**Intro Bio Questions and answer archive.** Use the find function to search for topics of interest

1        Q: What constitutes a monomer? And if steroids are not monomers, what are they

1        A: The use of the word \"monomer\" implies that there is a higher order form of the molecule consisting of more than one monomer linked together. The minimum use of a monomer would be to form a dimer, such as fructose in forming sucrose (forever a dimer). The word can also be applied to large molecules such as polypepides: thus hemoglobin is made up of 4 monomer subunits. So a monomer is not synonymous with \"small molecule.\" A small molecule could be a monomer if a polymer incorporatiing it exists, such as glycine being a monomer for a polypeptide. Other small molecules may never be incorpated into a polymer, such as folic acid (a vitamin) or testosterone (a steroid hormone). Such small molecules do not have the right to be called monomers. ',1),(###-09-22&&

2        Q: In Lecture 4, you mention that asp-asn is an H-bond to ionic, but then in 3i) of exam 2002, the answer says that no ionic bonds can be formed between peptides A and B. What about the \"H-bond to ionic\" asn-asp

2        A: An ionic bond is a bond between two ions. The electrical attraction between a polar (un-ionized) group (a dipole) and an ion is not really an ionic bond, not a full ionic bond. I have called it a combination ionic, but it may be better described as \"charge-dipole interaction.\" Such interactions are usually described in the context of \"solvation\" of an ion by water dipoles. So this less overlapping name is probably a better idea, and hopefully eliminated the apparent conflict. ',1),(###-09-22&&

3        Q: In 5a of the 2002 exam, how do you know that Vmax won\'t be reached at substrate concentration = 2Km

3        A: Because the shape of the Vo vs. S curve. Vmax is never reached, it is only approached asymptotically. 1),(###-09-22&&

4        Q: Is it safe to say that any cell-produced inhibitor is allosteric, unless it\'s the example you mention above? Or are there cell-produced, physiological competitive inhibitors, too

4        A: As far as I know all inhibitors used to control cell metabolism are of the allosteric type. *1),(###-09-22&&

5        Q: Is it true that all allosteric inhibitors are non-competitive, but that not all non-competitive inhibitors are allosteric

5        A: No, allosterics are in a separate class. They do not obey the simple Michaelis-Menten kinetics that we have presented here. That is, even the curves for Vo vs. S are different. The mechanism is more complex. Non-competitve inhibitors on the other hand do follow Michaelis-Menten kinetics, in the way shown in lecture. Indeed they are defined this way, by their kinetic behavior. A mechanism by which a non-competitive inhibitor blocks a catalytic site without blocking substrate binding and without distorting the enzyme would explain this kinetic behavior (lower Vmax, same Km).',1),(###-09-22&&

6        Q: One of my Chemistry professors made a specific point during our discussion that catalysts don\'t alter the activation energy, they alter the mechanism by which the reaction proceeds, and this alternate pathway corresponds to a lower activation energy. He was very emphatic about how

incorrect it was to state that \"catalysts act by reducing the activation energy\" - yet this is what is in your notes and the text. Can you reconcile these seemingly contradictory statements for me

6        A: I\'m glad you brought this up and got an expert opinion from a chemist. I have always felt a little uncomfortable talking about the activation energy being \"lowered,\" even though that\'s what it says in most biochemistry texts. I think your chemistry professor\'s explanation is much more reasonable and provides a better insight into what\'s going on. The energy needed to reach a transition state by the simple collision of the two molecules represents the activation energy for that situation and nothing can lower it for that situation. The catalyst brings the two molecules together by a different mechanism, (binding them side by side often) and a transition state can be reached in this situation with much less input of energy. We don\'t need the great kinetic energy necessary in the uncatalyzed reaction. So I like his phrasing. Nevertheless the fact remains that in the presence of the catalyst, the activation energy needed is less, it is lower, than in the uncatalyzed situation. So I can understand why one might say \"In the presence of a catalyst the activation energy to reach a transtition state is lower.\", and then \"Catalysts work by allowing a lower activation energy\" and then it\'s short step to \'Catalysts act by lowering the activation energy\" which could even be considered to be literally if loosely true, but I agree is misleading. Indeed, I plan to incorporate this way of explaining catalysts into future years\' lectures. Thanks. *1),(###-09-22&&

7        Q: I want to know more about the mechanism behind noncompetitive inhibition. It apparently doesnt seem to distort the enzyme in the slide, yet it\'s binded to another site. Does it repel substrate at the binding site? It\'s just unclear to me how it\'s lowering reaction velocity.&&

7        A: No. The substrate binds just fine in non-competitive inhibition. The inhibitor does not interfere with the substrate binding, it does not compete with it for the substrate binding site, thus it is called non-competitive, when this situation is seen. It could interfere with a side chain that is important for catalyzing the reaction once the substrate is already bound. Thus the catalysis would not take place, even though the substarte is bound. ',1),(###-09-19

8        Q: In problem 2-11b, why are the 26 spots if in part A, leu -> ala and both are nonpolar

8        A: Leu and ala are both non-polar. They both appear in the non-polar category of pages in books showing the categories of amiono acids. Why are they non-polar? Because they have side chains made up of hydrophobic pure hydrocarbons (-CH2- or -CH3). But leucine has a lot more of these non-polar groups than ala,which has but one. And the side chains must \"compete\" with the polar end of the molecules to establish the overall character of the molecule. So leu with its many hydrocarbon carbon atoms is a lot more non-polar than ala. All amino acids with predominantly non-polar side chains are non-polar but some are more non-polar than others.  ',1),(###-09-19

9        Q: In problem 1-23 (16th ed.rev.), how do you generate the chair form of galactose from the Haworth ring forms given in the texts

9        A: It is not necessary to do this to answer the question (part b). You know from the Haworth or even from the straight chain (Fisher) projections that glucose and galactose differ only in the orientation of the OH at C4. The chair form of glucose shows that the orientation of the OH at C4 in glucose is equatorial and that the H is axial up. . Therefore in galactose it must be the other way around: now it is the OH that is axial up. There are rules and conventions for following the H\'s from a Fisher projection all the way to a chair, but they require knowing (memorizing) the conventions and

probably the assumption that the most stable of the chair forms is the one that maximizies the nimber of equatorial OHs.  ',1),(###-09-19&&

10      Q: Are phospholipids the only structures that emulsify fat

10      A: Actually, the more important fat emsulfiers are the bile acids, which are acidic (-COOH) forms of steroids and not phospholipids at all. I didn\'t use these as examples because I didn\'t want to introduce too many different molecules.  ',0,10,1,1),(###-09-19&&&&

11      Q: Are trans-unsaturated fatty acids always solids or is the point that they are more likely to become solid whereas cis- unsaturated fatty acids will always be liquid

11      A: I always avoid the word always. \"Tend To\" serves nicely. Life is not black and white. You can imagine a fat with a mixture of many types of FAs, even a mixture within the same triglyceride, 3 different FAs. And unsaturateds could have 1, 2, 3 or more double bonds. So you could guess based on the majority; Wholly unsaturateds will be solids at room or body temperature. For the oils it depends. Trans-unstaurateds will tend to be less liquifying than cis, resembling saturateds more than unsaturated, it is thought. You can think of the mechanism, the facility with which the hydrocarbon side chains can get together without restraint will govern the tendency to form solid fat.  ',0,11,1,1),(###-09-19&&&&

12      Q: In problem 2-16, two proteins are subjected to ultracentrifuge. The question states, \"...the variant has 1 less ile and 1 more lys per 100 amino acids total.\" The word \"total\" at the end threw me off. Does this mean there are 100 amino acids in each protein, or does it mean simply 1 less ile and 1 more lys per every 100 amino acids contained in the protein

12      A: It\'s the second alternative -- there are NOT 100 amino acids in every protein. We are just normalizing by counting changes per 100, as you say.  ',0,12,1,1),(###-09-19&&

13      Q: Are domains equivalent to a protein\'s individual subunits

13      A: No. Protein subunits are separate polypeptides that are associated mostly with weak bonds (the exception being disulfides) to other polypeptide molecules to form the quaternary structure of a multimeric protein. A polypeptide is defined by amino acids linked to each other in a linear array via peptide (amide) bonds. Each polypeptide will have a free amino and a free carboxyl end. Domains, on the other hand, are PARTS of polypeptides. A domain need not have a free end, it represents a region of a continuos polypeptide, usually an internal region, although there could be two terminal domains for each polypeptide (an N-terrminal domian and a C-terminal domain). The domains are defined by their autonomously folded structure. That is, a region of contiguous amino acids along the polypeptide chain folds up on itself, forming a domian. Then these folded regions get together to form the overall 3-D shape of the polypeptide.  ',0,13,1,1),(###-09-19&&

14      Q: Is it possible that fingerprinting could also be used to separate amino acids rather than proteins and sub-peptides alone

14      A: But then it should not be called \"fingerprinting,\" should it, since all proteins would give the same pattern, the very antithesis of a \"fingerprint.\" So we have a semantic yet non-trivial issue here that could lead to conceptual confusion. Certainly the technique of 2-D separation used in

fingerprinting could be applied to the separation of amino acids rather than peptides.  ',0,14,1,1),(###-09-19&&

15      Q: Can entire proteins be subject to the 2D technique used in fingerprinting

15      A: Only fragments, up to 25, say.. Full sized proteins usually precipitate in the presence of organic solvents due to dentauration and the formation of entangled networks. ',0,15,1,1),(###-09-18&&

16      Q: I\'m still confused about what we need to memorize in terms of amino acid structure.&&

16      A: If there is a question requiring knowledge of an amino acid structure, you will be given than structure or, more likely, be given the same sheet that was given to you as a handout. You are expected to be able to recognize the properties of the amino acid side chains (charged, polar, non-polar, etc.) from examining their structure, fromyour knowledge (memorized) of the properties of the functional groups. For instance, we would expect you to be able to predict the properties of a new amino acid (from Mars or discovered in a fossil) that is not on your list of 20 but whose structure is presented to you and that contains only familiar functional groups. ',0,16,1,1),(###-09-18&&

17      Q: In the Becker book on page 65 looks to me like an equatorial C1 (beta) to axial C4 connection, which I thought would produce this kink and result in a helical structure, but I know that cellulose is straight chain because it produces microfibrils.&&

17      A: It is not equatorial- axial in Becker, it is eq-eq. You are being fooled by the Haworth pictures. What made you decide that a connection to a C4 OH that is depicted as down in Becker p. 65 is axial? Since in a Haworth projection ALL the bonds either point up or down they all have a connotation of being axial. But one is always equatorial and the other is axial, either up or down, in real life (chair forms). SO how do you figure it out, aside from memorizing? One way is to remember that the beta at C1 is by definition equatorial. So you know the H on C1 is axial. And since the Haworth picture shows it below the beta hydroxyl, then it must be axial DOWN. Now, you can also remember that the axials alternate their pointing direction as you go around the ring. So if the axial on C1 is axial down then the axial on C2 is axial UP, and it is the upper one in the Haworth projection, so we see that the OH on C2 is equatorial. Similar reasoning leads to the conclusion that at C4, the axial H is UP (as in C2) , and so the bond below it must be equatorial, and that\'s the one that\'s connected to the upstream glucose. So it\'s equatorial at C4 here, and the Haworth picture is not lying, it\'s just not depicting an equatorial orientation. ',0,17,1,1),(###-09-18&&

18      Q: The C6 CH20H groups seem to be in different places on alternating glucoses in the Purves book, whereas they\'re in exactly the same location in the Becker book.&&

18      A: Some authors like to flip every other glucose so that the bonds in the Haworth projections look like Z\'s instead of half-swastikas. But even Z\'s are not realistic, so I think it just adds opacity for not much benefit. ',0,18,1,1),(###-09-18&&

19      Q: Could the same technique used in fingerprinting be used to advantage to separate amino acids on basis of both polarity and charge

19      A: Yes, the 2-D separation could be used for simple amino acid separation, but 2 paper chromatographies would do also it without having to involve high voltage. ',0,19,1,1),(###-09-16&&

20    Q: Will we be required to know the properties of the amino acid side chains, as far as which are polar and nonpolar, and which are charged and uncharged, or will that information be provided to us

20    A: You will be given the structure of the 20 amino acids sheet that is a handout if relevant., but you have to recognize the properties of the side chain from knowing the properties of the functional groups and other atoms making up the side chain. That is, you should not memorize that serine is polar, but rather recognize it from knowing that the electrons are not distributed equally between the O and the H in an OH bond because of the difference in electronegatvity between O and H. It is this latter that you should memorize. For instance, the properties of an imaginary Martian (new) amino acid might be asked for, given the structure; then you\'d be out of luck if you just memorized the classification of the 20 earthly amino acids.  ',0,20,1,1),(###-09-15&&

21    Q: In lecture 3, you say, \"The monomers that make up proteins are the amino acids, of which there are 20. The same 20 in E. coli and in elephants and in tomatoes.\" I am a vegetarian, and several people have told me that it is unhealthy to be a vegetarian because only 19 of the 20 amino acids can be obtained from non-animal sources. What\'s the deal

21    A: Vegetables use all 20 amino acids, but most vegetables contain them in a different proportion than do mammals. You can synthesize 11 from glucose yourself, so you don\'t have to worry about those. But you need to eat the other 9 (the \"essential\" amino acids). Thus if you ate only certain vegetables you might be deficient in those essential amino acids that are low in popular vegetables but relatively high in mammalian proteins, like lysine and tryptophan. It\'s not a problem if you make sure to eat a good amount of vegetables that do contain decent amounts of these amino acids. A combination of rice and beans is the classic remedy. Of course if you drink milk or eat cheese or eat eggs you\'re also covered, all these foods are filled with animal proteins.  ',0,21,1,1),(###-09-14&&

22    Q: In my notes and in the Becker text, the maltose molecule is formed by an alpha/alpha glycosidic bond between two glucose molecules. However, on page 44 of Purves, the maltose molecule is formed between an alpha and a beta glucose. What gives

22    A: No, there is no alpha-alpha bond to speak of here. Maltose is formed via a bond between a molecule of glucose in the alpha form at carbon 1 and the C4 of a second glucose molecule whose oreintation at C1 is irrelevant. This it is not an alpha-alpha bond but rather an alpha 1- 4 bond. C4 has no alpha or beta, since that is not an anomeric carbon (its hydroxyl is fixed, doesn\'t change during ring formation). Confusion creeps in when one is forced to draw a molecule of the maltose disaccharide. The artist must do something with the C1 position of the glucose moiety that is contributing the C4 to the bond. Some authors will make it alpha and some will make it beta. In reailty in solution, the two forms slowly interconvert. So the \"right hand\" glucose is whatever the author commits it to be when placing pen to paper. And then she is stick with having to name it alpha or beta, admitting what she has done, feeding the confusion. Bu tthere\'s no other way to do it, I fear, short of speeded up computer animation.  ',0,22,1,1),(###-09-14&&

23    Q: In problem #2-2 (16th ed. rev), should we count the charge of the various amino acid groups? How do the relative pH values (say pH 6 vs. pH1--both acidic), influence the answer

23    A: pH6 is not very different from pH7 (10-fold). The carboxyl and amino groups will still be charged the majority of the time at pH6. At pH1, the concentration of H+ ions is now 100,000 times

higher than at pH6. This concentration is high enough to shift the equilibrium concentration of the carboxyl group far toward the COOH (protonated) form. At pH12, the concentration of H+ ions is only 10^-12, 100,000 times lower than at pH7. This low concentration is sufficient to swing the equilibrium concentration of NH2 group fartoward the uncharged (deprotonated form). The dissociation reactions we are talking about here are: R-COOH (protonated) <--> R-COO- (deprotonated) + H+ and R-NH2 (unprotonated) + H+ <--> RNH3+ (protonated). The equilibrium constant for the latter reaction is, for example: K= [RNH3] / [RNH2][H+], where the concentrations indicated are the concentrations at equilibrium. From the equilibriumm constant formula, one can see how the H+ concentration influences the ratio of the protonated to unprotonated forms of the acids and bases. You can\'t easily tell from looking at an acid or a base how strong it is, and thus exactly how it is affected by pH, but we tell you here that the carboxyl group is charged at pH7 and protonated at extremely low pHs and the amine group is protonated at pH 7 and unprotonated at extremely high pHs.  ',0,23,1,1),(###-09-14&&

24      Q: In problem 2-9F (16th ed. rev), could you please explain how one would arrive at determining that mutant #1 is the answer

24      A: Polypeptide overall folding is determined by the interaction among side chains. The normal protein has val at a certain position. If the is amino acid is important for proper folding, and therefore for proper function, then its substituion by a similar amino acid, such as leu, may not be that serious a change (you need your sheet of amino acid structures out to answer this.) For instance these very hydrophobic side chains may be burried in the interior of the protien, where the are acting to help keep the protein folded via hydrphobic forces. Even ala may not be too bad, but ser and lys are hydrophilc, so should abrogate this kind if interaction. Between these two, lys is charged and is therefore even more hydrophilic. So if we have to choose one (and we are asked to do just that) lys is the best choice: it represents the most extreme change from val among the changes listed. ',0,24,1,1),(###-09-14&&

25      Q: In Problem# 2-13 Part D (16th ed. rev), I tried working this out mathematically, but somehow I don\'t see how one gets +3 and at low pH.&&

25      A: At low pH,the carboxyls will be protonated and uncharged, while the amines will be charged. Peptide A has 3 amiono groups, one on the N-end and 2 from the 2 lysines side chains. Thse will give a charge of +3 altogether. There are 2 carboxyls, one from the C-end and one from the asp side chain. These will be uncharged at low pH. and so do not contribute to the net charge on the molecule. So the answer is +3. At high pH the answer would be -2, which is a lesser absolute charge than at low pH (+3), so high pH is where you will get the maximum absolute charge that is possible on this molecule (+3 vs. -2).  ',0,25,1,1),(###-09-14&&

26      Q: In Problem #1-21(16th ed. rev) I do not understand why *KH2PO4, *Na2HPO4, and Na2HP*O4 would not also work? Is the rule such that the radioactive component need be the phosphate molecule only (as indicative of the given answer), or can we suppose that any phosphate-containing molecule would suffice (present in the choices listed)

26      A: It is the atom that exists in isotopic forms; here 32P is the radioactive isotope that otherwise behaves as the predominant non-radiaoactive form (31P). In *KH2PO4, it is the potassium atom that is radioactive. Potassium would exist as dissociated K+ ions, not strictly bonded to any organic molecule. ',0,26,1,1),(###-09-13&&

27    Q: Do we have to worry about the pKas of protonating (or deprotonating) these R groups, or can we just assume that at low pH, negatively charged R groups will become neutral and at high pH, positively charged R groups will become neutral? Can we likewise assume that UNCHARGED, polar groups (like the amino acids serine, threonine, cysteine, tyrosine, asparagine, and glutamine) WILL NOT acquire charged R groups, regardless of the pH (high and low?), and this would allow us to make broad generalizations without worrying about specific pKas

27    A: Yes. ',0,27,1,1),(###-09-13&&

28    Q: In problem 1-22b (16th ed) why does the hydroxyl on Carbon #1 have to flip from alpha to beta in order for a 1-6 bond to form between two NSG molecules. Why couldn\'t it be alpha 1,4

28    A: Nothing complicated here. It is simply that this natural polymer, as reported in Science in 1999 was found to be one in which \"all linkages are beta 1 -> 6.\" It could have been alpha. It wasn\'t. I general, the monomers in polymers are linked in the same way, repeatedly: beta-1,6 - beta -1,6 - beta 1,6 here. Or one could imagine a more complex situation of, say: sugarA - beta-1,4 - sugarB - alpha -1,4 -sugarC alpha 1,4 - [ sugarA - beta-1,4 ... - sugarB - alpha -1,4 -sugarC alpha 1,4 ]n - ------- ; But that still a repeat of the same 3 linkages over and over: ABC, ABC. What you don\'t find is irregularity: A-B-C-A-A-A- B-C-C-C-C-B-C-A-A-C-B-A (not seen)  ',0,28,1,1),(###-09-13&&

29    Q: How often do the ring forms of gucose switch from alpha to beta, and what are their proportions

29    A: The half-life of a glucose ring form in water is about an hour. The equilibrium proportions of the different forms are approximately 60% beta, 40% alpha, and 0.1% straight chain. ',0,29,1,1),(###-09-11&&

30    Q: Why are the chair drawings named both Glucopyranose and glucose? Are they different variations of glucose? Is this a nonmenclature question

30    A: Yes, just nomenclature. Glucopyranose is more formally accurate, but we will use glucose. Since some texts use the fancier term, I put it in there.  ',0,30,1,1),(###-09-10&&

31    Q: About the answer to 1-5 B (which is \"Stationary phase\"). Doesn\'t the stated correct answer assume that the lag phase is the same for both medium? (If the assumption is correct, then I agree with the answer.) Could the lag phase theoretically be longer for the second medium? If so, then after the 10 hours, when measuring the number of bacteria in both media, medium 2 could be overtaking medium 1, even though both are in the growth/lag phase? It\'s a small chance (that the measured number of bacteria is the same) because of exponential growth, but theoretically could it happen

31    A: The lag time may be different for the two, but the difference (if any) should be small compared to the total time involved of 10 hrs. That should be enough time for both to reach stationary phase (given the short doubling time of bacteria). Note that the question asks about what phase the baceria are PROBABLY in. A lot of things are possible, but we are asking for the most likely answer. ',0,31,1,1),(###-09-09&&

32    Q: Regarding the1-8 and 1-9 questions. It refers to \"ordinary 5-carbon\" sugar. Does this include the aldosugar and ketosugar type

32      A: Yes. ',0,32,1,1),(###-09-09&&

33      Q: Is it only the aldosugar type that can form a ring

33      A: No. See handout for ring form of fructose, a keto sugar.  ',0,33,1,1),(###-09-09&&

34      Q: Can you give a more in depth definition of an anomeric carbon

34      A: It is the carbon with the double bond to oxygen. That double bond can be attacked by a hydroxyl, creating a new hydroxyl from the previously double bonded oxygen, and it is this new hydroxyl that can end up in either the alpha or beta conformation. ',0,34,1,1),(###-09-09&&

35      Q: Is it true though that this carbon in the ring form of sugar is no longer part of a carbonyl functional group

35      A: True, the carbonyl is C=O, which is no longer present in the glucose ring. The oxygen in the ring is said to be part of a hemiacetal, if you want to probe more deeply into the nomenclature (not recommended). ',0,35,1,1),(###-09-09&&

36      Q: Is the carbon that the 1-carbon bonds to through the O always the next to last as it was the 5-carbon in glucose, would it be the 4-carbon in an \"ordinary pentose sugar?\"&&

36      A: Yes, as it happens.  ',0,36,1,1),(###-09-08&&

37      Q: How can one experimentally find out if an organism is prokaryotic or eukaryotic (without looking at the cells with a microscope)

37      A: There are biochemical \"markers\" that are characteristic of cells with a true nucleus that one could analyze in the laboratory. Since eukaryotes have a true nucleus, it must be made of something, and most machinery in the cells has a protein component, so one could check for nuclear membrane specific proteins (there are 50 or more). Additonally, the DNA is packed into a true nucleus using packaging proteins that are distinct from DNA-associaed proteins in prokaryotes. These are the histones, and there are about a half dozen of them. I\'m sure there are more, but these come to mind as proteins that are common to all eukaryotes and are not present in prokaryotes.  ',0,37,1,1),(###-09-07&&

38      Q: Regarding problem #1-9 B: I don\'t see how the answer would coincide with the explanation given in the back of the book.&&

38      A: One way to do this problem is to compare the straight chain forms. There it is easy to see the differences between galactose and mannose: on C2, the OH in mannose is down and in galctose it is up. The opposite is true for C4. The realtive postions (up or down, left or right) in the straight chain form written in 2 dimensions reflects the relative positions above and below the ring. So if you must switch the OHs in the straight chain form on C2 and C4, do it also for the ring form, switching the positions of the H and OH on C2 and C4. C1 is given in diagram of this problem, so to find C2 and C4 just walk around the ring, but don\'t walk over the ring O, that would be in the wrong direction, since the numbered C\'s are contiguous. You can identify C1 in the ring as the carbon that is attached to the ring O and that has an OH as well. And you could find C6 easily in glucose if you remmber it is the one that hangs outside the ring.',0,38,1,1),(###-09-01&&

39      Q: What is the difference between steady-state and the state of equiliblrium

39      A: At equilibrium reactants are forming products at the same rate that products are forming reactants, so there is no net change in the concentration of either (R <--> P). In steady state, reactants are forming products at the same rate that products are going on to form something else (NOT back to reactants) so there is no net change in the concentration of products, the products ar said to be in a steady state (R--> P --> P2). What the two situations have in common is that the concentration of products (P) is not changing. But they are very different aside from that, for there is no net flow of material in equilibrium, whereas in the steady state there is a steady flow of material from R to P (and thence to P2). Life is a steady state situation, almost never an equilibrium situation, indeed it is a constant struggle against equilibrium.',0,39,1,1),(###-09-01&&

40      Q: How do you know whether two glucose molecules are alpha or beta linked

40      A: Alpha and beta refer only to the anomeric carbon. There is only one anomeric carbon in most sugars. It is the one with the carbonyl group in the straight chain form. If two sugars are linked together by each of their anomeric carbons (as in the s disaccharide sucrose, for example, then both anomeric orientations must be given in order to describe the bond. For instance, the designation glucose (alpha 1, beta 2) fructose would mean an alpha glucose bonded to a beta fructose. The glycosidic bond locks in the alpha or beta conformation, since the sugar ring can no longer open. On the other hand, in the much more common case, in which the anomeric carbon of one sugar is bonded to a non-anomeric carbon of a second sugar, such as in maltose, then the conformation about the anomeric carbon of the second sugar is not designated, since one can not know it, as that sugar ring is opening and closing, with both anomers represented in the population of disaccharides. Thus maltose is glucose (alpha 1, 4) glucose.',0,40,1,1),(###-09-01&&

41      Q: Are monoglycerides considered fats as well as triglycerides? If so, could a group of saturated monoglycerides could make up animal fats? If not, are mono and diglycerides just intermediates in the synthesis of triglycerides, or do they serve some purpose

41      A: Triglycerides are by far the most common kind of fat found in multicellular organisms. Mono- and di-glycerides are also considered fats, at least from a dietary point of view. They would, of course, also be intermediates on the way to triglcerides, which may be considered fats par excellence. One function of mono and di-glycerides is as emulsifiers, at least commercially. There may be other functions.',0,41,1,1),(###-09-01&&

42      Q: In the Polyacrylamide Gel Electrophoresis Lecture, the diagram showing the apparatus containing the gel and the highlighting the positions of the large and small charged and uncharged molecules post migration. Why are only positively charged species shown

42      A: The protein mixture is usually added to a well in the gel at the top, so the proteins with a net negative charge would migrate up into the buffer reservoir and never enter the gel, and are lost from the experiment.',0,42,1,1),(###-09-01&&

43      Q: Does succinic dehydrogenase use the same mechanism as platinum binding hydrogen iodide to catalyze its reaction

43    A: In general, enzymes do not act like the inorganic catalysts. They use more powerful and sophisicated mechanisms to achive a great rate of catalysis. A common feature is to provide a surface for binding the reactants, so that they can be placed in proximity to each other. But I would expect the affinity of the enzyme for fumarate would be much greater than the affinity of platinum for fumarate. (Pt will also catalyze the hydrogenation of C=C double bonds).',0,43,1,1),(###-09-01&&

44    Q: What is the difference between allosteric and non-competitive inhibition

44    A: The distinction is not so clear, especially because many texts do not make the distinction, describing non-competitive inhibitors as \"distorting\" the protein from a remote site (which is equivalent to allostery). Non-competitive inhibition is primarily defined on the basis of the effect on enzyme kinetics: insensitivity to substrate concentration. Such insensitivity could be brought about by interference with the activity of the enzyme without distortion and without affecting substrate binding, as I tried to explain in class: e.g., by blocking or masking a side chain necessary for the catalysis (Hg++ masking a sulfhydryl, or a small organic compound binding to a Cu++ which acts as a necessary prosthetic group). Allosteric inhibition on the other hand always involves a distortion or change in the shape of the protein. The distinction becomes easier when you consider the mechanism of allostery in more detail (not in this class): allosteric enzymes usually do not display Michaelis-Menten kinetics, yielding an S-shaped curve of dependence on substrate concentration. The S-shape implies cooperatively among substrate molecules: once one binds more bind. This in turn implies that allosteric enzymes must be multimers with multiple substrate binding sites (one per subunit, e.g.). So they are more complex. I find it useful to keep the distinction as distortion (allostery) or no distortion (non-competitive inhibition), even thought thisneed not not strictly be true: non-competitive inhibition could work through distortion. We could drop non-competitive inhibition from the discussion altogether, except it provides a nice comparison to the competitive mechanism. See also: <a href = \"http://users.rcn.com/jkimball.ma.ultranet/BiologyPages/E/EnzymeKinetics.html\">http://users.rcn.com/jkimball.ma.ultranet/BiologyPages/E/EnzymeKinetics.html</a>',0,44,1,1),(###-09-01&&

45    Q: Why is tryptophan classified as a nonpolar amino acid, as it has a partial charge separation

45    A: I admit it\'s difficult to categorize tryptophan, as it has a lot of hydrocarbon, yet it has a highly polar if not charged N atom. I would say it can behave as either depending on the exact immediate environment. We will not discuss try or his for these reasons, it\'s too complicated for a course at this level.',0,46,1,1),(###-09-01&&

46    Q: Becker (4th Ed., p. 35) and Purves seem to give conflicting accounts of the polarity (Becker) or hydrophobilcity (Purves) of cysteine.&&

46    A: Cysteine seems rather intermediate, halfway between serine and threonine on the one hand and valine, leucine etc on the other. See scales 2 and 3 at the URL below: <a href = \"http://prowl.rockefeller.edu/aainfo/hydro.htm\">http://prowl.rockefeller.edu/aainfo/hydro.htm</a>',0,47,1,1),(###-09-01&&

47    Q: The book highlights only one OH (from C2) in each glucose as being a participant in the H bonding. Is this so

47    A: I think they are just trying to keep it simple, uncluttered. Actually, in one form of crystallized cellulose, it is the C6 and C3 that do all the intermolecular H-bonding, with C2 and C5 involved intramolecular H-bonds. See: <a href =

48      Q: In his portrayal of cellulose, Purves (6th ed., p. 45) inverts every other glucose molecule such that on one, the C6 is up and on the other, it is down? Why

48      A: This strategy gets him out of the problem of having to draw the glycosidic bond with bends in it (on paper), certainly not the case in reality. This is a problem with the Haworth depiction:  /| O |/ ',0,49,1,1),(###-09-01&&

49      Q: In a cellulose chain, do all of the equatorial OH groups (C2 and C3) participate in hydrogen bonding with other chains in the formation of fibrils? Does this include the OH group on C6

49      A: It seems that the C6 is most likely to be involved in interchain H-bonding. One would have to do a detailed analysis of the structure to find out.',0,51,1,1),(###-09-01&&

50      Q: What prevents alpha 1,6 glycosidic linkages on cellulose

50      A: It\'s a good question since all plant cells make cellulose for their cell walls, and some of those plant cells are also making starch. I\'m not sure. I can think of three possibilities: 1) The enzyme (catalyst) that forms the 6,1 bond recognizes a large part of the sugar carrying the C6, and rejects it if it has a C1 in the beta position. 2) Cellulose is made in a special compartment within the cell, and the branching enzyme is not allowed in that \"compartment.\" 3) The C6 OH in cellulose is not accessible because it is involved in \"locked in\" hydrogen bonding with other cellulose molecules. In support of this idea, in cellulose crystals the C6 OH is found to be hydrogen bonded to a C3 OH of the neighboring strand.',0,52,1,1),(###-09-01&&

51      Q: You say that a sucrose disaccharide is built from a 1-2 alpha-alpha link between glucose and fructose., but Becker (4th Ed., p. 64, Fig. 3-23), shows sucrose as a 1-4 glycosidic bond and calls it alpha-beta. Which is correct

51      A: 1-2 is correct: the glycosidic bond in sucrose is from the C1 of glucose to the C2 of fructose. This is actually what the Becker figure shows: if you look closely, the fructose has been turned upside down to able to be next to the C1 of glucose (Hint: the anomeric carbon is the one that has the ring oxygen attached to it). Both are the anomeric carbons of their respective sugars. In sucrose, the C1 glycosidic bond comes from alpha glucose but the C2 from fructose comes from beta fructose (hard to tell, but that\'s what is known). Thus my notes and Becker 4th edition legend to Fig. 3-23 are incorrect in implicating alpha-fructose here. Becker\'s 5th edition, and my corrected notes, (thank you), have got it right.',0,53,1,1),(###-09-01&&

52      Q: Why is the ring conformation of glucose more stable in aqueous solution when this seems to minimize entropy

52      A: Stability and entropy are not equivalent. The direction of chemical transformations is influenced by considerations of entropy but are not solely determined by the movement to maximize entropy. And a good thing, too, or all would chaos by now. You can put in energy in the form of enthalpy to counteract the trend toward increased entropy, thus creating order out of disorder.

Ultimately we use the energy of the sun to effect this process as we create the highly ordered structures necessary for life. Despite it\'s more ordered structure, the arrangment of atoms (the hydrogen and carbon and oxygen atoms involved) in the ring for of glucose is more stable, the bonds are stronger, due to the particular arrangement of electrons around the atoms in the ring form as compared to the straight chain. This effect overrides the small entropic cost. ',0,54,1,1),(###-09-01&&

53      Q: What determines which conformation glucose decides to exist in, or does glucose constantly interchange between them

53      A: Glucose in an aqueous environment is constantly interchanging among the 3 predominant forms we discussed, the straight chain, the alpha ring and the beta ring. The 3 forms are in equiilibrium and each is present at a constant proportion of the total at any given point in time. The equilibrium concentration of the 2 ring forms are about equal but are much higher than that of the straight chain. The predominance of the rings lies in the fact that they are more stable: it is harder to break the ring C-O bond than it is to form it. Nevertheless, some of the large number of rings will break, by chance, giving rise to the straight chain, which will subsequently recyclize. At any given point in time there will be a certain concentration of the straight chains, molecules caught between ring breakage and re-cyclization.',0,55,1,1),(###-09-01&&

54      Q: In an aqueous solution of NaCl, are the water molecules forming hydrogen bonds with the Na+ and Cl- ions

54      A: The interaction of water with ions does not always involve hydrogen atoms as intermediaries (HOH with Na+), so can\'t be considered H-bonds, but rather simply a partial ionic bond, betwen a fully charged ion and a dipole (water). The true hydrogen bond involves a lengthening of the covalent O-H bond as the H is attracted to the neighboring molecule, and requires that all three concerned atoms be arranged in a straight line.',0,56,1,1),(###-09-01&&

55      Q: If the association between hydrophobic molecules in an aqueous medium doesn\'t really consist of bonds, where exactly does the 3 kcal/mol necessary to break this \"bond\" go? And when you say 3 kcal/mol-- per mole of what substance

55      A: Good question. I\'ve been using that number for years. It must mean that if you had a mole of octanes coalesced in an aqueous environment, it would take 3 kcal to disperse them. What doesn\'t fit so easily with that answer is the mole part, since if you had a mole of butane (a 4-carbon hydrocarbon) it seem likely that you would need less energy to disperse those, as they, being smaller, would have \"ordered\" less water molecules. The 3 kcal estimate may simply refer to a mole of a hydrophobic biological molecules of \"average\" size (10 carbons?)',0,57,1,1),(###-09-01&&

56      Q: I am fully self-taught and was never able to get anything from lectures; it is extremely difficult for me not to rely almost entirely on a good textbook. Should I take this Intro Bio

56      A: This course does not rely on the text as a primary source of information. Rather, it is the lectures that are emphasized, with readings in the text suggested to provide a larger overview and more detail to help you to understand the material in the lectures. So it is exactly the opposite of what you are looking for, according to your question. That said, there is a saving grace: all the material in the lectures is presented on the Web. The web lectures cover essentially all the important points in the course, although they are more succinctly presented than the live lectures. Some students prefer to take copious notes, learning best by writing down the  information; with other students it is just the

opposite. Thus if you have trouble taking notes and listening at the same time, you can sit back and listen, perhaps just jotting down a note or two on the handouts, with the assurance that everything you need to know will be there on the Web. Take look at least year\'s Web lectures, available via a link from this year\'s lectures index page. Finally, we\'d feel that the real learning of the course material takes place not in studying the lecture notes, but in doing the problems. A great number of problems are offered in the Problem Book and in a set of old exams.By doing all the problems, consulting the lecture notes and the text to do so, you gain an understanding of the concepts not usually possible from simple reading or thinking about the sentences you have read. Studying for the exams is also a time when much learning takes place. So there is a hierarchy here: you study for the exam problems by doing all the given problems, you listen to and study the lectures to do the problems, you read the text to help understand the lectures and finally you email questions to the instructors to deal with points with which you still have trouble.',0,58,1,1),(###-10-01&&

57      Q: In PCR, does the amount of primer added determine the number of replicated DNA sections that are produced

57      A: Yes. But usually you add lots and lots of primer.',0,60,2,1),(###-10-01&&

58      Q: Are primers destroyed after helping to intitiate replication

58      A: Yes if they are RNA ? in DNA replication. No if they are DNA used in PCR.',0,61,2,1),(###-10-01&&

59      Q: Why did Meselson and Stahl first grow the bacteria in N15 media for a long time and then in one generation in N14 media? We already know that the DNA in the E.coli contain N14 and not the N15 isotope, so why did they not just grow the bacteria in N15 for one generation and isolate/analyze that DNA

59      A: I\'m not sure about this one. I\'ve always assumed there was some technical reason why this worked better.',0,62,2,1),(###-10-01&&

60      Q: Regarding the Meselson-Stahl experiment: In density centifugation, why is CsCl in particular used? Given enough time, will all the CsCl fall to the bottom of the centrifuge tube

60      A: Partially, it\'s just arbitrary. CsCl solution happens to be just about the right density needed to separate the DNA. Any other similar molecule would work too. CsCl is so small and diffuses so rapidly that it will never all fall to the bottom.',0,63,2,1),(###-10-01&&

61      Q: Regarding the answer to problem 4-12: Are the two drawings of succinate correct? How could radioactivity be in both places

61      A: Top drawings in key: the radioactivity is not in BOTH places at once, but only on one carbon. In fact, as stated in the key the top two figures are identical as molecules; they\'ve just been slid around on the plane of the paper. Now when such SYMMETRIC molecules are converted to oxaloacetate (the lower figures, which are not citrate) a C=O has been substituted for 2 H\'s on one carbon in succinate. That carbon is one in from an end that is a carboxyl in succinate. But succinate has two symmetrically located carboxyls. So the conversion enzymes chooses one or the other, they appear identical to each other. They are identical to each  other .... UNLESS we biochemists add a radioactive label to one. Which one? We can\'t tell, they  are symmetric, but only one is labeled, not two. So when

the C=O gets put on, it has a 50-50 chance of being close to the radioactive carboxyl or close to the non-radioactive carboxyl, and you get the true mixture of (radioactively) distinguishable molecules shown in the lower figure. ',0,64,2,1),(###-10-01&&

62      Q: In problem 5-6 (F), is the carbon dioxide produced not be radioactive because it come from the oxaloacetate

62      A: In this case you have to examine the reactions and follow each carbon to see where it ends up. Since this is anaerobiosis, you need only look at the pathway of fermentation, say to ethanol and CO2 (although admittedly the problem stated bacteria, it\'s more interesting to entertain the possibility of ethanolic fermentation here). Use the handout that has the sugars in linear form to make it easier.  Label the top (C1) of glucose with a pink outliner and the bottom C (C6) with a blue outliner. Now label each intermediate as it is produced the same way. When you get to DHAP and G3P you will have the C bearing the phosphate group labeled. When DHAP get converted to more G3P, that is still the labeled carbon, the one attached to the P. It is the other 2 carbons that are involved in the isomerization, although even that does not change the relative positions of the C\'s. Maybe the label should be violet now as G3P continues (pink + blue). In 1,3 diPGA, the label is on the bottom, as the action (oxidation of the aldehyde to the acid took place at the top, where the old  P wasn\'t. So now we can describe the labeled C as the one at the opposite end from the carboxyl group. This definition stays with us until pyruvate. That carboxyl (Unlabeled) comes of on the next step as CO2, so you see it is not labeled.  The label in in the ethanol, at the end opposite from the carbon with the hydroxyl. Phew, it takes more time to explain it than to do it.',0,65,2,1),(###-10-01&&

63      Q: In problem 5-6 (E-F), how does one conclude anything about where the carbons come and go.&&

63      A: This one is actually easy because ALL the carbons and hydrogens are labeled. SInce all the CO2 comes from glucose and all the carbons in glucose are labeled then the CO2 must be labeled, there is no other source of NON-radioactive carbon after all. The hydrogen case is not so easy, since there is a great source of non-radioactive hydrogen, that is water. But the question  just wants to know if there will be radioactive water, and there must be, because the products of respiration are CO2 plus water, and the glucose is all gone, so those radioactive h\'s had to go somewhere, and water IS a product. So you can reason this answer without actually following the reactions one by one.',0,66,2,1),(###-10-01&&

64      Q: In fatty acid catabolism, it looks like in the first step we couple an ATP de-phosphorylation to adding on a CoA. Why is no ATP needed for coupling in the last step, when an acetyl CoA is split off and another CoA takes its place

64      A: The attachment of CoA to acetate is unfavorable (requires free energy) so the enzyme couples it (somehow) to the hydrolysis of ATP so that this new reaction will run in the required direction, like hexokinase in principle but more complicated mechanistically. For the next CoA addition, the coupling energy is coming from the hydrolysis of the C-C bond that is cleaved to form the 2 carbon unit, so no ATP is needed.  In general (although not always), cleavge reactions are free energy releasing and condensations to make a larger molecule are free energy requiring.',0,68,2,1),(###-10-01&&

65      Q: In problem 5-9, why do oxidized components build up after blocking the electron transport chain? (Since no electrons flow after the block, won\'t you get oxidized components BEFORE the block, but not after?) &&

65     A: Maybe you are fooled by the words. I am sometimes, and have to slap myself to back in line. When molecules GET electrons they get reduced. When they LOSE electrons they become oxidized. The downstream components can\'t get electrons and they\'ve given up their old electrons to oxygen down the line and they can\'t get any more because of the block, so they stay oxidized.',0,69,2,1),(###-10-01&&

66     Q: In problem 5-3B, why will electron transport stop entirely? Why doesn\'t it proceed to the point of the blockage and then stop

66     A: It will stop entirely at the point of blockage. No more electrons will be flowing, not one, so it is stopped, entirely. ETC components upstream of the block will be reduced (have the electrons). ETC components downstream of the block will be oxidized (have a dearth of electrons).',0,70,2,1),(###-10-01&&

67     Q: Is it true that at any given point in a double strand of DNA, only one strand can function as the template

67     A: That is correct. In other words, for a particular section of DNA, a \"primer could not attach to the 5\' end of one strand of DNA and another primer attach to the 5\' end of the complimentary strand (either at the same time or at different points in time)\". You are confusing DNA and RNA synthesis. You don\'t need a primer for transcription. Also nucleic acid synthesis starts at the 3\' end of the template (corresponding to the 5\' end of the newly made strand). However you are right that RNA synthesis does not start at one end of one strand sometimes and the other end of the complementary strand at other times.',0,71,2,1),(###-10-01&&

68     Q: It seems that only 50% of a double stranded helix contains relevant information for transcription. If this is true, why is it necessary for DNA to be double stranded

68     A: There are several answers to this. One is to facilitate replication. If the DNA were single stranded, you\'d have to make the complement before you could make more copies of the \"real\" strand. Another is to protect the DNA -- double stranded DNA is more resistant to damage and easier to repair than single stranded. However, from the point of view of information, one strand is all you need, and some viruses do use single stranded DNA (or RNA) as the genetic material. But all cells use only double stranded DNA.',0,72,2,1),(###-10-01&&

69     Q: Does DNA polymerase bind to origins, primers, or both

69     A: DNA polymerase (or the polymerase complex or more correctly the replicating machine or replisome) is a monster protein complex with many parts. The process of replication begins when part or all of the complex settles down on the DNA at the origin. We are treating it as one large unit and calling the whole thing polymerase. The actual polymerase part (the part with catalytic activity) may really bind at the 3\' end of primers (with the help of many other proteins in the complex that have already bound) since that\'s where the actual chain growth begins.',0,73,2,1),(###-10-01&&

70     Q: If it is true nucleotides are at least 3 nucleotides long, how does the tRNA know to read, say, a 4-nucleotide gene in its entirety instead of just the first 3 nucleotides in the gene

70	A: It\'s not correct that genes are always at least 3 nucleotides long. Codons, not genes, are 3 nucleotides long. Genes are usually thousands of bases long (or longer). There is a specific signal to start translation at a fixed place in the mRNA (details will be discussed; they differ between eukaryotes and prokaryotes) and then the mRNA is read 3 bases at a time to specify the chain of amino acids.',0,74,2,1),(###-10-01&&

71	Q: Why/How does the DNA during replication manage to unzip only a small amount at a time if DNA becomes completely denatured at its melting temperature (Tm)

71	A: Cells operate at a constant temperature. They don\'t unzip their DNA using heat -- if they did, they\'d cook all their enzymes. Specific enzymes help catalyze the breakage of H bonds (and unwinding of the DNA) ahead of the fork, sometimes using ATP to get the energy to unwind and unzip the DNA. There are some details in your texts and more in any biochem or mol. bio. book.',0,75,2,1),(###-10-01&&

72	Q: How do scientists build a length of DNA with a specific sequence entirely from scratch (i.e. with no prexisting template)? Is this how probes are made

72	A: Yes, this is how probes are made, usually, using chemical synthesis, not biological sources. There are chemical procedures (automated, too) for stringing together a series of nucleotides to make a probe. Each nucleotide is added to the growing chain, one nucleotide at a time. No template.',0,76,2,1),(###-10-01&&

73	Q: If prokaryotes don\'t have a nucleus, can they carry genetic information

73	A: Think about the implications -- if they had no genetic info, how could they produce offspring like themselves? If you don\'t have much background in biology, it might pay to take a look at one of the textbooks on basic cell structure, prokaryotes vs eukaryotes, etc. We skipped that for now, but it covers this sort of thing. Chap 4 in either text.',0,78,2,1),(###-10-01&&

74	Q: In the exam will the pathways handouts be annotated

74	A: I reserve the right to leave out a bit of annotation: e.g., ->NADH from NAD -> NADH, so you must know what in direction the oxidation-reduction is going at a particular step. But the exact steps where NAD and ATP are involved will remain annotated.',0,81,2,1),(###-10-01&&

75	Q: Why does starch, which is composed entirely of glucose, break down to G-1-P before entering glycolysis, whereas lactose or maltose are broken down directly to glucose

75	A: Actually it is glycogen that is broken down to G1P; starch is broken down to G1P in some bacteria, but in mammals it is simply hydrolyzed to glucose via the enzymes amylase (to maltose) and maltase (to glucose). I don\'t know the answer, but one rationale is that starch and lactose are ingested as food; the products are nutrients (glucose) that need to circulate (in the blood) to all the tissues. Phosphorylated compounds in general are not transported into cells. Thus the formation of G1P would be of no use here, and the hydrolysis reaction is given its full standard free energy change so that the reaction goes to completion. Glycogen on the other hand is an intracellular source of energy, at least in muscle. There the G1P generated in situ can be used (almost) directly in the cell\'s glycolytic pathway.',0,82,2,1),(###-10-01&&

76      Q: What happens to adenine when it\'s triphosphorylated? Is it ATP (adenosine triphosphate and not adenine triphosphate)

76      A: Adenosine triphosphate is A - Ribose - three phosphates. That is what we are talking about here in all cases. Adenine triphosphate would be A -phosphate-phosphate-phosphate without any sugar. That does not occur. Reaction 11-2 would be different if you got AMP from breaking down RNA. You would need one ATP to convert the AMP to ADP. Then you could use ox. phos. or substrate level phosphorylation to convert the ADP to ATP. (You wouldn\'t use one ATP to make ADP into another ATP. It makes no sense.) If you got deoxyAMP from breaking down DNA, you would use two (ribo) ATP to make deoxyATP.',0,83,2,1),(###-10-01&&

77      Q: Are 3 ATP produced per electron pair in oxidative phosphorlyation because each electron pair pumps about 10 H+ across the mitochondrial membrane, of which it takes 3 to bind to something?\r\n&&

77      A: The electrons flowing back through the FoF1 complex seem to bind to the c subunits in Fo, the part anchored in the membrane. Binding protons causes the complex of c subunits to rotate relative to the stationary a subunts (a, not alpha). This motion constitutes the motor. The motor turns the camshaft, which distorts the alpha-beta subunits of F1, generating the ATP from ADP + Pi there. It is thought that protons of some amino acid side chain at the interface between c and a subunits are involved: as c gives up a proton to a, it causes the rotation and then proton is lost from a and enters the matrix . Then c gets a new proton from the flow, and the process is repeated. For a nice PowerPoint explanation, see: <a href = \"www.ag.unr.edu/biochemistry/class/bch400-600/\">www.ag.unr.edu/biochemistry/class/bch400-600/</a>',0,84,2,1),(###-10-01&&

78      Q: If the outer membrane of the mitochondria is permeable to large molecules, why don\'t the protons pumped out of the matrix flow out to the cytoplasm

78      A: I have not been able to find an authorattive answer to this question. There are several possibilities. 1) Remember the inner mitochondrial membrane is in the form of highly invaginated cristae, so most of the inter-membrane space is really between two inner membrane \"walls\". That is, most of the H+ ions will be in a cove between two peninsulas, and not so close to the mouth of the cove) where the outer membrane is found (which is not invaginated, not part of the cristae). 2) There is an electrochemical gradient formed here, so perhaps the electric field (voltage difference, excess negative charge inside the mitochondria) is working against the diffusion away of the positive H+ ions. 3) It\'s too fast. The gradient is really high just outside the inner membrane and is instantly dissipated by flow back throught the ATP synthetase before much diffuson can occur. Perhaps it doesn\'t get much past 1/10 of the distance to the outer membrane. I have seen this explanation given but do not know the evidence. 4) They do. So what. Still plenty to flow back, and flowing back constantly, in a steady state.',0,85,2,1),(###-10-01&&

79      Q: If ATP is used all over the cell, how does ADP find its way back to the mitochondria to be rephosphorylated at a sufficiently high rate

79      A: Diffusion is fast enough (evidently); then there is an ADP-ATP exchanger protein (\"translocase\") in the inner mitochondrial membrane for the final leap.',0,86,2,1),(###-10-01&&

80      Q: Do yeast have lactate dehydrogenase, and, if so, why don\'t they utilize lactate fermentation

80    A: Yeast do have lactate dehydrogenase, at least Baker's yeast do. Yeast find themselves in many different environments, and lactate must be available sometimes. So they can use it for carbon and energy, but only in the presence of oxygen. In the absence of air, they would use NAD to convert lactate to pyruvate and then would have to convert pyruvate to ethanol + CO2 to regenerate the NAD. All this would get them nowhere as far as ATP production goes. Why don't they use LDH to regenerate NADH anaerobically, instead of going the ethanol route? I don't know; but there's probably a reasonable known answer,having to do with ecology, perhaps.',0,87,2,1),(###-11-01&&

81    Q: If the ordinary tRNA for met only fits in the A site of a ribosome, how would it carry a peptide chain into the P site

81    A: When it has an amino acid on it, it only fits in the A. Once it gets a peptide, it fits in the P like any other tRNA. But the special met-tRNA fits in the P when it only carries an amino acid.',0,89,3,1),(###-11-01&&

82    Q: When does AUG function as a start codon and when is it just another codon for an amino acid somewhere in the middle of a polypeptide chain

82    A: AUG also codes for plain old methionine in the middle of a peptide chain. You need a special tRNa to use AUG for start (and met simultaneously) and a second tRNA to use AUG to put met in the middle of a growing chain. Whether AUG is just met or both met and start depends on where it is in the mRNA.',0,91,3,1),(###-11-01&&

83    Q: In order to allow operons to be partially on or off, are there multiple copies of the same operon in DNA

83    A: There is only one copy of each operon per bacterium. But active repressor can be present at levels that are insufficient to tie up the operator in every cell all the time. Another explanation: we always measure enzyme production in a culture, not in a single bacterium. So it may be that every individual operon in each individual bacterium is either on or off (totally) but in the culture as a whole, at intermediate levels of inducer or co-repressor, some copies are on and some off.',0,92,3,1),(###-11-01&&

84    Q: If we insert an inverted gene into a bacteria's DNA via genetic engineering to produce antisense mRNA, do we also need to add a second promotor (one that binds to a different base sequence than the promotor for the the sense sequence)

84    A: Promotors are part of the DNA sequence. They are not additional. RNA polymerase binds to them. If you turn the gene around, you can use the same old promotor. Alternatively, you can move the promotor to the other end of the gene, and turn it around.',0,93,3,1),(###-11-01&&

85    Q: How does HIV produce the reverse transcriptase if it doesn't bring any enzymes, ATP, etc. with it into the cell

85    A: We never said it doesn't bring ANY enzymes in! It just doesn't have the ones for translation, energy generation etc. You have guessed correctly that it must carry reverse transcriptase in its head when it infects. The virus makes RT when it makes head protein, and packages some of the RT in its head to use in the next infection.',0,94,3,1),(###-11-01&&

86      Q: Since mRNA is degraded much faster than tRNA or rRNA, why is a mutation in mRNA worse than a mistake in a tRNA or rRNA molecule

86      A: There are many copies of each tRNA and rRNA. So if one is messed up, all the others will continue to function normally. That\'s why a \"bad\" tRNA or rRNA isn\'t so terrible, even if each individual molecule lasts a long time.',0,95,3,1),(###-11-01&&

87      Q: Should we understand how to design a tRNA so that it reads a UUG trp codon rather than a UGA stop codon (due to wobble)

87      A: You are expected to be able to do that IF you have a copy of the genetic code AND a copy of the wobble rules in front of you. Not by memory. But there are 3 stop codons, and the most important thing is to remember that there are NO tRNAs for the stop codons.',0,96,3,1),(###-11-01&&

88      Q: Do we have to memorize wobble rules

88      A: As always, the answer is NO. You have to know A goes with T/U and C with G but that is about it on the memorization front. No one really believes it, but understanding what all this means is way more important than memorizing the rules. You can always look up the rules!',0,97,3,1),(###-11-01&&

89      Q: Regarding the trp operon: When you add tryptophan, the enzyme production per cell declines, but why does it level out and reach a steady state rather than declining indefinately

89      A: Even if there is trp present, there is a little mRNA and a little trp synthetase made. The basal level of synthesis is very low, but not zero.',0,98,3,1),(###-11-01&&

90      Q: When performing PCR, it looks like you always get primer along with the target sequence. Is this true

90      A: If you consider the primer region to be outside the target, yes you get more than the target. But the primer is quite short relative to the target, so this fine pt. is usually ignored. (Primer is usually less than 20 bases long and target is usually thousands.)',0,99,2,1),(###-11-01&&

91      Q: In problem 7-5 A, I assumed that because DNA polymerase complex was added in excess that RNA would also be produced because primase (an RNA polymerase) is one of these components. Why isn\'t this true

91      A: Primase is a special RNA polymerase that only makes short primers. It will not transcribe a significant section of the DNA. The primers will be replaced anyway, and at the end you will have no RNA at all left.',0,100,2,1),(###-11-01&&

92      Q: Why is the promoter for DNA transcription double stranded

92      A: As I understand it, both strands of DNA are involved in binding RNA polymerase. This positions the enzyme so its \"front\" end faces in one direction or the other. Which ever direction it faces will be the direction in which transcription goes. (Not both). The promotor may be double stranded, but each promotor only \"promotes\" replication in one direction, and therefore on one strand.',0,101,2,1),(###-11-01&&

93      Q: How are RNA primers removed from the leading strand of DNA replication? This is clear in the case of circular DNA but what about linear unidirectional replication

93      A: You can remove primer on the end, but you can\'t replace the end primer on a linear molecule without enzymes we haven\'t discussed. This is the whole issue of telomeres and telomerase which we have postponed. (If you\'re curious, look in Becker.)',0,102,2,1),(###-12-01&&

94      Q: Are two versions of the genes on homologous chromosomes always transcribed simultaneously

94      A: In almost all cases, both alleles are transcribed (if possible). ',0,103,4,1),(###-12-01&&

95      Q: Would it be correct to say that a diploid organism with one allele coding for inactive enzyme ALWAYS produces 1/2 the amount of enzyme homozygous normal does

95      A: No. In some cases there is the equivalent of repression/induction -- regulation at transcription that compensates. But usually it is 50%.',0,104,4,1),(###-12-01&&

96      Q: About the female being \"mosaic.\" Assuming that in the beginning cells randomly inactivate one of the X chromosomes, is the only way to get more or less than 50% enzyme concentration per PERSON (not cell, of course) through unequal activation of the two X\'s, or do some cells divide more rapidly, and hence form larger \"patches\" -- so that inactivation of X\'s in some cells has more of an effect on overall concentration of active enzyme per organism.&&

96      A: Presumably, any patch has an equal chance of having either X on, so overall, the size of the patches doesn\'t make much difference in how much product of each X you have. (The size does affect the type of phenotype -- whether the mosaic is fine grained as in color vision or in large patches, as in cat fur color.) The random X inactivation is statistical, meaning that in some individuals more than half the cells use one X and less than half use the other. But in most people it comes out about 1/2 and 1/2.',0,105,4,1),(###-12-01&&

97      Q: Since the X and Y do have homologous regions, would a mutation on the X in one of these regions still cause a disease

97      A: There are other genes on the Y that are transcribed. Otherwise how would the genes there have any effect? The small number of homologous genes act in most ways like autosomal genes -- only if both copies are defective does a problem usually occur.',0,106,4,1),(###-12-01&&

98      Q: In problem 15-6A, I understand why you can select the mutant by slow growth on low substrate, but if you were able to detect slow growth on  high substrate, wouldn\'t that be a better indication of the mutant

98      A: On high substrate, both enzymes would be well above Km and both would work equally well (same high V) so you would not be able to tell the difference. There would be no difference in growth.',0,107,4,1),(###-12-01&&

99      Q: In problem 10-10 (A-2), why are the genotypes not 100% paternal if the genes are linked in cross of NnBb x nnbb? Wouldn\'t Meiosis only yield gametes NB and nb (N=normal, n=not, B=blue, b=white)

99    A: Linked does not mean 100% linked. It merely means that you get more parentals than recombinants. It could mean 5% or 22% RF which are different extents of linkage. It does not mean meiosis only gives parental gametes. Just more parentals than recombinants.',0,108,4,1),(###-12-01&&

100    Q: Are equilibrium and evolution mutually exclusive

100    A: <b>YES</b>.',0,109,4,1),(###-12-01&&

101    Q: The problem sets always seem to refer to a carrier as a phenotypically normal (healthy) individual (as of hemophilia).Isn\'t the person who actually has the disease a carrier as well

101    A: Carrier in this context always means a silent carrier -- one who can pass on the allele, but has no defective phenotype. It doesn\'t just mean \"someone who carries the allele\" = someone who many or many not have the condition. So a male can\'t be a carrier (in this sense) of hemophilia, but a woman can. There are female hemophiliacs -- they are homozygous.',0,110,4,1),(###-12-01&&

102    Q: If you have a male cell after DNA replication, should it have 2 X\'s and 2 Y\'s or just one X and one Y? Also how do you represent each Y (is it a straight line AND a half arrow)

102    A: One doubled X and one doubled Y. Doubled = 2 chromatids per chromosome. Don\'t think of an X as necessarily X shaped. Call it F for female or something else, and write the unduplicated one as a rod = 1 ds DNA molecule. The Y is just a smaller rod. Not Y shaped.',0,111,4,1),(###-12-01&&

103    Q: Regarding problem 8-9. Since just before meiosis, DNA is replicated, shouldn\'t we have an egg with 2 copies of X for 4 total chromosomes

103    A: Not 4 chromosomes. 4 chromatids, 2 chromosomes. In a cell about to produce eggs by meiosis. The eggs themselves are haploid and have one X, with one chromatid. (The term is \"X\" but it isn\'t X shaped.)',0,112,3,1),(###-12-01&&

104    Q: Regarding problem 8-9: When you have nondisjunction at the second division of meiosis, does it occur to each of the 2 cells that are formed after the first division, or only to one of the two

104    A: Only one. The two are not connected. When your twin slips, do you fall down too?',0,113,4,1),(###-12-01&&

105    Q: The Lecture 19 notes say  that the sex of a cell can be determined without doing a karyotype by the presence of a Barr body. The presence of the Barr body, however, doesn\'t distinguish between XX and XXY, which (in the case of humans at least) code for female and male, respectively. Doesn\'t this suggest that given the possibility of aneuploidy a karyotype really is necessary to make the dictinction

105    A: Aneuploidies are rare, so usually the presence or absence of a Barr body does the trick. However you are correct that you get more info from a karyotype. Even a karyotype doesn\'t always establish sex, as mutations in critical genes can produce a female even if a Y is present, by inactivating SRY, etc. This is a pretty typical situation where what we say in the general case always has exceptions. But we are trying to focus on what is common and leave the exceptions for later. Once you start considering exceptions, sex (and many other characteristics) start to get complicated. (This has practical applications in sports -- how do you decide who is really a male trying to win at a female sport??)',0,115,4,1),(###-12-01&&

106    Q: In problem 8-6, I know it is easier to draw out the cycle, but how do you figure out how many cells to draw? (i.e. how long the cycle is?)&&

106    A: You can draw out any number of cells -- they just represent different points in the cycle, which is essentially infinitely divisible. If you draw too few it is hard to see the process is continuous. If you draw too many, you are just wasting your time. (Unless you enjoy drawing that kind of thing.)',0,116,4,1),(###-12-01&&

107    Q: What exactly is a test cross? Is it specifically referring to the case of determining recombination frequencies, where one would cross a heterozygous F1 with a homozygous recessive, or can it also refer to a test to determine which allele is dominant and which is recessive

107    A: Both are test crosses if one parent is homozygous recessive for all genes involved. I think the term was originally used for your second case and then became generalized.',0,117,4,1),(###-12-01&&

108    Q: Is it possible for dominance to occur at the cellular level for a sex-linked trait?\r\n&&

108    A: No. Only for autosomal traits.',0,119,4,1),(###-12-01&&

109    Q: According to the Lyon Hypothesis, every female is a mosaic since some of her cells utilize the maternal X chromosome while other cells utilize the paternal X chromosome. How is it that with respect to some traits, like colorblindness, the mosiac is very finely grained with approximately every other cell using either the maternal or paternal X chromosome, but in other cases, like the case of black and orange tortoiseshell cats, large numbers of cells in proximity to one another seem to coordinate which X is utilized

109    A: It all depends on how the protein encoded by the gene is expressed -- whether it used locally or secreted, and how the cells that use the protein develop -- whether large areas of cells derived from a single cell stay together, as in skin, or whether cells of different origin get mixed together into a fine grain mosaic, as in the eye. In other words, there are additional factors to consider in each case.',0,120,4,1),(###-12-01&&

110    Q: In problems 9-1G and 9-5A, I thought there were 2 traits, but the answer for both is 1. However, the glossary of <i>Life</i>, the  says trait is one form of a character; so eye color is a character, and brown eyes and blue eyes are traits. Is \"trait\" the same thing as \"phenotype?\"&&

110    A: We are using \"trait\" the way they use \"character.\" Perhaps it would be better to be sure I agree with the text, but I think there is no agreement on this particular use of the terminology. (For example, what could be meant by \"sickle cell trait?\" It is used to mean an asymptomatic carrier for sickle cell, which doesn\'t fit either definition.) I hope that it is always clear from the context what we mean by \"trait.\"',0,121,4,1),(###-02-09&&

111    Q: How does RME count as moving substances across their gradient? It seems like the environment inside a vesicle would be the same as the environment outside the cell.&&

111    A: The receptors trap specific substances out of the extracellular solution. So the vesicle doesn\'t contain a sample of everything -- it\'s enriched for the substances that bind to receptors.',NULL,1484,1,2),(###-12-01&&

112     Q: About diseases caused by dominant genes, like Huntington\'s. How is it that the disease gets passed on, particularly if it\'s a serious one, if anyone with the allele inevitably gets the disease? Do people with diseases like Huntington\'s have children even though they know that, at best, (that is, assuming that they have one diseased chromosome and one normal one) their children have a 50-50 chance of avoiding it

112     A: Good Question. Usually people don\'t get the symptoms of HD until they are past reproductive age. Until recently, they usually didn\'t realize the risks until it was too late, so people with the dom. allele reproduced like the rest of us and the allele was passed on. (Dominant diseases that are serious enough and have a early onset are not passed on.) Now that people are aware, and there is a test for the allele, the situation is somewhat different. Individuals who are risk for the disease (had a parent or grandparent with it) have the option of being tested before they get symptoms. If they find out they are carriers, they can decide what to do -- different people have very different outlooks. (Some do not have kids, some play the odds, some do prenatal screening, etc.) Deciding to get tested is a complex decision, as the results can have far reaching implications. Do you want to know for certain you are going to get a fatal disease in the future? Or is it better to live with a 50-50 chance and hope for the best? What if you want to know, but your parent (who hasn\'t yet got symptoms) doesn\'t? And so on. So not all people at risk get tested. Everyone wants to know they DON\'T carry the disease allele, but not everyone wants to know if they DO.',0,123,4,1),(###-12-01&&

113     Q: How does crossing over with plasmids take place

113     A: The plasmid is still in a circle when crossing over takes place.',0,124,4,1),(###-12-01&&

114     Q: When a plasmid or fragment is integrated into a chromosome to make a bigger chromosome (i.e in conjugation or lysongenic cycle), why is the process called crossing over? How does the fragment integrate into the chromosome

114     A: For two circles, there is one actual cross over event, a cutting and splicing, between the two circles. The way I did it in class with the rubber tubing. The result is to join the two circles into one big circle. For a fragment and a circle, there are two cut and splice events. The result is to splice the fragment into the circle in place of a section of circle.',0,125,4,1),(###-10-04&&

115     Q: In question 2-7 B ii) in the problem sets, why does each subunit have 1/4 size

115     A: One experiment shows there are 2 kinds of subunits that differ in migration on a native gel. The difference could be in shape, mass or charge. The second says that both types of subunits have the same mol. wt. (So they must be different in charge or shape to migrate differently as in (1)). The third experiment says there are 4 free ends or 4 subunits (chains) per molecule. Since all types of subunits have the same mol. wt, that must be = 1/4 of the total.',0,126,1,1),(###-10-04&&

116     Q: Terminology question: what is a molecule (a protein molecule)

116     A: A molecule in biochemistry is what stays together in the cell and can be isolated such that all the subunits stay together when subjected to mild separation techniques. That is, if the atoms all stay together in the same superimposable way from one structure to another, then that structure is a molecule even though it is held together by non-covalent, weak bonds.',0,127,1,1),(###-10-04&&

117    Q: If a substance disrupts the primary structure of a polypeptide, does it also disrupt the secondary, tertiary, and quaternary structures of the polypeptide

117    A: In general, \"disruption\" of the primary structure really represent the destruction of the protein, which is a polymer. If the protein is hydrolyzed back to its constituent amino acids, then there is no more polymer, never mind one that could fold. That said, we could consider less severe disruptions of primary structure than cleavage of all the peptide bonds. Cleavage of many peptide bonds would also destabilize the structure, with small peptides not providing the cooperative effects necessary to keep the protein folded in the face of thermal agitation. However, if we go to the extreme low end of disruption of primary structure, the cleavage of just a single peptide bond, then there may be enough stability and cooperation among all the weak binds to allow a polypeptide to stay together in a proper tertiary structure (which now might be better considered a quaternary structure, since the cleavage would result in two polypeptide chains). This was the case for the problem in set 2 for ribonuclease. One would think a protein could better tolerate such a minimal cleavage event if there were disulfide bonds helping to stabilize the tertiary structure.',0,128,1,1),(###-10-04&&

118    Q: Wouldn\'t any two fingerprints be identical since both probably contain all twenty amino acids (albeit in different amounts)

118    A: You have the wrong idea about what a fingerprint is. It NEVER involves the separation of the 20 individual amino acids. Rather it is the separation of fragments of a polypeptide that have been produced by a limited cleavage of the polypeptide. If the proteolytic enzyme trypsin is used, fragments are produced by cleavage after lysine and arginine residues. The fragments are peptide of various sizes (the length being determined by the distribution of arg and lys residues in the polypeptide sequence). Every protein will have a distinctive combination of fragment sizes and compositions.',0,129,1,1),(###-10-02&&

119    Q: If endergonic means that a reaction is non- spontaneous, how can they be catalyzed by enzymes, which only \"increase the rate of spontaneous reactions.\"&&

119    A: The enzyme will catalyze the reaction in either direction. But the reaction will not proceed in the non-spontaneous direction because of the energy considerations. Thus as a practical matter, the enzyme speeds up the reaction in the direction dictated by the energy flow, in the direction where Delta G is negative. Note that the Delta G can be negative for a reaction with an unfavorable (positive) Delta Go, if, for instance, the products are kept low by rapid removal by a subsequent reaction. Is the reaction still endergonic? Not under those conditions, I would say. The terms exergonic and endergonic become confusing and not so helpful if pushed too hard when considering different situations. It is clearer to talk about Delta G and Delta Go.',0,130,1,1),(###-10-02&&

120    Q: What is this special regulatory site remote from the catalytic site to which the allosteric inhibitor binds

120    A: It is the specialized inhibitor-binding site; upon binding the inhibitor, the enzyme changes its shape slightly. The shape change includes the remote region encompassing the active site of the enzyme, and is sufficient to prevent the catalysis from taking place at the active site.',0,131,1,1),(###-10-02&&

121    Q: What are the regulatory and the catalytic subunits of the hetero-multimers referred to in allosteric inhibition

121     A: The protein has two different kinds of subunits: one polypeptide is in charge of carrying out the catalytic reaction, doing the catalysis. The other is specialized to bind the allosteric inhibitor (e.g., the end-product of a biosynthetic pathway) and in doing so inhibits the catalytic polypeptide from assisting the reaction.',0,132,1,1),(###-10-02&&

122     Q: What is the half-hydrogen, half-ionic bond formed between asp and asn

122     A: It\'s really more like a partial ionic bond. Polar compounds have a partial separation of charge, so a partially charged positive end of a polar group will be attracted by a completed charged negative ion, and vice versa. In the case you mention, the negatively charged asp carboxylate ion will attract the H on the amide nitrogen and the C in the C=O of the amide.',0,133,1,1),(###-10-02&&

123     Q: What is the difference between glutamine and glutamic acid/glutamate

123     A: Glutamine has an amide group on its side chain, which is polar but is not an acid or a base, so does not becomes ionized or charged. Glutamic acid has a carboxylic acid group on its side chain, which is an acid, can lose its proton and therefore take on a full negative charge.',0,134,1,1),(###-10-02&&

124     Q: At pH 1, glu and asp won\'t be charged. Why aren\'t these amino acids charged at this pH

124     A: At pH 1 glu and asp will be charged, they have a net charge of +1. The two carboxylic acid groups on these amino acid will not be charged. Carboxylic acids are not strong enough acids to remain charged (without a proton) in the face of such a high concentration of hydrogen ions (pH1 = $10^{-1}$ M). The acid is in equilibrium between its charged (deprotonated) and uncharged (protonated ) forms: RCOO- + H+<--> RCOOH. At pH 1 the protonated form on the right predominates. The amino group of glu and asp will be charged, however. Even at pH7, the amino group is a strong enough base to gain a hydrogen ion from solution (where the free H+ ion concentration is $10^{-7}$ M at pH7) and take on a +1 charge for almost allof the molecules.  That is, in the equilibrium R-NH2 + H+ <--> RNH3+, the form on the right predominates.  At pH1, where the free H+ concentration is a million times higher than at pH7 (a whopping $10^{-1}$ M), the equilibrium lies even more completely to the right.  So asp and glu have two uncharged carboxyl groups and one charged amino group at pH1.  Please note that for these reasons, at pH7, both the carboxyl and the amino groups of asp and glu are charged.',0,135,1,1),(###-10-02&&

125     Q: In our notes, in non-competitive inhibition, the substrate is still able to bind to the enzyme, but then the enzyme can\'t do anything with it. However, in Purves, it says that the presence of the non-competitive inhibitor in its own site on the enzyme distorts the shape of the enzyme and therefore makes it difficult for the substrate to bind to the enzme. Which is true

125     A: Yes, both. Purves does not offer the explanation I prefer, and vice-versa. I like to reserve the distortion case for allosteric inhibition, which is yet another situation (more complex).',0,136,1,1),(###-10-02&&

126     Q: Why does Km stay constant in non-competitive inihibition

126     A: The non-competitive inhibitor does not bind to the substrate-binding site. Those enzyme molecules that are still active are responding to substrate normally, according to the intrinsic Km of the enzyme.',0,137,1,1),(###-10-02&&

127     Q: Why does Vmax remain constant in competitive inhibition

127     A: Because you can always keep increasing the substrate concentration until the amount of inhibitor can\'t effectively compete, it\'s as if there were no inhibitor present. So the V reaches V max at this point, as the enzyme is saturated with substrate. It\'s just that you have to add a lot more substrate to get to this point if the inhibitor is present.',0,138,1,1),(###-09-30&&

128     Q: Should we have memorized structures to predict the charges according to the number of amino/carboxyl groups

128     A: You do have to memorize the properties of the organic chemical functional groups. These include one acid and one base. Carboxyls (R-COOH) are organic acids that will ionize at pH 7 to result in a negatively charged R-COO- group. Amines (R-NH2) are bases that will take up an H+ ion at pH7 to result in a positively charged R-NH3+ group. You can influence the charge by changing the pH: at a low pH the excess of H+ ions will protonate the carboxyl, so it is no longer charged. At hig pH, the low concentration of H+ ions allows the extra H+ ion on the amine group to be lost, so it loses its positive charge.',0,139,1,1),(###-09-30&&

129     Q: How do hydrophobic forces and entropy work in the formation of a \"water cage\" around nonpolar molecules?\r\n&&

129     A: It seems that the water molecules around the hydrophobic molecules get locked into a more specific arrangement with their neighboring water molecules, forming this cage (or \"clathrate\") or even ice-like structure in this microenvironment. It\'s not really ice since it does not last that long. But it\'s long enough to represent a considerably more ordered structure than the case in which the water molecules form more random partnerships, in the absence of the hydrophobic intruder.  Try these sites for more explanations: <a href = \"http://www.aw.com/mathews/ch02/clathrat.htm>http://www.aw.com/mathews/ch02/clathrat.htm </a>, <a href = \"http://wine1.sb.fsu.edu/bch5887/lecture_6/lecture_6.html\">http://wine1.sb.fsu.edu/bch5887/lecture_6/lecture_6.html</a>, <a href = \"http://www.harcourtcollege.com/chem/biochem/GarrettGrisham/HardToGrasp/Entropy/Entropy.html\">http://www.harcourtcollege.com/chem/biochem/GarrettGrisham/HardToGrasp/Entropy/Entropy.html</a>',0,140,1,1),(###-09-30&&

130     Q: Is this true that only the last monomer in a polysaccharide can be different in terms of alpha or beta conformation

130     A: Yes, the one with the free anomeric carbon, not yet connected in a glycosidic bond.',0,141,1,1),(###-09-27&&

131     Q: The Purves book says that a beta pleated sheet is formed from two or more polypetide chains. But don\'t beta pleated sheets pertain only to secondary protein structure

131     A: Further in the paragraph Purves points out that the participants in a beta sheet structure can be on the same polypeptide folded back on itself (the usual case, and the one depicted by Purves in Fig. 3.5d) or, (more exceptionally) between two separate polypeptide chains, thus contributing to quaternary structure. I see no contradiction here, do not get too concerned about pigeonholing terms. Secondary structure certainly contributes to tertiary structure (the overall 3-D folding) so why not

quaternary structure. There is a hierarchy here, where primary leads to secondary and tertiary, secondary to tertiary, secondary and tertiary to quaternary. My definition of secondary was just \"regular, repeated interactions between (solely) backbone atoms.\"',0,142,1,1),(###-09-27&&

132     Q: In problem 2-12, how is it possible that compound C only affected the quaternary structure and not the other structures

132     A: Depending on the action of Compound C, it could differentially affect the weak bonds holding one part of the protein together but not another. One could even imagine that one domain of a single folded polypeptide change becoming denatured under a particular condition, while the remaining domains stay intact. Furthermore, one can imagine treating a protein with increasing concentrations of one agent, say, urea. At some point the protein will start to denature, but not all at once, the parts that are most sensitive (say, relying most on H-bonds compared to hydrophobic forces) unfolding even as the rest iof the protein stays intact. So we can readily imagine a protein with a fragile quaternary structure and a robust tertiary structure. No disulfides need enter into the discussion.',0,143,1,1),(###-09-26&&

133     Q: Isn\'t it true that in order for a peptide to be polar, it must have both a positive and negative end

133     A: A peptide can be characterized as being hydrophilic or hydrophobic or something in between. Polar is probably not the best word to describe a peptide. A peptide will be more hydrophilic if it contains a lot of polar GROUPS on it, such as the side chain of asp, ser, or glutamine, for example (charged or partial separation of charge). The more non-polar groups (leu, val, phe) it contains, the more hydrophobic it will be. Depending on its composition, the peptide will represent a composite of the type of side groups it contains. Thus there is a lot of latitude, and so you can usually separate all the peptides (fragments) derived from a polypeptide using paper electrophoresis and paper chromatography, as in protein fingerprinting. The ends are usually not especially important in determining the overall hydrophobic or hydrophilic nature of a particular peptide.',0,144,1,1),(###-09-26&&

134     Q: How can we predict the charge of an amino acid in acidic, basic, and neutral solutions

134     A: Carboxylic acids are weak acids so they cannot lose a proton at low pH (1-2). Amines are weak bases and cannot take on a proton at high pH (10-12). At other pH\'s (neutral for both, high pH for acids, low pH for bases) you can figure they will be charged.',0,145,1,1),(###-09-26&&

135     Q: How we can determine if an amino acid is polar or nonpolar

135     A: Polar side groups can be recognized if they exhibit partial charge separation or become fully charged. Partial charge separation occurs when two atoms involved in a bond do not share their electrons equally, because one is better at attracting electrons (more electronegative). Pairs that exhibit this property are C-O, O-H, C-N, and N-H, where the O and N take on a partial negative charge and the H and C a partial positive charge. Full charge separation takes place when ions are formed. The only organic groups that we will discuss that can take on a full charge are the carboxyl groups (carboxylic acids, lose a proton and become negatively charged) and amines (accept a proton and become positively charged.',0,146,1,1),(###-09-23&&

136     Q: Why is the side chain of histidine charged rather than polar

136     A: Since it doesn\'t have any recognizable functional group, how do I determine its properties w/o having to memorize them? My initial thought before I looked at histidine in the text was that its side chain was polar because of the N-H bond but apparently that is not the case. Histidine is a weak base; at pH 7 about 1/3 of the molecules will be charged at any moment. The N\'s are related to amines: one is a secondary amine - primary amines have 2 H\'s while secondary amines have only one. I did not want to get into this finer organic chemical distinction in this introductory course, so I have chosen to sort of ignore the charged status of side groups like histidine and tryptophan. You are not expected to be able to figure out whether histidine is charged or not, but you should be able to see its polar character.',0,147,1,1),(###-09-23&&

137     Q: What is the significance of the sulfhydryl group, specifically in reference to cysteine

137     A: The sulfhydryl of cysteine plays a unique role in protein 3-dimensional structure. In the presence of oxygen (usually present), the -SH group is easily oxidized if it is in close proximity to another -SH. The two SH groups lose two hydrogen atoms; An oxygen atom accepts the two H\'s to form water. The two cysteine side groups become bonded to each other in a disulfide bind. The disulfide bond is a strong covalent bond. The disulfide bond stands out as the only strong bond that helps to maintain the 3-D structure of a protein, all the others being weak bonds (H-bonds, etc.). Two cysteines linked together by a disulfide bond constitute a new amino acid called cystine. Cystine has 2 less electrons than the 2 cysteines that went into its formation. Thus there was a net loss of 2 electrons; the molecules that lost the electrons are said to have been oxidized. Oxygen gained the two electrons; molecules that gain electrons in a chemical reaction are said to be reduced. When an oxidation reaction occurs, there is always a combination of oxidation and reduction, so the reaction is better called an oxidation-reduction. In the case of 2 cysteines being converted to a cysteine, the loss of electrons is accompanied by a loss of two protons (2 hydrogen atoms, each with one proton and one electron are transferred from cysteine to oxygen). Biological oxidation-reduction reactions often involve the loss of a proton along with the electron (a dehydrogenation). However, this combination is not always involved. It is the loss of the electrons, with or without a proton, that defines a reaction as an oxidation.',0,148,1,1),(###-09-23&&

138     Q: What is \"fingerprinting,\" and how does it differ from sequencing a polypeptide

138     A: Fingerprinting is the process of cleaving a protein with an enzyme that hydrolyzes a polypeptide at limited defined positions within the polypeptide (e.g., the enzyme trypsin cleaves the polypeptide after lysine and arginine residues) followed by 2-dimensional separation of the fragments to produce a series of spots that are characteristic of the protein. Each spot represents a peptide in the range of 5 to 20 amino acids in length. Virtually every different protein will result in a different pattern of spots; the the pattern is called a \"protein fingerprint.\" It does not involve carboxypeptidase, which is used in the sequencing of a polypeptide. The first method discussed in class was the use of carboxypeptidase to produce free amino acids from the C-terminus of a polypeptide. If the free amino acids are isolated sequentially as they are released, then the sequence of the peptide can be determined, if one can identify the amino acids. The methods used to identify the amino acids were paper electrophoresis and paper chromatography. The behavior (migration in paper electrophoresis and/or paper chromatography) of an unknown amino acid can be compared to the behavior of the 20 known amino acids to determine which amino acid corresponds to the unknown.',0,149,1,1),(###-09-23&&

139    Q: I am confused about the various protein purification and analysis methods. Are we separating individual amino acids, rather than proteins

139    A: The lecture covered two sets of methodologies within our discussion of proteins.  First, we discussed the monomers of proteins, the amino acids. Our discussion of PE (Protein Electrophoresis) and PC (Protein Chromatography)  showed how these free amino acids can be separated, one from the other. In thinking about these two methods, we became more aware of the properties of net charge and hydrophobicity/hydrophilicity that are dictated by the different amino acid side groups. Fingerprinting showed how molecules larger than individual amino acids could also be characterized by these two properties: peptides of modest size, say 5 to 20 amino acids in length, exhibited properties that were just the composite of their amino acid constituents. Later I talked about protein purification, so  hat we could appreciate the  complexity of a full protein (100 to 100 amino acids in length, and with a function in the cell). These methods brought new characteristics to consider: molecular weight and shape, as well as the old net charge. Our primary purpose in discussing these methods is to have you think about these small and large molecules in chemical and  physical terms, often at the level of the functional groups that are present. The division of the methods between the small molecules and the macromolecules is a natural one, as the methods used are different and the goals are usually different: for small molecules, the goal is usually analysis; for the proteins the goal is often purification as well as analysis.',0,150,1,1),(###-09-19&&

140    Q: Why in lecture 3 do you say \"an alpha bond in sugar is where the C1 OH that is formed ends up on the same side of the ring as the -OH of C2, or beta is where the C1 OH that is formed end up on the opposite side of the ring from the -OH of C2.\" In Becker p193, we see betas with the -OHs on the same side. Elsewhere in Becker, it defines beta as having the OH pointed up, and alpha as pointed down.&&

140    A: The lecture sentence is referring to the Haworth projection of the ring structure, which presents information on the relative positions of the H and OH at each carbon. In this type of flat ring diagram, alpha glucose has OHs below the Hs at both C1 and C2 (see glucose ring formation handout right side or Purves Fig. 3.10); thus they are on \"the same side\" in this type of figure. And in beta glucose the are on opposite \"sides\" of this depicted flat ring. In true 3D space, the situation is rather different, since what was below the Haworth ring at C2 is equatorial (out) while what was below the Haworth ring at C1 is axial and down. So in alpha glucose, the OHs at C1 and C2 are on the same side (below) in the Haworth diagram and oriented differently from each other (down vs. out, respectively) in the puckered chair depiction (reality). Likewise, although the C1 and C2 OHs must be placed on opposite side of the flat ring in the Haworth picture, they are both equatorial in the chair conformation. Gazing at the figure in the upper left of the glucose ring formation handout may help.',0,151,1,1),(###-09-11&&

141    Q: Do hydrophobic forces exist in polar solvents other than water (ammonia, for example)

141    A: Hydrophobic forces should also work in other polar solvents, such as the liquid formamide (HCONH2) for example, in which  many types of hydrogen bonds between formamide molecules could form. Ammonia is not a good example, because it is a gas; liquid ammonia is usually a solution of gaseous ammonia in water.',0,152,1,1),(###-09-17&&

142    Q: In the answer to #19 in problem set 1, where did the fraction > 63/64 come from

142     A: The cells increased in number from 100 to 6400 during the course of the experiment. Thus 6300 new cells were formed during this period. These cells are made up of molecules (polysaccharides, proteins, phospholipids, smalls, etc.). Almost all of these molecules contain carbon. The only source of carbon for all these molecules in these 6300 new net cells is the glucose in the minimal medium. So 63/64ths of the carbon in ALL the carbon-containing molecules in the cells was derived from the glucose on the medium (where else?). The remaining 1/64th represents the molecules that were present in the original 100 cells that started the culture, that were used to start the experiment, that were present at time zero. By the end of the growth period these original molecules have been distributed to many daughter and granddaughter cells as the cells keep dividing. But they are still there in the culture as a whole, even if they are spread out among all the cells. So at the end we have 1/64 old and 63/64 new, and all the new ones have been formed from the medium components.',0,153,1,1),(###-09-15&&

143     Q: In the linear form of glucose, does it matter whether the hydrogens or the hydroxyls are on the right or the left of each of the carbon atoms 2-6

143     A: Yes, it matters greatly. If there is a different arrangement of the hydroxyls around carbons 2-5 (left or right), then the hydroxyls will be differently oriented in 3-D space in the ring form; and indeed, the sugar will even have a different name. Switch one hydroxyl from left to right (or right to left) and you get mannose, or galactose, etc.). These different sugars are stereoisomers of each other, as in L and D amino acids. Because hexoses have 4 asymmetric carbons, there can be $2^4 = 16$ different arrangements, 16 different stereoisomers, each with its own name and its own distinctive 3-dimensional configuration. See text and problems.',0,154,1,1),(###-09-13&&

144     Q: Why is the side chain of histidine charged rather than polar

144     A: The nitrogens in the histidine ring are actually a form of amine (a secondary amine, with the N attached to two carbons instead of one). They act as a weak base, the two N\'s together being about 20% protonated (and thus positively charged) at physiological pH of 7.3. Because of this partially charged state, and not wanting to raise the issue of acid and base strengths in this introductory course, we prefer not to discuss histidine in the context of the charge on the side group.',0,155,1,1),(###-10-25&&

145     Q: In question 7-5B, if you have half the number of forks, won\'t the DNA replicate at a linear rate AND at half the speed

145     A: You will get linear replication. However for that part of the problem, I meant you to assume that DNA synthesis is normally unidirectional (one fork) and it takes 20 min to replicate using that one fork. If you took the problem to mean it takes 20 min. with 2 forks, then it will indeed take 40 min with one fork.',0,156,2,1),(###-10-25&&

146     Q: What properties of DNA make it less easily degraded and more easily repaired

146     A: The lack of a hydroxyl group at the 2\' positionand the presence of T instead of U. (Damage to C converts it to U. When a GC pair is damaged and generates a GU pair in DNA, the repair system can tell it\'s the U, not the G that is the error (since U is not supposed to be in DNA), so the repair enzymes can remove the U and replace it with C.)',0,157,2,1),(###-10-25&&

147     Q: Regarding the energy requirements for DNA synthesis in lecture 11, how can one say that reactions 1 and 2 are coupled when they are really just simply the net reaction of reactions 3-5

147     A: Reactions 3-5 have the same net effect as 1-2, but reactions 3-5 are linked and 1 and 2 are not. (There is nothing about 2 that indicates the energy from 2 can be used to drive 1 to the right.) It can take more than one enzyme to couple 2 or more reactions. The basic idea is that one reaction generates a product and that product is used by the next reaction. So overall, the entire process takes place together. It can take more than one step and more than one enzyme.',0,158,2,1),(###-10-25&&

148     Q: In given a replication fork, how can one determine the locations of the lagging and leading strands

148     A: Leading = synthesized continuously, 5\' to 3\' = goes in same direction as fork. Only one of the two parent strands can be replicated continuously if synthesis goes in the direction of the fork and new strand and template are antiparallel.',0,159,2,1),(###-10-25&&

149     Q: In  problem 5-12, why does decreasing the pH of the buffer help the mitochondria to produce ATP more efficiently

149     A: In this mutant, electron transport is too slow, so not enough protons are being pumped out of the matrix, and there is too small a proton gradient. So not enough protons flow back in to power the F1/F0 ATP synthase at max. speed. If you decrease the pH, you increase the protons available to flow through the synthase and power ox. phos.',0,160,2,1),(###-10-25&&

150     Q: Why don\'t we need pyrophosphatase to run PCR

150     A: Because in PCR you use relatively high concentrations of dXTPs. DNA polymerization has a standard free energy of about zero. You can \"pull\" the reaction by adding pyrophosphatase to remove the PPi product, or you can \"push\" the reaction by adding high concentrations of dXTPs = reactants. Cells do the first; in test tubes biochemists do the second.',0,161,2,1),(###-10-25&&

151     Q: Purves says DNA polymerase chews up the primer of the first Okazaki fragment, leaving a gap at its 5\' end. He goes on to say that it is ligase that fills that gap. How can this be

151     A: This is not right. DNA polymerase can chew a primer from the primer\'s 5\' end. But the DNA polymerase needs a 3\' end to extend the neighboring sequence to fill up the gap left by removing the primer. The DNA pol. chews primer from ITS 5\' end and replaces the gap from the 3\' end of the NEXT Okazaki fragment. Most of the texts are not clear on this point. (See the corrections page.) Purves is incorrect.',0,162,2,1),(###-10-25&&

152     Q: In problem 4-14B, it says that bacteria fed either glucose, 1,3 PGA or PGAld without O2 all provide the same energy (ATP) per mole.Doesn\'t glucose wind up as 2 moles of PGAld and therefore yield 2 moles ATP, providing more energy than 1 mole of PGAld

152     A: Glucose yields 2 net mole of ATP per mole of glucose, but actually produces 4 ATPs per mole. Since you had to invest 2 moles of ATP to get started (to get to F-1,6-diP), you only net 2 moles of ATP. P-d=gal or 1,3 PGA already have the phosphate on, and require no investment. Two moles of ATP will be produced per mole of pGal or diPGA tha travels down the glycolytic pathway (see pathway) minus zero investment yields 2 moles ATP per mole.  Glucose produces less energy per mole (and per gram)

that PGAld. Look at it this way: you've already invested some enrgy in producing PGAld. Somebody had to out that phosphate on, and now it's ready to roll, with no investment necessary. Glucose, ingetting to that point (PGald) only spent ATP, didn't produce it. So PGald is more energy rich.',0,163,2,1),(###-10-25&&

153     Q: In problem 4-8C, why is the reactant the only substrate if all enzymes catalyze both the forward and reverse reactions and can use both reactants and products as substrate

153     A: An enzyme is always capable of catalyzing the reaction in both direction. However, under a particular set of conditions, such as the ones in the problem, the enzyme may spend most of its time operating in one direction. If only substrate (and not product) is around, the enzyme will catalyze more S to P than the reverse.',0,164,2,1),(###-10-25&&

154     Q: In problems 4-5C and 4-10D, is it correct to think that, without enzyme, a given reaction will proceed very slowly but will still occur

154     A: The reaction rate without the enzyme, at intracellular pH, temp, etc. is so slow it is effectively zero (compared to the rate at which cells have to do things). This is what you are supposed to realize.',0,165,1,1),(###-10-24&&

155     Q: In exam 2 of 2000, question 1C, it says that succinate can go on to make more glutamate, but then says you cannot use pyruvate because the inhibitor is blocking the cycle. How can the inhibitor only block the cycle  for pyruvate but not for anything else

155     A: The inhibitor blocks the cycle of reactions between aKG and succinate. So there is no real \"cycle\" any more no matter what is added. Pyruvate needs OA  to get into the reactions of the KC. A very little could go to acetyl-CoA but this  would be only a little, limited by the stores of CoA, which would not be recycled since acetyl-CoA cannot condense with OA because there is no OA  because of the block. Any nutrient added that is upstream of the block will also not be able to yield any OA and so pyruvate will still be stuck. This is the  case with citrate, isocitrate, and aKG. However, succinate is another story: it  comes in downstream of the block and can get converted in a few steps to OA,  which now can condense with acetyl-CoA produced from pyruvate. So  succinate (or fumarate or malate or OA itself) will allow pyruvate from glucose  to get into the KC reactions as far as aKG (it cannot go further because of the  block).',0,166,2,1),(###-10-24&&

156     Q: On Handout 12, if the primer used has just one compliment, how is it that the new strands generated can bind the same primer \"upstream?\"&&

156     A: There are two different primers here, one complementary to each strand. One binds on one side of the target sequence and the other binds to the other strand, at the other side of the target sequence.',0,168,2,1),(###-10-23&&

157     Q: In problem 4-11C, since the enzyme for the conversion of pyruvate to lactic acid is present, can't NAD+ be regenerated and recycled, resulting in fermentation of 10 moles of glucose to yield a net of 20 moles ATP, not the .01 mol ATP as suggested

157     A: The question should say \"Suppose you try to ferment some glucose to ethanol...\" If that is omitted, then your solution is ok.',0,169,2,1),(###-10-23&&

158    Q: If the protons and electrons involved in electron transport originally come from NADH, where are the protons that are pumped out coming from

158    A: The protons from NADH are separated from the electrons in an early stage, even before CoQ. The protons that are pumped come from the pool of protons that are in equilibrium with water, due to the dissociation of water  (10^-7 M protons at pH 7). The energy for this pumping comes from the free energy changes associated with the electron transfers. (See also question 36.)',0,170,2,1),(###-10-23&&

159    Q: In respiration, we have 10 NADH2\'s and 2 FADH2\'s per glucose molecule that are giving up 24 electrons to 6 atoms of oxygen. Considering the stoichiometry of the overall reaction, why are only 6 water molecules produced instead of 12

159    A: In my over-simplification I presented only the NET reaction: glucose + 6 O2 -- 6 CO2 + 6H2O. But what is really going on is: glucose + 6 H2O + 6 O2 -- 6 CO2 + 12 H2O. H2O is added at several steps in glycolysis/KC; and those 12 pairs of electrons indeed end up on 12 newly minted molecules of water.',0,171,2,1),(###-10-23&&

160    Q: For the handout graph of energy levels in glycolysis, what does the y-axis represent

160    A: The y-axis represents changes in standard free energy for the reacions numbered in the glycolysis handout. The scale is set arbitrarily with glucose at zero. To determine the free energy change for a particular reaction, subtract the level of the product from the level of the reactant. Connecting lines going up represent a positive delta Go (unfavorable) whereas stepping down represents a favorable delta Go (release of free energy, a negative delta Go).',0,172,2,1),(###-10-23&&

161    Q: In Delta G calculations, are the concentrations of water and hydrogen ions also set equal to 1 as for the Delta Go calculations

161    A: A non-trivial question. For delta G, one should divide the concentration of products and reactants by the standard concentrations to obtain the \"activities\" of the reactants and products. It is these activities that are really meant to be used in the calculation of delta G. The starndard concentrations are 1 M for all but water and H+. For water the standard concentration is 55 M, and it is assumed that water is always at 55 M, so the H2O activity always comes out to 1, and is never a factor in the calculation. For H+ ions the concentration should be included and divided by 10^-7 M, which is the standard concentration of H+ (pH7) in biochemistry.  Such a pH convention means you are calculating delta G\'. This discussion goes beyond the level of this introductory course.',0,173,1,1),(###-10-23&&

162    Q: In problem 6-9C, shouldn\'t the answer be (G+C) and (pyrimidines), since G and C are pyrimidines, as opposed to A and T, which are purines

162    A: A and G are purines; C, T and U are pyrimidines.  G + C are NOT both pyrimidines -- they  are mentioned together because they form base pairs by hydrogen bonding.',0,174,2,1),(###-10-22&&

163    Q: Is the enzyme that removes the primer the same one (DNA polymerase) adds the nucleotides to fill in the gap

163     A: It isn\'t the same enzyme, but both enzymes are DNA polymerases and we are not making a distinction between them. (See Becker for the fine points if you are curious.)',0,176,2,1),(###-10-22&&

164     Q: In problem 6-11, why is ddNTP incorporated into the DNA at all, since the polymerase ordinarily binds the 3\' hydroxyl (which is missing in this case)

164     A: Note the problem says that the DNA polymerase cannot distinguish ddCTP and dCTP (which is actually the case). From first principles, you can\'t be sure if the enzyme will recognize (and incorporate) ddCTP -- you don\'t know enough about the fine points of the substrate binding site of the enzyme. It appears from the experimental results that the group at the 3\' site of the XTP is not critical to enzymebinding (or at least, that either a OH or H will fit).',0,177,2,1),(###-10-21&&

165     Q: In problem 5-1B(iv), why use glucose and not fructose, since fructose is the natural sugar in grapes

165     A: Yes, fructose would work. So would any other monosaccharide or polysaccharide (if the yeast have the enzymes to break them down and/or convert them to glucose or F6P.) The point here is you can\'t feed them lactate. (Note also that most grapes contain about equal amounts of glucose and fructose.)',0,178,2,1),(###-10-21&&

166     Q: In problem 4-2, what is the relation of Keq to the concentrations of starting material

166     A: Keq is independent of starting concentrations. Delta G-zero and Keq are both constants, defined independently of starting concentrations. Delta G is different -- it depends on what concentrations you start with. One point of the question is to drive home that Keq doesn\'t change if you change starting concentrations.',0,179,1,1),(###-10-20&&

167     Q: During DNA replication, when is the RNA primer removed and the resulting gap filled in by the polymerase

167     A: There are no gaps between the Okazaki fragments until the RNA primer is removed. Both removal of primer and filling up of gap probably occur simultaneously by DNA polymerase. In my opinion, the pictures in the texts are confusing or wrong on this point.',0,180,2,1),(###-10-18&&

168     Q: In problem 5-1, how can yeast carry out aerobic metabolism with poorly developed mitochondria

168     A: In part A, it says the mitochondria are poorly developed. But that is not a permanent state -- the mitochondria can be fully developed under other conditions. The point of part B is that the mitochondria have to be fully functional if the Krebs cycle etc. is going to work. So when the yeast are grown aerobically on lactate, the mitochondria are NOT poorly developed any more.',0,181,2,1),(###-10-18&&

169     Q: How many unique types of primers are needed in PCR

169     A: Two (for double stranded replication). Many copies of each primer are added at the beginning of the reaction. One copy is used per template for each cycle. The process is explained as if it happened step by step to make it clearer, but it is actually carried out for many cycles in a single sealed vessel.',0,182,2,1),(###-10-18&&

170     Q: Why does lagging strand have to be synthesized as shorter fragments

170     A: The critical fact is that chains can only grow from their 5\' ends toward their 3\' ends. So DNA polymerase can only make new chains as it passes from the 3\' end toward the 5\' on the template. As the fork moves down the DNA (or the DNA moves through the fork) both sides of the double helix (both templates) must be replicated at once. The only way to do this is to have the DNA polymerase slide in opposite directions on the two template strands. This is very hard to explain in words. I suggest you ask me or a TA or look at the pictures in the texts.',0,183,2,1),(###-10-18&&

171     Q: In fermentation, do bacteria have a choice between producing lactic acid and ethanol and CO2

171     A: Any given type of bacterium usually has enzymes for only one or the other -- converting pyruvate to lactate or ethanol + CO2. Different types of bacteria produce different end products (including some other choices we haven\'t discussed.) It all depends on what enzymes the bacterium can produce, which depends on its genes.',0,184,2,1),(###-10-18&&

172     Q: On handout 9-3, what if the difference between views 2 and 3 of the ETC

172     A: View 2 shows some of the discrete steps of electrons being transferred, but leaves out a few for simplicity. View 3 reflects how the electron carriers are organized into complexes. The 3 that are shown are those involved in the route from NADH to O2. There is an additional complex (II) when FAD is the source of electrons. Each complex contains several carriers, and the electrons are passed to each carrier in succession within a complex. CoQ carries electrons from the left hand complex (I) to the middle complex (III). Cytochrome C carries electrons from the middle complex to the right hand complex (IV), which finally gives the electrons to oxygen. So View 2 shows separate individual transfer steps and View 3 shows the physical organization. See your texts for better pictures and additional explanation. See also the link in lecture 9 for a more detailed but clear explanation.',0,185,2,1),(###-10-18&&

173     Q: In the electron transport chain, why are the electrons just passed along, and where do the protons go

173     A: The electrons are passed because their transfer is energetically favorable, and the proteins of the respiratory complex act as enzymes, reacting with NADH to start with and oxidizing the first internal component of Complex I, followed by similar reactions of oxidation and reduction with internal components (Coenzyme Q, cytochromes,etc.) as the transport proceeds. You can think of each step as an enzymatically catalyzed reaction. The protons go into the aqueous environment once the electrons are transferred.',0,186,2,1),(###-10-18&&

174     Q: In the electron transport chain, when are whole hydrogen atoms passed along, and when just the electrons

174     A: As the electrons from NADH are passed to the proteins of Complex I, the protons are quickly stripped off, as the electrons are passed to several Fe+++ atoms in succession within this complex (iron-sulfur centers, not heme). The protons join the pool of other hydrogen ions in the matrix, but many of these are pumped out concomitant with the transfer of the elctron. For instance, as a pair of electrons from NADH traverse Copmplex I, it is estiamted that 4 protons are pumped out of the matrix to the outside of the inner mitochondrial membrane.',0,187,2,1),(###-10-18&&

175    Q: In problem 5-1, shouldn\'t anything in the glycolytic pathway work to produce ethanol, not just glucose

175    A: Most of the glycolytic intermediates can not be taken up by whole cells. Phosphorylated compounds are found inside cells, but not (normally) outside, and they are not taken up if they are added. You may notice that many of the problems involving metabolism of intermediates involve test tube situations, not growing cells. (Although some impossible situations are made up for educational purposes.) So other mono- or di-saccharides will work instead of glucose, but not most intermediates of glycolysis.',0,188,2,1),(###-10-18&&

176    Q: What is meant by the terms \"tautomer,\" \"keto-forms,\" and \"enol?\" What is their relevance to the 3D structure of DNA

176    A: A tautomer is a type of isomer -- tautomers differ in where one of the H atoms is placed. It the H is on an N, leaving a double bond O (keto group) sticking out of the ring, the tautomer/isomer is called a keto form. If the H is on an O, so a hydroxyl (or enol) sticks out of the ring, then the tautomer/isomer is called an enol form. The two forms (see the two forms of T on the handout) differ in their ability to form hydrogen bonds. A keto is delta negative and forms an H bond to a group that is delta plus; the H on an OH is delta positive and forms an H bond to a group that is delta minus. So it matters whether an \"O\" or an \"OH\" sticks out at position 6 of the pyrimidine ring (in U or T). You can\'t see why A bonds to T and G to C (and not the other around) until you draw the bases in the correct tautomeric form. Watson and Crick were misled in the beginning because the books that they consulted had the bases written in the wrong (enol) tautomeric forms.',0,189,2,1),(###-10-16&&

177    Q: Where did the phosphates in glycolysis come from? The handout makes no mention of additional ATP molecules being used.&&

177    A: The 2 phosphates that are added in the phosphorylation of Gal-3-P to 1,3 diPGA came from inorganic phosphate (a.k.a. Pi, or H3PO4, PO4---). This phosphate is present as an inorganic component of minimal medium (potassium phosphate), an inorganic salt that is necessary for life (in part for this reason).',0,190,1,1),(###-10-16&&

178    Q: What exactly is a vesicle

178    A: A pinched off piece of membrane that reseals itself so that it represents a spherical space enclosed by a phospholipid bilayer membrane. What is inside a vesicle depends on what was just beside the membrane when the pinching off occurred.',0,191,2,1),(###-10-09&&

179    Q: Are high energy bonds -7 kcal/mol or -5 kcal/mol

179    A: There appears to be no exact consensus on this point, a state of affairs unfortunately reflected in my notes. Some say -7 and some say -5 and most sources are non-committal. So it seems to be somewhat a matter of taste in the region between -5 and -7. All agree that -7 is in the high category, but towards the low end. All agree that -3.5 is not a high energy bond. So it\'s fuzzy. I like -5 because I\'d hate to think that ATP hydrolysis just squeaks into this category. The exact demarcation is not important. The important point is that reactions that RELEASE a larger amount of energy can be used to drive reactions that REQUIRE a lesser amount, if the reactions can be coupled.',0,192,1,1),(###-10-09&&

180    Q: What is meant by \"steady state\" concentrations? How does it relate to the equilibrium state, if at all

180    A: In both steady state and equilibrium, the concentrations of a set of reactants is not changing. In equilibrium, there is no net change because products are being transformed into reactants at the same rate that reactants are being changed back into products. In a steady state the constancy of a particular component is due to the fact that its rate of formation is exactly balanced by its rate of conversion to another product (not back to the reactants). Thus, whereas equilibrium is a net static situation, a steady state is just the opposite, characterized by a constant flow of molecules down a pathway.',0,193,1,1),(###-11-19&&

181    Q: Can viruses transduce whole plasmids

181    A: Probably not, but you can artificaly make virus heads that enclose whole plasmids.',0,194,3,1),(###-11-19&&

182    Q: In problem 13-8 C, if there is only one cutting site for HindIII, why is the answer 1300 or 1800, not 5000, and how far apart are the two restriction sites

182    A: You are cutting a plasmid that has an insert, not just the plain plasmid.  The insert also has a HindIII site. The size piece you get depends on which way the plasmid is inserted into the Bgl II site, which is 800 BP from the Hind III site in the plasmid.',0,195,3,1),(###-11-19&&

183    Q: When a plasmid with introns is integrated into another plasmid, do transcription and translation occur normally

183    A: Transcription, yes, but only if there is a prokaryotic promotor.  Translation -- usually does not work properly, as introns often contain stop codons and/or cause frameshift and virtually never produces a functional product. Replication is normal.',0,196,3,1),(###-11-19&&

184    Q: In lysogeny, how does the repressor protein get destroyed

184    A: Damage to the DNA (often by UV light in a lab) triggers activation of an enzyme which destroys many proteins, including the repressor.',0,197,3,1),(###-11-19&&

185    Q: Do viruses ever contain plasmids or anything other than signle-stranded DNA

185    A: Viruses can contain circular DNA\'s, but that doesn\'t make them plasmids. (A virus is like a plasmid in some respects, but has the genes to package itself for release as a whole particle that can travel to another cell. A plasmid is a naked DNA molecule that cannot exist outside a cell. It has no packaged, dormant form.) The bacterial chromosome is circular, but it still isn\'t a plasmid. The critical thing about a fragment is that it has no origin of replication. It is usually circular.',0,198,3,1),(###-11-19&&

186    Q: What is the best way to approach problem 11-6

186    A: In general, you have to examine each experimental set up (in each problem, or in real life) individually and see what is going on. You can\'t use a simple easy to memorize rule to be sure what you are dealing with -- for example, the word \"often\" or \"common\" doesn\'t always mean it is

complementation, although it usually points in that direction. \r\n<br><br>\r\nYou have  to put all the facts together and not rely on one or two key words, which may be misleading. In other words, you have to look beneath the surface to be sure of understanding and solving the problem.',0,199,3,1),(###-11-19&&

187     Q: In problem 11-6, for the transduction procedure (c), is there a general rule about whether viruses can recombine and/or complement

187     A: In this case, the virus with bacterial DNA is transferring a fragment of bacterial DNA. So the only long term way to restore function is recombination between the fragment and the chromosome of the recipient. In 11-8, there is an actual viral infection with 2 dif. viral DNA\'s present throughout infection in a single host -- so you can have complementation.',0,200,3,1),(###-11-19&&

188     Q: How do you predict whether recombination or complementation happens in a given cell? For example, in problem 11-6, why can\'t the conjugation (a) and transformation (b) experiments work by complementation

188     A: You have to look carefully at individual cases to use the high/low rate criterion. In many of these cases, there has been selection for functional recombinants and/or functional complemetations so that the ones you get are only a small fraction of the total. You look at so many (total) that you still get a significant number even if the process was inefficient.',0,201,3,1),(###-11-19&&

189     Q: In problem 11-7A, why can\'t single-stranded DNA just be happy that it\'s getting a copy of itself every time the host replicates a double strand

189     A: The ss DNA does need to stand alone -- it has to be copied many times and packed into viral heads. You need special enzymes to make many copies of just one strand without the other.',0,202,3,1),(###-11-19&&

190     Q: In wobble, does the actual base at the 5\' end change to accomodate the different codons in can bond to

190     A: Once the codon is chosen, a particular anticodon must be chosen to match it. There are more choices for the anticodon, if wobble is allowed. However, once you have a real tRNA, you can\'t change the anticodon in it. It\'s just there are several different tRNA\'s you could think of that would work, with different anticodons in them.',0,204,3,1),(###-11-19&&

191     Q: In question 11-6A, why is (a) an example of recombination, and does (c) involve recombination because a gene from one strain infects the second cell instead of the viral DNA

191     A: In (a) you are transfering a fragment of the chromosome, not a plasmid. (must be Hfr and F-, not F+). The genes for making pro are normal bacterial genes and should be on the chromosome. Yes, you are correct about part (c).',0,205,3,1),(###-11-19&&

192     Q: Is integration of an F factor considered to be a recombination event

192     A: Recombination means any crossing over event that leads to a new combination of DNA pieces. If you cut and rejoin two circles to get a bigger circle, that\'s recombination.',0,206,3,1),(###-11-19&&

193     Q: Why does complementation have high efficieny and recombination low efficiency

193     A: Crossing over events are rare. So just putting two DNA\'s into a cell doesn\'t mean they will cross over. Even if they have the potential to form a recombinant, working molecule doesn\'t mean it will happen.  No cross over event (or other rare event) is required for complementation to kick in.',0,207,3,1),(###-11-19&&

194     Q: In problem 12-8E, couldn\'t Mutant 2 also have a defective promoter, or would that produce exactly zero enzymes

194     A: If it had a defective promotor, then the normal mut 2/wt combination wouldn\'t be permanently repressed. The normal operon would still be inducible.',0,208,3,1),(###-11-19&&

195     Q: In problem 13R-3, does \"complete and normal operon\" always mean that it includes the repressor gene

195     A: It has to. Otherwise how could the operon operate?',0,209,3,1),(###-11-18&&

196     Q: What is the difference between a cistron and a gene

196     A: The term \"gene\" can be used in different contexts to mean slightly different things. The term \"cistron\" is defined by complementation tests. Gene and cistron usually mean the same thing, but in some cases, they don\'t. This is largely a matter of fine points and special cases.',0,211,3,1),(###-11-18&&

197     Q: How does a strong promotor increase the level of transcription

197     A: A promotor only binds one pol. at a time, but a gene can have many RNA polymerases reading it at once -- each at a different point along the gene. (Each polymerase attaches at the promotor, and then proceeds down the gene.) A stronger promotor has a higher chance of \"catching\" a molecule of RNA pol at any particular time. So transcription starts more often from a strong promotor.  The rate of growth of the RNA chains is the same, no matter what the strength of the promotor. The strength determines how many polymerases (on the average) are moving down the DNA at any one time.',0,212,3,1),(###-11-18&&

198     Q: Is an splicesome an enzyme (i.e. protein) or a ribozyme

198     A: Strictly speaking, the answer depends on which part of the spliceosome (the RNA or the protein) is the catalytic part. Probably it is best to define the spliceosome as a ribonucleoprotein particle, and leave it at that. ',0,213,3,1),(###-11-18&&

199     Q: Does recombination involve only DNA fragments and chromosomal DNA (and not plasmids)

199     A: Any two homologous DNA\'s can engage in recombination. Plasmids can be involved. Crossing over between a plasmid and the chromosome to make one big chromosome also occurs.',0,214,3,1),(###-11-18&&

200     Q: Does the terminator or poly-A tail determine the end of transcription

200     A: Transcription is ended differently in prokaryotes and eukaryotes. In prokaryotes, there is usually a termination signal in the DNA. In eukaryotes, there isn\'t. Instead there is a signal to add poly A, and where the poly A goes determines where the end of the transcript is.',0,215,3,1),(###-11-18&&

201     Q: Does spliced mRNA have still have its G-Cap and Poly-A tail when it is being translated by the ribosomes? Does it ever lose them

201     A: An actively translating mRNA has both the cap and the poly-A tail. More than we covered:  In fact, both are necessary for efficient initiation of translation. Recent results indicate that the polyA tail is gradually shortened and when it is less than  ~20 A\'s, the mRNA is decapped and then rapidly degraded from the decapped 5\' end.  This evidence is strongest for yeast so far.',0,216,3,1),(###-11-18&&

202     Q: Does the primary transcript have a poly-A tail?  If not, when is the poly-A tail added

202     A: The definition of a \"primary transcript\" is not exact or universally accepted when one asks about the details. Strictly speaking, the primary trasncript is the transcript produced by RNA polymerase before any modification. The usual modifications are capping, splicing, and polyadenylation.  The problem is that capping takes place soon after the intiation of transcrption, when the transcrript is only about 20 nucleotides long.  Additionally, splicing usually starts happening before the trasncript is finished, while it is still \"nascent\" (being transcribed by RNA polymerase). So the primary trasncript, strictly speaking, does not usually exist as a complete unmodified copy of the gene. Nevertheless the term is useful in distinguishing the modified from the unmodified versions at a location in the RNA molecule. Thus \"the primary transcript gets spliced\", or \"the primary transcript is cleaved and then immediately polyadenylated.\"',0,217,3,1),(###-11-18&&

203     Q: What is the exact definition of nucleic acid hybridization

203     A: Hybridization is the annealing of two single strands of nucleic acid together so that they form a double helix according to Watson-Crick base-pairing rules.  The duplex nucleic acid thus formed can be DNA-DNA, RNA-RNA, or DNA-RNA in terms of the single strands it contains.  A common way to achieve hybridization is to slowly cool a heated solution containing complementary single strands.',0,218,3,1),(###-11-18&&

204     Q: When an operon is turned \"on,\" are there are always high levels of enzymes produced? In what case are there intermediate levels of enzymes produced

204     A: If there is only a little of the inducer added to a bacterial culture, there may not be enough inducer to inactive all the repressor molecules. So some operators may be blocked, part of the time, and induction will not be maximal.',0,219,3,1),(###-11-18&&

205     Q: Are two tRNAs that carry the same amino acid but have different anticodons--as 3\'CAA5\' (val) and 3\'CAG5\' (also val)--seen as different tRNAs

205     A: Yes. These would be two different tRNA\'s. They are different molecules (with different nucleotide sequences) and would have to be transcribed from different genes. However I think you may be thinking about this from the wrong angle. There usually would not be two such tRNA\'s, as their abilities to read codons overlap. The one with the 3\' CAG 5\' anticodon can read both GUU and GUC, so there is no need for the other tRNA. I think you may be thinking of this whole issue -- how to

match tRNA\'s and codons -- backwards. Generally it is clearer if you start with the codons, and figure out how many different anticodons are needed to match up with them. Don\'t start from the anticodon end -- not all possible anticodons are needed. (See answer to next question.)',0,221,3,1),(###-11-18&&

206     Q: If for every two codons coding for the same amino acid there is one tRNA, what are the 2 anticodon sequences for valine (I see four possibilites)

206     A: For any \"box\" in the genetic code containing 4 codons for the same amino acid, such as the 4 val codons GUX, you need 2 tRNA\'s to read all 4 codons.  One tRNA could have the anticodon 3\' CAG and one 3\' CAU. (Alternatively, one could have the anticodon 3\' CAI and the other 3\'CAC.) Check the wobble rules to see why this should work.  It is not correct that there are always 2 tRNA\'s per amino acid -- that\'s on the average only.  The number of tRNA\'s required depends on the number and type of codons.  ',0,222,3,1),(###-11-18&&

207     Q: In the process of producing a certain protein whereby we indroduce cDNA into bacteria and use a probe to see which colonies represent cells that incorporate the cDNA, where does the probe come from and how do we design a probe given the existence of introns

207     A: In the example I presented, we make the probe based on the amino acid sequence of a \"window\" in the protein, as you stated in your question. The probe is only 20 nucleotides or so, and since it  made synthetically, on a machine, and not isolated from a natural source. it includes no intron sequences (by design). So the probe has nothing to do with introns, and likewise the target here has no introns, since it is cDNA, made from mature mRNA, from which the introns have been spliced. So we are not \"dealing with restriction fragments of human DNA,\" we are dealing with a fairly complete cDNA copy of the mRNA (made in the test tube with reverse trasncriptase).',0,223,3,1),(###-11-18&&

208     Q: Can two or more restriction fragments, given that they have the same \"sticky ends,\" come together and all get inserted into a bacterial chromosome

208     A: In DNA cloning, the restriction fragments are inserted into a plasmid, not into the bacterial chromosome. In theory, more than one restriction fragment can be cloned at the same time in the same plasmid host molecule. Sometimes this 3-way cloningis desriable, but it is more difficult to achieve, since the collisions of the moelcules are rare events.  Most plasmids do not incorporate any foreign DNA, some incorporate one restriction fragment, and many fewer incorporate 2.  You can decrease the probability of the double event by using a low concentration of restriction fragment. Detail not provided  in the lecture or expected from you: Also, in practice, it is necessary to discourage the reannealing of the plasmid, which would produce an unproductive \"empty\" vector.  This unwanted reaction is minimized by treating the linearized plasmid with the enzyme phosphatase, which cleaves off the 5\' phosphate left by the restriction enzyme action.  The denuded plasmid sticky ends cannot become ligated without a phosphate there.  In contrast, the foreign restriction fragments still have their phosphates, so at least one of the two strands at the newly-forming joint can be ligated, and this single ligation suffices; the joint is further repaired by E. coli enzymes after entrance into the E. coli cell.',0,224,3,1),(###-11-18&&

209     Q: When crossing over occurs, does the entire gene have to be exchanged or can just a segment of a gene

209     A: Just a segment or a whole gene. Either way.',0,225,3,1),(###-11-18&&

210    Q: Suppose two chromosomes both have a sequence C that codes for protein A. If both chromsomes have a mutation in sequence C, but in different places, is it possible for recombination to produce normal sequence C

210    A: Yes, recombination can exchange genes in whole or in part.  ',0,226,3,1),(###-11-18&&

211    Q: Can recombination be used to refer to both crossing over and the act of cutting up DNA and inserting a sequence between the two pieces

211    A: Yes. People usually call it genetic engineering if it\'s done in a test tube in a biochemistry lab and genetic recombination if it occurs inside cells.',0,227,3,1),(###-11-18&&

212    Q: In problem 13-3 part A, why are the codons for the mRNA, not for the DNA

212    A: The sense strand of the DNA and the mRNA are the same, except for switching U and T. The sequences given there are those of the sense strand, not the transcribed (template) strand.',0,228,3,1),(###-11-18&&

213    Q: In problem 11-5, if strain B is leu- and has 2 identical copies of the gene (in which case both must be leu-), how can a \"-\" be transferred? In part B, how can there be more than one leu gene

213    A: You can transfer any piece of DNA. The trick is to be sure you have done it -- you have to be able to tell those who got the piece of DNA in question from those who didn\'t. When it is a defective gene, it is sometime hard to tell who got it. There is no single \"the leu gene\". There are a lot of leu genes, one for each peptide needed to help make leucine, (so a leu- phenotype can come from many genotypes).',0,229,3,1),(###-11-18&&

214    Q: In Figure 13.18 of the Purves text, on page 253 (6th Edition), if the DNA strand shown is written in the typical convention, 5\' to 3\', and if the promoter is present on the left side, then shouldn\'t the strand shown be the sense strand and the mRNA transcript should be coded for by the complementary template strand

214    A: I think they are trying to finesse the whole issue by drawing a double stranded DNA. (Note the fine white line down the middle.) Operons and genes are usually portrayed so that the 5\' end of the sense strand is on the left. That is what they mean in the pictures.',0,230,3,1),(###-11-18&&

215    Q: What exactly constitutes a \"purified\" gene

215    A: It usually means a test tube full of many copies of the gene in question. Without a lot of other DNA.',0,231,3,1),(###-11-18&&

216    Q: If restriction enzymes of bacteria don\'t cut their own DNA because of the methyl group, why is it that viruses don\'t methylate their DNA to prevent cutting as well

216    A: Viruses do not have the genes to code for the enzymes to methylate their own DNA. They have to rely on bacterial enzymes. Bacterial enzymes only methylate newly replicated DNA that is already methylated on one (the old strand) -- this is what you get when methylated DNA is replicated semi conservatively. Since the viral DNA is completely unmethylated, when it replicates it still has 2 unmethylated strands and is not methylated by bacterial enzymes.',0,232,3,1),(###-11-18&&

217    Q: In bacterial conjugation why can\'t the recipient be F+ or Hfr

217    A: The F+ or Hfr uses it\'s special genes to make a bridge structure. It must contact a cell surface that is complementary, and only F- is. In other words, you must have two structures that fit together. F+ or Hfr makes one; F- makes the other.',0,233,3,1),(###-11-18&&

218    Q: In problem 7-16, why is the promotor on the sense strand (the strand shown), not the antisense strand

218    A: The promotor is not on one strand or the other. It consists of both strands, and it binds RNA polymerase so that the enzyme faces in one direction or the other. Once the direction of transcription is fixed, which strand can serve as template is fixed too. We write the P on one strand for convenience, but that is probably just confusing the issue.',0,234,3,1),(###-11-18&&

219    Q: In problem 12R-2 (A), why is the repressor protein affected by a frameshift mutation in 5\' UTR

219    A: The UTR\'s in bacteria are relatively short, which is how the mutation can still have an effect if it is near the start of the gene.  However, the insertion must occur after the start of translation to have any effect.',0,235,3,1),(###-11-18&&

220    Q: In problem 12-8 (E), if WT is inducible and mutant 2 is super-repressible, doesn\'t the WT operon still produce normal repressor protein that, in the presence of sucrose, will become inactive thus allowing the WT operon to make sucrose

220    A: When the normal repressor binds with sucrose and gets out of the way, the super repressor moves in and binds to the operator that is now available. That\'s why super repressor overrides the effects of normal repressor.',0,236,3,1),(###-11-18&&

221    Q: Why does repression have a slow effect

221    A: When you stop further synthesis of new enzymes, the old enzymes are still there and continue to work. It takes a  while for the cell to grow and divide up the old enzyme among a large number of cells -- that\'s how the enzyme/cell faills.',0,237,3,1),(###-11-18&&

222    Q: If there can be two or more codons for one anticodon, is the converse true also? For example, if we have the codon 5\'UGG3\' is the corresponding anticodon 3\'ACC5\' or 3\'ACU5\'.&&

222    A: UGG and UGA cannot normally be read by the same tRNA -- one is trp and one is stop. So the normal tRNA in this case must have 3\'ACC5\' in the anticodon. (If it had 3\' ACU5\' it would put trp in in response to the stop codon.)  In some cases, you can think of more than one possible tRNA to read a particular codon. But when asked for the \"anticodon\" you generally give the perfect match to the codon. When asked what tRNA\'s can be used (with what anticodons) you have to check that particular case -- you have to be sure all codons that would be read by the tRNA in question are synonomous.',0,238,3,1),(###-11-18&&

223    Q: Is the sequence on one strand of the DNA where a restriction enzyme cuts considered one restriction site or are both DNA strands together considered the restriction site

223    A: A single restriction site includes both strands of DNA.  Cutting with a restriction enzyme implies cutting both strands. So both strands comprise one restriction site.',0,239,3,1),(###-11-10&&

224    Q: During the translation process, what if the effect of charging/loading on translation accuracy

224    A: The synthase must fit both AA and tRNA, but the accuracy of the fitting is a different question. The AA must fit correctly twice -- once when it combines with ATP to make AA-AMP, and again when it combines with tRNA to form AA-tRNA. So the tRNA fit on the enzyme surface is checked once and the AA fit is checked twice.',0,240,2,1),(###-11-10&&

225    Q: In translation, if the first tRNA binds the A site of the ribosome, why does Purves show the first tRNA upstream of the start codon

225    A: The first tRNA = met tRNA is not in the A site, but in the P. After that, all new tRNA\'s bind at the A site.',0,241,2,1),(###-11-10&&

226    Q: In the ribosome, is the mRNA groove on the small or large subunit

226    A: The groove is made up partly of each subunit.',0,242,2,1),(###-11-10&&

227    Q: During translation, why does adding a single amino acid to the chain not allow the translation machinery to read 2 adjacent codons at a time

227    A: If you added the single amino acid to the chain, the whole system of A and P sites wouldn\'t work at all. The tRNA with the growing chain would have to stay put and the new tRNA would have to bind to a codon in the mRNA that would be farther and farther away from where the chain was.',0,243,2,1),(###-11-10&&

228    Q: In DNA synthesis, how does editing effect the ability to start chains

228    A: The nature of DNA polymerase requires the last nucleotide (the one at the 3\' end that we are going to add to) be attached to a chain and RNA polymerase does not. Which means that the substrate binding sites of the two enzymes are different. (TA\'s note:  in a sense, having proofreading functionality precludes DNA polymerases from starting a chain de novo since they must first check that the previous nucleotide in the chain is correct.  RNA polymerases like primase have the ability to start an RNA chain from scratch, but at the expense of the inability to proofread in a 3\' to 5\' direction. Stryer \"Biochemistry\" 4th Ed. has a good explanation of this.)',0,244,2,1),(###-11-10&&

229    Q: How can the bond between AA-tRNA (a regular ester bond) provide free energy by its breakage

229    A: The AA-tRNA bond has a large neg. delta G zero of hydrolysis, but I don\'t know the chemical explanation for why it\'s \r\ndifferent from an ordinary ester.',0,245,2,1),(###-11-10&&

230    Q: Where does the energy for making peptide bonds come from

230    A: Hydrolysis of PPi provides the energy to make the AA-tRNA bond. The hydrolysis of the AA-tRNA bond provides the energy to make the peptide bond.',0,246,2,1),(###-12-23&&

231     Q: In problem 9-16B, why does ND have to be at division 2?  Wouldn\'t a ND-1 result in the 4 gametes and 2 empty cells?  &&

231     A: I think you are getting into trouble because you are confusing X shaped chromosomes (with 2 chromatids) and the X chromosome -- the one named X.  Draw out first and second div. again, using two chromatids per chromosome, and putting a cb or normal allele on each chromatid. (Be sure not to confuse chromatids and chromosomes.) You need to get a gamete from the mom with two X-cb chromosomes (each with one chromatid -- not X shaped). That can only happen if ND is at 2nd div.',0,248,4,1),(###-12-23

232     Q: What is genetic drift

232     A: Genetic drift is any change in allele frequencies due to statistical fluctuations (sampling errors) in small populations. Changes that are due to picking more or less of one genotype or another just by chance. Changes that are not due to any properties of the resulting phenotypes. If you flip a coin only a few times you don\'t get exactly 1/2 heads and 1/2 tails. If you have only a few people, their descendants will not carry a statistically valid sampling of their parents\' alleles. You are not expected to understand the various fine points and types of genetic drift.',0,249,4,1),(###-12-23&&

233     Q: Can a male be a carrier for a recessive sex linked condition

233     A: A disease like PKU is autosomal, so a male and female are the same with respect to it. If a gene is on the X, a male can not be a silent carrier of a recessive allele. If he has the recessive allele, it is expressed. If he has the normal allele, that one is expressed. Only a female can can be a silent carrier of a recessive, X linked condition -- carrying a recessive, disease causing allele, but showing no symptoms.',0,250,4,1),(###-12-23&&

234     Q: In the moss life cycle, where in the process do you have meiotic haploid products dividing by mitosis

234     A: The haploid spores land on the ground, germinate and divide by mitosis to produce the fuzzy stuff.',0,251,3,1),(###-12-19&&

235     Q: In problem 15-5 (C), why are the resistant rats able to degrade the drug rather than simply not absorb it

235     A: You are supposed to think here about the basis of dominance. If you have a heterozygote, there are two alleles present, so you should be producing two different proteins, one from each allele. So consider, what combination of enzymes/transporters should you have in the heterozygote? If you have a mix of active and inactive transporter proteins, will the drug get transported into the cells? If you have a mix of active and inactive enzymes (some can degrade the drug and some can\'t) will the drug get degraded?',0,252,4,1),(###-12-19&&

236     Q: Will we be supplied the meiosis and mitosis flow chart with the labeled steps

236     A: This one you have to memorize. It\'s like knowing the alphabet. Seriously, the basic features of meiosis vs mitosis are fundamental -- it\'s like knowing which is the RNA and which is the DNA. As you can tell from reading the book, we have left out many picky details which you DO NOT have to know.',0,253,3,1),(###-12-19&&

237    Q: In problem 10-7, how can the genes sort independently

237    A: If there are only 3 types of chromosomes, then 2 of the genes must be on the same chromosome. If they are far enough apart on the same chromosome, then multiple crossovers will give the same results as if they were on separate chromosomes, (i.e. independent assortment).',0,255,4,1),(###-12-19&&

238    Q: In problem 10R-6 (Part III), how do you determine epistasis

238    A: You write out all possible orders for the pathway. Then you ask, which one(s) do the results fit? In this case, you can block the pathway so that only met will do, or either met or Y. That means pathway must be X --> Y --> met not Y --> X --> met. (Try it and see.) The basic procedure is trial and error. For part H, you figure out from the complementation results which step is blocked (which enzyme is missing) in mutant 4. What ever mutations do not complement mutant 4 must be blocked at the same step.',0,256,4,1),(###-12-19&&

239    Q: If in a karyotype we see two chromosomes that have centromeres at the same locations and that are the same size, can we always assume that they are homologous

239    A: That is the usual assumption in the cases we discuss. Usually there are only 2 chromosomes that look the same. However in real life, you can\'t be sure without banding the chromosomes -- there are several pairs of homologs that have the same size and their centromeres in the same position. So two that \"look\" the same (without banding) may not be homologs.',0,257,4,1),(###-12-19&&

240    Q: Does the nature of DNA replication during interphase not allow the two sister chromatids ever to flip relative to each other

240    A: I am not sure what you mean here. Do they cross over? Probably not. At any one moment, which is on the \"right\" or \"left\" is arbitrary. Also which is which usually makes no difference, as both are genetically identical.',0,258,4,1),(###-12-19&&

241    Q: Given two homologous chromatids with 4 unlinked genes, can we treat it as if the 4 alleles were on different chromosomes even though they are not

241    A: I am not sure why you say 4 genes -- do you mean 4 copies of one gene or 2 genes, 2 copies of each?  In any case, if genes are far enough apart on the same chromosome, then outcome for gametes is the same as if they were on different chromosomes.',0,259,4,1),(###-12-19&&

242    Q: With independent assortment, do all gametes produced look like the parental cell in that they have one chromatid from each pair of chromosomes

242    A: Parental cell has two chromosomes from each pair, gamete has only one. Gametes do not have same genotype as parents unless parent is homozygous. The gamete gets one chromosome/chromatid from each pair, and it can have sections that were originally from grandma and sections that were originally from grandpa.',0,260,4,1),(###-12-19&&

243    Q: If it is specified that the two genes being considered are unlinked and autosomal during gamete formation, does this mean that subsequent gene formation will be done via independent assortment

243     A: Independent assortment can be due to multiple crossovers between genes on the same chromosome or to independent distribution of genes on separate chromosomes. This could be either case. Also genes specified are not on the X or Y.',0,261,4,1),(###-12-19&&

244     Q: Do some plants spend their lives in a haploid state, become diploid upon fusion, then almost immediately split via meiosis and again become haploid

244     A: True for very simple plants only. Not the average flowering plant.',0,262,3,1),(###-12-19&&

245     Q: If humans all begin as two gametes, become zygotes that have 23 homologous pairs of chromosomes, go through interphase and mitosis, and eventually undergo specialization, when is the DNA content 4C and when does meiosis occur

245     A: The DNA content is only 4C after S and before M. In G1, it is 2C. The germ cells that produce the eggs and sperm go through meiosis. The eggs and sperm are the product of meiosis.',0,263,3,1),(###-12-19&&

246     Q: In problems involving crosses, such as 10-15, cross do we always assume that the F2 was made by F1 X F1

246     A: Yes, unless it says otherwise.',0,264,4,1),(###-12-19&&

247     Q: In problem 10-12, why does one consider the original parents instead of the F2 parents as those to which the recombinants differ

247     A: The F1 look the same whether they are AB/ab or Ab/aB. Which is the right combination determines what is \"parental\" -- AB and ab or Ab and aB. Which is parental follows from what the parents were. That\'s why one refers back to the parents.',0,265,4,1),(###-12-19&&

248     Q: In problem 10-11, when it asks what the proportion of the gametes that carry dominant alleles of both genes, does it mean A_B_ or AABB

248     A: Gametes are haploid. It means what proportion of gametes are AB. I think you are confusing zygotes (which are diploid) with gametes.',0,266,4,1),(###-02-10&&

249     Q: Question 2 of exam #1 asked questions concerning glycosylation, a term whose meaning was not clear to me. I feel that the exam ended up testing my use of non-emphasized, esoteric vocabulary rather than my knowledge or ability to apply my knowledge.&&

249     A: I think the term is used pretty liberally in texts and in the problem book. It wasn\'t meant to be sneaky or technical -- it\'s the just the standard term for \"with sugars added,\" similar \r\nto \"phosphorylated.\" When students asked during the exam, it seemed to me that you ought to be able to figure out what it meant even if you didn\'t remember the exact term, given that we \r\nhave talked alot about adding sugars (glucose = most common sugar) and used many similar terms. However, I may be wrong, and I may have forgotten that it is not an obvious term that \r\neveryone knows because it is so familar to me. I\'ll have to see how most students answered the question. (We always check a reasonable number of papers before deciding on the grading \r\nscale. If no one knows what a term means, we adjust the grading.)\r\nIn any case, I think that it is only a few points and not worth worrying too much about.',NULL,1483,1,2),(###-12-19&&

250     Q: If a 2N sporophyte cell undergoes meiosis to make an N spore, why doesn\'t an N \"zygotaphyte\" cell undergo fusion to make a 2N zygote

250     A: Sporophyte means multicellular diploid; some of its cells will eventually undergo meiosis to make haploid spores. Gametophyte means multicellular haploid -- some of its cells will specialize to become gametes and these will fuse to form a zygote. (A gametophyte is haploid, but not just any old haploid cell can fuse with another -- some of the cells have to specialize first before they can fuse.)  You have to make gametes before you can get a zygote, which is why the multicellular haploid is called a gametophyte and not a zygotaphyte.',0,268,3,1),(###-12-19&&

251     Q: When you say \"parent\" does that always refer to the  P1?  In some cases it seems that what is really being  referred to is the grandparent.&&

251     A: It depends on the case. Hopefully, it is always clear from context. Parents may refer to the actual parents of the offspring being examined or to the parental generation in a series of crosses.',0,269,4,1),(###-12-19&&

252     Q: In lecture 21, how does a \"limiting amount\" of enzyme give rise to incomplete dominance

252     A: The presence or absence of an enzyme often controls some trait -- some aspect of phenotype, such as color. The enzyme catalyzes some reaction and the product of the reaction determines the phenotype (height, color, position of flower, etc.) If there is an intermediate level of enzyme, you may or may not get an intermediate level of product and an intermediate phenotype. It all depends on whether you have spare enzyme (in the normal state) or not. When you have less enzyme, then the max. rate of the reaction catalyzed by that enzyme will be lower. (Lower Vmax.) If the enzyme normally operates well below it\'s Vmax, then having less enzyme makes little or no difference. (If you have enough substrate, the enzyme can reach the same V as before, even with less enzyme. Each molecule operates closer to its max. rate. To look it at it another way, if you had excess enzyme, then having lass doesn\'t matter.) This is what I meant by \"the amount of enzyme is not the limiting factor in how much product is made.\" But if the enzyme is operating near its Vmax, if you have less enzyme, you won\'t have enough to convert S to P as fast as before. (In this case you didn\'t have extra enzyme.) So you will get less product, and the phenotype may be different -- less pigment, not as tall -- something intermediate between having more enzyme and no enzyme.',0,271,4,1),(###-12-19&&

253     Q: Do germ cells go through the same cell cycle as somatic cells? If so, where does meiosis fit in

253     A: Germ cells are diploid cells specialized to do meiosis, sooner or later. Somatic cells never do meiosis. (Sometimes people also refer to gametes as germ cells.) If germ cells divide by mitosis before they get to meiosis, their mitotic cell cycles are the same.',0,272,3,1),(###-12-19&&

254     Q: What is the difference between chromosomes and chromatin

254     A: Chromosomes = super condensed state of chromatin. However, when people talk about chromatin, they usually mean interphase chromatin. So they often use \"chromatin\"  meaning the loose form of the DNA/histone  complex. Alternatively, people use \"chromatin\" when they want to emphasize they are talking about DNA complexed to histones, not naked DNA without protein attached.',0,273,4,1),(###-12-19&&

255     Q: Does transcription take place during G2 and S of the cell cycle, or only during G1? If it does take place, does the RNA polymerase use both chromatids\' antisense strands or just one

255     A: Transcription takes place in every part of interphase, including S. It is not clear how the RNA polymerase and DNA polymerase manage not to interfere with each other in S. The polymerase probably uses as many copies of the gene as it can find. So in G2, it probably uses both copies on both sister chromatids. For some genes, there is regulation of transcription, something like repression, so that the total level of mRNA production stays the same in G1 and G2 (even with more copies of the DNA). In this case, less mRNA is made per gene copy. For some genes, transcription is constitutive, so more mRNA overall is made in G2.',0,274,4,1),(###-02-07&&

256     Q: If one were to remove kinase from the cell all together, would the Na+/K+ pump effectively shut down, preventing active transport since ATP would not be able to be split

256     A: The kinase is part of the pump. Without kinase activity, the pump doesn\'t run. If you pull the plug out of the wall, the mechanical pump doesn\'t run. Same idea -- no ATP hydrolysis, no \r\nenergy source.',NULL,1482,1,2),(###-12-19&&

257     Q: In problem 8R-2 (B),what is the difference between the choices (chromosome, chromatid, ssDNA)

257     A: A chromosome can have one or two chromatids, depending on whether it is before or after the S period of DNA replication. Your probe is hybridizing separately to each chromatid (= each ds DNA molecule carrying a copy of gene G). Your probe must be hybridizing to only one of the two strands of each copy of gene G, not to \"each single strand of DNA from gene G.\"  Remember your probe is single stranded, so it is only complementary to one \"side\" of the DNA in gene G.',0,276,4,1),(###-12-19&&

258     Q: If Lyon\'s hypothesis is true and extra X\'s are genetically inert, why should someone who has a Kleinfelter XXY be any different from a regular male with XY

258     A: There are 2 possibilities. Either the extra X\'s are not inactivated right away, and something goes wrong in the period before they are inactivated. Or, the problem is the consequence of the actions of the genes on the X that are not inactivated -- there are a few of these (the psuedoautosomal genes that have homologs on the Y.)',0,277,4,1),(###-02-07&&

259     Q: In the Na+/K+ pump, where the ATP is always on one side of the pump, when there is a high concentration of Na+ on the outside of the cell, why would all the ATP necessarily be on the INSIDE

259     A: Because the ATP is being broken down by the kinase on the inside of the cell (E1 side,\r\nwhich binds Na+).',NULL,1481,1,2),(###-12-18&&

260     Q: In problem 10R-6 part II (D): How can recombination/crossing over occur in the haploid cells before they fuse

260     A: Crossing over occurs in the diploid zygote when it goes through meiosis to make haploid spores. It can\'t occur in the haploids, as you\'ve noticed. It does occur in the diploid zygote during meiosis, but that affects the haploid spores that result from meiosis, not the zygote itself.',0,280,4,1),(###-12-18&&

261    Q: In question 10-2 (C), why do we need to look at the F2 to make sure that there are in fact two separate genes

261    A: From the information given in the problem, the most straightforward explanation is that there are two sep. genes controlling the 2 traits. However, I am sure there are other possible explanations, although I can\'t think up a good one at the moment. What I meant by the comment that you should do an F2, is that the best way to confirm a hypothesis is make a prediction and test it. Your hypothesis is based on previous results -- if you did a good job it fits, because you designed it to fit all previous results. To prove it is really correct, you need to predict the results expected in a new situation. (We usually don\'t go this far -- usually we ask for a good explanation of the results, and don\'t ask for suggestions about additional experiments.)',0,281,4,1),(###-12-18&&

262    Q: Why is the answer to problem 9-15 dominant

262    A: If condition were recessive, the would both be homozygous recessive and so should their child, who would then have the condition.',0,282,4,1),(###-12-18&&

263    Q: In problem 9-13, does \"carrier\" of a disease mean heterozygous for that disease

263    A: Yes. It means able to pass on the allele for the disease without having symptoms yourself.',0,283,4,1),(###-12-18&&

264    Q: In problem 9-12, can the male with brachydactyly be BB

264    A: Unlikely.',0,284,4,1),(###-02-07&&

265    Q: Can you have active transport that does not go against its gradient? For example, can you use active transport (primary or secondary) to move a substance from high concentration to low concentration

265    A: No. Why bother? Do you need a pump to move water down hill? I can conceive of a situation where you might want to pump the water very quickly, but as far as I know, there is no biological \r\nequivalent -- all transport down a gradient that I know of is passive.',NULL,1480,1,2),(###-12-18&&

266    Q: What is the difference between somatic cells and germ cells

266    A: Somatic cells = body cells = cells that reproduce only by mitosis. Germ cells = cells that will give rise to gametes or spores by meiosis.',0,287,3,1),(###-12-18&&

267    Q: In lecture 21, why don\'t we consider the possibility of crossing over between homologous chromosomes during independent assortment

267    A: If genes are on separate chromosomes, it makes no difference to the outcome whether crossing over occurs or not. You get the same gametes either way. Try it -- Put A and a on one pair and B and b on another. The chromosomes will look different at some stages of meiosis, if crossing over occurs, but what ends up in the gametes will be the same. (Still 1/4 each possibility.)',0,288,4,1),(###-02-06&&

268    Q: We note in lecture 5 that \"the concentrations needed to reverse pumps are not reached in cells, but can be achieved in test\r\ntubes (by adding ATP, setting up ion gradients, etc.)\" How can\r\nadding ATP affect the concentrations

268    A: If moving ions drives phosphorylation of ATP, than hydrolysis of ATP can push ions. But it takes a very large pile of ATP.',NULL,1479,1,2),(###-12-18&&

269    Q: What letter grade does the mean correspond to, and what does the standard deviation mean

269    A: The mean is usually around the B/B- cut off -- generally a low B. We ignore the SD; we just give it because people ask.',0,290,4,1),(###-12-11&&

270    Q: In crossing over, can a crossing event occur at any location -- even for genes on the same chromatid

270    A: Crossing over can occur at point along the chromosome, but it only makes a difference if it exchanges pieces of non-sister chromatids. (If it exchanges pieces of identical sister chromatids it makes no difference.)',0,291,4,1),(###-12-11&&

271    Q: In problem 14-5 (C), how is it possible to know the AB frequency in the next generation by just knowing that the previous generation reached genetic equilibrium

271    A: If a population is in equilibrium, you assume it will persist, unless something changes. One point of the Hardy-Weinberg equilibrium is to treat the whole population as one \"pot\" and not subdivide it into separate generations. (Of course, it is possible to disturb equilibrium, but in the absence of unknown factors, you assume you haven\'t.)',0,292,4,1),(###-12-11&&

272    Q: If a population is in a state of equilibrium wherein the frequencies of alleles don\'t change, can the frequencies of genotypes change

272    A: The genotype frequencies may or may not change, depending on the extent of nonrandom mating and how long it has been going on. However the critical point is that you know something is not standard because the genotypes won\'t fit the Hardy Weinberg  proportions.',0,293,4,1),(###-12-11&&

273    Q: Does the Barr body get transcribed and not translated or simply replicated in mitosis

273    A: Barr body is largely untranscribed. (A few areas of the \"inactive X\" are transcribed, but most of it is not.) It is replicated in S and passed on in mitosis.',0,294,4,1),(###-12-11&&

274    Q: How does the \"i\" allele for blood types create more phenotypes in addition to genotypes

274    A: I think  what I meant is that if you have only 2 co-dominant alleles, there are 3 genotypes corresponding to 3 different. phenotypes. If you add in a 3rd, recessive allele,  that increases the number of both phenotypes and genotypes. You now have the heterozygotes that are different in genotype but not in phenotype, but you also have the homozygous recessive, which is a new phenotype as well as genotype.',0,295,4,1),(###-12-11&&

275    Q: In problem 10-6 (D), since the 2 genes are very far apart, shouldn\'t there be multiple crossing over events and therefore 3 different possibilities of gamete proportions

275     A: The ans says that there will be 4 types of gametes in equal proportions. That is the result expected if there are lots of multiple cross overs.',0,297,4,1),(###-02-06&&

276     Q: When should I take into account the electrical gradient when solving a problem?\r\nShould I only worry about this gradient when working with ion channels

276     A: Only worry about it where ions are concerned.  With neutral substances, the electrical gradient is irrelevant -- it doesn\'t push them around, and their movement doesn\'t affect the gradient either.',NULL,1478,1,2),(###-02-06&&

277     Q: In problem 2-4, how exactly does the serine enter the sans-protein vesicle? Graph 1 suggests some serine uptake, but before the carrier protein P is inserted, shouldn\'t there be no serine uptake

277     A: There could well be a trace amount of uptake. The rate is meant to be neglible (but nonzero) compared to the uptake with protein.',NULL,1477,1,2),(###-02-06&&

278     Q: What exactly is the difference between an endosome and a lysosome?\r\n&&

278     A: Endosomes store and separate ligands and receptors by way of an acidic environment from H+ ions pumped in via active transport. A lysosome is more acidic, but it also contains active hydrolases. (An endosome may or may not contain hydrolases, but they won\'t be active.)  An endosome is a step on the way to a lysosome, and a stage on the way to other things as well. As vesicles develop, it may be one type of vesicle matures into another or one \r\nfuses with others or both to achieve changes in contents.',NULL,1476,1,2),(###-11-29&&

279     Q: Does \"c\" usually refer to one double stranded DNA molecule? For example, does \"4c\" mean that there are 4 DNA molecules present

279     A: No. \"c\" is not measured in molecules, it is measured in grams. It is the amount of DNA (in grams) in one set of haploid, unduplicated chromosomes. (The minimum amount of DNA to be found in a cell in any stage of the life cycle.) If a cell is \"4c\" it means that there are 4 copies of each TYPE of DNA molecule = 4 copies of each homolog = 2 homologs of each chromosome type, and each one has 2 chromatids = 4 chromatids of each type, total.',0,304,4,1),(###-11-28&&

280     Q: In problem 7R-1 (part V), why will nucleotides #25-27 be in the P site and not 22-24? Will #25-27 be added to the peptide

280     A: Ribosome is moving right to left, if mRNA = sense (bottom strand) with U instead of T. The last complete codon is 3\'ACG5\' = 22-24. Ribosome can be positioned with this codon in A site and previous in P site. When last bond is made, this codon (and tRNA with chain) will  be in the A site, not the P site. The previous tRNA that just gave away the chain will be in the P site. The ribosome cannot move to the left (relative to the mRNA) one codon because there is not another complete codon. So this as far as the ribosome can go before it starts to fall off. The codon corresponding to 25-27 is read -- it codes for the next to last amino acid. I think you may have trouble with this because we usually draw translation going left to right, and you haven\'t reversed everything properly.',0,305,3,1),(###-11-18&&

281     Q: When cDNA is transcribed off of mRNA using reverse transcriptase, are the UTRs always copied as well as the coding sequence

281    A: For the UTR\'s, it depends what primer you use. The transcriptase extends the primer to the end. If you use poly T, the poly A gets transcribed too. I don\'t think the cap is transcribed, but that is only one base. The reverse transcriptase does not always make it all the way to the 5\' end of the mRNA, but that is the goal. The cap is not copied and does not matter. If you use oligo T as the primer, you will get some random length of the polyA copied, depending on where the oligo T (~20-mer) happens to sit down.',0,306,4,1),(###-09-13&&

282    Q: In question 1.23(b) in the problem book, it asks for the orientation of the #4 carbon of galactose in the chair form, yet the answer key states that all -OH groups are equatorial for glucose. Is the question supposed to state glucose or galactose

282    A: Admittedly, the answer is a bit terse and even misleading, stating simply that in glucose all the OHs ore equatorial. Moreover, it is not even correct, as in alpha-glucose the the C1 OH is axial and down. Thanks for catching this. A better answer is: In glucose, the hydroxyls at C2, C3 and C4 are aways equatorial and out. (This fact need not not be meorized, as you are told to view a diagram of glucose in the chair conformation.) In galactose, it is stated that the orientation of the H and OH at C4 are opposite that of glucose. Thus in galactose, the the OH must be axial rather than equatorial, and so the H must be equatorial. ',0,307,1,1),(###-09-13&&

283    Q: In the web notes for lecture 2, you show the top H atom of an amide bonding to the oxygen atom of a water molecule. Would that still be hydrogen bonding since it isn\'t O-H-O (but rather H-O-H)

283    A: All the H-bonds can be interpreted as OHO or NHO (or OHN), with no HOH\'s implied. And almost all the O\'s and N\'s of the amide could and would be participating in H-bonds at any given moment. ',0,308,1,1),(###-09-14&&

284    Q: In reference to problem set 1-22C, why doesn\'t the N in NSG get protonated at pH7 while the N in glucosamine does

284    A: The N in NSG is part of an amide functional group, and the electronegativity of N in that environment is not strong enough to attract a proton. So an amine R-NH2 is not the same as an amide R-CO-NH2, which has its own characteristics as a different functional group (see list of functional groups). You could make an analogy to the OH of a corboxyl; without the C=O present, the -OH is not usually an acid, but when the C+O is attached to it, it is, and can lose the proton. ',0,309,1,1),(###-09-19&&

285    Q: What in the primary structure of a protein dictates whether a structure will form alpha-helix or beta-sheet for secondary structure

285    A: The side groups can influence how stable a particular secondary structure might be. For instance, if there are two glutamic acids 4 residues apart in an alpha helix, they would be placed near each other, one over the other, but they would repel each other because of their like electrical charges. In a beta sheet, on the other hand, they would not be near each other. So situations like this influence just what form of secondary structure is preferred for a given region. ',0,310,1,1),(###-09-21&&

286    Q: What is the charge on histidine? And regarding arg, does the R group get a single + charge or double/triple charge since it has three amino groups present

286     A: Arg has a single charge distributed over the 3 N\'s of the side chain (guanido group). Histidine, with a pK of ~6 is partially charged at pH7. As we will only treat all-or-none cases here, there will be no questions regarding the ionization state of histidine. ',0,311,1,1),(###-09-21&&

287     Q: Isn\'t glycerol, when part of a phospholipid, considered hydrophobic because it\'s non-polar? Why is just the H-C tail considered phobic when glycerol \"gives up its OH\" during dehydration synthesis

287     A: The glycerol residue in a lipid is considered to be contributing to the ester bond, which is a polar group. Since it is essential for the formation of the ester group, the ester group can be considered to be approximately half from glycerol and half from the carboxylic acid. ',0,312,1,1),(###-09-21&&

288     Q: In paper chromatography, what exactly is an Rf value and what does it signify

288     A: Rf is the ratio of the distance moved by your favorite compound to the distance moved by the liquid front at the time the experiment is stopped. A large Rf would be characteristic of a more hydrophobic compound, since hydrophobic compounds will be more soluble in the more mobile, non-polar solvent, and so will travel farther than hydrophilic compound. which spend more time in the stationary, polar solvnet. ',0,313,1,1),(###-09-21&&

289     Q: In fingerprinting, what exactly does a \"spot\" refer to? An amino acid or a protein

289     A: Neither. In fingerprinting the spots are small peptides (\"sub-peptides\"), fragments derived fromthe original large polypeptide by enzymatic hydrolysis at specific points (e.g. trypsin cleaving after lys and arg). If the spots were single amino acids, it wouldn\'t be much of a \"fingerprint,\" and nearly all proteins would give the pattern, since nearly all proteins are comprised of all 20 amino acids. ',0,314,1,1),(###-09-23&&

290     Q: In the web-lecture #5, the picture labeled \"Alpha-helix 3.6 aa\'s per turn\", the \"chain links\" are shown as NHCRHCOs (the R and one of the H\'s are implied), with the nitrogen being on the same plane as the <i>second</i> nearest carboxyl carbon. However, in the diagram immediately preceding this depiction, we see that the pivot carbon is actually the carbon that is attached to the R, suggesting that a nitrogen would be on the same plane with it\'s <i>nearest</i> carboxyl carbon, but not it\'s second nearest. Have I overlooked something or am I viewing it wrong

290     A: I can understand your confusion. The blue \"plates\" in the lecture diagram to which you refer are not meant to represent atoms in a plane but just amino acid residues, so that they would be easy to count in the search for the NH H-bond donor. ',0,315,1,1),(###-09-24&&

291     Q: About problem 1-23B: The key also states equatorial and OUT. What does in and out refer to in this case? I understand the difference between axial and equatorial, however it seems that there are still equatorial up and equatorial down subsituents.&&

291     A: Equatorial and out are redundant terms. Up and down refer to the flat ring representation and equatorial and axial up and axial down refer to the chair representation. And equatorial and in is nonsense. But equatorial and up is a contradiction: better would be equatorial and \"higher\" (or upper, relative to the othe non-ring bond, to hydrogen). And equatorial and \"lower\". ',0,316,1,1),(###-09-25&&

292    Q: How does treatment with urea change the MW of a protein if urea just disrupts weak bonds and interactions? I thought urea only changed quaternary, tertiary and secondary structure.&&

292    A: Because urea can disrupt quaternary structure, it can conver a native multi-subunit protein to its constituent subunits (as long as there are no disulfide bonds holding the subuints together). The subunit will have lower MWs than the the intact native protein. ',0,317,1,1),(###-09-25&&

293    Q: Does urea only act on hydrogen bonds by competing with the ability of side chains to h-bond? If so, how does it affect, or does it affect, ionic, VDW and hydrophobic interactions

293    A: The exact nature of urea dentaturation is not understood, as far as I know. Because of its ability to H-bond extensively, it is likely to disrupt H-bonds and ionic bonds. ',0,318,1,1),(###-09-25&&

294    Q: Can a strong salt denature proteins by disrupting ionic bonds? As in, will the salt ions compete with side chains to form ionic bonds

294    A: High salt can denature proteins, but more often it counteracts repulsive ionic charges that keep proteins from associating illegitimately with each other. Thus a high concentration of ammonium sulfate is often used in protein purification: at a given concentration (several molar), certain proteins will precipiate out of solution, while others will not, so you can collect the precipitate, return it to a low salt condition, and thus resolubilize the native (never unfolded) protein. ',0,319,1,1),(###-09-25&&

295    Q: In reference to beta pleated sheets--what is meant by parallel or anti-parallel

295    A: Parallel refers to two polypeptide chains that are both lined up in the same direction, e.g., considered as sections, N terminal on the left and C-terminal on the right for both. Antiparallel is the opposite case, where one strand can be written N to C and its neighbor C to N, and so on for more strands to make a sheet. Beta sheets can form either way, but the antiparallel case is more common, as the polypetide just keeps b doubling back on itself to form the sheet. See the ribbon models of protein 3-D structure in your texts. ',0,320,1,1),(###-09-25&&

296    Q: With amino acids that have acidic/basic functional groups, how do we know when they will be protenated or not

296    A: The guideline is that carboxylic acid groups are de-protonated at pH7 and that amines are protonated at pH 7. The protonation can be influenced by pH in the laboratory, but in the living cell, the carboxylic acid group and amines can be assumed to be very predominantly charged. ',0,321,1,1),(###-09-25&&

297    Q: When discussing hydrophobic interactions the lecture notes say, \"water molecules surrounding an apolar molecule take on a relatively ordered structure.\" This idea is also pictorially represented by all the water molecules around octane having the same orientation. Why does water naturally do this when it is surrounding \"an apolar molecule\"

297    A: The picture is not meant to imply a particular orientation for the water molecules surrounding the octane, but just that they are constrained from totally free movement: they can only H-bond to other water molecules on one side, but not on the other. Without the octane, they would have more possibilities, and so a higher entropy. ',0,322,1,1),(###-09-25&&

298    Q: What does one mean by the word \"extractable\" in the context of \"extractable in organic solvents?\" &&

298    A: Extractable means more soluble in the organic solvent than water, so that if you shake up a substance in a mixture of benzene and water (which do not mix much but rather form two layers, as in salad dressing), it will be preferentially soluble in the benzene and most of it will be found in that layer. Lipds are defined as being substances of this type, exteractable into organic solvents. ',0,323,1,1),(###-09-25&&

299    Q: Why is it called a phosphoESTER linkage if there is no carbonyl group in the bond

299    A: Carbonyls are not obligatory to the definition of an ester, which is the product of dehydration between an acid (any acid) and an alcohol. Thus in fats we have, to be more accurate, carboxylic acid esters; and in phospholipids, we have, in addition, a phospho-ester (an ester between phosphoric acid and an alcohol). ',0,325,1,1),(###-09-25&&

300    Q: Should we understand how to name phospholipids, like those that are discussed on page 7 of lecture three notes in the section, \"phospholipid and bilayer membranes?\"&&

300    A: You do not need to memorize the structure or name of particualr phospholipids (e.g., what is lecithin?). ',0,326,1,1),(###-09-25&&

301    Q: What types of molecule pairs are particularly likely to experience Van der Waals forces

301    A: Any two molecules that can approach each other within naroow distance limits at which VDW forces are effective. There is no specialization. ',0,327,1,1),(###-09-25&&

302    Q: Can catalysts can change equilibrium amounts of reactants and products

302    A: No, catalysts do not change the equilibrium. The equilibrium condition does not depend on time, and so it depends only on the relative energy levels of the reactants and the products, no matter what the route between them. If it takes ninety years to reach equilibrium, so be it. ',0,328,1,1),(###-09-25&&

303    Q: In Handout 2 on polysaccharides, the maltose sugar structure suggests that the second glucose can be either alpha or beta. Is this because of the fact that the OH on C4 for both beta and alpha glucose are equatorial out? But if the second one is a beta, wouldn\'t the second linkage be a beta bond instead of alpha? Then is maltose made of alpha or beta linkage

303    A: The right hand glucose is free to assume the straight chain form and the ring again and agian, so it is in the same equilibrium with respect to C1 as the free sugar. The anomeric C1 OH orientation is independent of the situation at C4.The enzyme that catalyzes the formation of starch (which is what you are starting to do if add a third sugar to maltose) will only accept an alpha orientation at C1. So it waits for that kind of ring to form (not long) before it springs into action. ',0,329,1,1),(###-09-25&&

304    Q: For problem 2-14 in the problem book, I was under the impression that the polypeptide chain, although denatured in urea, would fold back into its original position upon removal of the urea. The answer key however states that it will not refold itself the same way (ie. removal of urea does not rejoin bonds). Is this be the case with all chaotropic agents

304    A: In 2-14, the point in this made-up example is that the ribonuclease DOES return to its original 3-dimensional conformation, except for the reformation of the one covalent bond, the peptide bond, that was cleaved. All the weak bonds (non-covalent bonds), that are so important in holding the molecule in its distinct tertiary structure have returned. In a way, the protein has been converted from a single polypeptide protein to a multimer of 2 subunits, held together by weak bonds, and whose 3-dimensional structure is essentially the same as when that peptide bond was still there. So this example is sort of a super-Anfinsen experiment, renaturation was achieved even though a a covalent peptide bond was broken and not restored. Denaturation-renaturation never involves breaking or reformation of peptide bond; the only covalent bond allowed to break in denaturation-renaturation is the disulfide. ',0,330,1,1),(###-09-25&&

305    Q: I have a question about problem 2-3, part E. The answer states that the best method of separating 2 polypeptides (one with no overall charge, the other with unknown charge, connected by hydrophobic forces, and with equal MW and shape) is gel electrophoresis without SDS. I was under the impression that all of the protein purification methods we discussed in class worked for the separation of proteins, not polypeptides that make up a single protein. Is this indeed the case? And if not, would you not have to denature this protein in order to break the hydrophobic connections between its 2 polypeptides

305    A: Most of methods we discussed apply equally well to single polypeptide chain proteins or proteins that have quaternary structure (multi-polypeptide proteins). The exception is SDS-PAGE, since the denatures the proteins, most multi-polypeptide proteins would be converted to lower MW polypeptide chains. But I see the source of your concern: on the one hand you are told that the enzyme consists of 2 polypeptide chains, which would be held together by weak bonds. On the other hand, the answer says the best way to separate these 2 polypeptides would be native PAGE. Yet in native PAGE one would expect the quaternary structure to be maintained, since it is carried out under non-denaturing conditions. The resolution must be that you are given the 2 individual chains, already disassociated by an unnamed method, and now are asked to purify (separate) one from the other. Since they are the same MW and shape, one must rely on a possible charge difference, so native PAGE is called for. ',0,331,1,1),(###-09-25&&

306    Q: In problem 1-22A(5): Can you please explain how switching the hydroxyls on Carbons 3 and 4 will change the structure

306    A: You would have a different stereoisomer. Two sugars with one switched OH will not be superimposable. These stereoisomers are often given different names, like mannose, glucose, galactose... ',0,332,1,1),(###-09-25&&

307    Q: In problem 1-23B: The question asks about galactose, but the answer refers to glucose; which should we work with? Also, can you please explain the structure of galactose and how it differs from glucose.&&

307    A: As stated in the problem, galactose differs from glucose in the orientation of the OH on C4. They are switched on C4, galactose and glucose; that\'s the only difference. So if in glucose, all the non-anomeric OH\'s are equatorial, then in glucose, the OH at C4 is equatorial, then in galactose where the OH is switched relative to glucose, the OH must be axial, not equatorial, and so it is the H that is equatorial, which is the answer. ',0,333,1,1),(###-09-25&&

308     Q: In Exam 1, 1999 Question 2A: Can you please clarify why the L-amino\'s will not fit? I thought the only difference between L and D was the orientation in space

308     A: Yes, the only diffference and a critical difference if you are an enzyme that is designed specifically for a particular spatial orientation. It\'s like try to put a left-hand glove onto your right hand. The enzyme must cuddle up to the alpha C-H side of the D-amino acid. If there is a side group there instead of a little H, it will not be able to do so. Of course glycine is OK as it has nothing but an H on either side of the alpha C. ',0,334,1,1),(###-09-25&&

309     Q: In Exam 1, 1999 Question 2B1: Why do we not include the charge of histidine as +1

309     A: It\'s not quite a strong enough base to have a full charge at pH 7 and so we ignore it in the interest of simplicity in this introductory course. So I should have left it out of these proteins. ',0,335,1,1),(###-09-26&&

310     Q: I have a question about how paper chromatography impacts a peptide. In problem 2-13 E, I was surprised to see that PC was the answer. Wouldn\'t this technique tear the molecules apart, separating the polar amino acids from the non-polar? In the 1999 exam (problem 2B2), the answer key indicates that fingerprinting does indeed break apart peptides.&&

310     A: Fingerprinting involves several steps. You partially hydrolyze the protein (break it down into short peptides) BEFORE doing the separations. The separations themselves (electrophoresis or chromo.) do NOT break any bonds. They only allow you to separate molecules that have already been broken apart. (This answer could allow for the electrophoresis half of fingerprinting to be the part of the process which breaks apart the peptide.) ',0,336,1,1),(###-09-26&&

311     Q: Is the anomeric carbon the only one that can form a glycosidic bond

311     A: Yes.  Technically, the anomeric acrbon in the ring is part of a hemiacetal (OH + aldehyde).  It can go on to form a full acetal (2 OH\'s + aldehyde). The reaction mechanism involves the ring oxygen (transient C=O formation there). None of the other OHs have this possibility. See slides 24-26 at <a href = \"http://www.chem.uic.edu/wardrop/teaching/chem234/files/WARDROP-234-26.ppt\">www.chem.uic.edu/wardrop/teaching/ chem234/files/WARDROP-234-26.ppt</a> for more.',0,337,1,1),(###-09-26&&

312     Q: How do phospholipids emulsify fats

312     A: They use their hydrocarbon tails to bind to the fat globules and break them into smaller and smaller droplet, all coated with phopholipid. The charged head groups of the phospholipid now allow these small droplets to be semi-soluble in an aqueous environment and to be more easily assimilated into the tissues. ',0,338,1,1),(###-09-26&&

313     Q: In lecture 5 on the different types of weak bonds that contribute to 3 degree structure of proteins, I don\'t understand why the asp -asn asn interaction is called a \"H-bond to ionic.\"&&

313     A: True, it is not really an H-bond, but rather an electrical attraction between the fully charged arg side group and the partially negatively charged parts (delta minus) of the polar asn amide group (the O and the N). ',0,339,1,1),(###-09-27&&

314     Q: What sort of side chain reactions prevent beta-pleated sheets from forming

314     A: Interaction between side chain can pull the polypeptide chain out of a secondary structure. Imagine a horizontal beta pleated sheet with a glutamic acid side chain sticking up on one side of the sheet, and now another part of the polypeptide chain oriented above it with an arginine held above the sheet. If the arginine is far enough above it, it could attract the gluatmate carboxyl, thus pulling that residue up out of the sheet. The whole sheet could be weakened by this distortion, and the structure could be resolved by some alternative more stable arrangement, notinvovling secondary structure. ',0,340,1,1),(###-09-24&&

315     Q: I don\'t understand why the hydrogen bonded to the nitrogen on the pentane ring of proline is unable to hydrogen bond.&&

315     A: You have been misled by gazing at the free amino acid structure.  A proline residue peptide bonded withina polypeptide has no hydrogen on \r\nthe alpha nitrogen. It was used during the extraction of water in the formation of the amide bond.  ',NULL,1381,1,1),(###-09-27&&

316     Q: Does the formation of secondary protein structures have to do with the polarity of its side chains? Why do most proteins have extensive regions folded into alpha-helices and beta-pleated sheets

316     A: No. The secondary structures tend to form because of interaction between backbone atoms only. However, side chain interaction could either add to this stabilty or detract from it so as to prevent its formation. For example suppose you had a stretch of polypeptide with glutamates and aspartates at every 4th or 5th position. An alpha helix structure for this stretch would position these negatively charged groups above and below each other, close to one another. One could imagine that the electrical repulsion between these like charges would disallow the formation of such an alpha-helix. One could make an analogous argument about a beta sheet. ',0,341,1,1),(###-09-27&&

317     Q: In a peptide linkage, is the carbonyl double bonded O always on the opposite side of the molecule from the H bonded to the N in the amide bond.&&

317     A: Yes. These two atoms are part of the 6-atom rigid plane. ',0,342,1,1),(###-09-27&&

318     Q: Does glucose switch between alpha and beta constantly? What about from ring to straight chain

318     A: Yes and yes. As long as the anomeric carbon is not tied up in a glycosidic bond. ',0,343,1,1),(###-09-27&&

319     Q: When looking at secondary structure, is rotation stopped about the N-C bond where the C is the central carbon of an amino acid or is it about the N-C bond where C is from the carbonyl and has a double bonded oxygen.&&

319     A: The latter. There is (relatively) free rotation about the N- central C bond. ',0,344,1,1),(###-09-27&&

320     Q: Why does the one C-N \"double bond\" in a polypeptide due to electronegativity cause us to have 6 atoms in the plane, why not just those 2

320     A: Hard to do this without real graphics. For the following explanation consult the handout on polypeptides, the bottom figure showing the planes. If we take one plane, consider the first 4 atoms: CC(O)N. The partial double bond is between the second C and the N. These 4 atoms are in a plane, sort of Y-shaped, with the first C the bottom of the stem of the Y. So that.s one plane with 4 atoms. Now consider the last 4 atoms in the depicted plane: CN(H)C. The partial double bond is between the first C and the N. Again we have a flat Y with the first C at the bottom of the stem of the Y. These two Y\'s have two atoms in common, the C=N, Both Y\'s must lie in the same plane. So all 6 atoms of these two Y\'s are in the same plane. For better pictures consult a biochemistry text (e.g., Lehninger). ',0,345,1,1),(###-09-28&&

321     Q: How do you number the carbons of sugars

321     A: The rule is always to start numbering at the end; Start at the end NEAREST the Carbon with the least hydrogens. So if the Carbonyl is not on the end, you count from the end closest to it. ',0,346,1,1),(###-09-24&&

322     Q: Are prosthetic groups ever bound by covalent bonds

322     A: Occasionally.  If so, then they would not be released after denaturation of the protein, which occurs for prosthetic groups that are bound via weak bonds (most cases).',NULL,1382,1,1),(###-09-24&&

323     Q: Could you explain the answer to problem 2-18 about the ultracentrifugation of denatured proteins

323     A: Since the two concentrations of mercaptoethanol yielded 2 different results, the difference in concentration must be doing something. It must be that the two types of \r\ndisulfide bonds (H-H vs. H-L) are differentially sensitive, i.e., that one type requires a higher concentration of mercaptoethanol to get reduced, perhaps because it is more \r\nburied within the protein structure. If the H-L disulfides were the most senstive, then at the low concentration of mercaproethanol we would get  an H-H and 2 L\'s for bands \r\nof MW 120,000 and 20,000. We do see a tube with two bands, but neither looks really of high MW as they are closer to the top of the tube, so that\'s an argument aginst the \r\n120000 H-H here. A more compelling argument is that if this were true then when we raised the mercaptoethanol to 0.01M we would expect again 2 bands from the \r\ncomplete reduction of all disulfides, yielding bands of 60000 (H) and 20000 (L).  But no other tube has 2 bands, so this scenario seems wrong. If we take the opposite \r\npossibility, that the most sensitive disulfide is the H-H, then we would expect just one band of 20000+60000=80000  (HL) for the low mercatptoethanol and again 2 bands \r\nfor the high mercaptoethanol (60000 aand 20000). This idea fits with what we see, only one tube with 2 bands. So tube 4 must represent situation A.   Two of the 3 remaing \r\nsituations have no mercaptoethaol treatment, so the MW must be 160000 for both of those, and since tube 3 has a much lower sedimetation rate, it is likely to contain the \r\n80000 MW H-L molecule, so tube 3 represent situation B.\r\n<br><br>\r\nThe remaining two situations represent the 160000 molecules with or without urea. Since the denatured molecules is much more extended than the native compact folded \r\nform, it will sediment more slowly than the latter, so tube 2 must represent situation C (+ urea, denatured) and tube 4 represents situation D (native compact 160000 MW \r\nprotein).',NULL,1383,1,1),(###-10-03&&

324     Q: What does the term \"apparent\" mean, as in apparent Vmax or apparent Km

324    A: Apparent means the measured parameter, measured under a particular condition; inhibitor. Thus an enzyme will have an intrinsic Km for a substrate, defined as (k2+k3)/k1. This number, once determenined, can be written down and tabulated in a book. But if you run a Vo vs. S experiment in the presence of a competitive inhibitor, for example, and measure the Km using the graphical method of determining the [S} that yields 1/2 Vmax, you will get a Km that does not agree with the the listing in a book. The Km measured in the presence of a competitive inhibitor will be larger. It is not the real Km as described above but only an apparent Km, the Km that appears when an inhibitor is present. That apparent Km can change, for example, if you add a higher concentration of inhibitor: it will get larger. Analogously, you can get an apparent Vmax in the presence of a non-comptetitive inhibitor. ',0,347,1,1),(###-10-03&&

325    Q: What exactly does it mean when an allosteric inhibitor binds to a regulatory subunit in a reversible manner

325    A: The regulator (let\'s say an inhibitor) can bind the enzyme to form an enzyme-inhibitor complex. This complex can dissociate back to the free enzyme and the free inhibitor. Thus the free and bound forms can be in equilibrium. Much like the enzyme-substrate complex, except there is no subsequent chemical transformation of the regulator. ',0,348,1,1),(###-10-08&&

326    Q: In the equation calculating K(eq)for the hydrolysis of ATP -> ADP {-7 = RT x 2.3logK}, where does the number 2.3 come from

326    A: lnX = 2.3 logX ',0,349,1,1),(###-10-08&&

327    Q: In Step 6 of glycolysis where does the P(i) come from

327    A: The medium. Phosphate is one of the required salts in minimal medium (e.g., K2HPO4) ',0,350,1,1),(###-10-08&&

328    Q: Is the term for the reactions in Step 6 and Step 10 of glycolysis phosphorylation

328    A: Yes, any time a phosphate group is covalently attached to an organic compound it is called a phosphorylation of that compound. ',0,351,1,1),(###-10-08&&

329    Q: How does the drain of products push the unfavorable reactions in glycolysis? And what products are actually draining

329    A: The direction of a chemical reaction will be determined by delta G. Delta G has 2 components, delta Go and RTlnQ. Q is made up of the concentrations of the reactants and products under the conditions you are considering to calculate delta G. Say the reactants are A and B and the products are C and D. And further suppose that the delta Go for A + B =--> C + D is large and positive (unfavorable). Now Q = A*B/C*D (concentrations). Now suppose D is also a reactant for a second chemical trasnsformation (a downstream reaction), say C --> X + Y. If the delta Go for c --> X + Y is very favorable, then the delta G for that reaction will be favorable (i.e., large and negative) even in the presence of a low concentration of C. So the concentration of C will be greatly reduced by the running of the second reaction. This reduction in C concentration will greatly affect the delta G of the first reaction, by making Q very very small (e.g., 10-7) and thus making delta G for A + B --> C + D negative even though delta Go for a+B --> C + D is large and positive (unfavorable). The products tha are

draining are the porducts of the reaction under consideration for the determination of delta G. Note that, importantly, delta G and delta Go are different. ',0,352,1,1),(###-10-09&&

330     Q: Does the fact that a higher Km mean poor enzyme binding and complete ES dissociation mean that the substrate will sit in the enzyme\'s active site, but the enzyme will release it without forming product? If this is true, why or how does increasing the [S] kick the enzyme into gear

330     A: \"Completely\" is an exaggeration here. Poorer binding means that E*S/ES is lower, but there is is always some ES around at any moment. And yes, the enzyme isn\'t holding onto the substrate long enough for it to be changed into product in most bound cases. But increasing S will improve things: high S will produce more ES no matter what the strength of the binding is, since ES = (E * S)/Kd. The more ES there is, the higher the chance of getting some S converted to P (via k3). ',0,353,1,1),(###-10-10&&

331     Q: How much of glycolysis and the Kreb\'s cycle will we be responsible for on the next test? Should I simply learn what occurs in each reaction, or do I need to memorize complete structures and names of molecules in each step

331     A: All of both these pathways are among the topics to be covered. You need not memorize structures or names associated with structures, names and/or structures will be provided if necessary on the exam. But you should be able to recognize what is happening at each step, and know the significance of what is happening at each step (some more significant than others). ',0,354,2,1),(###-10-12&&

332     Q: Why does the electrochemical gradient that builds up outside a FoF1 structure translate into a relase of free energy

332     A: We did not treat this topic quantitatively in lecture. One reason is that we did not introduce the subject of membranes and molecular transport. This topic will be addressed in the second semester. Meanwhile you can read about it in Becker, p. 436-437 for mitochondria, referring to pp. 222-226 in Ch. 8 for background. ',0,355,2,1),(###-10-12&&

333     Q: Why are k1 and k2 faster than k3? Why is there a k2 reaction at all? Why would the ES just dissociate back into E and S without the S having been converted to product? It seems kind of pointless.&&

333     A: One can rationalize why k1 and k2 may be faster than k3:These reactions just involves a collision between two molecules (E asd S) and then their binding together via weak forces. The k3 reaction on the other hand requires the making and breaking of covalent bonds as S is turned into P. Nevertheless k2 is often slower than k3, making the Km a less than perfect indicator of binding ability of the enzyme. As for the reversibility of the E + S --> ES reaction, how could you you prevent it? What sticks together can come apart. All reactions must be considered to be reversible in principle. Now you could design an enzyme with such a strong binding site that once bound, the substrate has a high probability of staying on for a long long time (e.g., Km = 10^-10M). But then perhaps the product would also have a great affinity for the active site and would not come off so readily, thus clogging up the enzyme and preventing its renewed use. ',0,356,1,1),(###-10-12&&

334     Q: If Km = ~k2/k1 how can it have molarity as its units? I thought that it was just a constant

334    A: Formation of ES driven by S and E:<br>\r\n -dS/dt= k1[E][S], k1=(-dS/dt)/[E][S], units = sec^-1*M^-1*M^-1<br><br>\r\nDissociation of ES driven by ES:<br>\r\ndS/dt=k2[ES], k2=(dS/dt)/[ES], units=sec^-1*M^-1<br><br>\r\nProduction of P driven by [ES]:<br>\r\ndP/dt= k3[ES], k3= (dP/dt)/[ES], units = sec^-1*M-^1<br>\r\n(k2 + k3)/k1, units = sec^-1*M^-1/sec^-1*M^-1*M^-1 = 1/M^-1 = M ',0,357,1,1),(###-10-13&&

335    Q: Is it safe to say that the only difference between non-competitive inhibitors and allosteric inhibitors is that allosteric inhibitors bind to a remote site in the enzyme whereas noncompetitive inhibitors bind close to the active site

335    A: A consequence of the fact that allosteric inhibitors biond to a remote site is that they must distort the protein so as to \'telegraph\" their presence to the active site. In contrast, a non-competitive inhibitor need not do so: it could bind to the active site pocket, but not to the part of the active s ite that binds to the substrate. ',0,358,1,1),(###-10-13&&

336    Q: What does a given equilibrium constant tell us

336    A: Keq = [products] at equilibrium/[reactants] at equilibrium, and thus the direction tendency of a given reaction. And also: Keq = e^delta Go/-RT and reactions inside living cells are rarely at equilibrium. ',0,360,1,1),(###-10-13&&

337    Q: In problems 4-6 and 4-17, when do we add the standard free energys, and how are the two questions different on this respect

337    A: Not too different. But you should also be able to manipulate the equations. You often need to see how to arrange two reactions equations so that they simplify when combined (added), i.e., so that some reactants and products are the same on both sides so they cancel out in any consideration of net change. In this switching around, you need to change the sign of delta Go every time you switch. ',0,361,1,1),(###-10-13&&

338    Q: I think I understand the general idea of mass action, but what is it defined as actually

338    A: That the rate of a chemical reactionis propotional to the concentration of the reactants raised to their stoichiometric power. E.g., for aA = bB --> C ; Rate (ignoring back reaction) = k[A]^a[B]^b ',0,362,1,1),(###-10-13&&

339    Q: What does the picture 6.19 in Purves have to do with non-competitive inhibition

339    A: Non-competitive inhibition is defined as inhibition that is not affected by variations in the concentration of substrate. The example I gave in lecture was an inhibitor that prevented the k3 reaction (S -> P) without affecting k1 or k2. Such inhibition could be achieved by blocking an amino acid residue at the active site (but not at the substrate binding site of the active site) or by binding elsewhere, deforming the enzyme so as to inhibit the k3 reaction but not affecting its ability to biond substrate, as in the Purves example. I avoided this example, as it overlaps with a simple description of allosteric inhibition. ',0,364,1,1),(###-10-13&&

340    Q: Noncompetitive and allosteric inhibitors see very similar, but why do noncompetitive inhibitors leave Km unaffected but allosteric inhibitors can affect Km

340     A: Non-competitive inhibitors do not affect Km by definition. ',0,365,1,1),(###-10-13&&

341     Q: What do E and F represent in the energy diagram introducing free energy changes

341     A: The inclusion of a conversion pathway from A+B to E+F and thence to C+D was inserted to emphasize that the free energy change associated with the conversion of A+B to C+D is independent of the route. ',0,366,1,1),(###-10-13&&

342     Q: Delta Go is dependent on direction; is that true for delta G as well

342     A: The dependency goes the other way round: direction is determined by the delta G\'s. Delta Go specifies a tendency to go in a specific direction (i.e., defines the direction under standard conditions). Delta G defines the direction period (i.e., for the conditions under consideration). ',0,367,1,1),(###-10-15&&

343     Q: Why does using the same enzyme to catalyze both the hydrolysis of ATP and the synthesis of G-6-P mean that the energy produced by the ATP hydrolysis is be used to drive the G-6-P sythesis rather than lost as heat

343     A: If the reactions are physically coupled they are no longer two separate reactions but just one reaction: G + ATP <--> G6P + ADP, and the delta Go for that reaction is about -3.4 kcal/mole. ',0,368,2,1),(###-10-15&&

344     Q: In redox reactions, do electrons represent the energy, since energy goes from the reducing agent to the reduced product

344     A: In any oxidation reaction there must also be a reduction. The free energy change could be positive or negative, depending on the relative stabilities of the reactants and products, as written. Thus when electrons are transferred from NADH2 to pyruvate (in fermentation) there is a negative free energy change. But when electrons are transferred from Gal-3-P to NAD, there is also a negative free energy change. So in this example, electrons are flowing either to NAD or away from NADH2, yet free energy is released in both reactions. So yo cannot make a simple general rule about direction of electron flow and free energy without a specific reference to all the reactants and products. ',0,369,2,1),(###-10-15&&

345     Q: Why does step 6 in glycolysis (a phosphorylation) need NAD, but steps 1 and 3 do not, utilizing an ATP instead

345     A: Each reaction in a biochemical pathway involves a small specific change as a reactant is converted into a product. The chemical changes are diverse: some are phosphorylation, some are isomerization, hydration, hydrolyses, etc. Step 6 in glycolysis needs NAD because it involves an oxidation of an aldehyde into an acid. That is the change represesned by this step. Electrons must be removed and so there must be an electron acceptor, and NAD fulfills this role here. As to WHY these changes are orchestrated in this particualr sequence, the answer lies in the evolution of a series of steps that will allow a cell to produce net ATP from ADp + Pi via the breakdown of glucose. Thsi may not be theonly way to doit, but it is an efficient way, and once it got established (presumably billions of years ago), it spread throughout the living world, such that it now remains a universal biochemical pathway. ',0,370,1,1),(###-10-15&&

346    Q: Is O2 necessary for the oxidation of pyruvate to acetic acid in the lead-in step to the Krebs Cycle

346    A: O2 is not involved in this step or any step in the Krebs cycle. NAD and FAD are used as the oxidizing agent for the many oxidations of glucose metabolites that take place. ',0,371,2,1),(###-10-15&&

347    Q: How is Coenzyme A different from a regular enzyme? Why is it written on the Krebs diagram, but no other enzymes are

347    A: Coenzyme A is not an enzyme. It is a CO-enzyme, which is a small molecule co-factor in an enzymatic (enzyme-catalyzed) reaction. It facilitates the reaction, and actually is a reactant in the Krebs Cycle entrance step reaction. Thus it can also be considered a substrate for that enzyme. It is considered a co-factor because it is immediately regenerated, to be able to participate over and over in the reaction it facilitates by its participation. ',0,372,2,1),(###-10-15&&

348    Q: Why do we die when deprived from oxygen long enough if we still have the option of anaerobic respiration

348    A: The small amount of ATP produced by fermentation, while sufficient for the reproduction of single celled microorganisms like E. coli and yeast, is insufficient for the greater energy needs (e.g., constant heart muscle contraction) of larger multicellular organisms. ',0,373,2,1),(###-10-15&&

349    Q: In problem set 5-15C, how does the electron transport chain still function despite that oxidative phosphorlyation is not taking place

349    A: Because if the electron transport chain were not working, there \r\nwould be no way to re-oxidize the NADH2 and FADH2 that is produced in the Krebs cycle, so the reactions would grind to a halt very quickly. Since 1 ATP is continually produced per acetate, NADH2 must be recycling to NAD',0,374,2,1),(###-10-15&&

350    Q: When do we need pyrophosphatase when you are just adding a single nucleotide? Is it true that a high nucleotide concentration drives the reaction forward and thus does not require pyrophosphatase

350    A: Pyrophosphate hydrolysis accompanies the addition of each nucleotide. There is not an excess of nucleotides; provision for such an excess would be very inefficient, given the large amount of product nucleotides (DNA) in each cell (about 10 million per E. coli cell, at 5 million BP/genome). ',0,375,2,1),(###-10-15&&

351    Q: Is allosteric inhibition \"all or nothing\" in comparison to competitive inhibition, where enough substrate will mask an inhibitor\'s effect? What about noncompetitive inhibition

351    A: None of these inhibitions is all-or-nothing when considering a population of molecules (which we almost always should do). At certain intermediate concentrations of inhibitors, you always have partial inhibition overall. ',0,377,1,1),(###-10-15&&

352    Q: I am confused about the various components of a minimal medium, because I thought E. coli could replicate with just glucose.\r\n&&

352     A: Minimal medium contains more than just glucose; go back to the lecture 1 notes or handouts to see. Glucose is the only carbon-containing compound, and the great majority of the thousands of different types of molecules in E. coli (or any living cell) are carbon compounds, so the major problem is how you get the carbon in glucose transformed into thousands of other organic molecules. Some of those organic molecules also contain other atoms than C, H, and O present in glucose. Nitrogen, for example, in amino acids and nucleotides, must come from the NH4Cl in the minimal medium. And P, which must come from the K2PO4 in the minimal medium. E. coli is what it eats, at the level of atoms at least, and almost all of the types of atoms provided in minimal medium will end up being part of our E. coli cells. ',0,378,2,1),(###-10-16&&

353     Q: In Problem 4-5D, why isn\'t a mix of G-6-P, ATP, and hexokinase sufficient to hydrolyze the ATP to ADP

353     A: You forgot about the enzyme and rate issue. For a reaction to give a significant amount of product in a finite amount of time you have to worry about rate as well as yield. The reaction for hydrolysis of G-6-P has a neg. delta G (favorable yield), but if there is no enzyme present, the rate of G produced will be negligible. (At equilibrium there will be plenty of G, but you\'ll never get anywhere close to equil. without a catalyst.) To get a reaction going you need BOTH a favorable delta G AND an enzyme. ',0,379,1,1),(###-10-16&&

354     Q: I had a question regarding the Krebs cycle diagram on page 415 of Becker. The caption notes that the two orignal pyruvate C\'s are highlighted in pink until they are randomized by the symmetry of fumarate. But isn\'t the preceeding step, succinate, symmetrical as well

354     A: You are both right. Yes, succinate is symmetrical, but they are just trying to allow you to compare \"new born\" succinate (that hasn\'t had a chance to turn over) with the previous compounds/intermediates. They want to illustrate how you get from alpha keta glutarate to succinyl CoA to succinate. But once you have free succinate, the label is randomized, which is why the class handout has the labeling symbols in parenthesis for succinate. So by the time you get to malate and OAA, 1/2 the molecules have radioactivity in the top 2 carbons and 1/2 have it in the bottom 2. ',0,380,2,1),(###-10-16&&

355     Q: In problem 6-9D: If I have 2 different DNA molecules (2 strands) with the same %G+C, can I assume they are identical? And conversely, if %G+C different, can I assume no hybridization? What about partial hybridization

355     A: Same G + C does not mean identical. Bases could be in a different order. If % G + C different, will get at most partial hybridization. ',0,381,2,1),(###-10-16&&

356     Q: In problem 6-10A, aren\'t you supposed to consider thymine in the nucteotide form, or as TTP, which can be in other places in the cell

356     A: The problem means that the only macromolecule that is radioactive is DNA. RNA, protein, etc. are not radioactive. We\'re ignoring any small molecules containing T that are floating around, whether TTP, plain T, etc. ',0,382,2,1),(###-10-16&&

357     Q: In problem 6-12B, wouldn\'t you rather go both directions when replicating DNA (i.e. have as many forks as possible?)&&

357     A: This question is not about real DNA. It\'s about an imaginary molecule contructed for study purposes. So take it as a given there is one origin and proceed from there. Most real long linear DNA molecules do indeed have multiple forks with bidirectional replication at each fork. ',0,383,2,1),(###-10-16&&

358     Q: In the body, does over-production of ATP result in fattening (synthesis of fatty acids)

358     A: Yes. If there is excess ATP, it is used to drive synthesis of glycogen, fat, etc, thereby storing the excess energy to be used later. ',0,384,2,1),(###-10-16&&

359     Q: In DNA synthesis, how is the energy of the first breakage of phosphate atoms (reaction 4 on the handout) used? Is it used to bond the nucleotide to the DNA chain or to make up for any energy lost in combining the phosphate atoms when TMP becomes TTP

359     A: The overall energy (standard delta G) of reaction 4 is zero. The energy from breakage of the link between PP and XMP is used to form the phosphodiester bond in the growing chain, as you suggest. The energy \"lost in combining phosphate when TMP becomes TTP\" is provided by breaking down additional ATP. (Reaction 3). ',0,386,2,1),(###-10-17&&

360     Q: Regarding problem 5-13, C-1. When oxidative phosphorlyation is taking place, why can\'t electrons cross the inner membrane toward the matrix? (Based on the assumption that if oxidative phosphorlyation is taking place, then so is glycolysis, and hence the electrons of the reduced NADH2 from glycolysis must cross the inner membrane to be processed.)&&

360     A: The reduced NAD in the cytoplasm often stays there; it doesn\'t have to enter the mito. for ox. phos to occur. (Electrons do have to flow through the et chain for ox phos to work.) Also it wouldn\'t be loose electrons moving -- it would be reduced cmpds. carrying electrons. So choice (2) was not considered as an option. Your answer is ok, given the explanation, but what you really mean is that reducing power might be transported into mito, which is correct. ',0,387,2,1),(###-10-18&&

361     Q: What happens to gas exchange in muscle cells in anaerobic conditions

361     A: Although CO2 is released in the Krebs Cycle, there is no oxygen utiization there. The oxygen is used at the end of the E.T.C. When you talk of \"gas exchange\" you are referring to a gross physiological process of addition and removal of gases in the blood via the lungs. This exhange should not be related to the primary biochemical processes by which these gases are produced (CO2 in the Krebs Cycle) or absorbed (O2 in the E.T.C.). In large organisms such as mammals, there can be a temporary fermentation to lactivic acid under conditions of temoporary oxygen insuffuficiency. The lactate produced in muscle cantravel through the blood to the liver, where it is eventually converted back to pyruvate (now with oxygen present) and then metabolized to CO2 inthe Krebs Cycle. ',0,388,2,1),(###-10-18&&

362     Q: In a reaction A+B-->C+D that is catalyzed by an enzyme, would A and B both be considered substrates of the enzyme

362     A: Yes, since the enzyme has to bind both of them in order to bring them close together and avoid a high energy transition state.',0,389,1,1),(###-10-18&&

363     Q: In the reaction A+B-->C+D, how do we classify which reactants and products are the substrates

363     A: Depends on how you are writing the reaction. Reactants, on the left, are considered substrates in that context, and the compounds on the right are the products, by definition. ',0,391,1,1),(###-10-18&&

364     Q: Is the rate of an enzyme catalyzed reaction proportional to [ES], [E], or [S]

364     A: The rate of product formation (the overall reaction) is proportional to [ES]. However, under most (but not all) circumstances, enzyme (E) is limiting, and so the rate of ES formation will be proportional to [E]. That is, if you double the enzyme, you double the amount of ES formed in the quickly achieved steady state and the rate of product formation (V) then doubles. ',0,392,1,1),(###-10-18&&

365     Q: In addition to inhibiting the first direct step of a pathway, does a feedback inhibitor always inhibit the last step as well

365     A: The fact that a product is produced at the active site of the enzyme means that it must be bound there momentarily before being released. Such binding does not necessarily imply inhibition, as it may be much weaker than substrate binding. Addition of high amounts of product will reverse a reaction by thermodynamic (energy) considerations, but this does not constitute enzyme inhibition, just reversal of the reaction direction. ',0,393,2,1),(###-10-18&&

366     Q: If we say that the top and bottom strand of DNA are written according to convention, does this mean that the top strand runs 5\'---->3\' and the bottom strand runs 3\'-----> 5\'

366     A: Usually it says that ONE of the strands is written in the usual convention, namely, 5\' to 3\' left to right. (The other one is always antiparallel). If it says the molecule or two strands are written in the usual, etc., it means top strand is standard (5\' to 3\') and bottom is antiparallel. ',0,394,2,1),(###-10-18&&

367     Q: What is malonic acid

367     A: Malonic acid is like succinic except it has one CH2 in the middle, not two. It acts as a competitive inhibitor of Succinic DH. This is usually brought up in class, but may have been overlooked this year. ',0,395,2,1),(###-10-18&&

368     Q: For problem 3-7D, shouldn\'t the solution read \" .... If the mutant CANNOT grow on glucose + C, then B --> is blocked ...\" In the manual, the solution that is given says: \"if the mutant CAN grow on glucose + C, then B --> C is blocked....\"&&

368     A: I think the answer is right, but it may not be clear why. We already know that one of two steps is blocked, B --> C or C --> D. Given that, if we rule out one step, it must be the other that is blocked. If mutant can grow on glucose + C, then we know C --> D is ok, and by elimination, B --> C is blocked. If mutant cannot grow on glucose + C, we know C --> D is blocked, so B --> C is okay. It is true we have not tested B --> C directly by showing mutant can or cannot grow on glucose + B -- but either way, mutant won\'t be able to grow on glucose + B as block is after B. ',0,396,1,1),(###-10-18&&

369    Q: Regarding problem 5-1B. In regular anaerobic glycolosis, what drives the oxidation of NADH2, and why can\'t that mechanism be used when starting with lactate (which yeast can use to oxidize NADH2 only in the presence of O2)

369    A: Yeast can grow on lactate only if they can break it down first to pyruvate and then use the Krebs cycle to breakdown the pyruvate. That means they will generate lots of reduced NAD, which will require O2. When bacteria do glycolosis anaerobically, they generate 2 reduced NAD per glucose, and that is reoxidized by conversion of pyruvate to lactate. You can\'t do that here -- you can\'t use pyruvate to soak up the reducing power because you need to break down the pyruvate to get energy. You are skipping the whole glycolysis part of metabolism when you start with lactate.',0,397,2,1),(###-10-18&&

370    Q: Regarding problem 5-13C1: Since the electrons are transported through the respiratory complex and then given up to 1/2 O2 in the formation of H20, why can\'t electrons cross the inner mitochondrial membrane toward the matrix

370    A: The basic movement here is within the membrane. It is true the electrons eventually end up in water on the matrix side, but that is where most of them started out -- in the matrix, from reduced NAD and FAD. The electrons are not transported across the membrane. (Some reduced NAD or equivalents can come in from the cytoplasm and eventually end up in the matrix, but that is not required.) ',0,398,2,1),(###-10-18&&

371    Q: Is it possible that two proteins can contain the same amino acids, though in different sequences, but give the same fingerprint

371    A: A protein fingerprint does not result in free amino acids, but rather in peptides. These peptides are sub-peptides of the protein polypeptide, having been derived from it by cleavage at points where specific amino acid residues reside in the polypeptide chain. For instance, if the polypeptide were a sentence and the amino acids the 26 letters, and trypsin cleaved after K and R, then: \r\n\"The quick brown fox jumped over the lazy dogs\" would produce the peptides: \r\n1) The quick \r\n2) br\r\n3) own fox jumped over\r\n4) the lazy dogs\r\nwhile the polypeptide \"Too many cooks spoil the broth\" would give the peptides:\r\n1) Too many cook\r\n2) s spoil the br\r\n3) oth\r\nEach peptide will have a characteristic mobility in the combination of paper electrophoresis and chromatography, so that it produces a spot at a given X-Y coordinate. What\'s the chance that any two different 500 letter sentences would give rise to the exact same combination of words? Just about zero. That\'s why the pattern is called a fingerprint. If it gives the same fingerprint, it must be the same protein. ',0,399,2,1),(###-10-18&&

372    Q: Non-competetive inhibition can\'t be drowned out by adding more S but can be by adding a lot of E, correct

372    A: More S does not help restore acitivtyot an enzyme inhibited by a non-competitive inhibitor. Addng more E always helps (increases the rate of the reaction), no matter what the inhibiton, as long as the enzyme is not saturated with inhibitor. ',0,400,1,1),(###-10-18&&

373    Q: If we start with 1 M ATP with a Keq of 10^5 in the reaction ATP-> ADP+ Pi, how do we get 10^-5 M ATP

373     A: Keq = [ADP][Pi]/[ATP] at equilibrium = ~100,000<br><br>\r\nAlso: ATP + ADP = 1 (M); and [Pi] = [ADP]<br><br> \r\nSo: [ADP][ADP]/[ATP]= 100,000<br> \r\n[1-ATP][[1-ATP]/[ATP] = 100,000 <br><br>\r\nEstimate: the above relationship can be true only if ATP << 1.<br><br>\r\nSo: 1 x 1 /[ATP] ~= 100,000<br> \r\nAnd [ATP] ~= 10^-5, at equilibrium. ',0,401,2,1),(###-10-18&&

374     Q: When listing the receptors in the ETC, lecture 9 says the 1st acceptor is a Fe+++ complex, but on the handout (views 1,2), we see that CoQ is the first acceptor and cytB, which has the Fe+++, is the 2nd acceptor.&&

374     A: Actually the first acceptor is FMN, related to FAD. Then Fe++ in an Fe/S protein and then CoQ. The handouts have been simplified in that many intermediate steps have been omitted. See Becker Fig. 14-17 for a more complete (and accurate) story. ',0,402,2,1),(###-10-18&&

375     Q: Is it true that when delta Go is positive, then the reaction goes to the left

375     A: Tends to go left. The actual direction under any particular set of circumstances will can be influenced by the concentrations. ',0,404,1,1),(###-10-18&&

376     Q: Is it true that when delta Go is positive, Keq MUST be less than 1 as you will see much more reactant than product

376     A: Yes, less than one; and yes, at equilibrium. ',0,405,1,1),(###-10-19&&

377     Q: The answer to problem 6-5 refers to nonconservative replication; is this congruous with semi-conservative or dispersive replication? Also, is it typical for the DNA template to degrade after replication in conservative replication

377     A: This is a different model. No, it is never typical for the template to degrade after replication in conservative replication. If the answer were semi-conservative or dispersive I would expect an intermediate line (assuming the DNA was not denatured - if it was denatured, then there would be two). Please explain part B also. How would it be unchanged? I would think that we would have more light strands. This is a totally ridiculous, made up situtation to test your understanding. It is unheard of for the template to degrade or this type of replication to occur. In part B, and for every cycle of replication (in this artifical situation) you expect all new strands. If they are made of totally new nucleotides (newly made from materials in the medium), DNA will be all light. If the new strands are made of a mix of new (light) nucleotides and recycled old (heavy) nucleotides, then new DNA molecules will have intermediate density. ',0,407,2,1),(###-10-19&&

378     Q: In problem 6-12D, why don\'t we need pyrophosphatase and why is the delta Go of synthesis about zero

378     A: Look at the reactions on handout 11-2 or in lecture 12. Synthesis of DNA from dXTP\'s without pyrophosphatase = reaction 4 = delta Go of zero. You need the overall delta G (not delta G zero) of reaction 4 to be negative. If you remove the product (pyrophosphate) OR increase concentration of substrate (dXTP\'s) that will do the trick. In the cell the first strategy is used; in the experiment described in the problem, the 2nd strategy is employed. ',0,408,2,1),(###-10-19&&

379     Q: Does hydrolysis break base-base bonds in addition to sugar-phosphate bonds? And does it break both sugar-phosphate bonds (3 and 5), totally tearing the phosphate apart from the sugar

379     A: Hydrolysis as it is usually done breaks polymers down into their monomers. DNA --> nucleotides, starch --> glucose, etc. If the monomer is itself made of pieces, as nucleotides are, more extreme conditions (different pH, temp. etc. and esp. different enzymes) can break the compound monomers down into their parts, for example nucleotides --> sugars, bases, and phosphates. The base-base bonds in DNA are weak H bonds, and conditions of hydrolysis break these too, but not by \"hydrolysis\" -- no water is added across these bonds. The usual treatments hydrolyze DNA and RNA into 5\' mononucleotides, but some conditions will give you 3\' mononucleotides, as explained in the problem book. ',0,409,2,1),(###-10-22&&

380     Q: If all NAD processed by the ETC winds up in the matrix, how does glycolysis, which takes place in the cytoplasm, get the NAD it needs for step 6

380     A: NADH2 formed in glycolysis does not migrate into the matrix. It gives up its electrons to a cytoplasmic carrier which then transfers them to the mitochodria (at the 2 ATP yield level, like FADH2). The NAD regenerated is still in the cytoplasm, so can be used for more glycolysis. See Becker Ch.14 p. 443 for details of this \"glycerol phosphate shuttle.\" ',0,410,2,1),(###-10-27&&

381     Q: How well do we need to understand the genetic engineering involved in making antisense mRNA

381     A: You should understand what \"transc. etc.\" means. It means equivalent to starting at the other end of the gene -- which means transcribing the other strand. The same thing can be achieved by either leaving the gene alone and moving the promotor OR by leaving the promotor in place and inverting the gene. If this is still not clear (it really needs a diagram) ask Dr. M or a TA. ',0,411,3,1),(###-10-27&&

382     Q: In translation, does the fact that the enzymes involved recognize a unique feature of the tRNA which may or may not be the anticodon mean that the wrong tRNA can bind to the enzyme

382     A: Absolutely not. The point is that the enzyme does not always use the anticodon to recognize a particular tRNA. It may use a different feature of the molecule. The enzyme for loading, say, his-tRNA always binds the tRNA for his. But how does the enzyme \"know\" it has the right tRNA? Because some area of the enzyme binds to/matches some site on the tRNA. The site on the tRNA that is unique to his tRNA and binds to the enzyme does not have to be the anticodon. ',0,412,3,1),(###-10-30&&

383     Q: Regarding problem 7-2: Would the reactive OH on C2 of every ribose be considered a \"free hydroxl?\" If this is the case, wouldn\'t there be a free hydroxyl on every rXMP with two free hydroxls on the right end

383     A: This question is asking \"what is special about the ends?\" It is true that every ribose has an unattached 2\'hydroxyl. But that OH is not used to grow chains. In this context, we are concerned with the OH\'s involved in phosphodiester linkages, the ones that can be considered \"fasteners\" or \"hooks\" to lengthen a chain. So we are ignoring the 2\' OH\'s and considering only 3\' and 5\' OH\'s. This is supposed to be clear from the context. To put it another way, each ending nucleotide (on 3\' or 5\' end) has a ribose with a 2\' OH that is unhooked. But neither of these hydroxyls is involved at all in building the chain, so these are not considered \"ends.\" ',0,414,2,1),(###-11-02&&

384     Q: In problem 6-16 (E), doesn\'t a phosphate to sugar ratio of 1 (not counting the 5\' end) mean that the 5\' end is a diphosphate or triphosphate?\r\n&&

384     A: Newly made RNA has a triphophate on the 5\' end. (Think of the way the chain grows.) Eukaryotic mRNA is capped, and RNA\'s in general can be modified, but the very first nucleotide on the 5\' end should have retained all 3 phosphates, barring any modifications. Since DNA synthesis in vivo starts with RNA primers, and the primer is removed, the first deoxy nucleotide remaining in the DNA chain (after the primer is removed by exonuclease) should have one phosphate group left. ',0,415,3,1),(###-11-02&&

385     Q: How does tRNA know whether the AUG on the mRNA is a start codon? Would it ever start translation in the middle

385     A: 5\' AUG........AUG....... 3\' (codes for 1 protein) \r\n<br><br>\r\nIn order for translation to start, the parts of the ribosome and the special initiator tRNA have to bind to the mRNA. This requires special IF\'s (initiator proteins) and usually a special sequence on the mRNA near the start AUG. The binding of the IF\'s and/or ribosome to the special sequence specifies which AUG will be used. The internal AUG\'s lack the necessary signals for binding of ribosomes, etc. The details are different in eukaryotes and prokaryotes and are described in detail in Becker pp. 676-679. ',0,416,3,1),(###-11-11&&

386     Q: It seems like recombinant DNA and cloning can only happen if there are sticky ends on the fragments. Can\'t sticky ends only occur if the DNA has a palindromic sequence

386     A: 1- It is possible to ligate two pieces of DNA that have blunt ends, if high concentrations of DNA and enzyme are used; and 2 - restriction enzymes that recognize non-palindromic sequences usually make a staggered cut of the 2 DNA strands, often at points a few nucleotides away from the recognition site. For examples, look at the handout of the restriction map of the human interferon gene. ',0,417,3,1),(###-11-12&&

387     Q: Is plasmid DNA single-stranded

387     A: No. We draw a single line to mean one, double stranded, DNA. ',0,418,3,1),(###-11-12&&

388     Q: What kinds of probes are acceptable to use in Northern and Southern blotting

388     A: Either RNA or DNA can be used as a probe, but DNA is usually used because it is more stable (less easily degraded). ',0,419,3,1),(###-11-12&&

389     Q: What the role of the poly-A tail if not in signalling the end of transcription or translation

389     A: The signal to add poly A is also the signal to end transcription, either directly or indirectly. (It\'s not the poly A itself that is critical). ',0,420,3,1),(###-11-12&&

390     Q: When we see \"mRNA\" without any other specifications about the molecule, should this be interpreted as the FINAL mRNA transcript with exons (but no introns),  untranslated regions on the ends, and with a 5\'methylated guanine cap and a poly-A tail

390     A: Yes. The transcript containg introns or without poly A or cap is not considered a mRNA. It is a pre-mRNA. ',0,421,3,1),(###-11-13&&

391     Q: Does RNA processing happen to all eukaryotic RNA\'s or just mRNA

391     A: It happens to all, but details of how may be different. ',0,422,3,1),(###-11-13&&

392     Q: What is the relationship between editing and the fact that DNA polymerase cannot start replication

392     A: It has to do with the binding site of DNA polymerase. It must hold a nucleotide attached to a chain and the incoming nucleotide. (Otherwise there would be nothing to edit.) The enzyme checks the match between the new base and the template for both nucleotides. The binding site of RNA polymerase is a different shape and does not check that a chain is sticking onto one of the nucleotides. It only checks the match between the base to be added and the template. ',0,423,3,1),(###-11-13&&

393     Q: In problem 12-2 (C), how can you just convert an inducible operon into a repressible one

393     A: This problem describes an experimental lab situation, not a normal one. The investigator has engineered (or discovered) a rearrangement that hooks the regulator sequences of one operon up to the structural genes of a different one. This is not the normal state of affairs. It is as if the switch on the microwave were rewired to the toaster so that when you turn on the switch on the microwave, the toaster goes on. The operator and repressor that are specific to the lac operon (and bind lac, not his) are now hooked up to (and control) the structural genes of the his operon. ',0,424,3,1),(###-11-13&&

394     Q: How many high energy bonds does it take to shift a tRNA over one codon and likewise to add the chain to the amino acid

394     A: 5 total. It takes 2 ATP\'s to load up a tRNA but it takes additional ATP\'s to move the ribosome relative to the mRNA. ',0,425,3,1),(###-11-13&&

395     Q: Is the enzyme that couples the hydrolysis of ATP to AMP and tRNA loading aminoacyl synthetase? Do there need to be 20 versions of this enzyme

395     A: This is correct. There are 20 different aminoacyl synthetases.',0,426,3,1),(###-11-13&&

396     Q: What is the precise role of pyrophosphatase

396     A: The enzyme catalyzes destruction of the product (PPi) so the reaction is now irreversible. Destruction of P changes [S]/[P]. ',0,427,3,1),(###-11-13&&

397     Q: When a hybrid plasmid with a human gene is \"transformed\" into E. coli, is the human gene incorporated into the E. coli chromosome or just into the cell so that it remains on the plasmid

397     A: The point of using a plasmid is to keep the added gene separate, on a plasmid, so the gene (being on the plasmid) can easily be retrieved. (The plasmid is much smaller than the bacterial chromosome, so they are easily separable.) ',0,429,3,1),(###-11-13&&

398     Q: Is there a valid comparison to be made between cloning and PCR? Don\'t they both amplify genes

398    A: Cloning and PCR do overlap in their uses. You can\'t do PCR unless you know enough to make a primer. Yes, cloning gets the bacteria to work for you, making a protein. ',0,430,3,1),(###-11-13&&

399    Q: When preparing recombinant plasmids, isn\'t  it difficult to get the desired fragment/plasmid combination, since any plasmid or fragment can splice to any other?\r\n&&

399    A: You set up the concentrations so most plasmids get 1 insert or none. You discard those with none easily -- no drug resistance. ',0,431,3,1),(###-11-13&&

400    Q: Do plasmids have single promotors/origins

400    A: Single origin. Number of promotors depends on plasmid. ',0,432,3,1),(###-11-13&&

401    Q: Should we assume that a probe always hybridizes completely to sample DNA?\r\n&&

401    A: It depends how you set it up how much mismatching will be accepted. ',0,433,3,1),(###-11-13&&

402    Q: About problem 13-3 (A): Should the answer for the val in the sickle cell sequence be GTX rather than GTG

402    A: You assume the mutation causing sickle cell is a single base change. ',0,434,3,1),(###-11-14&&

403    Q: Why is cloning mRNA sequences a particularly good way of cloning a gene?\r\n&&

403    A: A cell that devotes 1% of its protein sythesis to a particular protein will in general have 1% of its mRNA as coding for that protein. So among all mRNAs in a preparation, 1 out of 100 will be the mRNA for that protein. In contrast, the gene for that protein will be 1 out of about 70,000. So the mRNA is a richer source of the nucleic acid that can specify the protein than is the total genomic DNA. Another advantage of the cDNA library is the small size of the cDNA version of the gene, enabling its isolation in one piece, as opposed to the real genomic gene, which may have to be cloned in pieces (e.g., could be 100 kb) A third advantge is that it can be expressed in E. coli if hooked up to an E. coli promoter, as it does not have to be spliced. ',0,435,3,1),(###-11-14&&

404    Q: Why can we rely on hybridization if probes don\'t need to be perfectly complimentary to hybridize

404    A: You can do hybridization under a wide range of conditions. Under certain conditions (refered to as \"stringent\") only perfect hybrids will hold together. Under other conditions a less than perfect match will hold up. You can manipulate the conditions to set the % match required (within reason). ',0,436,3,1),(###-11-14&&

405    Q: Regarding problem 12-1 (C). Don\'t we need to assume pathway also utilizes feedback inhibition? Do pathways usually involve both feedback inhibition AND repression/regulation, or only if so mentioned in problem

405    A: Problem doesn\'t assume feedback inhibition. It assumes addition of phosphate will stop production of new enzymes but not affect action of old ones. In real life you can have fbi, repression or

both -- it depends. In an exam question we will do our best to make it clear (from results or explanation, as the case may be). ',0,437,3,1),(###-11-15&&

406     Q: If I understand capping correctly, the cap is inserted approximately 35 nucleotides after the start of transcription. So what happens to those nucleotides before the cap

406     A: The cap is attached to the 5\' end of the transcript, NOT at a position 35 nucleotides downstream. The 35 nucleotide figure is a reference to a delay time, not a position. The cap is added to the very first nucleotide of the transcript at a time when ~35 nucleotides have already been polymerized. ',0,438,3,1),(###-11-15&&

407     Q: What happens to the cap and the poly-A tail during translation

407     A: The cap and polyA are not translated. Translation requires an ATG to get started,and the cap is at the very 5\' end of the mRNA, upstream of any AUG. And translation is terminated by a UAA, UAG or UGA triplet before you get to the polyA tail. ',0,439,3,1),(###-11-15&&

408     Q: How does inverting a sequence relative to the promotor cause the complement to be made if the sequence isn\'t symmetrical? Won\'t it actually just do the same sequence in the backwards order? (i.e. PROMOTOR-ACGTTAACG gives 5\' UGCAAUUGC 3\'; inverting relative to promotor is the same as, according to the lecture, moving the P to the other side of the gene giving PROMOTOR-GCAATTGCA and a transcript of 5\' CGUUAACGU 3\', which is just the reverse, not the complement, of the above sequence.)&&

408     A: Promoter-ACGTTAACG, written 5\' to 3\' represents the top strand. It is the bottom strand that is the template for trasncription, since the (antiparallel) bottom strand is 3\' to 5\' and the RNA is synthesized 5\' to 3\'. So the top strand represents the transcript, and is called the \"sense\" strand for that reason. So the transcript in your first example is 5\' ACGTTAACG 3\'. Now if you reverse the double stranded gene sequence relative to the promoter, you get promoter- CGTTAAGCA as the \r\np strand, 5\' to 3\': <br>\r\nprom-5\'ACGTTAACG 3\' <br>\r\nprom-3\'TGCAATTGC5\' goes to <br>\r\nprom-5\'CGTTAAGCA 3\' <br>\r\nprom-3\'GCAATTGCA3\' ',0,440,3,1),(###-11-15&&

409     Q: In lecture 13, you write delta Go is negative for reactions 1+2+3. Isn\'t it actually delta G that\'s negative, as it depends on the product being taken away by reaction 3 to push reaction 2 forward

409     A: Actually, the delta Go is negative because we are explicitly figuring in the pyrophosphatase reaction as part of the overall path we are considering. This may be a semantic problem. ',0,441,3,1),(###-11-15&&

410     Q: I don\'t understand how to tell the number of rRNA\'s in a ribosome.&&

410     A: An E. coli ribosome has a 23S, a 16S and a 5S species. That\'s what has evolved and there\'s no variation, or \"way to tell.\" Eukaryotic ribosomes have 4 rRNAs. See Becker p.557 for a table, also indicating which rRNA is associated with which ribsommal subunit. ',0,442,3,1),(###-11-15&&

411     Q: What\'s a somatic cell

411     A: Any cell in a multicellular organism that is not a germ cell. The germ cells, are used to produce a new multicellualr organism, and consist of cells that give rise to sperm and eggs, the gametes. ',0,443,3,1),(###-11-15&&

412     Q: When a plasmid and circular DNA combine and then come apart, can the plasmid sometimes end up with chromosomal DNA

412     A: Yes, this disassociation isn\'t always exact, and the plasmid can end up with parts of the chromosomal DNA.',0,444,3,1),(###-11-15&&

413     Q: What is the difference between transformation and conjugation

413     A: For conjugation you need contact between two living cells, and DNA is passed over the connecting bridge. Also the cells must be the right strains -- donor must be F+ or Hfr and recipient F-. For transformation, one cell dies (or is killed by the investigator) and releases its DNA. That DNA is then taken up by a living cell. In problem 11-6, for example, (a) involves two living strains in contact and (b) involves DNA from one added to the other. So (b) must be transformation. (a) is probably conjugation, unless cells are dying and releasing DNA for other cells to pick up. To be absolutely sure, you would have to do some experimental controls such as adding DNase to see if that would stop transfer. (DNA passed by conjugation would be protected and not degraded by the enzyme.) ',0,446,3,1),(###-11-15&&

414     Q: Regarding problem 12R-2, part E-2. How can the original haploid mutant 1 be F-. I thought all \"parents\" had to be either F+ or Hfr.&&

414     A: The recipient must be F-. DONOR must be F+ or Hfr. ',0,447,3,1),(###-11-16&&

415     Q: In problem 7-17, why does the met-tRNA hybridize to one or both of the DNA strands if the anticodon has U rather than T

415     A: The anticodon is not the point. The entire length of the tRNA hybridizes to the DNA strand it was transcribed from. Any RNA can hybridize to the complementary DNA strand. ',0,449,3,1),(###-11-16&&

416     Q: What does \"reading frame\" refer to

416     A: Reading frame refers to where we start, but where we begin counting depends on the set up. If RNA is say, AAAUGCCUAAG, you can read it AAA-UGC-CUA .... or AAU, GCC, UAA... or AUG, CCU, AAG. This would be the 3 possible reading frames. Which is the first is arbitrary. ',0,450,3,1),(###-11-16&&

417     Q: In eukaryotes, is the cap considered part of the 1st exon and the poly-A tail considered part of the last. If so, are they part of the 5\' and 3\' UTR\'s respectively

417     A: Yes, formally. But they are usually ignored when discussing particualr UTRs since they are they same for most mRNAs. ',0,451,3,1),(###-11-16&&

418     Q: Are sticky ends always palindromes

418     A: No. Some restriction enzymes produce a staggered cut in a non-palindromic region. See handout on the interferon gene restriction map for examples. ',0,452,3,1),(###-11-16&&

419    Q: Are the 5\' and 3\' UTR\'s coded for by DNA

419    A: Yes. ',0,453,3,1),(###-11-16&&

420    Q: Where are UTRs located on mRNA transcripts

420    A: Only on the outsides of whole mRNA transcripts. They are NOT located on the outsides of individual genes.',0,454,3,1),(###-11-16&&

421    Q: What\'s a terminator? Does it show up in the transcript and if so, is it the end of the 3\' UTR

421    A: It is not well understood (hardly at all) in eukaryotic cells. In bacteria, it is partly in the transcript, and includes double stranded stem-loop structures. There is more than one way to terminate bacterial transcription. You are not responsible for termination mechanisms; they were not discussed in lecture. On the other hand, you are responsible for the mechansim of 3\' end formation in euklaryotic transcripots (via cleavage and polyadenylation.) ',0,455,3,1),(###-11-16&&

422    Q: Generally, there are no operators in eukaryotes, right

422    A: There may be sites at which repressors sit, but they are not called operators. ',0,457,3,1),(###-11-16&&

423    Q: Within an operon and also within a mature eukaryotic mRNA, are there several start and stopping points? (For example, several initiator methionine points and several stop codons, thus giving several genes?)&&

423    A: True in prokayotes, where polycistronic messengers are common. Not true in higher eukaryotic organisms such as mammals, where virtually all messengers are monocistronic. ',0,459,3,1),(###-11-16&&

424    Q: Does circular DNA make circular RNA

424    A: No. Think of a yo-yo string. ',0,460,3,1),(###-11-16&&

425    Q: If there is a frameshift mutation in an intron, is the rest of transcription garbled? Or can the spliceosome still recognize the consensus sequence and cut the 3\' end at the appropriate site

425    A: An insertion or deletion of a single base within an intron will not affect translation. The exception is a base change in or near the consensus sequence, which could affect splicing, and thus affect the reading frame indirectly. ',0,461,3,1),(###-11-20&&

426    Q: If complementation fails, then the mutation has to be on the same cistron (but not necessarily in same place on the cistron). On the other hand, if restoring function fails via recombination, then what does it tell us

426    A: If recombination fails, then (if the enzymes were present) the 2 mutations were overlapping. ',0,462,4,1),(###-11-20&&

427    Q: It sounds REALLY unlikely that recombination \"fixing\" a mutation by crossing over just before and just after a mutation so as not to disrupt any other genes should take place. If the DNA is recombining enough that this beneficial double recombination occurs at just the right places, wouldn\'t many other disruptive crossings over occurring too? Is this mutating hazardous to perpetuation of the initial genotype

427    A: Crossing over can occur just about anywhere, and it generally does not mess things up as equivalent sections are exchanged. If you have exchange between a fragment and a chromosome, you do need 2 crossover events in fairly defined places, if you want to replace a mutant section, and it may be quite rare. However we are talking about lab situations, where the experiment is set up to allow only the recombinants to grow. So it doesn\'t matter if they are rare. You can still recover them, if there are any, as all the other millions of bacteria die and only the \"fixed\" ones survive. ',0,463,4,1),(###-01-20&&

428    Q: How do stabilizers work in keeping polymers of actin/tubulin- microfilaments/microtubules intact

428    A: They bind to the polymer and prevent monomers from coming off. For more details, see Becker or any cell bio book.(Not that I\'m not interested, but we have to stop somewhere.)',NULL,1455,1,2),(###-11-28&&

429    Q: On problem 13-12 (B), why do bands have to be more than 3000 bases? It says there are 4 bands that hybridize to the cDNA and, \r\nalthough these probably have some of the intron on them, why does all the intron necessarily have to be on these fragments

429    A: It doesn\'t say all the introns are included. It just says all the exons (3000 bases) and some extra is included.',NULL,1421,3,1),(###-11-28&&

430    Q: In problem 8-6 (A), what is a good way of figuring out that S is 6 hours long from the fact that the first cells pass the end of mitosis at 6 hours and the last at 12

430    A: Think of the cells as marching in step one behind the other, and all the ones that were in S at the time of the label have red shirts. You are at the viewing stand (mitosis) watching the shirts go by. The length of time it takes the red shirts to pass is the length of mitosis.',NULL,1422,3,1),(###-12-01&&

431    Q: Does sex-linked imply that the trait is recessive

431    A: Sex linked means \"gene is on the X.\" Either allele of the gene (normal or otherwise) can be dominant. It is true that most (abnormal) sex (X) linked traits that we have discussed are recessive. I think there are more well known recessive abnormal traits than dominant ones, and I think that is because most mutations cause a lack of function (not a gain or change), and most lack of function mutations are recessive. This leads to your question about the 2nd pedigree on handout 20B - is the second pedigree a \"sex-linked\" trait? We decided in section that it was not. However, if sex-linked is anything on the \"X\" including dominant characteristics, this could be sex-linked, autsomal recessive, or autosomal dominant. We decided it was not sex-linked, probably autosomal, but did not have enough info to know if it was dominant or recessive. Were we right? I promised the section that I would ask you to post some form of an answer to this. Trait in question can be autosomal rec. (if condition is common, so it is reasonable for I-1 to be a carrier). It can be autosomal dominant or sex-

linked dominant. It can\'t be X-linked recessive (or III-2 would have the condition). ',0,466,4,1),(###-02-06&&

432    Q: In problem 2-4 (C), could the reason for \r\nthe different graph result be that valine is nonpolar and can therefore diffuse through the membrane, and that becayse lysine and aspartic acid are charged, they can diffuse through the membrane via a channel

432    A: No. Threonine is similar enough to serine that they could use the same carrier. Other two are quite different (in R groups). There is no sign of a channel protein here -- only protein is protein P. Also only small ions like Na, K etc. usually go through \"ion\" channels. But in any case, there\'s no second protein to build a channel out of.',NULL,1473,1,2),(###-12-03&&

433    Q: Regarding lectures 20 and 21. Wouldn\'t black show up in a heterozygous O/Bl cat since there is still some enzyme making black pigment (for a cat with an overall black coat)

433    A: Both black AND orange show up in a heterozygous female cat. However, the overall cat is not a mixed color or light black, but patchy, with areas of orange and areas of black. That is because of two factors. First of all, it reflects the way cats are built, so that all the cells in a particular patch of skin are descended from the same cell and have the same X\'s on and off. Also the black pigment that is made is localized in the cells that make it. So \"blackness\" doesn\'t spread over the whole cat. (That is what I wanted to emphasize in the notes -- that even though some cells make black pigment, other cells do not, and those cells that do not make black pigment appear orange.) It really makes no sense to call either black or orange dominant (or recessive) -- the orange allele \"does its own thing\" in some cells and the black allele is active in others. Many mutations (like the one that causes hemophilia) affect phenotype in a different way. In a heterozygous female (for the mutation that causes hemophilia), some cells make active clotting factor, and some do not, just as some cells in the heterozygous cat make black pigment and some do not. The cells that make clotting factor do not retain the protein but secrete it into the blood. So the person, overall, has clotting factor, and the effects of the mutant allele are covered up. You can\'t \"see\" patches of cells or areas of the body that make no clotting factor. So in this case, it makes sense to call the mutant allele recessive. ',0,467,4,1),(###-02-06&&

434    Q: In problem 1-14 (C), if only the extracellular domain of the connexin is visible in the freeze-fracture, isn\'t the intracellular/transmembrane domain visible too

434    A: You can\'t see the cytoplasmic domain -- that\'s on the other side of the bread. You can\'t usually see the transmembrane domain -- that\'s still usually buried in the bilayer/bread. Exactly what \r\nyou see depends on how you do the freeze fracture and how you take the picture. ',NULL,1474,1,2),(###-12-04&&

435    Q: In problem 9-10 (B), how can two tall parents have many short children if all the males are tall and 1/2 of the females are tall

435    A: If they have 100\'s of offspring there will be plenty of short ones. ',0,468,4,1),(###-12-04&&

436    Q: Problem 9-13 (A) asks what the chance is of the male being a carrier. If SCD is an autosomal disease, does \"carrier\" refer to heterozygous

436    A: Carrier means heterozygous, whether disease is sex linked or autosomal. ',0,469,4,1),(###-12-04&&

437     Q: When is a cell considered two? Is it during anaphase or telophase or after cytokinesis is completed

437     A: Until cytokinesis is finished, it is still one cell. ',0,470,4,1),(###-02-06&&

438     Q: Receptor proteins are integral proteins, but yet they could move around \r\nwithin the plane of the membrane to slide over the areas with clathrin. Aren\'t integral proteins supposed to be anchored? Also, is clathrin all around the membrane, or just at specific parts

438     A: Not all transmembrane proteins are anchored. Clathrin usually gathers in limited areas; it\'s not spread evenly all around.',NULL,1475,1,2),(###-12-04&&

439     Q: In problem 8-4, when they say a diploid cell has 4 chromosomes, how do we know they don\'t mean 4 originals plus their homologs (for a total of 8 DNA molecules)

439     A: 4 means 4. A pair of homologs = 2 chromosomes, not one. 4 means 4 chromosomes, not 4 pairs. I think you are confusing pairs of sister chromatids with pairs of homologs. When it says \"4 chromosomes\" it could be 4 single chromosomes (before S) or 4 double chromosomes = 8 DNA molecules (after S). When we say chromosome #21, we mean pair but we don\'t say it so that is where my confusion lies. So when a question says chromosome should we assume chromosome pair as in \"chromosome #21\" or should we consider chromosome to be one of the homologs as in question 8-4? Chromosome does not mean pair. When we say \"chromosome 21\" we are refering to a type of chromosome, and there may be one or more of them. ',0,471,4,1),(###-12-04&&

440     Q: In problem 8-9, why is nondisjunction at first division more likely than at second divion

440     A: It isn\'t. But nondisjunction at first division produces 4 abnormal gametes (per ND) and ND at 2nd div. produces 2 abnormal gametes. ',0,472,4,1),(###-12-04&&

441     Q: I\'m confused as to why the answer to problem 8-13 (E), first blank, is 2. When it says chromosome, is it referring to chromosome pair

441     A: There are 2 DNA molecules per chromosome during metaphase. Chromosome here does not refer to a pair of homologs -- it refers to a pair of sister chromatids connected at the centromere. ',0,473,4,1),(###-12-04&&

442     Q: Referring to lecture 20: When we say that a cat is heterozygous, we mean that the X chromosome carrying a black gene is active in some cells, while the X carrying the orange gene is active in others, rather than that the cat is heterozygous at the cellular level. So does heterozygous refer to the whole organism

442     A: You have it right, except for some of the terminology. Heterozygous means two different alleles per cell. It doesn\'t matter whether one or both are expressed per cell -- the cat is still heterozygous if the two alleles are different. Heterozygous refers to genotype, not phenotype. Also I would say the whole organism displays both alleles, not both genes. Tortoise shell is the pattern you get when some cells make black pigment and some don\'t. ',0,474,4,1),(###-12-07&&

443     Q: If blood samples are so conclusive in DNA testing (1 in a million), then why was OJ Simpson not convicted after the positive results of his blood all over the crime scene were presented

443     A: Because he had a very very good lawyer who threw dust in the eyes of the jury and convinced them that the blood samples that were tested could have been planted by the police. To put it more soberly, the chain of custody for the evidence wasn\'t good enough. (By the way, DNA testing is better than 1 in a million but so was OJ\'s lawyer(s). ',0,475,4,1),(###-12-07&&

444     Q: When considering twins, is there the same chance for a given genotype/phenotype to be expressed in each fetus, or do the numbers change

444     A: Identical Twins do have the same DNA pattern. So it\'s a good defense to say \"my twin did it\" if you have a monozygotic (identical) twin. But most of us don\'t have one, so you better have a good alibi. ',0,476,4,1),(###-12-08&&

445     Q: Regarding problem 8-8F. I understand why the given karyotype could show chromosomes from Mitisis or meiosis 1 of a haploid cell. But why couldn\'t it also be Meiosis II (before interphase) of a tetraploid?\r\n\r\n&&

445     A: Because we are assuming this is an ordinary cell, namely haploid or diploid, and not one of the unusual cases.\r\n\r\n',0,477,3,1),(###-12-08&&

446     Q: Regarding problem 8-14, are there any triploid cells in humans

446     A: There aren\'t any normal cells that are triploid. ',NULL,1418,3,1),(###-11-29&&

447     Q: Will meiosis be on exam 3

447     A: Anything we covered in lecture 19 may be on the exam. See the exam info page for the nitty gritty. (this means an overview of meiosis but not the \r\ndetails.)',NULL,1419,3,1),(###-11-28&&

448     Q: How are kinetochores different from centromeres

448     A: The region where the two sister chromatids stick together is known as the centromere. The kinetochore is a structure made of protein that forms \r\nat the centromere and attaches to the spindle fibers. In some contexts, the two terms are used interchangeably, but they are distinct.\r\n(We have basically been ignoring the kinetochore.)',NULL,1420,3,1),(###-12-17&&

449     Q: If an organism is heterozygous for a sex-linked allele, will the  phenotype of the organism (displayed by this gene) always depend on which  X chromosome is turned on in a cell? It seems there is no concept of  dominance with sex-linked alleles, and phenotypes are always \"patchy\"  (using the cat example). However, in an experiment in my biology lab, I did  see dominance in a sex-linked trait in Drosophila, where the bristles on the flies  were either singed or straight, and heterozygous females always had straight bristles (this gene was sex-linked also). Could you clarify this for me

449     A: Good question. The expression of sex linked traits in female mammals will  depend on whether the phenotype is \"cell autonomous\" or not. For fur color in  a cat, each hair may originate from a single cell and so the hair will be either  pigmented or not. The fact that one sees large patches of a single color means that during development a single cell moved to a position in the embryo, then had one of its X chromosomes inactivated, then further divided many times to  give rise to a patch of cells in the adult, all of which inherited the same active  X chromosome through all these later mitoses. That is, there is a delay before  X chromosome inactivation occurs in development, but once it occurs in

a cell, it is fixed in that cells and all its cellular descendants throughout the life of the animal.\r\n<br><br>\r\nIf a phenotype is not dependent on a particular cell, however, the outcome is different. Consider the X-linked gene for clotting factor in humans. This factor is made by liver cells and secreted into the bloodstream. On average, a heterozygous female will accumulate only half as much clotting factor as a homozygous wild type (normal) woman. But that is enough for clotting to occur normally, so that the clotting mutation (i.e., hemophilia as a trait) is recessive despite the cellular mosaicism.\r\n<br><br>\r\nNow as to your lab experience, you would expect that something like fly bristles would be more akin to hair color in a cat, and so exhibit mosaicism on the surface of the fly. The answer here is that X-inactivation as a mechanism for dosage compensation between male and females is a mammalian phenomenon. Flies do it another a way: both X\'s are active in females and the single X in males is cranked up in activity so that each of its genes is transcribed at twice the rate as the female X genes. \r\n<br><br>\r\nSo there\'s more than one way to skin a cat (.. or a fly). ',0,478,4,1),(###-12-18&&

450     Q: In problem #1 of exam 3, 1998 (part 1): Why is the DNA content = to 3x(4e11) instead of 6x(4e11)? I thought that DNA content per cell = 2c when there is one chromatid per chromosome, as the case is in this part of the problem.&&

450     A: C is defined as DNA content per HAPLOID cell. Below is pasted a quote from lecture 19:\r\n<br><br>\r\n\"c\" is a measure of DNA content per cell, not the number of chromosomes or chromatids. \r\n<br><br>\r\nc = minimum DNA content per haploid cell of an organism = DNA content of haploid cell before S (with unreplicated chromosomes) = DNA content of one set of chromatids. C is NOT equal to N; c is the DNA content of N chromosomes (with one chromatid/chromosome). ',0,479,4,1),(##8-09-15&&

451     Q: To assist me in learning the names and chemical formulas of the various compounds we are studying, can you shed some light on naming conventions? For example, ethyl alcohol is CH3-CH2-OH while acetic acid is CH3-CH2-COOH. Why isn\'t the latter ethyl acid? Or why isn\'t the former acetic alcohol? Thankfully I see that CH3-CH2-O-CH2-CH3 is ethyl ether. Can I at least assume that if I see the word ethyl it will always mean CH3-CH2? All helpful hints in learning names and formulas would be greatly appreciated.&&

451     A: You actually wrote 3 carbons in the acid. Three carbons represent the propyl group, so you might expect the acid to be \"propyl acid\"(sic). Actually it\'s close: propanoic acid (like propane, CH3CH2CH3). What you really meant was why isn\'t CH3COOH called ethyl acid? The answer is probably historical. Perhaps acetic acid was recognized before modern day chemistry (e.g., in vinegar produced by fermentation of foodstuffs) and named then and it stuck. There are many exceptions of this type to a unified naming convention, and there\'s no way around it. If they are important to our discussion, then to know them you have to memorize them.You can always look up the name in an index and see the formula if it is an important molecule, probably in our texts, and if not, you can track it down in a biochemistry text. From the point of view of exams, to minimize memorization, you will not be required to produce the exact molecular structure from an English name of the molecule; nor will you be required to produce the specific English name for a specific molecular structure. You WILL be responsible for knowing the functional groups and the properties that each functional group imparts upon a molecule of which it is a part (e.g., an amine will ionize at pH7, an amide is hydrophilic, etc.). ',0,480,1,1),(##8-09-17&&

452     Q: Do alpha glucose molecules only bond with other alpha\'s and betas only bond with betas

452    A: The homogeneity of bonds (all alpha or all beta) to which I referred in lecture were meant to apply only to the specific ploymers under discusson, i.e, glycogen/starch and cellulose. In the former it is all alphas and in the latter all betas. But polysacchrides exist that contain a mixture of alphas and betas (dermatan sulfate). However, even here, that pattern is repetitious and thus still monotonous, i.e., a, and NOT a,b. ',0,484,1,1),(##8-09-17&&

453    Q: Why do phospholipids break down fats

453    A: Phospholipids (along with molecules that behave similarly, the bile salts), bind to the large globules of fat (triglyerides) that are in the stomach and the small intestine after a meal of chicken soup. This fat can only be chemically broken down (hydrolyzed) by reactions that occur in aqueous phase (like most biochemical reactions) , so that this hydrolysis would only occur at the surface of the large (e.g., 1 cm diam) fat globule. Phosphoplipids can associate with the fat through hydrophobic interaction of their fatty acid groups, but having done so, they contribute a highly hydrophilic (charged) end. If many such phospholipids surround a portion of the fat globule, a small piece can pinch off, covered with charged groups,so it has little tendency to reassociate with the large original globule. The result of this process is the conversion of the large globule to many small globules, which increases the surface area that react with the aqueous process of digestion (hydrolysis). The smaller globules also are more easily transported into the cells of the intestinal wall. ',0,485,1,1),(##8-09-17&&

454    Q: Can disaccharides only have alpha-alpha links or beta-beta links

454    A: The homogeneity of linkage type to which I was referring in lecture was in starch/glycogen (1,4\'s all alpha) and cellulose (1,4\'s all beta), specifically. I did not mean to rule out linkage of more than one type in other polysaccharides. On the other hand, even if such diversity were present it would not change the basically monotonous character of most polysaccharides, the linkage pattern would be repeated, eg., a,b and not a,a,b. As we will soon see, such monotony is NOT characteristic of most cellular polymers (i.e., proteins and nucleic acids). ',0,486,1,1),(##8-09-21&&

455    Q: In relation to the two questions about the uniformity (or at least repetitiveness) of bonds in the sugar polymers: ia this uniformity a result of anything inherent to the sugars themselves

455    A: No, it\'s a function of the enzymes that put the macromolecules together, which are \"programmed\" in a way to make only that kind (or series) of bonds. There are only a few enzymes to do this job, and an enzyme is usually designed to do just one job (and do it well, as we shall see). Hence the repetitiveness. ',0,488,1,1),(##8-09-22&&

456    Q: Why does the structural formula of alpha mannose display 7 oxygen atoms, where it is only supposed to have 6? Similarly, why does the structural formula for Fructose display 4 Oxygen atoms, where it is supposed to have 6 oxygen atoms

456    A: There is an error in Figure 3.11 on page 48 of Purves. There should be an H pointing down from position 2, not an OH. For fructose, there should be an OH above the ring on carbon 2 and an OH above the ring on carbon 3. ',0,489,1,1),(##8-09-22&&

457    Q: What is the purpose of adding urea to a dialysis sack with protein

457    A: The urea was added in the Anfinsen experiment to denature the protein (ribonuclease) in what turned out to be a reversible manner. The use of a dialysis sack was to allow the gradual change

in the denaturuing condition, especially in the removal of the urea. By letting the urea diffuse slowly out of the pores of the sack, its concentration gradually lessened, giving time for the polypeptide to explore different bonding possibilities, to settle on the most stable one, which turned out to be the one he started with, the native, functional form. ',0,492,1,1),(##8-09-28&&

458     Q: Precisely what is included in the generalization \"weak bonds and forces?\"&&

458     A: By weak bonds and forces, I refer to all the weak interatcions we discussed: ioninc, H-bonds, hydrophobic forces, and van der waals. VDW was the weakEST but they are all weak compared to covalent bonds, the only strong bonds we talked about. Hydrophobic forces will be important if, for example,  two amino acid side chains have several hydrocarbon groups (-CH2-\'s and -CH3\'s). VDW bonds can take place between any two groups, regardless of their polar or non-polar character, as long as they are postioned VERY close to one another. ',0,493,1,1),(##8-09-28&&

459     Q: What would happen if urea diffused extremely quickly or somehow instantaneously from a dialysis sack? Would there be a difference in renaturation of the protein compared to what happens with slow diffusion of the urea

459     A: Urea diffuses slowly into or out of a dialysis sack because the molecules must find the pores by random motion. The slow diffusion out during renaturation is thought to be important, as it would allow the polypeptide, through random motions, to explore different bonding possibilities while it is still partially denatured and settle on the strongest combinations. Thus when the urea finally gets to zero, the polypeptide has found its most stable state, presumably the native form. ',0,494,1,1),(##8-09-28&&

460     Q: In your lecture about gel electrophoresis with protein gels, as well as online, the anode is labled with a (+) and the cathode is labeled with a (-). Is it a typing error, or is a different convention being used

460     A: In an electrolytic cell, the positive electrode is the anode. That is the terminology we are following. In any case, we will always try to specify the positive electrode or negative elctrode in addition to or instead of using the words anode or a cathode. (The reason for the discrepancy is that anodes and cathodes aren\'t directly related to charge, but where oxidation and reduction occur. Oxidation always happens at the anode, reduction at the cathode.)',0,495,1,1),(##8-09-30&&

461     Q: What is the difference between endergonic reactions and endothermic reactions? (Likewise for exergonic and exothermic)&&

461     A: Endergonic and exergonic refer to free energy changes (delta G). Endothermic and exothermic refer to the changes in internal energy of molecules, measured as heat given off or taken up, delta H. Delta g takes into account not only changes in internal enregy but also changes in entropy (delta S) that may accompany a reaction. For most ordinary simple chemical reactions, the entropy factor is not great, so chemists usually talk about delta H. For many biological reactions,the entropy factor is significant, so biochemists usually talk about delta G. Delta G = delta H - (T x delta S ). Despite the signs, which have to do with the way these terms are defined, delta G is the sum of the 2 effects. Becker presents this very nicely. You may forget about delta H for this course. More in a later lecture. ',0,496,1,1),(##8-10-01&&

462     Q: With what properies of an amino acid side chain can I predict its behavior

462     A: You just have to memorize the properties of the functional groups as to their acidity or basicity: R-COOH groups are the only organic groups that we talk about that lose a proton and so become negatively charged, and amines take on a proton and become psotively charged. Amines include R-NH2 and the guanido group of arginine, R--NH-CNH-NH2, but NOT the -NH2 of amides, R-CO-NH2. Thus glutamine, asparagne, and the peptide bonds in polypeptides do NOT gain a full charge. You can figure ou tpolarity if you remember that O and N are more electronegative than H or C, so those combinations will result in partial charge separtations and polarity. ',0,497,1,1),(##8-10-02&&

463     Q: In the Purves book, tyrosine is listed as having a hydrophobic side chain, whereas in the Becker text it is listed as having a hydrophilic side chain. It would seem to be hydrophilic due to the -OH group, but does the ring change that

463     A: The tyrosine side chain has lots of CH groups, making the ring and stem very hydrophobic, but it also has that hydroxyl, which is actually VERY polar. So take your choice; Profs Purves and Becker picked the part they thought was most important and disagreed. My opinion is that tyr can be both: when approached from the stem side by a non-polar group, it could make a hydrophobicinteraction. But its hydroxyl could also H-bond very well to a partially negatively charged group it points at. So I say, both. Nature need not be simple (or unambiguous). ',0,499,1,1),(##8-10-02&&

464     Q: Do we have to know how to determine the original order of polypetide fragments subjected to fingerprinting or some other technique of protein purification

464     A: As I mentioned in class, since I did not go over the sequencing strategy for putting subpeptides back in order (on paper), I will not ask such a question on an exam. ',0,500,1,1),(##8-10-05&&

465     Q: How can I determine the form of an amino acid side chain? For example, some R-side chains with amine groups ionize to become basic whereas others do not (lysine vs. asparagine).&&

465     A: The asn (asparagine) side chain is not an amine, It is an amide: -CONH2 is not equal to -CH2NH2. The connection to a carbon bearing a double-bonded oxygen changes the basicity of the NH2 so that it does not ionize. You do have to recognize all the functional groups listed on the handouts, and recognize that an amide is an amide and an amine is an amine, etc. and which are ionized, which are polar, which are hydrophobic, the significance of the sulfhydryl, etc. ',0,501,1,1),(##8-10-06&&

466     Q: According to the answer of problem 2-14, the two polypeptide pieces DO NOT rejoin, and thus the renatured protein only \"looks\" like the original. However, the problem itself states that the restored protein has \"regained its full enzymatic activity.\" If so much as one amino acid substitution disables an enzyme, how can an enzyme which is not fully renatured be restored to its normal activity

466     A: Whereas a single amino acid change CAN disable an enzyme\'s cataltyic activity, such a disablement need not occur. Indeed, most single amino acid substitutions are benign. Especially those that conserve the character of the amino acid (e.g., val for ile, asp for glu). ',0,502,1,1),(##8-10-06&&

467     Q: Regarding problem 2-3E: In addition to gel electrophoresis without SDS, why would fingerprinting, which also separates by charge/solubility not work

467     A: Fingerprinting destroys the polypeptide, by definition, since the first step is to chop it up with protease. The separations desired here are for the purposes of purifying proteins and polypeptides, separating them away from each other. ',0,503,1,1),(##8-10-06&&

468     Q: The answer to 2-20 said that a triglyceride would be found in the benzene phase because it is nonpolar. However, if the triglyceride was part of a phospholipid, then couldn\'t it be found at the interface because bilayer phospholipids are both hydrophobic and hydrophilic

468     A: A triglyceride cannot be a phospholipid, because the tri- means 3, which means that all 3 of the hydroxyls of glycerol are esterified to a fatty acid. So there\'s no place to put the phosphate. Thus phospolipids are typically diglycerides (mono- is possible). I agree with you that a phospholipid would probably go to the interface. ',0,504,1,1),(##8-10-06&&

469     Q: What exactly is a chaotropic agent and how does it work? (You provided urea as an example and mentioned that it is very soluble in water and thus is good at disrupting hydrogen bonds. Is it therefore only involved in the disruption of secondary structure?) &&

469     A: Yes, urea disrupts secondary and tertiary and quaternary, but not primary structure nor disulfide bonds. How it works is not so clear, but probably by interfering with the proper formation of hydrogen bonds that are important for secondary structures and for interaction between side chains in tertiary structure. ',0,505,1,1),(##8-10-06&&

470     Q: The answer for number 4B of the 1997 exam #1 asks which enzyme has the higher turnover number, and the answer states that we can\'t tell because we have no idea what amount of enzyme is being used. Why isn\'t the largest turnover number for the SDH-M, since the problem specifies that 1 mL of enzyme is used for each reaction. (Can\'t we just divide Vmax by Eo to get k3?)&&

470     A: You cannot calculate the number of moles of enzyme from the number of mL. of enzyme unless you know the concentration of the enzyme in the solution. That is, ml is a unit of volume, not of moles or molecules or grams of enzymes. The solution could be very concentrated or very dilute - we just don\'t know from the info given. ',0,506,1,1),(##8-10-06&&

471     Q: For number 1C on the 1997 exam #1, how are we supposed to know that starch also makes 1 to 6 glycosidic bonds and that these dimers are preserved

471     A: Starch has a branched structure, and the branches must be at a position other than 1 or 4, as these are tied up in forming the straight chain. You did have to remember that starch, unlike cellulose, has a branched structure. ',0,507,1,1),(##8-10-07&&

472     Q: Does paper chromatography only separate individual amino acids that have been cleaved (like in fingerprinting), or can it also separate \"small peptides\" as it says in the lecture notes on the web

472     A: First, paper chromatography DOES separate small peptides (up to, say, 20 AAs). As a peptide gets longer, it tends to precipitate when exposed to the organic solvents used in paper chromatography. Second, fingerprinting doees NOT involve paper chromatography or electrophoresis of amino acids. If that were the case, ALL proteins would give the same 20 spots, and it wouldn\'t be in the least a \"fingerprint.\" Rather, fingerprinting involves the separation of small peptides produced by proteolytic cleavage of a polypeptide at defined sites in the primary structure (e.g., after aromatic

(ring-containing) side chains. That way, every different polypeptide yields a different set of small sub-peptides. ',0,508,1,1),(##8-10-07&&

473    Q: Noncompetitive inhibitors bind to a site other than the active site and render the enzyme ineffective. Allosteric inhibitors do the same thing. So, how are they different? And in what way can we apply the Michaelis-Menton equation to our understanding of allosteric inhibitors

473    A: I agree that at a simple mechanistic level non-competitive and allosteric inhibition appear the same. There are several differences, however. Allosteric inhibition generally acts by switching the enzyme between two alternative states, an active form and an inactive form. It usually works by binding to a sites in a specialized subunit of a mutlimeric protein, and thus binds at several sites. The more inhibitor that binds, the more then can bind, and vice versa with substrate. The kinetics are thus complicated, being cooperative, and non-Michaelis Menton and are beyond the scope of this course. So a qualitative understanding is all that is called for here. Allosteric inhibition is designed into the proteins and represents an important physiological process.<br><br>\r\n\r\nNoncompetiive inhibiton is more of a catch-all for non-physiological inhibition that does not compete with substrate for substrate binding to enzyme. In that, it is defined (and named) from a negative point of view. As described in your texts, a non-competive inhibitor may bind to a non-substrate site on a protein and distort it to the point of non-functionality, and adding more substrate will not alleviate this inhibition. Or, as in the example I used in lecture, it may simply block a catalytic site without interfering with substrate binding, an example that is more distinct from allosteric inhibition. ',0,509,1,1),(##8-10-07&&

474    Q: For problem 3-5C, why is it that the altered Km in the sick individual does not cause the disease? As I understand, noncompetitive inhibitors do not change Km but do change Vmax. In this case, Km has in fact changed. Why isn\'t this change substantial

474    A: The Km for the mutant indicates its affinity for substrate has decreased to one-tenth that of the normal enzyme. But this is probably of no consequence to the individual, since the steady state concentration of substrate is 10 times higher than even the mutant\'s high Km. Under this condition the mutant enzyme is operating at near Vmax, presumably the same Vmax as the normal enzyme (since turnover number was not said to be affected, just the Km). ',0,510,1,1),(##8-10-07&&

475    Q: Is the turnover number dependent on [E]

475    A: You are correct in thinking that the turnover # is a quality independent of enzyme concentration. The formula k3 = Vmax/Eo simply represents a way to calculate that quality, given data you can collect (measure Vmax, measure the amount of E that gave you that Vmax). ',0,511,1,1),(##8-10-09&&

476    Q: We\'ve seen alpha-glucose->alpha-glucose, and beta-glucose->beta-glucose: so what does alpha-glucose->beta-glucose make

476    A: The alpha or beta conformation is locked in only where a glycosidic bond is made. The C1 that is not inolved in bondong will be in equilibrium between alpha and beta (mixture) and just the the free monomer. Thus we can only talk about a glucose-alpha-glucose or glucose-beta-glucose. ',0,512,1,1),(##8-10-12&&

477    Q: I have read that gel electrophoresis without SDS and molecular sieve chromotography separate polypeptides only on the basis of size (MW) and charge, but the answers to question 8 of

problem set 2 claim that these techniques also separate polypeptides on the basis of their shape (diameter). Is this true

477     A: It is the effective diameter of the molecule that limits its ability to migrate throught the network of criss-crossed polyacrylamide fibers. The majoty of native proteins are globular and so in their native state (as in \"native\" PAGE), they will migrate according to their MW, and their net charge. If they are denatured without using SDS to charge up the molecule (e.g., using urea), then in their unravelled state their effective diameter will be larger than in their folded-up state, so they should havemore trouble negotiating the network of fibers, and thus migrate more slowly than their native counterpart. Molecular sieve chromatography (also known as gel filtration or Sephadex chromatography) does not separate on the basis of charge like electrophoresis, but only on the basis of size. The considerations above relating size to MW also apply here. ',0,513,1,1),(##8-10-15&&

478     Q: The mean on the exam #1 was 65, but is this score set at a B grade

478     A: Grades are given after the best 3 of the 4 exams are chosen for each student, i.e., dropping the lowest grade. Historically, the mean grade in this course has been at the B-/B border, or a low B. Around the time of the 3rd exam, we usually post guidelines indicating a 3-exam point totals that will guarantee each letter grade (e.g., B+,C-, etc.). ',0,515,1,1),(##8-10-06&&

479     Q: What exactly are the 2 ways to get an unfavorable reaction to go forward?\r\n&&

479     A: Two ways to get a reaction with a positive delta Go to go forward (from left to right).\r\n1. Couple it physically to an exergonic reaction (e.g., usually the hydrolysis of ATP) so that a new reaction, more complicated, is created, and the delta Go of this new coupled reaction is now negative (favorable). Example considered was Gluc --> G6P with ATP --> ADP. \r\n2. PULL a reaction with an unfavorable delta Go to the right by having a downstream reaction that is highly exergonic. The downstream reaction will deplete the products of the unfavorable reacton thus making the second term in the equation for delta G highly negative, so much so that the overall delta G becomes negative despite a positive delta Go. ',0,516,1,1),(##8-10-19&&

480     Q: If a nucleotide = sugar + base (no phosphate), then why are ADP/ATP called <i>nucleosides</i> on your handout

480     A: A nucleoSIDE has a sugar + base (no phosphate). A nucleoTIDE has a sugar + base + 1 or more phosphates. A nucleoTide can also be called a nucleoSide mono (or di or tri) phosphate. ADP and ATP are nucleosides plus 2 or 3 phosphates. So they are called nucleoside di (or tri) phosphates OR nucleotides. ',0,517,2,1),(###-10-27&&

481     Q: Regarding question 7-7A:  How can there be more RNA than DNA if RNA is transcribed from only one strand of DNA

481     A: Two issues here:<br><br>\r\n \r\n1. When most genes are transcribed, they are transcribed many times to make multiple copies of the RNA. That is one of the advantages of \r\nhaving RNA -- you can make as many copies as you like. (You need many copies of tRNA, rRNA and mRNA to have translation go fast \r\nenough.) So yes, there are multiple copies of the RNA.\r\n<br><br> \r\n2. We are talking about a test tube experiment. You break open a lot of cells and separate the DNA and RNA. Then you mix the DNA and \r\nRNA back in whatever proportions you like. Not necesarily the proportions found in cells.',NULL,1405,2,1),(##8-10-19&&

482    Q: What do you mean by Delta Go reflects the nature of reactants and products? (you mean, the direction of the reaction?)&&

482    A: Delta Go is a means of predicting the direction of a reaction. Both the Delta Go and therefore the direction of the reaction depend on the nature of the reactants and products: the reaction will tend to go in the direction of forming the most stable molecules. The Delta G depends not only on the the nature of the molecules involved, but also the amounts of each that are present at the time in question. ',0,519,1,1),(###-10-25&&

483    Q: What is the difference between unidirectional and bidriectional DNA replication

483    A: A bidirectional origin = two forks; it looks like two Y\'s with their open ends facing each other. (See bottom of handout 11-3 -- bidirectional, vs the top -- unidirectional.)',NULL,1404,2,1),(##8-10-21&&

484    Q: When you say that a DNA or RNA strand is 20 bases long, does that mean that it is 20 base pairs long, or 10 pairs of two bases long

484    A: If the nucleic acid is single stranded, and we say it is X units long, we mean X bases; if it is double stranded we usually mean X base pairs in the double stranded molecule or X bases in each strand.',0,521,2,1),(##8-10-21&&

485    Q: Will we be responsible for the information needed to answer question 5-10 on fatty acid catabolism

485    A: You would be expected to be able to determine the consequences of the metabolism of a new compund as long as you are given the enzymatic reactions that connect it to the pathways of energy metabolism that we went over in detail: i.e., glycolysis/fermentation and the Krebs cycle. Such connecting metabolic paths are given in probelms 5-10, 5-12, and 5-13. For these types of problems, the glycolysis/fermentation and Krebs Cycle handout would be made available on the exam. ',0,522,2,1),(##8-10-21&&

486    Q: The answer to problem 5-2B states that all ATP production will stop because NADH cannot become oxidized, thus halting glycolysis and the krebs cycle (from CN- addition). Focusing just on the electron transport chain, can we still say that no ATP is produced

486    A: If any one step in the electron transport chain is blocked, then the cytochrome just upstream of the block will not have a way to get rid of its electrons and so will be stuck in the reduced form. Soon, all the electron transport chain proteins and other carriers (e.g., CoQ) will fill up with electrons and also be stuck in the reduced form. And so NADH2 produced from this time on will have no acceptor available for its electrons, and will also be stuck in the reduced form. All NAD will end up this way, and so little NAD will be available for the oxidative reactions of the Krebs cycle or glycolysis. Until the accumulation of all carriers in the reduced condition, H+ pumping will continue. This continuation will probably be for a very short time. In addition, the steady-state H+ gradient that had been built up prior to the addition of cyanide could run down (into the mitochodria, producing ATP for a short time as the gradient is dissipated. Thus we would expect the continued production of ATP for a short time after cyanide addition. This situation was contrasted to the addition of another drug, dinitrophenol, in problem 2B of the 1997 exam #2. ',0,523,2,1),(###-10-27&&

487     Q: Does the fact that the curve from which Tm is derived will be biphasic if there\'s a mix of different molecules mean that Tm is actually the temperature at which the halfway point of each phase is reached, rather than the temperature at which half of all DNA present is denatured

487     A: Normally you have a pure sample that denatures at a fixed temp. If you have a mixture, you get a multiphasic curve. The midpoint of each \r\ntransition defines the Tm for a separate component of the mix.',NULL,1406,2,1),(###-10-27&&

488     Q: At the stem of a tRNA molecule, is the 3\' the left or right strand? To which strand (5\' or 3\') does an amino acid bond

488     A: The clover leaf of tRNA can be written either way. In standard convention, 5\' is always on left. When tRNA is pairing with mRNA, it\'s usually draw reversed to \r\nshow pairing to codon is antiparallel. AA is always on 3\' end. (no free hydroxyl on 5\' end)',NULL,1407,2,1),(##8-10-23&&

489     Q: Why don\'t you get deoxyribose backbones with U-bases, or ribose backbones with T-bases? &&

489     A: The enzymes involved in nucleotide synthesis are specific -- won\'t add ribose to T or deoxyribose to U. (Bases and sugars are made separately and then hooked up.) Then the enzymes for nucleic acid synthesis are specific too -- DNA polymerase won\'t use anything with U or ribose and RNA polymerase won\'t use anything with deoxyribose or T. ',0,524,2,1),(##8-10-23&&

490     Q: What happens if the primer sequence in PCR is a mutant in the individual you\'re looking at? Can you get 19 out of 20 matches? What if (as I know happens) some of the exon segments flip (and thus throw off the primer matching)

490     A: It depends how you set up the conditions for hybridization. You can set them up (stringently at higher temp) so only perfect matches stick together, or you can set them up (leniently, at lower temp) so almost perfect matches will stick together too. As to exons etc., you use a primer (usually) outside the area you want to copy, so order of exons/introns and so on doesn\'t matter if primer brackets the change. If it doesn\'t, you\'ll need a new primer. ',0,525,2,1),(##8-10-23&&

491     Q: How do you know you\'ve got a unique primer

491     A: You find out by trial and error. (You hybridize your primer to cut up DNA and look for a primer that sticks only to one piece.) ',0,526,2,1),(###-11-09&&

492     Q: About problem 7-12D. Since a\r\ntrailer usually comes after a stop codon that cannot be translated, how is it possible for a ribosome to start translating\r\nafter a stop codon

492     A: This question (except part G) is about artificial conditions where no start codon is required. Under normal circumstances, for a \r\nmonocistronic mRNA (coding for one peptide) there is no start codon after a stop, so the ribosome can\'t restart. (For polycistronic \r\nmRNA\'s there may be a AUG a bit further down after the stop, and ribosome may indeed restart.)\r\nThe parts of this question, except G, assume you don\'t have to worry about starting at an AUG.',NULL,1410,3,1),(##8-10-23&&

493     Q: How does a circular piece of DNA not get its double-helix all in a twist

493     A: There are enzymes that cut single strands so the mess can unwind. We have not paid much attention to the topology, but any biochem or molecular bio book will tell you the details. ',0,528,2,1),(##8-10-23&&

494     Q: Is a coupled reaction any reaction that combines an endergonic and an exergonic reaction

494     A: In the broadest sense, we can talk about two reactions coupled to contrast the same two reactions not coupled, that is, occuring independently. This is easy to see for the first such coupled reaction we considered, the phosphorylation of glucose by ATP instead by phosphate addition. In English terms, we can talk abstractly about the reduction of NAD to NADH2, and the oxidation of pyruvate to acetyl-CoA and CO2, as separate \"paper\" reactions, that are then coupled in the actual reaction leading to the Krebs cycle. ',0,529,2,1),(##8-10-23&&

495     Q: Is it the case that an oxidation releases energy and a reduction requires energy to accept electrons?\r\n&&

495     A: No, any particular oxidation-reduction reaction will have a particular delta Go, which could be positive or negative. It is the combination of the two that will determine the delta Go. ',0,530,2,1),(##8-10-23&&

496     Q: If 1 glucose ---> 2 lactates (delta Go = -45), What would be the delta Go at 2 pyruvates from glucose

496     A: The 45 figure was for the transformation without considering ATP coupling. I cited the figure of -18 kcal/mole of glucose in the lecture and handout, for the reactions from glucose to 2 pyruvates, including and taking into account the net production of 2 ATPs. If you want to consider the standard free energy change of glucose to 2 pyruvates without making ATP, it would be -18 -(7X2)= -32. ',0,531,2,1),(###-11-03&&

497     Q: How do I get to be a TA

497     A: You send me a brief email about why you want to do it and why you think you\'d be good at it. I collect the emails, and interview the \r\nprospective TAs at the start of the next year. I ask prospective TAs to stand at the board and explain a problem. I look for students who are \r\ngood explainers (clear but not boring) and seem to have a sympathetic, patient manner. I have no absolute grade cut off, but the better the \r\ngrade the more likely you\'ll end up as a TA. Another factor is scheduling -- I have to find TAs who can come at the times we offer recitations \r\nand attend a weekly TA meeting (usually Mon around 4 pm.)',NULL,1409,3,1),(##8-10-23&&

498     Q: What is meant by coupling formation of ATP? I though coupling usually refers to making ADP in an exergonic reaction.&&

498     A: No, coupling refers to two otherwise independent reactions that are obligatorily carried out together by the enzyme. Thus when ATP is produced by substrate-level phosphorylation, it is via coupled reactions producing ATP from ADP + Pi, as in the 2 relevant steps (7 and 10) in glycolysis. These are coupled reactions just like hexokinase (think of the hexokinase reaction in reverse, still coupled). ',0,533,2,1),(##8-10-23&&

499    Q: How many times do we use the breaking of Pi bonding to release the energy needed to make ATP from ADP

499    A: In glycolyis? 4 times per glucose, twice (per glucose) in rxn 7 and twice in rxn 10. ',0,534,2,1),(##8-10-23&&

500    Q: Does NADH2 give off 2 electrons to produce energy as a part of a coupling reaction

500    A: Not really. The coupling of energy production to NADH2 oxidation is very indirect, via the chemiosmotic mechanism. The term \"coupling\" could be used in English for this more indirect use of NADH2 oxidation for energy production. But the term coupling is usually reserved for a more direct linkage. ',0,536,2,1),(##8-10-23&&

501    Q: Does NADH2 provide energy for ATP to be made

501    A: Yes, via oxidative phosphorylation ',0,537,2,1),(##8-10-23&&

502    Q: What determines whether lactic acid or ethanol + CO2 is produced when oxygen is absent from the pathway

502    A: The pathway used for fermentation depends on the organism. Yeast will use the ethanolic route. Mammals and E. coli will go the lactate route. ',0,539,2,1),(##8-10-23&&

503    Q: I don\'t quite understand the purpose of the PS+ ---> PS- experiment. My understanding is that we wish to convert poisonous PS+ to PS-.&&

503    A: Comment: No, it\'s the other way around. (We could do it either way, but it\'s easier to detect a few PS+ in the middle of a lot of PS- than the other way.) \r\n\r\n<i>We tried PS+ with different other compound, but only PS+ plus DNA worked. (PS+ + DNA--->PS- This rxn is called transformation?)</i>\r\nComment: Yes, this is called DNA transformation, because DNA does the \"transforming\" or changing of properties.\r\n\r\n<i>A) We knew the genetic info is passed on from the one cell to the exact the identical cell when self copying one, but in the PS+ +DNA --> PS- experiment, the initial reactants and the final product are not the same material. So what does this still prove about DNA? DNA transformed PS+ to PS-. But what does this say about DNA?)</i>\r\nWhen one ordinary cell grows, it copies its DNA exactly and each descendant gets a copy. This case is different -- DNA from one organism (PS+) is chemically extracted (purified) and transferred to another organism (PS-) whose DNA is different. Some of the added DNA replaces some of the original DNA in the recipient. From then on, the recipient is PS+ and faithfully copies and passes on PS+ DNA.\r\n\r\n<i>B) In my notebook I wrote down PS- +DNA --->PS+ Is this what you wanted to write on the board?</i>\r\nYes, the PS- cell + DNA from PS+ --> cell that takes up PS+ DNA and becomes PS+.\r\n\r\n<i>C) How does DNA\'s transformation ability imply it can do the 2 jobs: to order the AA, and to make copies of itself? In this PS experiment specifically, what substance did DNA order of AA? What copies did DNA make? </i>\r\nChange from PS- to PS+ was permanent and inherited. Being PS+ is due to making a particular enzyme; PS- doesn\'t make it. The DNA from a PS+ must contain the info to make that enzyme and copy itself -- that\'s the only way transferring DNA could turn a PS- into a permanent, inherited state of PS+. ',0,540,2,1),(##8-10-23&&

504    Q: Why is thymine used so often to radioactively label DNA

504     A: T is unique to DNA. A is not -- it\'s found in RNA (and ATP) too. So that\'s why T is better if you want to label only DNA. ',0,541,2,1),(###-11-03&&

505     Q: Given that it\'s advantageous to have fewer different tRNAs, why not have just one codon for each AA, and therefore have just one tRNA for each AA (plus one exrtra for met)? What exactly is the advantage of degeneracy

505     A: No one really knows the answer for sure. One reasonable idea is that degeneracy serves as a buffer against mutation. If there were only one \r\ncodon per amino acid, then every base change would produce a change in the corresponding protein. Since most AA changes are not \r\nadvantageous, this would increase the consequences of mutations (relatively to the current system). Also, to cover 20 amino acids, you need \r\nat least 20 codons. You can\'t use dimers and have to use (at least) triplets. Once you have the extra codons, what will they code for? Having \r\nthem be nothing, or stop, makes the code even more sensitive to trouble from mutations.',NULL,1408,3,1),(##8-10-26&&

506     Q: Where exactly is the 1 ATP produced in the Krebs cycle

506     A: See lecture 9: One GTP is formed by substrate level phosphorylation in the coupled reaction forming succinate from alpha-keto-glutaric acid. This GTP is energetically equivalent to ATP, and there is an enzyme in the cell that will carry out the interconversion: GTP + ADP <--> GDP + ATP, Delta Go = ~0. ',0,542,2,1),(##8-10-26&&

507     Q: The answer to problem 5-5B states that since the yield of ATP/NADH2 oxidation is still normal in the mutant mitochondrion, oxidative phosporylation must be ok and that electron transport must be defective. Why is this so

507     A: If the ATP synthetase reaction were slowed, (e.g., by a leaky mitochondrial inner membrane) then NADH2 conversion to NAD would be normal but less ATP would be realized, and the ratio of ATP per NADH2 oxidized would go down. But such is not observed, the ratio is normal. So it must be that less NADH2 is being produced, to account for the low rate of ATP production. Also, the defect is stated to be due to a defective (though apparently not completely defective) cytochrome. The cytochromes are used for electron transport, not for ATP synthesis directly. The defect must limited to oxidative phosphorylation (the overall process) and could not, for example, be in a glycolytic step, for the same reason, as cytochromes are not involved in glycolysis. Also, most of the energy in this organism must come from oxidative phosphorylation, since it cannot grow anaerobically. ',0,543,2,1),(##8-10-26&&

508     Q: In problem 4-7C, why is it not possible for (ii) to be the answer if a positive delta G will prevent K from going to Q

508     A: Delta G will determine the net direction of a reaction, and the ratio of products to reactants at equilibrium will be detrmined by a delta Go. But as a reaction runs toward equilibrium or is even at equilibrium, reactants are still being converted to products and products are reforming into reactants. At equilibrium there is no NET change in the concentration of each, but much interconversion is still constantly taking place. In the cell, on the other hand, equilibrium is rarely reached, because the products of a reaction in a pathway are being swept away (consumed) by the next reaction in the path. Thus products often do not have much chance to reform into reactants. ',0,544,1,1),(###-11-16&&

509     Q: How does one know which mRNA to extract from an immature RBC to use as probe? Do we assume that we already know the amino acid\r\nsequence of the hemoglobin protein (like we do to make oligonucleotide probes)

509     A: You do need some trick to isolate the mRNA of interest. If I recall correctly, what you usually do is precipitate polysomes making protein using antibodies to \r\nHb. Then you extract mRNA from those polysomes -- it is mRNA for Hb.',NULL,1411,3,1),(###-11-16&&

510     Q: When cleaving DNA with restriction enzymes prior to a Southern blot,\r\nhow do you know which restriction enzymes to use? Do we assume that the entire DNA sequence is known

510     A: You try a bunch of different enzymes. Usually you do not know the entire DNA sequence. If so, you\'d use PCR instead of cloning to look at that section of the DNA. (You\'d make multiple copies by PCR, not cloning, and then cut them up \r\nwith restrict. enzymes to see which version of the RFLP you had.)',NULL,1412,3,1),(##8-10-27&&

511     Q: If primase is a form of RNA polymerase, how can it incorporate T into primer sequences? I was surprised to see that the answer did in fact include a T in the primer sequence.&&

511     A: Preformed DNA primers can have T, not RNA primers. If you were using RNA primers, they would have U instead. ',0,546,2,1),(##8-10-28&&

512     Q: I know that the 3\' end of a polynucleotide has a free OH group. But what about the 5\' end? Is there a \"rule\" regarding how many free phosphates are attached to the last nucleotide on the 5\' end of a strand

512     A: If polynucleotide is made continuously, starting from the 5\' end, it should have all P\'s (that came in with the first nucleotide) still left on the 5\' end. However, because of the fine points of synthesis and/or modification, the 5\' end may not retain all of those P\'s. Alternatively, polynucleotide may be obtained from cutting up a longer chain -- in that case, the 5\' end will only have (at most) 1 P. So it all depends, and that\'s why the problems are not all the same. ',0,547,2,1),(###-11-16&&

513     Q: Are all individuals\' RFLP\'s flanked with\r\nthe same restriction sites

513     A: Some restriction sites don\'t vary much and are almost always the same. The pieces you get are generally all cut at those sites. The variations in length come \r\nfrom variation at other sites. Your probe sticks to a site inbetween a fixed site and a variable one.',NULL,1413,3,1),(###-11-19&&

514     Q: If cDNA is copied from mRNA, doesn\'t \r\nit lack regulatory stuff like start codons (assuming initial met is removed), stop codons, promoters, terminators, 3\' UTRs, 5\' UTRs, operators, etc., rendering it useless

514     A: The start & stop codons are in the mRNA in the exons. (As are UTR\'s.) I think you are confusing the start signals for transcription and the start signals for \r\ntranslation. The met is removed from the protein, not from the mRNA. The signals for transcription are not in the cDNA -- they are in the plasmid that you insert \r\nthe cDNA into.',NULL,1414,3,1),(##8-10-30&&

515    Q: If you can take into account the concentration of enzyme while a cell multiplies, wouldn\'t it be more probable that its glycolytic enzyme concentration would slowly increase (for the preparation of its daughter cell), and then when division occurs, for the concentration to level off

515    A: If you are following a single cell or a bunch of cells dividing in synchrony, you are correct. But in a whole culture, different cells are at different points in the life cycle -- some are new (just finished division), some old (big and about to divide) etc., so the average amount of enzyme per cell stays the same. ',0,549,2,1),(##8-10-30&&

516    Q: What does \"expression\" mean in the context of protein or gene expression? That a certain protein is being synthesized by the cell, and is it confined only to certain molecules

516    A: You\'re right -- gene expression means making the gene product (usually a protein, but sometimes a tRNA, rRNA, etc.) so the effects of the gene and its product are measureable, determinable, etc. Some genes are only \"expressed\" at certain times or in certain tissues (in a multicellular organism) -- under other conditions the genes are effectively mute. ',0,550,2,1),(##8-10-30&&

517    Q: Are we responsible for knowing fatty acid metabolism

517    A: You are not specifically responsible for the nitty gritty of fat metabolism, but you should be able to figure out how to fit anything into the standard pathways -- if you are given the necessary reactions that connect whatever the substrate is to some intermediate of the standard pathways. Those problems were written assuming fat metabolism was not discussed in class. ',0,551,2,1),(###-11-18&&

518    Q: How can you turn the production of a single enzyme on and off if you only have one promoter

518    A: You don\'t usually turn on/off production of a single enzyme -- you turn on and off synthesis of a bunch that all are needed to carry out one function.',NULL,1416,3,1),(##8-10-30&&

519    Q: The book is somewhat confusing about the order and method of DNA polymersase\'s actions on a lagging strand and the ligase catalysed linkage. Suppose there is one Okazaki fragment with a primer at its 5\' end. The first action (upon completion of the fragment by DNA polymerase) is an excision of this primer by an exonuclease. This excised primer is then converted into DNA through the work of DNA polymerase from another fragment, the fragment \"behind\" (formed after) the first fragment in question continues to grow to the point where its nucleotides replace the excised primer: what was once a primer on the 5\' end of fragment 1 (the first fragment made) is now the last few nucleotides on the 3\' end of fragment 2. Once this is completed, ligase catylzes the connection between the 2 disjointed fragments. If this were not the case, the answer to problem 6-17C (part III) would be #10-#11 and the problem of telomers would not exist since, as it seems by the pictures in the book, DNA polymerase could somehow replace the excised primer by adding to the 5\' end. Is this the case

519    A: You are absolutely right. The pictures in the book (fig 15-9 and 11) show primer being replaced by DNA polymerase without the DNA being added to anything. As you say, if this would work, there would be no telomerase problem, and I assume it is an error. (I noticed the problem in 15-11, but

I didn\'t notice 15-9 before). Your statements are all correct, except that the removal of primer from 5\' end of frag. #1 and elongation of 3\' end of frag #2 may be simultaneous. ',0,553,2,1),(###-11-18&&

520     Q: If by coordinate control you repress the transcription of one polycistronic mRNA (say because you do not need to make trp. synthetase), wouldn\'t you also repress the production of another enzyme that perhaps you need

520     A: No. All the proteins made by that operon are needed to make trp. That\'s why it\'s called the trp operon. You don\'t just need trp. synthetase to make trp -- you \r\nneed all the other enzymes in the pathway. The trp operon codes for them all -- but not for enzymes of other pathways.',NULL,1415,3,1),(##8-10-30&&

521     Q: In problem 5-1, we are asked how many molecules of ATP are formed when the ETC and oxidative phosphorlyation are uncoupled. The answer key has 4 as the answer with no explanation, but wouldn\'t you get 2 molecules out if you consider pyruvate transport (2 from glycolysis + 2GTP from Krebs - 2 to transport Pyruvate)?\r\n\r\n&&

521     A: Transport of pyruvate is not an issue. It\'s transport of reduced NAD that \"costs\" ATP. Pyruvate goes freely into mitochondria. ',0,554,2,1),(##8-10-30&&

522     Q: Problem 7-2 claims the DNA strand GTAGCCTACCCATAGG (5\'-3\') would direct RNA (5\'-3\') as GUAGCCUACCCAUAGG. Shouldn\'t it be CCUAUGGGUAGGCUAC 5\'-3\'

522     A: It says the OTHER strnd is used as template. Not the one shown. ',0,555,2,1),(##8-11-06&&

523     Q: On question 3B of the test, shouldn\'t the answer be \"none of these\" because you cannot assume that each strand will break at the same place? For every AA/TT, the DNA could break between the two pairs to create two nucleotides, between the two pairs so that there is one nucleotide (A or T) and one dinucleotide (the other A or T, plus whatever is next on the strand), or on either side to create dinucleitides of AA and TT. Thus  wouldn\'t it be impossible to have AA=TT, even though the strands have equal amounts of AA and TT.&&

523     A: The assumption here (that we made) is that you are cutting up lots of DNA molecules, all identical, from many bacteria. (Because if you grow up a batch of bacteria and isolate their DNA, that\'s what you would have. You wouldn\'t have one molecule.) In that case, when you make a mix of mono and dinucleotides, they don\'t all come from the same molecule -- they come from randomly busting up a whole bunch of identical molecules. Therefore, on average, if A is next to A a large percent of the time in the original DNA, you\'ll get a lot of AA in the dinucleotide mix, and an equal amount of TT. Almost no one got this question right (since most people assumed there was only one DNA molecule to start), so we awarded half credit if you realized the original DNA had A=T and AA=TT (or even if you thought AT = TA) because then you were thinking about the consequences of double stranded DNA structure, which was the basic point. ',0,556,2,1),(##8-11-10&&

524     Q: Why are only 31 tRNAs used when there are 64 possible codons? Are the other 33 possible combinations used for something else completley different from protein production

524     A: ALL codons are used. (Stop codons are used, but have no corresponding tRNA\'s). Both UUU and UUA are used, but same tRNA reads them. (It\'s not that UUU is used and UUA isn\'t). But why bother to have extra codons? We don\'t really know, but can guess. We assume it is easier to

read/translate a code where all codons are the same length. If codons are 2 bases long, can\'t encode 20 dif. aa. So codons need to be 3 bases long. But that provides extra\'s. Either \"the waste is worth it\" in ease of translation, or more likely, having the extra codons acts as a \"buffer\" to reduce effects of mutations (mistakes). Many mistakes in a codon --> synonomous codon or a codon coding for a similar amino acid. So it looks like there has been selection for a code that is relatively easy to translate and minimizes the effects of errors. ',0,557,3,1),(##8-11-10&&

525     Q: Does the degeneracy of the genetic code consist of two parts: (1) wobble -- the potential of a single tRNA to bind to two or more codons, snd (2) each amino acid has more than one codon that codes for it

525     A: What you have called (2) is degeneracy -- more than one codon for (most) amino acids. Wobble is a way of dealing with degeneracy -- it takes advantage of degeneracy to cut down on the number of tRNA\'s needed for accurate protein synthesis. I\'m not sure what you mean by \"source of variability.\" There is variability in base pairing (relative to standard G-C and A-U base pairing) allowed by wobble. There is variability in the codons that code for the same amino acid due to degeneracy. If this issue is still unclear, please ask again for clarification. ',0,558,3,1),(##8-11-11&&

526     Q: What is the difference between wobble and degeneracy

526     A: Degeneracy = multiple codons per amino acid. Wobble means one tRNA can pair with more than one codon (because of additional pairing options in the 3\' end of the codon). You can (in theory) have degeneracy without wobble -- see problem set 7-22. You can\'t have wobble (more than one codon read by the same tRNA) if there is no degeneracy (without messing up the code). ',0,559,3,1),(##8-11-16&&

527     Q: How many genes are there in the human genome? Also, is the average gene size (i.e. number of base pairs) the same for all organisms

527     A: The number of genes inthe human genome is estimated at 70,000 to 100,000. The number of restriction fragments typically produced by a six-cutting restriction enzyme (such as E coRI or HindIII) is 750,000. The restriction fragments are not related to genes, other than by the fact that they are both derived from the DNA of the human genome. A 6-cutter will produce fragments whose average size is ~4000 bp independent of the gene size or number. The average gene size appears to be the same in all mammals (I would guess at about 20 kb without looking it up), but different in prokaryotes such as E.coli (~ 2 kb) and simpler eukaryotic organisms such as yeast (in which most genes do not have introns). ',0,560,3,1),(##8-11-17&&

528     Q: Gene cloning and PCR seem like similar technologies to achieve specific DNA sequence amplification. When is one technology employed over another? Also, is DNA cloning (a.k.a gene cloning) synonomous with DNA culturing

528     A: Gene cloning and PCR are indeed both able to give you large amounts of a specific DNA sequence. Their use would depend on the situation. Fragments larger than 1 kb are more problematic to amplify by PCR; cloning in bacteria on the other hand can accommodate up to 10 kb in plasmids and up to 200 kb in viruses. The colony hybridization used to identify the growth hormone gene that was used as an example in lecture 16 is convenient to carry out on thousands of bacterial colonies; it would be difficult to generate thousands of discrete spots of pure DNA fragment s using PCR. On the other hand, the search for VNTRs and RFLPs is easily carried out by PCR once you know the sequence in the

region in question. Although this problem was used to illustrate Southern blotting in lecture 17, in reality this type of analysis is now carried out more simply by PCR, and no hybridization to probes is necessary. ',0,561,3,1),(##8-11-18&&

529    Q: If the same tRNA can read more than 1 codon, ie. GGG and GGA, then which anticodon does the tRNA contain?? Will it be 3\' CCC 5\' or 3\' CCU 5\'

529    A: I think the exact rules depend on the organism. The usual ones are in Becker, table 18-2. U in tRNA can pair with A or G. ',0,562,3,1),(##8-11-19&&

530    Q: What does it mean when E. coli is lys+ or ser-, or anything similar to that? And what are progeny

530    A: Lys + usually means able to make its own lysine. Ser- usually means unable to make its own serine. lac+ usually means able to break down lactose. In general, the \"+\" means able to synthesize and/or breakdown (depending on context) some compound; the \"-\" means unable to carry out the necessary steps in metabolism to use and/or make the substance. progeny = offspring or descendants. ',0,563,3,1),(##8-11-19&&

531    Q: Regarding problem 7-5 (C), why are two different tRNAs needed for serine UCX? Shouldn\'t the 3\'AGX5\' anticodon suffice for any given UCX serine

531    A: Wobble allows limited deviations from the G only with C and U only with A, but not any old base pair will do. Only certain combinations are allowed, and as a result, no tRNA can read more than 3 different codons. So it takes two tRNA\'s to read all 4 UCX codons. ',0,564,3,1),(##8-11-23&&

532    Q: Can one \"take\" the DNA from two human female eggs and make a human

532    A: In theory, this should work and you would get an XX female. However because of the phenomenon known as imprinting, it won\'t work. Some genes must come from your dad in order to be transcribed, and some must come from your mom. The genes are marked, probably by methylation, so the zygote \"knows\" which parent they came from. If all the genes are from mom or from dad the embryo is abnormal. (We know about this from experiments in animals.) ',0,565,3,1),(##8-12-01&&

533    Q: In the notes for Lecture 16, how do we interpret the diagram illustrating the fragments produced by restriction enzymes

533    A: The top part of the diagram shows the fragments produced by the restriction enzymes the approximate cutting sites for EcoRI and the bottom the approximate sites for HindIII.',0,567,3,1),(##8-12-01&&

534    Q: In problem 12-7 (E), why is it that only the copy of the operon on the plasmid is constitutive

534    A: The repressor protein from the good R gene on the plasmid will work on the good operator on the chromosome. So the chromosomal operon will not remain constitutive. ',0,568,3,1),(##8-12-01&&

535    Q: Purves defines exon in the glossary as coding for a polypeptide, and intron as not coding for a polypeptide. What about the parts of the gene that code for things other than polypeptides (eg. tRNA)? Are they introns, exons, or neither

535     A: The definition of an exon given in the glossary of the Purves text is incorrect. The definition given in the text of Purves (p. 314, col. 2, para.1) is correct. ',0,570,3,1),(##8-12-03&&

536     Q: What does the \"coding region\" refer to

536     A: Usually the region of mRNA that is translated, NOT the region of DNA that is transcribed. But \"coding\" can mean coding for mRNA OR coding for protein. Usually it means the second. ',0,571,3,1),(##8-12-03&&

537     Q: What is the difference between a gamete and a spore

537     A: Gamete is specialized for fusion/fertilization. It never goes through meiosis. A spore is generally not specialized, but goes through mitosis and then some of the offspring are then specialized for various jobs. ',0,572,3,1),(##8-12-10&&

538     Q: With regard to the posted solution to problem 4 on the exam, shouldn\'t the translated initiator codon be located in the exon 3 region? Assuming that the DNA sequence on the exam is written from left to right, 5\' to 3\', a 3\' to 5\' transcribed mRNA region is read from left to right also. Since translation runs in the same direction as transcription, namely 5\'(right side of strand) to 3\' (left side) for this mRNA strand, the first encountered translated region is located at exon 3; therefore, the initiator codon should lie there.&&

538     A: DNA is normally double-stranded, and the gene map represents such double stranded DNA (with restriction sites). The top strand is the \"sense\" strand (= to the mRNA sequence). It is the bottom strand (3\' to 5\' left to right) that is the transcribed strand. Thus exon 1 is 5\'-most in the resulting transcript and in the mRNA. ',0,573,3,1),(##9-09-10&&

539     Q: From what I read, the macromolecules in a cell do not apparently have K and Mg as component elements in their monomers (sugars,lipids, amino acids, nucleic acids). What are these ions needed for

539     A: Mg++ is indeed a part of many proteins, intimately bound although not covalently, as we shall see. It is also bound to many phopsphate-containing molecules (e.g., RNA) helping to keep them in correct 3-dimensional form. Potassium is usually not so stronglty a part of any molecule, but is necseeary to maintain the correct osmotic balance between the inside and outside of a cell, and as a participant in some chemical reactions. ',0,574,1,1),(##9-09-10&&

540     Q: Are algae prokaryotic or eukaryotic

540     A: Algae are eukaryotic (I think I may have mistakenly put them into the prokayotic row in class); some species are unicellular others are multicellular. They come in different colors. Cyanobacteria are prokaryotic and unicellular. Many algae and the cyanobacteria can carry out photosynthesis. ',0,575,1,1),(##9-09-10&&

541     Q: What tells the DNA in E coli\'s nucleus to replicate? Does the impetus to replicate come from its chemical structure, or from its nucleic environment

541     A: The control of replication is still not understood. It is an intensely studied question in eukaryotes (esp. mammals; and yeast, as a model) as this process is intimately related to the

mechanism of tumor formation. It is less studied in E. coli these days, as far as I know. ',0,576,2,1),(##9-09-10&&

542    Q: I don\'t see why an acid is not always charged.&&

542    A: An acid is always in dynamic equilibrium between its charged and uncharged form$ a carboxylic acid:<br><br>\r\nRCOOH <---> RCOO- + H+<br><br>\r\nwith interconversions occuring continuously. A \"strong\" acid will be mostly dissociated at pH7 where the hydrogen ion concen$ 10-7 M. But if if you increase the [H+], say by adding a lot of an even strong$ like hydrochloric acid, HCl, and you raise the [H+] to, say, 10-2 M (pH2), then$ equilibrium reaction depicted above will be driven to the left by mass action. $ these conditions most of the carboxylic acid will be in its associated (protona$ and uncharged. At some pH, the amount of charged and uncharged forms will be j$ equal. One can gauge the strength of an acid by this pH, the pH which results $ concentrations of protonated and unprotonated forms. For many carboxylate group$ molecules we will be discussing, this pH (called pK) is around 3. ',0,577,1,1),(##9-09-13&&

543    Q: Are hydrophobic forces classified as Van der Waals bonds? What sorts of molecular interactions are involved when octane is placed in water

543    A: The hydrophobic force squeezes the octanes together, it does nothing TO the water, it is is force created BY the water. By cuddling up to each other, the octanes present a smaller surface area to the surrounding water molecules, and so more water molecules are free to be disorganized. Since disorganization (maximum entropy) is the preferred state, the octane stay together. Van der Waals bonding is not related to the hydrophobic forces. The van der Waals bonds are created when two atoms come into close proximity, as one atom\'s electron cloud induces an opposite charge on the other atom\'s periphery. The octane molecules that are pushed together by hydrophobic forces will interact with each other through van der Waals bonding, just as any two molecules that are closely positioned will do. ',0,578,1,1),(##9-09-15&&

544    Q: Are there specific enzymes for bonding alpha glucoses to beta glucoses, or are such combinations of alpha and beta linkages caused solely by the recyclization of the C4 glucose

544    A: I am not aware of a polysaccharide that alternates alpha and beta linkages, but I see no reason why one could not exist. I did find one that alternates the carbon\'s used in the glycosidic bond: b1,4 -- b1,3 -- b1,4 -- b1,3 etc. That is hyaluronic acid, slippery stuff in bone joints, and inside the eye. ',0,579,1,1),(##9-09-15&&

545    Q: How does one differentiate between alpha and beta glycosidic bonds

545    A: It is hard to tell. You need to look at the anomeric carbon that is contributing to the bond (e.g., C1 of glucose). Look at its position relative to the other hydroxyls on the ring. This is easy enough if the sugaar is drawn right side up as we have done in class. But occasionally the sugar is turned around in a figure. For glucose drawn the usual right side up way, if the OH of the C1 carbon is pointing down from a flat ring or out from a chair, then it\'s alpha. If its pointing up from a flat ring or out from a chair, then it\'s beta. ',0,580,1,1),(##9-09-15&&

546    Q: When an alpha-D-glucose forms a bond with a beta-D-fructose (p.65 of Becker), why is the bond labeled an alpha glycosidic bond? Is the bond between an alpha and a beta always an alpha bond

546     A: In most bondings between two sugars, only one partner is using the anomeric carbon. And it is only the anomeric carbon that is given the alternative name of alpha or beta. The othe carbon's OHs do have a positional status (above or below the ring, or axial or equatorial to the chair) but they are fixed for any given sugar with a given English name. So in the examples on page 65 of Becker, the top molecule in Fig. 3-20 can be called an alpha glycosidic bond, since the right side sugar is not contributing an anomeric carbon and has no Greek letter associated with it. In the case of sucrose, the bottom molecule in the figure, the anomeric carbon of each molecule is involved in the glycosidic bond. It's the alpha of C1 in glucose bonded to the beta of C2 in fructose. So this bond should be more properly called an alpha 1 - beta 2 glycosidic bond, as it is in some texts (Loewy et al., Cell Structure and Function, Saunders, 3rd Ed.). So Becker is a bit misleading here in his labeling. ',0,581,1,1),(##9-09-17&&

547     Q: Why are unsaturated fats recommended as more healthy than saturated fats

547     A: (This answer from Dr. Judy Gibber, who teaches Physiology here):\r\nThe major reason for the recommendation is the epidemiological data. In the 1950's Ancel Keys' studied people's diet and disease in 7 different countries, and found that people in the US with animal-rich diets had more atherosclerosis than in Japan and Yugoslavia where less animal fat was eaten. Many more carefully designed studies have confirmed this general relationship.\r\nThe understanding of the mechanism by which saturated fats have this effect keeps changing, since there's a lot that's not known about the mechanism of atherosclerosis in general.<br><br>\r\nIn the textbooks, the explanations focus on how saturated fatty acids increase the blood levels of LDL, which is the form in which fats are deposited in the walls of blood vessels. But since many people now consider athero to be an inflammatory disease, there have been studies showing the effects of the fatty acids on various steps in the inflammatory process.\r\nPeople with a variety of commercial interests may dispense nutritional advice! But if you use the term \"nutritionists\" to refer to professionals with training in the field, then I'd say that nutritionists ARE biologists. ',0,582,1,1),(##9-09-17&&

548     Q: Does tyrosine have a polar or nonpolar side group

548     A: I think it has BOTH characteristics, both of which might be exploited by a given protein in using tyrosine to help it fold in a certain way. Thus the body of the ring could be attracted to other hydrophobic groups, whereas the hydroxyl can interact with polar groups. Maybe even both at once. Nature may not agree with our tendency to make exact categories. ',0,583,1,1),(##9-09-17&&

549     Q: What exactly accounts for the behavior of an individual amino acid in paper chromatography

549     A: An amino acid always has two polar groups (the amino and carboxyl), a single H atom that is always nonpolar, and a variable side chain. Thus it is the composite polarity of all four groups connected to the alpha-carbon that counts for its behavior in paper chromatography.',0,584,1,1),(##9-09-17&&

550     Q: For the two acidic amino acids (asp and glu), why can't a peptide bond be mistakenly formed between its non-alpha carboxylic group and another AA's amino group? Likewise, can a peptide bond form at the non-alpha amino group of lysine

550     A: The process of polypeptide synthesis involves a complex and intricate orchestration of events, conducted by scores of proteins and RNA molecules. You will learn about it in a few weeks. Using this complex apparatus, proteins are put together solely by their alpha carboxyls and aminos.

That said, one can find examples of unusual peptide bonds of the type you propose tacked onto side chains here and there in nature. ',0,585,1,1),(##9-09-17&&

551     Q: Is Mg also a prostheic group like Fe or Zn? What does it do exactly? In the minimal growth medium you described for E-Coli, it seems Mg is needed substantially more than the trace amount of Fe and Zn. Does this mean Mg occurs a lot more often in protein than Fe or Zn

551     A: We will see examples of how prosthetic groups sit in proteins. Mg can be a prothetic group as can Fe and Zn and Co etc. Mg is also probably important for influencing the structure of small molecules such as nucleotides, where it can act as a specific counter ion of phosphate groups. And perhaps in DNA and RNA as well. ',0,586,1,1),(##9-09-17&&

552     Q: Why 20 amino acids and not some other number?\r\nDid someone really sequence all the different proteins found in all the organisms on earth and conclude there is no 21st amino acid

552     A: 20 seems a good number to me, but maybe 40 would have given us more complexity and subtlety :-). Yes, proteins have been sequenced from hundreds of organisms (not ALL!) and 20 is the number found. The genetic code codes only for these 20 (with some minor excptions, as usual, but these are variations on a theme) as we will see. ',0,587,1,1),(##9-09-20&&

553     Q: Is a glycosidic bond the same thing as an ester bond

553     A: No, a glycosidic bond is a C-O-C bond where the neighboring C\'s have OHs. It is formed by withdrawal of a molecule of water from two carboins carrying hydroxyl groups. An (carboxylic acid) ester has a carbonyl component (C=O), and has the structure:\r\n-CO-O-C (where CO means C=O). It is formed by withdrawal of water from two carbons, one of which has a hydroxyl and one of which is a carboxylic acid. ',0,588,1,1),(##9-09-20&&

554     Q: Does a molecule with a carboxyl group count as an alcohol? Is any molecule with an hydroxyl group an acid, since it loses the hydrogen

554     A: No, a carboxyl group does not count as a hydroxyl. An OH is a part of its structure, but the combination of =O and OH attached to a single carbon changes the characer of the functional group, such that the H of the carboxyl readily ionizes in water. Thus it is an ACID. The H of the simple hydroxyl almost never ionizes, it does not lose its hydrogen. It is polar, but not an acid. ',0,589,1,1),(##9-09-20&&

555     Q: Do anomeric carbons have to have a carbonyl group?\r\n&&

555     A: The anomeric C of sugars starts out as a carbonyl in the straight chain form. ',0,590,1,1),(##9-09-20&&

556     Q: Can steroids polymerize

556     A: No. A steroid is defined as a small molecule that is <i>not</i> a monomer. Despite being small, they do not go on to polymerize.',0,591,1,1),(##9-09-20&&

557     Q: H2PO4(-) is the phosphate group on a phospholipid. Does it only lose one hydrogen because the first hydrogen is the easiest to leave

557    A: The first 2 of the 3 OHs of phosphate are highly acdic and will dissociate the H in water. Even the third loses its H about half the time. ',0,592,1,1),(##9-09-20&&

558    Q: Why isn\'t an acid always charged in aqueous solution

558    A: An acid may not be charged if the hydrogen ion concentration is sufficiently high to drive the association-dissociation reaction (protonation) far towards association, by mass action. For many carboxylic acids, this associaiton will predominate at pH of 2 or lower. ',0,593,1,1),(##9-09-20&&

559    Q: Figure 3.15 in the Purves book depicting peptide bond formation seems to conflict with Fig 3.3 in the Becker book. Specifically, why does the amino group pictured in the Purves book have only 2 hydrogens bonded in the free amino group

559    A: Purves (Fig. 3.15) writes the free amino acid prior to peptide bond formation as having an amino group with 2 H\'s, and uncharged, as it might occur in the solid powder. Becker writes it as it exists predominantly in solution, with 3 H\'s and a positive charge, having taken up a hydrogen ion from water. He is consistent in writing the carboxyl as also ionized, with no H in sight. Either way, you can extract 2 H\'s and and O, forming a free water molecule and the peptide bond. Although the ionized form is more accurate, the withdrawal of water is easier to see in the un-ionized form, as I wrote in class. The result is the same. The actual mechanism of peptide bond formation is actually much more complicated than this, as you will soon learn. These reaction schemes merely summarize the molecules that you start with and end up with. ',0,594,1,1),(##9-09-20&&

560    Q: Since the carboxyl end (COOH) of a fatty acid is polar, and this end combines with glycerol to form a glyceride, is this end of glyceride polar

560    A: Yes, the glyceride part of a fat is polar, but the non-polar character predominates. Thus the fat globules, insoluble, floating in the chicken soup. Putting more polarity into that end with the phosphoglycerides, leads to more interesting effects (phospholipid bilayer formation). ',0,595,1,1),(##9-09-20&&

561    Q: What is the difference between an anomer and an isomer

561    A: Any molecule with an asymmetric carbon can form a stereoisomer (sometimes referred to as an optical isomer). Sugars that can form those special isomers that put a hydroxyl either above or below the hydrogen sharing that carbon are anomers of each other. Note that the carbon involved in anomer formation is asymmetric (has 4 different groups attached) after cyclization but was not asymmetric before cyclization, when it was a carbonyl carbon. ',0,596,1,1),(##9-09-22&&

562    Q: If hydrogen bonds may be readily broken at temperatures between 0 and 100 degrees, why aren\'t a lot of hydrogen bonds broken in our bodies (at 98 degrees)

562    A: Individual H-bonds <i>are</i> being broken (and re-formed) all the time, in the water present in your body, for example. But when these bonds are broken in a molecule like the alpha-helix of a polypeptide, at any moment in time there are still many others that continue to hold the alpha helix in its helical conformation. That is, at any given moment in time, only a minority of the H-bonds will be broken, while the rest hold the structure together. At the next moment it is another set that is doing the breaking and while others are doing the holding. Thus an alpha helix of some minimum length is stable at body temperature. ',0,597,1,1),(##9-09-22&&

563    Q: What is the difference between a carbohydrate and a polyhydroxy

563    A: Usually one C=O bond present in the carbohydrate. General formula CnH2nOn. For a poly-alcohol (e.g., glycerol) the general formula would be CnH2n+2On. ',0,598,1,1),(##9-09-22&&

564    Q: What is D-Glucose

564    A: D-glucose is themolecule we have been discussingin class, and is the abundant form of th esugar. L-glucose is a stereoisomer of D-glucose, having the opposite orientation of the hydroxyl groups on all 4 asymmetric carbons of the sugar (2and 5). ',0,599,1,1),(##9-09-22&&

565    Q: How do keto-sugars form a ring

565    A: In fructose, the OH on C5 attacks the C=O double bond on C2 to form a 5-membered ring (as shown on the carbohydrates handout). ',0,600,1,1),(##9-09-22&&

566    Q: Is dehydration the same as condensation

566    A: I believe a condensation reaction just puts two molecules together, necessarily via the withdrawal of water. So dehydration is a condensation, but not necessarily the other way around. ',0,601,1,1),(##9-09-22&&

567    Q: Why don\'t the hydroxyls on lipids make lipids polar? (i.e. cortisol, testosterone?)&&

567    A: The same could be asked of a fat. There\'s simply not enough polarity at one end to counteract the overwhelming majority of the atoms, which are plain hydrocarbon. The polarity of the OH group in cholesterol is nonetheless utilized, as it helps orient the cholesterol molecules that reside in the cell membrane, attaching them to the inside of the charged head of the phospholipid. ',0,602,1,1),(##9-09-22&&

568    Q: Do all sugars have an anomeric carbon? How can we tell on a sugar other than glucose

568    A: Any sugar that can cyclize by opening up a carbonyl group will have that carbon as anomeric. A very small sugar (triose) cannot cyclize (too small) despite the presence of a carbonyl, such as the aldehyde on glyceraldehyde (consult the index of your text to see the structure of glyceraldehyde). ',0,603,1,1),(##9-09-22&&

569    Q: Problem 2-5C: \"Would urea reduce the molecular weight of a protein composed of more than one polypeptide chains?\" Why is this so?\r\n&&

569    A: Say the MW of a tetrameric protein (i.e., a protein that has quaternary structure) is 64000 (e.g., Hb). Treat with urea. The 4 monomer polypeptides come apart. Now if you determine the MW (of the subunits), it is 16000. A protein is defined as the whole shebang, all the subunits together if there is quaternary structure. All this assumes no disulfides between the subunits. ',0,604,1,1),(##9-09-22&&

570    Q: On the functional group handout from 9/9, next to phenol, there is a parenthesis with an O crossed out connected to an -OH. What does this mean

570     A: The O with the line through it denotes the Greek letter phi, which has been adopted by organic chemists as a shorthand for the phenyl group, C6H5- . ',0,605,1,1),(##9-09-24&&

571     Q: When a catalyst increases the rate of reaction, does it increase the proportion of molecules that have the required activation energy to react via collision, or does it act to break and reform bonds faster

571     A: A catalyst can facilitate the breakage of bonds, as in the case where the bonds between H\'s are weakened upon binding to Pt. But once the bonds are broken, the catalyst cannot influence the probability that they will reform to make products vs. reforming to remake the reactants. That ratio is governed by the relative strengths (stabilities) of the bonds in the two alternative structures (reactants and products), which ultimately are in equilibrium with each other. The direction of the reaction is determined by this equilibrium ratio, as well as the relative concentrations of the reactants and products that you are starting from. We will discuss how energy changes determine directions in a reaction in more quantitative detail when we get to energy metabolism, right after enzymes. ',0,606,1,1),(##9-09-25&&

572     Q: Isn\'t it true that denaturing only breaks weak bonds (ie- H bonds, NOT covalent ones)

572     A: \"Denaturing agents\" break only weak bonds, not covalent bonds. Some might consider mercaptoethanol to be an aid in denaturation and so classify it as a denaturing agent. In that exceptional case, a covalent bond would be broken. But never a peptide bond. ',0,607,1,1),(##9-09-25&&

573     Q: Purves says that cysteine is very hydrophobic when not involved in disulfide bonds. But Becker puts it in the uncharged polar category, as it seems it should be. Which is more correct

573     A: Yes, cysteine is a problem with respect to polarity classification. The electronegativity of sulfur is greater than that of hydrogen, but not by much, so the -SH group should be only weakly polar. This weak polarity is utilized in protein folding, although usually in the interior of the protein, where the more strongly polar water molecules are not competing for the -SH\'s binding partner. So both characterizations are partially correct. ',0,608,1,1),(##9-09-26&&

574     Q: In problem 1-23C, I thought that alpha bonds in glucose made starch, but the answers to the problem set says that the beta linkage will make starch.&&

574     A: The beta bonds in glucose make cellulose when they are connected to the #4 carbon on other glucose molecules because they are both equatorial and so can connect in a (more or less) straight manner. The prinicipal is in the connection, either straight (equatorial-equatorial, cellulose-like) or with a bend (equatorial-axial, starch-like). You must understand this principal to solve the problem and not just associate words (alpha = starch). The beta bond from glucose in the poly-lactose polymer concocted in this problem is not from C1 of glucose to C4 of glucose as in a starch, but is from C1 of glucose to C4 of galactose. The C4 of galactose is axial, so you get an equatorial (C1 of beta glucose) to axial (C4 of galactose) bond, which puts the connected chairs at a severe angle to each other as in starch and not straight as in cellulose. ',0,609,1,1),(##9-09-29&&

575     Q: How do fingerprinting and paper chromatography differ in what you can learn about a polypeptide?\r\n&&

575     A: Fingerprinting itself does not provide the sequence of a polypeptide. It breaks the polypeptide into smaller peptides which are then separated by a combination of paper electrophoresis and paper chromatography. It provides a relatively quick way to identify a protein, since the pattern of spots produced is unique for each protein. Paper chomatography itself can be used to separate amino acids, small peptides, sugars, and many other small organic molecules. ',0,610,1,1),(##9-09-29&&

576     Q: Could you please explain the Svedbergs equation (Lecture #5)

576     A: The Svedberg unit is defined as $V/w2r$, where $w2$ is the square of the angular velocity (e.g., ~rpms), r is the distance from the center of rotation and V is the constant velocity at which the particle is sedimenting. This value will be higher for larger (higher MW) proteins. So a protein that yields an S value of $200 \times 10^{-13}$ sec will be much larger than a protein with an S value of $20 \times 10^{-13}$ sec. A Svedberg unit is defined as $10^{-13}$ sec, so a protein\'s sedimentation behavior can be more conveniently referred to as 200S or 20S. The sedimentation will also be influenced by shape if the protein is not roughly spherical: $S = M/fo$ where fo is the frictional coefficient and M is the molecular weight. This is all more than you need to know about Svedbergs. Just remember that a high S value means rapid sedimentation and a low S value means slow sedimentation and the rate of sedimentation is proportional to the MW and inversely proportional to the coefficient of friction. ',0,611,1,1),(##9-09-29&&

577     Q: What does one need to know the ring and chain formations of ribose, mannose, galactose, fructose, deoxyribose, etc.

577     A: You do not need to memorize any organic structures to bring up when given the English name (e.g., write the structure of mannose). Nor do you need to know the English name if presented with a stucture (e.g., is $H2NCH2COOH$ glycine or alanine?). You do need to memorize the functional groups and know their properties (polarity, hydrophobicity, charged or not, sulfhydryl properties, etc.). You may have to manipulate a structure if given directions (e.g., what would happen if the OH were on the opposite side .. etc.) ',0,612,1,1),(##9-09-29&&

578     Q: How on earth do you reassemble the components in problem 2-9(D)

578     A: This is difficult to answer by email, as I can\'t properly draw. You look for overlaps between 2 peptides, each produced by a different enzymatic cleavage. For example, start with 2-9Dv: ala-ala-val-lys . Now go to the upper group of 4 and look for part of that sequence. See ii, ala-val-lys, but we already know that. How about 2-9Biii, ser-lys-ala. That final ala must be the same ala as the one that starts the ala-ala-val-lys, because all of the other ala\'s in the B set are followed by amino acids other than ala. So the ala-ala part of ala-ala-val-lys must have come from a peptide region that was originally ser-lys-ala-ala-val-lys. ETC. ETC. ',0,613,1,1),(##9-09-29&&

579     Q: Does \"large pores\" in problem 2-9(E) mean that shape won\'t play a part in this type of gel electrophoresis

579     A: Yes. Neither MW nor shape, as the large pores present no impediment to migration. ',0,614,1,1),(##9-09-29&&

580     Q: In problem 2-14, how can an enzyme retain it\'s catalytic activity after a peptide bond has been cleaved

580    A: If the rest of the interaction between the side groups that hold the native protein in its tertiary structrue are strong enough, the cleavage may not necessarily affect the overall conformation of the protein. In a way, in such a case, quaternary structure has been added to the protein by this experimental intervention. If the peptide bond that has been cleaved is not in the active site, then the protein could retain all functions. One can readily imagine the opposite as well, where breakage of a single peptide bond causes the folded polypeptide to fall apart into 2 pieces. ',0,615,1,1),(##9-09-29&&

581    Q: In problem set 3R-1B, won\'t the second molecule, which has more carbon atoms, move more slowly in paper chromatography than the first molecule and therefore have a smaller Rf

581    A: Size is not a factor in paper chromatography. The distance between cellulose fibers is huge compared to the molecules. ',0,616,1,1),(##9-09-30&&

582    Q: For the 1997 exam, question 1D asks \"suppose cellulose is hydrolyzed using an enzyme that specifically cleaves the 1,4 gycosidic bond. The products of the hydrolysis will have their carbon #1 hydroxyl in the following conformations?\" Why does the hydroxyl exist in both the alpha and beta positions

582    A: The straight chain and ring forms are in equilibrium: that means they are constantly changing from one form to the other, but the net amount (concentratuion) of any particular form is not changing (at equilibrium). A free ring will be forming the straight chain, and when that straight chain reforms a ring, it will do so as either alpha or beta. So all three forms of the free sugar are present The internal glucose residues in the polysaccharide cannot open into the straight chain form because the C1 oxygen is tied up in the glycosidic bond. (One terminal residue has a free C1 OH, and it should be in equilibrium with the straight chain and the alterntative ring form.) ',0,617,1,1),(##9-10-03&&

583    Q: Can I say that a competitive inhibitor binds to an enzyme\'s active site about as well as the substrate

583    A: No. A competitive inhibitor will have its own intrinsic affinity for the substrate binding site, which may be better (tighter) or worse than that of the substrate. Even much better or much worse. ',0,618,1,1),(##9-10-03&&

584    Q: Can I say a noncompetitive inhibitor binds to the enzyme not only at a different site, but with a much better binding ability than the substrate? So much better that the binding of NCI to enzyme is almost \"permanent\" compared to substrate binding

584    A: No. There is no relationship between the binding affinity of a non-competitive inhibitor (NCI) and that ofthe substrate. In most cases the binding of the NCI by the enzyme is not \"permanent\" but rather the enzyme is in equilibrium between the bound and free state. The lecture diagram showing some enzyme molecules with the NCI bound is meant to represent only a snapshot of the situation at a given moment in time,or an average of many such snapshots. In reality the NCI is continually binding to the enzyme and dissociating from it. But for the period of time the NCI is bound (which can be considerable), the enzyme cannot function. ',0,619,1,1),(##9-10-03&&

585    Q: With an allosteric inhibitor can I say that the apparent Km will be the average of the weakened enzyme molecules and those remaining uninhibited, and thus the apparent Km will increase

585    A: From its remote binding site, an AI could affect the shape of the substrate binding site, thus changing the binding affinity andthe apparent Km. In a mixture of inhibited and non-inhibited enzyme molecules, I would expect the Vo vs. S curve to be complex (biphasic?) and not yield an \"average\" Km. ',0,620,1,1),(##9-10-03&&

586    Q: If, after an allosteric inhibitor binds to the enzyme on the allosteric site, the active site of the enzyme is so distorted that S can not bind, then it effectively serves as a \"competitive\" inhibitor.Will it therefore only affect Km but not Vmax

586    A: It affects Km in a manner different from that of a competitive inhibitor, since it binds at a different site. It is changing the Km of the enzyme by \"changing\" the enzyme. Actually it brings about this change by stabilizing a natural alternative inactive state of the enzyme, see Purves tet, p. 129-130. ',0,621,1,1),(##9-10-05&&

587    Q: I thought noncompetitive inhibitors don\'t change the active site of an enzyme, meaning that the substrate still binds but can\'t do anything. However, in the picture link from Purves (figure 19.C), the noncompetitive inhibitor changes the enzyme\'s conformation so that it can\'t bind the substrate. Does the substrate bind or not

587    A: Purves says the non-competitive inhibitor affects the active site, but not the substrate binding site (p.127, last paragraph). The picture from the publisher\'s Web site differs from that in the current Purves (4th Ed.) text Fig. 6.19C. I believe the text paints a less confusing picture and have removed the linked picture. The active site would comprise the the substrate binding site plus those elements that facilitate the reaction, that help catayze the reaction, that produce a transition state. The catalytic activity is inhibited, but substrate binding per se is not. The important thing is that substrates do not compete with a noncompetitive inhibitor, and that non-competitive inhibitors do not resemble substrates. ',0,622,1,1),(##9-10-05&&

588    Q: If there is no oxygen present, and if the Krebs cycle does not need O2, then how would the pyruvate know to go to an anaerobic process rather than the Krebs cycle

588    A: Many of the glycolytic enzymes and the Krebs Cycle enzymes are regulated by allosteric regulation. Several steps in the Krebs Cycle are inhibited by NADH2, including pyruvate dehydrogenase, the enzyme that takes pyruvate to the Krebs Cycle. So when the oxygen supply decreases, NADH2 begins to accumulate, and the pyruvate is shunted to the lactate dehydrogenas reaction. The system also monitors closely the levels of ATP, ADP, acetyl-CoA, citrate, etc. to balance the activity of the two pathways and to regulate glucose flow through them. ',0,623,2,1),(##9-10-06&&

589    Q: Why can\'t noncompetitive inhibitors be \"swamped out\" by excess substrate

589    A: Because they do no compete with substrate for binding. They are essentially oblivious to the substrate concentration. ',0,624,1,1),(##9-10-07&&

590    Q: What happens to the electrons in reaction 6 on the \"glycolytic pathway\" handout

590    A: Two hydrogen atoms have been withdrawn from the two reactants. The hydrogens leave as hydrogen atoms, not hydrogen ions; thus they leave with their electrons. So 1,3-diPGA has 2 less electrons that do the two reactants, G3P and PO4. These electrons are accepted by NAD. One of the

electrons is divorced from its proton in the process. That proton (hydrogen ion) joins the pool of H+ ions in the pH 7 milieu of the cell. ',0,625,2,1),(##9-10-08&&

591    Q: In problem 3-1, a series of values for substrate concentration and rates with and without inhibitor are provided. The first question asks for a value for Vmax and Km with and without the inhibitor. Should one always take the fastest velocity presented

591    A: You should get the same answer whether you take the top value (aprox.) as Vmax or whether you take a few intermediate values for V and plug into the formula. (We tried to set it up so that was true, as it should be.) Note the problem says you can do it without a calculator -- we want you to think about the meaning of Vmax, Km etc. and not just plug in. If you can\'t make it come out the same both ways, come see me and we\'ll go over it. It doesn\'t always work to take the top value measured as equal to Vmax -- it works here because you can see that V is leveling off -- adding more substrate fails to make much difference in V. Therefore you are not in the linear part of the curve (V proportional to S) but in the flattened out part where V is independent of S since enzyme is saturated. ',0,626,2,1),(##9-10-10&&

592    Q: In the problems 3-1 B and C, wow can I tell if the inhibitor is competitive or not

592    A: The (apparent) Km measured in a plot of Vo vs. S in the presence of the inhibitor will be higher than in the absence of the inhibitor. A non-competitive inhibitor will have no effect on Km measurement. A noncompetitive inhibitor will decrease the Vmax of a reaction in a Vo vs, S experiment. A competitive inhibitor will not do this. The structure of a competitive inhibitor will resemble that of the substrate. A non-competitive inhibitor need have no such resemblance, and usually does not resemble the substrate. ',0,627,1,1),(##9-10-10&&

593    Q: If lactate is accumulated in our muscle when we work too hard, how does it change or leave the muscle later? I heard too much lactate build up is a toxin, is it

593    A: It goes to the liver where it is converted back to glucose. ',0,628,2,1),(##9-10-12&&

594    Q: Becker points that life would be impossible if all reactions would go to equilibrium. What is an example that validates this statement, and what is the difference between steady state and equilibrium

594    A: Life is in a constant state of flux because it takes a lot of energy to maintain. A lot of energy must be constantly put in in order to grow, to construct complex structures out of simple ones (e.g., E. coli cells out of glucose) and even to maintain a living complex structure (e.g., muscles moving to obtain food, proteins being broken down to amino acids and new proteins resynthesisized). At equilibrium, no energy is being put in, being used, because at equilibrium there is, by definition, no change in free energy taking place (delta G = 0). At equilibrium, complex structures would represent only a tiny percentage of the material present with the great majority of atoms being in simple stable compounds such as CO2 and H2O. So to maintain and expand these complex molecules that are characteristic of life, we need a constant great input of energy. For us, this is in the form of the chemical energy in food. For the planet as a whole it is the from our constant bombardment with photons from the sun. All living organisms maintain a steady state of atoms flowing in to the system and atoms flowing out while energy is being consumed. As an example of steady state on an organismic basis, we can consider a human being being maintained alive even as its atoms are changing and great amounts of glucose and other energy sources are being consumed and CO2 being

released. At a microscopic chemical level, we can observe a constant amount of ATP per cell in an exponentially growing culture of E. coli, as great amounts of ATP are being produced by respiration and great amount expended in the synthesis of proteins and nucleic acids, etc. The ATP is constant in its steady state, but its concentration bears no relation to its equilibirum concentration, with ADP + Pi, for example. ',0,629,1,1),(##9-10-12&&

595     Q: Where are the electrons coming from in the reaction at the beginning of Krebs Cycle, where pyruvate is oxidized by NAD to form acetyl-CoA and CO2

595     A: The electrons are difficult to follow here, as this is an extremely complicated set of rections; understanding the mechanism is not the aim in our course.<br><br>\r\n\r\nThe enzyme pyruvate dehydrogenase contains about 42 subunits, and the overall reaction takes place in several steps with the involvement of 2 more vitamin cofactors (thiamin pyrophosphate and lipoic acid) in addition to CoA. The mechanism involves the addition and then subtraction of a molecule of water. One simplified way of looking at it in order to see some electrons being extracted is to imagine the CO2 of the caroxyl group extracted (no oxidation) , leaving CH3-CHO. Now add water across the aldehyde C=O double bond to get CH3-CH(OH)2. Now remove 2 hydrogen atoms with their electrons, one from the C and one from one of the OH\'s. This is the oxidation, and leaves you with CH3-COOH, acetic acid (equivalent to acetyl-CoA, oxidation--wise). Look in the index of any biochemistry text for pyruvate dehydrogenase to find the actual details of the reaction. ',0,630,2,1),(##9-10-12&&

596     Q: The Energy-level handout shows that reactions 1, 3, 7, and 10 of glycolysis are favorable,while reactions 2, 4, 5, 6, 8, and 9 are unfavorable. On Purves fig 7-7 , rxn\'s 1-4 are unfavorable and rxn\'s 5-10 are favorable. Is the chart in Purves just a simplification

596     A: I find the Purves chart confusing (Fig. 7.7 on the Web, Fig. 7.12 int he text). First. he refers to delta G, not delta Go. One advantage of using delta Go\'s for comparisons is that we will all be on the same page (standard conditions) even if this is not the condition in the cell. I prefer to use delta Go for comparisons of reactions, and then worry about cell conditions. Purves takes the other (valid) attitude that one should consider the cell conditions from the start, and not bother with the delta Go\'s. However, I have no idea what conditions he is using for Q in the equation for delta, so cannot easily see if there is any discerpancy between our numbers (if I would want to). Second, Purves considers just parts of the reactions, not always taking into account all of the reactants in a coupled reaction. So his reaction 6 (the oxidation) has a tremendous drop in free energy associated with it, whereas my characterization of that reaction shows a modest absorption of free energy. The difference here is that Purves does not take into account the free energy absorbed in reducing 2 NAD\'s to 2 NADH2\'s (106 kcal/mole). His way of looking at it has the advantage of showing the great amount of free energy that is being released from glucose at this step. So the two charts are showing two different views of energy issues in glycolysis and complement each other if you can keep from being confused. ',0,631,1,1),(##9-10-12&&

597     Q: In glycolysis, reaction 6 adds a phosphate group without using energy from ATP. Does this energy come from reducing NAD to NADH2

597     A: No, reducing NAD to NADH2 ABSORBS a tremendous amount aof energy (remember how much you get out (-53 kc/m) when the reverse process occurs in oxidative phosphorylation). The energy is coming from the oxidation of G3P to 1,3-diPGA (BPG). ',0,632,1,1),(##9-10-13&&

598     Q: How in depth should we know the pathways of energy metabolism? During the past two lectures, I felt overwhelmed with all of the detailed highlighted.&&

598     A: You should be familiar with the important steps in the pathways, where ATP and NAD is used or generated. You should have a complete understanding of the overview represented on the Outline of Energy Metabolism handout. You do not have to memorize individual reactants or reactions. In an exam, if that information is needed it will be provided (e.g., copy of the handout). But if given the reaction(s) you should understand what is going on and what the role of the individual reactions are in the overall pathway(s). For example, on the 1998 exam #2 that is on the course Web site, the glycolysis and Krebs Cycle handouts were provided, as indicated on Page 1. ',0,633,2,1),(##9-10-14&&

599     Q: It seems to me that noncompetitive inhibitors must bind irreversibly to the enzyme in order to exert their effects, and that substrate binding could be affected. What is wrong with this picture

599     A: Irreversibility has nothing to do with it. If a particualr enzyme molecule is (reversibly) binding a non-competitive inhibitor (NCI) , then while it is doing so, it will be inactive and will not contribute to the formation of product, not contribute to the velocity of the reaction. So the velocity of the reaction will be less in that test tube for that moment because of the presence of the NCI on that molecule at that moment. The next moment, the NCI will have become disassociated from its enzyme target, but another NCI will have become associated with another enzyme molecule, thus taking it out of action. So statistically, summed over all enzyme molecules and all the time going by, there are effectively less active enzyme molecules in a test tube in the presence of this dynamically binding NCI. The presence of substarte has no effect on this state of affairs, as it binds to a different site on the enzyme whether the enzyme is active or not (by definition for a NCI). ',0,634,1,1),(##9-10-14&&

600     Q: In problem 5-13, why are there 3 binding sites at ATP synthase? Does the enzyme need to \"grab\" an ADP and a Pi at different sites and then yield an ATP at a third

600     A: Yes, but I did not discuss this mechanism in enough detail for you to be able to deal with this part of the problem, so I suggest you ignore it. ',0,636,2,1),(##9-10-14&&

601     Q: In problem 5-14, why is metabolizing fat \"fattening\" here? I assumed that higher metabolism is good, since you break down the fat.&&

601     A: Yours was a natural alternative way of looking at it. We make our own fats (biosynthesis was not discussed in detail) and need energy to put them together. Excess energy (calories) will be stored as our fat. The yield of these calories from fat is greater than that from glucose, pound for pound. The control of this process is under intense investigation by pharmaceutical companies to cure obesity and allow weight loss, but is not well understood. ',0,637,2,1),(##9-10-14&&

602     Q: Why is it important to follow the identity of the individual atoms in problems where either H or C is labeled? It feels a little futile chasing the atoms around in the problems. Is it important in research

602     A: So you can \"see\" what is really going on in terms of the molecular transformations taking place. A bit of the chemical mechanism and not just succinate --> fumarate, one FAD used, etc. B. Such experiments are powerful tools in deciphering what is going on when you start from a position of ignorance. You can see what kinds of molecules are present at steady state (even then you have to know what to look for), but you don\'t really know the relationships among the compounds

(intermediates) unless you can demonstrate the flow of material from one to another. ',0,638,2,1),(##9-10-15&&

603    Q: How does is reaction time and Km for allosteric enzyme inhibition affected by competitive and noncompetitive inhibitors, and how that is shown on a graph

603    A: We will not treat the V vs. S curves for allosteric inhibition in this course. The enzymes we use as examples (those following Michaelis-Menten kinetics) are not changed by the competitive or non-competitive inhibitors we consider, they simply inhibit. In allosteric inhbition the enzyme itself changes from one form to another and this change is affected not only by the inhibitor but also usually by the substrate, resulting in complex (but understandable) kinetics. The mechanism and math is complex and is more suitable for a biochem course. If you want to learn more, consult any biochemistry text. For a simplified view of the mechanism, see Becker, p. 158. ',0,639,1,1),(##9-10-18&&

604    Q: Can we say that some of the H atoms given to NAD and FAD came from $H_2O$, and that as a consequence some of the electrons in the later ETC came not from glucose but from $H_2O$

604    A: I believe the electrons originate in the carbon compounds derived from glucose (glyceradehyde-PO4, pyruvate). The mechansims are quite complex, often involving covalent binding of the carbon compounds to the enzyme, with transfer of electrons temporarily to cysteine side chains, followed by transfer to NAD and water adding (hydrolyzing) the product-enzyme bond to release free product. Consult any biochemistry text for the exact mechanism of, for example, pyruvate dehydrogenase or glyceraldehyde phosphate dehydrogenase. ',0,641,2,1),(##9-10-20&&

605    Q: In problem 5-15A (ii), why is the answer that $CO_2$ will be the last compound to become radioactive

605    A: The $CO_2$ in the first turn is derived from the borrowed oxaloacetate, so none of the $CO_2$ is labeled with radiactive carbon inthe first turn. in contrast, all of the Krebs intermediates listed become radioactive during the first turn as the radioactive carbons from acetate are incorporated into their structure. The other choices never get radoactive carbons from these energy metabolism pathways. ',0,642,2,1),(##9-10-20&&

606    Q: If Km is a constant, does this mean that k1, k2, and k3 are fixed in relation to one another

606    A: k1, k2, and k3 are rate CONSTANTS. They are fixed values for any particular enzyme and substrates and products. They are defined by how the rate of a part of the reaction depends on the concentration of reactants, such that: k1[X] describes the influence of the concentration of X on the rate of disappearance of X in a reaction of X < --> Y. Since X can also be formed from Y as well as turn into Y, the total description of how X changes with time is given by dX/dt = k1[X] - k2[Y], where the lower case k\'s are the rate constants. The k\'s are not rates, they are multiplication factors: the rate constants must be multiplied by the concentration(s) of reactants present to get the individual rates for each part of the reaction. ',0,643,1,1),(##9-10-20&&

607    Q: I understand that Km is the [S] when V = 1/2 Vmax, but what is the significance of this with regards to k1, k2, and k3? In other words, why does [S] equalling (k2 + k3)/ k2 mean V will be 1/2 Vmax at [S] = Km

607    A: That Vo= 1/2 Vmax when [S] = Km is a simple consequence of the Michaelis-Menten equation. It is algebraically true. The fundamental definition of Km is (k2+k3)/k1. ',0,644,1,1),(##9-10-20&&

608    Q: For the reaction glucose--> 2 lactates, without considering the coupling to the formation of ATP\'s (delta Go = -45 kcal/mol), \r\ndo we know the delta Go just from some table or experimental value or is it calculated somehow

608    A: Standard delta G\'s are figured out by calculation or by experiment and published in standard tables. Virtually any biochem book has values for most reactions of biological interest. ',0,645,1,1),(##9-10-20&&

609    Q: Are we to always assume that  necessary molecules such as NAD and Pi are present in a minimal medium

609    A: A minimal medium containing glucose as the sole carbon and energy source was described in lecture 1 and in an early handout and referred to often subsequently. A minimal medium conaining a sole carbon and energy source other than glucose is referred to as glycerol minimal medium or DHAP minimal medium etc. In addition to the carbon and energy source, the medium always contains salts that serve a sources of other necessary atoms: N, K, P, S, etc. Potassium phosphate serves as a source of K as well as of phosphate (Pi), a necessary component. See lecture 2 minimal medium handout for more details. ',0,646,1,1),(##9-10-20&&

610    Q: In problem 4-2D, how do I get the reaction to go from G-6-P to F-6-P if Keq is < 1? Why can\'t the answer include using an enzyme rather than excess/removal to go from G-6-P to F-6-P, as happens in vivo

610    A: Enzymes don\'t influence the direction of reactions, just the rate,in either direction. Direction is dictated by delta G. In vivo G6P --> F6P directionality is brought about by the product-drain method, which produces a favorable delta G despite an unfavorable delta Go. You could devise perhaps another way to do it, by CHANGING the reaction to another one, more complex, that has a favorable delta Go, as is done in COUPLED reactions, but that is not what happens in glycolysis in real life for this conversion. ',0,647,1,1),(##9-10-20&&

611    Q: In problem 4-11C, why wouldn\'t you be limited by the ATP concentration since you\'ll need 2 ATPs before you even get to NAD requirement

611    A: You would use the 0.001 mole of ATP originally present to turn 0.0005 moles of glucose to FDP, which goes on to form 0.001 moles of G3P, which uses 0.001 moles of the NAD to go on to produce 0.002 moles of ATP by the time you go down to 0.001 moles of pyruvate. Now you can take those 0.002 moles of ATP and convert 0.002 more moles of glucose to FDP, etc. and end up with 0.004 moles of ATP by the time those 0.002 moles of glcuoce and up as pyruvate. So there\'s no shortage of ATP, you are making more and more as you go along ... until you run out of NAD. ',0,648,1,1),(##9-10-20&&

612    Q: What is the effect of inhibitors on the V vs. S curve in relation to the Michealis-Menten equation? (i.e. what terms in the equation are changing to have the corresponding effect on the graph?)&&

612    A: The inhibitors are not changing the Michaelis Menten equation, since that equation can still describe the response of Vo to [S]. That is, the shape of the curve agrees with the form of the equation. But in the presence of the inhibitors, it is AS IF one or the other of the constants has changed. Thus we get an apparent Km that is larger than normal in the presence of a competitive inhibitor. Let\'s call it Km\', so in this condition, Vo=k3Eo[S]/(Km\'+[S]). Similarly, in the presence of a non-competitive inhibitor, we can describe the curve by considering the amount of enzyme decreased, subtracting those enzymemolecules that are bound to the inhibitor at any given moment, so we have a new apparent Eo, let\'s say Eo\', that is less than the original Eo, so we can still write Vo= k3Eo\'[S]/(Km+[S]) or this condition and generate a curve that agrees with experimental data. ',0,649,1,1),(##9-10-21&&

613    Q: In problem 3-10, I don\'t understand why a low concentration of arg would activate the arg biochemical pathway at all, thus precipitating the accumulation of intermediates. Why are there any intermediated accumulated when low concentrations of arg are present

613    A: Arg doesn\'t deactivate the path. It is automatically \"on\" UNLESS high arg is present to shut the pathway down. Intermediates only accumulate in mutants that have blocks in the pathway. Because the pathway is blocked in the middle by the mutation (not inhibited at the start by high arg); intermediates enter but can\'t finish.\r\n<br><br>\r\nA more Detailed explanation: I think you are confusing what happens in the normal cell with what happens in a mutant (where one step of the pathway is blocked). The default state of the pathway is \"on.\" So if there is NO extra arg, in a normal cell, precursors are converted to arg. The precursors are present in low amounts, although the total flow through the pathway is high. In a normal cell, when arg is high, the pathway is shut down at the first step unique to that pathway, and no intermediates accumulate.\r\n<br><br>\r\nIn a mutant, when there is low arg (enough to live on, but no extra), the pathway is \"on\" (no feed back inhib. by arg) but one step in the pathway is blocked (by the effects of the mutation). In this case, precursors feed into the pathway but can\'t get by the block and so intermediates accumulate before the step blocked by the mutation. When large amounts of arg are added, that shuts down the pathway at the first step so no more precursors feed into the pathway and nothing gets \"stuck\" before the block in the middle. So no intermediates accumulate. ',0,650,1,1),(##9-10-21&&

614    Q: In problem 5-11B, why would there not be any ATP generated if oxidative phosphorylation depends on a proton gradient? The outside pH is rising so the inside pH must lower relative to the outside which would create a proton gradient and the F0F1 needs to be in the higher pH side and it is.&&

614    A: In this problem, I was envisioning a situation where (for some reason) ox phos and ETs were uncoupled. This is a common situation in real life, as damage to the membranes during isolation makes them leaky. Therefore it is like adding DNP -- the H+ gradient is generated, but it is dissipated without making any ATP because the H+\'s leak back into the matrix without passing through ATP synthase. The issue is not which way the membranes are orientated but whether the membranes are leaky or not. If everything is working normally and the situation is as described, ATP synthesis should occur. The question is, what could be wrong to give the results described? Since the proton gradient is generated, the problem must be after that step, not before it. ',0,651,2,1),(##9-10-21&&

615    Q: Problem 4-2B asks you to find the amount of product formed at equilibrium given a specific equilibrium constant. What if, however, the initial amount of reactant weren\'t so small

615    A: In general: Keq = [P]/[R], where [P] and [R] here are the final equilibrium concentrations, and: [P] + [R] = sum of the initial concentration of each (since nothing is lost between the two). So if

you know the Keq and if you know how much of each you started with (even if one of them is zero) then with two equations and 2 uinknowns, you can solve for P and R. ',0,652,1,1),(##9-10-21&&

616    Q: In Becker on page 364-365, the inside of chloroplasts is at pH 4, while the outside is at a pH of 8, and a burst of ATP was generated. Aren\'t things backwards here

616    A: Yes, it the gradient is backwards compared to mitochondria. ATP produced by oxphos in chloroplasts involves chemiosmosis also, but protons are kicked inside a part of the chloroplast and then allowed to flow back through FoF1 complexes oriented with their knobs facing out. So everything is backwards with respect to the inside/outside of the chlorplast itself but as expected it functions pefectly well with all components being turned around. ',0,653,2,1),(##9-10-14&&

617    Q: How exactly does DNA polymerase join 2 Okazaki fragments? Specifically, where does it start and what direction does it move

617    A: DNA pol. does not join the fragments. It extends the n+1 fragment at its 3\' end until it has filled the gap formed by removal of primer from the 5\' end of fragment n. Then ligase joins the 3\' end of fragment n+1 to the 5\' end of fragment n. (DNA polymerase or a different enzyme may remove the primer before or at the same time that fragment n+1 is being extended to fill the gap.) DNA pol. NEVER adds in the wrong direction, and no additional primer is needed. The idea is to use the 3\' end of the \"next\" Okazaki fragment instead of primer and just add on to the available 3\' end. Primer is removed first, or at about the same time, to make an empty space to fill in. ',0,654,2,1),(##9-10-14&&

618    Q: Why does C-G have 3 hydrogen bonds connecting them, while A-T bonds only need two. &&

618    A: Look at the structures of the bases, using your text or handout 10-3. You will find that there are only two groups (on the rings of the bases) with the right distances and properties to form H bonds between A and T but 3 between G and C. It is not a question of what is needed or optimal -- it is a question of what structures are available to make H bonds. ',0,655,2,1),(##9-10-15&&

619    Q: Which of the two DNA strands acts as template in transcription? Is there special header code sequence in one strand that tells the cell \"I am the postive copy and my brother in arms is the negative\"

619    A: Stay tuned! One strand of the DNA is transcribed (in any particular section) and one is not. (But remember, when DNA is copied to make RNA, the template is read 3\' to 5\'! That way, new RNA is made 5\' to 3\'. When info in RNA is used to make protein, the RNA is decoded 5\' to 3\'. I warned you in class it was a mess.) ',0,656,2,1),(##9-10-15&&

620    Q: Why is it that in E-coli, Chargaff got A=26%, T=24%, G=25%=C? Does that data have experimental error, or is E-coli an exception

620    A: That is a normal range of experimental error. I\'m sure the results are + or - a few percent. It\'s a difficult procedure. ',0,657,2,1),(##9-10-18&&

621    Q: In the Meselson-Stahl experiment, are we to assume that the dispersive method is \"random,\" and as such the results would show a variety of densities (as heavier, old DNA would be spread throughout new generation), or that the old DNA is somehow distributed evenly (albeit piecemeal) thus yielding only one band after centrifugation

621    A: The usual assumption is that the old and new sections are scrambled in some even handed sort of way so that all the DNA strands will become intermediate in density. (There are variants of this scheme that differ in the fine points.) It is possible that all the DNA will have exactly the same density, but it is considered more likely that the DNA will have a range of intermediate densities as each strand will have a slightly different proportion of heavy and light. The result will be a single band, but a broad diffuse one, of intermediate density. (There will probably not be discrete multiple bands since each molecule in the mixture will have a slightly dif. proportion of H and L.) ',0,658,2,1),(##9-10-22&&

622    Q: Are mRNA, tRNA, and rRNA replicated in the same way? Or is the description you gave for replication only for mRNA

622    A: All RNA\'s are made the same way -- not replicated, but transcribed, off a DNA template. (I drew a U in the template in the morning lecture -- that was an error.) I meant to say all RNA\'s come from DNA in lecture, but didn\'t get to it; we will summarize it all next week. ',0,659,2,1),(##9-10-22&&

623    Q: In lecture 10, the function of DNA is said to imply that the phosphates are located inside DNA molecules, with the nucleotides sticking out, whereas the structure of DNA implies that the phosphates are located on the outside of the helical structure. In this case, why does function imply one thing while structure implies another

623    A: Before the structure was figured out, people reasoned on theoretical grounds whether the bases stuck out or in. Some reasoned the bases must stick out in order for them to be read. Others reasoned the bases must be inside, in order for the hydrophillic (and charged) phosphate sugar backbone to be outside and interact with water. Experimental data has settled the question -- the bases are inside. This is compatible with function -- now that we know the actual structure, it is clear that the bases go in, but the double helix unravels (in short sections) to make the bases and the info the carry available. I think you are confusing the current state of knowledge with the historical line of reasoning that lead to it. ',0,660,2,1),(##9-10-29&&

624    Q: What is the difference between deoxycytidylate and deoxycytidine

624    A: \"ate\" means the ionized form of the corresponding acid. So cytidylate means the nucleotide dCMP with a phosphate (ionized). It is used synomously with cytidylic acid. Deoxycytidine means just the base + sugar. (cytosine is the plain base). The terminology is not consistent with respect to suffixes -- adenosine is the sugar + base but adenine is the base alone. We usually say C, C + ribose or CMP etc. to avoid these problems. ',0,662,2,1),(##9-10-29&&

625    Q: How many times will a DNA replicate before it is degraded into nucleotides

625    A: In real life, in a living cell, the answer is forever. The DNA is never degraded unless the cell dies. (In problem 6-5, the DNA duplicates only once, and then the template is degraded.) ',0,663,2,1),(##9-10-29&&

626    Q: Regarding problem 6-5: If the original \"heavy\" DNA is degraded into \"heavy\" nucleotides, can\'t those nucleotides be used for polymerization of new nucleotides that are heavier and therefore centrifugate to a position between light and intermediate

626    A: Yes. See answer to 6-5 B. ',0,664,2,1),(##9-10-29&&

627    Q: How can you tell DNA and RNA nucleotides from one another when they are represented in the shorthand of handout 10-2?\r\n\r\n&&

627    A: The shorthand method of writing the sugar doesn\'t allow for distinction between whether or not the sugar is ribose or deoxyribose because only the 3\' and 5\' carbons are shown, not the 2\' (which distinguishes). The only way you would know whether you were looking at DNA or RNA would be to look for either a U or T attached in a polynucleotide.',0,665,2,1),(##9-10-29&&

628    Q: On problem 6-8, does reaction 2 always require a coupled exergonic reaction, whereas reaction 1 provides its own reaction

628    A: Reaction 2 can occur as is (going spontaneously to right) -- it\'s used to hydrolyze polymers. If you want to synthesize a polymer, you have to couple reaction 2 to something exergonic, which gives you (overall) something like reaction 1. ',0,666,2,1),(##9-11-01&&

629    Q: In problem 4-1 (D), why do the chromosomes look like they do

629    A: At this level, we are just asking for number of chromosomes lined up in pairs and sorted properly with respect to size and with position of chromatids shown. A real karyo done in a lab would probably be done so as to show bands. In this case you don\'t need bands to tell chromosomes apart because all are different sizes. ',0,667,3,1),(##9-11-01&&

630    Q: When we use the term, \"degeneracy,\" are we specifically referring to the number of tRNAs (~30) compared to the number of amino acids (20), or do we mean the number of distinct codons (64) compared to the number of amino acids

630    A: Degeneracy refers to the # of codons vs # of a.a.. Code is degenerate because there is more than one codon per amino acid. The number of tRNA\'s per amino acid depends on wobble -- code could be degenerate with or without wobble. (But wobble isn\'t an issue unless the code is degenerate in the first place.)<br><br>\r\n\r\nSo in problem 7-25, degeneracy stays the same, but wobble doesn\'t. ',0,668,3,1),(##9-11-01&&

631    Q: In equilibrium density centrifugation, what exactly will happen to 14N and 15N DNA while the centrifuge is running, and what happens when it stops

631    A: Each type of DNA (containing N14 or N15) will rise or sink until it reaches equilibrium -- finds itself in an area of density that matches its own. Running the centrifuge is what sets up the gradient of density. Turning off the centriguge is just done at the end after everything has reached its final position. ',0,669,2,1),(##9-11-01&&

632    Q: Why does trp synthetase catalyze the LAST step in synthesis of the AA tryptophan. (i.e. A-->-->-->tryp)

632    A: That\'s just the way it is -- the enzyme that catalyzes that particular step has that particular name. Maybe you think some other enzyme in the series should have that name? Or all of them together? The person who named the enzyme called it trp synthetase because the product of the reaction is trp. Did s/he know there were other enzymes involved? Maybe, maybe not. ',0,670,2,1),(##9-11-01&&

633     Q: In a ribosome, what makes the P or A site specific

633     A: A peptidyl tRNA holds an amino acid that has something attached to its amino end (namely, the rest of the chain); an amino acyl tRNA holds an amino acid with a free amino group. That\'s how the sites tell the difference. The initiator tRNA in bacteria has a blocking group on it\'s amino end; the eukaryotic initiator does not have an actual blocking group, but mimics one. ',0,671,2,1),(##9-11-01&&

634     Q: Is there also a wobble phenomenon with the 20 aminoacyl-tRNA synthetases when they bind the 30 or so tRNAs and their specific AA

634     A: As far as I know, there is only one loading enzyme per amino acid. So one enzyme must usually bind several cognate tRNA\'s. It isn\'t called wobble or degeneracy -- it isn\'t uncommon for an enzyme to bind several similar substrates (like dif. tRNA\'s for the same AA). ',0,672,3,1),(##9-11-01&&

635     Q: Is RNA primer required for transcription or translation

635     A: You need RNA for primer to start DNA synthesis. You don\'t need it for standard transcription (to make mRNA, tRNA or rRNA) or to do translation. ',0,673,2,1),(##9-11-01&&

636     Q: On problem 6-13 (B-4) Wouldn\'t EXO1 also do this editing since it is going over the newly synthesized chain while removing primer

636     A: In this question we are assuming no repair (by any enzyme). ',0,674,2,1),(##9-11-01&&

637     Q: In problem 6-13 (A-3): Shouldn\'t the answer to this part of the problem be \"nucleotides containing ribose should be found in the middle and on the left end?\" Since ribose is the sugar of RNA, it should be found wherever there is primer, and in bidirectional replication, primer is needed to start the synthesis of the leading strand once (in the middle at the origin) and to start the synthesis of each Okazaki fragment.&&

637     A: The question asks you to \"consider a new DNA chain that is made continuously from the origin using the bottom strand as template.\" Given this constraint, there is only one primer (as there is only one growing chain to consider). If you consider all the possible growing chains, the answer is different. ',0,675,2,1),(##9-11-01&&

638     Q: Are ribosomal subunits and mRNA components that are \"re-used\" over and over always reused by the same cell, or can the parts be used by different cells over time

638     A: Ribosomes, mRNA etc. are too big to get in and out of cells easily. They would also be degraded by material on the outside. So Things like that are generally used only in the cell that makes/contains them. Certain proteins get secreted, but it is a complex and special process. (They usually act extracellularly; they don\'t get into a second cell, but just signal or act on the outside.) ',0,676,2,1),(##9-11-02&&

639     Q: What are the conditions under which DNA hybridization can form similar, but not identical DNA strands

639     A: Hybridization can be down under so-called \"stringent\" conditions meaning high temperature and buffers that do not stabilize base pairs. Under these conditions, only perfectly matched strands will form a hybrid. Imperfectly matched strands fall apart. Under less strigent

conditions of lower temp and different buffer, strands that are almost the same will form a hybrid. (Different degrees of mismatch are acceptable at different temperatures.) Depending on what you want to do, you use the appropriate conditions. ',0,677,3,1),(##9-11-02&&

640     Q: Regarding the 5 reactions to synthesize DNA/RNA: Are all 5 reactions really occuring or are only 3, 4, and 5 occuring (or only 1 and 2)? It seems that the overall delta G from adding 3, 4, and 5 is not the same as adding 1 and 2.\r\n&&

640     A: 1 plus 2 is the equivalent of 3 through 5. The actual rxns are 3-5 (for synthesis). According to my reckoning, net delta G\'s for 1-2 or 3-5 are the same -- they are -7Kcal/mole in each case. 3 and 4 are about zero; 5 is -7, 1 is +7 and 2 is -14. If it still doesn\'t add up, let me know. ',0,678,2,1),(##9-11-02&&

641     Q: Are germ cells the only normal human cells that have telomerase

641     A: As far as I know, yes. (I doubt if every type has been tested.) It is possible that some stem cells have it too. I will see if I can find out more details. ',0,679,3,1),(##9-11-03&&

642     Q: In PCR, after you denature the DNA and add primers, what keeps the two long strands from re-hybridizing

642     A: I think the two long strands of DNA do not renature during the short time at low temperature because of their extremely low concentration in the mixture. ',0,680,2,1),(##9-11-03&&

643     Q: When using PCR for identification, such as DNA from a crime scene, and you compare STRs, are all the 13 or so sections on a person\'s DNA exactly the same

643     A: You replicate only highly variable sections. You are looking at 13 DIFFERENT sections. A different repeat occurs at each section. ',0,681,2,1),(##9-11-04&&

644     Q: How to organisms regulate the amount of mutation going on in DNA replication

644     A: The organism has no idea what is \"good\" or \"bad.\" Different organisms have different rates of mutation, due to random differences in DNA polymerases, repair enzymes etc. (Diff. enzymes have dif. degrees of accuracy.) If a population of organisms has too low a mutation rate, it often dies out as there is no variation for selection to act on. If the mutation rate is too high, the population doesn\'t do well because too many organisms are sick or defective or just non optimal. The organisms that just happen to have enough, but not too much, mutation prosper and have many surviving offspring. So the organism doesn\'t plan ahead, but those who (by accident) do it right have the most offspring and their descendants (who do things more or less like their ancestors) take over. ',0,683,3,1),(##9-11-04&&

645     Q: In problem 7-12 (D), does it always have to start on either the 1st, 2nd, or 3rd base? IN part (G), where\'s the stop codon

645     A: Ans. for D: No. But starting on the first and the 4th are considered the same \"reading frame\" and to produce different versions of the same protein, not totally different proteins.<br><br>\r\nAns to G: If you use 1st or 2nd reading frame you hit a stop. In first frame stop is last full codon ; in 2nd frame stop is first. ',0,684,3,1),(##9-11-04&&

646    Q: What is genotype vs. phenotype

646    A: I forgot to bring it up. Genotype = state of genes and/or DNA; phenotype = state of function, appearance, etc. = state caused by expression of the DNA. See texts for more information; we\'ll go over it in detail later. ',0,685,3,1),(##9-11-05&&

647    Q: What is the difference, if any, between F+ and Hfr? Are they 2 names for the same thing

647    A: They are different -- see either text for detains and pictures. Both are able to copy DNA and transfer it to F-; I deliberately didn\'t want to get into the fine points. F+ transfers only copies of plasmids; HFr usually transfers fragments of bacterial DNA. In F+ the genes allowing copying and transfer are on a plasmid; in HFr the plasmid (through crossing over) has become part of the chromosome (is integrated). ',0,686,3,1),(##9-11-16&&

648    Q: In problem 6-16, is it possible that RNA can be double stranded

648    A: Double stranded RNA is found only in viruses and virus-infected cells. It is NOT found in normal, uninfected cell, but it does exist.\r\n',0,689,2,1),(##9-11-16&&

649    Q: In problem 7-3 (B), is the DNA polymerase recycled

649    A: DNA polymerase in an enzyme -- it was a catalyst that is used over and over.\r\n',0,690,2,1),(##9-11-16&&

650    Q: In problem 6-15, are the nucleotides indicated the ends of the DNA? That is, is GCCT at the right hand end of Crick the 5\' end or does it continue past GCCT

650    A: Notice the ....... on the ends. That means the DNA keeps going, so it is the second case.',0,691,2,1),(##9-11-16&&

651    Q: In DNA and RNA synthesis, are the substrates nucleotide triphosphates or nucleoside triphosphates?\r\n&&

651    A: There is no such thing as a nucleoTide triphosphate, whether ribo or deoxy. In both cases you need a nucleoSide triphosphate. ',0,692,3,1),(##9-11-17&&

652    Q: Is a test of mRNA an accurate indicator of a genetic problem

652    A: The state of the mRNA is assumed to reflect the state of the DNA. If it were a mistake in the mRNA, but the DNA were ok, then most molecules of mRNA would be okay. This is providing all the mRNA is changed. ',0,693,3,1),(##9-11-17&&

653    Q: In light of problem 7-23, how to similarities between amino acids translate to protein structure and function when one amino acid is substituted for another, similar one?\r\n&&

653    A: This sort of substitution would yield structurally and functionally similar proteins (and so a few incorrect amino acids in an enzyme won\'t usually have a noticable effect).',0,694,2,1),(##9-11-18&&

654    Q: What exactly do you mean by nonconservative replication

654    A: Nonconservative replication is a nonsensical invention for study purposes. It means the original template is destroyed after copies are made. ',0,695,2,1),(##9-11-18&&

655    Q: In the 1998 exam 2 question 3, why must the number of TT dinucleotides equal the number of AA\'s

655    A: DNA is very long, and you have many, many molecules (even at low concentrations). In a real sample, the proportions of dinucleotides will reflect their proportions in the DNA. You are correct that the results are different if you use a very short DNA or only a few molecules. ',0,696,2,1),(##9-11-18&&

656    Q: In problem 12-2 (C), are promoters non-specific and operators specific such that the lac repressor protein can affect the expression of his structural genes

656    A: You have hooked the switch mechanism for the lac structural genes up to the his structural genes. The switch mechanism (O-lac, P-lac, repressor protein for lac) acts exactly the same whether the O and P are next to the lac or his structural genes. ',0,697,3,1),(##9-11-22&&

657    Q: Are aneuploid, monosomic and trisomic always defined relative to a diploid genome? Would you say a chromosome in a haploid genome were trisomic if it appeared with 3 homologs? And are these aberrations usually corrected by a round of replication

657    A: Aneuploid means more or less than a multiple of N. Can be relative to N, 2N, 3N etc. Monosomic means there is one of that chromosome when there should be 2, 3 etc. Trisomic means there are 3 when there should be 1, 2 or 4. In a triploid, if you were missing one chromosome, that group of homologs would be disomic. These aberrations are usually not corrected -- mitosis just passes on copies of whatever is there. ',0,698,3,1),(###-12-08&&

658    Q: Why does a zygote have to be diploid? Is it simply this by definition, or could there also be tetraploid zygotes (since they go through meiosis according to problem 8-5).&&

658    A: Normally, only gametes (haploid) fuse, so zygotes are diploid . But it does happen rarely (in nature) or in the lab (if you interfere with the normal processes) that two diploids can fuse and then you get a tetraploid. But this is not a normal event in any organism\'s life cycle.\r\n',NULL,1417,3,1),(##9-11-28&&

659    Q: What is the difference between RFLPs and VNTRs? I realize that they are both different types of polymorphisms causing different individuals to have unique banding patterns, but what exactly distinguishes one polymorphism from another

659    A: A polymorphism is any difference found between individuals. If the difference is in the lengths of restriction fragments found (after identical treatment), it\'s called an RFLP. The difference can be caused by differences in the DNA in the actual restriction sites, OR in the sequences inbetween the restriction sites. A VNTR generates a RFLP because there is a difference in the number of repeats inbetween fixed restriction sites. ',0,699,4,1),(##9-11-28&&

660    Q: Since cDNA is trancribed from mRNA, is it possible for it to contain introns and exons

660    A: cDNA contains only sequences found in the mRNA. It contains the translated region plus 5\' and 3\' UTR\'s = \"leaders\" and \"trailers.\" Only exons; no introns. By definition, exons = what ends

up in the mRNA; introns = intervening sequences that are removed from the premRNA transcript before it becomes mRNA. ',0,700,4,1),(###-01-23&&

661    Q: Is the nuclear envelope considered to be part of the EMS

661    A: No, but in some sense the outer layer of the envelope is. You should consider the perinuclear space to be <i>effectively</i>\r\nconnected to the lumens of the golgi apparatus, ER, and vesicles.',NULL,1456,1,2),(###-01-23&&

662    Q: When a microtubule is stabilized, does it mean that it cannot be disassembled?  Similarly, when a microtubule is depolymerized,\r\ndoes it mean that it cannot be further assembled

662    A: Yes. Although usually stabilized means \"less likely to disassembled\" not that it can\'t disassembled at all, and depolymerized (by drugs) means that subunits released as monomers are less likely \r\nto polymerize.',NULL,1457,1,2),(###-01-23&&

663    Q: When we say that intermediate filaments consist of staggered protofilaments in a flat cable, 8 across, what exactly does the \"8 across\" mean

663    A: The IF is made of multiple protofilaments, either twisted together or in a bundle or arranged in a flat cable. It is thought that there are 8 protofilaments in one IF.',NULL,1458,1,2),(##9-11-30&&

664    Q: I have a hard time grasping the distinction between different types of media. In other words, how does bacteria respond based on the medium it\'s in? How does this work in regard to energy considerations

664    A: Small molecule end-products present in a more-than-minimal medium (e.g., a rich medium) will inhibit the first enzyme committed uniquely to its biosynthesis (e.g., the threonine deaminase inhibited by isoleucine, example) and also repress all the enzyme devoted to the biosynthesis of those molecules (e.g. the tryptophan operon example). Note that a Rich medium is made up of <b>small</b> molecules, not macromolecules like enzymes. Enzymes (proteins) usually have no way to get into cells intact (i.e., without being broken down to amino acids) and so are not usually included in the medium for bacteria.\r\n\r\n<p>About energy considerations.\r\nMost biosynthetic pathways use reactions coupled to ATP hydrolysis to provide energetically favorable steps, so feedback inhibition saves using this ATP. Repression results in less proteins being made. The polymerization of amino acids into proteins (translation) uses a lot of ATP, so much is saved by not making unnecessary enzymes (e.g. beta-galactosidase in the absence of lactose, tryptophan synthetase in the presence of tryptophan. ',0,702,2,1),(###-01-26&&

665    Q: If immunofluorescence is used to locate a target protein (say Protein A), how do you isolate protein A to begin with in order to \r\ncreate an antibody to it

665    A: You have hit on a serious problem! Sometimes you have antibody to a protein #1 from source #1, and you use that to see if there is a similar protein (like #1) in cell or organism # 2. Alternatively, you may have antibody to protein #1, but are not sure exactly where in the living cell protein #1 is found. But if you have no idea what the protein is, you can\'t make antibody to it and find it using immunofluourescence.',NULL,1460,1,2),(##9-12-07&&

666    Q: Problem 13-9 (B-2), indicates that the hybrids of genomic DNA and cDNA are treated with DNAase and then denatured. Wouldn\'t both the single stranded genomic DNA and cDNA being degraded by DNAase? If so, wouldn\'t the gel reflect this by having both radioactive and non-radioactive bands of equal lengths (i.e. the length of the exons of the genomic DNA)

666    A: It depends on how the cDNA probe and the genomic DNA pair up. If the introns in the genomic DNA loop out, then a single molecule of cDNA probe can hybridize to many exons and the probe will not be degraded. (See picture in the key.) If a different molecule of probe hybridizes to each exon in the genomic DNA, then only parts of the probe will be double stranded, and parts of the probe will be degraded, and answer will be as you say. ',0,703,3,1),(##9-12-07&&

667    Q: Is it safe to assume that only \"even-ploidied\" cells can divide by meiosis

667    A: Yes -- if you want products of meiosis to be euploid. If ploidy is odd, most of meiotic products will be aneuploid (and these are not viable in most organisms.) ',0,704,3,1),(##9-12-07&&

668    Q: In problem 11-15 (B), is it possible for leu and his to be on a different chromosome

668    A: No, because Bacteria have only one chromosome. ',0,705,4,1),(##9-12-07&&

669    Q: Why does the answer to problem 8-2 state there are chromosomes, etc. in the G1 and S phases of the cell cycle? Isn\'t the DNA too loose for transcription or replication to occur

669    A: The DNA (+ histones) is there during interphase, it just isn\'t condensed. You can\'t SEE chromosomes, chromatids, etc., but a chromosome in an unfolded form is still considered a chromosome, chromatid, etc., and the section that connects (or will connect) the two sister chromatids is still there too (and considered a centromere.) ',0,706,3,1),(##9-12-07&&

670    Q: Why can\'t the answer to problem 13-7 (A) be that we made the plasmid without sticky ends on the human DNA or by using different restriction enzymes to cut the human and plasmid DNA

670    A: The plasmid holds together unless you heat it. This implies there are complementary sticky ends, and that they hybridize. However the sticky ends are not sufficient to hold everything together at high temperature when weak bonds are easily broken. So it must be the ligase that was left out. ',0,707,3,1),(##9-12-07&&

671    Q: In problem 8-9, why doesn\'t nondisjunction at the second division produce XY gametes? Also, when considering nondisjunction at second division, don\'t you need to mention the fate of all 4 gametes formed from the original cell (not just 2)

671    A: Question asks for ABNORMAL combos only.\r\n\r\n<p>If ND occurs at second division, only 2 of the 4 cells are affected; the other 2 will be normal. The answer is only concerned with ABNORMAL gametes. ',0,708,4,1),(##9-12-07&&

672    Q: In problem 13-8, why is it that in the right end of the restriction fragment (part A), the bottom strand is only CCTAG, not CCTAGG (to complement GATCC)

672    A: The restriction enzyme cuts between the two G\'s on each strand. If you make a cut, the two halves will look like this:\r\n\r\n<table width = 20%>\r\n<tr><td>5\'XXXXXXG</td>

<td>GATCCXXXXXX</td></tr> \r\n<tr><td>3\'XXXXXXCCTAG</td><td>GXXXXXX</td></tr>\r\n</table> ',0,709,4,1),(##9-12-07&&

673    Q: When does complementation occur over recombination? How can you tell the difference by just observing phenotype

673    A: If function is restored without any opportunity for crossing over, you know it was complementation. Otherwise, it is sometimes difficult to tell. ',0,710,3,1),(###-01-23&&

674    Q: Are secondary antibodies specific to a primary antibody? Do they bind to the variable part of the primary antibody

674    A: Secondary antibodies are not grabbing the grabbers, or variable part, of the first antibody. They are grabbing the constant part, or back end, of the first antibody. So the secondary antibody is not \r\nspecific for a particular primary antibody -- it is specific for all the antibodies from a particular animal (and of a particular class). We\'ll explain about classes later -- you can ignore that part for now.',NULL,1459,1,2),(##9-12-07&&

675    Q: Are people albino due to a recessive gene or due to a mutation? If an albino man and an albino woman have children, will their children necessarily be albino? If albinism is due to a mutation, will this mutation be in the gametes, too

675    A: Albinism is usually due to a recessive gene; that is, an albino is homozygous for the recessive allele of a gene for making melanin. The recessive allele causes albinism because it has a mutation that causes the enzyme it codes for to be defective. There are two genes that can cause albinism when they are defective, so an albino is aa or bb. Usually albinos inherit mutant alleles from their parents; they are usually not new mutatants -- they don\'t usually carry mutations that they did not inherit. Albinism is caused by a mutation, but the mutation usually occurred many generations back, not in the person who has it now. If an aa marries an aa, or bb X bb, kids will be albino. If an aa (BB) marries a bb(AA) kids will not be albino; they will be AaBb. ',0,711,4,1),(##9-12-07&&

676    Q: Can sex-linked alleles be dominant or recessive (i.e. is the black coat allele considered to be dominant)

676    A: On a cellular level, there is no dominance or recessiveness for sex linked alleles. Neither O nor black is considered dominant, since the effects of the two are independent. Neither allele over rides the effects of the other. The situation is somewhat different at the organismic level for some sex linked genes. A female heterozygous for the hemophilia or color blindness allele will not be a hemophiliac, or color blind, because the effects of the normal allele (working in half the cells) produces enough clotting factor or pigment to override the effects of the mutant allele (maybe working, but producing nothing, in the other half of the cells). ',0,712,4,1),(##9-12-07&&

677    Q: Why do some people have eyes that are neither blue nor brown, such as green eyes or a combination of colors? Also, is eye color constant or can it change over time, or even over the course of a few minutes

677    A: Eye color is constant over short periods but looks different in different lights. Eye color can change over long periods as pigment production changes with age. There are many genes that affect

eye color. If all the others are held constant, one gene causes blue or brown as described. If the other genes vary as well, additional colors are possible.',0,713,4,1),(##9-12-09&&

678    Q: Am I observing correctly that \"normal\" alleles are generally written as capital letters and \"altered/deficient\" alleles as lowercase letters

678    A: Usually upper case is used for dominant alleles, whether normal or mutant. Lower case for recessive. The system varies for different organisms -- diff. groups of researchers have different conventions. ',0,714,4,1),(##9-12-09&&

679    Q: In problem 11-14 (D), why does the percentage of recombinants need to be doubled before the correct map distance can be found? How are the values in the table different from the recombination frequency

679    A: Any time there is crossing over, you get two recombinants from every crossover (cut and splice) event. You often detect or count only one of the two (because it\'s easier to find a normal, say, than a double mutant -- normal often grows where others won\'t but double mutant has same phenotype as original mutants). In this case you count the number of normal sized plaques = number of normal, wt recombinants = 1/2 total number of recombinants. Total recombinants = double mutant recombinants + normals.',0,715,4,1),(##9-12-09&&

680    Q: Problem 13-3 asks for the DNA sequence given the amino acid sequence, but the answer seems to give the RNA sequence (the exact codons for the AA sequence). Why is this

680    A: The sequence given is the sense strand of the DNA. Restriction enzymes actually cut double stranded DNA, but only one strand is given in the answer (the other strand is the complementary one). You are probably thinking in terms of the transcribed strand instead of the sense strand of the DNA. If the transcribed strand has the complement to the mRNA, the sense strand has the sequence shown.',0,716,3,1),(##9-12-14&&

681    Q: What is the significance of a population being \"inbred?\"&&

681    A: Inbred implies homozygosity.',0,717,4,1),(##9-12-14&&

682    Q: In what stages of mitosis and meiosis do homologs pair up, and when does DNA coiling occur

682    A: Homologs pair up in pro I of meiosis; that\'s when crossing over is. They also pair up in metaphase I, so they can separate (disjoin) properly. In mitosis, there is no pairing of homologs at all -- chromosomes do line up at metaphase, but not in pairs. See texts for pictures of all stages in more detail than discussed in class. Condensation does occur in prophase of both mit. and mei.',0,718,3,1),(###-11-29&&

683    Q: Does the poly-A signal sequence (that signals the cleaving of the transcript and the \r\naddition of the poly-A tail) replace the terminator in Eukaryotes, or is there a terminator in addition

683    A: There isn\'t any terminator for transcription for mRNA\'s with poly A (most of mRNA\'s). A few mRNA\'s don\'t have poly A, and I don\'t know how their 3\' end is determined. In any case, when there is no terminator, the RNA polymerase just keeps going, but once the transcript (still with introns)

is \r\ndetached, any remaining fragments of RNA and the polymerase tend to fall off the DNA.',NULL,1423,3,1),(##9-12-14&&

684     Q: In problem 8-6, how do you determine the G1 and S times without knowing that it is a 20 hour cycle? And do the \"beads\" in the answer key represent anything

684     A: The beads are the cells going around the cycle. The problem tells you how long it takes the first cell to go from end of S to M (---> length of G2) AND how long it takes to go from end of S to M and then all around again to M -- this gives the total length of the cycle if you subtract the length of G2.',0,719,3,1),(##9-12-14&&

685     Q: In Problem 13-11 (B), why is the number of amino acids resulting from the mRNA = 2667 rather than 4833 if the mRNA is 14.5 KB in length? Wouldn\'t you theoretically divide 14.5KB by 3 (nucleotides/codon)

685     A: There are always untranslated regions (UTR\'s) on the two ends of the mRNA in eukaryotes -- an extensive \"UTR\" on the 5\' end before the first AUG and one on the 3\' end after the stop codon. You can see how long they are here but looking for the sections before the start and after the stop codons. You divide the # of bases between start and stop codons by 3, not the total number of bases in the entire mRNA.',0,720,3,1),(##9-12-14&&

686     Q: In problem 13-13 (C-1), why is it that only 1/2 of the mRNA\'s that result from transcription of the transformed plasmid are translated

686     A: You can insert your gene into the plasmid two ways at each restriction site. In terms of sites in the gene, it can go A -B - C etc. or it can go C- B - A. The promotor is in the plasmid, not attached to the gene (before you ligate). When you transcribe, starting at the promotor, you always go in the same direction. If the gene is inserted one way you transcribe A then B then C using \"Crick\". If the gene is inserted the other way you transcribe C then B then A using \"Watson.\" (You use the complementary strand, and start from the other end.) One strand of the gene codes for a sensible mRNA with a start codon, no internal stops, etc., and one doesn\'t. If this still isn\'t clear, ask me or a TA. ',0,721,3,1),(##9-12-14&&

687     Q: How can you tell whether complementation or recombination has occurred

687     A: If function is restored without any opportunity for crossing over, you know it was complementation. Otherwise, it is sometimes difficult to tell. Additional hints: You have to look closely at the details in the question to see how to rule out one or the other in any particular case. In 11-10, it says all progeny are mutant. Therefore there was no crossing over, and function must be restored by complementation. in 11-8, most of the progeny are mutant, and only a few are recombinant. So the rest (non mutant) must be the product of complementation. Complementation allowed infection to proceed to the point where some crossing over could occur.',0,722,3,1),(###-01-28&&

688     Q: If 4.1 is attached to glycophorin, and actin is attached to 4.1, then would actin \"light\r\nup\" if there were a labeled antibody attached to glycophorin? How about 4.1

688     A: 4.1 is attached to glycophorin, but the 4.1 may be attached to spectrin which is in turn attached to actin, or the 4.1 may be attached to actin and spectrin. In general, the protein target

\"lights \r\nup\", not what\'s attached to it. However when you use immunoprecipitation to find protein A, what is attached to it also precipitates.',NULL,1461,1,2),(##9-12-14&&

689    Q: When the term linkage is used in the problem book, does that always mean that some recombination can occur, but mostly parental gametes are obtained, or does linkage imply that the two genes are on the same chromosome and thus physically linked? (For example, can you explain how to arrive at the correct conclusion for problem 11-15 (A) )

689    A: Genetic Linkage means that you get less than 50% recombinants = parental combinations tend to stay together more often than they get switched. Genetic Linkage implies physical linkage, but the reverse is not always true as genes far apart on the same chromosome do not always act genetically linked (because of multiple crossovers).\r\n\r\n<p>In 11-15, the leu+ allele is linked to the arg- allele, so if you select for leu+ recombinants, you tend to get arg- along with leu+ -- the two genes tend to be on the same fragment of DNA and incorporated together. Note the recipient is arg+; this is different from the usual set up where the donor is + for everything and the recipient is -. When arg- is incorporated (by crossing over) the arg+ allele ends up on a fragment and is lost. ',0,723,4,1),(##9-12-14&&

690    Q: Can you explain how to find G1 in problem 8-6 (1)? .&&

690    A: If you have the whole cell cycle, and G2, M and S, you subtract to get G1. In this problem, you get G2 first, then M, then S. Then it tells you how long it takes to go from the end of S to M and all the way around again to M. This gives you the total length of the cycle + G2. You subtract G2 and that gives you the whole cycle. Then you subtract G2, S and M and you are left with G1. ',0,724,3,1),(##9-12-16&&

691    Q: Can you explain the answers further for problems 11-15 (C) and (D)

691    A: Part C: The his and leu genes are on the same chromosome (E. coli only has one) but they are far apart. You know this since they are not genetically linked from data given. If you break the DNA into short fragments, and you get a fragement of the chromosome that carrier an allele of his, it is unlikely to include an allele of leu.\r\n<p>Part D. It\'s a topology question. You have to draw the chromosome and plasmid involved and see what gene orders you will get if you \"cross in\" the plasmid. ',0,725,4,1),(##9-12-16&&

692    Q: What is the difference (phenotypically) betweeen epistasis and partial dominance

692    A: Dominance involves effects of one allele of a gene vs another allele of the same gene. Does effect of A override effect of a (partially or completely)?\r\n\r\n<p>Epistasis involves effects of one gene vs effects of another, different gene. Does state of gene A (aa or A_) override effects of gene B (whether B_ or bb)?',0,726,4,1),(##9-12-16&&

693    Q: What exactly is the Founder effect? Are we expected to know this for the final exam

693    A: We did not discuss Founder effect in class and I said then (I think) and in the notes not to worry about the fine points. It is a form of genetic drift and is explained in the notes. All you have to know is that it is an effect of small population size -- it can cause changes in the alleles in the gene pool due to picking a small sample of individuals (to \"found\" a new population). How it differs from the

other effects of chance and small samples is a very interesting issue but will not be on the exam. ',0,727,4,1),(##9-12-16&&

694     Q: In problem 13-13 (A), why does the bacteria\'s DNA hybridize to the gene on the plasmid if plasmid DNA is double stranded

694     A: It is assumed that you will denature the double stranded DNA\'s involved before attempting any hybridizations. You are correct that plasmids (and bacterial chromosomes) are not single stranded. ',0,728,4,1),(###-01-28&&

695     Q: How do the sugars on glycophorin of RBCs determine blood type? Do the sugars act as receptors

695     A: Sugars determine ABO, not MN. Amino acid differences in glycophorin A determine MN. I\'m not sure if ABO sugars are on glycophorin or not. I doubt it. Sugar/protein combos (glycoproteins) can act as receptors .',NULL,1463,1,2),(##9-12-16&&

696     Q: In problem 11-10 (A), why can\'t the mutation be in the same base pair

696     A: In the experiment described in this question, you are getting only complementation, not crossing over (recombination). It says all progeny are mutant, so no crossing over is occuring at all. All the positive results are due to complementation, and all the negative results are due to failure to complement, which means the pair of mutations tested is in the same gene. You can\'t tell from results given if 2 mutations in the same gene are in the same spot or not. You\'d have to have a set up that allows crossing over in order to tell. So in part A, the two mutations could be in the same base pair, but you have no proof that they are or not. All you know is they must be in the same gene. ',0,729,4,1),(##9-12-16&&

697     Q: Why go through all the cloning procedure when it is possible to obtain the RNA sequence for the desired protein (as done for a probe) and then use reverse transcriptase to obtain the gene

697     A: Reverse transcriptase gives you the cDNA, not the gene. The gene is different -- it has introns, regulatory sequences 5\' and 3\' etc. You can\'t get untranscribed sequences from looking at cDNA. ',0,730,4,1),(##9-12-16&&

698     Q: In problem 14-1 (B), what do you mean by mutation balances selection

698     A: The effects of mutation (mutations --> new defective alleles) is counterbalanced by effects of selection (loss of defective alleles when aa don\'t reproduce). ',0,731,4,1),(##9-12-16&&

699     Q: Please explain the answers to problem 10-14  (A), (C), (D) and (E).&&

699     A: Part A. You did a test cross here of double heterozygote X double homozygous recessive. If two genes involved were not linked, offspring should be all 4 types in equal proportions. AaBb gives 4 types of gametes, all in equal amounts. aabb gives only one kind. Results do not match this -- instead you get 83% of two kinds and 17% of the others. This makes sense if genes are linked and two common kinds are the parentals; the other two are the recombinants.<br><br>\r\n\r\nPart C. The two rare types must be recombinants. (see D). RF = % recombinants.<br><br>\r\n\r\nPart D. Red goes with short and white with tall (phenotype). These are the majority of the offspring, which means they must

be the parental combos. This means original parents must be red short (AAbb) and white tall (aaBB). F1 is all Ab/aB. F2 is Ab/ab mostly red short and aB/ab white tall = parentals. Recombinants are ab/ab or AB/ab. Only other possibility is original red parent was tall or AABB. If that were the case, red short and white tall in the F2 would be recombinants, and there shouldn\'t be so many of them.\r\n<br><br>\r\n\r\nPart E. You expect aprox. equal proportions of the two types of recombinants. ',0,732,4,1),(##9-12-16&&

700     Q: Please explain the answer to 9-16 (B).&&

700     A: If ND occurs at first division, one cell gets both homologs and one gets none (for a single pair). Therefore gamete gets two alleles for every gene on that pair of homologs. If parent was heterozygous, gamete will be \"heterozygous.\" If ND happens at second division, gamete gets two sister chromatids instead of the usual one. Even if parent was heterozygous, gamete will be homozygous -- it will get two identical copies of the same chromosome (barring crossing over). In this case, if ND happened in mom at first division, son would get two X\'s -- one with cb allele and one with normal vision allele. Son would not be colorblind. If ND happened in dad, resulting XY would have a normal vision allele on the X and son would not be colorblind. If ND happened in mom at second division, son gets two chromatids from one X (instead of the usual one). If both are from normal X son has normal vision; if both are from X with cb allele, son is colorblind. So ND must be at 2nd div. in mom. ',0,733,4,1),(##9-12-16&&

701     Q: Why does a zygote produce a ball of cells that inactive one X chromosome? Why not inactivate one immediately, producing a uniform phenotype

701     A: We can\'t really say \"why\" anything happens; that\'s just the way it is. Sometimes there are evolutionary or functional reasons that explain how it got that way, sometimes not. In this case, it is probably advantageous to use both X\'s if you have two -- that way any defective alleles on one X are compensated for by good alleles on the other X. This is probably why inactivating one X half the time and the other half the time is better than inactivating one X or the other all the time. You might inactivate the \"good\" X and be stuck with the bad one. A uniform phenotype is not necessarily a good thing.',0,734,4,1),(##9-12-16&&

702     Q: What is the fastest way to find the frequency of an allele given the frequency of its carrier

702     A: If allele in question is rare, you can approximate as explained in the key in the problem book. Otherwise, you are stuck with a quadratic equation and you have to use brute force. ',0,735,4,1),(###-01-28&&

703     Q: In tissues, $CO_2$ is converted to $HCO_3^-$, which in\r\nturn is exchanged for $Cl^-$,  but what happens to the $HCO_3^-$

703     A: It is dissolved in the plasma (liquid part of blood). $Cl^-$ leaves plasma and bicarbonate enters. Remember that the opposite is true in the lungs, and that bicarbonate leaves the plasma and returns into the cells to be converted to $CO_2$.',NULL,1462,1,2),(##9-12-19&&

704     Q: What constitutes a primary transcript

704     A: Primary transcript is unfortuately not well defined, as it is somewhat subject to context. The usual meaning is in the context of the splicing process, in which it means a capped polyadenylated RNA

that is not yet spliced. But it could mean non-polyadenylated if one is talking about polyadenyaltion and wants to distinguish the not-yet-polyadenylated RNA (primary transcript) from the polyadenylated species. ',0,736,4,1),(###-01-30&&

705    Q: What is the significance of being connected to intermediate filaments in the case of desmosomes vs. being connected to\r\nmicrofilaments in the case of adherens?  Are desmosomes junctions for support while adherens junctions for contraction and\r\nmovement

705    A: Maybe. MF may be useful in both support and movement. But IF are clearly for support.',NULL,1464,1,2),(###-09-22&&

706    Q: Do ribosomes count as organelles

706    A: Ribosomes are usually categorized as organelles although they do seem quite distinct from the embrane-delimited organelles. I don\'t care for \r\nthe classification for that reason, preferring clean boundaries. They could also be considered \"supermolecular structures\" (another term used) or\r\n\"machines\" (more vernacular). But they make most organelle lists. Nucleolus is another with no membrane. But there is no reeason that E. coli could not have an organelle or two.',NULL,1384,1,1),(###-09-29&&

707    Q: I don\'t see the H from the water ending up anywhere in the some of the diagrams of the hydrolysis of ATP.&&

707    A: The H atom that goes to the phosphate that is formed immedialty dissociates its proton (its electron stays there) because phosphoric acid is a strong acid.',NULL,1385,1,1),(###-09-29&&

708    Q: In problem 3-11, is the histidine also competing with the inhibitor thiazolealanine

708    A: Histidine is not competing with thiazolealanine. Histidine is running around the block, not dealing with it or reversing it directly, just providing the endproduct (i.e., histidine). Histidine \r\nbiosynthesis, the biosynthetic pathway, is stll blocked by thiazolealanine, but the E. coli do not care since histidine is being provided in the medium.',NULL,1386,1,1),(###-10-01&&

709    Q: Problem 2-6 of the problem book states that in general the frictional coefficient of an enzyme composed of a single polypeptide chain is larger when it is denatured than when it is in its native state. If the denatured protein more or less resembles a sphere it should have a comparatively low fo, so is this inconsistent

709    A: Although the composite of all the structures taken on by an assortment of random coils can be considered a sphere, the radius of this composite sphere (about 2/3 of the exxtended length, I think) \r\nis much greater than that of the real sphere that is the result of a protein packing into a tertiary structure.  Thus although both can be considered spherical in this sense and of the same MW, the \r\ndenatured protein will have a much greater coefficient of friction.',NULL,1387,1,1),(##7-09-19&&

710    Q: Is the bond formed between a fatty acid and glycerol a glycosidic bond or an ester linkage?\r\n&&

710     A: The bond between the fatty acid and glycerol is an ester and not a glycosidic bond. If you compare it with the glycosidioc bond connecting two sugars in a disaccharide you will note the difference: there is no trace of the acid remaining in the glycosidic bond, while the C=O is stil there in the ester. A dehydration betten any acid and any alcohol produces an ester. The fatty acid is a carboxylic acid and it forms a carboxylic ester, a sub-category, or one kind of ester. Phosphoric acid plus and alcohol produce a phospho-ester. Since phosphoric acid has 2 acidic OH\'s, it can form two esters on the same phosphate, as in phosphatidyl ethanolamine, a phospho-di-ester.',0,739,1,1),(##7-09-23&&

711     Q: Can you tell me what form or forms of the Michaelis-Menton equation we\'ll be seeing on the exam? Will we see Km = k2 +k3/k1? Also will we see the Lineweaver-Burke equation

711     A: You would be given the basic form of the Michaelis-Menton equation, but you are expected to know the other relationships (e.g., Vmax). No Lineweaver-Burke plots will be used.',0,740,1,1),(##7-09-20&&

712     Q: I saw something about \"quizzes\" start next week. What does this mean

712     A: Yes, the quizzes are only for the morning class, C2005. No quizzes for F2401.',0,741,1,1),(###-10-01&&

713     Q: What is the difference between allosteric inhibition and noncompetitive inhition

713     A: Allosteric inhibition occurs via binding at a site other than the active site. It is not at the active site. It works from afar by inducing a change in the tertiary structure of the protein that results in a change in the conformation of the active site to affect either its ability to bind substrate (Km effect) or its ability to catalyze the conversion of a bound substrate to a product (affects the turnover number or Vmax). A noncompetitive inhibitor binds at or near the active site so as to block the ability of the enzyme to catalyze the conversion of substrate to product but does not (by definition) block the ability of the enzyme to bind the substrate at the active site.',NULL,1388,1,1),(##7-09-23&&

714     Q: Is it valid to call the building blocks  of a given lipid, monomers (e.g glycerol, fatty acids, phosphate, alcohol)? If so, then for example, can a monoglyceride be called a dimer? Or, are dimer, trimer, polymer, etc., terms that are reserved for things like polysaccharides or any chain built up using the same or very similar monomers? \r\n\r\n&&

714     A: Yes, strictly speaking a monoglyceride is a dimer of lipid building blocks. In the case of phospholipids or triglycerides, the total number of \'monomers\' reaches all of 4 or 5, so you are right is being suspect of the macromolecule moniker here. Yet when you think of the phopholipid bilayer, all lined up in the cell membrane it sure looks sort of macro. Lipids do not fit well in this classification scheme, but it is not too important as long as we all know what they are and what they are used for. ',0,743,1,1),(##7-09-23&&

715     Q: On page 176 of Becker, he states that glycophorin and the anion channel segement are integral proteins, while proteins such as spectrin, ankyrin and actin are peripheral proteins. However, he goes on to state that ankyrin (peripheral?) links the anion channel protein (integral?) to the tetramers of spectrin (peripheral?), and thus anchor the membrane to the cytoskeleton. How can a peripheral protein link a integral protein to another peripheral protein and thus, according to the logic of Becker, anchor the membrane to the cytoskeleton (interior of the membrane)

715     A: I think you and Becker are defining peripheral proteins a bit differently. You are trying to be very distinct, while he is calling peripheral membrane proteins all proteins that are found associated with the membrane, yet do not permeate into the interior of the lipid bilayer, sort of an operational definition. Thus he can call both ankyrin and spectrin peripheral membrane proteins, with ankyrin effecting this association via binding to the integral membrane protein anion channel and spectrin gettinig associated via binding to ankyrin. The picture on page 176 shows it all nicely, whatever you choose to call them you can see what's going on there. Just think of peripheral membrane protein as proteins that associate with the membrane indirectly,through protein-protein interactions. ',0,744,1,1),(##7-09-23&&

716     Q: What does the term \"ala\" mean

716     A: Ala is the 3-letter abbreviation for the amino acid alanine. Each of the 20 amino acids has a three-letter abbreviation. See your text for these. They also have one-letter abbreviations, but wee are not using these in this course. ',0,745,1,1),(##7-09-23&&

717     Q: How does one determine the form of an amino acid at a given pH

717     A: The only acids we have discussed are carboxylic acids and phosphoric acid. These acids are fully ionized, i.e., they lose their acidic proton (H+) at pH's above 4. At pH's less than 2, the carboxylic acids will not lose their protons, as the high concentration of H+ at this pH drives the association reaction back to the protonated form. Intermidiate situations exist at pH's between 2 and 4, where a fraction of the total number of molecules inthe solution are ionized and a fraction un-ionized at any given moment, but we will not concern ourselves with those cases here. The only base we have considered is the amine group. The amines willtake on a an H+ from the solution at pH's less than 10, and so will be positively charged. At higher pH's the reaction is driven toward dissociation by the scarcity of H+'s in solution. Almost all of the molecuels will be uncharged, divested of their extra protons, at pH 12. As above, we will ignore the intermediate cases at pH's 10-12. Polypeptides have lots of amino groups and carboyxylic acid groups among their side chains. The net charge on a polypeptide will be depend on the number of acidic and basic groups it contains, and the pH atwhich it finds itself, according to the rules above. For more information, it would be best to consult a general chemistry book, rather than our texts. ',0,746,1,1),(##7-09-23&&

718     Q: Are \"polypeptide\" and \"subpeptide\" also acceptable answers for problem 2-13A (which shows an octapeptide described as having been derived from a protein by hydrolysis)

718     A: Subpeptide is fine, but polypeptide is not. First it is a stretch to call a mere 8 amino acids strung together \"poly\" rather than \"oligo.\" More importantly, in the context of the question, the molecule from which this octapeptride was derived was itself a polypeptide, or a group of polypeptides that constitued a protein. Thus there would be no distinction between precursor and product, which is what is being sought here, so that we can communicate about this procedure (the hydrolysis) ',0,747,1,1),(##7-09-23&&

719     Q: Does the -OH have to be terminal for something to be considered an alcohol? For example, is CH3-CH2-CHOH-(CH2)3-CH3 an alcohol

719     A: No, it does not have to be terminal. And yes, that is an example of an alcohol. ',0,748,1,1),(##7-09-23&&

720    Q: In problem 2-14: Upon removal of urea and renaturation, why doesn\'t the peptide bond between ser and ala reform

720    A: For all the macromolecules we are discussing, the formation of the linking bond is a complex, energy-requiring process that we will discuss in the coming weeks. Very few of the reactions we are discussing will take place at a practical rate without the aid of enzyme catalysts to speed things up. These enzymes are not present in the experiment (only the protease was added, to effcet the specific hydrolysis as described.). ',0,749,1,1),(##7-09-23&&

721    Q: Is there ever a situation that a hydrogen or hydroxyl is equatorial and in, or does equatorial always coincide with out

721    A: There is no \"in,\" as there is no room in there. Also think of the tetrahedral arrangement of the bonds about carbon: all the ins are used to form the ring in the first place. So equatiorial is always out, and axial can be either up or down. ',0,750,1,1),(##7-09-23&&

722    Q: Concerning question 1c of 1996 C2005 exam 1: Is the bond this disaccharide is refering to an alhpa or a beta bond? Also, I thought that alpha glycosidic bonds were always for starch and glycogen (storage polysaccharides), while beta bonds were for cellubiose (structural polysaccharides). Is this correct

722    A: That is correct for the polysaccharide made up of glucose connected to glucose in 1,4 linkages. But in theory, we could think of molecules that are connected in other ways, between other hydroxyls, e.g., using the 1,6 linkages seen in the starch branches. Or think of molecules that are not just glucose, but contain other sugars, like galactose, as I did in this question. The term alpha and beta have been used here to denote the conformation of the OH at the anomeric carbon, that is, the carbon that can form in 2 alternative conformations when the ring closes. We can extend this nomenclature to any sugar that closes into a ring. ',0,751,1,1),(##7-09-23&&

723    Q: In question 4 of the 1996 exam, could you please tell me what the difference between glucose and ATP are in this problem? Are they both substrates? Also what is ADP

723    A: Yes, they are both substrates. ADP is a product ',0,752,1,1),(##7-09-23&&

724    Q: In part 4D of the 1996 exam, why is the answer #200,000 instead of 200,000. What does the # sign represent

724    A: The answer is less than or equal to 200,000. That\'s what comes up in the answer box on my computer when I click for the answer. AH, I see, when I cut and paste it here in Courier font, it comes up as #. \'(#200,000) [sic. should be less than or equal to 200,000]. If 0.001 M were a saturating concentration of ATP, then the conditions would be the same as the initial description, which yielded a rate of 200,000. However, since we do not know the Km of the enzyme for ATP, it could be that 0.001 M ATP is less than saturating, in which case the rate of the reaction should decrease according to the Michaelis-Menton relationship between Vo and S where S refers to ATP (rather than glucose). [That is, there should also be a Vo vs. S curve in which the x-axis is ATP concentration rather than glucose concentration. Without further information, the simplest assumption is that each substrate binds to the enzyme independently.] It cannot be >200,000, since the reaction rate is limited by the

concentration of glucose, present at its Km concentration. I\'m not sure how to fix this, but we will try ... ',0,753,1,1),(##7-09-23&&

725     Q: Are we to assume that the proteolytic enzyme remains in solution with the ribonuclease, or, once a peptide bond is broken, can it be reformed without outside help

725     A: It would be simplest to assume that the protease has been removed in an experiment of this type, testing renaturation of the target polypeptide. But it should make no difference here, as the peptide bond that was broken is not reforming. It cannot reform without outside help. ',0,754,1,1),(##7-09-23&&

726     Q: What about our mantra of \"1 (primary) structure determines 2, 3, and 4 structure\" when broken primary structure doesn\'t seem to upset the other levels of structure.&&

726     A: That\'s the trouble with mantras, mind-blanking repetitions can be hazardous here. Primary structure can be and often is sufficient to detrmine higher order structure, But is it also absolutely necessary? Yes, in the extreme: hydrolysis all the way to individual amino acids (via concentrated acid treatmnet, e.g.) would destroy any chance of assembling into a specific structure, the bonds are too weak, and the possinbilities are not constrained. But atthe other extreme, considered here, of just one little cut ... it leaves two good sized polypeptides, each of which could fold into perhaps the same structure thay had in the original protein, with their outside surfaces able to interact with each other to hold then together. ',0,755,1,1),(##7-09-23&&

727     Q: I\'m having trouble identifying phosphate. In Becker on pg. 57, the picture of a phosphate group includes 4 oxygens. In our multi-page handout (pg.2), the picture of phosphate includes only 3 oxygens. In the problem book, the answer to question 1-14B (i) shows that the phosphate contains only 2 oxygens (if bonds 6 and 7 are broken). What am I missing

727     A: Phsphoric acid contains 4 oxygen atoms; its formula is H3PO4. When it is esterified to an alcohol like ROH an oxygen is lost from the the two partenrs getting together (it is a dehydration), so you end up with R-O-PO3. Although we may call it the \"phosphate,\" what is really meant is what is left of the phosphate after it has been esterified. It is now a phosphate ester, not a phosphate per se, but we need to talk about the residue (like an amino acid residue in a polypeptide), so we call it the \"phosphate.\" The oxygen between the R and P is shared by what was the alcohol and what was the acid, that is, the phosphate. The oxygens \"belong\" to the phosphate part of the ester as much as to the alcohol, but we may sometimes carelessly exclude them when it is not important to think about it, or when we are indicating where an addition of water might take place to regenerate the phosphate. ',0,756,1,1),(##7-09-23&&

728     Q: How can I determine the polarity of a molecule? For example, a molecule of alcohol, a molecule of acid, and a molecule of ester, which is most polar, and which is the least polar

728     A: It is difficult to predict the relative polarities among various polar compounds, but some distinctions can be made. Copmound with fully charged groups, such as an ionized acid, like a carboxylic acid or phosphate or sulfate, or an ionized base such as an amine willbe more polar than molecules carrying only a partial separation of charge such as hydroxyls, esters, and amides. Moleucles carrying more than one such group will be more polar than those carrying only a single polar (or charged group). Thus I would expect an amino acid like glycine to be more polar than acetic acid, and glutamine to be more polar than threonine (checkout the structures in your text). Without being told,it

would be difficult to predict some cases, such as cyteine with an R-SH group (not very polar I think). ',0,757,1,1),(##7-09-23&&

729     Q: When glycosidic bonds form in glucose-glucose polysaccharide or glucose-fructose (or any combination) through the process of dehydration, why is it that hydrolysis will break these bonds when the bonds themselves look like ether (-C-O-C-) bonds (which normally are hydrophobic)? Why isn\'t water attracted more to the hydroxyl groups on the glucose rings

729     A: Speaking strictly of the chemical reaction (uncatalyzed) of hydrolysis, I am not sure why the glycosidic bond is hydrolyzed more readily than other bonds in the disaccharide. Perhaps it is because that carbon (carbon 1) is the only carbon bound to 2 oxygens. I do know that the glycosidic bond is subject to hydrolysis by acids. In any case, for BIO-chemical reactions, the reaction is almost always catalyzed by an enzyme. The enzyme acts very specifically to catalyze just one reaction. So the hydrolysis of a polysaccharide in the body or in a cell would be catalyzed by a glycosidase which works specifically on the glycosidic bond and no other. For instance by the enzyme amylase in saliva which starts hydrolyzing starch you eat even as you chew. Similarly, the dehydration that puts the sugars together is effected by a rather complex set of reactions; the 2 sugars are not simply brough together and water removed. Rather, one of the sugars is first attached to a \"carrier\" molecule and the second sugar then replaces the carrier. All steps here agaian are catalyzed by specific enzymes. We will touch briefly on this reaction later in the semester. ',0,758,1,1),(###-10-02&&

730     Q: Do hydroxyl groups ever ionize

730     A: Sometimes, if attached to an aromatic ring (DNP example in respiration lecture); for our purposes we will assume they never ionize.\r\n ',NULL,1389,1,1),(###-10-02&&

731     Q: Is there any difference between paper electrophoresis and gel (no sds) electrophoresis except for the medium in which the two separations take place? Can they be used interchangably

731     A: PAGE presents a sieving effect as the proteins have to worm their way through small passages. The spaces between the cellulose fibers in paper are like the the Grand Canyon compared to \r\nthe size of proteins. Notwithstanding, paper electrophoresis is never used for proteins. Perhaps they bind to the cellulose in disagreaable ways (smear?). And diffusion would be great (large \r\nblobs instead of bands in the end. The peptides and amino acids do this. (Start spotted the size of pea, end up the size of a large grape).',NULL,1390,1,1),(##7-09-24&&

732     Q: Since the polypeptide implicated in sickle cell amemia has a substitution of val, which is hydrophobic and uncharged, instead of glu, which is negatively charged, wouldn\'t paper electrophoresis alone be enough to determine the difference in these charges at this point? In other words, wouldn\'t the (normal sample) peptide containing the neg charged (glu) have moved to the cathode (+) side and the mutant peptide containing the val just stay in the middle

732     A: To answer the last part first: the peptide containing the mutant valine would not mecessarily stay in the middle: one has to know what the other amino acids are in this peptide. There could be a bunch of arginine and lysines, in which case it would move toward the cathode. You need to know the net charge, a composite value. As for the first part, in paper electrophoresis the mutant peptide will certainly behave differently from the normal, as it have one less negative charge. But there will be many peptides produced, and separation in one dimension will probably produce a crowded lane. Your moved spot may end up hidden under as normal unrelated spot. That\'s why the second dimension of

separation by paper chromatography is added: to increase the resolution of the system. ',0,761,1,1),(##7-09-24&&

733     Q: In the \"Glucose\" handout, the glucose cyclization picture shows a bond forming between C1 and C5. However, most other references seem to be to be a 1-4 bond. Which is correct

733     A: The cyclization takes place between C1 and C5 of the SAME glucose molecule; these two carbons join via an oxygen bridge to form a ring structure. In this ring there are more hydroxyls, one on each carbon except C5. These carbons can form bridges between different separate molecule to form dimers (disaccharides), trimers, etc. It is here than the 1,4 linkage comes into play. Glucoses can become attached to one another via the C1 of one molecule (molecule #1) connected to the C4 of the next molecule (moelcule #2). Then the C1 of molecule #2 can connect to the C4 of molecule #3, and so on. So you get a polymer of glucose attached to one another via the 1,4 linkage. Within each glucose ring the C1 is attached to C5 to close the ring. ',0,763,1,1),(##7-09-24&&

734     Q: Is there a difference between alpha and beta glucose? (Does alpha form 1-4 bonds and beta form 1-5 bonds?)&&

734     A: When glucose cyclizes to form a ring, the hydroxyl that is created by this process on C1 can lie either above or below the ring that can be drawn. Above is called the beta conformation of OH on C1, or just beta-glucose. Below is called the alpha conformation there, or just alpha-glucose. The alpha and beta molecules are distinct, although they are interconvertible in the free (unpolymerized) sugar. Either the alpha or the beta form of glucose is know to form 1,4 bonds with another glucose molecule. Some cells (e.g., in plants) will put glucoses together with 1,4 linkages, but only use beta-glucose. The result is cellulose. Other cells (e.g., in liver) will put only alpha-glucoses together to form a polymer called glycogen. The physical properties of cellulose and glycogen are quite different even though they both consist of glucose molecules strung together. ',0,764,1,1),(##7-09-24&&

735     Q: Will glucose connected via alpha-alpha linkages always form an alpha helix? And likewise, will glucose connected via beta-beta linkages form a beta pleated sheet

735     A: The alpha helix and beta pleated sheet are examples of secondary structure in proteins and have nothing whatsoever to do with alpha and beta glucose. The terms alpha and beta are simply ways to enumerate different form of similar things, such as the alpha, beta, gamma, etc. carbons of amino acids; or alpha and beta versions of software; or alpha, beta,and gamma rays of radioactivity, etc. ',0,765,1,1),(##7-09-29&&

736     Q: Do you have any tips or advice for pacing ourselves during an exam

736     A: I suggest bringing a watch, budgeting equal time for all questions for 3/4 of the exam time (~20 minutes each for a 4-question exam), then use the remaining 20-30 minutes to go back to finish, review, and/or polish your answers. ',0,767,1,1),(##7-09-30&&

737     Q: After the hydrolysis of cellulose, why does glucose exist in BOTH the alpha and beta comformations

737     A: After hydrolysis, the product is the free sugar. It WAS in the beta form when it was in cellulose, but the free sugar is in equilibrium betwen the alpha, the beta, and the straight chain form of the sugar. You need the hydroxyl on C1 to effect the cyclization,and that hydroxyl is tied up in the

glycosidic bond in cellulose, where it is locked into the beta form, because the enzyme that puts the polysaccharide together will only accept the beta form as a substrate. Once freed by hydrolysis, the sugar is free to resume its cyclization-decyclization equilibrium. ',0,769,1,1),(##7-09-30&&

738    Q: Is the cell membrane permeable to small ions? If this is true, H+ (the smallest possible ion) should be able to readily pass through the cell membrane, but then how is a concentration gradient maintained

738    A: The membranes are not permeable to ions, just to very small, even polar, molecules (see Becker p.203) ',0,770,1,1),(##7-10-06&&

739    Q: Is there any other source for supplemental problems that you know about? The problem sets are great, but I\'d like to have the opportunity to do more--I\'ve found that this is the best way for me to study.&&

739    A: There is a solution manual to Becker\'s 3nd ed. If it is not on reserve in the bio library, I can put it there. As to more problems, there are not very many books that have problems at the right level. Most problems are either too easy or too hard. We have chosen to limit the # of problems in the problem book to a large but doable number. If you really understand them all, you should be fine. ',0,771,2,1),(###-10-24&&

740    Q: What does the DNA at the telomeres code for

740    A: It doesn\'t code for anything, so it\'s loss is no big deal.',NULL,1396,2,1),(##7-10-09&&

741    Q: Why can\'t allupurinol be oxidized when hypoxanthine can

741    A: The site of oxidation (resulting in the addition of a hydroxyl grouip) on the smaller ring is a carbon atom in hypoxanthine but a nitrogen in allopurinol. This is apparently too much of a surprise for the catalytic site to handle.',0,772,2,1),(###-10-20&&

742    Q: On problem 6-5 B, why can\'t the H on C2 of the inosine make a H-bond to the carbonyl group on cytosine in a 3rd hydrogen bond between these two base pairs

742    A: H\'s on carbons don\'t form H bonds. The H has to be attached to an electronegative atom, usually N or O. (If the H is attached to a C, the bond isn\'t polar.) Also the distances are wrong -- \r\nthe H can\'t reach that far. (G has a NH2 in that position, and the H\'s from that group can H bond.)',NULL,1397,2,1),(##7-10-09&&

743    Q: On the noncompetitive inhibition diagram, if only 1/3 of the enzymes are affected (i.e. 2/3 are normal), why does it only reach 1/3 V.Max and not 2/3 V.Max

743    A: You have discovered that the picture of the molecules in the beaker is drawn incorrectly. The words are correct and consistent, presenting an example in which 2/3 of the molecules are inactive and 1/3 are normal at any given moment. I have corrected the picture to agree withthe text. ',0,773,1,1),(##7-10-09&&

744    Q: Why isn\'t the bond between the 1st and 2nd phosphate of ATP used for energy (to get AMP)

744     A: Sometimes it is, but indirectly. One way is in DNA synthesis, in which a di-phosphate is split off (\'pyrophosphate\'). The free energy is used to attach the AMP (or TMP, etc.) othe growing new DNA strand. The pyrophosphate is subsequently cleaved by the enzyme pyrophosphatase. That packet of free energy is not copuples to tha transfer of anything, but its release assures that the overall reaction will go forward (pulled). A second way the energy in the second phosphate bond is utilized is also indirect. There is an enzyme (myokinase) that catalyzes the reaction ATP + AMP <--> 2 ADP, Delta Go = ~0. Now the 2 ADPs can be brought back to ATP by the usual energy metabolism. If thisreactiion isconsdiered inreverse, it is a way to get useful energy (ATP) out of ADP molecules. The chain of events would be 2 ADP --> AMP + ATP; ATP --> coupled reaction + ADP; so net reaction would be 1 ADP --> useful energy + AMP. ',0,774,1,1),(##7-10-09&&

745     Q: What is the conceptual and mathematical difference between apparent and intrinsic values for rate constants for enzyme catalyzed reactions? What is the exact effect of each kind of inhibitor on the turnover number

745     A: Apparent here means that the Km, in the presence of a competitive inhibitor APPEARS to be a certain value based on its graphical measurement as the substrate concentration needed to drive the reaction at 1/2 its maximum velocity. This concentration is not a true indication of the affinity of the enzyme for the substrate, because substrate molecules mustwait artound for enzyme\'s substarte binding site to rid itself of inhibitor. Then the substarte nmust compete for binding with the inhibitor. So you need more than you would otherwise. The Km is DEFINED as (k2 + k3)/ k1. This number exists for any enzyme and its substrate,and can be recorded for posterity n a book. You just can\'t measure this number from an experiment in which a competitive inhibitor is present; you get the wrong number, a higher number than the real one. This wrong number is the \'apparent\' Km in this type of experiment. \r\n\r\nIn an analogous way, the turnover number of an enzyme would be measured incorrectly from an experiment in which a noncompetitive inhibitor were present. The Vmax wouldbe depressed, and if you knoew how much enzyem you added and then calculated k3 fromthe relationshipo k3 = Vmax/Eo, you would get an apparent k3 that was lower than the number determined inthe absencei of any inhibitor,and which can also can be written down for posterity. A competetive inhibitor would have no effect on the measurement of Vmax and therefore on the calcuation of of a turnover number.',0,775,1,1),(##7-10-14&&

746     Q: In the Krebs cycle, alpha-keto-glutaric acid goes to succinic acid with the loss of a CO2 (looking at the handout); however the succinic acid picked up an extra oxygen from somewhere (look at the bottom carbon). Where did it come from

746     A: The extra O\'s that appear from nowhere in the Krebs cycle all come from water. In this case, the AKG is coverted to an enzyme bound intermediate so the C=O at the bottom is connected to a R group on the enzyme. (Simultaneously, the C=O is oxidized to the COOH level). Water hydrolyzes the C=O enzyme bond, giving free succinic. The ultimate result is to oxidize the C=O on the end to a COOH. The reaction is similar to the entry step of the Krebs cycle and is quite complex; any biochem book will show you all the details. ',0,776,2,1),(##7-10-15&&

747     Q: Why isn\'t beta-pleated sheat should be more elastic than an alpha-helix

747     A: The beta pleated sheet is more flexible in the plane of the sheet, but is not stretchable further because all peptide chains are fully stretched out. In the alpha helix, you have something like a slinky which can still be stretched further but can pop back into place. If this is not clear, come by my office -- I have a neat toy which demonstrates this well. ',0,777,1,1),(###-10-24&&

748    Q: Becker says that the Krebs cycle generates 1 ATP in a step between succinyl CoA and succinate, whereas on the handout, there is \r\nno such step, but instead GTP generated (the text does not have GTP). Which is true

748    A: Either diagram is okay. They are essentially the same, but the two were designed to emphasize different points. Becker is right about where the phosphorylation happens, but \r\nyou get GTP, not ATP. The two nucleoside triphosphates are exactly equivalent energywise. (There is an enzyme to catalyze ADP + GTP <--> GDP + ATP, 0 delta G0.)  So in \r\ncalculating energy yields, we count the GTP as if it were an ATP, and get the same totals as Becker. (You may notice on the overall energy handout it says GTP [ATP] to make clear \r\nthey are equivalent.)<br><br>\r\nWe combined two steps and put in the net result, instead of the intermediates.  Becker put in two steps separately (although it is one enzyme) to emphasize that it is an \r\nexample of substrate level phosphorylation. Once you do that, our steps and Becker\'s are the same.<br><br>\r\nOn an exam, we would supply the handout from lecture.',NULL,1398,2,1),(##7-10-15&&

749    Q: Are the words \"origin\" and \"primer\" synonymous

749    A: No. The origin is the spot where the DNA polymerase complex binds and starts opening up and replicating the DNA. It is a DNA sequence. The primer is a short stretch of RNA, made by primase, every time a new DNA chain starts. A primer is needed at the origin and at the start of every Okazaki fragment. Remember a primer is not in the DNA; it is complementary to it. ',0,779,2,1),(##7-10-17&&

750    Q: What do you mean by the statement, \"You can only hybridize 2 chains at once?\" &&

750    A: Two single strands can hybridize to each other. A third one cannot stick to a part of either of the original two strands unless the two strands are separated (denatured) first. The picture I drew on the board ignored this fact.',0,780,2,1),(##7-10-22&&

751    Q: Is a promoter found on both DNA and RNA, or just RNA? What about a terminator

751    A: The promotor is usually not transcribed -- transcription starts nearby. So no promotor sequence is found in RNA. The terminator sequence is transcribed, and it is thought that it is the structure of the RNA (probably a self complementary hairpin) that leads to termination. ',0,781,2,1),(##7-10-22&&

752    Q: Succinic dehydrogenase converts succinate (-OOC-CH2-CH2-COO-) to fumarate (-OOC-CH=CH-COO-) by subtracting two hydrogen atoms. What exactly is the mechanism by which malonate (-OOC-CH2-COO-) inhibits succinic dehydrogenase

752    A: Shorter than succinate, it apparently still fits in the substrate binding site, where it acts as a competitive inhibitor. It cannot be oxidized as there are no H\'s on two adjacent carbons to extract. See Purves p. 127 for this and other inhibitors of SDH. ',0,782,2,1),(##7-10-22&&

753    Q: Since nucleic acids have basic side chains and phosphate \"connectors,\" are they pH sensitive, similarly to proteins

753    A: Yes, but not as much as proteins. Extreme pH\'s will denature nucleic acids.',0,783,2,1),(##7-10-22&&

754     Q: Is there a connection between the basic side chains in DNA found in the mitochondria, and the ETC system which pumps H+\'s out of the mitochondria

754     A: I don\'t think so. There is relatively little DNA in mitochondria, and protein pumps are found in other membranes in the absence of any DNA. The fact that the bases in DNA are \'basic\' has little consequence for most biological processes. ',0,784,2,1),(##7-10-27&&

755     Q: The promoter is the start sequence for RNA polymerase. Is the primer the actual RNA sequence that is built at the the promoter

755     A: No primer is made at the promotor. The promotor is (aprox) where transcription starts. The RNA chain being transcribed is made continuously from start to finish; no primer is needed. The primer is built each time a DNA chain is started during replication, not transcription. ',0,785,2,1),(##7-10-27&&

756     Q: The origin has been variously defined as the starting sequence for DNA polymerase and as the unzipping point for DNA replication. Which is it

756     A: The origin is where the DNA first unzips and the DNA polymerase complex, meaning DNA polymerase and associated enzymes, gets started. You are right that DNA polymerase itself can\'t do beans until a primer is made first (& all the unzipping and winding enzymes work too.) But the whole process of opening up and replication starts at the origin. ',0,786,2,1),(###-10-24&&

757     Q: About problem 7-7B. I don\'t see how the presence of excess denatured DNA allows all RNA from an E. Coli to hybridize to it. Wouldn\'t you get only one hybrid if all RNA is transcribed from the same sequence?\r\n\r\n&&

757     A: The trick is that there are many copies of the DNA. That\'s what it means by \"the DNA is in excess.\" You are doing this experiment in a test tube, so you can adjust the proportions of RNA \r\nto DNA. (In cell, on the other hand, you generally have multiple copies of the RNA but only one copy of the DNA.)',NULL,1402,2,1),(##7-10-27&&

758     Q: Is it correct that, at the origin, the RNA polymerase creates the primer which starts DNA replication

758     A: True, The first primers are formed at or near the origin, but additional primers are needed on the discontinuously synthesized (lagging) strand as fork moves down. And primase is considered different from the ordinary RNA pol used in transcription. The origin IS a specific sequence of bases which signals the unzipping and DNA polymerase beginning, and IS NOT any sequence the DNA \'chooses\' to unzip at. ',0,788,2,1),(##7-10-27&&

759     Q: Problem 4-11B says that the concentration of NAD in the cell will decrease when glycerol is metabolised through the glycolitic pathway, causing the ratio (NAD)/(NADH) to decrease. However, isn\'t the cell membrane impermeable to NADH, which is the reason that the net ATP yield is 36 instead of 38 (due to the expenditure of 2 ATP in order to shuttle the NADH across the energy expensive membrane)

759     A: NAD and NADH2 are synthesized and interconverted within the cell. The permeation that costs one ATP per pair of electrons is the transport within a eukaryotic from the cytoplasm into the mitochondria. No such trasnport is necessary in prokaryotes in order to access the electron transport

chain. The ratio of NAD to NADH2 within the cell will be sensitive to the metabolism that is producing or using up one or the other. In the case of an attempted anaerobic metabolism of glycerol via lactate fermentation, NAD is used more than it it produced, thus the ratio NAD/NADH will decrease (to zero). ',0,789,1,1),(##7-10-27&&

760    Q: Problem 4-9C says that NADH cannot be oxidized back into NAD due to the lack of the enzyme that catalyzes the pyruvate-ethanol reaction, meaning that ATP production is limited by the concentration of NAD (ie 1:1 yield of ATP to NAD). However, even if ethanol is not produced, can\'t fermentation still occur and produce lactate instead

760    A: In this case, there is one source of NADH oxidation: the loss of one NAD and then gain of NAD from lactic fermentation would mean that the yield of ATP would be independent of NAD concentrations, and therefore increase with each cycle of glycolosis...or else be dependent on the initial limiting concentrations of ATP to phosphorylize the glucose and fructose-6-phosphate. for example. Usually, a given organism will only use a single fermentation pathway.',0,790,1,1),(###-10-24&&

761    Q: When the H+\'s are coming through the ATP synthetase, how many of them does it take to get the complex to do ADP + P --> ATP

761    A: I do not think it is known how many H+\'s are needed, but it seems reasonable to think it is one for each C subunit in the base.  This number is 10 to 14 depending on the species.  So if it\'s \r\n12, that would be 4 H+\'s for each of the 3 ATPs that is produced in one turn.',NULL,1401,2,1),(##7-10-27&&

762    Q: Problem 5-6E assumes that the two protons gained from the Krebs cycle (first from the conversion of succinate to fumerate and then from the conversion of malate to oxaloacetate) are the actual protons that attach to the oxygen after the ETC is completed and forms water. How are we to know that the protons that were given up by the oxidating agents are one and the same which form water later

762    A: Oxygen will just pick up protons from the general pool of these hydrogen ions after it accepts the electrons from the last cytochrome. Along the same lines, the protons from glucose join this pool when they are removed from NADH2 and FADH2 as these compound donate their electrons to the cytochromes. Actually, in the caseof NAD, since NADH2 is really NADH + H+, half the protons go into this pool concomitant with NAD reduction. But the pool of protons is in constant equilibrium with water: H+ + OH- <--> H20. (And at pH 7,the equilibrium is far in favor of water, although this is not strictly relevant). So the 3H label ends up in water anyway. ',0,792,2,1),(##7-10-27&&

763    Q: Problem 5-4 did not specify that the inner membrane is turned outwards when forming a discrete vesicle on its own. Is this assumed, general knowledge that we should have learned

763    A: You can deduce the inside-out nature of these vesicles from the information provided; that is part of the question. 1) NADH2 is being oxidized,so it must have access to the electron transport chain proteins, which are located on the inside of the mitochondrial membrane; if we assume that there is no mechanism for NADH2 transport into these vesicles (all components are defined: just the vesicles plus small molecules present), then the vesicles are probably inside out. 2) More importantly and clearly, the problem states that the pH of the solution outside the vesicles RISES concomitant with NADH2 oxidation. Thus there si a drop in hydrogen ion concentration outside the vesicles, so the

hydrogen ions must be being pumped INTO the vesicles. Since in right side out mitochondria, the hydrogen ions are pumped OUT,these vesicles must be inside out. ',0,793,2,1),(###-10-23&&

764    Q: For question 6-9B, why isn\'t the 2nd generation lighter than the 1st generation

764    A: You can\'t get lighter than all light. Just because 1st generation is lighter than 0th, it doesn\'t mean you can get any lighter the next time. You may get heavier, as explained in the key, but you can\'t \r\nget lighter. Once you have made a new chain out of all light raw materials, that\'s as light as you can get.',NULL,1400,2,1),(##7-10-27&&

765    Q: How is the Gibbs free energy of glycolysis -18 kcal/mole after the production of pyruvate

765    A: The delta Go for the reaction glucose --> 2 pyruvates is -18 kcal/mole. One way this value can be calculated is by adding up all the individual reactions that take glucose to pyruvate in the glycolytic pathway. ',0,794,1,1),(##7-10-27&&

766    Q: Where does the extra H on carbon #3 of pyruvate come from, after the production of ATP in the final step of glycolysis

766    A: You may think of it as the H atom from ADP, as it is displaced by the phosphate group in the formation of ATP. After formation of CH2=COH-COOH,this compound rearranges to form CH3-CO-COOH. In reality, that acidic hydrogen of phosphate is in equilibrium with water, so the actual hydrogen atom is drawn from a pool. Also, the mechanism of the final rearrangement probably involves intermediate reaction with water (addition across the C=C double bond followed by elimination across the C-O bond). But we are not delving into the chemical mechanisms at this depth here. ',0,795,1,1),(##7-10-27&&

767    Q: 3-Phosphoglyceric acid has an extra H attached to carbon #1 where phosphate used to be (when the molecule was 1,3-diphosphoglyceric acid. Where did this extra H come from

767    A: Do you mean the H of the COOH group? Then you should know that this H is not really there most of the time, as the carboxylic acid group is mostly ionized to COO- at pH7. If you wish, you may trace this H back to the OH on the end of ADP,whichgives it up upon accepting the terminal phosphate to become ATP. But this H would not stay long on either phosphates or carboxyls. ',0,796,1,1),(##7-10-27&&

768    Q: How does pyruvate permeate the inner membrane of a mitochondrion in order to go from the final step of glycolysis in the cytoplasm, to the first step of Krebs Cycle in the matrix

768    A: Good question. In fact, the transport in and out of the mitochondria of many of the different molecules we have discussed is a problem for eukaryotic cells, which solve it by using some of the energy inherent in the proton gradient. For example, the import of pyruvate into the mitochondria is apparently directly coupled to the export of hydrogen ions. Thus the number of 34 ATPs from electron transport-driven oxidative phosphorylation is a maximum that is probably not in fact entirely realized. See Becker, p. 368 (and p. 372 prob. 12-9). ',0,797,2,1),(##7-10-27&&

769    Q: Why does the enol form of pyruvate LOCK? What is the chemical significance of -OH on C2

769     A: The keto form of pyruvate is actually much more stable, and most molecules of pyruvate will have carbon 2 as a ketone. But in phosphoenolpyruvate, carbon 2 cannot form a ketone because the phosphate is there, so it is phosphoenolpyruvate that is locked in to the enol form, not pyruvate. It is this difference that makes the hydrolysis of the phosphate from phosphoenolpyruvate so exergonic: the resulting pyruvate can now take on the more stable keto form. See. Becker p. 311 for half a page on this subject. ',0,799,2,1),(##7-10-27&&

770     Q: What does Becker mean when he says \"fermentation...is energetically wasteful because the cell has access to only a limited portion of the total free energy that is potentially available from the oxidizable molecules it uses as substrates\" (ch. 12, p. 327)

770     A: Just that oxidation of glucose all the way to $CO_2$ and $H_2O$, as occurs when you set glucose on fire (combustion) produces 686 kcal/mole, whereas the limited oxidation represented by the conversion of glucose to 2 lactates releases only ~46 kcal/mole (32 after harnessing to the production of 2 ATPs). If you would burn the lactates you would get the remaining 686-46 = 640 kcal/mole released. (But you only get the lactates in the absence of oxygen, and you can\'t burn them without oxygen.) See Becker p. 314 first column. ',0,800,2,1),(##7-10-27&&

771     Q: How do specific tRNAs recognize their respective amino acids

771     A: The tRNA\'s don\'t recognize their aa\'s directly; the proper loading enzyme recognizes both the right tRNA and the right aa and pairs them together. How this works varies -- different enzymes recognize different features of their cognate tRNA\'s. Yes this is very important -- how it works has been called \"the second genetic code\" (the codon-anticodon match being the first code). The second code is still not completely worked out, which is one reason we said less about it. ',0,802,2,1),(###-10-23&&

772     Q: About Problem 6-6C. There is a line that reads:\r\n\"When you break down the DNA to get 3\' monophosphates, the\r\nradioactivity is found in all 4 different monophosphates - dGMP,\r\ndCMP, etc. When you break down the DNA to get 5\' monophosphates,\r\nall the radioactivity is in dGMP.\" How does cutting to make\r\n3\' monophosphates make everything radioactive

772     A: There are different enzymes and/or uncatalyzed chemical reactions that break the bonds differently. The usual enzymes & reactions in cells give you 5\' phosphates, but using different \r\ntest tube conditions, you can get 3\' phosphates.',NULL,1399,2,1),(##7-10-27&&

773     Q: In problem 7-6 A.(ii), is another possible answer that the gene which makes this specific tRNA is mutated and is making a tRNA which holds glu but has the anticodon for asp

773     A: No, that isn\'t reasonable. The mutation you described would cause glu to replace asp in many dif. proteins. Problem says only one protein is changed -- In this protein only, one asp --> glu. The same tRNA is used over and over, to translate different mRNA\'s for different peptides. If something is wrong with a tRNA, many different protein molecules will be defective, not just one kind. ',0,803,2,1),(##7-10-28&&

774     Q: In Purves it mentioned that an organism could not live long in a closed system, because it would soon come to equilibrium. Is this true, even under the conditions of, say, yeast on a minimal medium in a glass jar

774     A: Yes. Once the yeast uses up all the nutrients in the glass jar, there will be no more growth. Usually, the medium is replaced before that happens, so you don\'t really have a closed system. ',0,806,1,1),(##7-10-28&&

775     Q: Since telomerases attach at the 5 prime end of the complementary strand, do they copy 3\' to 5\', or are they large enough to cover the whole telomere and copy it without moving

775     A: The mechanism of action of telomerase is very complex. The enzyme does not use the DNA as template at all -- it contains its own template, made of RNA. For details, see Becker or Alberts. ',0,807,2,1),(##7-10-28&&

776     Q: What is the relevance of enol and keto forms of the nucleic acids in relation to bonding, structure, role in DNA, etc.

776     A: The structures are there for demonstration purposes. The alternative forms make different H bonds, which was important in figuring out the structure of DNA (historically). Until Watson and Crick picked the right tautomers, they couldn\'t figure out why A= T and G=C. Secondly, the tautomers may be important in mutation -- if the bases take the enol forms now and then, they will mispair and cause mistakes in DNA replication. That\'s basically it, for this course. ',0,808,2,1),(###-10-25&&

777     Q: About #4B on last year\'s exam. It is unclear in the question whether this G is being added in the beginning (i.e. the primer) or to the rest of the strand. Does the fact that it says a \"growing DNA \r\nchain\" imply that it has passed the primer stage of growth and is now using DNA polymerase to catalyze growth

777     A: The short answer is Yes. Since it says \"growing DNA chain,\" you must be growing DNA, not RNA. So you must be past the primer. This sort of uncertainty can sometimes be handled \r\non exams by asking the proctor.  If s/he won\'t clarify the question for you, then answer what seems to be the obvious meaning, and then add, BUT if you mean....\r\nJust be sure to explain the obvious answer first, and not as a last resort.',NULL,1403,2,1),(##7-10-28&&

778     Q: In problems that ask for the number of ATP molecules produced, should we typically specify the net or total production

778     A: We usually mean net in such questions, as it usually was when presented in lecture. But if it is unclear on an exam, that\'s a valid question to ask during the exam.',0,810,2,1),(##7-10-30&&

779     Q: We\'ve learned that the tRNA translates the mRNA from the mRNA\'s 5\' end to its 3\' end. However, figure 11.19 in purves (page 261) suggests that the tRNA itself is running from its 5\' end to its 3\' end, which would mean that the tRNA anticodon is hybridized with the codon in the mRNA, and that the two fragments are parallel and not antiparallel. How is this explained

779     A: All nucleic acids always pair antiparallel. This is sometimes ignored in pictures to keep things simple, but pairing is always antiparallel. The picture on p. 261 is showing the tRNA all by itself, not paired to mRNA. It shows the tRNA 5\' on left because that is the usual convention for nucleic acids.',0,811,2,1),(##7-10-30&&

780     Q: Are the tRNA and the mRNA antiparallel during translation

780    A: tRNA--mRNA pairing is antiparallel. See web notes (lect 13). ',0,812,2,1),(##7-11-10&&

781    Q: Regarding exam 2, question 2C, in which one is asked how much ATPs will be produced in the presence of cyanide and dinitrophenol. In the presence of cyanide, why are 2 ATP rather than 0 formed? Since cyanide stops the Krebs cycle, glycolysis, and ETC, it seems that, although 2 ATP can be produced in the first run, this would not be possible afterwards since there is no fermentation due to the presence of oxygen.&&

781    A: The answer of 2 ATPs in the presence of cyanide assumes that fermentation takes over once oxphos is blocked by cyanide. A clue to this switchover lies in the fact that it is stated that the cells grow in the presence of cyanide (and air, although the air here would be making no difference). If no net ATP were being made, then the cells would not grow. If you clearly explained that you were assuming that oxygen would inhibit fermentation and therefore no ATP would be produced, then you should have received some partial credit, depite the growth discrepancy. ',0,814,2,1),(###-11-29&&

782    Q: In 11-6 (C), to which colonies do the statement \"if you select for pro+ colonies, you get a significant number\" refer

782    A: It refers to the recombinant bacteria. Recombination does, in fact, produce a signif. number if you select for recombinants -- that is, test enough progeny so even a small % of the total is a \r\nbig number.',NULL,1424,3,1),(###-11-30&&

783    Q: Are caps and tails considered to be part of the UTRs, or are they added ON TOP OF the UTRs

783    A: They\'re add on top of the UTRs.',NULL,1425,3,1),(###-11-30&&

784    Q: Does the twisting of DNA matter, such as when a plasmid is incorporated into a chromosome and it does a figure-8

784    A: All DNA is really twisted (super coiled). The amount of twisting can matter -- depends on the situation. (There are enzymes to control the increase or decrease of twisting as needed.)',NULL,1426,3,1),(###-11-30&&

785    Q: In lecture 18, the picture of \"splicing details\" says that this is DNA strand shown is transcribed starting at the 5\' end. Doesn\'t transcription always go 3\' - 5\'

785    A: The picture shows the sense strand. Any time when only one strand is shown, it is virtually always the sense strand. ',NULL,1427,3,1),(###-11-30&&

786    Q: What is a complementation test

786    A: When you put two mutants together and see if they restore function, it is a complementation test IF you can rule out crossing over.',NULL,1428,3,1),(###-11-30&&

787    Q: In a complementation test, how do you rule out crossing over

787    A: You often can infer from the set up whether recombination is possible or likely to explain the results. If every cell with two copies has restored \r\nfunction, it must be complementation.',NULL,1429,3,1),(##7-11-11&&

788    Q: Why do chromosomes exist as sister chromosomes in meiosis? Why can\'t they line up as single chromosomes, cross-over and form haploid cells, thus going through a shorter cycle

788    A: If you or I were planning meiosis, we would do it as you have discussed. However, that\'s not the way it is! Meiosis starts with doubled vchromosomes, and once that happens, there have to be two divisions etc. Current opinion is that meiosis evolved from mitosis, and therefore starts the same way, with doubled chromosomes (2 chromatids per \'some). In other words, what we have is the result of random mutation plus selection for what works, not a system that was planned or designed in advance. A variant of mitosis occurred that allowed reshuffling of genes and chromosomes, and the advantages of the reshuffling were so great that organisms that did the \'shuffle\' out reproduced the others. So here we are! ',0,818,3,1),(###-12-01&&

789    Q: The answer to problem 12-2 C (ii) says that the number of repressor proteins bound to the\r\noperator will remain the same at zero. But since the operon is now inducible and no lactose is present, wouldn\'t the\r\nrepressor protein be bound to the operator, inhibiting transcription

789    A: There\'s no operator to bind HIS repressor. Only lac repressor has any effect. Two separate repressor proteins!!',NULL,1430,3,1),(###-01-30&&

790    Q: In problem 2-2 from, how do you know ions are not being moved through the protein (e.g. through channels or pumps/permeases/carriers)

790    A: First of all it says F and G are neutral. That kills ions. Second of all,\r\ntransport of both F and G is  dependent on energy. That kills channels.',NULL,1465,1,2),(###-02-06&&

791    Q: Is the luminal concentration of glucose  always strictly lower than the concentration in the epithelial cells lining it, or is it dependent on diet

791    A: This is something I should really discuss in class. Some people think there is a standard glut transporter on the apical surface. When Glucose in the lumen is very high, for \r\nexample, as you say, after eating cotton candy, then that transporter may be sufficient to let the glucose into the epithelial cells. But most of the time the glucose in the lumen is low \r\n(remember anything you eat is diluted in the entire volume of your stomach, intestine, etc.), and then the co-transporter takes over.\r\n\r\n<p>I do know that when you have cholera, and the contents of your guts are washed out by frequent diarrhea, replacing the contents with plain sugar isn\'t sufficient -- you can\'t take \r\nup the sugar efficiently. You have to drink a mixture of sugar and salt for the sugar to be taken up efficiently. This implies that either there is no \"downhill\" transporter, or that it can\'t \r\ntransport enough glucose to make a significant difference. It\'s the co-transporter, which is dependent on sodium, that seems to be the major mover of sugar into the cells.\r\n ',NULL,1466,1,2),(###-12-18&&

792    Q: I\'ve always gotten the impression that the real test of a theory is its ability to make accurate predictions. Are evolutionary scientists able to make and test such predictions? If so, what sort of predictions do they make/test? If not, is that considered a weakness of the theory

792    A: We can\'t do macroevolution experiments. We can do microevolution\r\nones. We\'re in the same position as historians -- can you say what\r\nreally happened? Can you say something are much more likely to have\r\nhappened in the past than others? However as we keep uncovering data\r\n(like more protein and DNA sequences) evolution by nat. selection\r\nkeeps looking better

and better. But we do not have specific\r\npredictions about what we will find (except that all living things\r\nwill work about the same).',NULL,1438,4,1),(##7-12-07&&

793     Q: Where do biologists stand on more ambiguous phenotypes such as alcoholism, perfect pitch, or a tendancy to anger easily

793     A: Most biologists feel that genes influence behavioral traits and personality just as they influence everything else. After all, what does a gene do? It codes for a protein and the sum total of actions of all the proteins determines (in large part) how a cell and/or person is constructed and/or acts. However, the genes do not determine the final outcome completely -- they set the possibilities and then the environmental conditions (including the effects of other genes) determine whether the genetic limits are reached or not. There is considerable argument over what % of say, musical ability, is genetically determined and how much depends on other factors. Perhaps the most important thing to keep in mind is that most interesting traits (such as musical ability, personality, etc.) do not depend on the state of a single gene but on the states of many different genes acting in concert. So there probably is no one \"gene\" for perfect pitch, or schizophrenia, etc. even if genes greatly influence musical ability and/or mental function. These issues are discussed in many venues; you might try any standard human genetics book. Of course the problem is that almost everyone who writes on these subjects has an axe to grind and makes the genetics fit their theories instead of the other way around. ',0,822,4,1),(###-12-02&&

794     Q: In problem 13-3, the restriction site for Dde sequence isn\'t a palindrome, so how can the sequence be split

794     A: Some restriction sites are almost but not quite perfect palindromes.\r\nLike this one.',NULL,1436,3,1),(###-12-18&&

795     Q: Do creationist criticisms that the theory of evolution is untested and perhaps untestable have any merit

795     A: There is no evidence whatsoever for creationism. There is excellent\r\nevidence for evolution. Creationists generally find the theory of\r\nevolution unacceptable for one nonscientific reason or another and\r\ntherefore assume it must be wrong. Therefore they try to pick holes\r\nin evolutionary theory, but they have no evidence at all for\r\ncreationism (or that evolution is really wrong).',NULL,1437,4,1),(##7-12-07&&

796     Q: It is naive to believe that students are purely driven by the satisfaction of learning new material. The student\'s primary concern is the final grade he or she will earn by the end of the semester.&&

796     A: We have no illusions that students are purely driven by the love of learning, however we consider that at least for some students, this might be a factor. We understand that for many (most?) students the primary concern is the grade. Therefore we do our best to make it pay to learn as much as possible -- in other words, in order to get a good grade (so you\'ll be pleased) you have to learn a lot (so we\'ll be pleased). As we see it, it is our job to set the incentives so that you learn the maximum amount, not so you get the max. grade for the least work. If there are ways to do this that you think are fairer and/or involve less stress, we would be happy to hear them.',0,823,4,1),(##7-12-09&&

797     Q: Would you recommend revision of old material for the final exam

797     A: Only go over old material as it is needed to answer new questions. For review, try problem set 15 -- start with the end and work backwards, as earlier questions (in the prob. set) are from older exams and less like our current thinking. ',0,824,4,1),(##7-12-09&&

798     Q: Problem 10-18 (E) says that \"crossing over does occur (rarely) between the alk. phosphatase gene and the WS gene.\" Does this  mean that the alleles of each gene may cross with each other, or that one allele from each different gene can cross over

798     A: It means crossing over can occur in the space between the two genes. That means that either allele of either gene can end up on the same chromosome with any allele of the other gene, due to exchanges.',0,825,4,1),(##7-12-09&&

799     Q: What do you mean by \"Mendelian gene?\"&&

799     A: Mendelian gene means acts like the genes discovered by (really inferred to exist) by Mendel. He was the first to posit that genes existed and had certain properties; that\'s why he was honored. A Mendelian gene controls a single trait, has 2 alleles (unless specified otherwise) with one allele completely dominant over the other, and is not sex linked. In other words, if you have two forms of the trait in two inbred strains, say tall and short, and you cross the two to get first an F1 and then an F2, and the F2 is 3:1 then you say height is controlled by a single M. gene. ',0,826,4,1),(##7-12-09&&

800     Q: The solution to problem 14-4 (A) states that the frequency of Rh- is O.3, the square root of 9%. However, doesn\'t the frequency of Rh- also include the number of recessive alleles of the carriers, i.e. f(Rh-)= q + 2pq

800     A: No. You are confusing the freq. of Rh- with the total number of Rh- alleles. ',0,827,4,1),(##7-12-09&&

801     Q: In problem 14-4, why don\'t you include the carrier alleles in the frequency of Rh-

801     A: The total number of Rh- alleles includes those in heterozygotes and those in homozygotes. However, the frequency of the Rh- allele can be calculated directly from the freq. of homozygotes if the population is in gen. equil. Given gen. equil, There are two ways to get the frequency of Rh-. It\'s the total number of Rh- alleles over the total number of alleles (whether there is g.e. or not) OR it\'s the sq. root of the number of Rh-Rh- homozygotes (only if there is equil.) The second way means you can calculate the freq. of Rh- from the chance of picking two Rh- in a row; you don\'t need to figure out first the number of Rh- in heteros. If it still isn\'t clear, look at the derivation or ask me. ',0,828,4,1),(##7-12-09&&

802     Q: What is cDNA

802     A: cDNA is complementary DNA = DNA made using mRNA as a template. (Using the enzyme reverse transcriptase.) cDNA can be single stranded or double stranded, depending on how you make it. (first mRNA acts as template --> ss cDNA; then you can use the ss cDNA --> ds cDNA). ',0,829,3,1),(###-12-18&&

803     Q: What are the units for allele frequency?  Should we report it as a percent or as a decimal, or does it not matter

803     A: You can report it either way, but in doing calculations, always use\r\ndecimals or fractions, not %.',NULL,1441,4,1),(###-12-18&&

804     Q: Does nondisjunction in meiosis (in both divisions) ever occur for more than one chromosome at once

804     A: Nondisjunction usually only occurs for one chromosome out of many, not for all of them at the same time.',NULL,1442,4,1),(###-12-18&&

805     Q: If a gamete ends up losing a chromosome to another in nondisjunction, is it still considered N?\r\n&&

805     A: You would call an aneuploid gamete something like \"nullisomic\" or\r\ndisomic if it has none or 2 when it should have one. It is still basically N, but with one chromosome extra or missing.',NULL,1443,4,1),(##7-12-09&&

806     Q: In problem 10-18 (E-2), why are there no 2-2 normal progeny. The parents are 2D/1d x 2d/1d, so if we cross and get a recombinant with the final genotype of 1D/1d, then shouldn\'t there be a resulting offspring with genotype 2d/2d

806     A: A cross over in the affected parent gives a 1-D gamete --> type 1. You would also expect to get some 2-d gametes from the same parent --> type 4. It\'s just that the sample is so small, that you don\'t get any. (If you expect 1 or 2, getting zero is not a big deviation.) ',0,831,4,1),(###-12-18&&

807     Q: The answer to problem 14-1 says that selection balances out the mutation, but I thought selection can\'t occur in genetic\r\nequilibrium. Is the large population \"compensating\" for the selection

807     A: G.E. was originally defined for cases in which all the assumptions\r\nheld. We now know that it can also hold for cases in which the\r\nvarious factors compensate for each other. So it\'s like chemical\r\nequilibrium -- it\'s a condition where \"concentrations\" of the\r\nvarious genotypes and alleles remain constant overall, even if\r\nthere is actual increase of some genotypes/alleles due to one\r\nfactor and a decrease due to another. In this case it is selection,\r\nnot the large population, that compensates for the mutation.\r\nMutation adds some mutant alleles but selection removes some. You\r\nget G.E. when the two rates balance out.',NULL,1439,4,1),(###-12-18&&

808     Q: In problem 14-7, how can you assume that q = 1

808     A: p is so small that 1 - p = q is close to 1.',NULL,1440,4,1),(##7-12-10&&

809     Q: Problem 10-18 (E) states that we have one recombinant which is clear, but then goes on to say that there must have been one and exactly one recombinant that looks like the parental phenotype. However, the sample size is too small, so how can you assume that there would be one and only one recombinant that took on the parental type? Why not two or zero

809     A: You\'re right the statistical validity of the sample here is lousy. Note that the question says, what is the best estimate? Given the data, you can assume that you didn\'t detect (necessarily) all the recombinants. That\'s really all we want you to notice. I agree that any estimate based on this data is

likely to be way off, and the best way to get a real estimate is to look at more people. (If you can find another family with the same condition.) ',0,833,4,1),(###-12-18&&

810    Q: What do you call the non-empty daughter cell product of a Meiosis 1 nondisjunction?  Is it 4C/2N just like its predecessor? If so, is the other one 0C/0N

810    A: The multiples of C and N we discussed apply to normal haploids and\r\ndiploids. A cell with missing or extra chromosomes will not have an\r\neven multiple of C (or N). These cells are aneuploids, and the\r\nmissing or extra chromosomes have to be included into any\r\ncalculation of chromosome number, chromatid number or DNA content.',NULL,1444,4,1),(###-12-18&&

811    Q: When exactly in the course of the development of the zygote/cell differentiation process does the extra X in all the cells become\r\ninactivated

811    A: Inactivation occurs when the embryo has a few hundred cells. Once\r\ninactivation occurs, it is maintained through mitosis. Cells can\r\ndivide after inactivation, before (or sometimes after)\r\nspecialization. That\'s why heart cells are mixed. Remember an\r\ninactivated X can be replicated and passed on by mitosis.',NULL,1445,4,1),(###-12-18&&

812    Q: Since diploid organisms have two alleles of each gene, does that mean that only one of them gets transcribed and translated

812    A: Generally no. Both alleles are transcribed and translated if the genes are autosomal. (Only one is generally used if the gene is on\r\nthe X.) One copy may be so messed up it can\'t be transcribed, but there is no inactivation of autosomal genes.',NULL,1446,4,1),(##7-12-12&&

813    Q: In problem 11-7, how do you know that complementation is not going on

813    A: Because the DNA that has been transduced into the cell is degraded by nucleases eventually, and hence you would not get ser+ colonies from complementation.',0,836,4,1),(###-12-18&&

814    Q: If you are homozyogous recessive for an\r\nenzyme, does it mean that neither one of the two ds-DNA molecules will get transcribed or translated

814    A: It means you can\'t get a working enzyme out of either copy. Either transcription or translation fails, or the protein is made but\r\ndoesn\'t work.',NULL,1447,4,1),(##7-12-12&&

815    Q: In problem 10-8 (D), why is map distance 6 but not 3

815    A: Map distance depends on % recombinants. arg- strep-S is a recombinant, but so is arg+ strep-R. Since recombination is reciprocal, every cross over event that gives a arg- strep-S also gives an arg+ strep-R. So total number of recombinants should be 2X 3%. ',0,838,4,1),(##7-12-15&&

816    Q: I am a premed struggling in your class. I go to all of the lectures, read all the readings, read all the online notes and exams and do all of the problem sets. I go to every test thinking that I am really well prepared, yet I am getting horrible grades in your class. Is it ridiculous for me to still want to be a biology major

816    A: No. It depends on how you did in other science courses here, whether you are interested in the material, and whether you think there is any likelihood of \"catching on.\" It takes some students a

term or two to adjust. I suggest you come see one of us and let us look at your exams to see where you are having trouble and to see if we can give you some specific advice. ',0,840,4,1),(###-02-06&&

817     Q: For problem 2-1, if the ghost starts with 10 mmol outside, and the inside ends up with about 10 mmole after the long period of time, doesn\'t that mean all the 10 mmol from outside has been moved inside

817     A: The assumption is that the volume of material outside the ghosts is much much larger than the total volume inside all the ghosts.',NULL,1467,1,2),(##7-12-15&&

818     Q: In problem 10-16 (C), how do you arrive at the number 39% as the percent of offspring,  and how was the map distance of 22 units used to arrive at this answer

818     A: In this problem, you are looking for one of the classes of parentals. You crossed an Ab/aB with an ab/ab and are looking for an aB/ab offspring -- or chance of getting an aB gamete from the hetero. parent. Since RF is 22 %, that means all parental gametes = Ab + aB = 100-22 = 78%. 1/2 of those are Ab and 1/2 aB, which means 39% are the ones you want. (I am assuming A is gene for eye color with a --> green and A--> red. If assume A/a are alleles of gene for tail length, not eye color, then you want an Ab gamete, not an aB, but freq. is the same. ',0,842,4,1),(##7-12-16&&

819     Q: What do you mean when you say that a final grade above an A and below a C- are decided on an individual basis

819     A: We decide A+ on the basis of the overall grade distribution for the whole class -- only students who do outstandingly well relative to the class get A+, and there is no set cut off. (Some years we don\'t give any A+\'s at all.) Similarly, we have no set cut off for D and F but look at the overall point distribution and look at individual cases to see if there are any extenuating circumstances. ',0,843,4,1),(##7-12-16&&

820     Q: How are the grading guidelines configured

820     A: We used grade distributions from the past, plus our ideas of what is a reasonable performance. We grade on a curve in the sense that if the scores are too low, we adjust our cut off points. We do not grade on a curve in the sense that we do not have a predetermined limit of what % should get A, B, etc. We have an idea of the minimal percent (who should get A\'s), but not the maximum -- should everyone score in the 90\'s, everyone would get A\'s or A-\'s. If everyone did well, we would not raise the cut off points to ensure that no more than a certain percent got an A. ',0,845,4,1),(##7-12-17&&

821     Q: In problem 11-7, why isn\'t complementation a possibility. Is there some distinction between \"progeny\" and \"colonies?\" How does one approach a problem such as this one in determining complementation versus recombination

821     A: Complementation won\'t (in this case) give you colonies. The added DNA is a fragment, and must recombine with the chromosome to set up permanent (heritable) residence. Complementation could occur in the original recipient, but not in all its descendents. ',0,846,3,1),(##7-12-17&&

822     Q: In problem 14-1 (B), how exactly does the frequency of the recessive allele stay constant if PKU individuals do not reproduce

822     A: There is a low rate of mutation which balances the low rate of selection. (By the way, PKU individuals do now reproduce.) Remember selection is not against the recessive allele directly but against aa individuals.',0,847,4,1),(###-12-18&&

823     Q: If you are homozygous recessive, does this mean that that particular gene and its allele don\'t do anything in the cell except serve as a sort of placeholder and contribute genetically to the gametes

823     A: Yes. In the \"eye color locus,\" for example, you have a version of the information\r\nthat is not decipherable. There is DNA there, but it can\'t be read\r\nout to make enzyme, pigment, etc.',NULL,1448,4,1),(###-12-18&&

824     Q: Problem 8-8 (E) says that the zygote is monosomic and that there is nondisjunction in the gametes. Why doesn\'t this mean that there is only one chromosome of a particular pair, so so 11 chromosomes of 6 types in the zygote

824     A: For this organism, N = 3, not 6. There are 3 pairs in the cell shown, which is diploid. (It isn\'t a haploid with N=6.)\r\nSee part D.',NULL,1449,4,1),(###-12-18&&

825     Q: On problem 9-13 (A), why isn\'t the chance that the man is a carrier 1/2 but rather 2/3? Why is the condition on him having SCD not included in the denominator

825     A: The man cannot be a recessive homozygote. He must be either a heterozygote or a normal homozygote. So the chance is only 2 out of\r\n3, not 2 out of 4. (For a cross of Aa X Aa, there are 4 possible outcomes. But one of those outcomes is impossible. So his chance is 2 out of 3, not 2 out of 4.)',NULL,1450,4,1),(##7-12-18&&

826     Q: Where do plasmids come from? Were they originally viral DNA that circularized? Do eukaryotes ever have them

826     A: Not clear what origin of plasmids is, but do occur in nature. Maybe stripped down viruses that have lost ability to make coats. Not so common in eukaryotes, as far as we know.',0,849,4,1),(###-12-19&&

827     Q: The answer to problem 15-9 says that we can delete the viral origin OR the gene for viral DNA polymerase. However, if we only delete the gene for viral DNA polymerase, isn\'t there a chance that human DNA polymerase could be used to replicate

827     A: The virus codes for it\'s own viral DNA pol, so we assume that means it can\'t use the host one.',NULL,1451,4,1),(##7-12-18&&

828     Q: Are retroviruses present only in mammals? Why? What would happen if a prokaryotic cell got ahold of a retroviral RNA? If it also got the reverse transcriptase, would it lyse

828     A: I don\'t know. RNA viruses are found in bacteria, but I don\'t know about retroviruses.',0,850,4,1),(###-02-06&&

829     Q: In problem 2-4 (B), how can you conclude that facilitated diffusion is involved

829     A: You can tell from graph #2 that a carrier is involved that does not use ATP (for primary active transport). Since there is additionally no ion gradient, you can also rule out secondary active transport.',NULL,1472,1,2),(###-02-06&&

830     Q: In problem 2-3 (A-3), how do we know that Na+ binds on the outside, facilitating a symport transport with the amino acid? Isn\'t it possible that K+ could bind from the inside and facilitate antiport transport

830     A: I don\'t think K+ ever does that. Also a Na+ ionophore messes up transport, and nothing is said about K+ ionophores. Also answers do not give the alternatives of a K+ gradient being \r\nresponsible.',NULL,1468,1,2),(###-02-06&&

831     Q: In problem 2-5 (B), why does Cl- flow out if there is no gradient

831     A: Adding extra bicarb disturbs the equilibrium.',NULL,1469,1,2),(###-02-06&&

832     Q: When nerve cells release neurotransmitters, do they use exocytosis, and if so, how do they compensate for the loss of their\r\nmembran

832     A: They do use exocytosis and reclaim the membrane by endocytosis.',NULL,1470,1,2),(###-02-06&&

833     Q: In problem 1-6, why is the M/N ratio = 4?  Does the monolayer area mean the area including the pores

833     A: If you ignore the pores, you have two bilayers, not too monolayers, surrounding the nucleus.',NULL,1471,1,2),(##7-12-18&&

834     Q: Is the corrected final exam going to be handed back next semester or should I come see you to take a look at it

834     A: We\'ll hand back exams next term and will post answers online when they\'re ready.',0,853,4,1),(##8-01-07&&

835     Q: Do the Postbac\'s exams scores affect the C2005 curve

835     A: The grading scale is based on C2005 exam scores, which is all undergrads. ',0,854,4,1),(###-12-21&&

836     Q: Does selection affect the proportion of genotypes or allele frequency

836     A: Selection affects relative reproduction of the different phenotypes. It acts directly on phenotype, not genotype or allele frequency. However, selection on phenotype ultimately causes changes in the alleles and genotypes (& phenotypes of course).',NULL,1452,4,1),(###-12-21&&

837     Q: In problem 14-14 (C), what does increased randomization of mating have to do with a decrease in the number of homozygous recessives (people with elbow spots)

837     A: If there is nonrandom mating, the recessive alleles are there, but they don\'t necessarily meet each other, so (as in this problem) there can be less homozygous recessives than expected. With

random mating (or NMR), the same alleles are there, but in different combinations.',NULL,1453,4,1),(###-12-21&&

838    Q: Question 4 of sample exam 4, part A-1, says that you would expect that 78 represents and the answer is 2N. Why not it 2N or 2C

838    A: C is an amount of DNA -- in grams or some unit of weight. It isn\'t a number. 2C is the amount of DNA in 2N unduplicated chromosomes. 2C is NOT the same as the number of chromosomes.',NULL,1454,4,1),(##7-11-13&&

839    Q: Regarding problems 12-2 (C) and 12-9 (A). Does a structural gene include the operator and promoter? Are the stop and start codons also considered part of a gene

839    A: The structural gene is the part that codes for a peptide (or its mRNA) or that codes for an RNA like a tRNA or rRNA. The promotor is usually considered a separate part, as it does not code for a product (and is usually not transcribed). In 12-9A what is meant (regarding the promotor) is that the structural gene must have a promotor associated with it. The stop and start codons are definately considered part of the gene -- usually the entire transcribed part is considered part of the structural gene (even the untranslated parts such as leaders and trailers). ',0,856,3,1),(##7-11-13&&

840    Q: If chiasma formation is the only means by which two homologous chromosomes are held together during Metaphase I, is genetic recombination essential to normal gamete formation

840    A: The answer seems to be yes, most of the time. But there are cases where there is no recombination (as in male fruit flies and female silk moths) so there must be other ways of ensuring proper segregation without recombination. ',0,857,3,1),(###-12-01&&

841    Q: What is the point of inserting DNA fragments into plasmids that have promoters if the fragment can be incorporated into chromosomal DNA and use bacterial promoters

841    A: Because you can\'t cut up the bacterial DNA and add stuff to it and put it back. The bacterium is dead.',NULL,1432,3,1),(###-12-01&&

842    Q: In a prokaryote, if a DNA fragment is in the same environment as the chromosomal DNA, why is the fragment degraded and the chromosome not

842    A: The chromosome is a circle with no loose ends.',NULL,1433,3,1),(###-12-01&&

843    Q: Can you use a double-stranded probe?\r\n\r\n&&

843    A: No, as this would prevent hybridization. You can, however, use a probe that contains a mix of two DNA strands by denaturing double stranded DNA.',NULL,1434,3,1),(###-12-01&&

844    Q: Does a DNA fragment inserted into a bacterium have a promoter? If not, does it use the bacterial promoter?\r\n&&

844    A: The fragment usually does not have its own promoter, and it will only use a bacterial promoter if inserted into the chromosomal DNA.',NULL,1431,3,1),(##7-11-14&&

845     Q: For pedigree questions, when it asks whether it is sex linked or not, are we supposed to analyze for the case on the Y chromosome, or do we just stick to X

845     A: Stick to the X (when it asks if something is sex-linked). Sex-linked virtually always means \'on the X\' or X-linked. In this class, it always means that. When people mean \'on the X or Y\', or \'on the Y\' they usually say so -- they use a different term than \'sex linked\'. ',0,861,4,1),(##7-11-17&&

846     Q: The answer to problem 9-10 says in part B that the \"female parent = XY and the male parent = XX.\" How can this be

846     A: In part B we are NOT talking about humans -- that\'s why you can have a heterogametic female and a homogametic male (the reverse of the human situation). Using a chromosome pair to determine sex is very common, but in some species the female has a matching pair like XX and the male has a pair that look different, like X and Y; in other species the situation is reversed. When the situation is the reverse of the human, geneticists often use different symbols than X and Y (they usually use Z and W) so you won\'t get confused. I neglected to do that, and I think that may be the source of the confusion. In the species described in part B, The male is ZZ and the female is WZ, where W and Z are the two sex chromosomes. (See Purves, p. 228.) ',0,863,4,1),(##9-12-07&&

847     Q: What exactly is a \"functional unit? Can there be more than one functional unit in a gene?\r\n&&

847     A: Functional unit means DNA coding for one peptide, one tRNA, etc. We are assuming there is one functional unit per gene (and per peptide), by definition, since mutations in the same gene (affecting the same peptide) do not complement. This is usually true, but there are exceptions that we are ignoring on purpose. There are proteins with more than one functional domain per peptide and mutations affecting separate domains sometimes complement each other. IN this course, we are ignoring that possibility.\r\n',NULL,1435,3,1),(##7-11-18&&

848     Q: For pedigree questions, when it says the disease is rare, are we always supposed to assume that if it is a dominant disease that the genotype will be Aa

848     A: Yes, unless there is evidence in the problem to indicate that the geno (this particular time) is AA. ',0,867,4,1),(##7-11-18&&

849     Q: When discussing family trees and tracing genotypes from disease expressions, why can\'t one show the autosomal example as a sex-linked disease (i.e. put the gene on the X chromosome)

849     A: The woman has a normal son. If it\'s a sex linked recessive, her son would have to have the condition. ',0,868,4,1),(##7-11-19&&

850     Q: When a question refers to repressor proteins, does that include the \"inactive repressor proteins\" involved in induction, or just those involved in repression

850     A: The \"repressor protein\" refers to any protein involved in either induction or repression that sticks to the operator and turns transcription off. Some people use the term \"regulator protein\" to avoid confusion. ',0,869,3,1),(##7-11-19&&

851    Q: On the lecture 15 handout or the euk primary transcript, there is no leader/trailer sequences; is this contradictory to the 1996 c2005 question (with the 140 and 50 base pairs on either end of the exons)? Is this leader/trailer just more junk that never makes it out into the cytoplasm

851    A: The \"leader\" and \"trailer\" shown in the figure in the 1996 C2005 exam refer to those portions of the mRNA that are not translated, since the AUG intiation codon is not located at the physical 5\' end of mRNA, but rather some distance in; and similarly, the translation termination codon is not at the very end of the coded mRNA but rather some distance upstream of the polyA site. Thus almost all mRNAs have untranslated 5\' and 3 \' regions (usually abreviated as 5\' UTR and 3\' UTR). Not all diagrams of mRNAs or of primary transcripts will denote these landmarks, which are relevant only if one is considering issues of translation. Thus Purves does and Becker does not in the figures you mention. The handout on splicing, however, DOES have these landmarks shown in the final mRNA. These 5\' and 3\' UTRs are part of the exons, and must and do indeed go out into the cytoplam as part of the mature mRNA. RNA sequence elements that regulate the translatability and degradation rate of an mRNA often reside in these 5\' and 3\' UTRs. ',0,870,3,1),(##7-11-19&&

852    Q: Can promoters or operators be inverted and still function correctly?\r\n\r\n&&

852    A: A promoter will not work if inverted in an operon, but an operator will.',0,872,3,1),(##7-11-19&&

853    Q: In lecture 19, the example for hemophelia says that 1/2 the cells will make good clotting factor and 1/2 will not. Doesn\'t this mean that different X\'s would have to be activated in different cells

853    A: No, different X\'s are inactivated in different cells. It happens in the early embryo, and it\'s random which X is inactivated in each cell. ',0,873,3,1),(##7-11-19&&

854    Q: What\'s a nonsense mutation

854    A: A nonsense is a premature stop codon. The original sequence coded for some amino acid; the new sequence codes for \'stop\' instead and translation cannot continue beyond it. ',0,876,3,1),(##7-11-19&&

855    Q: What\'s the importance of the \"strength\" of promotors for operons

855    A: The stronger the promotor the more transcription per unit time --> more mRNA from the operon. ',0,877,3,1),(##7-11-19&&

856    Q: Is the second division of meiosis simply mitosis

856    A: No, but the mechanism is virtually the same. (But remember, no DNA replication before the second division) .',0,878,3,1),(##7-11-19&&

857    Q: In problem 8-6, what does \"after 4 hours\" mean? After four hours from the beginnning of the experiment, or from the beginning of the last time phase

857    A: After 4 hrs from the start. ',0,879,3,1),(##7-11-19&&

858    Q: What\'s the importance of having two chromatids per chromosome for nuclear division? Why not just separate/uncoil one strand of DNA (of a single-chromatid chromosome) and then base pair in each daughter cell after division

858    A: This way the DNA replicates first, and then the two double stranded copies separate after. There is never a stage where DNA is single stranded (and more likely to be damaged) for an extensive period. Also, to separate the strands requires that the DNA be relatively loose; to divide the chromosomes/chromatids between daughter cells requires that the DNA be very tight. ',0,880,3,1),(##7-11-19&&

859    Q: Is there a name for one chromatid-chromosome complex, to distinguish from a two chomatid-one chromosome complex

859    A: You said it. It\'s a chromosome with one chromatid, or an unduplicated chromosome.',0,881,3,1),(##7-11-19&&

860    Q: On the handout from lecture 16, does \"start over\" refer to mitosis division in zygote, or reproduction of gametes? Which cells/gene packets are destined to be gametophytes

860    A: Start over refers to fuse (with another haploid cell) --> zygote --> meiosis --> haploids again and so on. Gametes never divide by mitosis -- only reproduce in sense that they fuse to give a zygote which in turn gives more gametes. Gametophytes are stages of plants that will produce gametes by specialization of cells that are already haploid. Meiosis --> haploid cells (spores) --> more by mitosis --> multicellular stage (haploid = gametophyte) --> some cells of this stage specialize to make gametes ',0,883,3,1),(##7-11-19&&

861    Q: What is the difference between a chromosome and a DNA molecule? Are the upstream activating elements of DNA the sites where the activators bind, or the general transcription factors bind

861    A: A chromosome contains a single, double stranded DNA molecule (usually circular in a bacterium, linear in a eukaryote). The chromosome contains structural (and some regulatory) proteins in addition to the DNA. The DNA is the information part, but it is packaged with proteins. The upstream regulator sequences, upstream of the TATA box, are where the activators bind. ',0,884,3,1),(##7-11-19&&

862    Q: In questions 12-4 and 12-8, why can the repressor protein bind to the operator of two operons

862    A: Only because the the operons are either of the same kind or a mutant. That is, a lac repressor protein can only bind to an operator of a lac operon or a mutant lac operon right and not, say, a his operator. ',0,885,3,1),(##7-11-19&&

863    Q: In the case of cat fur color, where the gene is on the X chromosome, why is dominance not involved? Is there no dominance in sex linked traits

863    A: At the cell level, there is no dominance for genes on the X. Only one allele, the one on the active X, is expressed in each cell in a heterozygous female. At the organismic level, there is dominance in some cases. The cat coat color gene acts locally, that is, the protein it makes stays in the cell that

makes it and affects only the color of a small section of skin. Therefore a heterozygous cat has some black skin areas and some orange areas. In this case, there is no dominance. Some genes have an effect on the entire organism, not just the cell they are in. The clotting factor gene produces a protein that is released into the blood and works throughout the entire circulation. The protein doesn\'t stay in the cell that makes it. So a female heterozygous for the clotting factor gene (hemophilia gene) has some clotting factor in her blood (everywhere). In this case, the effects of one normal allele override the effects of the mutant allele -- a heterozygous female doesn\'t have hemophilia. Each individual cell either makes clotting factor or not, but the person overall has enough clotting factor to function. So in this case, there is dominance for normal allele over hemophilia allele.',0,887,4,1),(##7-11-20&&

864     Q: Is more DNA theoretically transcribed in human males than females, since males probably transcribe the 22 autosomes the same, but females only transcribe 1 X and males transcribe both the X and Y

864     A: There are only about 20 genes on the Y. So the difference here is small. Also there are other genes in adults that are transcribed in one sex or other only. ',0,889,3,1),(##7-11-20&&

865     Q: Because of sex chromosomes, are males considered diploid 24N and females diploid 23N, since N is equivalent to the number of different chromosomes

865     A: Both are considered to be diploid, with N = 23. The last pair is just XX or XY. The terminology is 2N or N for diploid and haploid. ',0,890,3,1),(##7-11-20&&

866     Q: What does \"interrupted\" mean when describing eukaryotic genes

866     A: It means they have introns.',0,891,3,1),(##7-11-20&&

867     Q: In problem 12-8, what does \"partially diploid\" mean

867     A: A bacterium that has one complete chromosome + a plasmid, and the plasmid carries a second copy of some of the bacterial genes. The bacterium is basically haploid, but diploid only for the genes on the plasmid. ',0,892,3,1),(##7-11-20&&

868     Q: What\'s a test cross

868     A: A test cross is when you cross a homozygous recessive with a dominant phenotype to see if the genotype is homozygous dominant or heterozygous.',0,893,4,1),(##7-11-24&&

869     Q: For 1997 test #3, question 1 (B), why couldn\'t the answer be 1, 0, 1, where the cell is eukaryotic with alternative splicing? There would then be no operator and only one promoter. &&

869     A: Operons are rare in eukaryotes, so you should think prokaryote first. Alternative splicing to produce totally different enzymes in a pathway is unlikely -- alt. splicing is usually used to produce variants of the same peptide. In other words, although your answer may be possible, it is much more unlikely than the answer given. In terms of both truth and exam taking, it is best to go for the simple answer first (if there is one).\r\n \r\nRe: Web notes for next term: I have received several queries and comments about the web notes and next term. I have not replied to all of the messages here, but I am taking note of all comments, and will look over the web questionaires too.',0,896,3,1),(##7-11-25&&

870    Q: On page 575 of Becker, operon is defined as the cluster of genes in regulating the transcription of mRNA, used in both eukaryotes and prokaryotes. However, Purves  outrights states that eukaryotes do not have operons.  What is the single cluster of genes called that are involved in transcription of eukaryotic mRNA? Is the primary difference, for our purposes, the difference in regulation and processing

870    A: Purves gives the correct definition, and you are correct about the difference between Eukaryotes and Prokaryotes. Only there virtually aren\'t any single clusters of genes. In eukaryotes, genes of related function are generally not clustered; each is in a separate location and is transcribed separately. In other words, most eukaryotic mRNA\'s are monocistronic, not polycistronic. (The exceptions are virtually all in unicellular eukaryotes.) Eukaryotes have \"transcription units\" which usually means a single gene, introns and all. The primary transcript from the transcriptional unit may be processed in more than one way. ',0,897,3,1),(##7-12-01&&

871    Q: About the Venezuelan family with Huntington\'s disease. I understand that a mutation high up on the family tree converted the normal hd allele to the disease-causing HD allele, and therefore if someone inherits a C allele from outside that genetic line then he/she doesn\'t get the disease. However, I saw that person V-22 was the only person on the pedigree with a D allele, and she doesn\'t have Huntington\'s disease even though she does have a C allele. Since A/B/C/D is \"a gene of unknown function,\" does the D allele code for something that combats HD, just as the sickle-cell allele resists malaria? It would follow that people with AC, BC, or CC genotypes would get Huntington\'s disease, but people with the D allele would not. As for person VI-5, who is AC but does not have the disease, I assumed she was the product of crossing over. Is this hypothesis a valid one

871    A: Unfortunately we misled you by saying the A/B/C/D gene is a gene -- it\'s just a sequence of DNA (a spacer) that varies from person to person. (It\'s an RFLP -- to be explained Tues.) So there\'s no reason to think the state of the A/B/C/D \"gene\" has any effect on function, much less on HD. The person who is CD married in to the family, and since most C alleles are on chromosomes with normal HD alleles, the simplest explanation is that the CD person has normal HD alleles on both her chromosomes. (However, to test your hypothesis, you would need to look at a family with lots of people with the D allele in it, and lots with HD too.) VI-5 may or may not be the result of a cross over -- it depends where her C allele comes from, mom or dad (see key). ',0,899,4,1),(##7-12-05&&

872    Q: A boy was recently diagnosed with Gaucher\'s disease, which is recessive. His DNA testing reveals two mutations, where substitution occurred, and he was determined to be \"double heterzygous.\" At first, I assumed it was like being AB/ab, where he had two recessive alleles and two normal alleles, from two different genes. But this did not make sense since the disease is recessive and he has the disease. I also thought about complementation; but, again, he has the disease. The only thing I can think of is that he has two mutations on one gene, where one of the mutations occurs on one allele and the other mutation occurs on the other allele. In a sense, he would be heterzygous per mutation location, thus double heterozygous. He would thus not make the \"good\" enzyme from either allele, giving him the disease.<br><br>\r\n-----x----------- <br>\r\n------------x----<br><br>\r\nIs this correct, or is there something else going on? I\'m told that the mutations occurred on two different genes, but I don\'t see how this is possible. &&

872    A: As far as I know, this is a standard example of failure of complementation -- the kid with Gaucher\'s has two different mutant alleles of the same gene, just as you drew, and so he can\'t make the appropriate enzyme. If the mutations were in different genes he should be okay. However the

standard terminology used for cases like this can be confusing, and that may be the problem. If you can get a reference to the place where the case is described, that would help. By the way, this sort of thing is pretty common -- there are many different defective alleles that cause CF and at least 2 that cause Tay Sachs. Until DNA analysis came along, all nonfunctional alleles where indistinguishable, and all were assumed to be the same. However we now know this is not the case from direct analysis of the DNA (by restriction enzymes, sequencing, etc.) ',0,901,4,1),(###-09-24&&

873    Q: How can lipids be soluble in organic solvents if they\'re extractable in the same solvent

873    A: They are extractable because they are soluble. They are extracted from the generally aqueous environment of the cell by treating the cell(s) with an organic solvent.  The organic solvent will form a non-miscible layer with the aqueous phase (water). The lipid will be found in the organic phase (i.e., benzene).  On the other hand, hydrophilic substances such as polysaccharides would be in the aqueous phase. The lipids will have been extracted, but the polysaccharides not. I guess the trouble was in the definition of \"extraction,\" right?  I would have done well to define that operation in lecture.\r\n',1076525324,903,1,1),(###-10-03&&

874    Q: The answer to problem 4-13C is that bacteria will not grow under the conditions given.  But it seems that the bacteria WILL grow, though only for a while until the NAD runs out.\r\n&&

874    A: If cells will run out of something, it is generally very fast, as stores of small molecules (pools) are usually low. So we don\'t count the little bit of growth that could take place using these stores (there\'s virtually no lower  limit if we reason this way: one molecule?). If they can\'t grow then usually they die, but we don\'t usually think about death, just non-growth, as death raises more complex issues (e.g., how long until they die).',0,904,2,1),(###-10-03&&

875    Q: How does E. coli get ATP in the first place if it runs out and only has glucose to work with

875    A: This is a bit of a chicken and egg question. The same could be asked about NAD, CoA, oxaloacetate, etc. One simple answer is that we are always starting with one cell, one pre-formed, existing cell. This cell contains a bit of everything, so we don\'t run into a situation where the pump needs priming. The E. coli cell synthesizes ADP like everything else: there is a pathway of many steps starting ultimately from glucose if it is in minimal medium. Some of these steps will require energy in the form of ATP. So if every single molecule of AMP, ADP and ATP were gone, we would have a dilemma and death. But that is rarely the case. If E. coli is starving, it cannot make ATP, but then it accumulates lots of ADP as pathways run down.  When food (glucose) is once more available, it can not only make ATP from this store of ADP but it can make many many more new ADP (and thence ATP) molecules from scratch (from glucose)\r\n',0,905,2,1),(###-10-04&&

876    Q: Why does the handout for energy changes associated with reactions in glycolysis not at all match what Purves (6th Ed.) presents in Fig. 7-6

876    A: Purves does not consider the energy change of the coupled reaction (which is the actual reaction) but rather that change in the free energy of the glucose derivatives alone.  We learned that the delta Go for glucose  + ATP  -> G6P is quite favorable (-3.4 or so); that was our reward for coupling glucose phosphorylation to ATP hydrolysis.  So reaction 1 viewed as a coupled reaction (which it IS) is not energy-requiring because the energy is right there in the total  transformations of the components. I chose not to artificially break apart this coupling, so hard-earned through the evolution of energy metabolism. I thought it might be confusing and detract from the important concept of coupling

reactions to make new reactins. The way Purves presents this story has its merits, as it nicely points out the places where energy is required and where energy is given off if one considers the chemical transformations of the glucose-derived components in isolation.  These sinks and sources of energy are hidden in my presentations of the actual coupled reactions. This is most evident in the oxidative step, where the cost of making NADH is not figured into the Purves view. I just said that oxidative reactions are often associated with large free energy changes. Later in discussing the electron transport chain the cost of making 2 NADH\'s per glucose became revealed: 2 x 53 kcal/mole = 106, accounting for most of the y-axis in Purves Fig. 7-6 (6th ed). So it\'s useful to look at both graphs and gain a realization of what\'s going on in each.',0,906,1,1),(###-10-06&&

877     Q: In problem 4-12, why is only half of the radioactive carbon is lost during A-K-G production and half at succinate production

877     A: Because succinate is symmetrical, the population of asymmetric malic acid molecules subsequently produced will have the radioactivity distributed 50% on C1 (near the C with the OH) and 50% on C4 with have have the molecules with radioactivity on the middle COOH and half on the bottom COOH.  The middle COOH is converted to CO2 at the isocitrate dehydrogenase step and the bottom COOOH is removed in the alpha-ketoglutarate dehydrogenase step.',0,907,2,1),(###-10-07&&

878     Q: In problem 4-2 (C,D), why is \"adding energy to the system\" to make an otherwise thermodynamically unfavorable rxn go an insufficient answer

878     A: \"Adding energy to the system\"  is a vague and uninformative answer.  What do you mean by that? How do you \"add energy\"?  That\'s what\'s being asked, how?  We talked of just 2 ways to do this: change the reaction to a coupled reaction in order to get the product you want (here, F6P). Usually, but not always, changing the reaction to one coupled to ATP hydrolysis is used.  This strategy operates on delta Go: the new reaction has a favorable delta Go. But in this case this reaction itself (uncoupled, not changed)  is what is used, and the standard free energy change is somewhat unfavorable (Ke = 1/2.3) .  The other way is to work on the second term of the free energy change equation, the RTlnQ term.  You can do this by altering the concentration of the products and reactants, if you can.  Either increasing the concentration of reactants or decreasing the concentration of products will do the job: both decrease Q, and can turn RTlnQ into a large negative number, large enough to offset and unfavorable delta Go and then some.  It is this second strategy that must be being used by the cells to get this reaction to go, since the first  strategy has not been invoked.  Also, in order to use  to reduce the concentration of products there must be subsequent (downstream) reactions that ARE favorable, so that the products can be swept away.  Thus the overall delta Go for the entire series of reaction of the which the one in question is one part must be favorable , large and negative.  This is the case for this pathway (glycolysis, glucose --> pyruvate -18 kcal/mole). A long answer, I got carried away.',0,908,1,1),(###-10-07&&

879     Q: In problem 4-7, I understand that Keq = .5 (not 1) and that delta G is 165 cal/mol. But how can a Keq of 0.5 be considered close to one

879     A: Yes, it is close to 1 on the scale of Keq\'s . Given that delta Go\'s are usually in the range +6 to +6 kcal/mole, this corresponds to a range of Ke\'s form about  10^-6 to 10^+6. in this context the difference between 0.5 and 1 is small.',0,909,1,1),(###-10-03&&

880     Q: Can you provide a refernce for the experiment in which an actin filament was rotated by the FoF1 ATP synthetase acting in reverse as an ATPase

880    A: Here\'s the reference. below. See also Dr. Junge\'s web site.\r\n<a href = \"http://www.biologie.uni-osnabrueck.de/biophysik/junge/overheads.html\">http://www.biologie.uni-osnabrueck.de/biophysik/junge/overheads.html</a><br><br>\r\n\r\nThe actin filament was 2 um, which is 200 times the diameter of the gamma subunit that was twirling it around \r\n',0,910,2,1),(###-09-14&&

881    Q: Will a cell wholly commit to either lactate or ethanol and CO2, or can you end up with both

881    A: Almost always, a given  organism contains the enzymatic path to lactate (E. coli, elephants) OR ethanol and CO2 (yeast) but not both.',0,916,2,1),(###-09-14&&

882    Q: On the Krebs cycle handout you have asterisks on the labelled carbons coming from acetyl-CoA that disappear all of a sudden at fumararte with no CO2 being produced. Why

882    A: The labeled carbons are still there after succinate, but their position is ambiguous because succuinate is symmetrical.  So we don\'t know whether to draw the labe;ls at the top or the bottom of the pictured molecule.  When the 4-carbon derivatives become assymetic again at malate, the malate omlecules  consist of a mixed popualtion. Half have the two lables on the OH-containg section and half have the labels on the other side.\r\n',0,917,2,1),(###-09-14&&

883    Q: Is it true that if you introduced labelled C in pyruvate it would totally be shed by the time you reach oxyaloacetate?\r\n&&

883    A: No. Like malate, oxalo acetate still hase the pyrvuate derived carbon at this point.  They do not start to be removed as CO2 until the next turn of the cycle, when this labled oxaloacetate condenses with a new acetyl-CoA.',0,918,2,1),(###-09-14&&

884    Q: In problem 4-11C, I don\'t understand why the 0.001 moles of ATP isn\'t the limiting factor here.&&

884    A: You will make NET ATP by running this pathway to the extent of the NAD limitiation. You could start with two molecules out of your stock of ATP (0.001 moles = 6 x 10^20 molecules). After using these to prime a glucose and running glycolysis down to pyruvate, you will have (6*10^20) +2 ATP molecules. Note that you have ADP available for this net ATP production. After another round you will have (6*10^20) + 4 molecules of ATP. So ATP is not limiting, you are not running out  of ATP; on the contrary, you are producing it. Until you run out of NAD in this case. By the way, I think at least 0.01M Pi should have been included here.\r\n',0,919,1,1),(###-09-13&&

885    Q: How exactly does DNA degradation occur? Do we need to eat these nucleotides like we need to eat amino acids

885    A: We all eat DNA and RNA but I think we can make all the nucleotides from smaller molecules if necessary. We normally use a hydrolase to break the 3\' to 5\' phosphodiester bonds in DNA and RNA and release 5\' mononucleotides in the stomach. These go through the blood to cells that need them. The cells do not break the nucleotides down further for energy-- the nucleotides are used by the cells to make their own DNA and RNA.\r\n',1076515785,920,2,1),(###-10-19&&

886     Q: In the structure of reduced NADH in the lecture notes and handout, shouldn\'t both free electrons be on the nitrogen? (one is shown on carbon.) \r\n&&

886     A: The red denoted electrons are not meant to be free electrons, but rather are the electrons that came from the substance being oxidized (the sourceof +2H. in the handout). One electron is used to neutralize the + charge on the ring N of NAD and the other comes in with its proton to constitute the new additonal C-H arrangement on the top ring carbon. You may be thking of the unshared pair of electrons often associated with N, which is not what is being depicted here.\r\n',0,922,1,1),(###-10-19&&

887     Q: When we amplify DNA obtained from a crime scene, don\'t we need to sequence the DNA before we decide on what primer to use

887     A: You don\'t have to sequence the DNA first to decide what primer to use. All human DNA\'s are similar enough so you can use the same primers. You need PCR to find out how long the sequence is between the primers. That\'s what varies.',0,923,2,1),(###-10-20&&

888     Q: When are the Okazaki fragments put together with ligase and exonuclease

888     A: The ligase/exonuclease continually work. following shortly behind the polymerase so Okazaki fragments are connected as soon as possible.',0,924,2,1),(###-10-20&&

889     Q: Is the CO2 we exhale the same CO2 that is released after the production of pyruvate, during Krebs cycle when isocitrate goes to a-ketoglutarate and finally to Succinyl CoA

889     A: Yes. Our pathways are the same.',0,925,2,1),(###-10-20&&

890     Q: How is cellular respiration related to human respiration-- breathing in and out? Is this where the CO2 we exhale COMES FROM? A sideline process to do with metabolism that merely floats off somewhere?  Or does hemoglobin pick up the CO2 and head out to a vein

890     A: Hemoglobin picks up some of the CO2, the rest is converted to bicarbonate and is dissolved in the blood. (See text or wait until next term for details.)\r\n',0,926,2,1),(###-10-20&&

891     Q: Is RNA composed of ATP, minus two phosphates?\r\nThey look identical in the book and are even named identically. But how can something that is charged with the job of energizing life double as a building block for DNA and RNA? Why ATP and not some other molecule

891     A: They are indeed the same molecules--you use what you have to do new things. Did you ever open a can with a screw driver? ATP is the main player in energy metabolism, but some of the others are used too for some processes. Why ATP is prefered is not known. It may be a historical accident. Why do we drive on the right side of the road or use the English system of measurement? Other ways or systems would have worked, but for reasons having nothing to do with the superiority of those methods, we use them.\r\n\r\n',0,927,2,1),(###-10-20&&

892     Q: Are the bands for the semiconservative and dispersive replication at the same level?\r\n\r\n&&

892    A: I assume you mean after one duplication. It depends on how dispersive replication works. Bands could be at the same level but be more broad for dispersive if not every molecule has exactly the same proportions of heavy and light.',0,928,2,1),(###-10-20&&

893    Q: In problem 6-11, why would DNA synthesis stop? ddCTP has a Hydrogen at the 3\' position, all that is needed for the condensation reaction between that H and the OH from the phosphate group on the next nucleotide. According to Becker, the H comes from the sugar and the OH from the phosphate. If this is true, wouldn\'t it make sense that replication would continue with ddCTP

893    A: You need an OH for the condensation reaction. You can\'t lose an H attached to a carbon -- the H isn\'t reactive. The H has to be attached to an OH to react here.\r\n',0,929,2,1),(###-10-20&&

894    Q: In Problem 7-6, how do you determine the percentage of the other bases given only that \r\nthe RNA molecule is 30% A and 10% U

894    A: The RNA must be complementary to one strand of the DNA. So template strand of DNA is 30% T and 10% A. Other strand is 30% A, 10% T. That means overall, DNA is 40% /2 = 20% A = T etc. If you don\'t see it, write out a short DNA strand with 10 bases that is 30% A etc. and it will be clearer.',0,930,2,1),(###-10-20&&

895    Q: How does using T instead of U make DNA more easily repairable

895    A: There are enzymes for DNA repair that scan the DNA for bases that don\'t fit, etc. A section of the damaged strand is degraded and the other strand\r\nis used as template for repair -- in this case a section containing the U is degraded, and replaced with the proper sequence containing C.',0,931,2,1),(###-10-21&&

896    Q: In problem 4-16, why is it that the Keq for the second equation (the one with the enzyme) is used to calculate the delta Go and not the Keq that can be determined from the first three concentrations given

896    A: The concentrations given at the top are the concentrations found in red blood cells, the actual concentrations abserved. There is no reason in the world why these concentrations should represent the EQUILIBRIUM concentrations of this chemical trasnformation. The red blood cell, like most living cells, is in a STEADY STATE, converting one compound into another at some steady rate such that the concentrations are constant. These compounds are not at equilibrium with each other, they are flowing through the system. See Powerpoint slide 25 and 26 in lec. 7 for anillustration of the difference between steady state and equilibrium. Both states result in a constant amount of a given compound, but for very different reasons.',0,932,2,1),(###-10-21&&

897    Q: Is the equilibrium constant Keq = 1 or can it be several different values for the reaction R <--> P

897    A: If Keq=1 it means the the ratio of P to R AT EQUILIBRIUM is 1. Then it can\'t be 0.01 or any other number for those particular P\'s and R\'s.  For the reaction R <--> P there is one and only one Keq, and it could be 1 or 0.01 or 500, but it is something and it is a constant value you can look up in a book. The Keq reflects the tendency of  R to turn into P, which in turn depends on the relative stabilities of  R and P, or their electronic arrangements. A reaction will BE at equilibrium if the concentrations of the reactants and products are such that their ratio  equals the equilibrium constant.

If they are not at such values, then the reaction is NOT at equilibrium and will tend to go in the direction that gets it there, gets it to the point where the ratio does equal the equilibrium constant.',0,933,1,1),(###-10-21&&

898    Q: In problem 4-14C, can we assume that the presence or lack of ADP is not a factor in determining what part of a cycle will still be functioning in the absence of other materials like production of ATP, recyling of NAD, NADH

898    A: If one is considiring a living cell (as in problem 4-14c), then all the usual pathways are present, including one to synthesize ADP from glucose, so ADP is present and the only problem is having a way (a source of carbon and energy) to generate ATP from it.<br><br>\r\n\r\nIf, on the other hand, one is considering a test tube, where one is testing what is necessary for an experiment or for a made-up problem, then you do definitely have to worry about the provenance of each component. If you do not add any ADP (or ATP) to a reaction mixture, then no ATP can ever be present. For instance, if you add ATP but have no way to hydrolyze it to ADP + Pi, then if you need ADP for some other reaction you will not have it available.\r\n',0,934,2,1),(###-10-21&&

899    Q: Does the fact that our muscles resort to anaerobic respiration during strenuous exercies mean that the more poorly we breathe while running, the more weight we\'ll lose/prevent (since fermentation will use a lot more glucose to produce comparable ATP)

899    A: I think the weight issue is not real, because we catch up when the acute excercise is over -- we take the lactate and either break it down further (and make more ATP) or use it to resynthesize glucose to save for later. So we may temporarily use more glucose per min, and break it down less, but we make the same amount of ATP, more or less, in the end, per glucose. Now if we could eliminate the lactate directly, without breaking it down more, and always ate the same amount of glucose, then we would lose weight.\r\n',0,935,2,1),(###-10-21&&

900    Q: In problem 5-17, why are the coupled reactions involving the conversion of glucose to G6P via ATP hydrolysis not uncoupled by the uncoupling proteins

900    A: The use of the English word coupling is the only thing these two circumstances have in common. For a strategy to allow the formation of G6P from G, an unfavorable reaction, the cell has evolved a mechanism of \"coupling\" that energetically unfavorable reaction to a second biochemical trasnformation, ATP to ADP, which is a very favorable reaction. \"Coupling\" is perhaps not the best use to decribe this process, because what the cell does is carry ourt a brand new reaction, whose reactants aand products are the same as if the two separate reaction had been somehow coupled. But it is a new reaction G + ATP -> G6P + ADP. This strategy is called coupling an unfavorable transformation to a favorable transformation to come up with what is in the end a favorable reaction also.\r\n<br><br>\r\nThe word coupling can also be used to describe other disparate process that are tied together physiologically. Thus pyruvate reduction is coupled to NADH oxidation ot regenerate NAD. And ATP synthesis is coupled to the use of a proton gradient across the mitochondrial membrane in oxidative phosphorylation. And also in oxidative phosphorylation the transport of electron down the electron transport chain is coupled to the formation of the proton gradient mentioned above. There are proteins that uncouple this last process, sometimes called \"uncouplers\" without further specification, and that is unfortunate. But it is usually clear from the context as to just what they are uncoupling. In this case (problem 5-17) electrons travel down the chain, but no proton gradient is formed because the protons are let back across the mitochondrial membrane by a route other than the ATP synthetase (F1Fo).\r\n',0,936,2,1),(###-10-22&&

901     Q: I read that strong covalent bonds that hold stacked nucleotide rings together. If instead it is weak bonds that hold them together, then why don\'t they break apart when the DNA is denatured by heating

901     A: You have the covalent bonds connecting the parts of the chain confused with weak bonds that stabilize 3D structure.',0,937,2,1),(###-10-22&&

902     Q: In problem 4-14C: How can you get to pyruvate from 1,3PGA if you\'re not generating ADP through this part of the glycolysis cycle?  Is ADP is being released by other pathways

902     A: You are worried about the need to put in ADP to get the reaction from 1,3 PGA to pyruvate to run. Because after all, I kept harping on these loose ends. If you borrow it, you have to pay it back, etc. A legitimate concern.  But where was this concern when we discussed glycolysis in general? Why only for problem 4-14C? We invested 2 ATPs and got out 4 ATPs, for a net gain of 2 ATP. Which means a net loss of 2 ADP, where are they going to come from? You answered your own question, they are being generated elsewhere. Think back to the simple diagram that introduced all this discussion of energy metabolism: If we only had a way to generate ATP from the ADP that was the product of these coupled reactions, we would be all right. And that ATP is the energy \"currency\" of the cell , constantly being spent, and generating ADP in  the process.',0,938,2,1),(###-10-22&&

903     Q: Should we be able to recognize the structures of adenine, thymine, guanine, or cytosine? Or any other molecular structures, for that matter

903     A: No. But you should understand the significance of the various parts of the structures.',0,939,2,1),(###-10-23&&

904     Q: When RNA replicates, what does the very first nucleotide look like? Is there a single phosporus on the 5 prime, or is it a triphosphate? Is this ever a question in DNA?\r\n\r\n&&

904     A: RNA doesn\'t replicate, except in rare cases with viruses. You mean has RNA just been made by transcription? Then there would be a triple P on the 5\' end. In the case of DNA: because DNA polymerase always always needs to combine 3\' to 5\', there are no \"initial\" loose 5\' ends. ',0,940,2,1),(###-10-23&&

905     Q: Is the same DNA polymerase which has exo 3\' 5\' capability during editing the same one that removes primer 5\' to 3\' and adds 3\' to 5\'? (i.e. 3 functions)?\r\n&&

905     A: Probably not. Probably one DNA polymerase does the first round, including editing, and a second DNA polymerase removes primer and fills the gap.',0,941,2,1),(###-10-23&&

906     Q: Problem 7-7 suggests that you can separate radioactive DNA from nonradioactive DNA (denature, hydrolyze, etc.) and then measure their relative proportions. But problem 7-R2 (part D iii) says it is very difficult to separate radioactive and nonradioactive molecules. Is it possible or no

906     A: No you can\'t separate radioactive and nonradioactive. You have to use other methods to separate double stranded from single stranded or RNA that sticks to DNA from RNA that doesn\'t. I think the answer to 7-7 refers to measuring amounts of radio left after some proceedure, not to separating radioactive from nonradioactive.',0,942,2,1),(###-11-10&&

907     Q: In the colony hybridization technique, in which different plaques are formed, how do we know that each plaque is formed by bacteria of same DNA

907     A: You mean colonies, not plaques. If the solution that you spread on the Petri dish is dilute, only a few bacteria land on the dish, and each bacterium divides to form one colony = a clone.',0,943,3,1),(###-11-12&&

908     Q: Why does rate of transcription of histidine repressor gene stay same in problem 12-2 (1)

908     A: The repressor gene is always transcribed -- production of the repressor protein is constitutive. It\'s the shape the repressor is in -- with or without effector -- that determines what happens. Not the presence or absence of repressor protein.',0,944,3,1),(###-11-12&&

909     Q: When we refer to a hybrid cell infected by two different viruses as in problem 11-4, is it always the case that the coat of one and nucleic acid of other hybridize

909     A: It\'s viruses. Always coat of one and NA of one. NA\'s don\'t stick to each other (unless denatured) and proteins here don\'t stick to each other. But the coat and NA pair up and match. To make an analogy, suppose you have two people and two coats. You can put either coat on either person (assuming the sizes are right) but not a coat on a coat or a person on a person.',0,945,3,1),(###-11-09&&

910     Q: In problem 12-2D, why is it that synthesis of enzymes will also decrease (though more slowly) when feedback inhibition occurs in addition to repression

910     A: It means synthesis of enzymes will decrease more slowly than synth of his. Not more slowly than before.',0,947,3,1),(###-11-09&&

911     Q: If production of his is being inhibited immediately, wouldn\'t there be less repression of the his operon in the long run? It seems like the fine control should have some impact on the coarse control here. Or should we think of the processes as completely independent

911     A: There will be enough his, even if you stop making more, to kick in repression. Repression, and fbi, will end once the his falls below a critical level. Fbi won\'t stop repression from happening, but it may cut\r\noff more his production sooner, so that the critical level is reached sooner.\r\n<br><br>\r\nThere should be enough his added to inhibit the enzymes and to act as co-repressor and stop synthesis of additional enzymes. The two processes do act independently, as long as there is enough his to bind to both enzymes and repressors. FBI has an immediate effect on his synth, not on\r\nhis levels.',0,948,3,1),(###-11-10&&

912     Q: Regarding problem 12R-4 (B). Does the inactivation of the repressors occur when the effectors (corepressors = pro) are detached from the repressors? If so, then will the start of the mRNA transcription occur when all the pro are removed

912     A: Yes, the inactivation occurs when effectors are detected from the repressors (meaning that all the pro are removed). Non-bound pros are removed before the bound ones. It works like this: There is an equilibrium between bound repressor (bound\r\nto operator) and free repressor (off the operator). There is also an equilibrium between free repressor complexed with pro and empty repressor. (See below) When co-repressor (pro) is used up, the equilibrium shifts to the right (see

below) and repressor is pulled off the operator.\r\n<br><br>\r\nbound rep (+pro) <---> loose rep (+pro) <----> separate rep and pro\r\n',0,949,3,1),(###-11-12&&

913    Q: I was wondering what the 5S portion of the ribosome referred to (in the answer to 7-14 A of the problem set)

913    A: 5S is the name of a type of rRNA. Not of a subunit or RNP. 16S and 23 S also refer to types of rRNA, not subunits of the ribosome. See handout 13A or table 20-1 in Becker.',0,950,2,1),(###-11-12&&

914    Q: With problem 13-4C, won\'t BugII cut at different places so that we don\'t know whether it inadvertently cuts our drug resistance gene

914    A: You are only cutting up the DNA of the gene here with Eat III and BugII. You are not cutting up the plasmid -- You already cut up the plasmid with Eat III. All you care about is that the two enzymes you use give the same sticky ends.',0,951,3,1),(###-11-13&&

915    Q: Can recombination and integration can occur for lytic bacteriophages as well as viruses

915    A: As I intended it, 11-11 doesn\'t involve integration of the phage. The phage accidently packs some loose bacterial DNA into its coat instead of its own\r\ngenome -- it doesn\'t acquire the extra DNA by recombination with the phage chromosome during integration or excision.',0,952,3,1),(###-11-04&&

916    Q: In problem 11-2 C, why isn\'t the answer \"assay for enzyme made by gene L\" also viable in addition to \"growth on medium containing lactose as only carbon source?\"&&

916    A: To assay the enzyme in a bacterium, you have to grow up a separate culture from each bacterium you want to test. Then for each separate culture, you\r\nbreak the cells up, prepare an extract of the cells, add substrate, and measure formation of product. This will work for bacteria with the normal (but not mutant) plasmids. However it is a lot easier to spread the bacteria on an agar plate and see if they form colonies or not.',0,953,3,1),(###-11-04&&

917    Q: On page 600 Becker makes it sound as if a copy of the F factor is replicated during the transfer process from F+ to F- during conjugation and that a copy is left in the original donor cell, thus allowing it to retain its F+ status. Is this true

917    A: I think he means that given semi-conservative replication, there is no such thing as a copy and a original. Both daughter molecules are 1/2 and\r\n1/2. The donor keeps one and transfers one. He is trying to emphasize that \"donation\" during conjugation doesn\'t mean giving away your one and only copy.',0,954,3,1),(###-11-04&&

918    Q: Is it correct that cells are either inducible, repressible and constitutive, but not a combination

918    A: Those terms refer to genes, enzyme synthesis from these genes, or to operons, not to cells. Production of a particular enzyme (or set of enzymes) is inducible etc. One may also refer to a particular gene or operon as inducible, repressible, etc. However a bacterial cell has some enzymes and operons that are inducible, some that are repressible, and some that are

constitutive.\r\n<br><br>\r\nThere are operons that are both inducible and repressible -- one small molecule acts as a co-repressor and turns off the operon and a different\r\nsmall molecule acts as an inducer and turns on the operon. But I think these are few, and we have ignored them.',0,955,3,1),(###-11-04&&

919    Q: Is the only difference between induction and repression them is a difference in repressor protein

919    A: Each operon has its own repressor protein that binds to its respective effector (co-repressor or inducer) and its respective operator. If the\r\nrepressor protein is inactivated by the effector (so it falls off the operator), the operon is inducible.\r\nIf the repressor protein is activated (so it binds to the operator) by the effector the operon is repressible.',0,956,3,1),(###-11-14&&

920    Q: If only one tRNA for met fits in the P site and one in the A site, what happens when met is inserted in the middle of  the peptide chain and that tRNA is moved to the P site

920    A: Every tRNA fits in the P site when it has a peptide chain attached to it. Only the special initiator met tRNA fits into the P site WITHOUT an attached chain. I suspect that was not clear from the way I said it.',0,958,2,1),(###-11-14&&

921    Q: With regard to handout 17A: Along step 4, you have included tetracycline in the medium, meaning that all those cells that did not get plasmids died in this step. So what is the relevance of trp+

921    A: You want to be sure everything that got an insert grows, even if the insert doesn\'t convert the cell to trp+. So you include trp in the medium.',0,959,3,1),(###-11-14&&

922    Q: With regard to handout 17A: In Step 5, you make a replica that shows those colonies that don\'t have inserts. Isn\'t this step redundant

922    A: Yes & No. It allows you to discount all colonies with a plasmid but no insert. You are right that the two methods (colony hybridization and replica plating to medium with amp) overlap. If you did all the colony\r\nhybrids you wouldn\'t need to do the replica plating. But often you do the replica plating first and throw out the colonies with no inserts before doing additional screening.',0,960,3,1),(###-11-14&&

923    Q: In problem 13-7A, are the sticky ends too short because of the weight of the DNA pieces

923    A: The weight doesn\'t matter here, although I know that thinking of this in terms of models might lead to that conclusion. The issue is that complementary chains do not form stable double stranded section unless there are a minimum of H bonds. (It doesn\'t matter how long the rest of the chain is -- it still takes about the same number of H bonds to hold two single strands together.) The number of H bonds formed by the sticky ends is quite small.',0,962,3,1),(###-11-04&&

924    Q: I thought cutting and splicing a chromosome would kill the cell. How do bacterial cells survive the cutting and splicing that occurs when plasmid DNA is integrated into their chromosomes

924    A: If you have 2 circles, one cut and splice just makes a bigger circle. So that\'s ok. One big circle, one origin of replication. But if you have a linear molecule and a circle, one cut and splice makes

a broken circle. The \"circle\" now has an origin, but it\'s ends are not connected to each other, so replication will not occur properly with correct joining up of all loose ends.',0,963,3,1),(###-11-04&&

925    Q: What is a \"cutting and splicing\" event? Why are there two Xs on the recombination handout

925    A: Each \"X\" or \"cutting and splicing\" event means you cut two molecules (cut each once) and then join the loose ends crosswise. It involves two molecules, two cuts, and two joins, but it is considered one event. If you cut only one molecule you can\'t recombine anything. It always takes two to tango. You need two tapes to splice them together.',0,964,3,1),(###-11-16&&

926    Q: Suppose a lysogenic virus invades a prokaryotic cell. If repressor protein stops the RNA polymerase from transcribing the DNA, is it true that the repressor protein also stops the DNA polymerase from replicating the DNA

926    A: It may prevent synthesis of the specific DNA polymerase that uses the origin of the viral DNA. So unless the virus DNA has joined the chromosomal DNA, the virus won\'t be replicated. If the virus integrates, the viral DNA is replicated every\r\ntime the chromosome is replicated, using the ordinary bacterial enzymes of replication.',0,965,3,1),(###-11-16&&

927    Q: What mechanism is used to remove the DNA bound repressors before replication? Would this mechanism also remove the repressor protein that keeps a virus dormant during the lysogenic cycle

927    A: Repressor doesn\'t need to be removed during replication of an integrated, lysogenic virus. Or from any repressed operon.\r\nNote that there is an equilibrium between bound and free repressor. It isn\'t all on or all off. If the free repressor is removed or destroyed, that shifts the\r\nequilibrium and more repressor falls off, etc.',0,966,3,1),(###-11-16&&

928    Q: If repressor protein is not removed during replication, are the repressor proteins re-made for every replication of lysogenic bacteria? Does this mean that the repressor protein in a daughter cell gets degraded later than that of the parent cell.&&

928    A: No. But as bacteria grow, more repressor protein is made so both daughter and parent are repressed.',0,967,3,1),(###-11-16&&

929    Q: In problem 11-1, how do we know that experiment 2 involved conjugation, rather than no transfer of genetic material happening at all

929    A: The problem asks which processes could be going on here to transfer plasmids. Experiment two, if it works at all, is conjugation. Is there a possibility nothing will get transferred? Of course. But the question asks how you would set it up so as to detect transfer if it occurred.',0,968,3,1),(###-11-16&&

930    Q: Regarding question 13-9 (B-2). Why don\'t introns cleaved with DNase show up as separate fragments in gel electrophoresis

930    A: DNase degrades the SS regions down to nucleotides. It doesn\'t just cut off the loops -- it chews them to bits. So there are no fragments\r\ngenerated to show up on the gels. DNase is not like a

restriction enzyme that cuts once at a particular spot -- it cuts many times at random.',0,969,3,1),(###-11-16&&

931     Q: If a protein\'s function is closely related to its structure, how can anitbodies be used to detect proteins based on structure if the protein doesn\'t function correctly

931     A: There are at least two reasons. One is that very subtle changes in structure (changes of a few AA) can ruin enzyme activity and have only small effects on overall structure. The second reason is that an\r\nantibody doesn\'t react with a whole protein.  Each antibody reacts to one small part of the protein -- one small region with a particular structure.\r\n(Called an epitope or antigenic determinant.) When the protein is altered, usually most of the areas or epitopes remain the same, and only a few fold\r\ndifferently. One alteration is enough to make the protein lose function, but not enough to lose all aspects of structure. So you can mess up enzyme\r\nactivity and still have most of the antibody still match most of the parts of the protein.',0,970,3,1),(###-11-16&&

932     Q: In problem 12R-2 C, wouldn\'t the levels of enzymes A and B be \"can\'t predict,\" since the maximum rate of production depends on the promotor, and the promotor could\r\nbind RNA synthethase either well or poorly

932     A: High and low here refer to induced/nonrepressed levels vs uninduced/repressed levels. That sidesteps the issue of the promotor\'s strength.',0,971,3,1),(###-11-16&&

933     Q: In problem 12R-5F, I don\'t see why the answers involving viruses are \"out\" and restriction enzyme methods are \"in.\"&&

933     A: A lysogenic virus can act like an F and integrate, forming something like an Hfr. When the virus loops out, it can carry off bacterial genes near the place where it integrates. Just as with an F.  But no virus is mentioned here -- you aren\'t making a virus carrying extra genes but a plasmid carrying extra genes. So anything involving viruses is \"out.\"',0,972,3,1),(###-11-16&&

934     Q: What is the difference between an allele and a mutation

934     A: Alleles are alternative versions of genes. A mutation is a change in the DNA. A mutation can convert one allele into another. A mutant has an abnormal allele of a gene, an allele with a mutation that changes the function of the gene. Which particular allele(s) is \"normal\" or \"wild type\"  and which is (are) \"mutant\" depends on the situation.',0,974,3,1),(###-11-20&&

935     Q: Aren\'t ribosomes needed for both eukaryotic and prokaryotic RNA transcription?\r\n&&

935     A: Ribsosomes are for translation, not transcription.\r\n(The two processes are simultaneous in prok. but separate in euk.)',0,975,3,1),(###-12-17&&

936     Q: During crossing over, can one particular chromatid from one homolog have a crossover event with either of the chromatids from the other homolog or just one

936     A: A chromatid can cross over with either chromatid of its homolog.',0,1007,4,1),(###-12-17&&

937     Q: In problem 15-2 (E), why is the rate of NADH2/NAD not correct

937   A: NAD is constantly recycled, so in a normal cell the ratio of reduced and oxidized forms stays the same. Red. NAD would be made faster if metabolism goes faster, but then the oxidation of NAD will go faster too. Otherwise oxidized NAD will run low and start to limit energy metabolism.',0,1008,4,1),(###-12-17&&

938   Q: In problem 13R-4 (B), does an insertion or deletion always cause a frameshift mutation? Does this matter if we\'re only considering restriction enzyme fragments

938   A: An insertion or deletion MAY cause a frameshift or may not, depending on where it is. But that doesn\'t matter either way here, as you say, because we are only looking at cutting by restriction enzymes, not function of the protein.',0,1009,4,1),(###-12-16&&

939   Q: The solution to problem 9-7 (A-4) says that A = active enzyme that turns white to red, and a = inactive enzyme (so Aa = pink). Can you also say that A=red, a=white, and Aa=pink due to incomplete dominance

939   A: There are cases like this where the heterozygote phenotype is pink and other cases where the heterozygote is red. I think there are more \"red\" than \"pink\" cases, meaning that the heterozygote is usually similar to the dom. homozygote. But partially it depends on how you look at it. If you see it in terms of color vs no color, or able to make pigment vs unable, it\'s different than if you look at it as red vs white vs pink.',0,1010,4,1),(###-12-15&&

940   Q: In problem 10-18, the allele 2 of alk. is linked to D (in the relevant family). Does that mean that for individuals outside this family, the D should be linked to allele 1, or is it that the D is linked to either (1 or 2) if and only if a mutation occurred at some point? \r\n\r\n\r\n&&

940   A: The D can be linked to either, depending on which allele was there when the mutation occurred.<br><br>\r\nThe gene or locus for alk. phosphatase has to be linked to the gene or locus that causes WS\r\nin everybody. However the \"WS gene\" only causes WS when the gene is mutant-- the normal allele of the gene codes for a protein that is involved in hearing, pigmentation, etc. The gene should be called the \"hearing/pig. gene\" but it is called \"the WS gene\" because it was first noted when a\r\nmutant form of it caused WS.  Everybody has the alk. phos. gene and the \"WS\" gene. But which allele of each locus is on each chromosome varies from person to person and family to family. A person from a different\r\nfamily is likely to have only normal alleles of the \"WS\" gene.<br><br>\r\nI think you have confused linkage -- where the two loci are relative to each other, with what geneticists call \"phase\" -- which alleles are on one homolog and which alleles are on the other homolog.',0,1011,4,1),(###-12-09&&

941   Q: In problem 10-4, what part does the excess phenylalanine play in the pathway? (It could play several possible roles--acting as an inhibitor on enzyme 1 and thus shutting down the entire pathway and the production of tyrosine, or perhaps working as an inducer on the production of enzyme 2 and thus the production of tyrosine.)&&

941   A: All you have to know is that phe can be made into tyr. So if you have enough phe, you don\'t need to eat tyr. But it doesn\'t work the other way around. The pathway is phe --> tyr. (It\'s presumably irreversible because of energetics.) You don\'t need to consider regulation to answer this question.',0,1012,4,1),(###-12-04&&

942    Q: Have scientists actually done experiments inserting haplotype DNA segments into bacteria to verify that haplotypes indeed have no effect on phenotype

942    A: That\'s not the right test. But are these differences really 100% neutral? It\'s hard to know. But it is clear that some differences really make very little difference to growth, survival, reproduction, etc. Proving the\r\ndifference is zero is really impossible. It is true that some differences thought to be neutral turn out not to be so afterall. But some differences may be truely neutral.\r\n\r\n',0,1014,4,1),(###-12-04&&

943    Q: I don\'t understand why evolution, in all its efficiency and elegance, would select for random strings of nonsense. What are the\r\ntheories for what these guys are doing in the cell? Do they really serve no purpose

943    A: There are many theories, some of which claim the \"nonsense\" has a function and some of which claim it is parasitic and of no function at all (to the host). Who knows? I don\'t think every feature of every organism is adaptive, i.e. useful. Some just got there by chance.',0,1015,4,1),(###-12-03&&

944    Q: Regarding standard written practice with\r\nsex-linked genes. Is it wrong to label the normal alleles as well as the affected ones? It makes it easier for me to see how a woman must be either a carrier or affected to have an affected kid. It also gets a little wacky without the normal alleles when you\'re thinking about recombination.&&

944    A: You are absolutely right that it is clearer if you write out both alleles for each gene. However it is difficult to show where the\r\nalleles all go if you write the chromosomes as X\'s. That\'s why I wrote the chromosomes as they are shown in the answer to problem 10-9 under the picture. There the chromosomes are drawn as straight lines, not X\'s.',0,1016,4,1),(###-12-02&&

945    Q: What exactly do the boxes represent in a Punnett square

945    A: The boxes inside the square represent the types of zygotes and their proportions. (In the case on 20A there are 4 types in equal\r\nproportions.) The zygotes are not the products of meiosis -- the gametes or spores are the productions of meiosis. The zygotes are the combinations that you expect when you mix the gametes of one parent with the gametes of the other. The gametes of one parent are written on the top of the square; the gametes of the other parent are written\r\non the left side of the square.',0,1017,4,1),(###-11-26&&

946    Q: Out of curiosity, how exactly does the body get rid of a virus? And has there been any experimentation with using a virus to\r\nfight a bacterial infection in the body, or is that silly, since it will just make the body more vulnerable

946    A: Stay tuned -- next term we\'ll talk about the immune system. Or check it out in the texts. People have thought of using viruses to kill bacteria, but viruses don\'t prey on other viruses. You can use some viruses as decoys to mess up reproduction of others or cause production of antibody to the \"real viruses\" -- that\'s what some vaccines do.\r\n',0,1018,4,1),(###-11-24&&

947    Q: How does increasing the dosage of antibiotics \"beat\" the resistance of certain bacteria if they are resistant

947     A: Bacteria are not absolutely resistant or not resistant at all. They vary in their resistance. If you use a lot of antibiotic, for a long time, you kill them all. If you use less antibiotic and/or don\'t add it for long enough, you allow the more resistant bacteria to outgrow the others but do not kill the resistant bacteria. This makes matters worse, not better, as you have\r\neffectively selected for the partially resistant bacteria and given them a leg up.',0,1019,4,1),(###-11-24&&

948     Q: Is the fever a defense mechanism in killing off bacteria and/or viruses

948     A: Fevers are often treated because they feel awful and very, very high fevers can kill us as well as the bacteria. No one is sure how important the fever is in helping us, we have other medical treatments, and fevers make us miserable. So the usual thing is to treat. But we may eventually find it better not to treat regular fevers and only treat very high ones.',0,1020,4,1),(###-02-19&&

949     Q: Why is it that when you add DNAses to the nucleosome and linking DNA you get not just 200bp, but multiples of 200bp. Why don\'t you just get equal amounts (i.e. all 200 bp) if it cuts every 200 bp

949     A: If you add enough enzyme, you get all 200 size pieces. If you add a little enzyme, you don\'t cut at every possible site.',NULL,1021,1,2),(###-02-19&&

950     Q: Are there two carrier proteins involved in Na/glucose co-transport

950     A: The same protein facilitates movement in either direction. Either the protein flips back and forth (from T1 to T2) spontaneously, or binding glucose on either side (in either conformat on) flips it to the other side and the other conformation. The protein acts like a revolving door -- it helps glucose move either way.',NULL,1022,1,2),(###-02-19&&

951     Q: Why aren\'t peroxisomal localization sequences removed

951     A: I don\'t know. It may be that the enzymes are trapped inside the peroxisome in some way (by forming assemblies?) so that they can\'t get out, and there is no need for a peptidase. It looks like peroxisomal proteins can fold up and enter their organelle without unfolding, somewhat the same way proteins enter nuclei. It may be that once you fold a protein, you don\'t or can\'t cut anything off.',NULL,1024,1,2),(###-02-19&&

952     Q: If the signal peptide is downstream from the start of a growing peptide, does that mean that the protein has to be a transmembrane protein with its amino end in the cytoplasm

952     A: The way we explained it, if all transport into the ER is co-translational, yes. However you can insert the protein the other way if you don\'t push the growing end in, but push the part that\'s already made into the lumen. We are ignoring this possibility, but advanced texts deal with it (there is some post translational import into the ER which we are ignoring).',NULL,1025,1,2),(###-02-19&&

953     Q: Are the ribosomes on the rough ER only attatched to the ER by the proteins that they are co- or post-translationally importing? If so, are all ribosomes the same except for their location

953     A: Yes, all ribosomes are the same. The sequences in the newly growing protein determine the fate of the ribosome.',NULL,1026,1,2),(###-02-10&&

954    Q: Why can\'t lysosomal storage diseases besides Gaucher\'s (or I disease) be treated by providing the missing enzymes? Is it a matter of substrate not being able to reach the target cells? In the case of Tay-Sach\'s, is the blood-brain barrier the problem

954    A: I assume the target cells don\'t have the scavenger receptors on their surfaces or the added enzyme can\'t get to the cells at all, or you can\'t add enough enzyme to make a difference. In other words, I don\'t really know for each case, but assume it is one of these. (In I cell disease, so many enzymes are missing that they can\'t all be replaced. Even if it were theoretically possible, it\'s prohibitively expensive.)',NULL,1027,1,2),(###-02-10&&

955    Q: When using the scavenger pathway to treat Gaucher\'s Disease, how does addition of hydrolase help if only a small fraction of the M6P receptors are \"misdirected?\"&&

955    A: A little hydrolase goes a long way. Since it\'s an enzyme, you only need a little.',NULL,1028,1,2),(###-02-19&&

956    Q: In post-translational import into the mitochondria/chloroplasts, the localization sequence is called a transit peptide. Is the peptidase in this case called a transit peptidase, or is it still a signal peptidase

956    A: Becker calls it transit peptidase; I assume that is standard terminology.',NULL,1029,1,2),(###-02-13&&

957    Q: When we talk about glycophorin being a glycoprotein but also a transmembrane protein, is it a part of the ECM or not

957    A: A protein counts as being in the ECM if it is entirely outside the cell. If it has a domain outside the cell, it is not considered part of the ECM.',NULL,1031,1,2),(###-02-13&&

958    Q: How is glycophorin on the RBC involved in aging the cell

958    A: Sugars on the protein are gradually lost with time. So the state of the glycosylation of the protein matches the age of the cell. Degradation may be triggered by loss of sugars (beyond a critical point).',NULL,1032,1,2),(###-02-13&&

959    Q: In problem 1-2, is it correct to say that lysed means to cut a cell to remove contents and then resealed, whereas disrupted means cut multiple times to allow small vesicles to reseal

959    A: Yes. Or lysed means cracked open and resealed back the way it was; disrupted means fragmented into little pieces that reseal randomly (right side out or inside out) into minivesicles.',NULL,1033,1,2),(###-02-13&&

960    Q: During RME, is the clathrin somehow responsible for the migration of receptors, or does the receptor just know to migrate without a signal from the clathrin

960    A: My sources say it differs for different receptors. Some migrate spontaneously to the coated pit; others migrate only if they have bound their ligand. (Until they have bound ligand, they are anchored in place to the cytoskeleton and can\'t diffuse in the lipid bilayer.) There is no signal from clathrin, but once the receptors \"slide into the pit\" meaning arrive at the area where the clathrin is,

there is no escape. The receptors probably move around randomly in the membrane (if unanchored) until they hit an area with clathrin.',NULL,1034,1,2),(###-02-09&&

961     Q: Is it possible for the answer of problem 2-8 (C) to be \"is recycled back to plasma membrane OR be degraded?\" Both seem to be possibilities depending on the receptor-ligand complex (recycled as for LDL receptor or the entire receptor-ligand complex degraded as for EGF)&&

961     A: It says in the problem that the uptake protein (the receptor) is never found in lysosomes. That\'s how you know it isn\'t degraded.',NULL,1037,1,2),(###-02-09&&

962     Q: In graphs of rate vs. time for ion channels, will a plateau ever indicate equilibrium

962     A: You are mixing up two kinds of curves. Reaching equilibrium because concentration on both sides is equal (or has reached a stable level because rate in = rate out) occurs when you plot conc. inside vs time. That kind of plateau is reached for every kind of transport. Reaching saturation of rate v. conc. of X is another matter. (That\'s the kind of saturation I was talking about.) That kind of plateau occurs when you plot the rate of uptake of X vs. the conc. of X. A plateau means that adding more X doesn\'t make X move any faster. It does NOT mean that concentration of X on the two sides of the membrane is constant with time. For ion channels, the rate of X going in (as a function of concentration of X) does plateau at enormous conc. of X.',NULL,1038,1,2),(###-02-09&&

963     Q: In the answer to problem 2-4, secondary active transport is ruled out because there is no ion gradient, not because of the lack of energy metabolism (hydrolyis of ATP). What would fund the energy to maintain an ion gradient in secondary active transport if it\'s not ATP

963     A: As far as I know, secondary active transport always uses ATP but it is ALWAYS indirect. The ATP is needed to set up a gradient, but once the gradient is made, no more ATP is needed. I think you are confusing the short and long range situations. You can use up all the ATP to create an ion gradient. The gradient can then persist fora while and drive secondary active transport in the absence of any ATP. In the long run, you can\'t make or maintain the gradient without ATP, but in the short run, once the gradient is set up, you can run active transport without any ATP around at all. (The \"short run\" here can be quite a while, but the gradient will run down eventually.)',NULL,1039,1,2),(###-02-05&&

964     Q: How is hypercholesterolemia inherited

964     A: It\'s a dominant disease -- if you have one mutant allele, you have half the usual amount of working LDL receptor (from the normal allele), and low levels of uptake of LDL. So cholesterol accumulates in your circulation and causes problems. This condition is fairly common, and predisposes to early heart attacks (in 30\'s or later). It can be treated by altering the diet and taking cholesterol lowering drugs. If you have two mutant alleles, you don\'t have any receptor, and this leads to very high levels of circulating cholesterol and very early heart attacks. This state is pretty rare, but the consequences are very serious.',NULL,1041,1,2),(###-02-04&&

965     Q: What\'s the difference between transferrin and ferritin

965     A: The Fe binding protein I was talking about that enters the cell by RME is transferrin. Transferrin is the iron binding protein that carries Fe in the blood. Ferritin is a different (intracellular) Fe binding protein that stores extra intracellular iron. For some unknown reason I slipped up and switched them.',NULL,1042,1,2),(###-02-03&&

966     Q: Why is there alow concentration of Na+ but high K+ in the cell? What\'s the difference between the contributions of Na+ and K+ to the cell or to our bodies? We get Na+ mostly from the table salt, but where do we usually get K+ from

966     A: There are several kinds of \"why\" answers -- how it happens, what good it does, and how this particular system evolved. How it happens (in this case) is a function of the protein pumps in the cell membrane -- they pump Na+ out and K+ in at the expense of ATP. But why do cells operate that way? I can\'t answer that one. It may have to do with the relative sizes and properties of the two ions, so that it makes more sense to do it this way than the other way around (or to use some other combination of ions.) But I don\'t know really know -- I\'ll have to see if I can find a good argument one way or the other.  There are two possible standard evolutionary explanations for the current state of affairs (whatever it happens to be). One is that the current system works well, and better than other possible system. So it was \"selected for\" that is, it won the survival race -- the organisms that did it other ways didn\'t reproduce as well. The other evolutionary explanation is that the current system works ok, but no better than several other possibilities. It is just an accident that one of our ancestors (who was \"selected for\" because of other important talents) just happened to use this system of ion pumps, and so all the descendants of this common ancestor do so too. Other systems would work, but we just happen to be descended from an ancestor that did it this way. Just as the USA uses the English system of measurement because our ancestors did. It works, but it certainly isn\'t better than other systems. We just started using it and it\'s too hard to change. K+ also comes in food, like Na+. Most people get enough, but some people who don\'t eat enough variety don\'t get enough. I don\'t know off-hand what good sources of K+ are, other than bananas. \r\n<p>Dr. G adds: Bananas are a very good source! Exercise, and certain drugs deplete the body of K+ and people are told to eat lots of bananas. But it\'s found in other fruits and vegetables as well, and, of course, in smaller quantities, in any living thing (meat or vegetable) that has a Na/K pump.',NULL,1043,1,2),(###-02-03&&

967     Q: What does it mean that a \"small amount of DNA\" is found in the mitochondria

967     A: By \"amount\" we are talking about the total # of base pairs in the DNA, or total length of DNA  -- not the amount in grams or the number of copies. The longer the DNA, the more different proteins it can encode, and the more information it contains. The total length of DNA, and # of genes, in the nucleus is much much larger than the amount in the mitochondria. There are no chromosomes in mitochondria -- the DNA there is additional to the chromosomal DNA. Also the DNA in mitochondria is not in \"chromosomal\" form -- it is circular and naked, like bacterial DNA, not linear and associated with histones (proteins) like chromosomal DNA.',NULL,1044,1,2),(###-02-03&&

968     Q: How does recent evidence for the existence of mitochondrial networks respond to endosymbiont theory? The book points out that part of the supporting data for mitochondria as bacterial in origin is their size and shape. How are these seemingly contradictory theories being rationalized

968     A: If they are endosymbiants, they have been so for hundreds of millions of years. Major changes have occurred during that time, so I don\'t think this is any big deal. The size argument inspired the original theory, but there are many more reasons to suspect endosymbiosis besides the size of the organelles.',NULL,1045,1,2),(###-02-03&&

969     Q: Would it be possible for you to provide all of the problem sets from the book via the web? This would save us the money in purchasing it from the Village Copier.&&

969     A: I\'ve posted it on the web before, but it is a royal pain. Not just for me, but also for the students. The amount of work it takes to print it all out is not worth the cost saving, and then you are left with umpteen loose pages to deal with.',NULL,1046,1,2),(###-02-03&&

970     Q: If a rabbit protein is injected into a mouse, and the mouse produces antibody #1 to it, what would happen if the antibody were injected back into the rabbit

970     A: Rabbit will produce antibody (#2) to antibody #1, which is a foreign protein (antigen) as far as the rabbit is concerned. It takes a while to make antibodies. In the meantime, some of antibody #1 may find its antigenic target in the rabbit and stick to that protein.',NULL,1048,1,2),(###-02-03&&

971     Q: How does a vesicle membrane fuse with the plasma membrane? Does it have P and E sides

971     A: All membranes contain bilayers. When a vesicle fuses with the plasma membrane, the two bilayers of both membranes fuse -- the two P sides join and the two E sides of the two membranes join. Draw it out and see.',NULL,1049,1,2),(###-02-02&&

972     Q: In regard to vesicle/membrane fusion, you say that the two P layers and the two E layers always end up joining each other. Does this mean that the E layer in vesicles is the inward facing one because the endomembrane system is functionally continuous with the exterior of the cell

972     A: Yes, you have it right. The lumenal spaces and the space outside the cell are functionally continuous.',NULL,1050,1,2),(###-02-02&&

973     Q: What good is Cl- in the body besides its excjange for bicarbonate in RBCs

973     A: Cl- has many uses (and it is exchanged really for HCO3-, not CO2 directly). You have to have negatively charged ions to balance the positively charged ones, and in some cases, to attract positive ones. For one example of the uses of Cl- ions, see box 8B in Becker.',NULL,1051,1,2),(###-02-02&&

974     Q: If a protein is embedded in the membrane but only on the P side, where was it produced

974     A: Proteins that are on the P side are generally peripheral, not embedded. (Most embedded proteins go all the way through.) The peripheral proteins on the cytoplasmic side are made on free ribosomes, not the ones attached to the ER.',NULL,1052,1,2),(###-04-01&&

975     Q: Which hormones use the IP3/DAG signal transduction pathway

975     A: Some examples include TRH, angiotensin, vasopressin (ADH), and oxytocin.  However, we are still learning the details about which hormones use which pathway, and it seems that a single hormone can use multiple pathways, so it\'s not useful to memorize these at this point.  If there is any sort of generalization, it might be that IP3/DAG is used by hormones that lead to smooth muscle contraction, such as oxytocin, which stimulates uterine muscles to contract, and vasopressin, which stimulates vasoconstriction. ',NULL,1053,2,2),(###-04-01&&

976     Q: Could the answer to problem 4-9 (B) also be \"initiation of rate of translation?\"&&

976     A: The question is not \"How could it happen?\" but \"What is the most likely explanation?\" Then answer given is the most likely (if not the only) explanation.',NULL,1055,2,2),(###-04-01&&

977    Q: Couldn\'t answer to problem 4-11 (A) also be \"modification of protein post translation\" since this could inhibit the ability of type A to polymerize

977    A: Note that the question says that diff. types of fibronectin vary in amino acid sequence. Since this is sufficient to account for the differences in dimerization, there is no need to invoke an additional factor such as modification. If different fibronectins have the SAME amino acid sequence and dimerize differently, then protein modification would be a sensible explanation.',NULL,1056,2,2),(###-04-01&&

978    Q: Why is this problem 4-11 (B) a case of alternative splicing and not a gene family

978    A: The problem says that there is only ONE gene for fibronectin. So the DNA must always be the same. You are not dealing here with several genes that are part of the same gene family and that have diverged to give a family of proteins. You are dealing instead with only one gene that gives a family of proteins by alternative splicing of the same transcript.',NULL,1057,2,2),(###-04-01&&

979    Q: Why isn\'t the thyroglobulin chain hydrophobic? Is it made of anything other than tyrosine

979    A: Thyroglobulin is hydrophilic like other glycoproteins. It\'s a long protein, a chain of over 5000 amino acids, of which only 140 or so are tyrosine.',NULL,1058,2,2),(###-04-01&&

980    Q: Regarding alternative splicing: Is there any reason for one poly-A site to be utilized over another

980    A: In terms of cell and organismic function, yes. If one poly-A site is used, the cell has a membrane bound antibody which can detect the presence of  antigen; if the other polyA site is used, the cell produces large amounts of soluble (secreted) antibody which can be used to inactivate the antigen. The presence of antigen is what switches the system from the first state to the second. This response to antigen is \"useful\" to the organism overall -- it allows a multicellular organism to produce the \"right\" antibody to destroy a specific infectious agent, but only when that agent comes along. The organism doesn\'t intentionally or consciously switch gears -- this is a mechanistic response which has evolved over millions of years. The organisms that responded in this way survived infection, flourished and left many descendants who could mount the same immune response; those that didn\'t respond in this way (by making antibody in response to antigen) didn\'t reproduce as well and died out.',NULL,1059,2,2),(###-03-30&&

981    Q: Why are people in India developing goiter when they use the cheap salt made by evaporating seawater. Shouldn\'t this contain iodine

981    A: Seawater does contain iodine, but the concentration is somewhat low, and people don\'t eat a huge volume of salt. When iodine becomes incorporated into groundwater, seafood, and plants, you can get a lot in your diet.  But when salt is produced by the evaporation of seawater, what crystallizes is mostly sodium chloride, with some magnesium, calcium and potassium.  Whatever iodine was in the seawater is lost in the purification process, so even salt made from seawater needs to have iodine added.',NULL,1062,2,2),(###-05-07&&

982    Q: Do the peripheral chemoreceptors sense $CO_2$ levels or H+ levels in addition to $O_2$ levels

982    A: Summarized in Table 13-9, p. 472 Sherwood:  H+ is major stimulus for chemoreceptors. $CO_2$ can weakly stimulate them, and they respond to $O_2$ only when it\'s extremely low.',NULL,1063,3,2),(###-05-07&&

983    Q: Is CSF also considered ECF? Would the equation then become ECF = CSF + Plasma + interstitial fluid

983    A: You could consider the extracellular fluid to consist of plasma, interstitial fluid, lymph, cerebrospinal fluid, and even the aqueous humor inside the eye.  However, plasma and ISF make up about 99% of the ECF, so the others are usually  omitted from this sort of discussion.',NULL,1064,3,2),(###-05-07&&

984    Q: I am going over respiration and I realize that I might be a wierdo! How come my tummy goes in when I breathe

984    A: Because you\'re in better shape than me. I sit here slumped back in my chair, and each time I inhale, I see my belly move outward. When we stand upright and inhale in a relaxed, quiet way, we use mostly the intercostal muscles, so there is less movement in the abdominal area.  If you\'re especially trim, when you inhale and raise the chest cavity with the intercostals, this might pull up on the abdominal area and make it seem to go in. <p>If you lie on your back, you use the diaphragm more than the intercostals, and you should see the outward movement of the abdomen when you inhale. Note:  This explanation does not mean that you are not a weirdo, simply that we haven\'t been given sufficient evidence to decide about that.',NULL,1065,3,2),(##9-01-29&&

985    Q: Are lysosomes and perioxosomes also vesicles, or are they just called organelles

985    A: Lysosomes and Perox. are both types of vesicles and organelles.',NULL,1066,1,2),(###-02-20&&

986    Q: If we can treat Gaucher\'s disease by taking advantage of a scavenger pathway, why is there no way to do something similar for Tay Sach\'s Disease

986    A: I think the problem with Tay Sach\'s is that it affects the brain, and the enzymes are not taken up into the brain. They are taken into the liver and spleen, which are the primary problem \r\nin Gaucher\'s. In other words, the difference is in the tissue that is primarily affected, not in the ability to produce the enzymes.',NULL,1486,2,2),(###-02-18&&

987    Q: What is facultative heterochromatin

987    A: The DNA in constitutive heterochromatin is never transcribed. That in facultative is transcribed, when the DNA is not heterochromatic. By definition, facultative heterochromatin is a section \r\nthat can be non-heterochromatic under some conditions.',NULL,1487,2,2),(##9-01-26&&

988    Q: In your description of the evolution of protein families, you said that the gene code is duplicated before it mutates. Why is this an important/essential step? Couldn\'t a family evolve just by mutation

988     A: If the one and only copy of the gene mutated, then the cell would contain only one member of a protein family at a time. The protein and gene could change over time, but you couldn\'t get multiple different versions all at once.',NULL,1068,1,2),(##9-01-28&&

989     Q: Can you either recommend reading or summarize what you said in class regarding how junction function in epithelial cells because I\'m not sure I wrote down everything that was important. &&

989     A: I think the table in the outline summarizes the critical points. If you want more info, see Becker ch. 10 (pp 271-278)',NULL,1069,1,2),(##9-01-28&&

990     Q: I don\'t understand how the antibodies for any specific protein (G-actin, etc.) are made in the first place. Do you just inject antigen into another species

990     A: The basic ans is yes. You inject the antigen, wait a week or so, inject it again (to get a better response) and then bleed the animal. The serum will contain a mixture of antibodies, including the ones you want.',NULL,1070,1,2),(###-02-18&&

991     Q: Is the signal peptide always a hydrophobic localization sequence

991     A: Yes, as far as I know.',NULL,1489,2,2),(###-02-18&&

992     Q: Does the signal peptide bind the pore protein or does it move out of\r\nthe pore protein into the lipid bilayer?  Where is it anchored when\r\nthe protein is being pumped into the ER

992     A: SP probably does both -- binds to pore to start and then slides out. As protein is being pumped in, it could be on side of pore or in membrane.',NULL,1490,2,2),(##9-01-28&&

993     Q: If membranes have the hydrophilic sides out and the hydrophobic sides in, why are lipids absorbed and water soluble materials not? Does the hydrophilic head actually absorb, but then the hydrophobic rejects it and \"pushes\" it back out? And does the hydrophobic \"pull\" for the lipid overpower the hydrophilic \"push\" and thus still aborb the lipid put on top of the hydrophilic layer

993     A: I think the thin hydrophyllic layer on the outside of the membrane is so thin that water sol. molecules don\'t seem to go in at all. Or a few go in and stick to the hydrophylic heads, but they soon saturate the layer as no molecules continue in, across the layer. Lipid sol. molecules continue to be absorbed as they don\'t just build up on the outside, but cross the bilayer.',NULL,1071,1,2),(###-02-18&&

994     Q: Does the signal peptide contain a special sequence within it that allows signal peptidase to cut it off

994     A: There is a separate sequence, after the SP, that contains the site (or not) for signal peptidase.',NULL,1488,2,2),(##9-01-28&&

995     Q: I am curious about the difference in explanation of the structure of MT in Becker and Purves 5th edition. Becker is as you described it in class, but 5th ed. Purves says it is a cylinder of \"9 + 2,\" specifically 9 sets of 2 filaments forming the cylinder and 2 separate filaments running down the middle. &&

995     A: I think Becker is talking about microtubules in general (13) and Purves is talking about the kind found in cilia and flagella (9+2). I suspect both texts discuss both structures, but in different contexts.',NULL,1072,1,2),(##9-01-28&&

996     Q: I read about desmosomes in animal cells and plasmodesmata in plant cells, and one of the protein\'s IF is made of is desmin. What is the desmin root referring to? Is there a connection (linguistically or physiologically) between the desmin and desmsomes/plasmodesmata

996     A: I really don\'t know. Desmin got its name because it\'s associated with desmosomes. But why desmosomes? I don\'t know enough Greek or Latin. (If any reader knows the answer, please e-mail Dr. M). It could mean layer, or attachment, or skin etc. I don\'t know if the origin of plasmodesmata is related although it sounds like it should be. If I find this one out, I\'ll let you know.',NULL,1073,1,2),(##9-02-01&&

997     Q: Are collagen and elastin glycoproteins? Also, are proteoglycans actually a member of the glycoprotein family, or are they entirely seperate

997     A: Virtually all (all?) extracellular proteins and extracellular domains have carbohydrate attached to them. So yes, coll. and elastin are glycoproteins. Proteoglycans are not glycoproteins -- they are a different thing entirely, as I hope the table in the lect. #2 outline makes clear. Proteins are considered part of a family when their amino acid sequences and 3D shapes are similar. This is not true for these two classes of thing. Glycoproteins are very different from proteoglycans, and glycoproteins themselves have a vast array of functions; not even all glycoproteins are part of the same family. All Proteoglycans have certain structural features in common, and they might be considered to make up a family.',NULL,1074,1,2),(##9-02-04&&

998     Q: Does continuous labeling reveal things that pulse labeling does not? Does the maximum value for grains/organelle on the continuous graph have any particular significance

998     A: Continuous is much easier to do, which is why it is done. The max. value could have meaning if you had enough information about what a grain represents -- how much radioactivity per molecule, etc. Usually the actual value has no obvious significance.',NULL,1075,1,2),(##9-02-04&&

999     Q: Is there a connection between down regulation and things like drug or allergy desensitization

999     A: Down regulation of receptors can lead to decreased sensitivity of response. Whether this phenomenon is primarily responsible in the cases you mention, I don\'t know. I will see if I can get some more info on this.',NULL,1076,1,2),(##9-02-08&&

1000    Q: In the handout on glucose/Na+ co-transport, how does the sodium fall off at the part where the enzyme faces in

1000    A: The Na+/K+ pump keeps the Na+ concentration inside the cell very low. Therefore, inside the cell, the following goes to the left:<p>Na+ + cotransporter < ----> complex <p>(Outside the cell, [Na+] is high and reaction goes to right). The continual action of the Na+/K+ pump prevents Na+ from accumulating and keeps the reaction shown favorable to the left inside the cell. ',NULL,1078,1,2),(##9-02-15&&

1001    Q: In problem 3-14 (C), Hexo, though it goes straight into the cytoplasm and doesn\'t need a localization sequence for that, still needs to get inside a mitochondion, so wouldn\'t it require at least one localization peptide? And EX, though it could get out of the cell via the default pathway, don\'t proteins targeted for secretion have signal peptides to get them secreted faster

1001    A: Glycolysis takes place in the cytoplasm, not the mitochondrion. About the EX: It all depends what you consider \"secreted\" and what you consider \"dumped.\" If the EX is released only in response to some specific signal (what I meant by secreted), than it would need a sequence to get it into appropriate vesicles. If it is released constitutively (\"dumped\"), the additional signal should not be necessary. The term \"secreted\" is sometimes used to mean \"pushed outside\" and sometimes used to mean \"released in response to a signal.\" I intended secreted to be limited to the second meaning. (If you have any texts that say ECM material has special targeting sequences, I would love to know about it.) ',NULL,1079,1,2),(##9-02-15&&

1002    Q: In problem 1-12, if the contents of the vesicle are topologically equivalent to the outside of the cell, then the glcoprtoeins should be inside the vesicle when the vesicle is normal. Why is it that in the question the carbs are where they are supposed to be only when the vesicle is inside out.&&

1002    A: The vesicles you are looking at in this problem are made from fragments of plasma membrane. So \"right side out\" means has same stuff on the outside as the plasma membrane does. You may be thinking of vesicles from inside the cell, say those transporting the protein to the plasma membrane, and these would be different, in terms of what is \"right side out.\" \"Right side out\" intracellular vesicles have proteins meant for the extracellular surface on their inside. They are topologically the opposite of vesicles made from the plasma membrane itself.',NULL,1080,1,2),(##9-02-17&&

1003    Q: Is M6P added to a protein in the Golgi or ER

1003    A: This is a point not clearly settled by research. Most results point to addition in the Golgi, but there are some that can be taken to indicate addition in the ER.',NULL,1082,1,2),(##9-02-17&&

1004    Q: Can you please expain how <i>in situ</i> Labelling works

1004    A: You provide substrate to fixed or living cells. The substrate diffuses in and meets an enzyme that can convert S --> insoluble product. The product forms where the enzyme is. You locate the product by color in light microscope or density in EM. That tells you where the enzyme is.',NULL,1083,1,2),(##9-02-17&&

1005    Q: In co-translational import, do proteins that get inserted into the ER go there after modification in the Golgi, and if so, how

1005    A: Some proteins are probably retained in the ER -- they don\'t move on to the Golgi. Others are thought to go to the Golgi for modification, and then vesicles bud off carrying the proteins back to the ER to do their job.',NULL,1084,1,2),(##9-02-17&&

1006    Q: Are glycoproteins and proteoglycans only found in the extracellular domain of the membrane

1006    A: As far as I know, the answer is a qualified yes. The carbos have to be added in the Golgi and ER so they are only added to proteins that end up outside the cell or in the topologically equivalent lumen of the endomembrane system (lysosomes, etc.).',NULL,1085,1,2),(##9-02-17&&

1007    Q: What is the difference between an endosome and a coated vesicle

1007    A: The use of the term endosome varies from person to person. It is sometimes used as a synonym for endocytic vesicle (at any stage) and sometimes only for specific stages, usually the ones after uncoating or even later. It is not clear if endosomes should be considered separate vesicles or part of a larger network of tubules (like the ER). So if you are unclear, so is everyone else. Since the mechanism of uncoating and sorting is not entirely clear, we will have to wait for more experiments to clear this up. In this class \"endosome\" is usually used to mean the vesicles or cell compartment in which sorting of ligand and receptor occurs. If you read various texts, you have to be alert to context to be sure how they\'re using the term.',NULL,1086,1,2),(##9-02-17&&

1008    Q: The answer to problem 2-6 says that the most likely protein to be defective is the receptor protein for substance E. Why is clathrin not a possibility for the defective protein

1008    A: If clathrin were defective uptake of all substances using RME would be affected. Problem says this is not the case.',NULL,1087,1,2),(##9-02-18&&

1009    Q: I\'m confused about when glucose enters the cell via facilitated diffusion, and when via active transport (Na+/Glucose pump). Is there a difference

1009    A: Fac. diffusion uses one type of transport protein, and allows glucose to flow across a membrane down its gradient, never up. Active transport uses a dif. type of transport protein and pushes glucose up its gradient while using the energy provided by Na+ flowing down ITS gradient. The handout shows when each type is used.',NULL,1088,1,2),(##9-02-24&&

1010    Q: If water is small enough to pass through lipid bilayers by simple diffusion, how are the membranes fused in tight junctions to make it water tight? Can\'t water get through the nonpolar areas just as easily

1010    A: There are proteins in the bilayer that form the tight junctions. It is sort of as if you have a line of connexons with no holes sticking out of one bilayer, and a similar line sticking out the other bilayer. The two sets of proteins fuse, making a water impenetrable layer.',NULL,1090,1,2),(##9-02-24&&

1011    Q: What exactly is the difference between apoptosis and necrosis

1011    A: Necrosis and apoptosis both are terms for cell death. Necrosis means that the dead cells release their contents and trigger an inflammatory response. (see below or text for description). This can cause damage to neighboring cells. Apoptosis means that the dead cells destroy themselves from within (it is an active process that requires transcription and production of appropriate break-down enzymes) and apoptotic cells do not release compounds that trigger an inflammatory response. The inflammatory response = redness, heat and swelling due to presence of white blood cells (that engulf invaders and damaged tissue) and some blood cell proteins and is triggered by release of chemicals (mostly histamines) from damaged tissue. It is necessary for certain kinds of wound healing and destruction of invading micro-organisms. If it gets out of hand, it can be very unpleasant and/or destructive. In some cases, the response is worse than the insult that triggered it (as in some allergies).

Antihistamines are used to combat the excesses of the inflam. response. Apoptosis is important when unnecessary or redundant or rogue cells need to be eliminated without triggering destruction of the neighboring cells, as for example, during development when you eliminate the cells inbetween your digits (so people don\'t have webbed feet and hands).',NULL,1091,2,2),(##9-03-03&&

1012   Q: When the RP binds to mRNA of ferritin, why and how is the iron released from the ferritin? \r\n\r\n&&

1012   A: Release is not direct or immediate. What is meant by the arrows on the handout is that synthesis of ferritin will decrease, so in the long (ish) run the storage will decrease and Fe will be released. You are quite right that no immediate release will occur -- but future storage will decline.',NULL,1093,2,2),(##9-03-04&&

1013   Q: Although cascades offer an efficient and quick response to environmental stress, isn\'t it true that they also increase the potential that something can go drastically wrong (i.e. faulty signals causing a large problem)

1013   A: Virtually all cascades have a shut off mechanism or are self limiting as in blood clotting -- once the clot forms, and fills the space, that\'s the end of it. In other cases, as in the immune system, there are secondary cascades triggered by the primary cascade that conteract the earlier effects! Which I assume is a mechanism that has evolved to keep things in hand. However you are quite right that there is a potential problem. Continual exposure to stress can cause overstimulation of responses that are fine in emergencies but no good in the long run. The usual argument is that there has been continual selection of mechanisms that fine tune the system and improve regulation so signaling does not get out of control. However Modern society has moved too quickly for a proper evolutionary response to continual stress to evolve. So we need a societal fix, not a biological fix.',NULL,1094,2,2),(###-03-07&&

1014   Q: Is aspirin an antagonist to prostaglandins, or whatever it\'s supposed to block? And are ibuprofen, tylenol, and all those newish medications agonists of aspirin

1014   A: Aspirin isn\'t considered an antagonist to prostaglandins because it blocks their synthesis, not their action. The newer pain killers could be considered more or less agonists of aspirin--some inhibit a different enzyme than aspirin does, but another one in the same pathway.',NULL,1495,2,2),(###-03-07&&

1015   Q: What is the different between normal and small G proteins (e.g. ras) in terms of their activation and ability to exchange GTP and GDP

1015   A: I don\'t think I was clear enough.\r\nNormal G proteins need to be activated, but once they are, they can catalyze GTP/GDP exchange. The G protein itself is the catalytic part. For small G proteins, another protein does the catalytic part (& also activates). Both types of G proteins can hydrolyze GTP to GDP.\r\nAdditional proteins (in both cases) can speed up or slow down (\"modulate\") either GTP binding or hydrolysis.',NULL,1496,2,2),(##9-03-11&&

1016   Q: If muscle cells do not divide, like neurons, after a certain point in development, then how does a bodybuilder get big muscles

1016    A: Muscle cells can\'t divide by mitosis, but you can increase the amount of protein and mass per cell (by excercise). In some cases, over large multinucleate muscle cells can divide into two, but this is thought to have only a small effect on muscle mass.',NULL,1097,2,2),(##9-03-11&&

1017    Q: Can you explain the difference between the DNA binding and trascription activating site of a transcription factor

1017    A: All TF\'s have two domains -- one for binding DNA and one for affecting (inhibiting or activating) transcription. The second domain mentioned is called the trans activating or transcription activating (or inhibiting) domain. TF\'s that respond to ligands (steroids or similar compounds) have a third domain that combines with the ligand. The DNA binding allows the TF to stick to the DNA at an enhancer, silencer, etc. Once the TF is stuck to the DNA, its trans acting domain can combine with other TF\'s and influence transcription. If there is no DNA binding domain, the TF can\'t get close enough to the DNA to do anything. If there is no trans acting domain, binding to DNA has no effect. If this is still not clear, ask again. ',NULL,1098,2,2),(##9-03-22&&

1018    Q: In the glucose cycle, which is considered the effector(s) in maintaining homeostasis: the pancreas which monitors (the sensor) but then puts out either insulin or glucogon in response, or the liver, muscle, adipose cells, which finally \"effect\" the final desired effects

1018    A: Liver etc. are usually considered the effectors. But you can think of this as a two cycle system -- in cycle one, pancreas is the effector to give insulin etc. and this then signals the second set of effectors that actually change the glucose levels. The second way of thinking may be helpful in understanding what is going on, but is not usually the standard terminology.',NULL,1100,2,2),(##9-03-22&&

1019    Q: Is phospholipase C always embedded in the membrane? In Becker, it clearly indicates that can also be found in the cytosol (and thus be activated by Tyrosine Kinase). But in Problem 6-17 you stated that its only found in the membrane. &&

1019    A: Note that in my picture PLC, unlike AC, is not a transmembrane protein. I think it is a peripheral membrane protein than when activated binds to the inside of the plasma membrane, where it\'s substrate is. There is more than one PLC, and some may be permanently membrane bound.',NULL,1101,2,2),(##9-03-22&&

1020    Q: What exactly is the role of Ca++ once it is released into the cell

1020    A: Ca++ has many effects, many of them through calmodulin. Activated calmodulin binds to an activates/inhibits many other proteins. For more details, see Becker.',NULL,1102,2,2),(##9-03-22&&

1021    Q: Does only IP3 open Ca++ channels (considering there are channels on the cell membrane also)? Are the Ca++ channels that lie on the cell membrane all voltage gated channels, or can they be opened by a secondary messenger

1021    A: Many things open Ca++ channels. The details depend on cell type.',NULL,1103,2,2),(##9-03-22&&

1022    Q: How does more DNase lead to bands of 145 bp

1022    A: More DNase digests all the relatively exposed \"linker sections\" that go inbetween the nucleosome octomers. That leaves only the DNA that is tightly wound on the octomers, which is two turns worth or 145 bp.',NULL,1104,2,2),(##9-03-22&&

1023    Q: Does a hypersensitive region mean where basal or regulatory transcription factors bind

1023    A: Hypersensitive could have either type of TF\'s instead of nucleosomes.',NULL,1105,2,2),(##9-03-22&&

1024    Q: What is the difference between DNAases

1024    A: They have different specificities or talents -- tend to digest or grab DNA at different spots. Which one \"works\" for each type of experiment was largely decided by trial and error, not any theory.',NULL,1106,2,2),(##9-03-22&&

1025    Q: When is G1 cyclin made

1025    A: You mean the start cyclin for G1--> S? Probably in G1 or during the G0 to G1 transition. ',NULL,1107,2,2),(##9-03-22&&

1026    Q: What are the different types of diabetes and their relations to the production of insulin and regulation

1026    A: The short answer is type I = no insulin; type II = no (or reduced response to) insulin. Type I is treated with insulin injections; type II with diet and excercise (to reduce insulin secretion and upregulate receptors). Type I = juvenile; type II = adult onset. ',NULL,1108,2,2),(##9-03-23&&

1027    Q: Lectures 7 & 8 talk about an experiment using DNAse and a restriction enzyme to splice a gene. Do the presence of histones affect the work done by the restriction enzymes as it does with DNAse

1027    A: In all these experiments you isolate chromatin, mess around with it, and then isolate the DNA (by removing all protein). Then all further steps involve purified DNA. I think that point was not clear.',NULL,1109,2,2),(##9-03-23&&

1028    Q: In Exam #2 \'98 problem 4-C, we get different results on the blot due to the different states of the chromatin in the different cell types. Why doesn\'t this also affect the blot in part B of that same problem? Is the difference simply because in C we \"do a limited DNase treatment of chromatin\" and therefore don\'t get into the tight spots

1028    A: In part B you are cutting up DNA, not treating chromatin with DNase and THEN cutting up DNA.',NULL,1110,2,2),(##9-03-23&&

1029    Q: What\'s the difference between a Northern blot and a Southern blot

1029    A: Southern == separate DNA pieces, blot etc. Northern = separate RNA\'s, blot etc.',NULL,1111,2,2),(##9-03-23&&

1030    Q: How does insulin remove glucose from the blood and how does the pancreas \"slow down\" the production of insulin

1030    A: High glucose triggers movement of glucose transporters in muscle and adipose. The transporters are already made, but are in vesicles near the plasma membrane; high glucose in blood --> release of insulin --> signal for vesicles to fuse with plasma membrane, inserting glucose transporters into plasma membrane where they promote uptake of glucose. \r\n\r\n<p>How is insulin itself released? It is normally made (& receptors for it are too) whenever you eat. The insulin is inside vesicles near the plasma membrane (in pancreatic cells) and the receptors are on the surface of the target cells. When you heat --> High glucose in blood, and this causes the vesicles containing insulin to fuse with the plasma membrane, releasing the insulin to the outside of the cell --> blood --> target cells. So there are two sets of vesicles here, one (in pancreatic cells) containing insulin (to be secreted) and one (in the target cells) containing glucose transporters (to be inserted in the membrane as needed). Insulin has other effects that promote glucose uptake and utilization -- it stimulates many enzymes of glucose metabolism as shown on the handout.',NULL,1112,2,2),(##9-03-24&&

1031    Q: What exactly is coded for in the \"linker\" DNA between the histone octet? Are all exons only found around the histones and all intron stuff in the linker DNA

1031    A: Nucleosomes form more or less irrespective of the content of the DNA. So exons, introns, spacers -- it makes no difference, all are packed into nucleosomes in a regular way.',NULL,1114,2,2),(##9-03-24&&

1032    Q: Are there two different kinds of CDK (in addition to the two different kinds of cyclin) -- one for each of the checkpoints

1032    A: Yes. (In most organisms.)',NULL,1115,2,2),(##9-03-24&&

1033    Q: If estrogen down-regulates prolactin receptors causing no response to prolactin and allowing lactation to occur only after birth when estrogen levels fall, then how come you don\'t have lactation before estrogen levels rise before pregnancy

1033    A: Prolactin levels are too low before pregnancy to trigger lactation. PL rises during pregnancy, but doesn\'t work because estrogen suppresses the receptors.',NULL,1116,3,2),(##9-04-06&&

1034    Q: What\'s the difference between iodine and iodide

1034    A: The two terms are often used interchangeably.',NULL,1117,3,2),(##9-04-06&&

1035    Q: Do hormones always have to travel in the blood. Purves says that some are taken up so quickly by neighboring cells that they don\'t. &&

1035    A: Hormones can travel in the blood or act locally. The terms endocrine, paracrine and autocrine are used to distinguish the types on the basis of where they act (far away, locally, and on the cell that makes them, respectively). The classic hormones are endocrines that travel in the blood, so the term \"hormone\" and \"endocrine hormone\" or \"endocrine\" are sometimes used as synonyms, especially in older books.',NULL,1118,3,2),(##9-04-06&&

1036    Q: Are any cells that are replaced by stem cells, such as skin cells, not created through the process of mitosis of an existing skin cell? Can you fill out this picture for me

1036    A: The stem cells divide by mitosis. One daughter remains a stem cell and is ready to divide again. The other daughter differentiates (specializes) to become skin, or RBC or whatever, depending on what type of stem cell it is and what signal it gets. Some stem cells can only give rise to one cell type; some can give rise to several cell types. Not all tissues are replaced/grow using stem cells -- In some tissues there are no stem cells, and the specialized cells just divide to make more of the same. But for many tissues, specialization and division are incompatible -- once a cell is differentiated, it can\'t divide any more and stem cells are needed to replace it if it dies.',NULL,1119,3,2),(##9-04-13&&

1037    Q: What does teleological point mean? Is it the overall function served by hormone action

1037    A: Yes. It means, \"what is the rationale of having this function, structure, etc.?\" Often the answer is only a guess or a rationalization after the fact.',NULL,1120,3,2),(##9-04-13&&

1038    Q: In problem 7-1 (D), why is it correct that a single hormone may be secreted by more than one endocrine gland? Because androgens are secreted by both the ovaries and testes? What about estrogen and testosterone? Are both these hormones secreted by both the ovaries and the testes

1038    A: Some sex steroids are made by the adrenal cortex, as well as the gonads. There are multiple examples of more than one gland --> the same hormone.',NULL,1121,3,2),(##9-04-13&&

1039    Q: In problem 7-3, why is (a) follicle cells of thyroid, (b) hormonal stimulation only, and not also (c)change in concentration of a chemical in its surrroundings right? Doesn\'t iodine concentration influence the secretion of TH

1039    A: Iodine is needed to make TH. But the amount of TH you make and release (under normal circumstances) does not fluctuate with the amount of I around. The amount of I can limit the amount of TH made, but adding I won\'t cause release of TH (unless it relieves a deficiency of I). An analogy: You don\'t regulate the stove according to wood available, but according to temperature. (Even though wood supply influences how much heat you can get.)',NULL,1122,3,2),(##9-04-13&&

1040    Q: In problem 15-8, why should CDK be considered a proto-oncogene

1040    A: Because CDK stimulates cell division normally, but when it is mutated (as in 4-11) is causes uncontrolled growth.',NULL,1123,3,2),(##9-04-19&&

1041    Q: How is it that goiter is caused both by overstimulation of and understimulation of the thyroid gland

1041    A: This is not the right way to put it, and I apologize for causing confusion. What would be better is to say that overstimulation (by TSH or other factors) or failure to feedback inhibit (due to underproduction of TH) causes goiter. What I should have said is that goiter can be associated with either over production or under production of TH.',NULL,1124,3,2),(##9-04-19&&

1042    Q: Problem 7-*7 (B), it seems to contradict what was said in the notes. Is it true that  failure of thyrotropin (TSH) receptors won\'t cause goiter

1042    A: This is correct; if no TSH reaches the thyroid, the thyroid will not respond.',NULL,1125,3,2),(##9-04-19&&

1043    Q: How is it that understimulation of the thyroid produces goiter through hyperthyroidism

1043    A: What is meant here is that failure to produce TH will cause lack of feedback inhibition of the anterior pituitary and HT and cause overproduction of TRH and TSH, thereby causing goiter. (This is assuming TSH receptors work.)',NULL,1126,3,2),(##9-04-19&&

1044    Q: Problem 7-5 says that low levels of TSH and low levels of TH caused by pituitary damage cause hypothyroidism. Does this cause goiter

1044    A: I think not. You get goiter when there is too much TSH (or over stimulation of receptors). This can be due to either of two things: failure of negative feedback by TH or overproduction of TSH (or agonists).',NULL,1127,3,2),(##9-04-19&&

1045    Q: Sherwood says that, by definition, a hormone is secreted into the blood and travels a long distance, but Purves speaks of hormones acting locally and not entering the blood stream (paracrine). Which is right

1045    A: The term paracrine was invented to solve this problem; endocrine is reserved for the classic long distance hormones.',NULL,1130,3,2),(##9-04-19&&

1046    Q: What does problem 7-4 (6) mean when it says \"the follicle cells\"? Is that shifting the focus to inside the thyroid and thereby talking about thyroglobulin, which does have an amino acid attached? But does one really refer to thyroglobulin as a hormone, which the question/answer would imply

1046    A: TH is NOT a steroid, but has some of the same properties. Thyroglobulin is a protein containing modified Tyr\'s, NOT a hormone, but a potential source or storehouse of hormone. Thyroglobulin is a precursor to the hormone. When the thyroglobulin is cut up into individual amino acids (inside the follicle cells), some of those AA will be TH (= the actual hormone) and will diffuse out of the cells into the blood stream. The follicle cells form spheres inside the thyroid, and the modification of thyroglobin and production of TH takes place in the cells or in the hollow middle of the sphere (the \"colloid\"). Both the hormone (TH) and its precursor (thyroglobulin) are made in the follicle cells.',NULL,1131,3,2),(##9-04-19&&

1047    Q: Is T4 the same as Thyroxine

1047    A: Yes, but both T4 and T3 are known as thyroid hormone(s). Thyroxine refers only to T4. ',NULL,1132,3,2),(##9-04-20&&

1048    Q: Referring to problem 7-2 (A), is there EVER a case in which hormones are secreted through ducts into the blood

1048    A: Hormones are defined as being made by \"ductless\" glands. Glands with ducts make secretions, but they aren\'t considered hormones. If there is an exception, I don\'t know about it either. ',NULL,1133,3,2),(##9-04-21&&

1049    Q: What\'s difference between ACh and AcCH. When is each used

1049    A: They are exactly the same. Both are abbreviations for Acetyl choline.',NULL,1134,3,2),(##9-04-21&&

1050 Q: How does the pituitary lead to the production of cortisol? Does it secerte steroid hormones, or is it that peptide hormones such as ACTH stimulate the cortisol? Is it safe to say that peptide hormones transform cholesterol into a steroid? &&

1050 A: No steroids from the pit. ACTH stimulates the production of cortisol etc. It does so by affecting enzymes that make and interconvert the steroids. See exam #3 of \'98 for the pathway. (Cholesterol is a steroid too, but not a hormone.)',NULL,1135,3,2),(##9-04-26&&

1051 Q: In problem *8-27, shouldn\'t there be four types of channels? The three the answers given plus a K+ voltage gated channel (for the refractory period of the AP)? Or does the mechanically gated channel operate as both

1051 A: This comes from a real situation, and my sources don\'t reveal the answer. Either there are additional channels, or K+ leak channels take care of restoring the RP (it just takes longer). I doubt if channels could be both mechanically and voltage gated, but as you suggest, that would do it too.',NULL,1137,3,2),(##9-04-26&&

1052 Q: What is the difference between Growth hormones and growth factors

1052 A: Growth factors are a class of proteins that usually act as paracrines or autocrines. They are not usually made by glands, just by \"ordinary\" cells. They stimulate growth, differentiation, etc. Growth hormone is a specific, endocrine hormone that affects certain aspects of growth and metabolism (see a text for details).',NULL,1138,3,2),(##9-04-26&&

1053 Q: Dr. Firestein said in lecture that the resting potential of a neuron was -90mV, the threshold was -60mV, and the peak of the action potential was at +55mV. In Purves, however, it says that the numbers are -60mV, -40mV, and +50 mV respectively. Which is correct

1053 A: Different cells and organisms have different values, which is why the books are different. It depends what you take as your standard. It\'s the approximate and relative sizes (and signs) that matter.',NULL,1139,3,2),(##9-04-26&&

1054 Q: In problem 9-2, isn\'t it possible that calsequestrin could be located in the intracellular space of a muscle cell, since the molecule could be used to quickly bind several Ca++ ions to stop a twitch and take the Ca++ back into the SR for reuse

1054 A: Virtually all the Ca++ is stored in the SR. So the Ca++ \"sink\" is likely to be there. You could have a middle man, but why bother? And how would you get the Ca++ from calseq. back into the SR without having any lying around?',NULL,1141,3,2),(##9-04-26&&

1055 Q: The answer to 1999\'s exam 3, question 2 says that the production/release of prolactin would decrease in response to suckling because input from the HT would be cut off when blood supply was blocked. However, I thought that the HT produced Release INHIBITOR for prolactin, in which case I would have thought that the amout of prolactin produced/released would increase when the supply of HT hormones were cut off from the pituitary.&&

1055 A: There are TWO factors in the HT that affect prolactin release -- an inhibitory factor (usually called PIH) and a stimulatory factor (PRF). Suckling would cause the HT to produce PRF which would increase secretion (normally) of PL.',NULL,1142,3,2),(##9-04-30&&

1056    Q: What determines the extent of its refractory period in muscle? Do the threshold voltages of the voltage gated channels determine this

1056    A: Muscle does have to fire a full action potential, but it takes less input (in number of impulses) to fire one. A single AP in the neuron releases enough transmitter (gives one EPP Not an EPSP) to depolarize enough to cause an AP. The AP is very short, and as soon as cell repolarizes, a second AP can fire. I assume that the voltage gated channels in muscle have a short refractory period. The critical feature is that the AP is much narrower than the twitch, so the second AP can be fired before the first twitch is over.',NULL,1143,3,2),(##9-05-03&&

1057    Q: What is the distinction between secretion and excretion

1057    A: I was afraid this point was confusing and was not sure what to say to make it clearer without going off on a long tangent. So I\'m glad you asked. The process of dumping unwanted stuff out in the urine (overall) is known as excretion. However, when the cells surrounding the kidney actively pump stuff into the tubule (against a gradient), that is called secretion. Cells secrete substances so the substances can be excreted in the urine. What process you say is going on tends to depend if you are looking at individual cells or the body or kidney overall. Perhaps it is best to say you have both going on at once.',NULL,1144,4,2),(##9-05-06&&

1058    Q: I thought that exhaling to blow up a balloon would be something requiring only voluntary contraction of skeletal muscle. Am I thinking about this the wrong way

1058    A: Remember that you breath involuntarily, using signal from the medula oblongata to trigger motor neurons that trigger skeletal muscle. If you consciously need to breathe harder, you increase the contractions of those muscles and call in additional ones. Inhaling uses the contraction of the diaphragm and intercostals; exhaling normally occurs when those muscles relax. (The air is not actively pushed out by any contractions -- it flows out as a result of muscle relaxation.) To exhale forcefully, you contract muscles -- another set of intercostals and abdominal muscles -- to force air out. ',NULL,1145,4,2),(###-05-02&&

1059    Q: In cross-bridge formation, what does it mean when myosin ATPase is \"actin activated\"

1059    A: He means that there is no breaking of ATP unless actin is around. Myosin by itself doesn\'t split ATP. He is not refering to the exact position of \r\nactin vs. myosin in the bridge cycle.',NULL,1524,4,2),(###-05-02&&

1060    Q: Since cytotoxic T-cells differentiate into memory cells and effector cells (just like Helper T-Cells and B-cells), is it possible that the cytotoxic T-cells have a problem that prevents differentiation into memory cells even though their effector variety \r\nis effective in destroying the virus infected cells

1060    A: Memory cytotoxic T-Cells would not be necessary to attack secondary infected cells because memory B-Cells would have eliminated the viral antigen in the humors before infection of cells is possible.',NULL,1525,4,2),(###-04-20&&

1061    Q: About marathon runners drinking too much water. How does hyponatremia work

1061   A: I apologize for the delay in answering.\r\nI think the major problem is that water diffuses from the dilute plasma into cells, causing swelling. This is especially a problem with the brain because it is encased in bone.\r\n<p>For more details see\r\n<a href = \"http://www.emedicine.com/emerg/topic275.htm\">http://www.emedicine.com/emerg/topic275.htm</a>\r\n<p>The original article is about runners is\r\n<a href=\"http://content.nejm.org/cgi/content/full/352/15/1550\">http://content.nejm.org/cgi/content/full/352/15/1550</a>',NULL,1523,4,2),(##9-05-07&&

1062   Q: Why would B cells have MHC I? I thought they only had MHC II. &&

1062   A: They have both. All nucleated cells have MHC I, but only cells of the immune system ALSO have MHC II.',NULL,1147,4,2),(##9-05-10&&

1063   Q: Which is the primary pacemaker? The SA node or AV ndoe

1063   A: The SA node is the primary one and the AV relays the message to the ventricles and acts as a back up. ',NULL,1148,4,2),(###-05-03&&

1064   Q: The answer to problem 13-13 (C-2), says that cytotoxic T-Cells \"lyse\" infected cells with perforin, but in fact perforin creates pores that allow other proteins to enter the \r\ninfected cell and trigger \"apoptosis.\" Is apoptosis a type of \"lysis\" or is the answer key based on older data about the function of perforin

1064   A: According to most elementary texts, perforin lyses eukaryotic cells. However we now know the destruction is by apoptosis. Thanks for pointing \r\nthis out -- I\'ll fix the next edition of the problem book.',NULL,1527,4,2),(##9-05-10&&

1065   Q: What do the two ATP do in the contraction-relaxation cycle of smooth muscle?\r\n\r\n&&

1065   A: It takes two ATP rounds of ATP for smooth muscle contraction because one phosphorylates myosin (to activate it) and then additional ATP\'s are required to make and/or break and reform bridges with actin. It isn\'t two ATP per bridge cycle -- for a single myosin molecule it takes one round of phosphorylations by ATP to activate the myosin, and then that particular myosin can bind and rebind to actin (using one ATP per cycle) without additional activation. (The stochiometry is complex because there are actually two sites of phosphorylation of myosin heads involved in activation, not one; see Becker fig. 21-23 for details of activation part.)',NULL,1149,4,2),(###-05-02&&

1066   Q: Do helper T-cells activate cytotoxic T-cells (via cytokines) by a MHC I/CD8 binding? Since all nucleated cells have MHC I, do Helper T\'s and B cells also have it

1066   A: Helper T\'s and cytotoxic T\'s do not form a complex. The helper T\'s secrete IL\'s that help the cytotoxic T\'s. the signaling is paracrine, not \r\njuxtacrine.\r\n<p>Helper T\'s and B cells do have MHC I.',NULL,1526,4,2),(##9-05-10&&

1067   Q: Do we have to know all the properties of the five antibody classes (Problem 13-1 B-2)

1067   A: No. You should realize which part of the Ab is responsible for the differences and be able to answer the questions with the table in hand.',NULL,1150,4,2),(##9-05-11&&

1068    Q: Why does a high temperature lead to more $O_2$ dissociation than a low temperature

1068    A: We\'re talking cellular temperature. If cell is working hard, it will be warm, and that\'s a signal to pick up more $O_2$. If you are shivering over all, the individual cell is generating a lot of heat.',NULL,1151,4,2),(###-05-05&&

1069    Q: Are the different kinds of memory cells generated by stopping transcription at various sites on the DNA or by splicing one\r\nRNA transcript in different ways

1069    A: The cells with IgA, IgE or IgG on their surfaces have their DNA recombined differently. It\'s not a difference in transcription or splicing using the \r\nsame DNA.',NULL,1528,4,2),(###-05-05&&

1070    Q: What is the difference in function of the different light chains (lambda and kappa)

1070    A: No one knows -- the two types of light chain seem to be interchangeable.',NULL,1529,4,2),(###-05-05&&

1071    Q: Does the fact that B cells may change the antibody they product imply that they\r\nare not specific for a single antigen, but rather for a type or class of antigen and then evolve to be specific for only one antigen

1071    A: First of all, you can Change the class of antibody, which does not change the antigen binding part. Superimposed on all this is somatic \r\nmutation which slightly changes the antigen binding part. As a result there are multiple versions of the original ag binding part. When ag comes \r\nalong the next time, it binds to the one that has the strongest affinity.',NULL,1530,4,2),(##9-06-03&&

1072    Q: Regarding question 12-12 (B), since low aldosterone will cause a decrease in Na+ reabsorbtion, won\'t water also be lost because it follows the Na+ out? In part (C), will vasopressin help too

1072    A: The primary problem is the Na+; you are correct that water follows salt. Vasopressin is presumably already being made in max. amounts (in response to low BP); if that could fix the problem it already would have.',NULL,1157,4,2),(###-01-27&&

1073    Q: According to Becker Chapter 4 (p.88), ribosomes are transported across the nuclear envelope. I thought there were no ribosomes in the nucleus.&&

1073    A: Ribosomes are transported OUT of the nucleus, not in. It is really newly made ribosomal subunits that are transported out. There are no complete working ribosomes (2 subunits together, attached to mRNA) in the nucleus. We will discuss this in more detail in a week or so.',NULL,1158,1,2),(###-01-27&&

1074    Q: Why can only water-soluble molecules move through nuclear pores? Is it because of the phospholipid bilayers of the membranes

1074    A: The bilayer itself is a barrier to water soluble molecules. The pores are holes or breaks in the bilayer that contain water filled spaces. So water soluble materials that are small enough diffuse through the pores.',NULL,1159,1,2),(###-01-27&&

1075    Q: How does immunofluorescence allow for more fluorescent signal

1075    A: One secondary antibody binds to each antigenic site (on the primary antibody), but there are multiple antigenic sites on each primary antibody. So many molecules of secondary antibody stick to each molecule of primary antibody.',NULL,1160,1,2),(###-01-27&&

1076    Q: Which organelles have only one membrane and which have two?\r\n\r\n&&

1076    A: The nuclear envelope, mitochondria, and chloroplasts have two membranes, each made up of a bilayer, and the perioxisomes, lysosomes, ER and Golgi bodies each have one membrane of a single bilayer.',NULL,1163,1,2),(###-01-27&&

1077    Q: Do the same type of vesicles transport things into and out of the cell as those that transport things throughout the cell

1077    A: There are many different kinds of vesicles. They have to be different in order to be specifically directed to different places. Secretory vesicles have to go to the plasma membrane; transport vesicles have to go to the right target organelle. The different types contain different membrane proteins and different cargo (contents). We will get to more details later, but will leave some of the aspects for an advanced cell biology course. For more information on transport, see Becker esp. chap. 12.',NULL,1164,1,2),(###-01-28&&

1078    Q: Are peroxisomes not considered part of the endomembrane system because they don\'t bud off from the Golgi

1078    A: Yes. Also vesicles do not exchange material between any part of the endo. mem. syst. and peroxisomes. The enzymes that are inside peroxisomes are not made on the ER.',NULL,1165,1,2),(###-01-28&&

1079    Q: Is a lysosome defined as an endosome with its enzymes acting on a substrate, or can a lysosome be considered the organelle containing the hydrolases, even if they are not currently acting on a substrate

1079    A: The definition of a lysosome is variable -- it depends what you read. Some texts distinguish primary lysosomes (with hydrolases only) from secondary lysosomes (those containing substrate as well as enzymes). Others call the vesicles with just enzymes \"late endosomes\" and the vesicles with substrate \"lysosomes.\" Note: more info on these organelles is to be found in chap. 12 of Becker, and will be discussed later in the term.',NULL,1166,1,2),(###-01-28&&

1080    Q: Box 12A, figure 3 (p. 333 in Becker), shows organelle densities and sedimentation coefficients. Shouldn\'t the density and sedimentation coefficients (S values) have a positive, proportional relationship

1080    A: No. Density depends on protein/lipid ratios. It is independent of size (mass, friction due to shape, etc.) S values depend on the mass/friction ratio which depends on size, shape, etc.',NULL,1167,1,2),(###-01-28&&

1081    Q: If you transfer a patch of membrane in addition to its contents, every time a vesicle fuses with a plasma membrane or another vesicle, won\'t the area of membrane decrease and eventually destroy the cell

1081    A: The whole system is constantly in a dynamic equilibrium, with membrane being added to and removed from each organelle, plasma membrane etc. Vesicles fuse with each membrane, but vesicles bud off from each one too. Otherwise, you are quite right -- the whole system would self destruct.',NULL,1168,1,2),(###-01-28&&

1082    Q: What does it mean that a vesicle is continuous inside and outside of the cell

1082    A: The inside of each vesicle or sac of the endomembrane system is topologically equivalent to the outside of the cell. Consider what happens when a vesicle fuses with the plasma membrane or invaginates. What was outside the cell becomes inside the vesicle and vice versa.',NULL,1169,1,2),(###-02-12&&

1083    Q: When do we assume that P stands for the inside of bilayer? It seems like the definition changes for different problems.&&

1083    A: The P face/side always means the side closer to the cytoplasm. Whether that is the \"inside\" or \"outside\" depends on how you look at it. It\'s always the outside (cytoplasmic side) of a vesicle or the ER but the inside (cytoplasmic side) of the plasma membrane.',NULL,1170,1,2),(###-02-04&&

1084    Q: Can neutral molecules ever pass through channels

1084    A: Channels are specific, and most channels are specific for transport of ions. You probably can\'t build a channel that specifically transports anything much bigger, although there are channels specific for water. Since most channels transport ions, many texts use the terms \"channels\" and \"ion channels\" interchangeably.',NULL,1171,1,2),(###-02-04&&

1085    Q: Are ion channels exactly the same as gap junctions? If not, what are the differences

1085    A: Gap junctions are usually placed in a class by themselves, or considered to be more like \"pores\" than \"channels.\" Pores and gap junctions have properties similar to that of many channels, but are not specific -- anything small enough can flow through.',NULL,1172,1,2),(###-02-04&&

1086    Q: What types of molecules can pass through channels

1086    A: The \"holes\" in channels and pores are water filled, that is, hydrophilic. Hydrophobic materials don\'t pass through channels, but they don\'t need channels or pores anyway -- they can dissolve in the lipid bilayer of a membrane and diffuse right through. Hydrophilic materials can\'t diffuse across the lipid bilayer, so they need a water filled area, which the pores and channels provide. Most channels are highly specific for one or a few ions or for water; most pores allow anything up to a certain size to pass through as explained above.',NULL,1173,1,2),(###-02-04&&

1087    Q: What is the structural/functional relationship between the ECM and the plasma membrane? What does Becker mean when he talks about the \"alignment\" of the intracelluar cytoskeleton and ECM (fig. 11-8)

1087    A: The ECM is outside the plasma membrane. Components in the ECM may be bound to integrins or other proteins of the plasma membrane. These membrane proteins may in turn be linked to the cytoskeleton. In some cases, the components of the ECM and parts of the cytoskeleton can be aligned, as in Becker fig. 11-8. That means they are laid out in the same pattern. The ECM is outside the cell, and the cytoskeleton is inside, but they are lined up in parallel in the same direction. Presumably, they are lined up in parallel because they are connected to (and aligned by) the same transmembrane protein. The intracellular domain of the transmembrane protein is connected to the cytoskeleton while the extracellular domain of the transmembrane protein is linked to the ECM.',NULL,1174,1,2),(###-02-14&&

1088    Q: What is the difference between a phosphatase and a kinase

1088    A: They are both enzymes (or parts of complex enzymes, pumps, etc.) but they catalyze different reactions. Phosphatases catalyze removal of a phosphate, or the reaction: <br>X-P (+ water) ---> X + P<br><br>Kinases catalyze the transfer of a phosphate from ATP so you end up with phosphorylated X and ADP, or the reaction:<br>X + ATP --> X-P + ADP.<br><br>If X is a protein, you are dealing with a protein kinase or protein phosphatase.',NULL,1175,1,2),(###-02-12&&

1089    Q: In RME, are receptors transmembrane proteins

1089    A: Yes.',NULL,1176,1,2),(###-02-12&&

1090    Q: What triggers the joining of the ligand to the receptor in RME

1090    A: Same as enzyme and substrate -- they bump into each other and since they fit/match, they stick, due to formation of multiple weak bonds.',NULL,1177,1,2),(###-02-12&&

1091    Q: Is RME considered a type of active transport? Which of the 5 categories of transport would it be in, if it would be in any

1091    A: Yes it\'s active, but not really one of the 5 categories. It\'s usually considered in a separate category. Types 1-5 are for small molecules; RME is for macromolecules.',NULL,1178,1,2),(###-02-12&&

1092    Q: Can glucose diffuse into cells without a carrier protein or channel

1092    A: No. Glucose diffuses through the liquid in the spaces BETWEEN the cells, not through the cell membrane. The cells surrounding the capillary are not tightly joined and there are liquid filled spaces between them big enough for small molecules (but not proteins) to diffuse through.',NULL,1179,1,2),(###-02-12&&

1093    Q: If lipids are made and then inserted into ER, how do new lipids reach organelles that do not recieve vesicles from the ER or Golgi complex (e.g. mitochondria)

1093    A: There are transport proteins that bind and carry lipids to otherorganelles.',NULL,1180,1,2),(###-02-12&&

1094    Q: Are new lipids also inserted into cytoplasmic side of SER in the same way they are in the RER

1094    A: Yes.',NULL,1181,1,2),(###-02-12&&

1095    Q: What happens to the stuff that gets broken down by the lysosome and not used by the cell

1095    A: Some big undigested stuff in the lysosome is exocytosed and some just lies around. Depends on the stuff and the cell type. Small stuff that cells don\'t use, either gets stuck in membranes (if it is hydrophobic) or washed out into the blood and then into the urine if it is water soluble.',NULL,1182,1,2),(###-02-12&&

1096    Q: Do vescicles bud off the lysosomes and go to the ER, Golgi, and plasma membrane

1096    A: No. Vesicles don\'t carry digested stuff away from the lysosome. The products of digestion are generally small and diffuse out of the lysosomes and into the cytoplasm.',NULL,1183,1,2),(###-02-14&&

1097    Q: What exactly is a localization sequence and how does it differ from a signal/start sequence

1097    A: The terminology for this is not perfect and varies a lot from person to person and text to text. Localization sequence or signal is  generally used in a broad sense to mean any sequence that directs a protein to a particular part of the cell. Sometimes the term \"signal sequence\" is used in the same general meaning as localization signal. More often it (or the term signal peptide) is used for the specific type of localization seq. that directs insertion into the ER. (Or in a similar way, into the inner membrane of mito & chloro.) Start seq. refers to a hydrophobic sequence, which may include the SP or not, depending on context, that allows (re) start of transfer of a peptide across the ER (or inner mito) membrane.',NULL,1184,1,2),(###-03-03&&

1098    Q: How does G-protein (with GTP, active form) activate adenylate cyclase

1098    A: When the alpha subunit is bound to GTP, it dissociates from the beta/gamma subunits, and diffuses through the cytoplasm till it meets adenylate cyclase. The cyclase is usually in an inactive form, but when G-alpha binds to it, its conformation changes to become active. The exact details of the conformational change are not known.',NULL,1185,2,2),(###-03-03&&

1099    Q: What hydrolyzes the G-proteins alpha subunit back to GDP?\r\n&&

1099    A: The alpha subunit is a GTPase, so it hydrolyzes the GTP to GDP. G(alpha)-GDP then dissociates from the enzyme (phospholipase C) and reattaches to the gamma/beta subunits. ',NULL,1186,2,2),(###-03-06&&

1100    Q: Are signal peptides considered localization signals

1100    A: Yes. I guess you could call them \"endosomal membrane system\" localization signals.',NULL,1491,2,2),(###-03-06&&

1101    Q: Question #4 A-1 from Exam 1 states that \"you make an organism that has a fusion protein consisting of GFP attached to one end of pepT.\" Since we know that both ends of the protein are in the cytoplasm, why is the answer that fluorescence is found in the apical part of the membrane (as opposed to in the cytoplasm?)&&

1101    A: The protein is embedded in the membrane. The size of the part that sticks out on either side, relative to the part in the membrane, is trivial.',NULL,1492,1,2),(###-03-06&&

1102 Q: Are there any TF\'s that are cis-acting

1102 A: There aren\'t any cis acting proteins to speak of. We consider them ALL to be trans acting. The DNA elements can be cis or trans, depending on whether they code for a protein or not, but the proteins are all trans.',NULL,1493,2,2),(###-03-04&&

1103 Q: Do you expect us to know the cAMP and Ca++ pathways by heart

1103 A: You should know receptor > G protein > adenylyl cyclase > cAMP > protein kinase, phosphorylates something. And you should know what glycogen synthetase and glycogen phosphorylase do. So it\'s okay if you forget that the enzyme in between is called phosphorylase kinase.',NULL,1187,2,2),(###-03-05&&

1104 Q: Can you clarify if the suckling infant  conveys nerve impulses to posterior pituiary or to the hypothalamus that in turn stimulate the posterior pituitary to release oxytocin

1104 A: The cells that release oxytocin have their origin in the hypothalamus and their terminals in the posterior pituitary.  The part of the cell that\'s in the hypothalamus includes the cell body and dendrites, which is where the cell receives signals from other neurons, so the nerve impulses initiated by the pressure of sucking will come to this part of the cell in the hypothalamus.  This cell then develops a nerve impulse, which travels down the cell to the terminal of the cell, which is in the posterior pituitary, and oxytocin is then released from the terminal.',NULL,1188,2,2),(###-03-07&&

1105 Q: We learned that the fate of hormones deals with enzymes that make the hormone less active. Why would the hormone become less active? Wouldn\'t a cell want the hormone to be as active as possible for as long as possible

1105 A: If a cell secreted a hormone and the hormone were never inactivated, the cell would have no control over communication with distant targets. If it\'s necessary for a target cell to get a message for a long time, then the signalling cell just keeps sending out the signal, ie, secreting more of the hormone. Newly-secreted hormone stimulates the target as old hormone is inactivated, but there\'s a certain steady state concentration of hormone which continually stimulates the target cell. When the signal\'s not needed any more, the signalling cell just stops secreting, and the inactivation process continues, such that a new, lower hormone concentration is reached.',NULL,1189,2,2),(###-03-13&&

1106 Q: Why does the signal peptide of a peptide traveling to a mitochondrion get chopped off when the same isn\'t true for a peptide going to a peroxisome

1106 A: That\'s just the way it is! There are several after the fact rationales. For at least some mitochondrial proteins, the TP must be removed so that additional localization signal(s) can be revealed and used.',NULL,1190,2,2),(###-03-13&&

1107 Q: Is it correct to say that hypersensitive regions of DNA do not have nucleosomes ONLY when nearby gene is being transcribed and otherwise they do

1107 A: Yes, except that a \"hypersensitive region\" that has nucleosomes on it will not be hypersensitive! So it should be called something else. \"Hypersensitive\" refers to a state of sensitivity to degradation by nucleases. It is not a permanent condition -- it is a condition that certain sections of

chromatin/DNA are found in at specific times only. (Other sections of the DNA are never hypersensitive at any time.)',NULL,1191,2,2),(###-03-13&&

1108    Q: Can you explain what the differences are between mRNA and primary transcript? Is the primary transcript the mRNA before any splicing events occur

1108    A: Basically, your answer is correct. However, splicing, poly A addition and transcription all can overlap, so it is hard to make a strict definition that never fails. Usually, primary transcript refers to RNA that still needs to be spliced, but has its cap and polyA tail. The point is to make a distinction between mRNA (which is completely processed -- cap and poly A added, introns removed) and \"pre-mRNA\" or RNA that is not yet completely processed. The situation is complex because there are many steps in processing and so a nucleus can contain many different intermediates -- different pre-mRNA\'s at different states of processing/splicing.',NULL,1192,2,2),(###-03-13&&

1109    Q: Do all regulatory TFs ALWAYS have a dimerization domain that may be reposnsible for binding to other molecules of itself? Also, wouldn\'t the dimerization, and not the transcriptional regulation domain be responsible domain for homodimerization

1109    A: As far as I know, not all TF\'s have to dimerize in order to work (although I think most do). So a TF that works as a monomer does not need a dimerization domain. If dimers form, that is due to the dimerization domain (by definition) whether homo or hetero dimers form. But if additional sections of the protein are still needed to activate transcription, those would be called activation domains. The question is, what do they do?? Probably they bind to other proteins, but how does that help? No one knows all the answers, but the \"other proteins\" are thought to loosen up chromatin or causes changes in conformation of RNA polymerase or other critical factors. Details are known in some cases but there is no overall simple answer.',NULL,1193,2,2),(###-03-13&&

1110    Q: The article titled \"DNAs Wrapping Holds Key\" says \"sometimes, both the histone protein and its competitor are hooked onto the DNA simultaneously, apparently as a way of activating the gene - but only modestly or in spurts.\" In this case, would you only transcribe stuff that\'s in  the linker region , since the rest of the DNA is still wound up in the nucleosome? I can\'t imagine anything that useful coming out of 50 bp sequence.&&

1110    A: I don\'t think they mean that only the linker is transcribed. They mean that binding is a signal to open/loosen up (briefly) the promotor so the corresponding gene can be transcribed.',NULL,1194,2,2),(###-03-13&&

1111    Q: Can you clarify the meaning of \"core promoter?\"&&

1111    A: What is meant by \"the core promotor\" varies from person to person. Some use it to mean the place where polymerase binds (not including the start of transcription) and some use it to mean the area around where the polymerase binds, including the start of transcription. That\'s why your sources are contradictory. I generally use it to mean the RNA pol binding site, but you have to be alert to other uses.',NULL,1195,2,2),(###-03-13&&

1112    Q: When a protein gets inserted into the ER, why is it that a hydrophobic region is the reason it gets stuck when such sections are presumably lipid soluble

1112   A: A hydrophobic section is soluble in the lipid bilayer. If the entire molecule is hydrophobic, it diffuses right through. But if the hydrophobic section is connected to a hydrophilic section, then the hydrophobic section passes into and floats in the lipid bilayer but the remaining section can\'t pass through and the protein is stuck.',NULL,1197,2,2),(###-03-13&&

1113   Q: In alternative splicing, how exactly does the cell avoid poly-A addition after a particular exon

1113   A: No one really knows. If the splicing enzymes get there first, there is no poly Aaddition site for poly A to add to. But no one is sure what determines that splicing enzymes get there first after exposure to antigen but not before.',NULL,1198,2,2),(###-04-08&&

1114   Q: What exactly is the difference between \"induction\" and \"regulative development\"? The line between the two is quite hazy to me.&&

1114   A: Induction is the mechanism responsible for achieving regulative development. Signaling between cells leads to induction; the series of inductions produces regulative development.',NULL,1199,3,2),(###-04-15&&

1115   Q: What are pupa in drosphilia

1115   A: Pupa = chrysalis = stage during which metamorphosis takes place (when larva becomes fly).',NULL,1200,3,2),(###-04-15&&

1116   Q: In the notes and class you said differentiation occurs by progressively turning off genes. But aren\'t your really turning genes on, since the default state is off

1116   A: You are turning more and more genes \"irreversibly\" off (baring a nuclear transfer into an egg). The selection of genes that can be turned on is narrowing with time.',NULL,1201,3,2),(###-04-15&&

1117   Q: After the dimerization of TK-linked receptors, are the adapter proteins recruited or only the proteins the adapter proteins bind to

1117   A: Anything that binds to/is localized to the site of an activated protein is considered \"recruited.\"',NULL,1202,3,2),(###-04-15&&

1118   Q: What would the results of the fusion experiments (at different stages of cell cycle) have been if control was via release of an off switch? I don\'t understand how removing an inhibitor would be different from adding a positive signal like phosphorylation.&&

1118   A: If you needed to remove an inhibitor to enter S, then that inhibitor would be present after fusion, and it would prevent entry into S (unless it was diluted too much to work).',NULL,1203,3,2),(###-04-15&&

1119   Q: Which checkpoint do rb-EF and pP53 affect

1119   A: Only the between G1-S, <i>not</i> the checkpoint at G2-M.',NULL,1204,3,2),(###-04-15&&

1120   Q: In cancer cured by CML, is the cancer caused by autocrine stimulation of receptors for growth? Is that why blocking receptors stops growth of tumor

1120    A: CML is the name of a cancer, not a treatment. In cases where  paracrine/autocrine stimulation of receptors triggers growth, blocking receptors should stop/slow growth of tumor. (Erbitux is supposed to do this.) In CML it\'s actually a different case -- there is a constitutive TK inside the cell that triggers unregulated growth. Gleevec is supposed to block the activity of the TK.',NULL,1205,3,2),(###-05-09&&

1121    Q: Since MHC I is on the cell surface of all nucleated cells, is it true that the immune system cells (B cells, macrophages, etc) have both MHC I and MHC II their surfaces? If this is so, can you explain how this impacts the specificity of cellular response with respect to cytotoxic T vs. helper T binding

1121    A: Yes. Cytotoxic T\'s only bind to cells with MHC I that contains an epitope -- piece of antigen. A cell that is not infected or \"gone bad\" has MHC I, but no antigen attached to it. (The MHC I will have bits of protein attached to it, but the protein will be normal -- not an antigen.)  So cytotoxic T\'s do not respond. If the cell is infected, even if it has MHC II also, the cytotoxic T\'s will bind to the MHC I + antigen and destroy the cell. Helper T\'s only bind to cells with MHC II + antigen. (If cells have MHC I + antigen, as well as MHC I + antigen, the cytotoxic T\'s should get to the cell first and zap it.) So only uninfected APC\'s should be \"helped\" by binding to helper T\'s.',NULL,1206,3,2),(###-05-09&&

1122    Q: How do helper Ts fit into the cell mediated response

1122    A: T helpers secrete a cytokine (IL-2) that is needed by cytotoxic T\'s. I don\'t think it is known quite how the helpers find the \"right\" cytox. T\'s. You don\'t need the helper to bind to the cytotoxic Ts.',NULL,1207,3,2),(###-05-09&&

1123    Q: For problem 13-8 (A) (no immunity to virus infections), I am not sure how the B cells are eventually  activated. What are the steps involved (in other words, what cytokines released from which cells stimulate B cell activation)

1123    A: The idea here is that B cells don\'t get activated and don\'t make humoral antibody. The person recovers from viral infections using cytotoxic T\'s.',NULL,1208,3,2),(###-05-09&&

1124    Q: I thought antibodies only fight bacteria and soluble proteins. Do they attack viruses as well (which I thought only  the cytotoxic T cells attack?)&&

1124    A: Antibodies can attach to virus particles just as they attach to soluble proteins and/or bacteria. This is what happens if you are immunized or had a viral infection before -- the antibodies from memory cells (produced in response to the first infection or immunization) attach to the virus and the antibody coated virus is engulfed by macrophages. The virus never gets a chance to get inside cells and cause disease. However, if the virus is not intercepted by antibody and gets inside a cell and starts to replicate, the cytotoxic T\'s take over -- they destroy the infected cell and you recover (hopefully) from the infection. Cytotoxic T\'s do not attack virus particles themselves-- they attack cells infected by viruses.',NULL,1209,3,2),(###-05-10&&

1125    Q: Where does T helper fit in with activation of B cells?  &&

1125    A: The B-cell presenting the antigen to the helper T cell causes the helper T cell to release B growth factor, which in turn causes B-cell to differentiate into memory cells and plasma cells. In the case of most antigens, a B-cell with antigen attached to its receptor can\'t divide into memory and

plasma cells without helper T cells; however a few antigens can stimulate B cells without them.',NULL,1210,3,2),(###-05-10&&

1126    Q: Where do helper T cells fit in with activation of cytotoxic T cells

1126    A: There is no antigen presentation here (in the usual way). The two types of T\'s do not bind to each other through MHC, TCR, etc. However the helpers do provide cytokines that activate the cytotoxic T\'s. (I don\'t know how the helper T\'s know which cytotoxic T\'s to help. Last time I looked it up it wasn\'t known.)',NULL,1211,3,2),(###-05-10&&

1127    Q: How are helper T cells activated? Does a helper T stimulate itself

1127    A: Not exactly. Some other APC must initially activate the Th. After that, the helper T makes its own cytokines and receptors.',NULL,1213,3,2),(###-05-10&&

1128    Q: Why does a macrophage need to present antigen to a B-cell? Is this mostly in the primary response when lack of specialized memory cells forces more reliance on macrophages

1128    A: A macrophage does not present antigen to B cells. Both macrophages and B cells are classical APCs that present antigen to helper T cells. In the primary response, the macrophage presents antigen to a helper T. That helper T then activates a B. In a secondary response, the macrophage is dispensible and a B cell and a helper T can activate each other directly.',NULL,1214,3,2),(###-05-13&&

1129    Q: Sherwood says on p. 410-411 says that macrophages digest Ag and display it to B cells, activating the B\'s. Is this right

1129    A: As far as I can tell, this idea is incorrect. The standard immuology text that I consulted does not agree and neither does the prof. who teaches immunology. As we understand it, macrophages and B cells both display antigen to helper T cells. In the primary response, the macrophage displays to the T cell and activates the T cell and then the T cell finds a B (displaying the same antigen) and activates the B. In the secondary response, the B displays directly to a T and the B and T activate each other. So the B always needs a T to activate it. A macrophage cannot directly activate a B.',NULL,1215,3,2),(###-01-28&&

1130    Q: Does problem 1-2 (A-2) mean 100% for A, B, and C

1130    A: Yes. Answer says \"nothing is left.\" That is equivalent to saying \"100% of A, B and C is accessible\", meaning all lipids are accessible to degradation by the enzyme.',NULL,1217,1,2),(###-02-04&&

1131    Q: How do you visualize problem 1-2

1131    A: Once you open up (lyse) the RBC, disrupt, and reseal them, 1/2 the pieces of membrane reseal as they were originally,and 1/2 reseal inside out. So any particular lipid finds itself on the outside half of the bilayer about 1/2 the time. The other 1/2 of the time, it is protected, on the inside. So the enzymes can always digest 1/2 of each lipid -- the half in the outer bilayer (or leaflet). This is not the case in A, where the original orientation of the lipids is preserved. In the normal membrane, some types of lipid are more common on the inner half of the bilayer and some are more common on the outer half.',NULL,1218,1,2),(###-02-06&&

1132    Q: What is the difference between cell lysis and disruption

1132    A: Lyse means make a tear in the cell but leave the membrane all connected. Disrupt means break up the membrane into smaller pieces. Lyse = make a rip in a balloon so all the air leaks out; disrupt = blow it up until it explodes into small pieces.',NULL,1219,1,2),(###-01-28&&

1133    Q: Could you explain exactly why the ratio of monolayer to nuclear surface is 4 in problem 1-6

1133    A: The nucleus is surrounded by the nuclear envelope, which is equivalent to being surrounded by two membranes. Each membrane contains a bilayer of lipid. Each bilayer, consists of two, facing monolayers. So 2 membranes (bilayers) = 4 monolayers worth of lipid. In other words, there is enough lipid to coat the nuclear surface 4 times.',NULL,1220,1,2),(###-02-10&&

1134    Q: In cases of poisoning, why wouldn\'t evidence  be found in the smooth ER because it helps detoxify drugs and other poisons by adding hydroxyl groups, increasing their solubility and making them easier to flush from the body

1134    A: It\'s true that smooth ER helps detoxify, but I\'m not sure how that would help to find evidence of poisoning. I don\'t think you\'d find high levels of poison associated with the ER. (The drugs are probably not bound to the ER; if detoxed they\'d be gone.) You might find high levels of detoxification enzymes in the ER. In this problem, you are supposed to assume that the answer has something to do with the issues raised in the previous parts of the problem about trapping substances in acidic or basic environments.',NULL,1221,1,2),(###-02-04&&

1135    Q: I am a bit unsure how problem 2-5 relates to  \"theoretical reversibility\" versus \"practical reversibility.\" Can you give me an example of a situation where a pump the faces opposite its normal direction yet still pumps in the original direction

1135    A: The notes refer to keeping the membrane in place, in its usual orientation, and forcing the pump to run backwards, relative to its usual (chemical) direction. For example, using a very high ion gradient to drive ATP formation instead of using ATP hydrolysis to move ions. (This is unlikely for the Na/K pump in a living cell, but other pumps in the cell are actually used different ways at different times -- they can go in either direction, depending on the various gradients and concentrations.) The situation in problem 2-5 refers to physically turning the pump around, not reversing the chemical reaction. The pump still works in the usual way (from its point of view). The chemical reaction that is being catalyzed is not reversed -- the pump is still using ATP hydrolysis to move ions. However,what is \"in\" and what is \"out\" is reversed. (This doesn\'t happen naturally, only in laboratory situations.) ',NULL,1223,1,2),(###-02-10&&

1136    Q: The answer to problem 2-2 states that primary active transport is one of the possible methods of transporting hydrophilic and neutral substances F and G. However Purves states that primary active transport only works for cations. Please clarify.&&

1136    A: I believe that Purves is incorrect. A lot of primary transport involves cations, but there are some significant exceptions, primarily the family of so-called ABC transporters, which are largely (but not entirely) found in prokaryotes. These use the energy of ATP hydrolysis to transport a large variety of different substrates -- different proteins in the family transport widely different substances from ions to proteins. These transporters are important in drug resistance -- they help the target cells pump the drugs out of the cell.  ',NULL,1224,1,2),(###-02-12&&

1137    Q: How does one conclude from the graphs in problem 2-4 (B) that passive, not active transport is involved

1137    A: An unlabeled graph of conc. vs time doesn\'t tell you if conc. in = conc. out. The curve has the same shape whether conc. in and out at the end are the same or not. What gives it away that this is passive transport is that no ATP is present. So ser must be moving down its own gradient until equilibrium is reached with conc. in = out.',NULL,1225,1,2),(###-02-10&&

1138    Q: Can MF and MT both anchor proteins to the cytoskeleton

1138    A: There are no MT in RBC, but there are MT in normal cells. Transmembrane proteins could be anchored to the MT or MF in normal cells. I think that plasma membrane proteins are more likely to be attached to actin, as the support under the plasma membrane is thought to contain actin, as in RBC. However transmembrane proteins of organelles that are being transported are more likely to be attached to tubulin, since MT are thought to guide most vesicle transport through the cytoplasm.',NULL,1226,1,2),(###-02-12&&

1139    Q: In problem 3-3, does the protein insert into the ER first or directly into the plasma membrane? Is the approach of the protein to the plasma membrane via the cytoplasm or a secretory vesicle

1139    A: Proteins destined for the plasma membrane always insert first into the ER, and then travel to the plasma membrane via a vesicle.',NULL,1227,1,2),(###-02-12&&

1140    Q: In problem 3-13, can the imaginary protein #1 be thought of as a receptor protein? If not, why

1140    A: Doesn\'t seem like the best term for it to me. It links two other proteins (or a protein and a lipid) -- clathrin and something in the membrane. Such proteins are usually not called receptors. Receptor is usually used for something that is waiting to bind to something (= protein, hormone, etc. = ligand) that is added and trigger an appropriate response. Not everything that binds to another thing is called a receptor.',NULL,1228,1,2),(###-02-12&&

1141    Q: In problem 3-14 (D), why is hexokinase not included in the answer

1141    A: Hexokinase is an enzyme of glycolysis which is in the soluble cytoplasm -- that\'s where glycolysis is done.',NULL,1229,1,2),(###-02-14&&

1142    Q: The answer to problem 3-16 says that in I  disease, acid phosphatases aren\'t misdirected, but acid hydrolases are because they don\'t get M6P bound to them. What\'s the difference between an acid hydrolase and an acid phosphatase (and why don\'t acid phosphatases require M6P)

1142    A: Acid phosphatase is an acid hydrolase. However AP gets to the lysosomes by a different method than the other hydrolases, as indicated by the information in the problem. The localization signal that sends most hydrolases to the lysosome is the amino acid sequence/patch that triggers addition of M6P. The LS that directs AP to the lysosomes is an amino acid sequence in the cytoplasmic tail of the enzyme.',NULL,1231,1,2),(###-02-14&&

1143    Q: In problem 3-17, what is the difference between radiation and fluorescence

1143   A: It\'s like this: Fluorescence and radioactivity are two different ways of tagging molecules so you can \"see\" where they are. The detection methods for the two are different -- for fluorescence, you shine one color (wavelength) of light on the sample; the fluorescent material absorbs that light, and it emits light of a different color (which is what you detect -- you filter out the wavelength of the light used to excite the sample).  For radioactivity, you can\'t \"see\" directly where it is -- you have to use autoradiography or other techniques to detect it.\r\n\r\n<p>In both cases, you supply a tagged molecule and follow it \"like the mouse down the boa constrictor.\" Sometimes you supply small tagged molecules, like amino acids, and then follow the tagged macromolecules (here proteins) made from the small molecules. (You wash away any unused, tagged, small molecules.) In other cases, you provide (or get the cell to make) macromolecules that are tagged by having some extra stuff (radioactive or fluor.) added on to one end of the macromolecule. Then you follow them down the snake.',NULL,1232,1,2),(###-03-13&&

1144   Q: Is it really true that the number of nucleosomes stays the same during transcription (on the transcribed part of the DNA)

1144   A: Current thinking is that the nucleosomes are not removed but are loosened up by so-called \"histone remodeling\" proteins. This allows the polymerases to transcribe the DNA without kicking off the histones/nucleosomes completely. We are assuming that this is correct, and therefore the number of nucleosomes DOES remain the same in transcribed regions.',NULL,1233,2,2),(###-03-13&&

1145   Q: What happens to lamins in the different stages of the cell cycle

1145   A: Lamins depolymerize during G2 to M (because of extensive phosphorylation) and repolymerize during M to G1 (when phosphates are removed). Histones tighten (so DNA shortens) during G2 to M and loosen during the transition from M to G1.',NULL,1234,2,2),(###-03-13&&

1146   Q: In problem 4-4 (C), how can we be sure that the site to which probe #1 hybridizes is not a distal regulatory site

1146   A: First of all, the sad truth is you can never be 100% sure of anything in biology. You have to settle for what is MOST LIKELY. I suspect this makes bio and this course a lot harder, but that\'s how it is. Second of all, the DNA that hybridized to probe #1 could be a distal control element (given the distances involved), but the data on sensitivity to nuclease indicate that it isn\'t. Since this upstream region is NOT sensitive to nuclease, you know it is tightly wound. That\'s how you know it\'s not a distal control element. What I meant by the  \"too far\" argument is that it is too far away to be directly affected by TF\'s bound to the proximal sites.\r\n',NULL,1235,2,2),(###-01-27&&

1147   Q: In problem 1-11, in the drug A case, the cells are multinucleated and large apparently due to the drug\'s effect on actin and cytoplasmic division. How are the nuclear membranes formed and replicated

1147   A: The nuclear membrane doesn\'t grow bigger and pinch in half. As far as I know, there is no actin associated with or supporting the nuclear membrane (there is lamin supporting it.) During division, the nuclear envelope comes apart into membrane vesicles, and after the chromosomes have been divided, the vesicles reform into two smaller nuclear envelopes -- half of the vesicles associated into an envelope around each bunch of chromosomes.',NULL,1236,1,2),(###-03-13&&

1148　Q: In problem 4-16 (B-2), why would the more degraded segments be lighter

1148　A: Intensity of a band depends on the number of DNA molecules in the gel (at that particular spot) that bind the probe. It doesn\'t depend on the length of the DNA molecules in the gel or the length of the probe. The number of DNA molecules that end up a particular spot (band) depends on how many are any particular length. If you degrade the DNA and then run it on a gel, the fragments will not be in the original place on the gel, so you won\'t have the same number of DNA pieces to bind the probe and form a band (at the original position). You will get a lighter band in the original spot (if some undegraded DNA remains) or no band at all (if all the DNA is degraded. The smaller degraded fragments may run off the gel or spread out and form a smear when they bind probe.',NULL,1237,2,2),(###-03-13&&

1149　Q: The answer to this problem RP 5-2 says Estrogen OR Cortisol depending  on where the HRE is, assuming that the HRE for both hormones is at the same type of control region (either distal or proximal). What if the cortisol RE for the stress gene (Q) is in the proximal region and estrogen RE for the other gene is in the distal region? Wouldn\'t adding two hormones to the hybrid cell at the same time increase the stress gene production even further?  &&

1149　A: You are right that the answer assumes that all HRE\'s are in the same place. As it happens, I think most or all known ones are proximal. So it seems a reasonable assumption to me, but you are correct that the situation could be more complex. The way you would find out if the two HRE\'s are (or are not) in the same relative position would be by doing the experiment.',NULL,1238,2,2),(###-03-06&&

1150　Q: What is the difference between a secretory vesicle and a default vesicle

1150　A: Secretory vesicles are part of \"regulated secretion\" and default vesicles are part of \"constitutive secretion.\" No special signal required for const. secretion -- release is automatic. The signal is \r\nnot a sure thing -- it may never come or wait a week. Released now and next week are not equivalent.',NULL,1494,2,2),(###-01-27&&

1151　Q: What happens to the accumulated Cl in RBCs

1151　A: It goes back out when the cells reach the lungs. (At lungs, it\'s Cl- out and bicarb. in -- the reverse of what happens in the tissues.)',NULL,1241,1,2),(###-01-28&&

1152　Q: In problem 1-13, if water cannot pass through connections connecting two cells, then why doesn\'t increase of connexons decrease the likelihood of water passing through the extracellular space

1152　A: No. I think you misunderstand what is meant by the question. Suppose you have a layer of cells, all connected by gap junctions. The question asks about water moving from one side of the layer to the other. (Not from one cell to the next.) Water does not pass through gap junctions to get from one side of the layer to the other -- the water moves (if at all)  through the spaces between the cells. (It won\'t move through the spaces if the cells are connected by tight junctions.) The presence of gap junctions does not prevent water from moving across the layer.',NULL,1242,1,2),(###-01-27&&

1153　Q: Is the reason that transmembrane proteins play a role in support because all the transmembrane proteins lie on a cytoplamic support protein

1153  A: I meant that some transmembrane proteins are primarily important because of their supporting functions. For example the cadherins of desmosomes and adherens junctions that link the cytoskeleton of one cell to the cytoskeleton of another. Other transmembrane proteins are involved in other functions (and many have multiple functions).',NULL,1243,1,2),(###-01-23&&

1154  Q: Where do proteins made on the rough ER end up after the enter the lumen

1154  A: It depends. Sometimes to the Golgi, sometimes outside the cell.',NULL,1245,1,2),(###-01-23&&

1155  Q: Where do proteins manufactured in the rough ER ultimately end up

1155  A: Only the endomembrane system (remember that proteins destined for secretion are manufactured by free ribosomes in the cytoplasm). This usually involves going to the Golgi for modification, then sometimes back to the ER, sometimes to other places in the cell. ',NULL,1246,1,2),(###-01-23&&

1156  Q: Where are proteins manufactured in the cytoplasm meant to go

1156  A: The cytoplasm, nucleus, mitochondria, and peroxosomes.',NULL,1247,1,2),(###-01-22&&

1157  Q: In immunofluorescence, how do you wash off unattached antibodies while retaining those that are bound to antigens

1157  A: You literally wash the cells (or protein sample or whatever) -- you add a large volume of solution to the outside of the cells (or to the sample). Any unattached antibody diffuses out of the cells or washes off of the sample. (If you are using whole, live cells, they must have been\r\npermeabilized -- treated so molecules as large as antibodies can cross the membrane.)',NULL,1248,1,2),(###-01-22&&

1158  Q: How does immunofluorescence work

1158  A: You add the antibody, give it a chance to \"stick\" to its antigen, and then wash out \"loose\" antibody. So the only antibody left is the stuff that found its antigen.',NULL,1249,1,2),(###-01-22&&

1159  Q: In lab we used ethiduim bromide to label DNA that we were runing on a gel. The EtBr is structured so that it fluoresces more when its inserted among the base pairs because of excited elecrons (or something like that).\r\nAre the tags that are used on the antibodies similar in that they will only fluoresce when the antibody is bound

1159  A: No.  With antibodies the trick is washing out the unattached antibodies.',NULL,1250,1,2),(###-01-20&&

1160  Q: Which part of Becker or Purves explains on the topic of \"in situ\" labeling

1160  A: See \"Cytochemical localization of enzyme activity\" in Becker, Ch. 12, pp 353-54, and fig. 12-20.',NULL,1251,1,2),(###-02-01&&

1161   Q: In problem 1-2 (B), if you can control whether erythrocytes seal \"in/out\" or \"out/out\" by controlling the ionic strength of the solution, why would a person infer a 50/50 chance of \"out/out\" or \"in/out\" since nothing is said about the ionic strength

1161   A: The results given only make sense if (under the conditions used) you get 1/2 the ghosts resealed each way. I assume you can control it, but in this expt. they picked conditions that do not favor one way or the other. If conditions had favored one way of resealing over the other, then some proteins would be less accessible and some more.',NULL,1253,1,2),(###-02-05&&

1162   Q: Where does all the energy come from for ALL sorts of movement in the cell, as movement of organelles, etc

1162   A: It all comes ultimately from ATP which comes from.... Moving vesicles along microtubles etc. uses ATP. Moving within organelles probably is driven by concentration gradients.',NULL,1255,1,2),(###-02-06&&

1163   Q: In problem 2-4 (B), isn\'t serine a charged molecule at cellular pH, meaning that the electropotential gradient will come into play here and that ser-in is not necessary equal to ser-out

1163   A: Serine is a \"Zwitterion\" -- it is neutral over all, even though the carboxyl and amino are charged. (The R group is not.) The charges on the amino and carboxyl groups cancel each other out.',NULL,1256,1,2),(###-02-07&&

1164   Q: In problem 2-9 (C), how are we to know that the \"specific membrane protein\" required for uptake are the receptors as opposed to clathrin or some other abnormal coat protein

1164   A: The question says that all other substances are taken up normally. If clathrin were abnormal, all substances taken up by RME would be affected. If only some substances taken up by RME are affected, the receptor for those substances must be the altered protein.',NULL,1257,1,2),(###-02-11&&

1165   Q: What is the difference between continuous labeling and pulse-chase labeling? Why use one over the other

1165   A: With continuous labeling, the whole pathway of the labeled material eventually tagged, whereas with pulse-chase experiments, only the exact location of the material is lit up at a particular time. It\'s easier to follow what is happening with pulse-chase, but it\'s technically more difficult to do it that way.',NULL,1258,1,2),(###-02-17&&

1166   Q: Problem 3-16 (C) says that drug C inhibits acid phosphatase from separating from its receptors. If the ligands and receptors don\'t separate, how can you tell that the AP will be recycled back to the Golgi instead of going to the lysosomes with the AP where the receptors would be degraded and the APs used as normal

1166   A: The receptors are transmembrane proteins that stick out into the cytoplasm. Therefore it seems reasonable to me, that they will direct or determine where the vesicle goes, and not what\'s inside. However, I don\'t have any data to prove that I am right, and it is possible that the vesicles containing both receptors and ligands will go to lysosomes, where the ligands belong, and not vice versa.',NULL,1259,1,2),(###-02-16&&

1167    Q: When you have secondary transport of something into the cell, what keeps what's doing the primary active transport from re-\r\nestablishing normal cellular graidents in experiments? (For example, in cases where intracellular pH = extracellular pH, why wouldn't Na+ be pumped out via the Na+/K+ pump, then exchanged for H+ repeatedly for overall transport of peptides inside? Just a matter of competition between two things that use H+?)&&

1167    A: Under normal circumstances, what you say is right -- if the pH gets off, the Na+ is exchanged for H+ to straighten out the pH. (Usually Na+ drives H+, but if the balance gets sufficiently out of wack, the H+ can drive Na+.) However in a lab situation you can alter the pH or Na+ by other means that overwhelm the effects of the usual transporters.\r\nThere are set ups with automatic feed back for maintaining a constant pH inside or outside the cell. Alternatively, you can measure what happens right after you artifically change \r\nthe pH -- before the system can self correct.',NULL,1485,1,2),(###-02-17&&

1168    Q: What do the enzymes do that are added in the salvage pathway to treat Gaucher's diesease? Do they attach M6P to hydrolase or do they cause M6P and hydrolase to bind to the receptor

1168    A: The added enzymes have M6P already. (I disease is the only one where M6P\r\ncan't be added.) The M6P allows the added hydrolases to bind to receptors and to be internalized by RME and sent to lysosomes. In Gaucher's disease, the lysosomes are missing a particular hydrolase (just one, not all) and the added enzyme is the missing one.',NULL,1260,1,2),(###-02-17&&

1169    Q: Is simple diffusion specific to certain molecules

1169    A: No.',NULL,1261,1,2),(###-02-17&&

1170    Q: If you have different types of molecules that are able to diffuse across a membrane, each with the same charge and density, will they act together to equalize <i>total</i> solute concentration inside and outside the cell, or separately equalize their respective concentrations?\r\n&&

1170    A: Each molecule acts on its own to equalize it's own concentration in and out.',NULL,1263,1,2),(###-02-17&&

1171    Q: In problem 2-9 (A), is the carrier protein found only at the basolateral surface and not the apical surface

1171    A: A protein can be on just one surface or the other. (Just because the carrier is on the BL surface doesn't mean it has to be on the apical) In this case, there is some evidence for a carrier for G on the apical surface, but we are ignoring it, because it is thought most entry of G is by secondary act. transport with Na+.',NULL,1265,1,2),(###-02-17&&

1172    Q: In problem 2-9 (A), shouldn't the new high concentration of glucose in the intestine render a secondary Na+ gradient unneccesary for transport

1172    A: Yes, IF there is passive carrier for G on the apical surface.',NULL,1266,1,2),(###-02-17&&

1173   Q: When you refer to \"right side out\" and \"inside out\" vesicles made from the plasma membrane, will you always be referring to vesicles made NOT in the usual lumen-side in, cyto side out way but so that the outside of the vessicle corresponds to the outside of the plasma membrane

1173   A: Yes. The problems try to make clear when vesicles are forming by RME etc. and when the experimenter is just breaking up membranes into fragments and reforming them.',NULL,1267,1,2),(###-02-18&&

1174   Q: Are all proteins that are completely released into the lumen of the ER bound to be secreted from the cell

1174   A: They don\'t have to be secreted. Some proteins have signals that direct it to stay in the ER or go to the Golgi.',NULL,1269,1,2),(###-02-18&&

1175   Q: Are all parts of the extracellular domains of multipass proteins automatically glycosylated or do they only have the potential to be

1175   A: Most of them are. I don\'t know the actual percent.',NULL,1270,1,2),(###-02-18&&

1176   Q: What is the role of nucleoside diphosphatase as it is specific to the Golgi

1176   A: No one knows.',NULL,1271,1,2),(###-02-18&&

1177   Q: In problem 3-12, I thought a reaction in a peroxisome would make B MORE soluble.&&

1177   A: The normal function of peroxisomes is to make things more soluble, but this is an artificial substrate used for identification. The substrate\r\nwas chosen because it\'s product is LESS soluble.',NULL,1272,1,2),(###-02-18&&

1178   Q: In problem 3-15, should we assume that the signal peptide is not removed

1178   A: No. Usually (but not always) the signal peptide on the amino end is removed and a different stop transfer sequence anchors the protein in the membrane.',NULL,1273,1,2),(###-02-18&&

1179   Q: How do amino acids enter the cell in the first place

1179   A: Amino acids are small, like glucose. They use carrier mediated transport, sometime passive, and sometimes active (often Na+ cotransport.)',NULL,1274,1,2),(###-02-19&&

1180   Q: Are peroxisomes part of the EMS

1180   A: The peroxisomes are not part of the EMS. However they are vesicles -- a small blob surrounded by a membrane -- none\r\nthe less -- just a different kind.',NULL,1275,1,2),(###-02-19&&

1181   Q: Is there a difference in being attached to the outer nuclear membrane or the ER

1181   A: Being attached to the outer nuclear membrane is no different than being attached to the ER. The proteins end up in the same place.',NULL,1276,1,2),(###-03-04&&

1182    Q: Why is there a G-protein instead of just having the receptor directly interface with the AC/PLC?  Is the only reason that it can divide and act as two signals (basically multiplying the signal)? Why doesn\'t the G-protein make cAMP or IP3/DAG itself after it divides instead of signaling something else to do it

1182    A: All these questions are really \"why is it the way it is?\" We can make rationalizations after the fact, and some are better than others, but the best answer is \"that\'s the way it is.\" Probably some of the features have to do with the way the system evolved (in steps, from simpler things) and some have to do with selection for superior function -- it is the way it is because it works (better than the alternatives previously available). When that is the case, we can sometimes guess why the current system pays. But sometimes we can\'t tell.\r\n \r\n<p>In both the cases you asked above, the answer is probably a mix of both -- it\'s that way because of evolutionary origin and it\'s still around because it works well (if in a somewhat indirect, Rube Goldberg style).',NULL,1278,2,2),(###-02-29&&

1183    Q: Is the chain of nucloesomes the same thing as \"beads on a string,\" meaning no other folding or coiling except for the 200 BP DNA around every histone

1183    A: Usually when people say \"chain of nucleosomes\" they do indeed mean the \"bead\" stage. However, in context, they may mean it\'s more folded,\r\nalthough that should be clear from the other info.',NULL,1281,2,2),(###-02-27&&

1184    Q: Why is secondary transport inhibited in the long run if primary active transport is inhibited?\r\n\r\n&&

1184    A: If active transport stops (perhaps due to depletion of ATP), secondary transport must stop as well, since the ion gradient that powers it will run down and cannot be replenished.',NULL,1282,1,2),(###-02-26&&

1185    Q: Why do we need two separate enzymes for catalysis of the same reaction (say phosphorlyation)

1185    A: It isn\'t the same reaction. One is phosphorolysis and the other is\r\nsynthesis (without obvious phosphate -- it uses another source of energy).\r\nThe same enzyme can be used for both directions of any reaction, but in\r\nreal cases there is often one enzyme to get from A to B and another to get\r\nfrom B to A. But the two reactions are different -- one may use ATP, for\r\nexample. (If you write out all the reactants and products the two reactions are not the same. See problem 4-5 of first term problem book for an example.) It\'s like making recordings and playing them. You could\r\nsay it\'s the same reaction (music <---> tape) running in two different directions, but it isn\'t. Different equipment/enzymes are required for the two directions.',NULL,1283,2,2),(###-02-26&&

1186    Q: Why do we say that kinase adds phosphate?  Doesn\'t it also remove phosphate? Is it a matter of what conditions under which the enzymes are working

1186    A: It can catalyze the reaction in either direction. However the delta G is\r\nvery negative in the direction of adding P, so given the usual conditions -- concentration of substrate and product -- the reaction almost always goes in one of the two possible directions. Also product is usually\r\nremoved.\r\n\r\n<p>It <i>is</i> a matter of what conditions the enzumes are in. Some enzymes are used in both directions -- the direction depends on\r\nthe supply of reactants and/or products. This is usually the case for reactions without large delta G\'s.',NULL,1284,2,2),(###-02-26&&

1187   Q: About an article in <i>Nature</i> this week. It said that they found a gene that codes for a protein that prevents human cells from contracting HIV in vitro that comes from some monkey that can\'t contract HIV. Does this mean we have a potential cure for AIDS?  Is there any way to give an adult human being a new gene

1187   A: Gene therapy for humans is theoretically possible and practically (for now) almost impossible to use. It is more likely that someone will try to figure out what the protein does and how it does it, and then try to find something injectible or swallowable that has the same effect. In the Times (I\r\ndidn\'t read the Nature article either) it says the protein works in human cells, but that\'s in vitro, in Petri dishes, not in a person. It\'s one thing to get the gene/protein into cells in a bottle and some thing else to get it into the right cells in people.',NULL,1285,2,2),(###-02-24&&

1188   Q: What is the meaning of RNA dependent DNA polymerase

1188   A: It means reverse transcriptase -- an enzyme that makes DNA using an RNA template.',NULL,1286,2,2),(###-03-16&&

1189   Q: Are there any theories as to why muscle can\'t break down the glycogen that they have right there into glucose, but instead have to go\r\nthrough the liver first

1189   A: The muscle does use its own energy sources. It gets the energy from breaking the\r\nglycogen down to lactate (or CO2 if there\'s enough oxygen). The glycogen in muscle is for running the muscle, not buffering the glucose in blood. So the muscle doesn\'t really go through the liver first -- the muscle\r\nsoaks up glucose in the absorptive phase, making glycogen, and uses that glycogen during the post absorptive. What\'s \"left over\" from breaking down the glycogen, namely the lactate, goes to the liver.',NULL,1288,2,2),(###-03-16&&

1190   Q: In the positive feedback circuit for lactation, are the\r\neffectors respectively the smooth muscle around the alveolus, which constricts in response to oxytocin, and the anterior and posterior\r\npituitary, which secrete prolactin and oxytocin? Or would you count the anterior pituitary and posterior pituitary as effectors here

1190   A: Usually the Ant. Pit. and Post. Pit. are not considered effectors, as they simply relay the message to the tissues (smooth muscle etc.) that actually get something done. The PP and AP help send the word around the arrows to the effectors. In this case there is a sort of relay represented by the arrows, not one straight race stretch.',NULL,1289,2,2),(###-03-16&&

1191   Q: Exactly what is the determing cause of Goiter

1191   A: If the thyroid is over stimulated, too much TSH or equivalent, you get goiter. This can happen for more than one reason -- lack of neg. feedback by TH or too much stimulation of thyroid.',NULL,1290,2,2),(###-03-16&&

1192   Q: When does glycogen breakdown come into play if the postabsorptive phase involves the synthesis of glucose from smaller stuff (gluconeogenesis). Does gluconeogenesis occur only when there\'s no more glycogen

1192    A: In the postabsorptive state, glycogen is broken down in liver and glucose is released into the blood; after a few hrs the glycogen is exhausted and gluconeogenesis etc. take over. Glycogen is also broken down in muscle,\r\nbut doesn\'t produce free glucose -- it produces lactate. The liver uses the lactate & other stuff for gluconeogenesis.  You are correct that the liver glycogen is broken down first, and then when the glycogen runs out other processes take over.',NULL,1291,2,2),(###-03-11&&

1193    Q: Are the terms \"trophic\" and \"tropic\" interchangable

1193    A: The word generally used these days for hormones is \"tropic\" -- same root\r\nas tropic for going towards (as in phototropisms of plants). Trophic comes\r\nfor the root for nourishment as in eutrophic etc. Both used to be used interchangeably,  but nowdays scientists prefer tropic for\r\nhormones (and trophic for energy levels in ecosystems).',NULL,1292,2,2),(###-03-11&&

1194    Q: What changes physiologically in the see-saw balancing act or circuitry of temperature regulation that\r\nmakes the elderly more sensitive to cold?  (Or is it a decrease in subcutaneous adipose tissue, etc.)

1194    A: As to the cold sensivity of the aged, it is partically due to less adipose tissue, but also to less response by the autonomic nervous system overall-- the system declines in responsiveness with age.\r\nSee <a href = \"http://www.eurekalert.org/pub_releases/2001-01/PS-Amwe-2501101.php\">http://www.eurekalert.org/pub_releases/2001-01/PS-Amwe-2501101.php</a>',NULL,1293,2,2),(###-03-09&&

1195    Q: I\'m still not sure I understand why these scientists were surprised that it (Cp) was regulated at the transcriptional level and not at the translational level, since\r\nmost eukaryotic regulation is at the transcriptional level in any case.&&

1195    A: The scientists were assuming that the mRNAs for the proteins of iron metabolism were regulated at translation. They assumed there was something special about the regulation of synthesis of the enzymes of iron\r\nmetabolism. (Other wise you are right, that regulation at transcription is the usual case.)',NULL,1294,2,2),(###-03-09&&

1196    Q: The answer to problem 4-16 (B-3) says that the band should be lighter for NONE of the choices even though the liver cells exposed to Drug D make protein X. Since they\'re transcribing the gene, why isn\'t the DNA more degraded by DNase since the chromatin is clearly looser

1196    A: The cells in experiment #3 and 4(d) both make protein X; the chromatin in both should be in the same state.',NULL,1295,2,2),(###-03-25&&

1197    Q: What are the  water soluble hormones that don\'t use G-protein linked\r\nreceptors

1197    A: There\'s GH, Prolactin and insulin. They all use TK or TK linked receptors.',NULL,1296,2,2),(###-03-25&&

1198    Q: Is a neurotransmitter the signal that prompts posterior pituitary to secrete either oxytocin or ADH

1198    A: HT gets more than one kind of input. Neurotransmitter would be one. Signal would come to HT end of neuron.',NULL,1298,2,2),(###-03-24&&

1199    Q: In the case of the intracellular receptor of cortisol, after the inhibitory protein detaches from the receptor there is no\r\ndimerization, so is the receptor-cortisol complex is taken to the nucleus directly

1199    A: I think when I explained this I was trying to emphasize other things and didn\'t mention dimerization. The cortisol receptor binds cortisol, translocates to the nucleus, and then dimerizes in the nucleus; the dimer binds to DNA.',NULL,1299,2,2),(###-03-24&&

1200    Q: Can you get a smear when doing electrophoresis if you have not removed the histones (in other words, if the nucleosomes are still present and the chromatin is losely wound)

1200    A: Yes.',NULL,1300,2,2),(###-03-24&&

1201    Q: In answer to problem 7-16, why is TSH not considered a neuroendocrine hormone

1201    A: It isn\'t secreted by neural tissue. It is an endocrine but not a neuroendocrine.',NULL,1301,2,2),(###-03-24&&

1202    Q: Why doesn\'t the liver increase blood AA levels in response to glucagon, since it has the necessary enzymes to degrade proteins

1202    A: Most blood AAs come from muscle.',NULL,1302,2,2),(###-03-24&&

1203    Q: Why doesn\'t epinephrine have an effect on blood AA levels if muscles have receptors for it

1203    A: I assume epi doesn\'t affect protein breakdown significantly. That makes sense to me as protein breakdown is only needed in the longer term.',NULL,1303,2,2),(###-03-24&&

1204    Q: If in Grave\'s disease the antibodies act as antagonists, blocking receptors for the TSH, how does one get goiter when the thyroid isn\'t being stimulated at all

1204    A: In Grave\'s disease the Ab act as <b>agonists</b>. You don\'t get low TH -- you get too much TH.',NULL,1304,2,2),(###-03-24&&

1205    Q: Thyroid tumor leads to high T3 and T4 even though there are very low levels of TSH and TRH. Why doesn\'t one get goiter here

1205    A: There\'s no stimulation of other nontumorous cells in the thyroid. ',NULL,1305,2,2),(###-03-23&&

1206    Q: In problem 7-4 (1), why doesn\'t follicle cells of the thyroid count as a duct

1206    A: There is no tube connecting the follicles to anywhere. The TH diffuses out of the follicle into the blood stream.',NULL,1306,2,2),(###-03-23&&

1207    Q: Are there any structures that release into blood via a duct? Or they all via some capillary bed arrangement a la hypothalamus

1207    A: I don\'t think there are any ducts to the blood. The ducts go to a lumen (internal space) or outside.',NULL,1307,2,2),(###-03-23&&

1208    Q: I assume ducts are involved in sweat, tears, saliva, digestive acids, but what about blood bound secretions

1208    A: Blood bound secretions are not from ducts, as far as I know. (Since everything else has exceptions there may be one here, but I don\'t know of one.) The cells secrete materials by synthesizing lipid soluble stuff that diffuses into the blood or by exoocytosis of water soluble stuff.',NULL,1308,2,2),(###-03-22&&

1209    Q: It doesn\'t make sense that a loss-of-function mutation will be dominant in an TK receptor gene. If 50% of the TK proteins are defective, 50% will still be able to dimerize, triggering some effect.&&

1209    A: If one allele is defective, and both alleles are equally expressed, there will be 50% defective subunits. If dimerization occurs, only 25% of the molecules will have two\r\ngood subunits and work. In the usual terms\r\nof inheritance we use, the correct term is partially dominant. But because of the way these tests are done (by transfection -- I\'ll be happy to supply details if you like) the mutant alleles are often\r\noverexpressed, so dominance is complete. Also complete and incomplete dominance are often not distinguished -- the surprising thing here is that the loss of funct. mutation has any effect at all.',NULL,1309,2,2),(###-03-22&&

1210    Q: What regulates the types of regulatory\r\ntranscription factors that the cell produces?\r\nIs this a case of genomic control (i.e. tightness of chromatin) making certain parts of the DNA strand more/less available

1210    A: This is the $64 question. (or the $64,000 question in a different era). Exactly how you set up a stable situation with some genes for TF\'s on and some off is not understood. (A few specific cases are known, but the overall general principle seems to elude us.) If tightness of DNA controls expression, what governs the production of the proteins responsible for folding?  The short answer is that certain TF\'s stimulate their own production, so once you make them, the\r\nsystem maintains itself. The process of development involves a series of\r\nTF\'s turning on others that turn on others until you reach the stable state. We\'ll try to address some of this at the end of the term.',NULL,1310,2,2),(###-03-21&&

1211    Q: Can you clarify the terms \"isolate,\" \"extract,\" and \"purify\" as used in problem set 4?\r\n&&

1211    A: This is an interesting question. I never thought about why I use one term or the other. I think the words come close to being synonyms but are not exactly the same. Which one you use is partially just linguistic preference but partially has to do with the procedure and substances involved.\r\n \r\n<p>Isolate means you fish out whatever it is you are talking about -- could be chromatin, nuclei, cell, or DNA. You would say you isolated nuclei, but you might say you isolated or extracted DNA.\r\n \r\n<p>Purified usually means you took (additional) steps to get the stuff you\r\nwant away from other things. For example suppose you isolated chromatin, treated it with something, and then wanted to examine just the DNA. You would separate the\r\nDNA and protein, and throw the protein away. You would say you had purified or isolated the DNA. Which term you would use would depend on\r\ncontext. You might also say you extracted the DNA (from the chromatin).\r\n \r\n<p>Extracted also means fished out from a mix, but I think it implies you are\r\ntalking about a particular substance, not a cell or cell fraction.',NULL,1311,2,2),(###-03-21&&

1212    Q: Are enhancers always in \"loosest\" state

1212    A: No. Only when they are being used.',NULL,1312,2,2),(###-03-20&&

1213    Q: If you inherit a malfunctioning Rb allele, wouldn\'t it be almost inevitable that you\'d have some form of cancer some time in your life considering the countless number of cells in your body and the fact that selection is for lack of growth control

1213    A: The answer is yes. That\'s why the disease  caused by an inherited malfunction in rb is\r\nconsidered dominant -- if you get one defective rb allele you almost always get cancer. Why you usually get a retinoblastoma, and not some other type of cancer, is not so clear.',NULL,1313,2,2),(###-03-19&&

1214    Q: Problem 4-5 (B-1) says that the number of different kinds of histones / nucleosomes is 5. However, H1 is in linker and not part of nucleosome, so doesn\'t that make it 4

1214    A: H1 is considered a part of the nucleosome. It is optional in the bead on a string stage, but required for tighter folding.',NULL,1314,2,2),(###-03-19&&

1215    Q: In problem 4-5 (B-2), why is the number of nucleosomes in M and G-1 = zero

1215    A: It isn\'t. The number of nucleosomes is large, but it is the same in M and G-1.',NULL,1315,2,2),(###-03-19&&

1216    Q: In problem 4-5, C-1, what is depolymerization of lamins and how does it change from G2 to M, or G1 to S

1216    A: Lamins are nuclear IT\'s that support the nuclear envelope. Lamins must depolymerize for nuclear envelope to dissasemble for mitosis and must repolymerize for nuclear\r\nenvelope to reassemble. See notes or text for more details. (Laminins are part of the ECM.)',NULL,1316,2,2),(###-03-19&&

1217    Q: In problem 15-8 (C-3), is there anything in the information provided that\r\nrules out the possibility that phosphorylation allows the regulatory protein to increase initiation of translation? It is unclear to me why both answers aren\'t equally valid possibilities. &&

1217    A: If you look again at the answer, you will see that either of these solutions is a reasonable solution, and neither\r\nsolution is untenable. However the information in the earlier parts says there is already some regulation of stability of the mRNA. So (by Occam\'s razor) it seems more reasonable to me to think that\r\nthe protein described in C affects the step already known to be controlled\r\n(stability of the mRNA). It could be a 2nd regulatory protein controling a\r\ndifferent step, but that seems less likely. You have to assume that two steps, not one, are controlled, and there are 2 regulatory proteins.',NULL,1317,2,2),(###-03-18&&

1218    Q: What is the difference between metabolic rate and basal metabolic rate? Also, if your basal metabolic rate is higher than normal how would that affect the point at which you would sweat or shiver based on the outside temp.&&

1218    A: The BMR is the rate when you are at rest. The MR is the rate at any particular time -- it can be higher (if you are moving) than the BMR or lower (if you are asleep). The BMR is the base rate. If your BMR is higher than normal, you use more energy and generate more heat. So you will not need to start shivering\r\nunless it gets colder than normal. In other words, you will tend to overheat, which is fine if it is cold outside, but will cause you to sweat at what other people consider normal temperature.',NULL,1318,2,2),(###-03-18&&

1219    Q: I know messages from the hypothalamus travel to the posterior pituitary via nerves, but what is it that travels to posterior pituitary? Electrical Nerve impulses or hormones that originate in HT

1219    A: The actual hormones are made in the cell bodies of the neurons, which are\r\nin the HT. The hormones are released from the ends of the axons of the same neurons -- those axon endings are in the post pit.',NULL,1319,2,2),(###-04-02&&

1220    Q: Do you have a higher number of nucleosomes if a gene is being transcribed, or does the replacement of histones not consitute a loss in the nucelosome

1220    A: If it were a nontranscribed section, without nucleosomes, you wouldn\'t see the necklace structure. Addition/loss of H1 doesn\'t change the number of nucleosomes. Change of\r\nothers does. But I don\'t think you can argue that you would expect to see a change in this case. There were nucleosomes in interphase, and they should all be retained (and not added to) in mitosis.',NULL,1323,2,2),(###-04-11&&

1221    Q: Problem 8-3 states that since potassium is positive, more potassium inside the cell would increase the resting membrane potential. However, if we look at the problem quantitively through the GHK equation, the resting membrane potential would decrease since the variable for potassium inside the cell is in the dominator. Who\'s right?\r\n\r\n&&

1221    A: I think you misread the answer in the back of the book. (Or maybe it wasn\'t clear enough.) More K+ will end up inside, and that will make the resting potential LESS negative. In\r\nother words, K+ inside will increase, but rest. potential will decrease. This is exactly what the GHK says.',NULL,1324,3,2),(###-04-11&&

1222    Q: In the kidney, does aldosterone force Na+ out of the distal convoluted tubule in a nephron, causing reabsorption into circulation followed by H2O, thereby increasing blood pressure

1222    A: Aldosterone stimulates synthesis of the pumps and channels for removing Na+ from the filtrate (reabsorption), and water follows.',NULL,1325,3,2),(###-04-11&&

1223    Q: Does ADH case vasodilation in nephron causing water reapsorption also leading to increased blood pressure

1223    A: As far as I know, no. ADH works in the nephron to insert water channels in the plasma membranes of the collecting ducts (so more water is reabsorbed). It does cause vasocontriction (not dilation) of arterioles,\r\nwhich also raises blood pressure.',NULL,1326,3,2),(###-04-12&&

1224    Q: I\'m confused about how to view problem 8-3 quantitatively, which states that a less negative Vm equates to a decreasing resting potential. &&

1224　A: This is an issue that is perenially confusing! The problem is caused by the terminology. Resting potential is the size of the difference in voltage between the two sides of the membrane, and is negative in\r\nvalue. When the difference is smaller, the resting potential is smaller,\r\nwhich means it is still a negative value, but less negative -- smaller in\r\nabsolute value. For example, if the RMP goes from -70 mV to -50 mV it is\r\nsaid to be smaller and the cell less polarized = more depolarized = closer to threshhold.',NULL,1327,3,2),(###-04-17&&

1225　Q: What is meant by actin accesibility

1225　A: Actin and Myosin bind to each other only at special points on the actin (& matching points on the myosin). If those spots on the actin are covered up, the actin is \"inaccessible.\"',NULL,1328,3,2),(###-04-18&&

1226　Q: Is a calcium pump activated by a kinase, which in turn was activated by a cAMP? Is cAMP production what makes the removal of Ca++, and hence muscle relaxation, possible

1226　A: It depends on the tissue. Removal of Ca++ always requires ATP and pumps\r\nof some kind. Whether that pump is activated by cAMP depends on the type\r\nof cell.',NULL,1329,3,2),(###-04-26&&

1227　Q: The answer to problem 8-15 (4) is a \"receptor potential,\" potential in a \"separate receptor cell,\" but the cell in question is an olfactory receptor, thus\r\na \"modified neuron.\" So shouldn\'t the answer be \"generator potential?\"&&

1227　A: The term \"receptor potential\" is used in a general sense to mean both receptor and generator potential -- especially when you don\'t want to specify which case you are dealing with, or you want to keep the\r\ndiscussion general. Some texts don\'t even use the term generator potential, and just stick to receptor potential. That\'s the way the\r\nterm is used here. In the next edition of the book, I\'ll make it clear that what is here called a \"receptor potential\" is also a\r\ngenerator potential. (That the receptor is a modified neuron isn\'t always\r\nthe distinguishing feature, although it usually is. What\'s critical is if the receptor cell can fire an AP or not. The photoreceptor cells are modified neurons, but don\'t generate action potentials.)',NULL,1330,3,2),(###-04-26&&

1228　Q: In problem 12-12, how can you lose salt without also losing too much water

1228　A: You do lose both, that\'s why the blood pressure is too low.',NULL,1331,3,2),(###-04-27&&

1229　Q: In problem 8-18,(C), what allows us to conclude this isn\'t a graded potential, where the size of the stimulus is proportional to\r\nthe extent of depolarization? Why can we conclude that there are Ca$^{2+}$ channels all over the cells, since one graph shows repolarization only while the other shows a more normal curve

1229　A: The curves on the post. end look like graded potentials. The higher one on\r\nthe anterior end, caused by the larger stimulus, looks more or less like\r\nan AP. (The assumption here is that both smaller stimuli are the same size\r\nand both larger stimuli are the same size -- but the larger stimulus only\r\nproduces a spike on the ant. end.) The AP doesn\'t have all the usual\r\nfeatures, but it shows a spike when you go over a certain value. So it\r\nindicates a self regenerating stimulus, which requires voltage gated\r\nchannels all over so the spike can spread.  The fact that the spike does\r\nNOT

hyperpolarize on the way down is a good argument against voltage gated\r\nK+ channels. In this case, closing the Ca++ channels ends the AP, not\r\nopening K+ channels.',NULL,1332,3,2),(###-04-29&&

1230    Q: What prevents the heart from being in a permanently contracted state? Is it just the activity of the pacemaker cells that does it

1230    A: The AP lasts as long as the twitch (contraction). So you can\'t start a\r\nsecond twitch until the first one is completely over and the heart has\r\nrelaxed. (As long as the AP lasts, the cell is in a refractory period and\r\nyou can\'t fire a second AP and start a second twitch.) You can\'t build on\r\nthe first twitch and start a second one while the heart is already partly\r\ncontracted. No matter how fast the pace maker fires, you can\'t start one heart beat until the previous one is finished.',NULL,1333,3,2),(###-04-29&&

1231    Q: Is there a book you would recommend I read or a chapter in a textbook that I can use as a reference for immunology

1231    A: Purves isn\'t bad, but it is so condensed that it is hard to follow. Once you know what he is talking about, it all makes perfect\r\nsense. I haven\'t looked closely at Sherwood on this topic, but there is at\r\nleast one thing in her text that is misleading at best. It\'s a pretty\r\nsmall issue, so the text is probably worth reading. I think a look at any\r\nphysio book is worth it if you have one around. I suggest you try a\r\ndifferent intro bio book, like Campbell, and see if it makes more sense to\r\nyou. Most immuno books are too detailed, although they usually have intro\r\nchapters that are helpful.\r\nAnother thought -- try Kimball\'s online bio book. I haven\'t looked\r\nat all of the immuno section, but I have found some of it very\r\nhelpful. It\'s easy to use and it\'s free! It\'s at\r\n \r\nhttp://users.rcn.com/jkimball.ma.ultranet/BiologyPages/\r\n<br><br>\r\nI\'m sorry not to have a particular source ready, but I suspect\r\nthat different ones are useful to different people, and that the first one\r\nyou read never makes any sense anyway, because the topic is so complex.\r\n(That\'s why we do it last.) If all else fails, go through the notes, and\r\ncome to me with a list of questions and I\'ll see what I can do. (I also\r\nhave a big collect of intro books if you want to come by and see if any of\r\nthem make more sense to you than Purves.)',NULL,1334,3,2),(###-05-03&&

1232    Q: For exam purposes do we need to know the equations and how to calculate equilibrium and resting membrane potentials using the Nernst or GHK equations

1232    A: You will not be given any problems actually using the equations. I think understanding the equations helps you understand the issues\r\ninvolved, but any problems you have to solve will be solvable without them. There are problems in Becker if you want to test your understanding.',NULL,1337,3,2),(###-05-03&&

1233    Q: Since B cells can alternatively splice mu and delta to get M or D, if there\'s an activated B cell (memory cell) that has both epsilon and alpha that\'s already deleted mu, delta, gamma, can it alternatively splice to\r\nchurn out A? Or can it only churn out E like we learned in class

1233    A: As far as know, alt. splicing alows an M/D switch, but all the other switches require DNA rearrangements. This is related to the structure of the DNA -- the spacing between the segments and the positions of\r\npromotors, terminators, etc. is such that alt. splicing won\'t work for the\r\nother cases.',NULL,1338,3,2),(###-05-03&&

1234    Q: How does pre-synaptic modulation work, and how can the adjoining nerve cell communicate via neurotransmitter by its terminal bulb getting cozy with the receiving cell\'s axon if there are only voltage-gated ion channels in the axon

1234    A: You have picked up what was either a misstatement or a generalization that\r\nhas exceptions. Pre-synaptic modulation involves the standard chemical\r\nsignaling found at virtually all synapses. As I understand it, most of the\r\naxon has only\r\nvoltage gated ion channels, but there are ligand gated channels where ever\r\nanother axon synapses on the first axon. Most synapses end on the cell\r\nbody or on the dendrites, but there are some that end on a axon, as in\r\npre-synaptic modulation.',NULL,1339,3,2),(###-05-03&&

1235    Q: Which cytokines and interleukins are we responsible to know?  &&

1235    A: The important point is that\r\ncytokines are major players, and they often determine the next step in the\r\nimmune response. Which ones do what (except for the two mentioned) is\r\nsomething you\'ll learn later (or look up) on a need to know basis.',NULL,1340,3,2),(###-05-04&&

1236    Q: How do B cells work in fighting viral infections

1236    A: For viruses, the B cells don\'t do much in the primary response. By the time B cells make antibody, it\'s too late to help much. (It may help prevent virus spread from infected cells to other cells.)  It\'s the cytotoxic T\'s that kill the infected cells. The second time, the Ab made by memory B\'s attacks the virus before it can infect cells, and hopefully,\r\nthe immune response destroys the virus.',NULL,1342,3,2),(###-05-04&&

1237    Q: For problem 13-8 (C), could an alternative answer be that a primary response is generated, and effector B cells are made that lyse the bacteria

1237    A: This is a virus (intracellular) -- no bacteria. Viruses generally aren\'t\r\nlysed -- they are clumped by antibody and devoured by macrophages.',NULL,1343,3,2),(###-05-04&&

1238    Q: For problem 13-13 (B-2), since the proteins on the helper T cell and B cell are juxtacrine and both cells activate each other, shouldn\'t the protein on the helper T cell be a ligand and a receptor

1238    A: B and T cells can activate each other, but in this case we are looking at a later step in B cell development that T cells control. Also remember that there are multiple proteins on each cell surface, so although some\r\njuxtacrine signaling is clearly bidirectional, some juxtacrine interactions are unidirectional.',NULL,1344,3,2),(###-05-04&&

1239    Q: In Purves, there doesn\'t seem to be mention of helper T cells being involved in the cytolytic T-cell killing function. Are they involved

1239    A: Helper T cells are needed to secrete IL-2 and stimulate killing function of CD8 T-cells.',NULL,1345,3,2),(###-05-04&&

1240    Q: What is the order of events in the T helper/T cytotoxic cells in dealing with infected cell? Is it that helpter T cells have encountered Ag from APC\'s via MHC-II then go on to stimulate cytotoxic T cells that have independently encountered Ag presented on MHC-I of some infected cell

1240    A: You probably have it right. No one is exactly sure about how this works, especially how the helper T\'s find the appropriate cytoxic T\'s and help them out. That\'s the main reason we\r\ndidn\'t go over it.',NULL,1346,3,2),(###-05-04&&

1241    Q: In doing Recitation Problem #12, I see that one of the questions from 2000 states that the IgM and IgD are rearranged at\r\nmRNA splicing level and not in rearranged DNA. Being that it is 4 years later, is still the accepted view? Also, is this arrangement is restricted to IgM and IgD, but does not extend to IgG, IgA or IgE.&&

1241    A: It\'s still correct. (If there are significant changes due to new findings\r\nI usually alter the problems to match reality or add a note. But I do miss\r\nsometimes!) If you look at the actual arrangement of the gene segments on\r\nthe DNA you will see that the M and D segments are relatively close to\r\neach other and far from the others. So it makes sense that M vs D is in\r\na separate category.',NULL,1348,3,2),(###-05-05&&

1242    Q: In class switching, I know that going from IgM and IgD to the other classes involves DNA rearrangement, but what happens after that?\r\n&&

1242    A: A switch from, say, IgG to IgE involves alternate splicing of RNA/further DNA rearrangement. It requires removal of some DNA so the C region used is next to the V region.',NULL,1349,3,2),(###-05-05&&

1243    Q: Why don\'t TCRs bind antigen

1243    A: T cells are \"MHC restricted\" which means they don\'t bind to free antigen.\r\nThe antigen must be stuck to MHC first. ',NULL,1350,3,2),(###-05-06&&

1244    Q: We said that the Hb saturation curve shifts to the right (lower affinity) at high altitudes because of the need to dump off more $O_2$ in the tissues, but doesn\'t it need to shift to the left (higher affinity) so that it can pick up $O_2$? even at low atmospheric $pO_2$

1244    A: This turns out to be a sticky question. The llama, shifts to the left,\r\nloading up better. We pile up the DPG and shift to the right, unloading\r\nbetter. Balancing the two issues is the problem -- you have to have a high\r\nenough affinity to fill up the hemoglobin in the lungs and a low enough\r\naffinity to release the oxygen in the tissues.',NULL,1351,3,2),(###-05-06&&

1245    Q: Why don\'t T-cells undergo somatic mutation like B-cells? If T-cells also are involved in the secondary response, shouldn\'t they\r\nbecome more effective via somatic mutation just like B-cells in secondary response

1245    A: That\'s just the way it is!',NULL,1352,3,2),(###-05-06&&

1246    Q: In problem 8-18 (C), isn\'t there also need to be K+ and/or Cl- channels on the anterior end to allow for re-polarization and a return to resting potential after the action potential? Also, how do we know that the action potential spreads across the entire cell as opposed to just staying in the anterior end of the paramecium

1246    A: I really don\'t know if there are additional channels. With an AP, just closing the open Ca++ channels will restore the resting potential although it may take a while.\r\nThe shape of the curve on the right shows an AP if stimulus is larger. No point in having an AP if it doesn\'t spread. If all you want is a local signal, a graded potential will do the trick.',NULL,1353,3,2),(###-05-06&&

1247    Q: In problem 8-14 (E), what is the significance of vesicle transport being faster? When do we even see vessicle transport down an axon

1247    A: Many proteins are made in the cell body and transported down MT to the\r\nsynapse. The point of the question is that the rate of diffusion of large\r\nmolecules like proteins is so slow that the proteins would never make it\r\nto the end of the axon if there were only diffusion. We never explicitly\r\nsaid this, but we did discuss the fact that there are extensive \"railroad\r\ntracks\" of MT in the axons of neurons.',NULL,1354,3,2),(###-05-06&&

1248    Q: The answer to problem 8-15 (E-3) says\r\nwe will see a receptor potential along with an EPSP. Isn\'t a receptor potential the graded potential in a sensory receptor cell

1248    A: The term \"receptor potential\" is sometimes used in the general sense to include generator potentials.',NULL,1355,3,2),(###-05-06&&

1249    Q: In problem 12-13, why do we expect to see proline in the urine

1249    A: When you inject pro, it goes to the blood and is filtered into the urine. It should be reabsorbed, but it isn\'t. Therefore there\'s a problem with the urinary system. (No reabsorption = no transport across the\r\nepithelium lining the kidney tubule.) When you drink pro, it should be absorbed from the lumen, go to the blood and reach the urine. But it doesn\'t. So there\'s a problem with the digestive system. (No uptake = no\r\ntransport across the epithelium lining the gut.)',NULL,1356,3,2),(###-05-06&&

1250    Q: Do the terms generator and receptor potentials apply ONLY to afferent neurons and not generally to both afferent and efferent?\r\n&&

1250    A: Only afferent. Only for sensory neurons.',NULL,1357,3,2),(###-05-08&&

1251    Q: What is the mechanism for how TH and B cells activate each other

1251    A: In the primary response system,\r\nTH cells are activated by phagocytes via matching surface protiens and antigen, and IL-I. TH cells then produce IL-II (autocrine and paracrine signalling) and can match up with and activate B cells. Note that TH cells provide IL-2 (and/or other cytokines) not IL1.',NULL,1358,3,2),(###-05-08&&

1252    Q: Does a TH cell produce IL-I as well as IL-II

1252    A: APC\'s make IL-1 which activates T helpers to make their own IL-2.\r\nT\'s activate B\'s with various cytokines, including IL-2.',NULL,1359,3,2),(###-05-08&&

1253    Q: Why do you need to split ATP to maintain cross bridges

1253    A: You can\'t maintain a crossbridge as is -- you have to split ATP and keep\r\nforming and breaking and reforming the bridge. So ATP is needed to\r\nseparate the actin and myosin for another

round. You need ATP to\r\nkeep breaking and reforming the bridges, not to hold the ones you have\r\ntogether.',NULL,1360,3,2),(###-05-08&&

1254    Q: For the variable region on H chains,\r\nare there many genes for V (200-300), many genes for D (4-6), and many genes for J(10-20), all of which can be rearranged in  VDJ regions

1254    A: Yes. But people usually call them \"gene segments\" since none of them code\r\nfor a whole peptide.',NULL,1361,3,2),(###-05-08&&

1255    Q: Is the the whole H chain a single gene

1255    A: There is a section of the DNA that codes for the H chain. Whether it\r\nshould be called a gene is debateable.',NULL,1362,3,2),(###-05-08&&

1256    Q: What does inexact joining mean

1256    A: It means that extra bases may be inserted between the V, D, and J regions at the joints. These regions are always in the same order-- so you don\'t get things like JDV.',NULL,1363,3,2),(###-05-08&&

1257    Q: In the transcription of the variable region of an antibody, what do you mean by \"D goes to J and V goes to DJ\" in the H chain rearrngement

1257    A: That\'s the order in which you hook up the three segments. First you join a\r\nD to a J, removing the extra D\'s and J\'s. Then you join that DJ combo to a\r\nV and dump the other V\'s.',NULL,1364,3,2),(###-05-08&&

1258    Q: How are H and L chains put together? Why is lambda shorter than kappa

1258    A: Lambda (chain or gene) is not shorter than kappa. There are more\r\nalternatives for the kappa segments than the V segments.',NULL,1365,3,2),(###-05-08&&

1259    Q: What is the mechanism by which the cell membrane returns from hyperpolarization to resting potential

1259    A: When the volt. gated K+ channels open, the cell temporarily goes to the\r\nequil. value of the potential for K+.  In other words, a little too much\r\nK+ goes out (beyond the rest. pot. amount). So less K+ than usual leaks\r\nout of the leak channels/more leaks back in until the resting potential\r\nis restored. That\'s the point at which all the forces due to Na+ as well\r\nas K+ balance out.',NULL,1366,3,2),(###-05-08&&

1260    Q: On exam 3 of 2003, problem 2B states that transverse tubules have many voltate-gated Na+ channels. If this is skeletal muscle (is it?), wouldn\'t there also be mechanically gated channels from the DHP receptors, or are there not many of these

1260    A: The mechanically gated channels are part of the ER membrane, not part of\r\nthe T tubule membrane. The DHP receptors are part of the plasma membrane,\r\nbut not the channels they gate.',NULL,1367,3,2),(###-05-10&&

1261    Q: Are infected cells also considered APCs

1261   A: Infected cells are equivalent to APC\'s in many ways, and they \"present\r\nantigen\" in the common sense of the term, but they are NOT considered\r\nAPC\'s by immunologists. They are\r\nnot necessarily part of the immune system, and are more properly called\r\n\"target cells.\"',NULL,1368,3,2),(###-09-17&&

1262   Q: Can I take this course concurrently with General Chemistry

1262   A: If you had a strong high school course in Chemistry and rememeber it, then you could be all right.  We recommend college \r\nchemistry as a prerequisite because we use the language of chemistry in the course, discussing processes in termsof chemical \r\nstructures and reactions.  We assume you know concepts and terms such as molarity, molecular weights, pH, chemical equilibrium, \r\nstoichiometry, basic atomic structure, covalent bonds, salts, ions, logarithms, exponential notation. We do not assume organic \r\nchemistry and teach that at the level needed as we go along.  You could look at sample exam #1 on the course Web site to get a \r\nfeel for the level. You could consider the Barnard Intro Bio course as an alternative.  Feel free to come talk to Dr.Mowshowitz or \r\nme about it if you wish.',NULL,1369,1,1),(###-09-17&&

1263   Q: Are viruses or even prions considered to be living? Viruses in particular seem to meet all the criteria.  &&

1263   A: Viruses are a matter of debate, not very productive though. In my view they are not living if they can\'t do most of the 3 things on their own.  \r\nThere will be gray areas. You could decide viruses are alive and I could decide to disagree and then we could go on with the course and it \r\nreally wouldn\'t matter that much.  Prions are much less alive: they are not that complex and they do not really metabolize.  Their \"reproduction\" \r\nseems to me more like crystal growth than living cells.',NULL,1370,1,1),(###-09-17&&

1264   Q: I assume that the two ring conformations of glucose result from two different \"opportune\" conformations of the straight chain molecule. Is it as simple as that the aldehyde (anomeric?) end bending \"left\" or \"right\" from the straight chain conformation diagram when the opportunity arises

1264   A: \"Opportune\" throws me a little. It is by chance that with all that movement the two concerned atoms wll collide, not so eventually. Some of these collisions will result in a new bond being formed (cyclization).  Why is one ring is slightly favored over the other? I\'m not sure if it a greater likelihood of new bond formation or if one ring is less likely to break open again.',NULL,1371,1,1),(###-09-17&&

1265   Q: What parts of the \"Functional Groups\" handout should we memorize;  details, like amine structure

1265   A: Name, group, + details.  \"examples\" will be \"learned\"as we go along, but need not be memorized per se. Amines at pH 7 act as bases so \r\ntake up a proton to become a positively charged ions, as discussed in lecture 2.  The\"detail\" shows them in this charged state, which is \r\nthe usual state they are in in biological environments (which for the most part are maintained at pH 7).  This detail is exactly the fact that \r\nshould be memorized, so that when you see an amine you should know it is likely to be charged positively and be able make prediction \r\nabout its bahavior (e.g., attraction to a negatively charged ion, repulsion from a positively charged ion) ) .',NULL,1372,1,1),(###-09-15&&

1266    Q: If hydrophobic forces hold large fat globules together and hydrophobic forces hold smaller globules of fat inside spheres of \r\nphospholipids, why will fat be broken into small globules in the presence of phospholipids?  Why can\'t small globules \r\nsurrounded by phospholipids aggregate into a large globule? Is entropy driving the emulsification process

1266    A: I have done a bit or research on this question. If the micelles are small enough (the size will depend on the ratio of \r\nemulsifying agent to fat) then an entropic factor does come into play, making the dispersion enrgetically favorable. But the \r\nmore common situation arises from a mechanical dispersion into smaller globules, or you could think of it as a natural \r\nstatistical budding off of small globules of fat. In the absence of an emulsifying agent, these small globules rapidly re-\r\ncoalesce with the mother globule  and so dispersion  is minimal. In the presence of an emulsifying agent like lecithin, the \r\nsmall globules get coated with  lecithin.  These micelles now have a problem coallescing because all the fat will be coated \r\nwith lecithin molecules displaying their natively charged ends on the exteriors.  Their charged polar groups will repel each \r\nother, thus slowing down re-coalescence.  The re-coalscence is thermodynamically favored but hits a kinetic bottleneck. \r\nThius the emulsion is in an unstable themrodynamic state. During this time lipase can act to start the process of fat \r\ndigestion (breakdown to faty acids). Bile salts aid in this process and are not really phospholipids but like them have a \r\ncharged end and a hydrphobic end. Soaps and detergents act similarly, with a charged end (usually negative) and a long hydrocarbon region.',NULL,1373,1,1),(###-09-17&&

1267    Q: The answer to prob. 1-24C states that  the alternating polymer would have 0 net charge, but if a molecule contained an odd \r\nnumber of sugar residues, couldn\'t it be  +1 or -1

1267    A: The question is geared toward differentiating a whopping negative or positive charge, with each monomer contributing, but legally (and \r\nlogically) you are correct about the odd-even situation yielding moelcules with non-zero net charges. But your answer should not be to throw in the towel (\"unable to predict\"). Your argumnet leads to the conclusion that the polymer (assuming the polymerization ended arbitrarily) would \r\nbe constituted of a mixture of molecules carrying +1, -1 or 0 net charges.  ',NULL,1374,1,1),(###-09-20&&

1268    Q: In an amino acid, why is the amino group charged (NH3+) at pH7

1268    A: The H+ ions are picked up from the aqueous environment (water) as water is in equilibrium between HOH and H+  +  OH-. As a result, if you put an amine in water, the pH will rise. As to why such functional groups are bases or acids, that is beyond the chemistry we treat in this course.',NULL,1376,1,1),(###-09-17&&

1269    Q: In prob. 1-25, the hydroxyl groups of both carbon 1 and 3 are equatorial, so the molecules line up in a \r\nstraight line quite nicely. So how is this helix is formed?  &&

1269    A: You say the 1,3 connected molecules can line up nicely and that the book\'s answer says no. Consider the eq-eq 1,3  bonds. The \r\nniceness is that they allow the the two connected rings to lie in about the same plane; imagine a trimer viewed from the \r\nside--nothing is hanging down. Now view it from above:<br>\r\no-o<br>\r\n   \\<br>\r\n    o<br><br>\r\nThe turn is imposed because unlike c1 and c4, c1 and c3 do not lie at opposite ends of the chair.',NULL,1375,1,1),(###-09-20&&

1270    Q: How does the emulsification of fat work

1270   A: A molecule that has along hydrocarbon chain and a charged group at one end can use its hydrophobic\r\npart to bind to a fat (triglyceride) and its charged end to \"drag\" the fat into solution in water.\r\nSeveral together can encircle a small piece of fat that has broken off from a large fat globule\r\nand get it soluble in the water before it has a chance to coalesce back to the mother fat globule.',NULL,1377,1,1),(###-09-20&&

1271   Q: What is the reason the glucose chair does not flip to form other conformations

1271   A: In other conformations, hyrdroxyls are more pushed together. The more stable chair conformation minimizes these steric conflicts.',NULL,1379,1,1),(###-09-20&&

1272   Q: Are trans fatty acids liquid or soild

1272   A: Triglycerides containing trans fatty acids tend to be solid.',NULL,1380,1,1),(###-10-12&&

1273   Q: How does DNA, with its relatively large number of phosphrous groups in an ionized state, balance its charge

1273   A: In all eukaryotes it is folded up in association with a special class of positively charged proteins (histones). In prokaryotes, it is associated with small molecules and maybe some proteins with a \r\npostive charge. Details on eukaryotes and histones next term. Details are in both texts.',NULL,1391,2,1),(###-10-18&&

1274   Q: Why couldn\'t Meselson-Stahl have used standard sedimentation velocity centrifugation? Shouldn\'t DNA with only N-15 sediment faster because of its extra mass

1274   A: The extra mass is less than 1% and wouldn\'t make enough of a difference.',NULL,1392,2,1),(###-10-19&&

1275   Q: In the Meselson-Stahl experiment, is it possible to definitively say what dispersive replication would look like after 2 generations

1275   A: The critical point here that none \r\nof the 4 DNA molecules after 2 generations would contain only N-14 (as they would in semi-conservative replication).',NULL,1393,2,1),(###-10-19&&

1276   Q: Why can\'t DNA have parallel strands

1276   A: If you try to build a model that fits the data, it has to be antiparallel. If you don\'t want to try it for yourself, you have to take it for granted.',NULL,1394,2,1),(###-10-19&&

1277   Q: Lecture covered why DNA uses T instead of U, (due to a C often wrongfully turning into U), but why does RNA use U rather than T

1277   A: You need something to tell DNA and RNA apart. U vs T seems to be as good as anything. (Same base pairing, but one methyl difference to act as tag.) The issue is, if this \r\nis the difference, why is the U in the RNA and the T in the DNA and not vice versa?',NULL,1395,2,1),(###-03-31&&

1278    Q: If you had a problem \"downstream\" in the Rb gene so that Rb does not inhibit E2F, wouldn\'t p53 catch this as it \"scans\" the DNA upstream and notices the mutation, sending the cell into apoptosis

1278    A: p53 does not scan anything directly. It responds to a signal, not really known, that is generated when there are lots of mistakes or damage to the DNA. It does not pick up that one particular gene is mutated.',NULL,1497,3,2),(###-03-31&&

1279    Q: Are oxytocin and vasopressin made in\r\nthe posterior pituitary or are they made in the nerve cells in the hypothalamus? What does the PP receive from the HT

1279    A: The hormones are made in the hypothalamus. The hormones are packaged into vesicles and the vesicles move down the MT inside the axons until they reach the nerve endings in the PP. The vesicles then release their contents from the endings in the PP by exocytosis.',NULL,1498,3,2),(###-04-01&&

1280    Q: In class, we said that only the liver responds to glucagon to dephosphorylate G-P and to release glucose back into the blood when glucose levels fall. However, doesn\'t adipose tissue also respond to glucagon to break down fat

1280    A: Adipose cells do have glucagon receptors, at least in some species. However in most cases, the response to glucagon is mediated \r\nprimarily through the liver. (That is, the effects in liver are much larger and more important that the effects in adipose.) So we have \r\nbeen ignoring its effects on other tissues.',NULL,1499,3,2),(###-04-01&&

1281    Q: If it\'s possible to inherit a predisposition for a certain cancer, isn\'t it also possible to inherit a mutated oncogene resulting in the onset of cancer or would the fetus immediately abort

1281    A: There are a few cases where you do inherit a mutated oncogene. As you pointed out, most cases like this are lethal. But some are not. \r\nThe conditions differ in the chances that the person with the mutated gene will get cancer. The question is how this really works -- why \r\nthe same mutation causes cancer in some cells and people (but not others) and only after a long delay.',NULL,1500,3,2),(###-04-01&&

1282    Q: If two adults were predisposed to cancer and had a child, wouldn\'t the child automatically have a tumor suppressor related cancer

1282    A: If the adults have specific alleles of genes that predispose to cancer, they can pass on those alleles. But kid might not get them unless parents are homozygous.',NULL,1501,3,2),(###-04-03&&

1283    Q: If hormones are made in the axons of \r\nthe hypothalamus and then travel down neurons to ultimately end up in the posterior pituitary, then what is the role of the action potential? Do both action \r\npotentials AND hormones travel through the axon of the neurons

1283    A: Action potentials travel down the membrane. Vesicles containing transmitter, hormones, etc. travel through the cytoplasm on MT tracks. Both go from \r\nthe cell body to the synaptic terminal, but by different routes and they deliver different things -- chemicals or potential differences. An AP is needed to \r\nopen Ca++ channels and signal release of a transmitter. (The transmitter usually was made in the cell body and transported in vesicles to the \r\nsynapse, where the vesicles wait for a

signal to release their contents.) I don\'t know if an AP is needed to signal release of the neuroendocrine \r\nhormones (oxytocin and vasopressin) from the Post. pit. The release is controlled by the HT, in response to the signals it receives, but I don\'t know \r\nhow the HT triggers release of the hormones. An AP traveling down the neurons from the HT to the synapse in the Post. Pit would do it.\r\n(The books I have consulted are vague about this point. If I have more time I\'ll hunt it down.)',NULL,1502,3,2),(###-04-03&&

1284    Q: If only one cell of the thyroid becomes tumorous and overproduces TH, is it possible that this doesn\'t cause goiter because the TH produced is an endocrine \r\nhormone (not paracrine) and thus won\'t stimulate growth of other nearby thyroid cells? Also, if you consider a huge tumor in the thyroid that\'s almost the size of the original thyroid, would this be considered \r\ngoiter (enlarged thyroid), or just a tumor?\r\n&&

1284    A: TH doesn\'t stimulate cells of the thyroid. It stimulates other body cells. Overproduction of TSH (not TH) is what causes overstimulation of thyroid growth.\r\n\r\n\r\n<p>What to call a growth is usually clear (tumor or not) but sometimes it isn\'t totally obvious. A large mass of cells whose growth is dependent on TSH (or stimulation of TSH receptors) would be considered a goiter. I think a growth of cells that is not reliant on TSH or outside signals would be \r\nconsidered a tumor.',NULL,1503,3,2),(###-04-03&&

1285    Q: Regarding Problem 15-5: Shouldn\'t all of us get cancer if it only takes inheriting one mutation of a proto-oncogene to significantly increase our chances of another, cancer-causing mutation?\r\n\r\n&&

1285    A: One possibility is that proto-oncogenes usually change cells in ways that are recognized by the immune system. So the cells are destroyed. A gain of \r\nfunction mutation (producing an altered protein or a gross excess of protein) is more easily targetted than a lack of function one (that simply fails to \r\nproduce a normal protein). In other words, most of us do develop plenty of cancer cells, but they are zapped by the immune system.',NULL,1504,3,2),(###-04-03&&

1286    Q: On handout 15A (how TG is modified in the lumen of a thyroid follicle and in thyroid cells to make T4), is it correct to assume that the thyroid cells are not specialized and that some cells can both take up iodine AND export proto-TG into the lumen

1286    A: Yes, that is correct. The picture is drawn that way just to make it easier to follow. All cells carry out both processes.',NULL,1505,3,2),(###-04-04&&

1287    Q: Is there a difference between a Growth Hormone (GH) and a Growth Factor (GF)

1287    A: Yes. GH is a particular hormone. GF\'s are a class of paracrines and autocrines.',NULL,1506,3,2),(###-04-04&&

1288    Q: For problem 2 of Recitation Problems #10, what is the significance of the fact the amplitude of the depolarization at the membrane potential of -100mV is smaller than that at -90mV

1288    A: There isn\'t any. Whoever made the picture for me made a mistake. The two pictures should be reversed!',NULL,1507,3,2),(###-04-05&&

1289    Q: In problem 8-8 (E), I don\'t understand why  an IPSP is expected at point A in the diagram but not point B. What is the significance of the + and - signs in the diagram, and does the answer have to do with one synapse being involved in a gap junction and the other a cleft

1289    A: Both synapses should have a space. No gap junctions. That\'s my lousy drawing, and I\'ll fix it next time. + means excitatory synapse and - means \r\ninhibitory.',NULL,1508,3,2),(###-04-05&&

1290    Q: Is there an integrative center for glucose regulation

1290    A: Not separate IC different from the sensors. An integrative center only involves multiple inputs (i.e brain and skin thermorecptors). For glucose, the pancreas is the only place that checks to make sure the level of glucose in the blood is ok.',NULL,1509,3,2),(###-04-05&&

1291    Q: We said that Ras requires other proteins for its activation and to control the rate of deactivation. Does this mean that it cannot hydrolyze GTP itself

1291    A: Ras can hydrolyze GTP, but relatively slowly. So other proteins have a big effect on the rate. I think the ras does the actual hydrolysis, but proteins called GTPase activating proteins (GAPs) speed it up.',NULL,1510,3,2),(###-04-05&&

1292    Q: In Grave\'s disease, is the problem \r\nthe lack of negative feedback because the antibodies to TSH receptors act as agonists

1292    A: No. The problem with Graves is that the TSH receptors are always stimulated by the Ab, whether TSH is there or not.',NULL,1511,3,2),(###-04-05&&

1293    Q: In the cases of thyroid tumor and Graves\' disease, TH increases while TRH and TSH decrease. Why does a goiter develop from Graves\' disease and not for thyroid tumor

1293    A: Goiter is the result of overstimulation of the gland by any means.',NULL,1512,3,2),(###-04-05&&

1294    Q: Is cyclin important in activating CDK or is an important part of the complex itself

1294    A: Both. CDK is the catalytic part, but cyclin must be part of the complex for CDK to work.',NULL,1513,3,2),(###-04-06&&

1295    Q: In Problem 8-8 A, can\'t there be ligand-gated ion channels at C? I thought you could have synapses anywhere over the cell body.&&

1295    A: The picture means to imply only two synapses. If there are other ones, you are right.',NULL,1514,3,2),(###-04-26&&

1296    Q: Why is there only one twitch per action potential if there\'s Ca++ in the cytoplasm after a single AP and ATP around? When we say that a second AP can trigger another \r\ntwitch before the first is over, are we talking about another twitch in the same muscle fiber, or an adjacent one

1296    A: A twitch is from one fiber -- all sarcomeres contract. There is a pump in the SR membrane that removes cytoplasmic Ca++ quickly. The \r\nremoval of Ca++ ends the twitch.',NULL,1515,4,2),(###-04-23&&

1297   Q: How can the parasympathetic nervous system produce inhibitory effects if its neuron-effector synapses use mainly acetylcholine, an excitatory neurotransmitter

1297   A: Acetyl choline is always excitatory at synapses with nicotinic receptors. It is not always excitatory at synapses with muscarinic receptors. There are\r\n 5 different types of muscarinic receptors that all use 2nd messengers.',NULL,1516,4,2),(###-04-22&&

1298   Q: If the rhythm of the SA node\'s action potentials is faster than the\r\nAV\'s, the bundle of His or the Purkinje fibers, how can the\r\nheart maintain a regular beat

1298   A: The SA node fires and triggers an AP in the AV which fires and triggers an AP in the Purkinje fibers. The AV and the Purkinje fibers normally \r\nnever get a chance to fire on their own -- the SA node triggers an AP before they can trigger one spontaneously. After the AP, each cell type \r\nreturns to RMP and startd to depolarize. But before the AV and P fibers can depolarize on their own, they are triggered again by the SA node. \r\nAnd so on. You are thinking the AV node would fire by itself after the SA node. But that doesn\'t happen. Once the AV node fires, because of a \r\nsignal from the SA, the AV node can\'t fire by iself right away. It doesn\'t continue to depolarize -- it starts over.',NULL,1517,4,2),(###-04-22&&

1299   Q: In having \"pacemaker activity,\" do the bundle of His and Purkinje fibers all have the\r\nability to initiate their own AP or are they \r\ninvolved with regulating the heartbeat (i.e. setting the pace)

1299   A: All have pace maker cells, but usually the first one to fire is \"in charge\" and the others never get a chance to initiate their own AP\'s.',NULL,1518,4,2),(###-04-26&&

1300   Q: When someone who has too much sugar in their blood (as in diabetes mellitis), why isn\'t it excreted

1300   A: Sugar accumulates in blood because of lack of insulin. That sugar then gets into the filtrate. But the carrier can\'t remove it all in the proximal \r\ntubule.(Normally there\'s only a little sugar in blood, and the carrier can remove it all.)\r\nSo hyperglycemia -- high sugar in blood -- is due to lack of insulin.\r\nSugar in urine is a secondary effect of that.',NULL,1519,4,2),(###-04-26&&

1301   Q: Is the thing that TCRs hit called an antigen

1301   A: Yes, but it has to bound to MHC on the surface of a eukaryotic cell in order to be recognized.',NULL,1520,4,2),(###-04-27&&

1302   Q: On problem 8-3 (C), why doesn\'t changing the permeability of the membrabe to K+\r\nchange the threshold itself (because it would change resting potential)

1302   A: Changing resting potential is different than changing threshold. That\'s two different items. Changing RMP may change the distance to \r\nthreshold, but it doesn\'t change the value of threshold.\r\nIn this case, threshold stays the same, but distance from RMP to thresh. changes.',NULL,1521,3,2),(###-09-17&&

1303   Q: We said in lecture that cellulose consists exclusively of glucose connected in a straight chain through beta C1-C4 connections, both of whose OHs are equatorial, leaving the C6 OHs sticking out on

either side of the polymer for interchain hydrogen bonding (Purves 3.14a1). In Purves, it looks like the C2 OHs are highlighted rather than the C6, and that both alternate up and down. Can you clarify this?\r\n\r\n&&

1303    A: The cellulose in the Purves diagram is depicted using the Haworth, or flat ring, projection.  I have emphasized that this \r\nkind of depiction does not yield the the true orientation of the molecule in 3-dimensional space.  Indeed an entire handout \r\npage is devted to this point (2-8).  Thus when you write of groups being alternating up and down you may be visualizing \r\nthe molecule incorrectly.  Both the C6 and the carbon 2 hydroxyl are oriented equatorially in glucose in the usual chair \r\nconformation.  In cellulose the question is more difficult and somewhat ambiguous, since there will be free rotation  round \r\nthe glycosidic bond, so that in the context of the polymer, a group could be up or down relative to  another glucose \r\nmonomer, a question that never arises when considering just a single glucose molecule. The picture in Purves has each \r\nmonomer upside down relative to its neighbor. It would be difficult to predict such an arrangement.  However, it does \r\nmake sense, in that the C6 the extends out on either side of the polymer, and since it has a  longer reach than the ring \r\nhydroxyls, it is better suited for making the intermolecular hydrogen bonds that hold the individual cellulose starnds \r\ntogether.  The alternation allows the molecule to hydrogen bond to 2 different neighbor strands using its C6\'s .\r\n \r\n<p>Here is another depiction of the cellulose showing inter- and intra-strand hydrogen bonding (although it is difficult here to \r\nsee the orientation of the groups relative to each rinig (axial or equatorial) :\r\n \r\n<a href = \"http://www.steve.gb.com/images/molecules/sugars/cellulose_(hydrogen_bonding).png\">http://www.steve.gb.com/images/molecules/sugars/cellulose_(hydrogen_bonding).png</a>\r\n \r\n<p>All this goes beyond what I chose to present.',NULL,1531,1,1),(###-09-17&&

1304    Q: I know that two equatorial hydroxyls bonded together form a flat glycosidic bond. Does the same apply to two axial hydroxyls

1304    A: That\'s more difficult to visualize.  It seems to me that once you have a bend, the most likely structure will be a helix, with \r\nintramolecular H-bonding (easier and quicker to form). But I am not aware of a simple example with a repeated alpha \r\nhexose-axial bond (e.g., galactose(alpha 1,4) galactose).',NULL,1532,1,1),(###-09-20&&

1305    Q: We said that the proteins in a boiled egg are irreversibly denatured in a tangled web of \"incorrect\" linkages. If you put the egg in \r\nurea and allowed all the tangles to denature back to unlinked polypeptides, gradually removed the urea, and exposed to oxygen, will you recover the uncooked egg

1305    A: Sounds like a great experiment. Got urea?',NULL,1533,1,1),(###-09-25&&

1306    Q: For problem 2-14, I am unclear about how the enzyme ribonuclease should be able to function as a whole when the peptide bond is cleaved between a serine and alanine residue.&&

1306    A: One way to think about it that the two separate fragments are behaving like two subunits if a heterodimeric protein, exhibiting a quaternary structure.  After all, the bonds that hold two polypeptides together in a quaternary structure are the very same types of bonds that hold a single polypeptide in a unique 3-dimensional structure. ',NULL,1534,1,1),(###-09-25&&

1307    Q: For problem 3-5 (A), the answer key states that Vmax is the only value that depends on Eo. I thought that just Km was dependent on Eo, so why is Vmax dependent on Eo?\r\n&&

1307  A: Eo is the amount of enzyme in the system, as moles or as a concentration, moles per liter. Km represents a quality of the enzyme that is related to its ability to bind its substrates and its products, defined as Km = (k2+k3/k1). So where is the AMOUNT of enzyme in that definition? It is not there. Surely, Km depends on the enzyme in the sense that it depends on what the enzyme IS, what properties the enzyme has. But it is independent of the AMOUNT of enzyme, which is what Eo denotes. The same kind of argument applies to k3. But Vmax is a value that characterizes a particular experiment, not the enzyme in general. It tells you what is happening, the initial velocity of product formation in a particular experiment in which you have a particular amount of enzyme, Eo. It\'s like k3 tells you the top speed of a truck, and Km the size of its bed, but Vmax depends on how many trucks you have to move all that material.',NULL,1535,1,1),(###-09-25&&

1308  Q: For problem 3-5 (C), I understand that \r\nbecause the Km is higher, the enzyme of the sick person functions worse, but why do we assume that Vmax is unaffected

1308  A: The higher Km for the enzyme from the sick person indeed makes it potentially worse, but it depends on the circumstance. Think of the Vo vs. S curve. At 10^-6 M substrate the normal enzyme is operating at half its maximal initial velocity, but the enzyme from the sick person is operating at only about a tenth of this velocity, a big difference. But at 10^-4M (the concentration cited in the problem) both enzymes are operating near their plateau (Vmax), so there is only a small percent difference in the velocities (91 vs. 98% of Vmax if you calculate it, which is not necessary if you simply stare at the curve). Thus the altered Km is unlikely to explain the patient\'s symptoms. \r\n\r\n<p>Perhaps the Vmax is also affected in the patent\'s enzyme, we don\'t know. If there was a substantial difference, that could implicate the enzyme as a cause in the illness. But that was not the question. The answer goes on to ponder this possibility by considering a case where the Vmax is NOT affected, just for the sake of argument. If that case, the enzyme\'s properties would be unlikely to be a factor in the disease, and the altered enzyme in the sick person may just be a coincidence.',NULL,1536,1,1),(###-09-28&&

1309  Q: In problem 2-11(B), does the phrase \"a mixture of HbA and HbX\" mean\r\nthat entire subunits are taken from each protein (ie, 2 alpha or beta chains from HbA, and two alpha or beta chains from HbX)

1309  A: It means you take whole HbA and whole Hb X and mix them up. Then you hydrolyze to get peptides, and separate \r\nthe peptides in the usual 2D manner for a fingerprint.',NULL,1537,1,1),(###-10-08&&

1310  Q: In the Kreb\'s cycle, if alpha-ketoglutarate is oxidized and CO2 is released, it seems\r\nthat you\'d be left with only one oxygen on the second oxaloacetic acid carbon, but succinic acid, the next compound in\r\nthe cycle, has a carboxyl group and thus one more oxygen on the third carbon. Where does this oxygen come from

1310  A: The oxygen comes from water. Hydration is a part of 3 reactions in aerobic pyruvate metabolism: the oxidations \r\nof pyruvate and alpha-ketoglutarate and the addition of water across the double bond of fumarate. That\'s 6 \r\nmoelcules of water used per glucose metabolized. In the end, water is produced by the electron transport chain, \r\none molecule of water per pair of electrons taken from NADH and FADH2. Since there are 10 NADH\'s and 2 FADH2\'s \r\ngenerated per glucose, that\' comes to 12 molecules of water generated by the ETC. 12 generated, 6 consumed, so \r\na net of 6 water molecules are produced per glucose, just as was written in the simplifying overall reaction of \r\nglucose going to 6 CO2 and 6 H2O. This level of detail was purposely not included in the lectures,

both to \r\nsave time and to make sure the emphasis is on seeing the overall strategy and not focus on the organic chemical \r\ndetails in this introductory course. However, it is satisfying to see the stoichiometries work out.',NULL,1538,2,1),(###-10-08&&

1311    Q: Concerning the distinction between steady-state and equilibrium. Is it safe to say that an intermediate in a reaction pathway will never be at equilibrium, but will instead always be in a steady-state? For example,in A->Q->K->B, will K ever be in equilibrium with Q, or do we simply say that it is at a \r\nsteady-state concentration? If in a steady-state, does K ever get converted \r\nback to Q, or is the reaction always from Q->K due to the fact that K is constantly being removed

1311    A: Never say never.  This gets complicated, but I can give you my take on it.\r\n<p>1) First of all, in a test tube, reactions certainly can and do run to equilibrium. So if you had A, Q, K, \r\nand B in a test tube in the presence of the interconverting enzymes, all those reactions would run until \r\nequilibrium is indeed reached.\r\n<p>2)  Inside a cell there are in fact many types of molecular interactions going on that are probably at \r\nequilibrium.  Proteins are binding each other, small molecules are binding to signaling molecules, proteins \r\nare associating and dissociating with DNA, etc. and the reactants and products are achieving their \r\nequilibrium concentrations. 3) Most individual reactions in metabolic pathways on the other hand are in a \r\ndynamic steady state, with products being removed by a downstream reaction.  However, on a microscopic level,\r\n products are still capable of being converted back to reactants, even if the net flow is in the direction \r\nof products.  It gets complicated:  To achieve equilibrium in real time, you need an enzyme catalyst, and an \r\nenzyme may be a good catalyst for the forward reaction and a poor one for the reverse reaction (poor Km for \r\nproduct, for example).  Furthermore the enzymes are often controlled, so that once a product builds up \r\ncatalysis can become rate-limiting due to feedback inhibition.  In these situations equilibrium is probably \r\nnot reached because the necessary reactions in certain directions are too slow. Is pyrophosphate, the \r\nhydrolysis of which helps drive DNA synthesis forward,  in equilibrium with inorganic phosphate?  I don\'t \r\nsee why not.  So I would say most pathways in general are in steady state, but equilibrium situations are \r\nnot precluded.  ',NULL,1539,1,1),(###-10-17&&

1312    Q: Why is it that only a catalytic amount of NAD is needed to keep the Krebs cycle going? I\r\nunderstand that NADH2 is oxidized via fermentation, but I don\'t see\r\nhow only a small amount of NAD is needed per mole of glucose when 2\r\nmoles of ATP are needed. Doesn\'t the ATP cycle as well

1312    A: Think of billion glucose molecules ready and willing into be taken apart to release energy. Then think of \r\nmillions of ADP  molecules produced from running coupled reactions millions of times.  Now think of just 2 \r\nNAD molecules that are indispenable for accepting electrons from glucose as part of the energy-yielding \r\npathway. Those same 2 NAD molecules can be used to accept electrons early in the fermentation pathway to \r\nbecome NADH and then give them up to pyruvate at a later step in the same fermentation pathway to become NAD \r\nagain and be used again and again and again.  From the point of view of the fermentation pathway (glucose to \r\nlactate), NAD/NADH molecules ar being use catalytically, over and over again.  ADP is a substrate in this \r\npathway:  four times (per glucose molecule) ADP\'s are used as substrates for reactions that require their \r\nparticipation as reactants.  Two ATPs are also required reactants in reactions that convert them to ADP. So \r\noverall the fermenation pathway necessarily converts 2 ATPs to 2 ADPs and 4 ADPs to 4 ATPs, for a net change \r\nof 2 ADPs going to 2 ATPs.  Although 2 of the ATPs that are formed can be considered as cycling, two others \r\nare not. They are being produced in a net synthesis of ATP from ADP, and there are plenty  ADPs waiting for \r\njust this conversion.  Now if you

consider the whole complex cell, the whole E. coli cell, these net ATPs are \r\ngoing out and participating in coupled reactions and in doing so are being converted to ADPs, and it is these \r\nADPs that are awaiting reconversion into ATP in the fermentation pathway.  So you can consider ATP/ADP to be \r\nrecycling, but only in the context of the grand scheme of one E. coli cell becoming two E. coli cells, NOT in \r\nthe context of the fermentation pathway by itself. There they are flowing through to form net final products, \r\n2 ATPs.',NULL,1540,2,1),(###-10-17&&

1313    Q: In reaction 5 on Handout 10-2, is it that the delta G naught -7 kcal/mol, providing the requisite energy for the polymerization, or does it create a delta G of -7 kcal/mol by virtue of its \"pulling away\" of the P-Pi, shifting the equilibrium to the right

1313    A: The answer is that rxn 5 itself has a delta G naught of -7. Since it goes strongly to the right, the effect \r\nis to make the delta G (not delta G naught) of rxn 4 very negative. (Although the delta G naught of rxn 4 is \r\n0.)',NULL,1541,2,1),(###-10-18&&

1314    Q: In problem 4-17 (A), using radioactive labeled carbons to monitor the process of the Krebs cycle, it appears that the citrate molecule is symmetrical and that the two terminal carbonyl groups are equivalent. Doesn\'t this mean that once isocitrate is formed, half the radioactivity will be on each of the terminal carboxyl groups and therefore that half of the radioactivity will be lost to CO2 when alpha-ketoglutarate is converted to succinic acid?  &&

1314    A: Because citrate is a 3-dimensional molecules (not planar like fumarate), a smart enzyme can \r\ntake advantage of this fact by making sure to contact the substrate using 3 points of attachment. In this way \r\nit can distinguish between two otherwise identical groups attached to a centeral carbon.\r\n<p>See:\r\n<a href=\"http://www.bambed.org/cgi/content/full/30/1/27/F2\">http://www.bambed.org/cgi/content/full/30/1/27/F2</a>',NULL,1542,2,1),(###-10-22&&

1315    Q: Why does DNA replication have to start at the same end of the molecule such that one strand is replicated continuously and the other discontinuously, rather then on opposite ends for two continuous strands?\r\n&&

1315    A: Most DNA molecules are extremely long, and so you can\'t take it all apart and replicate the two halves separately. The single \r\nstrands would get all tangled up (and single strands are more easily damaged). If you replicated the two strands separately, from \r\nthe two ends, you would have large sections of single strands lying around for a longer period. This is probably what has been \r\nselected against -- there has been selection for a mechanism that leaves most of the DNA double stranded almost all of the time.',NULL,1543,2,1),(###-10-22&&

1316    Q: I understand that PCR is\r\nneeded to amplify a target sequence, but\r\nwhy do you need to make so many copies? Isn\'t it equally possible to\r\ndetect just a few copies

1316    A: Most chemical tests can\'t be done on very small samples -- the results aren\'t detectable. If the light is too dim, so to speak, \r\nyou can\'t see it.',NULL,1544,2,1),(###-10-22&&

1317    Q: How can allosteric inhibition change both the apparent Km and Vmax? Can allosteric inhibition be thought of as a combination of competitive and non-competitive inhibition?\r\n\r\n&&

1317    A: Allosteric inhibitors can affect the apparent Km of an a enzyme but not at all in the same way as a competitive inhibitor. \r\nAllosteric inhibitors can distort the active site of an enzyme,and in doing so can decrease the affinity of the enzyme for its \r\nsubstrate. The k2 reaction would be changed, and thus the Km. Substrate  may or may not be able to reverse this inhibition.  \r\nSince the active site is distorted, the k3 of the reaction can also be affected.  The enzyme structure is being changed by the \r\nallosteric inhibitor. Competitive and non-competitive inhibitors do not work this way. Moreover, enzymes that are subject to \r\nallosteric inhibition typically do not respond to substrate in a simple way, that is, they do not even exhibit the types of \r\ncurves we have discussed in the first place (usually an S-shaped curve for Vo vs. S). A quantitative treatment of allosteric \r\ninhibtion is beyond the scope of this course.',NULL,1545,1,1),(###-10-23&&

1318    Q: In ATP synthetase complex, do the protons move through the c subunits and up through the a, b, and delta subunits, or just \r\nthrough the c subunits

1318    A: It has been proposed that the a subunit acts as a proton \"wire\" that guides the proton to and from the c-subunits.  I know of no \r\nproposed involvement of any other subunits in proton transport.',NULL,1546,2,1),(###-10-23&&

1319    Q: Do we manufacture all our nonessential amino acids from scratch, or do we use some of the ingested amino acids directly, as \r\nwith essential amino acids

1319    A: Unlike E. coli, we can only synthesize about half of the 20 amino acids, with the other half being \"essential\", i.e., essential \r\nthat we get them from our diet.',NULL,1547,2,1),(###-10-24&&

1320    Q: How is it that nucleotide bases hydrogen bond if they\'re located on the inside of the DNA strand? I thought that the insides of such molecules were hydrophobic, and we said in lecture that the shape of DNA strands are partially determined by their hydrophobic character.\r\n\r\n&&

1320    A: The rings themselves are partially hydrophobic, but the groups that stick out and H bond are polar. Most biological molecules have \r\npolar parts and hydrophobic parts -- it\'s not all one or the other.',NULL,1548,2,1),(###-10-24&&

1321    Q: Why is the answer to problem 4-2 (C) only \"remove product in another reaction\" as opposed to this AND \"add an excess of reactant (F-6-P)?\"&&

1321    A: In a test tube, either high reactant, low product or both would achieve the same thing. In a cell, it\'s more likely to be low \r\nproduct. It\'s true that reactant is always being generated, but the level of reactant usually doesn\'t reach high steady state levels.\r\n Pathways tend to be pulled more by removing product than pushed by having high levels of reactant. If the reaction needs a push, it \r\nusually comes from ATP.',NULL,1549,2,1),(###-10-25&&

1322    Q: When calculating delta Go, why don\'t you use STP (273.15 K) instead of 298 K (room temp)?\r\n&&

1322    A: In biochemistry the standard state for calculating delta Go is taken to being close to ambient temperature, 25 deg C.  None of these \r\nvariation makes a great deal of difference in the calculation of delta Go, since 273 is solarge to begin with. But they are \r\ndifferent I agree. I think the the 25 deg standard is also used in organic chem, but there I am not sure. If you want to calculate \r\ndeltaG in red blood cells in the body, then 37 deg C would be correct to use in the RTlnQ part of the equation,

although delta Go \r\nwould just be delta Go by definition. In general one usually ignores this 4% difference in the calculation of delta G\'s, unless a \r\ntemperature change is the focus of the question (experimental or examinational).',NULL,1550,2,1),(###-10-26&&

1323    Q: In problem 6-14 (A), why isn\'t the first part synthesized C? I thought  the primer was placed onto the 3\' end of the template and synthesized in an overall 5\' --> 3\'\r\ndirection in discontinuous synthesis of the lagging strand.&&

1323    A: You only unzip the DNA a bit at a time. For disc. synthesis, You put the primer at the 3\' end of the unzipped part, not at the extreme 3\' \r\nend of the template.',NULL,1551,2,1),(###-11-02&&

1324    Q: If antisense RNA can be made by genetically modifying the cell -- by leaving the promotor and flipping the gene -- how exactly is\r\nthe gene flipped? If rotated around a vertical axis, won\'t the\r\npromotor be on the wrong strand? Rotating around the horizontal axis\r\ndoesn\'t seem to work either. &&

1324    A: You cut out the gene, and leave the promotor. Then you flip the gene 180 degrees. You put the gene in so if it was 5\'ACG-------TTC 3\' \r\non the top strand it is now 3\'CTT ----------GCA 5\'on the bottom (and 5\' GAA ......CGT 3\' on the new top.) ',NULL,1552,2,1),(###-11-02&&

1325    Q: If you can cut out and rotate the\r\nsection of DNA for which you want to make antisense RNA, why don\'t you just\r\ncut out that part of the DNA and eliminate the \"bad protein

1325    A: The real answer is you can add another copy of the DNA, which can be manipulated in a test tube before adding, but you can\'t delete \r\nthe one that\'s there. ',NULL,1553,3,1),(###-11-15&&

1326    Q: Is it possible for more than two viruses to infect a cell? Can more than one virus of the same type infect a cell

1326    A: Can be more than 2. Sometimes more than one virus of the same type can infect the same cell, although some viruses have a system so infection by the first virus inhibits further infection.',NULL,1554,3,1),(###-11-16&&

1327    Q: If tRNA has a strong tendency to fold up, won\'t the tRNA molecules be unable \r\ninto hybridize to the DNA, thus lowering the percentage? Or is there just such an excess of mRNA that the tRNA is negligible

1327    A: I assume that under the right conditions of concentration, temp. etc., the tRNA will hybridize to the DNA. Some may fold back on itself, \r\nbut if tRNA hybridizes to DNA it forms more base pairs (all sections pair, not just the bases in the hairpins) so I assume the DNA/RNA \r\nhybrid is more stable than the self folded RNA.',NULL,1555,3,1),(###-11-17&&

1328    Q: Purves Chapter 13 states in a diagram that in the lytic cycle, the host DNA can be digested. How can the cell continue to survive if its DNA is being degraded in this\r\nmanner? Wouldn\'t the mRNA, which is needed for protein synthesis, be\r\nused up and degraded rather quickly and the cell would be left with no\r\nenzymes? Or is the cell able to survive because the lytic cycle is so\r\nshort

1328    A: A cell that lyses is dead. So whether it\'s DNA or mRNA is shot makes no difference -- it won\'t survive the infection anyway. ',NULL,1556,3,1),(###-11-17&&

1329   Q: If DNA fragments\r\nare normally degraded and can\'t replicate, why is it that when a virus\r\ninjects it\'s DNA into a cell, it is\r\nnot only not degraded right away, but it is also able to replicate and be\r\ntranscribed

1329   A: The viral DNA is usually circular. It acts more like a plasmid.',NULL,1557,3,1),(###-11-18&&

1330   Q: In blotting, why is it\r\nthat DNA sticks to the filter but the excess probe doesn\'t (unless\r\nit\'s hybridized)

1330   A: The Cells/DNA are treated to denature them and bind them to the filter. The probe is added after that treatment -- after the glue has \r\ndried, so to speak. So there is nothing to make probe stick to the filter itself -- it only sticks to the complementary DNA.',NULL,1558,3,1),(###-11-18&&

1331   Q: In problem 7-17 (B), why does the met-tRNA hybridize only to the DNA it came from instead of every gene that has a met codon?\r\nI thought that the only thing\r\nthat tRNA had in common with genes that code for it was complementary base pairs from the anti-codon.  &&

1331   A: tRNA is a stretch of bases (really nucleotides) 75 nt long. So there are 75 bases available to hybridize to the DNA that coded for that \r\nparticular tRNA. That\'s what hybridizes -- the whole thing, not just the anticodon.\r\nEach tRNA has a unique base sequence that matches that of the corresponding gene.',NULL,1559,3,1),(###-11-18&&

1332   Q: Concerning enzyme repression vs. feedback inhibition, why does repression affect \"all enzymes\" of pathway in repression?  I understand \r\nthat repression inhibits particular genes involved in the transcription and translation of proteins (enzymes), but why would a single \r\nrepressor affect more than one gene

1332   A: If you don\'t need, say, to make trp, you don\'t need any of the enzymes in the pathway. But having all genes for all enzymes transcribed as \r\na single unit, you control transcription and translation of all enzymes at once. That\'s one point of repression/induction. You aren\'t just \r\nstopping (or starting) production of trp -- feedback inhibition can do that. You\'re stopping (or starting) the making of the enzymes that \r\nMAKE the trp.',NULL,1560,3,1),(###-11-26&&

1333   Q: The answer to problem 11-1 (B)  says that experiment 1 is an example of \r\ntransformation. Does this mean that just mixing purified plasmids and bacteria is an example of transformation even though it isn\'t \r\ntechnically the release of DNA from dead cells that is picked up by the bacteria

1333   A: Any time cells take up DNA from the outside, and the added DNA is recombined or permanently passed on, that\'s transformation. The \r\nsituation described here isn\'t \"natural\" but it\'s still transformation.',NULL,1561,3,1),(###-11-26&&

1334   Q: In problem 11-3, how do we know that the bacteria acquire their double antibiotic resistance through conjugation rather than transformation

1334   A: The question asks how the cells that acquired a plasmid passed that plasmid on, not how they got the plasmid in the first place.',NULL,1562,3,1),(###-11-26&&

1335   Q: Is it because of the high usage of drugs in hospitals that there tend to be more drug resistant bacteria in \r\nhospital rooms

1335  A: You got it. We spread the drugs around, and that kills the sensitive normal cells and allows the rare mutant resistant ones to multiply and\r\n take over. Not that the drugs shouldn\'t be used -- they are life saving -- but they should be used more carefully and selectively.',NULL,1563,3,1),(###-11-30&&

1336  Q: Can a virus give a bacteria a plasmid instead of a fragment

1336  A: Not normally. A virus doesn\'t carry a plasmid inside. But by genetic engineering you can make hybrids.',NULL,1564,3,1),(###-11-27&&

1337  Q: In problem 11-8 part B, are the recombinant \"double mutants\" considered \"normal\" or are normal and double mutants separate

1337  A: Normal has no mutations, double mutant has both. Both are recombinant. They are reciprocal recombinants -- if you cut and splice the DNA \r\nto get one, you have to get the other. So the number of double mutants and the number of normal recombinants is about equal.',NULL,1565,3,1),(###-12-12&&

1338  Q: We said that in the lysogenic cycle, one of the triggers for a provirus to enter the lytic cycle was poor health of the host. Is there evidence for this in infections of eukaryotes (e.g. is there evidence of \r\na viral infection appearing in a sick or dying individual when they could not possibly have been exposed recently)

1338  A: There are many cases known of reactivation of dormant viruses in people (for example, Herpes) by stress or environmental conditions. In \r\nsome cases a general weakening of the immune system can allow dormant viruses to start replicating. However I don\'t think people who \r\nare dying have an eruption of dormant viruses.',NULL,1566,4,1),(###-12-15&&

1339  Q: Is part of the reason that nondisjunction occurs  because of multiple crossovers leading to very tangled chromosomes that won\'t separate during meiosis I

1339  A: It actually goes the other way -- the tangling up caused by crossovers probably helps align the chromosome properly in homologous pairs \r\nand decreases nondisjunction. There is an untwisting process for the chromatids that follows after the cutting and splicing part.\r\n(People used to think that the twisting caused cutting and splicing, but we now know it\'s the other order.)',NULL,1567,4,1),(###-12-15&&

1340  Q: Can you explain the solution to problem 10-8 E

1340  A: The problem tells you the genotypes of each parent for for arg synthesis, drug resistance etc., so that you know that the \r\ndiploid is a triple heterozygote, and you know which alleles are on one chromosome and which alleles are on the other.  You know there \r\nare 3 genes involved, but the problem doesn\'t specify what order the genes are in on the chromosome. (The order listed isn\'t \r\nnecessarily the correct order.) You have to figure out the order.\r\n<p>The best way to do it is to write out what the genotypes of the diploid would be for all 3 possible orders. (Put the alleles on \r\neach chromosome in the right order for each case.) Then figure out how you can get a recombinant chromosome that has the alleles for \r\ngreen, arg- and strep-s (or the other combo given). Of course the order may be arg- green strep-s or arg- strep-s green. These are \r\ndifferent orders, but all have the same combo of alleles.  Which is the right order depends on which case it is. In one of the three

\r\ncases, it will take two cut and splice events to get the combo\'s given. Since these are the rarest combo\'s in the products of meiosis, \r\nthat must be the right order. In the other two cases it will take only one cross over to get the combo\'s, so they wouldn\'t be the \r\nrarest ones.',NULL,1568,4,1),(###-12-15&&

1341   Q: The answer to problem 10-15 B states that coat color is controlled by 2 indepedently segregating genes. Does this mean that both genes control the same step or two separate steps (co-dominance or epistasis)

1341   A: Two separate genes (epistasis) is not the same as two alleles of the same gene (co-dominance).\r\nTwo separate genes means, say, gene alpha and gene beta, with alleles A &a and B & b respectively.',NULL,1569,4,1),(###-12-15&&

1342   Q: In problem 10-8 E, I just don\'t understand how the order of the alleles impacts the cut and splice events. Why does arg-strep-color necessarily indicate that those exact two cases will occur after 2 events

1342   A: If order of genes is A-B-C and you have a heterozygote, it takes two cuts and joins to make a chromosome with alleles A, b & C. If \r\norder is BAC, it takes on one splice to get a chromosome with A,b & C.\r\nYou may need to see a picture.',NULL,1570,4,1),(###-12-16&&

1343   Q: In the answer to problem 10-16, it seems Ab/aB is always the case.  Why can\'t it be AB/ab

1343   A: To find out whether heterozygote is Ab/aB or AB/ab you have to consider the parents of the heterozygote.\r\n<p> Were they AB/AB and ab/ab? etc. Remember no crossing over in heterozygote until it makes gametes. No crossing over during mitosis and \r\ndevelopment from zygote to adult. So genotype of zygote (which depends on which gametes fused to make the zygote) determines what \r\nhetero will be -- AB/ab or Ab/aB.',NULL,1571,4,1),(###-12-18&&

1344   Q: In problem 10-12, how is it that OW/ow does not equal ow/ow if the\r\nsame crossover event led to the creation of OW from Ow, and ow from oW, respectively

1344   A: You are looking at a sample of many zygotes that came from many  gametes from many meioses. The real results of the meioses is 1/2 and \r\n1/2, but the sample does not have to be exactly one to one. If you have a huge pile of gametes that really are in one to one \r\nproportions, and you pick say 30, you don\'t always get exactly 15 and 15. Just as when you flip coins, you don\'t get always exactly 15 \r\nheads and 15 tails',NULL,1572,4,1),(###-12-18&&

1345   Q: When trying to identify recombinants in F2, when do you compare F2 to F1, and when do you compare F2 to grandparents

1345   A: When looking at recombinant gametes, you always take their heterozygous parent(s) as the \"parentals.\" In most cases, the parents and \r\ngrandparents have the same parental set up, but not always. If you start with parents, and get an F1, and then cross them to get an F2,\r\n what is \"parental\" is the same in parents (f1)  and grandparents (F2). But if you take a recombinant individual from the F1 and use \r\nIT to be parent of the F2, then the \"recombinant\" is recombinant relative to the parents, but becomes the parental for the F2.',NULL,1573,4,1),(###-12-18&&

1346   Q: In problem 14-4 (B), I understand why we cannot use HW since we are not in genetic\r\nequilibrium, but how is the value of 0.5 reached?\r\n \r\n&&

1346   A: You are assuming each individual gives one \"slip\" into the pot. The heteros give 1/2 Rh-slips and 1/2 Rh+ \r\nslips for 41 each. The homozygotes give 9 slips -- Rh- Rh- give 9 Rh- and Rh+ Rh+ give 9 Rh+. For 50/100 slips each.\r\nAnother way to see this: Heteros give 1/2 Rh- and 1/2 Rh+. The homozyugotes give equal amounts of Rh- and Rh+ (from the two types of \r\nheteros.) So over all, it\'s 1/2 each allele.',NULL,1574,4,1),(###-12-18&&

1347   Q: Say I have a cat that has an X chromosome that carries a gene for orange and \r\nanother that codes for black. If each individual cell expresses either X, why is it that the trait is expressed as patches, and not, say, each individual strand of fur

1347   A:  It\'s because of the way a cat grows its skin. One cell divides by mitosis to give a contiguous patch of skin. So all the cells in \r\nthat patch have the same X working and the same X off. In some tissues, the cells are all mixed up and each cell is different from its \r\nneighbor. But not in the skin.',NULL,1575,4,1),(###-12-19&&

1348   Q: In problem 15-2E, why does rate of CO2 production incrase if ATP falls?\r\n\r\n&&

1348   A: If ATP falls you need to break down more glucose to get more ATP. CO2 is one of the products of glucose breakdown in the presence \r\nof oxygen.',NULL,1576,4,1),(###-12-19&&

1349   Q: Why is the answer to problem 15-4 (C) 1/4 x (.42/.91)^2?  I get the\r\n1/4 part, but if you don\'t know whether the parents are carriers or not, do\r\nyou always use that ratio of carriers/(carriers + homozygous)

1349   A: Yes. You want the proportion of people of normal pheno who are carriers, not the proportion of carriers out of the total population. You want to leave out the homozygous recessives.',NULL,1577,4,1),(###-12-19&&

1350   Q: Why isn\'t problem 15-7 (C-i) a case of repression because you are\r\nshutting down the synthesis, not slowing it down as you would do in inhibition.&&

1350   A: The question is about what step is broken, not what step still works. mutants in B1 have a problem with inhibition, not repression.',NULL,1578,4,1),(###-12-19&&

1351   Q: Why isn\'t problem 15-7 (C-ii) a case of inhibition because\r\nthe rate of synthesis was being slowed down, but not completely shut off. &&

1351   A: Mutants in B2 have a problem with repression, but inhibition is okay.',NULL,1579,4,1),(###-01-24&&

1352   Q: Why do antibodies have two variable sites? Is it only because it allows a single antibody  to bind two antigens? What\'s to say that the antibody\'s variables won\'t attach to the same antigen at different sites

1352   A: a. That\'s how it is! b. It\'s probably an advantage (selected for) to form clumps. c. Each variable section on a single Ab is \r\nidentical, so the Ab can\'t attach to two different parts of the

same Ag. (Only to the same piece, or epitope, if it occurs more \r\nthan once.)',NULL,1580,1,2),(###-01-24&&

1353    Q: Shouldn\'t microtubules, microfilamnets and intermediate filaments be found in\r\nboth prokaryotes and eukaryotes

1353    A: Eukaryotes only.',NULL,1581,1,2),(###-01-24&&

1354    Q: About kinesin and dynein. Does\r\ndynein move things towards the nucleus of a cell and therefore\r\ntowards a cell if it is coming from the outside? And does kinesin move things to the plasma membrane of a cell and therefore away

1354    A: Both only move things inside cells. But dynein moves things toward the - ends of MT\'s and kinesin moves things toward the + ends.',NULL,1582,1,2),(###-01-27&&

1355    Q: If red blood cells only have the actin/spectrin/ankyrin web\r\nthat supports the plasma membrane, does this mean that they don\'t contain any microtubules or intermediate filaments

1355    A: Yes, that\'s right. The other stuff goes when the internal membranes, nucleus, etc. go.',NULL,1583,1,2),(###-01-26&&

1356    Q: Are their any instances where an active transporter can be a uniport

1356    A: Yes. You just use ATP to move one thing against its gradient.',NULL,1584,1,2),(###-01-27&&

1357    Q: Is there a reason that part B of problem 1-12 doesn\'t contain the answer \"adherens junction?\"  I thought that adherens\r\njunctions formed a belt around the cell and to me this satisfies the question: \"fusion of neighboring cells along an entire surface.\"&&

1357    A: There is a connection along a band, but not along the entire side of the cell. Thinking of the orange juice cell model, you don\'t \r\nhave the entire side of one tightly joined all over to the side of the next.',NULL,1585,1,2),(###-01-30&&

1358    Q: Problem 1-12 (E) states \"Involves fusion...\" In class I remember we said that tight junction is not fusion, but rather a lock between the ridges. Can you clarify

1358    A: People think of a tight junction as having a fusion between the two membranes because the two sets of intramembrane proteins are so \r\ntightly locked, forming a barrier. They don\'t mean a single bilayer forms, as far as I can tell.',NULL,1586,1,2),(###-02-01&&

1359    Q: In Problem 1-11 (B), the MT spindle fibers fail to form/slide\r\nproperly as a result of Drug B binding to tubulin and the chromatids aren\'t pulled apart in Anaphase. How does this imply that the number of chromosomes has doubled? It seems that the chromatids\r\nare still attached via their centromere, and therefore that the number of chromosomes is unaltered (although DNA content per nuclei\r\nhas doubled).&&

1359    A: Centromere splitting is a separate phenomenon than pulling of the chromosomes to opposite poles. So if the MT fail to \r\npull the sister chromatids far apart, they will still separate, and the number of chromosomes, not just chromatids, will double.',NULL,1587,1,2),(###-02-01&&

1360    Q: How does glucose get into capillaries if it\'s too big to diffuse across the membrane of an RBC?\r\n&&

1360    A: The glucose diffuses through the spaces between the endothelial cells that line the capillary. It does NOT cross a membrane, either \r\nthe membrane of the endothelial cells or of the RBC. (The RBC are inside the capillary, the endothelial cells are on the outside.)\r\nI realize that some of this is hard to follow if you don\'t know the basic set up (anatomy) of the circulatory system. It might be a \r\ngood idea to take a look at Purves. See p. 042 or 950. I realize a lot of people are confused about this, and I will try to find a \r\ngood picture to link in to the web notes or hand out in class.',NULL,1588,1,2),(###-02-01&&

1361    Q: Regarding problem 1-5, how do we judge whether or not certain acids and other  molecules will diffuse across a membrane?\r\n&&

1361    A: The question is about weak acids -- the kind in most cells (that don\'t ionize completely). The idea is that an un-ionized molecule might \r\nmake it across a bilayer if it\'s small or nonpolar enough, but a ionized molecule/ion would never make it. \r\n\r\n<p>Exactly what size molecule would make it across the bilayer is a good question. Glucose is big, compared to water, but it\'s usually considered a \"small\" molecule -- because it\'s not a polymer, or macromolecule. \r\n\r\n<p>In general, anything as big as glucose \r\ndoesn\'t pass through a membrane, but glucose is pretty polar, so maybe it isn\'t just size that\'s a issue here. As for the strychnine in the proble, I think the author of the problem thought it might be hydrophobic enough to pass membranes -- it looks vaguely like cholesterol.',NULL,1589,1,2),(###-02-05&&

1362    Q: Do both classes of motor molecules (dynein/kinesin and myosin) work\r\nboth as a ratchet between two polymers and along \"railroad tracks,\"\r\nor does only myosin work in-between and only dynein/kinesin work\r\nalong the tracks?\r\n &&

1362    A: Myosin moves relative to actin; in muscle it\'s the myosin itself that has to be moved.  The motor or half rachet is built into the \r\nmyosin. (But I suppose the myosin could be attached to something else, which is pulled by the myosin. However this is usually not \r\nthe main point.)\r\nD & K work inbetween -- they connect to something and pull that something relative to MT. As far as I know, D & K don\'t move for \r\ntheir own sake (Unless it\'s to get back to where they started so they can pull something else.)',NULL,1595,1,2),(###-02-05&&

1363    Q: In the notes, it says that spectrin and ankyrin comprise the peripheral cytoskeleton. Does that mean that they are microfilaments or intermediate filaments

1363    A: No, they are additional periph. proteins.',NULL,1591,1,2),(###-02-05&&

1364    Q: With ion channels, how can chemical potential and voltage balance out if they are both in the same direction?\r\n\r\n&&

1364    A: They don\'t have to balance. It\'s just that net ion flow stops when (or if) they ARE balanced, not just when they are closed.  Many ion channels open and close, and that\'s what starts or stops the flow, not reaching a balance.',NULL,1592,1,2),(###-02-05&&

1365    Q: How does the sodium/potassium pump run in reverse

1365    A: If Na is very very high outside, and K very very high inside, and ATP low, whole process works in reverse. ATP is synthesized,\r\n and Na goes in, K goes out. Ions go down their gradient, and the push makes ATP. If the head on a pump gets high enough, \r\nsometimes the pressure can push the pump backwards. That\'s the idea here.\r\n<p>\r\nYou can calculate the concentrations with standard delta G calculations that are described in Becker.\r\n',NULL,1593,1,2),(###-02-05&&

1366    Q: What structures inside the\r\ncell have a lipid monolayer as opposed to a bilayer?  &&

1366    A: EVERY membrane bound organelle is surounded by at least\r\nbilayer. One membrane = one bilayer. The question is whether one bilayer or more. The only thing with a monolayer we discussed is \r\nLDL. You are confusing a bilayer with a double membrane.',NULL,1594,1,2),(###-02-05&&

1367    Q: Let\'s say you have a protein\r\npump that pumps sodium from inside the cell (low concentration) to\r\noutside the cell (high concentration). What would happen if you\r\nplaced this cell in a solution where the concentration of sodium\r\nwas greater on the inside compared to the outside

1367    A: If only thing changed was Na, would probably continue to pump in the usual direction. It\'s a waste, but it doesn\'t happen in cells. (Even if it is a waste, it couldn\'t be helped -- a cell can\'t think. A protein that \r\nacts as a pump can\'t suddenly turn into a carrier. Two different proteins required.',NULL,1596,1,2),(###-02-05&&

1368    Q: When problem 2-7 asks what protein is probably abnormal in the mutant, why does it necessarily have to be the receptor and not the clathrin coating

1368    A: It says that all other substances are taken up normally. If clathrin were abnormal, all RME would be messed up, not just uptake of \r\nsubstance E. \r\n<p>The mutant protein has to be specific to uptake of E -- need to take in E but not anything else.',NULL,1597,1,2),(###-02-16&&

1369    Q: In the lecture it says, repeatedly, that \r\nperixisomes are thought to grow and split, similar to mitochondrion. However, in live lecture you said that perixisomes DID NOT grow \r\nand split, they were more like lysosomes in the matter. Please clarify this for me.&&

1369    A: I mispoke. there is some controvery on this point, but general opinion is that peroxisomes do grow and split. Remind me to say this \r\nin class.\r\n ',NULL,1598,2,2),(###-02-20&&

1370    Q: Regarding problem 3-7 (B), does the nuclear export signal not count as a localization signal? I realize it\'s on the mRNA, but \r\nwouldn\'t the gene still code for it

1370    A: We are ignoring any localization signals in the mRNA, and sticking to those on the proteins. It is assumed that all mRNA\'s have \r\nnuclear export signals, presumably because they bind to proteins that have the signals. But we are ignoring the details of mRNA \r\nexport to the cytoplasm. In part that\'s just because we can\'t cover everything; in part it\'s because all mRNA\'s are presumed to be \r\nmore or less the same in terms of localization signals, but all proteins are not.',NULL,1599,2,2),(###-02-20&&

1371    Q: In lecture 7 when we were comparing lysosomes, mitochondria,\r\nand peroxisome localization signals, you said that lysosomes do not grow and\r\nsplit, but can make new ones from

scratch, and that peroxisomes grow and split, but also may make new de novo. What do you mean by grow and split? Don\'t all cells grow and split? Can they make cells from scratch

1371    A: All CELLS grow and split, and none are made from scratch. But organelles are not cells. They are parts of cells. The question is how \r\ndo the individual parts inside the cell increase in number when the cell grows. Lysosomes, mitochondria and peroxisomes don\'t just \r\nget bigger -- they increase in number. A growing cell gets more of them. So how does that happen??',NULL,1600,2,2),(###-02-22&&

1372    Q: In the lecture 8 notes, II-B-7 says that mitochondrial proteins may\r\nnot need to enter all the way into the matrix before making their way\r\nto their final destination (i.e. intermembrane space, inner membrane,\r\netc.), but in lecture 6, didn\'t you say that in fact mitochondrial\r\nproteins always do go to the matrix before going to their final\r\ndestination? Which model should I go by

1372    A: We used to think that mito proteins always went to the matrix and then went \"back\" to the other parts. We now know that some \r\nproteins go \"part way in.\" So I think that\'s the right model. At least one problem in the book assumes the older model (I think \r\nit has enough data so that\'s the only solution.) I\'ll try to check the problem book and fix it for future editions. The real \r\nissue is how you tell what case you are dealing with from the results, and/or how to predict the results from either model. If \r\nyou can do that, you\'re okay.',NULL,1601,2,2),(###-02-22&&

1373    Q: Are DNA hypersensitive sites (regulatory regions) hypersensitive\r\nbecause there are never any nucleosomes there or because the\r\nnucleosomes that are normally there are displaced when transcription\r\nfactors bind to the DNA

1373    A: Nucleosomes were displaced. (At least that\'s the majority opinion.)',NULL,1602,2,2),(###-02-22&&

1374    Q: We know that signal peptides are hydrophobic and lodge in the ER\r\nmembrane. This isn\'t necessarily the case for mitochondrial transit\r\npeptides, peroxisomal localization signals, NLSs, etc, is it? Are all of these transport signals always hydrophobic sequences\r\nwhich can get lodged in membranes, or is this only the case of ER/EMS\r\nlocalization signals

1374    A: The other signals are different -- what there properties are depends on how they are \'read.\' For example, NLS binds to importin, \r\nbut a signal peptide has to bind the SRP.',NULL,1603,2,2),(###-02-23&&

1375    Q: In problem 3-10, by \"mix,\" does that mean\r\nsome of the golgi sacs were from the healthy and some were from the\r\nviral infected cells, or does it mean he actually fused viral and\r\nhealthy sacs? Why was the viral protein/ viral involvement important

1375    A: He broke open the two types of cells, isolated the respective Golgi apparati, and mixed them in a test tube. He did NOT fuse \r\nthe sacs -- he had no way to do that. He wanted to see if the modification enzymes and the cargo could end up in the same sac, \r\neven though the enzymes came from one Golgi apparatus (from the uninfected cells) and the cargo came from the Golgi from the \r\ninfected cells. His \"assay,\" or test was the formation of modified viral protein. That is, if the cargo and enzymes got together, \r\nthen the viral protein would get modified (by enzymes from the normal cells). He used virus protein because he wanted to be sure \r\nthe modification enzymes and the cargo

(virus) protein came from different sources -- different cells and therefore different \r\nGolgi apparati.',NULL,1604,2,2),(###-03-04&&

1376 Q: In problem 4-9 (A-3): Why doesn\'t treatment of the DNA of cell type 2 with increasing amounts of DNAse result in a labeled band on the Southern blot that has simply migrated further than for cell type I?\r\n\r\n&&

1376 A: As you increase the amount of DNase, you don\'t get discrete smaller bands, because the DNA is degraded at random spots. So you \r\nget a mixture of smaller pieces, and much less distinct bands or no bands at all. If you have pieces of assorted sizes, they are \r\nspread over a wide area of the gel, and there are not enough in any one place to bind enough probe to produce a band. Also if you \r\nuse enough DNase, the DNA is broken into such small pieces that they run off the gel.',NULL,1605,2,2),(###-03-05&&

1377 Q: In the pathway: hormone binds to\r\nreceptor --> G protein exchange --> G protein activates PLC --> IP3 split\r\nfrom PIP2 --> IP3 binds to Ca++ channels in ER, is the \"big bang\"\r\namplification caused by PLC splitting many molecules of PIP2, thereby\r\ngenerating many molecules of IP3 which can in turn activate many channels

1377 A: One receptor can activate more than one mol. of G protein. Other than that, you\'re right. Except of course that allows the passage \r\nof many ions of Ca++.',NULL,1606,2,2),(###-03-05&&

1378 Q: It seems like the amplification in the IP3 pathway is not as impressively large\r\nas the cAMP pathway, for which the second messenger is just the beginning of\r\na grand enzyme activation cascade.  In the IP3 pathway, it seems like the\r\n2nd messenger is pretty much the end product, since one molecule of IP3 can\r\nreally only activate one Ca++ channel at a time.&&

1378 A: Yes, but lots of Ca++ can come in one channel.\r\nThe amplification may be less, or it may be compensated for by the fact that the Ca++ gradient is so large that you get a whopping \r\nspike of Ca++',NULL,1607,2,2),(###-03-06&&

1379 Q: In Becker 5th edition, on page 279, it states the alpha adrenergic\r\nreceptors bind both epinephrine and norepinephrine and the beta-adrenergic\r\nreceptors bind epinephrine much better than norepinephrine. This is the\r\nopposite of what is in the table of Lecture Notes for Lecture 12. Can you clarify

1379 A: No two books really agree on this one. I think the reason is that there are subtypes of both alpha and beta. I was debating \r\nwhether to put this in the table or not. The point is that each receptor type has a different set of affinities for the two \r\nligands.\r\nMy physio book says alpha binds norepi over epi. Becker says alpha binds both. The physio book says Beta either prefers epi or \r\nbinds both about equally, depending on type. My Becker says beta binds epi better. I think the table matches the physio book.',NULL,1608,2,2),(###-03-07&&

1380 Q: Although heterochromatin (and particularly constitutive heterochromatin,\r\nsuch as telomeres and centromeres) is never transcribed and remains in a\r\nconstantly tight state, does it unwind at some point in order to be\r\nreplicated

1380 A: Yes. It replicates late in S. Otherwise it wouldn\'t be inheritable.',NULL,1609,3,2),(###-03-08&&

1381   Q: For question 4-16 (A), if we assumed that trans-acting referred to the gene that codes for a regulatory protein, \r\nwould the answer still be incorrect? Is \r\nthere a reason why trans-acting can\'t be the correct answer, regardless of whether it\'s referring to the protein or the gene

1381   A: It is very unlikely that a region that close to the start of transcription codes for a regulatory protein (or RNA). It\'s much more \r\nlikely that the region in question is part of the regulatory region of gene X to which a regulatory protein binds. However to nail \r\nit down, you\'d need to show the region wasn\'t transcribed or do some other experiments.',NULL,1610,2,2),(###-03-08&&

1382   Q: At the \"start\" check point of the cell cycle (\"restriction point\") external factors determine\r\nthe passage from G1 to S, i.e. GF. Is the G2 checkpoint dependent only on\r\ninternal factors

1382   A: The regulation of the cell cycle is much more complicated than we have let on. As far as I know, there are no external factors \r\ninvolved at the G2 checkpoint in adult animal cells. I think synthesis of one (G1) cyclin leads to synthesis of the next, etc. \r\nYou would have to consult advanced texts or the original literature to be sure of the details.',NULL,1611,2,2),(###-03-08&&

1383   Q: We said that regulatory \r\nregions for genes turned \"on\" will not have nucleosomes. Should we also assume\r\nthat transcribed DNA will also not have nucleosomes

1383   A: No. Transcribed DNA has virtually all of its nucleosomes at all times. Regulatory regions have their nucleosomes removed (or so \r\nloose they can\'t protect the DNA) as long as the gene they regulate is being transcribed. It is assumed the nucleosomes are removed \r\nand replaced by TF\'s. The transcribed DNA keeps most of its nucleosomes in place and only moves one or two at a time as polymerase \r\nproceeds. So large numbers of nucleosomes are not removed at at one time. Almost all are in place almost all the time.',NULL,1612,2,2),(###-03-08&&

1384   Q: In question 3-17, we are dealing with protein X \"receptors.\" In the case of a protein hormone, for example epinephrine = protein X, the protein might not actually be absorbed by RME. Is this a flaw in my thinking (as in, does the problem say something that would discount the possibility that RME doesn\'t happen), \r\nor can the answer to B-1 be \"can\'t predict\"

1384   A: The way the problem is written, it implies that RME is going on. (Or at least, it was supposed to. I also think the problem comes \r\nbefore exposure to hormones, so you are supposed to assume it is about RME.) You are correct that not every protein that binds to a \r\nreceptor triggers RME of the receptor.',NULL,1613,2,2),(###-03-08&&

1385   Q: On question 4-16 (B-3): Why wouldn\'t the band be lighter for D

1385   A: You are comparing the intensity of the band d in expt. 4 to the intensity of band d from experiment #3. You are not comparing the 4 \r\nbands in expt. 4 to each other. In expt. 4, Band d will be lighter than  bands a-c  in the same experiment (#4). But band in d will \r\nbe the same in experiment 4 as it is in experiment 3.',NULL,1614,2,2),(###-03-29&&

1386   Q: How does insulin both inhibit enzymes that break down glucose stores and assist in the breakdown of glucose? These seem to be opposing processes.\r\n\r\n&&

1386    A: Insulin promotes pushing glucose in two directions, both of which remove glucose. One is to polymerize the glucose into glycogen. \r\nThe other is to break down the glucose (that isn\'t polymerized) for energy. You are thinking that either the glucose is burned \r\nfor energy, OR the glucose is extra and is stored. But it isn\'t either one or the other -- it\'s both. When insulin is absent, and \r\nintracellular glucose is low, you burn other things for energy if possible, and break down glycogen (either to get free glucose \r\nor to burn the glucose -- depends on the tissue). When insulin is present, and free intracellular glucose is abundant, you break \r\ndown glucose for energy and store the extra.',NULL,1615,3,2),(###-03-30&&

1387    Q: Is it true that adrenals secrete androgens in small amounts

1387    A: I don\'t know if I remembered to say it, but I have a note to be sure to add it to the handout next time! The \r\nadrenal cortex secretes small amount of androgens (mostly dehydroepiandrosterone) that are converted to estrogen and/or \r\ntestosterone. The amounts are small and their significance is not completely clear.',NULL,1616,3,2),(###-04-03&&

1388    Q: Where do ras and p53 fit into cell cycle regulation

1388    A: In the progression from G1 to S, TK receptors will activate ras, which activates MAP kinases and leads to increased transcription in the nucleus. Cyclin-CDK activation inactivates Rb and releases E2F, and the cell progresses to S. p53 comes in fairly late-- it\'s stabilized when DNA is damaged and prevents cyclin-CDK activation.',NULL,1617,3,2),(###-04-05&&

1389    Q: How is it that negative feedback isn\'t always the same as inhibition?\r\n\r\n&&

1389    A: Neg. feedback means making a correction. That can include inhibition or stimulation, depending.',NULL,1618,3,2),(###-04-05&&

1390    Q: What kind of K+ channels remain open during RMP

1390    A: Ungated leak channels.',NULL,1619,3,2),(###-04-05&&

1391    Q: How do ligand-gated Na+ channels open in order to reach threshold

1391    A: Ligand binds to channel or generates 2nd messenger that opens channel.',NULL,1620,3,2),(###-04-05&&

1392    Q: When do voltage-gated channels close? Does it depend on the type of voltage-gated channels

1392    A: All types of channels exist. Generally, there is a fixed time lag for when channels close; in some cases it depends on the voltage.\r\n For AP, both close after a time, Na first, then K.',NULL,1621,3,2),(###-05-02&&

1393    Q: What genes does Pax6 activate? Exactly what role do the K4 and K27 methylations play in this process

1393    A: 1. Pax6 is activated in the head ectoderm by this regions previous\r\ninteractions with other tissues that where adjacent to the head ectoderm\r\nduring previous stages of development - we did not go into the molecules\r\nnor the names of the specific tissues involved here.\r\n<p>2. By the time

the optic vesicle - a protrusion of the brain (neural\r\ntube that just pinched off from overlying ectoderm) is underneath the\r\noverlying head ectoderm, Pax6 is already activated in all of the head\r\nectoderm.\r\n<p>3. Pax6 activates genes such as the tyrosine kinase receptor necessary\r\nto respond to BMP4 and FGF8 if they will be secreted in the vicinity.\r\n<p>4. Activation of the TK receptor expressed in the head ectoderm cells by\r\nBMP4 and FGF8 secreted from the optic vesicle, activates additional TFs\r\nthat work together with Pax6 to induce all of the proteins necessary to\r\nproduce the lens of the eye.',NULL,1622,4,2),(###-04-06&&

1394    Q: In problem 8-18, how does the cell return to resting \r\npotential without other ion channels than the three listed

1394    A: You can return to RMP without channels to counteract the AP -- it just takes longer.',NULL,1623,3,2),(###-05-03&&

1395    Q: Does water flow out of the lumen in the descending portion of the loop of henle by virtue of the Na+ gradient established in the rising portion, or simply because the osmolarity is higher in the epithelial cells that line the tubule

1395    A: It\'s the Na+ gradient that\'s primary. That pulls water out of the epithelial cells, which in turn pulls water out of the lumen.\r\nWhat is the steady state osmolarity of the cells lining the tubule? Probably the same as in most other cells, and not unusually \r\nhigh.',NULL,1624,4,2),(###-05-03&&

1396    Q: The answer to problem 8-16 (B-3) seems to say that receptor cells that cannot generate their own APs do not get graded\r\npotentials at all. Is this true

1396    A: In this case, the receptor cells release transmitter that causes a graded potential in the next cell, which does or does not fire \r\nan AP in response. This is a made up case.',NULL,1625,4,2),(###-05-05&&

1397    Q: What is the equivalent of the axon hillock of a neuron in a muscle\r\ncell?  Where do the voltage gated channels start relative to where\r\nthe acetylcholine receptors are

1397    A: Remember there is no summation here in the neuron sense -- every EPP generates an AP. The voltage gated channels that generate the \r\nAP in the membrane of the muscle cell are everywhere except at the endplate -- so they surround it.',NULL,1626,4,2),(###-05-05&&

1398    Q: In the class notes, it states that an EPP is the depolarization of the muscle membrane, but it also states in the next line that \r\nan AP in a neuron leads to 1 EPP, which in turn leads to 1 AP in a muscle. So does an AP in a neuron leads to an EPP, which is an AP of a muscle membrane

1398    A: An EPP is NOT an AP.\r\nAn EPP is like an EPSP -- you\'ve opened ligand gated channels but you haven\'t opened voltage gated channels and triggered an all \r\nor none response. The EPP at the endplate area triggers an AP in the surrounding area of the muscle membrane by opening voltage \r\ngated channels in the surrounding area of membrane.',NULL,1627,4,2),(###-05-04&&

1399    Q: Does humoral immunity only kick in if there\r\nis a bacterial infection, or for a viral infection as well

1399    A: Viruses can trigger the humoral system  if there is viral protein in the blood, or viruses are engulfed by macrophages. Viral \r\ninfection often triggers both halves of the system.',NULL,1628,4,2),(###-05-04&&

1400    Q: Are memory cells are still created during a viral infection to \"prevent\" re-infection, i.e. lead to a faster secondary\r\nresponse in case you encounter the same virus again

1400    A: Yes, there are memory B and T cells after most viral infections.',NULL,1629,4,2),(###-05-05&&

1401    Q: When an EPP reaches the endplate of muscle tissue, is it strong enough to always trigger an AP? Is the endplate similar to\r\nthe axon hillock

1401    A: Yes, EPPs in muscle alwayus trigger an AP, but it\'s more similar to the synapse. For this, a picture in one of the texts may help',NULL,1630,4,2),(###-05-06&&

1402    Q: Low blood pressure causes dilation of the afferent arterioles, leading to increased flow through the kidneys and thus increased GFR. Does this cause an increase in overall blood pressure by selective reabsorption of water? What about angiotensin II, a vasoconstrictor also released in response to low BP

1402    A: This causes an increased local blood pressure at the glomerulus, but not overall BP. Filtration is increased, not reabsorption. Since aff. arteriole is now \r\nlarger, relative to efferent, pressure at glomerulus is larger -- more pressure to push liquid into the tubule.\r\n<p>angiotensin II increases overall PB by constricting periperal blood vessels and cutting down the length of tubing, so to speak, so same \r\namount of blood fills less volume of blood vessels. Both vasodilation and vasoconstriction can increase blood pressure because they involve different blood vessels.',NULL,1631,4,2),(###-05-07&&

1403    Q: Do all 3 types of muscle have gap junctions

1403    A: Smooth and cardiac have gap junctions to help spread the good news. Skeletal muscle cells have solved the problem of coordination \r\nby fusing many  cells into one.',NULL,1632,4,2),(###-05-08&&

1404    Q: In problem 8-15 (D), the answer says that cell #1 would fire the AP first because\r\nbinding of AcCh to its receptor would lead directly to the opening of\r\nligand-gated Na+ channels (implying that the receptor is ionotropic). But,\r\ncell #1 is a postganglionic cell of the PNS. I thought that the AcCh receptor for a postganglionic cell\r\nin the PNS is muscarinic and metabotropic, in which case binding would initiate a 2nd messenger system and ligand-gated channels would not be\r\ndirectly opened.&&

1404    A: It asks which cell will fire an AP first, not which cell will cause its target to respond first. The synapse we are talking about \r\nis not the one made by the axon of the post ganglionic cell -- it\'s the one made by the axon of the pre-gaglionic cell on the \r\ndendrites or cell body of the post ganglionic cell.',NULL,1633,4,2),(###-05-08&&

1405    Q: In problem 13-13 (B-2), why is a defect in a surface receptor protein in the helper T cell not possible? Helper T cells secrete IL-2, which binds to a\r\nreceptor on the surface of the helper T cell. Without IL-2, doesn\'t\r\nthis mean that you do not activate the signaling cascade that is\r\nnecessary

to activate the B cells in order to create the memory\r\ncells? If there is a defect in the IL-2 receptor, then wouldn\'t\r\nadding IL-2 still not rectify the siutation

1405    A: The defect has to be a problem with a T cell surface protein. If it were the IL2 receptor, the T cells \r\ncouldn\'t be activated themselves, and so they couldn\'t activate B cells to secrete Ab either. In other words, more things than \r\njust class switching would go wrong. You wouldn\'t get a better 2ndary response either.',NULL,1634,4,2),(###-05-08&&

1406    Q: Is the epitope for a TC cell receptor \r\nalways a piece of protein from a viral-infected cell (vs. microbe-infected)

1406    A: It doesn\'t have to be viral, but it has to made inside the cell. Microbes multiple outside the cells.',NULL,1635,4,2),(###-05-08&&

1407    Q: In humoral response, can the epitope for a TH cell receptor be\r\nEITHER a piece of virus OR a piece of a microbe

1407    A: Depends on what the TH is helping. What ever it is, it has to be on the outside of a cell attached to MHC I or II.',NULL,1636,4,2),(###-09-07&&

1408    Q: Why are hydrophobic bonds stronger at higher temperatures

1408    A: Because the gain in entropy for the freed up water molecules is even greater at high temperatures, at which there is more molecular motion and so more states that can be occupied (or less accurately, more \"disorder\" if you will). Consider the opposite extreme, there would be little entropic gain for the freed up water molecules in solid ice.',NULL,1637,1,1),(###-09-07&&

1409    Q: If straight-chain glucose molecules jiggle around and most often the C5 and C1 (although sometimes C4 and C1) touch and collide, why isn\'t this true of C6 and C1

1409    A: I could not readily find an answer, but in looking at a small model (much firmer than the one in class), it appears that formation of a 7-membered ring puts some of the hyroxyls awkwardly close to other ring constituents (other hydroxyls or hydrogens).',NULL,1638,1,1),(###-09-07&&

1410    Q: When an ionic salt dissolves in water, the charges interact with the water molecules via polar interactions, and the salt dissociates. However, aren\'t polar attractions weaker than ionic attractions, in\r\nwhich case the NaCl should remain as an ionic unit

1410    A: The water molecules are just overwhelming in their numbers. As long as the Na and Cl ions don;t become too concentrated (able to find each other) then they stay hydrated and apart. Eventually a limit is reached (~ 6M). Beyond that, C Na+ and Cl- ions find eachother too often (as often as they are hdrated, and so the crystals remain (although in dynamic equilibrium). See <a href=\"http://www.mpcfaculty.net/mark_bishop/NaCl_dissolves.htm\">http://www.mpcfaculty.net/mark_bishop/NaCl_dissolves.htm</a> for a fun animation.',NULL,1639,1,1),(###-09-10&&

1411    Q: For cellulose, if a beta-glucose can bond to\r\nneither an alpha or a beta, how does the second glucose (if it is an alpha) form a beta-link to the next glucose

1411    A: One answer is to point out that \"being alpha\" is a tempoary state, as the free end, the free C1, can open up to a straight chain and then cyclize again to either alpha or beta, so we could just wait

around a bit.\r\nIn reality, things are a bit more complicated: <p>\r\n\r\nFor glycogen (like starch) synthesis: First a group (X for now) is added specifically to the C1 of alpha glucose so we get 4G1aX, where the \'a\' signifies an alpha conformation. This group is next displaced by adding 4G1aX to another molecule of glucose at its C4 to yield 4G1a-4G1 and X\'. Now a third 4G1aX can add to the only free C4 OH of the dimer, to yield 4G1a-4G1a-4G1 and so on.  The X is only added to C1 beta, and once it\'s added the ring can no longer open (no OH there any more).  The one free anomeric C1 on the polymer can equilibrate between the open chain and alpha and beta, why not. \r\n<p>Starch and cellulose synthesis is not as well understood, but all proceed through the initial additon of a temporary group to the anomeric carbon (also called the reducing end).',NULL,1640,1,1),(###-09-18&&

1412    Q: Is finger-printing simply the first part of the protein-sequencing process

1412    A: Actually, no.  The first step is the same, cutting the polypeptide into smaller fragments so that they can be individually sequenced in their entirety.  These fragments are then separated by a column chromatography method that we will not discuss (ion exchange chromatography). The separated peptides are then conveniently in tubes that were move sequenctially under the column as it was eluted. A small portion can be removed to treat with a color-producing reagent so you know what tubes contain the peptides. The peptides are then sequenced usually by a chemical method that chews in from the amino end (the Edman degradation procedure). That method can go further than carboxypeptidase, to maybe 50 residues. The whole process is repeated after cleaving the polypeptide with a different cleaving agent (enzymatic or chemical) to produce overlapping peptides.',NULL,1641,1,1),(###-09-18&&

1413    Q: According to lecture, C-terminal sequencing\r\nis \"less robust\" and yields fewer residues than N-terminal sequencing.\r\nSo why use C-terminal as an example in class instead of N-terminal\r\n(which I\'d guess is more commonly used?)&&

1413    A: A) Because I would have to spend more time explaining the organic chemical reaction that underlies the method (so as to avoid mysteries).  That would take time away from a more important topic down the line. \r\n\r\n<p>B)  Because I am foreshadowing the the action of enzymes as specific catalysts that carry out a particular job (in the cell as well as in the lab).  I do feel a little bad about sidestepping the real world, but Edman will be learned by those gwho go on to take biochemistry, and will make little difference to those who do not.',NULL,1642,1,1),(###-09-19&&

1414    Q: Why can\'t you fingerprint a protein or peptide by digesting it down to amino acids, which are then separted by electrophoresis and chromatography

1414    A: If protein A is hydrolyzed all the way to its consituent amino acids (NOT peptides), it will most likely contain all 20 AAs and these could be all separated from one another by the 2-D methods used in fingerprinting. Each amino acid will end up at a characteristic place on the paper. A place for lysine, a place for valine, etc.  Now if you take protein B, with a completely different sequence of amino acids, and hydrolyze it down to its constituent amino acids, it will also yield the same 20 amino acids, as most proteins (not all) contain at least one of each of the 20 different amino acids.  So its lysine will go to its spot, valine to its spot, etc.  The 2 patterns will look identical, so what has one learned? The distinctiveness of the 2 proteins has been obliterated by the complete digestion  down to single amino acids.   It is the peptides that maintain and display the distinctiveness; one must not lose that by further digestion.  Certainly all 20 amino acids can be separated, one from theother,  by the 2-dimensional separation methods USED in fingerprinting. (electrophoresis followed by paper chromatography) . But that would not be \"fingerprinting.\"',NULL,1643,1,1),(###-09-26&&

1415  Q: We explained a steady-state system with a tub of water and 2 pipes: in order to reach a constant level in the tub, the rate of water flowing out of the upper pipe is equal to the rate of water flowing\r\nout of the lower pipe. Is there more to this

1415  A: The system will reach steady state by itself. If you have a staedy state condition, and then the water inflow is slowed down, then the water level will drop, and as it drops, there will be less water pressure pushing it out at the bottom, until finally the level is low enough to just balance the inflow. The system will once again be at steady state, but  the steady state level in the reservoir will be lower. And vice versa if the inflow is increased.  If the inflow in increased so much that the outflow cannot keep up with the inflow even when th elevel reaches the top,  then the water will spill over the sides, then you have  ... a messy state to mop up.',NULL,1644,2,1),(###-09-27&&

1416  Q: Vmax is clearly dependent on Eo. Is k3 also  dependent on Eo? I understand that k3 is a constant for a particylar enzyme and that initial enzyme concentration will not alter its value, but in order to determine k3, you need Eo. &&

1416  A: No.  You can USE your measurement of  Vmax AND Eo to calulate k3, but k3 is noty dependent on theocnsentration of Eo.  That is, k3 will always be the same no matter what the concentration Eo is.  The fuel efficiency of your car is not dependent on how many miles you have traveled or how many gallons of gas were used. It is a characteristic of the automobile itself.  You of course USE the gallons and the miles to determine just what that characteristic number is.',NULL,1645,2,1),(###-10-03&&

1417  Q: In non-competitive inhibition, Vmax is affected but not Km.  I understand that the Km is the measure of the (K2+K3)/(K1), which\r\nshould remain the same, but I also know that we learned that the Km is measured by taking [S] at half the Vmax.  If the Vmax is changed, how does the Km stay the same? &&

1417  A: If you look at Handout  5-2d  you can see graphically how the Km can stay the same even if Vmax is very different.  If you are arguing from the point of view of the Michaelis-Menten equation, and think that if you change Vmax you must necessarily change Km to maintain equivalency, then you must realize that Vo is quite different in the presence of the inhibitor, the velocity being, well, inhibited.  So Vo changes to accomodate the change in Vmax and Km stays just the same.  This constancy of Km is indeed how non-competitive inhibiton is defined.',NULL,1646,2,1),(###-10-04&&

1418  Q: Enzyme inhibition occurs naturally in cells via allosteric inhibition, whereas most examples of non-competitive inhibition involve processes that are not natural, e.g. poisonous. But are there any examples of non-competitive inhibition in the cell that\r\nare natural, i.e. an NCI inhibiting a metabolic reaction as part of\r\nthe cell\'s metabolic design

1418  A: I do not recall hearing of this. Unlikely.  NCI is rare, as the \r\n\"facilitator\" is usually part of the substrate binding site, thus: CI.',NULL,1647,2,1),(###-09-20&&

1419  Q: Why doesn\'t the amino group in glutamine does take on a proton even at a low pH

1419  A: An amide is not the same as an amine. When you have C-double bond O-amino it\'s a single group with properties different from that of an amino plus a carbonyl. Same argument as to why a carboxyl is not the same as a hydroxyl plus a carbonyl.\r\n ',NULL,1648,1,1),(###-09-25&&

1420    Q: In problem 2-9 (D), I am having trouble\r\ndetermining the sequence of amino acids.\r\nI understand that I must look for where the substance cleaves the peptide according to the sub-peptides given, but I still don\'t\r\nunderstand the answer and the sequence given in the back of the book.&&

1420    A: Treat this problem like a puzzle. Assume you had the set of pieces given in B, and the set of pieces given in D. Both were derived from the same original sequence. How? Suppose you had two copies of the originial sequence.  One was cut up to give the pieces in B, and the other copy was cut up in a different way to give the pieces in D.  What original order of amino acids is compatible with both sets of pieces? You get the answer by trial and error. If you can\'t see how to do it, ask a TA or a prof.',NULL,1649,1,1),(###-09-28&&

1421    Q: Regarding problems 3R-2 and 2-9E. Both questions ask what will change about the peptides if amino acid sequence is changed. 3R-2 says charge but NOT SHAPE, 2-9E says shape. I would think that because the sequences are different, the\r\ndifferent orietations of the R groups will alter the shape. Do those answers contradict each other

1421    A: 2-9E refers to a very different order of amino acids. Note that the answer says that the peptides are so short they are unlikely to differ in shape -- they are both probably to small to have unique, 3D folded shapes. However, shape is the only property they MIGHT not share. In 3R2, you have a few amino acids different, and the enzyme still works. Therefore the normal and variant enzyme are likely to have very similar shapes and molecular weights. However, they could have different charges.',NULL,1650,1,1),(###-10-07&&

1422    Q: Problem 4-2 (A) discusses the radioactivity of G-6-P at equilibrium and whether F-6-P would therefore also be radioactive.  The solution says that radioactivity would be distributed evenly between the two since \"equilibria are dynamic.\" I thought the main difference between equilibrium and steady-state was that equilibrium was held static while steady-state experienced the dynamic emptying and filling.&&

1422    A: Steady state implies that there is a net flow in one direction of substrate in (from another reaction) and product out (to another reaction). So levels of P & S stay about the same, but there is unidirectional flow from S to P. It\'s an open system -- stuff comes in from the outside and other stuff goes out.\r\n<p> \r\nEquilibrium means there is conversion of both product to substrate and vice versa. But net concentrations of both P and S stay the same. (The levels of P and S are static, but the individual molecules are not -- some P is made into S and some S into P all the time. But the rates balance out.) It is a closed system; nothing is coming in from the outside or leaving.',NULL,1651,2,1),(###-10-11&&

1423    Q: Why does the answer to problem 5-11 show a mitochondrial inner membrane with only one lipid layer instead of a bilayer

1423    A: All biological membranes are lipid BI-layers. There is now way that one whole surface of hydrophobic fatty acid tails will just sit there in water without:\r\n\r\n<p>\r\n1) binding to itself, forming a spherical \"vesicle,\" or 2)  Binding to another MONO-layer sheet to form a bilayer.\r\nIt is the second that occurs durning membrane biosythensis in  cells. The first can be effected by experimentalists in a test tubes under the appropriate conditions  (perhaps low lipid concentrations?) ',NULL,1652,2,1),(###-10-02&&

1424    Q: In problem 3-7 D, if its the case that B-C and C-D are blocked, does introducing D to the growing material solve the problem\r\n(assuming C in not essential to anything but for synthesizing D)?  &&

1424    A: In most cases, mutants have only one mutation. If you go with that assumption, then only 1 enzyme is missing and one step is blocked. If both steps are blocked, providing D will still allow growth.',NULL,1653,2,1),(###-10-13&&

1425    Q: Suppose a poison blocks the electron transport chain and the mitochondria\'s NAD+ supplies are stuck as NADH and the Krebs shuts down. If an organism can\'t live without oxygen, but can live on 2 ATP per glucose, can the organism survive from the NADH that are produced by glycolysis

1425    A: This all depends on whether the cell can sense the presence of the poison and turn off respiration, sticking to fermentation/glycolysis. If the pyruvate is metabolized to acetate etc., then the pyruvate  is not available to reoxidize the NADH from glycolysis, and the cell will be stuck. If the cell can use the pyruvate to make lactate or ethanol, and prevent the pyruvate from entering Krebs, the cell will be okay. As far as I know, most cells cannot respond in this way. If they are making the enzymes for oxidative metabolism, and you add such a poison, they are stuck.',NULL,1654,2,1),(###-10-25&&

1426    Q: In problem 5-7 (C), I\r\nunderstand that in the absence of air glucose produces 2 ATPs, but\r\nhow does glutamic acid produces more than this

1426    A: Glutamate can enter metabolism at one point on the KC after it is first divested of its N atom via the oxidative demaination reaction described in lec. 9.  The result is aKG + ammonia, and the aKG is the same as the aKG one finds in the KC, normally in small amounts regenerated by the cyclic nature of the, er,  cycle.  Now we have lots of aKG comiing in from glutamate. The oxidative deamination reaction itself generates NADH (see handout) and that\'s worth 2 ATPs right there, which is already more than glucose can do in the absence of air. But furthermore, the aKG can now proceed through more reactions of the KC, why not, generating 2 more NADHs and one FADH2 along the way to ... OA. The OA has nowhere to go, as there is no acetyl-CoA to condense with to give citrate as it does when glucose is present. But here we have only glutamate, whose path we have just traced and there is no acetate or pyruvate or acetyl-CoA in sight. So OA would be the expected end product here, and it is simply excreted along with the ammonia.  3 NADs + 1 FAD have been reduced. Their re-oxidation in the ETC will yield $3 \times 3 + 1 \times 2 = 11$ ATPs by oxphos plus 1 ATP as GTP by substrate level phosphorylation in the GTP step of the KC. So grand total is 12, it seems.',NULL,1655,2,1),(###-10-29&&

1427    Q: If the F1 subunit of ATP synthetase is prevented from distorting by a drug or a mutation that changes an amino acid, could the Fo continue to rotate

1427    A: No, the C rotation and the alpha-beta conformational changes are obligatorily coupled. The gamma  subunit must be held very tightly by the C-subunts; otherwise how could it force the gamma to distort the alpha-betas?  Similarly, each alpha-beta must be deeply engaged with each of the 3 gamma lobes it can bind; otherwise, there would be no force behind the gamma engagement to push the ADPand Pi together or to spring the ATP loose.',NULL,1656,2,1),(###-11-02&&

1428    Q: Do the linked genes of an operon code for one enzyme, multiple enzymes for one pathway, or both

1428    A: Could be either of the above, or some combo. Usually there is more than one enzyme involved in any particular pathway or process, but one or more of those enzymes could have multiple subunits (polypeptides).',NULL,1657,3,1),(###-11-02&&

1429    Q: Becker says that peptide bond formation does not require \"nonribosomal protein facotrs.\"\r\nWhat does that mean? Is he referring to enzymes

1429    A: He means that the enzyme (really a ribozyme) for catalyzing formation of the peptide bond is part of the ribosome. For starting and stopping growth of peptide chains, and moving the mRNA relative to the ribosome, you need proteins that are not part of the ribosome that are called initiation factors, elongaton factors, etc., in addition to the ribosome.  You don\'t need any of those extra factors to form the peptide bond.',NULL,1658,3,1),(###-11-07&&

1430    Q: How quickly will a cell culture lose the plasmid if there is no selection\r\nfor keeping it

1430    A: The error rate is not high, so it takes many generations before there is signif. loss of plasmids (unless there is strong selection against having them). Bacteria grow so quickly that the passage of many generations takes a short time, especially compared to multicellular organisms. So it takes a lot of generations but a relatively short amount of time compared to our generation time.',NULL,1659,3,1),(###-11-14&&

1431    Q: How do plasmids initially acquire\r\nthe gene for drug resistance

1431    A: To start, some cell somewhere has a mutation in a gene on its chromosome, so that the cell becomes drug resistant. (The enzyme for degradation of drug gets better at it, protein for uptake fails to take up drug into cell, etc.) Plasmid in this cell integrates into chromosome and then crosses out, picking up a piece of bacterial DNA -- one carrying the altered genes that cause Drug resistance. This plasmid then passes a copy of itself to another cell and so on.',NULL,1660,3,1),(###-11-17&&

1432    Q: RFLPs are used in forensics because restriction enzymes will cut up a person\'s DNA into characteristically-sized fragments. \r\nBut because there are so many different DNA fragment sizes and they all run in a blur on a gel, how do we use them to distinguish between individuals

1432    A: The trick is having a probe that allows you to figure out which size pieces came from a particular section of the DNA. The overall DNA is a blur, but the bands in the blur that hybridize to a particular probe are clear. ',NULL,1661,3,1),(###-11-18&&

1433    Q: In an Hfr,\r\ncopying of the chromosome starts in the middle of the integrated F\r\nand goes in one direction. Does this mean that replication isn\'t bidirectional in this case? Is there still an origin? How does the cell know whether it should preform bi-directional replication for binary fission, and uni-directional relication in\r\nthe case of conjugation

1433    A: The mechanism of DNA replication during conjugation is different than the usual mechanism used for DNA replication. I think the origin is the same in both cases, although I\'m not sure. I assume that contact with an F- triggers the unidirectional type of replication and transfer over the mating bridge.',NULL,1662,3,1),(###-11-20&&

1434    Q: In problem 13-4(A), how would you know that the plasmid has a copy of the gene by the blotting/probing method? Couldn\'t you also use EatII-->add plasmids to bacteria-->grow on drug-

plate-->blot onto drug+ plates--> lyse cells from colonies that didn\'t grow-->electrophorese (to account for colonies that recived no plasmid at all)

1434    A: If you use Eat II, you will ruin the drug resistance gene. You can test indirectly for lack of drug resistance, but the idea is to select directly for drug resistance (and then check later for lack of insert\r\n-- you can\'t use the lack of drug resistance procedure described in class.) It\'s easier to select cells that got plasmids and screen out those that didn\'t get inserts than to do it as you suggest.',NULL,1663,3,1),(###-11-20&&

1435    Q: How can you ensure that an insert will be placed between a\r\nselectable marker? Isn\'t it possible that the piece of DNA that you\r\nare trying to clone will insert itself into a region that cannot be\r\nselected for

1435    A: You need matching sticky ends. The trick is to have a site for the restriction enzyme in a selectable marker gene. Either you get it by good luck or you use genetic engineering to make a deliberate change in the base sequence of the marker that doesn\'t ruin function but provides the restriction site.',NULL,1664,3,1),(###-11-25&&

1436    Q: We learned that there is a different repressor protein for each operon. For example, with a lac operon on the chromosome, does the only repressor that will fit into the lac operator have to have been created by the repressor protein sequence on the chromosome? Or will any lac repressor protein from any plasmid fit into any lac operator

1436    A: All lac operons have the same repressor. But usually there is only one per cell. When I said \"there is a different repressor protein for each operon,\" I meant a different repressor for each type of operon -- one for the lac operon, a different one for the trp operon, etc. Usually the cell has only one of each type of operon.',NULL,1665,3,1),(###-11-25&&

1437    Q: Do deletions or insertions always generate a stop codon

1437    A: No. But they often do if they are not a multiple of 3.',NULL,1666,3,1),(###-01-19&&

1438    Q: Are proteins made on rough ER ribosomes always transported to and used in the\r\nER

1438    A: They are used in some part of EMS, not necessarily the ER, or secreted by the cell. We will go over the process in some detail in a few lectures.',NULL,1667,1,2),(###-01-19&&

1439    Q: When adding substrate to an enzyme, how can you be sure that the product\r\nwill be insoluble and thus precipitate

1439    A: Products are usually soluble. The trick is to find just the right chemical that will produce an insoluble product. It isn\'t always doable, which is why only some enzymes can be localized this way.',NULL,1668,1,2),(###-01-19&&

1440    Q: Do the membranes of the peroxisomes and\r\nlysomes consist of one bilayer (as in one\r\n\"sandwich\") or does the membrane consist of one half of a sandwich,\r\nlike a unilayer instead of a bilayer

1440    A: A membrane is ALWAYS a bilayer, as in one sandwich, not a 1/2 sandwich.  A double membrane, such as the one that surrounds the nucleus is TWO bilayers.',NULL,1669,1,2),(###-01-21&&

1441    Q: We said that each antibody has two variable parts and two constant\r\nparts. Obviously the two constant parts are always identical. Are\r\nthe variable parts always identical to each other or can one\r\nantibody have two different variable parts

1441    A: The two variable parts on one antibody molecule are always the same. (Some antibodies have multiples of the basic structure, so they have more than 2 grabbers -- multiples of 2.) If you want to look at a better picture, look in your textbook or on line. We\'ll discuss more on this at the end of the term.',NULL,1670,4,2),(###-01-29&&

1442    Q: The answer to problem 2-4 (B) states that graph 3 does not indicate that\r\nthe two concentrations are equal at equilibrium. Doesn\'t that [ser] in = [ser] out imply that the concentrations are equal

1442    A: The fact that the graph reaches a plateau does not mean conc. on both sides are equal. (It just means the rate in = rate out.) You figure out that conc. are equal on both sides from other factors.',NULL,1671,1,2),(###-01-27&&

1443    Q: For channel proteins, why don\'t the substrate concentrations inside and outside the cell lead to a constant rate of uptake within a quick period of time

1443    A: We are always looking at the initial rate of uptake for a fixed conc. of solute. Not at steady state concentrations.',NULL,1672,1,2),(###-01-29&&

1444    Q: Are the connections between gap junctions of adjoining cells fleeting or permanent? Do they involve covalent bond or weak interactions

1444    A: Permanent as far as I know; weak forces are usually responsible for all protein-protein interactions.',NULL,1673,1,2),(###-01-29&&

1445    Q: Are connexons bound to microfilaments, microtubules, or intermediate filaments

1445    A: Exactly how they are connected, I don\'t know. But the freeze fracture pictures imply some indirect connection. There is no direct connection -- not like adhesive junctions.',NULL,1674,1,2),(###-01-30&&

1446    Q: Is it right that for facilitated diffusion through a channel, Keq is NOT equal to 1,\r\nand the delta G is NOT equal to 0, as where the situation is the reverse for\r\nfacilitated diffusion using a carrier protein

1446    A: For a NEUTRAL molecule, Keq is equal to 1 and delta G zero is zero. It\'s different for an ion. For neutral molecules the situation is the same for facilitated diffusion using a channel or carrier.',NULL,1675,1,2),(###-01-30&&

1447    Q: Is it right that glucose enters erythrocytes with the help of the GLUT1\r\ntransporter, but in the case of epithelial cells in one\'s intestine,\r\nit\'s taken up from the environment into the cells with the help of the\r\nGlucose/Na+ co-transporter

1447    A: Yes. It\'s down the gradient into RBC but up the gradient into epithelial cells around the intestine.',NULL,1676,1,2),(###-02-01&&

1448    Q: Why does the clathrin \"coat\" of a vesicle get\r\nlost so quickly once the vesicle enters the cytoplasm

1448    A: The only point of the coat (clathrin or other coat protein) is to help the membrane curve into a vesicle. Once that is done, the coat can be ditched.',NULL,1677,1,2),(###-02-01&&

1449    Q: And how does the clathrin know how to get back to the membrane once\r\nit falls off the vesicle

1449    A: I don\'t know the fine points, but I think it is known. There is an adapter protein that sticks to the membrane on one side and clathrin on the other.\r\nSee problem 3-13.',NULL,1678,1,2),(###-02-04&&

1450    Q: After EGF receptors are degraded, how does the cell know to put receptors back on the membrane? We said that the cell will produce new receptors when it needs to get the signal again. Are there other receptors that tell the cell that it should grow

1450    A: The control of growth is very complex. Probably some step in growth that signals that the cells have divided stimulates production of new receptors.',NULL,1679,1,2),(###-02-04&&

1451    Q: About radioactive labeling. I understand that we are able to determine intervals between benchmark organelles, and observe, say, the time to go from precursor/radioactive AA, to protein, through organelles, and then outside the cell. Can these techniques be used to \"clock\" the speeds of transport? If these proteins are flowing through the cytoplasm, would it be possible to determine how fast proteins are traveling between two given organelles

1451    A: Yes, you can get speeds. But most proteins travel inside vesicles, not by themselves. So many of the factors you are thinking of may not be relevant.',NULL,1680,1,2),(###-09-20&&

1452    Q: 1-11 B Part II: Which bonds must be broken in order to get a phosphate group?\r\n&&

1452    A: If you add HOHs to bonds 6 and 7, and figuring you add the OHs to the P\r\nand the Hs to the O, you\'d get HO-P-OH in place of 6 and 7 (omitting for\r\na moment the other bonds already on P).\r\n\r\nSo the freed phosphate would  be     \r\nO                                                    ||\r\n HO-P-OH\r\n                                            |\r\n                                                                  O-\r\nThe status of the H on the newly formed OH\'s would be determined by the\r\npH, as for any acid. At pH 7, two of the 3 hydroxyl\'s would be almost\r\nentirely ionized, and the third one about half ionized. (All 3 are\r\nequivalenrt with regard to position.  That is, once two protons lave\r\nbeen lost, the thrid one is more reluctant to leave.)   So the more\r\naccurate structure would for the freed phosphate might be:\r\n\r\n                                        O\r\n ||\r\n                              HO-P-O-\r\n                                                                  |\r\n O-\r\nBut we can also just write :\r\n\r\n                                        O\r\n ||\r\n                              HO-P-OH\r\n                                                                  |\r\n OH\r\nin many contexts, as long as we are aware that the phosphate would have\r\na net charge of  ~ -2.5 at pH7.\r\nIn most cases (e.g., to understand phospholipid behavior)  it is\r\nsufficient to recognize that the group is highly negatively charged at pH7.\r\n',NULL,1681,1,1),(###-09-23&&

1453   Q: 2-15 Would the molecular weight (MW) of a non-spherical molecule be underestimated or overestimated during ultra centrifugation? Gel filtration

1453   A: In ultracentrifugation, non-spherical molecules will travel more slowly due to inordinate frictional drag. In ultracentrifugation,lower MW molecules sediment more slowly because the have less mass acted on by centrifugal force. So the non-spherical molecule looks smaller than it really is, due to its slow sedimentation. \r\n\r\nIn gel filtration, spherical molecules more easily enter the sephasdex beads so non-spherical molecules would come out faster resulting in a higher MW.',NULL,1682,1,1),('0000-00-00&&

1454   Q: A. Problem 2-12 asks which separation technique(s) would reveal the presence of native protein in a sample treated with \"Compound C,\" which only disrupts quaternary structure. Is it possible for a protein\'s quaternary structure NOT to involve disulfide bonds, while its tertiary structure DOES?\r\n\r\nB. In the case of complete quaternary disruption of this hypothetical protein, could tertiary disulfide bonds in the subunits remain? How is this possible?\r\n\r\nC. How do subunits with intact disulfide bonds separate from other proteins in PAGE + SDS assay

1454   A: A. Yes. Indeed, this is a common situation.\r\n\r\nB. Yes. Let\'s say the weak bonds holding the subunits together are very weak indeed, are mostly H-bonds that are sensitive to urea so are disrupted at a relatively low concentrations of the chaotropic agent.  The subunits themselves on the other hand may be held in the tertiary structure mainly by relatively strong hydrophobic forces. Disulfides need not even be involved in this picture but if they were present as intrachain covalent bonds they would further the resistance to disruption of the tertiary structure while not contributing to the quaternary structure. \r\n\r\nC. Disulfides would not be broken by SDS, only by mercaptoethanol. Migration in SDS+PAGE might be affected, as the cross-linked polypeptide subunits would be more compact than a random coil capable of full extension, so they would appear to have a lower MW than the actually have.',NULL,1683,1,1),('0000-00-00&&

1455   Q: At pH 7, both the amine and carboxyl end become positively and negatively charged, respectively.  Do the protonated amine/deprotonated carboxyl form an ionic bond during the formation of alpha and/or beta sheets?\r\n&&

1455   A: Generally, no. They could for an ionic bond, but the chance that they happen to lie next to each other is small. In any case, such an\r\noccurrence would not be related to secondary structure.\r\n',NULL,1684,1,1),('0000-00-00&&

1456   Q: How do inorganic solvents denature proteins

1456   A: If the protein is placed in an inorganic solvent as opposed to water, then there are no longer any hydrophobic forces possible. Without this important driving force, most proteins unravel.  So it is the absence of water that is important.',NULL,1685,1,1),('0000-00-00&&

1457   Q: Why are the amino residues in the beta sheet pleated? Why is there the ups and downs we see in the pictures

1457   A: Many of the C-C bonds along the backbone are tetrahedral, so the chain is crooked much like the straight chain of glucose was shown to be in the model.',NULL,1686,1,1),('0000-00-00&&

1458　Q: So in problem set 2, question 14, the question asks how it is possible for a protein to regain its structure after it has been cleaved in two. The answer said that without the urea to disrupt the bonds that make up the secondary and tertiary structure the protein would conform to the proper conformation. I said however that in a protein, all of the necessary information to conform was in the primary structure and any disruption of the primary structure would distort the protein. The text book and in class we said that primary structure was where all the necessary information was and a disruption of that would mess up the rest of the conformation

1458　A: Yet the question states that enzymatic activity was restored, as was the molecular weight. If your answer explained this somewhat unexpected result, then you\'d be OK, even if your answer included a characterization that stated that the restored enzymes is not identical to the original (it is not, as it now consists of two chains, not one). The overall conformation was restored, as the enzyme regained catalytic activity. The 2 chains were associated with each other (much as in the original) since the MW returned. The focus here is not on the disruption (expected) but in the restoration (a surprise). The problem presents a new situation, one you have not encountered before, to test your understanding of the principles (not the \"definitions\") involved.',NULL,1687,1,1),('0000-00-00&&

1459　Q: In lecture you said that gel electrophoresis could only be used to separate proteins of the same charge. We put the proteins near one end so if we put it near the anode, anions would migrate right off the gel. However, if the proteins started off in the middle though, could you separate proteins of different charge

1459　A: Yes. This strategy can be implemented by using a horizontal gel electrophoresis apparatus with a well in the middle. Vertical gels make it easier to load and to get sharp bands.',NULL,1688,1,1),('0000-00-00&&

1460　Q: The lecture notes state: If you calibrate the column by noting the behavior of spherical proteins of known size, you can determine the MW of your protein by comparison, if it is also spherical. If it is not spherical it will appear to have a higher molecular weight than its true MW (imagine a pancake being excluded from a channel while a sphere of the same MW gets in).  However, in problem 2-15 regarding ultra centrifugation we found that if it is not spherical you will UNDERestimate the MW. &&

1460　A: Ultracentrifugation is a different kettle of fish in this regard.  In ultracentrifugation, non-spherical molecules will travel more slowly due to inordinate frictional drag. In ultracentrifugation, lower MW molecules sediment more slowly because the have less mass acted on by centrifugal force. So the non-spherical molecule looks smaller than it really is, due to its slow sedimentation. In the gel filtration case the spherical molecules enter the Sephadex beads more easily than the non-spherical molecules of the same MW, so they come out sooner making one think they has a high MW.',NULL,1689,1,1),(###-09-28&&

1461　Q: Are two enzymes needed to break up glycogen completely down to glucose (one to break the (1-4) alpha linkage and one to break the (1-6) alpha linkage)? Are the same two enzymes that break down glycogen also responsible for reforming the bonds? \r\n&&

1461　A: Yes. No.',NULL,1690,1,1),(###-09-28&&

1462　Q: In glucose ring formation, is it the forces from H-bonding in the surrounding water molecules interacting with the polarity of the glucose that create this thermal motion and promote \"bumping\"

1462    A: The thermal motion is simply related to the temperature and not to any intermolecular bonds. At 0 deg the motion would be less, the molecules move less, the intramolecular motion is less, so the chance of a productive collision is less. All just the opposite at higher temperature.   At body temperature, there is plenty of molecular motion, so in the course of this motion the C1 O and the C5 will meet.  Due to the length of the tether between them in a single molecule, it is the C5 oxygen that is most likely to collide with the C1.  Water is a bystander in this meeting, providing the medium for all this motion but it does not play a key role.',NULL,1691,1,1),(###-09-28&&

1463    Q: I found an answer on the Q&A database explaining that an increase in temperature leads to stronger hydrophobic forces because of an increase in entropy for the freed up \r\nwater molecules. This makes sense, but I\'d like to ask an additional question. At some point, will the additional molecular motion and increase in hydrophobic force strength be countered by an increase in nuclear-nuclear repulsion from the grouped non-water molecules?\r\n\r\nTHE RESPONSE OF SUCH SYSTEMS TO TEMPERATURE TURNS OUT TO BE MORE COMPLICATED THAN I IMPLIED AND IS STILL AN AREA OF SOME CONTROVERSY. THE EXPERIMENT ALLUDED TO (FROM 1959) IS APPARENTLY TRUE ONLY OVER A \r\nRESTRICTED TEMPARTURE RANGE, BEYOND WHICH OTHER FACTORS BEGIN TO play a role or even dminate.\r\n\r\n\r\nI\'m still confused as to why \"apolar groups will tend to associate with other apolar groups in aqueous solutions\".  &&

1463    A: Here\'s someone else\'s explanation:\r\n\r\nAnd, from http://www.cmbi.kun.nl/gvteach/alg/infopages/hydrophobicity.shtml:\r\n\"So, what is hydrophobicity? Many people describe it indirectly by saying things like \"Hydrophobic residues attract each other via \r\nhydrophobic forces\". Although this is a handy way to think about things, it is not correct. There doesn\'t exist such a thing as an \'attractive hydrophobic force\'. All atoms attract each other via the Van der Waals \r\ninteraction. The force associated with this Van der Waals interaction is very weak at large interatomic distances and still weak when the atoms get closer.\r\n\r\nSo why do hydrophobic atoms want to sit close to each other inside a protein? For that we have to think again about the name of this effect: hydrophobicity. Hydrophobicity does not mean \"love for hydrophobic atoms\", but \"fear of water\". And that also holds the other way around. Water doesn\'t like hydrophobic atoms.\r\n\r\nA water molecule that swims around in water at room or body temperature has on average about 3.5 hydrogen bonds, and it has nearly 6 degrees of freedom. Degrees of freedom (like rotational freedom and translational freedom) add to the entropy and the hydrogen bonds add to the enthalpy. \r\nSo in:\r\n\r\ndG = dH - TdS\r\n\r\nthe hydrogen bonds add to dH and the freedom adds to dS.\r\n\r\nNow imagine a water sitting against the side of the benzene ring in phenylalanine. This water can form only about two hydrogen bonds, and its freedom is also severely restricted. This water molecule compares unfavourably with one in bulk water.\"\r\n\r\n\r\nIs any one way of drawing a sugar preferred (Haworth vs. chair vs. Fisher)? \r\n\r\nNo. the use of a particular depiction depends on the context. If we are considering 3-D structure, the chair conformation is almost essential.  \r\n\r\nWhy is the carbonyl carbon in glucose symmetric before cyclization since the groups attached to it are =O, -H, and -C?\r\n\r\nYou can slice the aldehyde group into two halves by placing a plane through the center of the molecule:\r\n\r\n  O\r\nR-C-Q\r\n\r\n\r\nWith the C=O group, this arrangement of atoms is planar about the central carbon, no longer tetrahedral. Imagine the atoms resting on the \r\nplane of screen. Now slip a plane through them parallel to the screen but half an atom\'s width in front of the screen, so that it is cutting through the middle of all the resting atoms. The parts of the molecule \r\non  one side of the plane are the same as those on the other side: The molecule has a symmetry about it, a plane of symmetry can be passed through it. Such a plane is not possible with a tetrahedral carbon bound \r\nto four different

groups, no matter how it is resting on the screen.\r\n\r\nYou drew maltose and cellobiose disaccharides with the second glucose molecule indicated as alpha or beta glucose. Is the reason the glucose molecule was indicated\r\n\r\nthis way because one end of the molecule is free to alternate between ring and straight form and can switch from alpha to beta?\r\n\r\nGlucose is found mainly in one of the ring forms in solution. I had to write something out there on the right (arbitrary choice).\r\n\r\nI was confused into thinking that each amino acid has its own characteristic and that we should have them memorized for the exams.\r\n\r\nMemorize the functional groups. Look on the side chain for functional groups. Then draw your conclusion about the character of the side chain.\r\n',NULL,1692,1,1),(###-09-28&&

1464    Q: How could Anfinsen have determined where in the ultracentrifuge tube the denatured protein was located, since he could not assay here for\r\nenzymatic activity

1464    A: You can check the tube for where the protein is (how far and therefore how fast it has migrated) by several methods that do not depend on enzymatic activity. For instance, a color reagent that reacts with proteins ( as was seen in the lecture (ppt) photo of the gel after SDS-PAGE) or even amino groups (as was used to visualize the spots of the amino acids in paper chromatography)  or by its absorbance of\r\nultraviolet light (not discussed).',NULL,1693,1,1),(###-10-01&&

1465    Q: Regarding Recitation Problem Set 2, number 3B, I conceptually  understand that only the D-isomers of glucose would be found in the  hydrolysate immediately after hydrolysis. However, could the D-isomer rings  convert to straight chain glucose, allowing rotation around the C-C bonds to change into other 6 carbon sugars (including L-glucose)? In other words, what  stabilizes glucose in its straight chain form

1465    A: Rotation around the C-C bonds in the straight chain does occur, but this rotation between 2 C\'s does not change the position of the four groups attached to each of the carbons. If you remember the models of the D and L amino acids I showed in class, you may see that no matter how I moved these around in space, there wa no way I could get them to be superimposable. Thus glucose and galactose will never be superimposable no matter how much rotation and swinging around you do; they are distinct molecules, stereoisomers with different English names. .D- and L-glucose are similarly non-superimposable molecules, except they differ at all the asymmetric carbons, more than the difference between glucose and galactose (only one C different) LAC',NULL,1694,1,1),(###-10-01&&

1466    Q: I\'m struggling with the concept of an assay.  Can you expand at all? I think I understand the concept of assaying after gel filtration, but in problme 2-6(c) it spoke of assaying for catalytic activity of an enzyme--I\'m a little lost in how the same process can do both.&&

1466    A: I like this definition from the American Heritage Science Dictionary:\r\nA quantitative determination of the amount of a given substance in a particular sample.\r\n\r\nIt is very general, in that the method used for the "determination\" can be very different indeed. We can assay the amount of E. coli that has grown up in 100 ml. of culture medium in a flask by:\r\n- Centrifuging them down, letting them dry and weighing (dry weight)\r\n- Taking out a measured volume, diluting it in a measured way, and counting the number of bacteria in a new measured volume (a few microliters).\r\n- Using a reagent that produced a color in the presence of magnesium \r\n(e.g., flame photometry) in a measured amount of washed bacteria, and knowing the average amount of Mg per E. coli cell, back calculate the number of cells that were present.\r\n\r\nSimilarly, the amount of beta-galactosidase enzyme in a sample of E. \r\ncoli could by assayed by measuring the rate of hydrolysis of lactose in \r\na test tube containing a measured amount of cell cytoplasm (cell \"extract\")  or by

purifying the protein and then weighing it (difficult but theoretically possible) or reacting the purified protein with a reagent that turns blue in the presence of amino groups and measuring the amount of blue color produced.\r\n\r\nMeasuring the rate of lactose hydrolysis in an extract made from a measured amount of the culture.',NULL,1695,1,1),(###-10-05&&

1467    Q: Lec 8: 1a) If the cell uses the method of \"pulling\" to get a reaction going in the right direction, how does the unfavorable reaction get started

1467    A: The unfavorable reaction in question will produce a bit of product no matter what the delta Go is (none are + or - infinity), For instance, if it +7 (like ATP formation)  then at equilibrium we would have about 1/100,000 product compared to reactants.  This tiny amount of product then can react in the next step (the next step \"downstream\"), reducing the 1/100,000 to much less than that (say, 1/1,000,000). The upstream unfavorable reaction will then produce more of its product to maintain the 1/100,000 ratio.  This process continues, such that there is a constant stream of material through the unfavorable reaction, the product being constantly removed by the favorable downstream reaction.',NULL,1696,1,1),(###-10-05&&

1468    Q: 2) In the beginning of Krebs when pyruvic acid is changed into acetyl-CoA, where do the 2 H\'s come from that change NAD from NADH2? I see one H comes from the carboxyl group but where does the other come from

1468    A: In oxidations it is the electrons that matter, protons can usually be found in water to keep things balanced. Moreover, there aren\'t really 2 protons to find in NADH2, because NADH2 is a moisnomer, as it really only has one new proton to go with the 2 electrons it acquired. (NADH); the use of the NADH2 name is only to remind us that there were 2 electrons transferred.  Anyway, in this case we can look to the CoA molecule (lec. 8 graphic 33), where acetyl-CoA is pictured. However, the reactant in the entrance reaction to the Krebs Cycle is free CoA (no acetate yet) and ends in an -SH group, so there is a proton we can use.  In general I personally don\'t find it a useful exercise to spend time looking for the protons, unless you are in a chemistry course.\r\n',NULL,1697,1,1),(###-10-10&&

1469    Q: In the glycolytic pathway diagram from class, 1,3 DiPGA has 2 phosphate groups bonded to it, but only one of these is a high-energy bond. Why is one bond high-energy, but not the other? Also, when the molecule is converted from 3-PGA to 2-PGA to phosphoenol-pyruvic acid, the phosphate group moves, and the bond holding the phosphate group goes from a non-high-energy bond to a high-energy bond. What exactly is causing the bond in this case to go from low to high-energy

1469    A: The high energy bond in 1,3 diPGA involves the hydrolysis of an acid anhydride (acid-acid), whereas the non-high-energy bond is a phosphate ester (acid-alcohol) . We rationalized the cleavageof the terminal phosphate of ATP as being high-energy since the cleavage released an electrical strain. In the cleavge of 1,3 diPGA, the products of the hydrolysis are a free charged phosphate (true in both cases), and a carboxylic acid. The carboxylic acid can exist in two forms, with the electrons associates with either oxygen (tautomers). The electrons are really delocalized around both oxygens of the carboxyl group. This delocalization is a favored state (increased entropy?), so the products are greatly favored over the reactants in which such delocalization does not occur.  There is a similar delocalization of electrons possible when a hydroxyl lies next to a C=C double bond (pi electrons in the C=C double bond can migrate to the electronegative O to give a CH3 - C = O  <--> CH2=C-OH   molecule (keto=enol tautomerism). All this is way too much chemirstry for us here.  To read more detail about each of the glycolytic steps see: http://web.virginia.edu/Heidi/chapter19/chp19.htm,  and reactions 9 and 10, in particular, for the latter case.',NULL,1698,2,1),(###-10-19&&

1470   Q: Referring to the overall structure of the ribosome, can we conclude that rRNA and the ribosomal proteins exist in quaternary structure to form the ribosome? Is this the same in prokaryotes and eukaryotes

1470   A: I would call it a 5th degree of structure. The proteins have quaternary structure, and then they combine with the RNA, but you have the right idea. The structures are similar, but not identical in prokaryotes and eukaryotes.',NULL,1699,2,1),(###-10-19&&

1471   Q: In terms of function, does the rRNA do all the work while the protein stabilizes the structure, or is the ribosomal protein also involved in function

1471   A: Both RNA and protein are involved in both structure and function.',NULL,1700,2,1),(###-10-21&&

1472   Q: On Handout 13 A, Length of product, it indicates that in DNA synthesis, the new DNA is as long as the template, but I am wondering how that is possible for eukaryotes, taking into consideration that on the lagging strand, we are always losing a section due to the last primer. They would only or maybe be the same length if telomerase comes in and forms a telomere. When putting this on the sheet was it referring to a situation where telomerase is concerned

1472   A:     Linear DNA molecules do get slightly shorter with each replication. However, we are ignoring the shortening due to primer (for this discussion), since it\'s length is trivial compared to the length of the entire DNA molecule. We are trying to make the point that DNA replication goes from one end of the molecule to the other, copying both sides, but RNA transcription starts at multiple points, copies one side, and ends at multiple points.\r\n     Another point -- for now, we are talking about prokaryotes who have circular DNA molecules, and don\'t have a telomere problem, even though I wrote a short linear molecule on the handout (for convenience and clarity).',NULL,1701,2,1),(###-10-21&&

1473   Q: I have a question about the convention of drawing nucleotides generally with a phosphate on the 5\' end and an -OH group on the 3\' end. I understand that sometimes the phosphate is on the 3\' end, for instance. Is it convention to draw an XMP with the phosphate on the 5\' end

1473   A: As to nucleotides, it is customary to draw a phosphate the 5\' end and a OH on the 3\' end because that is the way they are usually found in cells. (That\'s what you get if you hydrolyze DNA that was injested.) However in labs, it is possible to end up with phosphates on the 3\' end or OH on the 5\' end. (Not sure it ever happens in living cells.)',NULL,1702,2,1),(###-10-21&&

1474   Q: In problem 4-15c, according to the explanation, pyruvate would be the only major product formed in the absence of oxygen, since NAD runs out. Why wouldn\'t you get lactate, as in fermaentation

1474   A: No, it\'s because NADH2 runs out ( =NADH; = \"reduced NAD\"). Since there\'s no constant source of NADH2, there\'s no substrate for the pyruvate dehydrogenase reaction that would produce lactate. There are no ATP ramifications for this non-conversion; you would just excrete pyruvate as the end-product rather than lactate. By supplying the\r\ndiphosphoglycerate as a foodstuff, you are supplying two phosphates that can be used to create ATP from ADP via the hydrolysis of hugh-energy bonds.',NULL,1703,2,1),(###-10-25&&

1475    Q: Does the word fermentation mean the whole process starting from glucose, or just the last steps that proceed from the end of glycolysis

1475    A: Fermentation = the whole pathway, glucose to lactate for example in E. coli; glucose to ethanol + CO2 in yeast (whether or not oxygen is present, actually)',NULL,1704,2,1),(###-10-26&&

1476    Q: How does primase know when to stop? How does primase know the primer it just created is the necessary length

1476    A: Presumably primase stops after a short distance, and whatever that distance is, is a property of the enzyme. Unlike regular RNA polymerase, the enzyme can\'t keep going indefinitely -- it falls off the DNA after 10-20 nucleotides have been put in.',NULL,1705,2,1),(###-10-26&&

1477    Q: Is the point of transamination to catabolize the proteins that we eat

1477    A: It is also used to synthesize amino acids, reversing things.',NULL,1706,2,1),(###-10-26&&

1478    Q: What about the other amino acids other than alanine? When we remove NH2 from  them, they do not resemble pyruvate because of their different side  chains. Is the process of transamination enough to get them into a form compatible with our metabolic cycles

1478    A: Most go through multiple transformation following a transamination that exchanges their NH2 for a C=O.  Transamination is usually (not always) the first step.  For instance tyrosine is transaminated and then goes through 5 more reactions before ending up as fumarte and acetate.',NULL,1707,2,1),('0000-00-00&&

1479    Q: Re: e-site in a ribosome. Must a tRNA be in this E-site for the ribosome to move down the mRNA? Is it \"necessary\"? What does it accomplish

1479    A: It\'s actually the other way around -- the movement of the ribosome kicks the empty tRNA into the E site and moves the tRNA with the chain into the P site. The addition of AA-tRNA leads to changes in shape that bump the empty tRNA out of the E site. You can design a system without an E site, but that is the way it happens to be!',NULL,1708,3,1),('0000-00-00&&

1480    Q: In problem set #7, question 19, do you need a different tRNAsynthetase for each tRNA or can a single tRNA synthetase connect an AA to all of its tRNAs

1480    A: You definitely need a different synthetase for each AA - cognate tRNA pair. Each synthetase is specific for one AA and it\'s matching tRNA.',NULL,1709,3,1),(###-11-26&&

1481    Q: Must a tRNA be in this E-site for the ribosome to move down the mRNA? Is it \"necessary\" and what does it accomplish

1481    A: It\'s actually the other way around -- the movement of the ribosome kicks the empty tRNA into the E site and moves the tRNA with the chain into the P site. The addition of AA-tRNA leads to changes in shape that bump the empty tRNA out of the E site. You can design a system without an E site, but that is the way it happens to be!',NULL,1710,3,1),(###-11-26&&

1482    Q: In problem set #7, question 19, do you need a different tRNA synthetase for each tRNA or can a single tRNA synthetase connect an AA to all of its tRNAs

1482    A: You definitely need a different synthetase for each AA - cognate tRNA pair. Each synthetase is specific for one AA and it\'s matching tRNA.',NULL,1711,3,1),(###-11-26&&

1483    Q: Why does the Feedback inhibitor in 12-2(D) slow the rate of synthesis of mRNA from HIS enzyme-genes?  Feedback inhibitors for HISTIDINE pathway inhibit enzymatic activity. They do not decrease the concentration of HIStidine enzymes (as I understand). So how can F.B.Is affect transcription or translation rates of these enzymes

1483    A: The answer means that both the deletion mutant and the normal will respond to fbi by his. But the mutant will not be subject to repression by his. The answer does not mean to imply that his is slowing transcription by acting as a feedback inhibitor. It is slowing transcription (in the normal) by acting as a co-repressor. But it can\'t act as a co-repressor in the deletion mutant.\r\n',NULL,1712,3,1),(###-11-26&&

1484    Q: I just wanted to clarify that the final is NOT cumulative, correct? The material on the final will just cover everything after EXAM #3 right

1484    A: All the exams are cumulative in the sense that you are expected to know about\r\nthe material covered earlier.  E.g., so we can mention the Km of RNA polymerase and you should know what it means. But the emphasis is always on the material since the last exam. The final will be just exam #4, concentrating on the material covered after the topics of exam 3.  One difference is that you will have 3 hours to complete exam 4, even though it will be no longer than the other exams.',NULL,1713,4,1),(###-11-26&&

1485    Q: Q 13R-4B: But by the explanation for the problem, I\'m guessing I have been reading lec 17 incorrectly. I thought it referred specifically to terminology, saying that only a substitution can result in a loss or addition of a restriction site. But clearly, insertions or deletions can also result in a change. Is that correct

1485    A: Yes. Insertions, deletions OR substitutions can cause an addition or loss of a restriction site.\r\n',NULL,1714,4,1),(###-11-26&&

1486    Q: In the lecture 17 notes it says: The \"addition\" or \"loss\" of a restriction site does not mean the insertion or deletion of bases. An \"addition\" means a change in base sequence so a particular stretch of DNA is now recognized by a particular restriction enzyme; a loss means a change so that the DNA is no longer recognized by the restriction enzyme\".&&

1486    A: It should say the \'addition\' or \'loss\' does not NECESSARILY mean the insertion......An \'addition\' means ANY change in base sequence.',NULL,1715,4,1),(###-11-26&&

1487    Q: Q 12-4 Part A: It says that the mutation in the constitutive lac operon must be from a defect in the repressor protein. I thought, since genes for repressor proteins are not part of the operon for the gene they repress, that the cell would not contain two copies of the gene for the repressor protein. If a cell has two of the same operon, should I also assume that it has two genes for the repressor protein

1487    A: Yes. The repressor gene is usually considered part of the operon. The gene just isn\'t always linked to the others (not next to it on the DNA).',NULL,1716,4,1),(###-11-26&&

1488    Q: Q 12-4 Part B: I don\'t understand how enzymes from a constitutive operon can affect an inducible operon.&&

1488    A: They don\'t. The statement means the constitutive operon remains constitutive and the inducible operon remains inducible. It means that the state of the regulator elements of the constitutive operon (not its enzymes) don\'t affect the regulation of the inducible one.',NULL,1717,4,1),(###-11-26&&

1489    Q: Q 13-13:\r\nA) cDNA contains only exons. Why does this mean it can only bind to exons?\r\nB)It contains at least one specific exon (for gene K).\r\nC)Why would it not then bind only to this one exon? Exons don\'t just bind to any old exon.&&

1489    A: A) It has no stretches complementary to introns.\r\nB) No. Gene K contains multiple exons, all needed to make peptide K. I think you have genes, peptides, and exons confused.\r\nC) The cDNA has stretches complementary to all the exons. See above.\r\n',NULL,1718,4,1),(###-11-26&&

1490    Q: If a single sister chromatid carries 1 copy of a gene, does this mean that haploid chromosomes always carry 1 chromatid (since haploids have only 1 copy of their genes) at the end of mitosis? Question 8-13, part D-1, made me wonder about this.&&

1490    A: It doesn\'t matter if cells are haploid or diploid. The chromosome still has two sister chromatids before mitosis and one after.',NULL,1719,4,1),(###-11-26&&

1491    Q: What are plasmids made of? We basically don\'t\r\ncare about the plasmid, only the insert

1491    A: Plasmids are made of DNA (and so are the inserts). What else could it be?',NULL,1720,4,1),(###-11-26&&

1492    Q: If the plasmid has a promotor of its own to transcribe (make mRNA of its DNA, that promotor is often used to transcribe/make mRNA of the insert

1492    A: A promotor works to start transcription, which then continues until the polymerase hits a transcriptional terminator. If the terminator is after the insert, the insert will be\r\ntranscribed too.',NULL,1721,4,1),(###-11-26&&

1493    Q: Say there is an hybrid plasmid in a bacteria which a euk. gene.\r\n\r\n1. What is the gene of the plasmid? Is it from prok. or euk? We seem to assume it is prok. gene.\r\n\r\n2. 13-14* B. In order for a euk. insert to be translated, does it always have to  be close the plasmid\'s promotor?\r\n&&

1493    A: 1. If it is a euk gene, it came from a euk.\r\n\r\n2. Yes',NULL,1722,4,1),(###-11-26&&

1494    Q: If there was no insert, all of the plasmid will replicate using the promoter/RNA Polymerase right?\r\n\r\nWhy, then, if there is an insert, that the transcription ends as soon as it has reached the end of the insert gene

1494    A: Promotors are for transcription, not replication. I think you are confusing RNA and DNA synthesis. There are terminators for transcription in bacteria, so not all would be transcribed.\r\n\r\n It doesn\'t have to end exactly there, but it ends somewhere. See above.',NULL,1723,4,1),(###-12-05&&

1495    Q: 1. In lecture 20, you described the haploid life cycle as the following: the zygote immediately undergoes meiosis to form haploid cells, or spores, that then grow by mitosis.  There are no germ cells. Do you mean that there are no GAMETES or do you mean that there is no DIPLOID PRECURSOR that gives rise to gametes..?  From googling the term \"germ cells\" it seems like one can loosely use the term to mean both.\r\n\r\n2. Is it correct for me to think that in the haploid life cycle, you DO get specialization of haploid cells to become gametes that then fuse to become the zygote which immediately undergoes meiosis?  So when you said there are no germ cells, did you mean no diploid precursors that DIRECTLY give rise to gametes by meiosis

1495    A: 1. Yes there are gametes, but I mean the second explanation, see below.\r\n\r\n2. Yes, exactly.',NULL,1724,4,1),('0000-00-00&&

1496    Q: If an individual is a carrier for sickle cell, the phenotype is not affected in the sense that the individual does not have the disease; however, the individual IS affected in the sense that his/her cells are a different shape and he/she is malaria resistant. We classify Sickle Cell as Recessive, but I don\'t see how a carrier is any different than a pink flower. I thought the conclusion was that in recessive conditions, the \"good\" gene covers for the bad. BB or Bb both give equally blue eyes. In incomplete dominance, the \"good\" gene only half covers for the bad and the individual is somewhere in between (be it pink or odd shaped blood cells).&&

1496    A: It all depends on how you define \'in-between.\' The carrier\'s cells do not sickle under the same conditions that make a homozygote sickle. (They only sickle under extreme conditions.) However SCD is the classic case that can be considered co-dominant, incompletely dominant, or recessive, depending on your point of view. It all depends on how you define the phenotype. If it is \"sickle cell disease\' or vs none, the condition is\r\nrecessive. If the phenotype is sickling or not, it depends on the oxygen conditions chosen whether hetero will sickle or not. (So you can classify the condition as either recessive or showing intermediate dominance.) If phenotype is defined by the type of hemoglobin made, the condition is codominant (heterozygotes make both HbA and HbS). If it\'s defined in terms of malaria resistance, it is considered dominant since one allele for HbS confers resistance (although homozygotes for HbS are probably not resistant). So this is a sticky case, and not at all typical.\r\n',NULL,1725,4,1),(###-12-14&&

1497    Q: I know that all females are mosaics for genes on the x-chromosome. You explained that heterozygous females will usually still be able to see color because enough of the cells in her eyes will be using the x-chromosome with the non-mutated gene and will correctly respond to light. In the case of tortoiseshell cats, the x-chromosome destined to be a Barr body is chosen when the fetus is a small group of cells hence the large patchy spots.  Since a heterozygous human female has correct function in every other (or so) eye cell does the switching off of the x-chromosome happen much later in human fetal development (making the \"patches\" much smaller)

1497    A: I think switching off happens at the same stage in all cells. However in some tissues the descendants of different cells \'mix\' and in some they don\'t. In the eye, the cells get all mixed up but in the skin they don\'t -- all the clonal descendants of a single cell stay in the same area. That\'s why patchiness is clear in tortoise shell cats but not in heterozygous color blind females. There are human skin conditions that show the same pattern as coat color in cats.',NULL,1726,4,1),(###-12-14&&

1498   Q: Question about 10-2.\r\nIf you have two inbred strains (AABB and aabb) and cross them, the problem set writes the F1 as AaBb. Is this really AB/ab? I assume it comes from a haploid gamete AB (where A and B are on the same chromosome) fusing with a haploid gamete ab.\r\n\r\nIf this is true, then where does the 3:1 ratio in the answer to 10-2 A come from? I was thinking the F2 would form from combinations of 2 AB gametes and 2 ab gametes.&&

1498   A: You are correct if A and B are alleles of two linked genes. If they\r\nare unlinked, or the traits in question are controlled by a single gene, then answer is\r\ndifferent. In part A, cross is Aa X Aa, not AaBb X AaBb. A single gene controls both traits. In part B, the 2 genes are not linked.\r\n\r\nF2 Comes from A and a gametes meeting more of the same. Standard Aa X Aa\r\ncross.',NULL,1727,4,1),(###-12-14&&

1499   Q: Question 10-17 - Why isn\'t V4 = A-h/B-h?  If it is A-h/C-h (as indicated in solutions), then some of the progeny would be AC normal? In addition, then where will the B allele in AB come from

1499   A: The AB person married into the family; he is not a descendant.  VI-5 is AC normal.',NULL,1728,4,1),(###-12-14&&

1500   Q: If the RF= 10% then the number of recombinants are 5% each and number of parental gametes is 90% (total). Then why is it that in 10-12, the numbers of recombinant of one type do not equal recombinant of another type and the parental gametes numbers do not match either? The same is true in mouse example in the \'How to measure RF\' handout.&&

1500   A: When you flip coins do you get exactly 50- 50 or is it approximate? Say 6 heads and 4 tails? You are looking at the results of a sampling of the total pool of gametes. The sample shows statistical variation from the expected just like coin flips do.',NULL,1729,4,1),('0000-00-00&&

1501   Q: What is a test question in this context

1501   A: A test question in this context is a question added by an administrator to test the Q&A database system.',NULL,1730,1,1),(###-09-19&&

1502   Q: test2&&

1502   A: answer to test2',NULL,1731,2,1),(###-09-22&&

1503   Q: Is there an easy way to tell if alanine is more or less non-polar than leucine?  My question is in context of resolving alanine and leucine in paper chromatography (problem 2-20).&&

1503   A: In general, all other factors being ~ equal, the more hydrocarbon groups present in a molecule, the more hydrophobic it should be. So leucine should be more hydrophobic than alanine, and an 18 carbon fatty acid should be more hydrophobic than a 10-carbon fatty acid.  But tyrosine should be less hydrophobic than phenylalanine despite the same number of hydrocarbon carbons, because it has that extra hydroxyl on the ring.\r\n',NULL,1732,1,1),(###-09-22&&

1504   Q: I\'ve been reading the lecture notes and slides and have noticed that you have labeled the anode as the positive (+) terminal and the cathode as the negative (-) terminal. In every other science class I have taken, the convention was that the negative terminal is the anode and the positive end is the cathode. Why is the convention different here

1504   A: Inside a battery (a galvanic or voltaic cell) chemistry is causing the anode to produce electrons so it is considered negative. But when current is drawn from the battery (or power source) during external electrophoresis (an electrolytic cell), using the battery, the anode is positive relative to the cathode. In chemistry there is a lot of interest in making batteries. In biology there is a lot of interest is using batteries. So the anode is positive in biology and most of life (e.g., Ipods).\r\n',NULL,1733,1,1),(###-09-22&&

1505   Q: What\'s the difference between Vmax and turnover number?\r\n&&

1505   A: Vmax = k3 * Eo\r\n\r\nk3 is the turnover number, which is characteristic of an enzyme with a particular substrate.  That number can be written down in a book and looked up.\r\nEo is at the mercy of the experimenter. Or of the cell. It is the amount of enzyme present in the situation you are analyzing. Usually, that is a test tube, and you have added the enzyme Vmax therefore is determined by the experimenter or by the cell\'s accumulation of the enzyme. It remains dependent on the turnover number as well.\r\n\r\nSuppose alcohol dehydrogenase will oxidize ethanol with a turnover number of 100 but n-propanol with a turnover number of only 20.\r\nYou add 1 nmole of enzyme to tube A with a very high (saturating) concentration of ethanol.\r\nYou add 5 nmole of enzyme to tube B with a very high (saturating) concentration of n-propanol.\r\nWhich tube exhibits the higher Vmax?\r\nNeither. It\'s a tie.  Vmax(A)  = 100*1 = Vmax(B) = 20*5 Vmax is operationally defined.  k3 is a constant.\r\n',NULL,1734,1,1),(###-09-22&&

1506   Q: All amino acids have charges amino and carboxyl groups attached to the alpha carbon, yet many are classified as non-polar. How can a molecule have an overall charge and not be polar

1506   A: We are most concerned about the amino acids as they contribute to the \r\nfunction of proteins of which they are a part. For almost all the amino \r\nacid residues in a polypeptide, there is no longer an amino or a \r\ncarboxyl group present (they have been converted to amides) and so there \r\nis no full charge and thus much less polarity.  The amide group remains \r\npolar, it is true. However, in comparing and categorizing amino acids, \r\nwe ignore the common alpha carbon and its attached group except for the \r\nside chain. So we are really speaking about the side chains when we \r\nclassify amino acids, and when we try to rationalize their contribution \r\nto portoen function.\r\n',NULL,1735,1,1),(###-09-22&&

1507   Q: After hydrolysis, a glucose chain\'s free molecules can exist as alpha or beta glucose, straight chain glucose, mannose, galactose, or any variant where the anomeric carbon is carbon 1. But can they become fructose molecules? Fructose has its double-bonded O on the 2nd carbon, and uses this as its anomeric carbon. \r\n&&

1507   A: Glucose after hydrolysis, i.e., free glucose, can undergo ring opening and ring closure continuously, because the C=O  of C1 is easily attacked by the OH of C5. The rings that form can be either alpha or beta. So free glucose exists as an equilibrium mixture of the straight chain, the alpha ring, and the beta ring. Transformation to mannose, galactose or any other stereoisomer of glucose is quite another story, and does NOT occur in a solution containing just pure glucose (i.e., without some considerable help from an enzyme). The transformation of\r\nglucose(D-glucose) to mannose requires the breaking of the the stable hydroxyl group at C2, the breaking of the the C-H bond at C2, and reconnecting those atoms in the opposite orientation. So pure glucose (or any other hexose) does not transform itself into any other sugar with another name.  It is only the different forms of 3-D

orientation around the anomeric carbon that is in equilibrium (C1 in glucose, C2 in mannose).\r\n',NULL,1736,1,1),(###-09-22&&

1508    Q: Question 1-18 a) in the problem book says that two macromolecules are hydrolyzed in separate test tubes, and afterward each tube contains only fructose. I was wondering what kind of macromolecule contains only fructose?  I am aware of only the glucose-fructose alpha 1-2 bond in the sucrose dissacharide. \r\n\r\n&&

1508    A: It doesn\'t matter, for the usefulness of this problem. The documented existence of polyfructose is immaterial to respond to the logic of this question, which is about the different forms of glycosidic bonds that COULD form, or be drawn. Indeed, if the problem is seen in the context of an experimental result, then it could represent the discovery of polyfructose.  Nevertheless, Googling polyfructose yields 4020 hits.',NULL,1737,1,1),(###-09-22&&

1509    Q: Are all of the carbons 1 through 5 in a hexose capable of forming glycosidic bonds? \r\n&&

1509    A: Yes. Why not? the non-anomerics can hook up with an anomeric of a second molecule, and the anomeric can hook up with any hycroxyl of a second sugar (including the heretofore neglected carbon 6).',NULL,1738,1,1),(###-09-22&&

1510    Q: Does steric strain affect where two monosaccharides will bond

1510    A: It could influence the favorability of the reaction.  But glucose hexoses are good at flipping around until they minimize such strain. I believe I\'ve seen virtually all the positions of a hexose used in one polysaccharide (or disaccharide) or another.',NULL,1739,1,1),(###-09-22&&

1511    Q: Do glycoside bonds in a polysaccharide have to follow a general pattern, or can alpha and beta bonds form in random order? \r\n&&

1511    A: The polysaccharides are put together by the repeated action of a small number of enzymes, the same process iterated faithfully. So the linkages are not at all random, but prescribed and orderly. That said, there is no reason that linkages cannot alternate in an orderly way between alpha and beta, and/or (1,4) and (1,3), for example. \r\n',NULL,1740,1,1),(###-09-22&&

1512    Q: If whether the molecule is alpha or beta is dependent on the equatorial or axial positioning of the hydroxyl group on the anomeric carbon, then what happens when the ring flips, thus rendering what was axial equatorial? Does that flip change the identity of the glucose?  I understand that for glucose in particular this may be less of an issue due to the steric advantage of having most of the hydroxyls in the equatorial position and therefore not interacting as much, but what about those sugars that do not have the majority of hydroxyls in the equatorial position?\r\n&&

1512    A: Since the position of the anomeric hydroxyl is locked in for alpha and for beta glucose, I\'d say they therefore retain their identity regardless of the state of the chair or boat. The chair we talk about is the most stable structure and as such represents the majority of the molecules at any given time.  So when we say alpha glucose, we show the most common conformation in space, but we admit that other conformations are possible for alpha glucose. Other sugars will adopt their most stable conformation, which may be different from that of D-glucose.  For instance, beta-L-glucose would have all of its hycroxyls axial if left in the chair we use for D-glucose. It would flip into the opposite chair

(headrest vs. footrest) and restore equatorial hydroxyls throughout.\r\n',NULL,1741,1,1),(###-09-22&&

1513   Q: Why are the glucose residues in the crystal structure of a cellulose fiber (slide ~58 in lecture 2) alternating between right side up and upside down?\r\n&&

1513   A: There is free rotation about the glycosidic bond, so the individual glucose residues can swing around as need be. Since the crystal structure of cellulose shows the very regular repeated pattern of right-side-up followed by upside-down etc. , I conclude that this is the arrangement that maximizes the number of H-bonds between adjacent strands of cellulose polymers as they associate to make a fiber, i.e., the most stable structure will prevail. I choose to link them right side up for clarity.\r\n',NULL,1742,1,1),(###-09-22&&

1514   Q: Can any assortment/order of hexoses form a polysaccharide (in a manner analogous to RNA bases)

1514   A: That is, the formation of a particular linkage, and its repeated nature in polysacchraides, depends on the presence of enzyme catalysts that can facilitate the particular reaction. Many such enzymes have evolved in the last 3 billion years, forming a modest variety of  different polysaccharides, most of the variety being in different organisms. I would guess on the order of hundreds of different polysaccharides exist through nature. RNA is much more complex, and the order if the bases is not determined by specific enzymes that catalyze the specific linkages. Rather, a template is used (DNA), another  subject.  E. Coli alone has thousands of different RNA polymers, and humans have hundreds of thousands. \r\n',NULL,1744,1,1),(###-09-23&&

1515   Q: I\'m having a hard time predicting what shape a polysaccharide will form.  Question 1-25 asks about glucose molecules linked only by beta (1 -->3) glycosidic bonds.  This bond will be equatorial - equatorial as all the hydroxyls in beta glucose will be equatorial. However, the shape that is formed is closer to a helix than a straight chain.  After reading the solution, I realize that you cannot simply see an equatorial - equatorial bond and assume it is a straight chain like cellulose, however, I\'m having trouble visualizing this one.  How can you predict what form a polysaccharide will assume if it is not one that we have already seen (eg: starch, glycogen, cellulose)

1515   A: Any bond that creates an angle will tend to form a helix. Cellulose is special in that regard as it forms not much of an angle up and down (as we usually depict the sugar rings) due to the beta configuration of equatorial to equatorial but also not if viewed from above, seeing the rings as flat hexagons (instead of coming out towards you). There because the inding is 1 to 4 , you get a head to tail arrangement again with no little or angle. Not so here for 1 to 3, where the connections lead to a constantly turning chain as seen from above.\r\n\r\n O-O   1,3\r\n      \\   1,3\r\n      O\r\n      /   1,3\r\nO - O   1,3\r\n',NULL,1745,1,1),(###-10-02&&

1516   Q: The equation N = N02t/tD does not seem correct to me in general.  In the  t/tD term, if t is not an integer, no new cells would be produced.  Only when a new generation time (multiple of tD) was reached would N rise â€" a true \"double or nothing\" situation.&&

1516   A: Unless extraordinary measures are taken, cells in a culture or cells in a growing tissue are not really dividing synchronously (as simplified in class). Even if they would be somehow synchronized, the synchrony rarely lasts for more than 1 to 2 generations, as the generation time is a probabilistic number. With an average doubling time of 60 minutes, some cells will divide after 70 minutes and

some after 50 minutes, etc. And in the typical asynchronous situation, some cells will divide a minute after time 0, others 2 min., etc. The equation using t as a continuous variable still give accurate predictions of cells N at time t, and a good measure of the doubling time. \r\n',NULL,1746,2,1),(###-10-07&&

1517    Q: In problem set five, question 1.B.v. - asking what you could feed yeast other than glucose or lactate to make wine, the answer given is any mono/poly - saccharide.  I am confused as to why we could not also feed the yeast a triglyceride or protein?\r\n&&

1517    A: Those compounds are not generally used as energy and carbon sources unless sugars are absent. In any case, their breakdown requires oxygen, so you get CO2 instead of ethanol. If you want to make wine, you have to feed something that can be fermented (broken down anaerobically). \r\n',NULL,1747,2,1),(###-10-13&&

1518    Q: I understand that it is the hydrogen bonding between the base pairs that hold the single strands of DNA together, but why does the helical structure form?\r\n&&

1518    A: If you make all the correct distances and angles for all the bonds, you get a helix, not a straight ladder.\r\n',NULL,1748,2,1),(###-10-13&&

1519    Q: The Key to the Problem 5-2 states  \"...assuming no energy cost for transporting reduced NAD into mitochondria so it can be reoxidized by electron transport..\" I was wondering, is the NAD actually transported \"into\" the mitochondria? I thought it was just electrons and protons that were pumped into and out of the mitochondria. Can you clarify?\r\n&&

1519    A: Some compounds actually are transported in or out, for example, pyruvate. NAD isn\'t. Reduced NAD on the outside reduces another compound on the outside; the other compound (carrying the reducing power) is transported into the mitochondria;  the other compound reduces NAD on the inside. There are several different so called shuttle systems that carry the reducing power.  This is discussed in more advanced texts.\r\n',NULL,1749,2,1),(###-10-16&&

1520    Q: Can yeast live without making ethanol and CO2?  (Prob. 5-1B)&&

1520    A: In the absence of oxygen, yeast must use a fermentation path (2 steps) to regenerate NAD, which it does by reducing pyruvate to ethanol and CO2. In the PRESENCE of oxygen this fermentation path is not needed and IS NOT used. So if lactate is provided with oxygen, then the provided lactate -> pyruvate -> Krebs.  Yeast have no need of the ethanol and CO2.  They are waste products to yeast even as they are valuable yeast products for us humans.\r\n',NULL,1750,2,1),(###-10-16&&

1521    Q: Why don\'t we use the carboxyl from acetyl CoA to make CO2 in the first turn of the Krebs cycle

1521    A: Once the acetate group joins the KC, it becomes part of citrate, which has 6 carbons total. Two things are going to happen to that citrate in one turn of the cycle. Two of its carbons will become CO2 (toward the goal of converting glucose to CO2) and 4 of its carbons will become oxaloacetate (to pay of that debt of using oxaloacetate to get the acetate into the cycle). All that takes chemical reactions catalyzed be enzymes, and the pathway has evolved to use the acetate to help pay off the debt and to use the carbons from the original oxaloacetate to produce the CO2 (2 of them). It could have been otherwise, but it isn\'t. \r\nThe two objectives of one turn of the cycle have been met this

way, so there is no disadvantage except that some humans had a hard time figuring it out and others have a hard time understanding it in Intro Bio. As more cycles turn, the original acetate carbons do wind up eventually as CO2. But not after one cycle.\r\n',NULL,1751,2,1),(###-10-16&&

1522    Q: In the problem book, 4-17-C the answer as to where a radioactive carbon is first detected in fumarate  is \"you will get a mixture of the two following molecules, 1/2 of each kind...\" But when the radioactive molecule is FIRST detected it\'s either going to be the carboxyl on the top OR on the bottom, right?\r\n&&

1522    A: There is no top and bottom really for such a symmetrical molecule in this case.  You are allowed to slide the molecule around on the paper as much as you wish. So top and bottom are meaningless. That is, I could DRAW it on the top, then slide it around (or turn the paper upside down) and it would be on the bottom. But it\'s the very same molecule. I can\'t even number 1. I could call the carbons end carbons and internal carbons, but there would be 2 of each, indistinguishable. \r\n\r\nAlso, technically, you cannot detect radioactivity in a single molecule, a separate issue.\r\n',NULL,1752,2,1),(###-10-20&&

1523    Q: how are bases synthesized in the cell?  That is, if I  wanted to make just the DNA radioactive in line of cells, could I add radioactive molecule in the medium as we saw in earlier problems about glucose or phospholipids?  Or would I have to grow cells in radioactive medium, extract a base from those cells, and then add those radioactive  bases to the target cells?  Is is clear what I am trying to get at?\r\n&&

1523    A: To get radioactive bases, you either make the radioactive bases chemically, or as you say, grow cells in radioactive medium and then isolate the bases from the cells (or their DNA). To get radioactive DNA, you would add radioactive T to the cells.',NULL,1753,2,1),(###-10-20&&

1524    Q: Question 6-1 part f states that if you denature the helix that H-bonds will be broken between the base and its complement on the other chain of the helix...my question is are the Van der waals bonds secondary to base stacking within each single chain also broken due to denaturation?\r\n&&

1524    A: Yes. Van der waals forces are weaker than H bonds.\r\n',NULL,1754,2,1),(###-10-20&&

1525    Q: For 5R-3 Part C, the answer says at the very end \"When [ADP] falls, enzyme should release ADP, enzyme (& glycolysis & glucose consumption) should slow down, and rate of ATP production should also slow down.\" Is it talking about the rate of ATP production only in glycolysis?(Please correct me if I am wrong) The rate of ATP production should be the same in fermentation as in oxidative metabolism. The rate of glucose consumption will be higher for fermentation, but the rate of ATP production should still be the same. However, the rate of ATP production in glycolysis will be slower in the presence of oxygen. Is my theory wrong?\r\n&&

1525    A: The assumption is that the rate of ATP production will overshoot at first, as glycolysis will be going very fast (due to activation by ADP). \r\nOnce the ADP stops activating glycolysis, the overshoot will be corrected. You are right that overall ATP production in the steady state should be the same (if growth rates are the same) whether cell is using fermentation or respiration.\r\n',NULL,1755,2,1),(###-10-20&&

1526    Q: In problem 6-12B, wouldn\'t you rather go both directions when replicating DNA (i.e. have as many forks as possible?)(exam 2) \r\n\r\nThis question is not about real DNA. It\'s about an imaginary

molecule constructed for study purposes. So take it as a given there is one origin and proceed from there. Most real long linear DNA molecules do indeed have multiple forks with bidirectional replication at each fork. \" My question is: Doesn\'t bidirectional replication have one origin as well? Or does it have two origins?\r\n&&

1526   A: Bidirectional eplication of small molecules starts from only one origin. \r\nBidirectional replication of large linear molecules starts at multiple origins. (In some years we discussed this earlier. This year we put it off until next term.) However, as it says below, this is an imaginary situation anyway. Also notice it is single stranded DNA, not double stranded, so you are making the complement to only one strand. There is no ordinary fork.\r\n',NULL,1756,2,1),(###-10-20&&

1527   Q: I\'m having a hard to understanding exactly what is going on in the electron transport chain. I understand that electrons are being passed along through the oxidation/reduction of the compounds that make up ETC, and that in the process protons are pumped out into the intermembrane space.  But where do these protons come from?  It looks like only NADH2 and CoQ can give up protons, so do all 10 protons that are generated from one pair of electrons passing through the ETC come from NADH2 and CoQ?  Or, are there other sources of protons within the matrix mitochondria?\r\n&&

1527   A: There are always some H+ in solution at pH 7. It\'s those H+ that get pumped. They aren\'t necessarily the same protons that came from metabolism.\r\n',NULL,1757,2,1),(###-10-20&&

1528   Q: I have a general question that came up while doing problem 6-17 that I wanted to clarify.  In PCR, we do not use Ligase because we first degrade the double stranded DNA into single-strands, and thus replication occurs continuously in both directions and no Okazaki fragments are created.\r\n\r\nYou denature the DNA. You do NOT degrade it. That means break phosphodiester bonds.\r\n\r\nIn \"DNA synthesis in a test tube\" (as is the case with this problem), DNA is replicating in its normal \"zipper-like\" fashion so Ligase IS necessary to connect the Okazaki fragments of the lagging, discontinuous strand.  Do I have this right?\r\n&&

1528   A: Yes. DNA synthesis/replication is not the same as PCR.',NULL,1758,2,1),(###-10-20&&

1529   Q: In question 6-12 (E) the answer states that pyrophosphatase is not needed in the synthesis of the complementary strand of DNA.  I was under the impression that pyrophosphatase was necessary to provide the energy needed for DNA synthesis.  If this is not so, when/why DO we need pyrophosphatase?  Also, does this mean that DNA synthesis can occur with the energy provided from ATP hydrolysis alone?\r\n&&

1529   A: Pyrophosphatase (PPtase) is normally needed for all DNA synthesis. It can be omitted in some test tube situations when the concentrations of dXTP\'s are high enough to drive the polymerase reaction to the right. The polymerase rxn has a standard delta G of about zero. The actual delta G has to be negative, either by removing the product (by\r\nPPtase) or piling up the concentration of the reactants (dXTP\'s), as in this problem. In a real cell, the dXTP concentration isn\'t that high, so PPtase is needed.\r\n',NULL,1759,2,1),(###-10-23&&

1530   Q: This question is with regard to PS# 6-13 (C-2). I am having a hard time picturing PCR. After we denature 1 molecule of DNA and anneal 2 primers to 2 L strands, we make 2 M copies from our two original L strands.  Do we then lower T to renature 2L strands (into helix) and isolate the newly synthesized M strands?  If so, do we then anneale primer to those 2 M strands and replicate, creating 2 S strands?\r\nAnd following that, do we then lower T again enough to renature the 2 M strands (into

helix) thereby isolating the 2 S strands that we can then replicate continuously? (I am trying to understand how many L strands, M strands and S strands we would end up with after a given # of steps, and am not understanding how we wouldn\'t simply keep on replicating the L or M strands, unless we \"get rid of them\" i.e. renature them so that they are no longer being replicated).&&

1530    A: I may not have made it clear that you continually raise and lower the temperature to denature, hybridize, and elongate. You repeat this cycle over and over. The 2 M or 2 L strands never meet because there are so few of them, so they never hybridize to each other. They hybridize to another molecule of primer and repeat the previous cycle, making S and M strands respectively. You keep making more M & S strands, but only at a linear, not exponential rate. You make additional S exponentially from the pre-existing S. \r\n',NULL,1760,2,1),(###-10-23&&

1531    Q: In problem 6.16 A, we hydrolize nucleic acid into nucleotides. I thought this means would have broken the \"base stacking\" base to base bonds, but that answer is not given as correct. Why is base stacking not a factor here?\r\n&&

1531    A: Hydrolysis means the breaking of covalent bonds by adding water. The point of that problem is that covalent bonds are broken. Of course under those conditions weak bonds are broken too, but that is taken for granted, and not counted as part of the hydrolysis process. \r\n',NULL,1761,2,1),(###-11-05&&

1532    Q: I have a question about the heads and tails present on DNA and RNA. By heads and tails I mean the non-coding regions upstream and downstream of the coding regions.\r\nThese sequences are usually called other things, but ok. \r\n DNA heads have promoters, operators.\r\n DNA tails have terminators.\r\n\r\nRNA heads have 5\' caps, ribosome binding sequences, and the start/met codon. RNA tails have poly-A tails and a stop codon.\r\n\r\nHow do the DNA head/tail become the RNA head/tail? Are the DNA head/tail actually transcribed - if so, what becomes of them? Where do the RNA head/tail come from?\r\n\r\nGiven all the various forms of post-transcriptional and post-translational processing that occur, how close is the association between nucleotide sequences on DNA and AA sequences on proteins, really?\r\n&&

1532    A: There are also spacers between genes.\r\nAll these are nontranscribed sequences in the DNA. Some consider them part of the corresponding gene and some consider them regulatory sequences that are outside the gene proper.\r\n\r\nIn RNA, the \'heads\' are called leaders or 5\' UTR\'s. \'tails\' are called trailers or 3\' UTRs. 5\' caps & poly A in euk. only. Start codon is translated. Stop codon is not.\r\n\r\nThe DNA head/tail are not transcribed. The RNA heads and tails are \r\ntranscribed from parts of the gene that are not translated. Genes don\'t \r\njust include the translated part -- they include more. (Exact details \r\ndiffer between eukaryotes and prokaryotes.)\r\n\r\nEvery RNA sequence is transcribed from a DNA sequence. The importance \r\nof later modifications vary with the case.',NULL,1762,3,1),(###-11-05&&

1533    Q: I\'m a little bit confused about the microRNAs: when you talk about the precursor RNAs that fold back on themselves to form double stranded hairpins, this precursor RNA is anti-sense or sense? Is it just that this folding back shows the spots where the sense strand is complementary to itself?\r\n&&

1533    A: The ds RNA is both sense and anti-sense. The sense part is degraded, leaving the anti-sense part to pair up with mRNA (sense).',NULL,1763,3,1),(###-11-06&&

1534    Q: In the answer to ps 7-19, you write that there are 3 diff types of rRNA- and it says in the lecture notes \" *rRNA* --   You need several kinds (3-4)to make one ribosome; exact # depends on whether it\'s a eukaryotic or prokaryotic ribosome. \"\r\n\r\nWhat are these diff types for?  Does the difference between them refer to the different densities of rRNA in the sml (5S and 5.8 S) vs large (16-18S) subunits?\r\n&&

1534    A: The differences are in size, but they matter because the different sized rRNA\'s have differences in sequence, folding, and  function. Remember \'S\' doesn\'t refer to density -- S value is measured by sedimentation velocity centrifugation, not equilibrium density centrifugation.\r\n',NULL,1764,3,1),(###-11-11&&

1535    Q: I see from handout 14A that inosine can be in the anticodon, why can\'t inosine be in a codon as well?\r\n&&

1535    A: There are enzymes that modify specific bases in the tRNA; these enzymes don\'t work on bases in mRNA. The answer is the substrate specificity of the modifying enzymes.',NULL,1765,3,1),(###-11-12&&

1536    Q: A few details about transduction and transformation have left me\r\nwith some questions.  I would appreciate your clarification of these ideas. Is transduction a sometimes-helpful byproduct of virus replication? There is a lot of info about the virus reproduction cycle in the lecture notes under transduction; I want to make sure that the focus is still on bacteria.  So the main idea: Viral replication via the lytic cycle sometimes causes transduction, which in turn may sometimes be helpful in creating more favorable genotypes/phenotypes in host bacteria. Do I understand this correctly?\r\n&&

1536    A: Yes, from the bacterium\'s point of view. However there is also the virus\'s point of view, which was discussed too (in a different class).\r\n',NULL,1766,3,1),(###-11-12&&

1537    Q: How likely is it that a transduced DNA fragment is recombined? You seem to imply in the notes that DNA fragment recombination is very rare (regardless of the method of transference). If that is true, then transduction seems likely to be the rarest of the three sources of genetic variation in bacteria. Is this true?\r\n\r\n&&

1537    A: Recombination overall isn\'t that rare. Recombination in any particular spot IS rare. However most viral and bacterial genetics involves selecting the progeny that come from rare events. Often the question is, can I get any progeny (with some particular property) at all? Or how many can I get (compared to some standard). You aren\'t expecting a lot of offspring or survivors with the desired characteristics. You are setting up conditions that select for growth of the rare desired offspring (and not for the growth of others).\r\n',NULL,1767,3,1),(###-11-12&&

1538    Q: The lecture notes specify that transformation is detected by a change in the phenotype. Do scientists who artificially induce transformations rely only on phenotype expression to measure their success? In other words, does no change in the phenotype preclude a successful (laboratory) transformation?\r\n&&

1538    A: Usually you have to be able to select for a change in properties (phenotype) to detect successful genetic (genotypic) change. However, sometimes, especially with genetic engineering, you test directly for the presence of the desired genotypic change using hybridization. That will be discussed in lecture 18.\r\n',NULL,1768,3,1),(###-11-17&&

1539    Q: In problem 11-7, it says that the \"simplest\" bacteriophage contains ds DNA.  I would think that ss RNA (positive sense) would be the simplest because it could be immediately translated into proteins by using host cell\'s enzymes.  I don\'t understand why ds DNA is easier for the host cell to work with.\r\nAlso, how are we to assume that the virus particle contains 1 coating protein.  I don\'t remember learning about viral coating, except in the case of \"phony phages\".\r\n&&

1539    A: Replication of RNA using an RNA template requires an enzyme not found in normal cells, and the virus would have to code for that enzyme. \r\nReplication of DNA and transcription of DNA can be achieved using cellular enzymes. As far as we know, the \'real\' simplest viruses are RNA, but in theory, it seems to me a ds DNA virus could get by by coding for the smallest number of \'extra\' proteins not found in an uninfected host.\r\n',NULL,1769,3,1),(###-11-18&&

1540    Q: With regard to question 12-9 (C), I know we always have a constant amount of repressor, but as inducible enzyme III is being made, wouldn\'t we have a constant level of inducer as well?  (Until enough enzyme III is made, and then the level of inducer would decrease, eventually being detached from the repressor, and the repressor would rebind to the operator, shutting down production of the enzyme)? In other words if the level of inducer is not constant during synthesis of an inducible enzyme, what does the map of the change in inducer level look like?\r\n&&

1540    A: If not much enzyme III is being made, there probably is a suboptimal level of inducer around. Turn on of the operon isn\'t all or nothing.\r\n',NULL,1770,3,1),(###-11-19&&

1541    Q: I seem to recall that a promoter and operator have some overlap on the DNA strand. Is this true? If we destroy the operator does that mean this could prevent RNA Polymerase from binding to the promoter

1541    A: Sometimes they overlap and sometimes they don\'t. For the problems, keep it simple and assume they are separate.',NULL,1771,3,1),(###-11-20&&

1542    Q: Problem 13-14b.  Are terminators for transcription always in every cistron?  Why does transcription that starts to the right end before it gets to the eukaryotic gene end? Does transcription of plasmids not usually make it all the way around a plasmid?\r\n&&

1542    A: Transcription usually ends after each eukaryotic transcription unit. Given real distances, it would never go all the way around.',NULL,1772,3,1),(###-11-20&&

1543    Q: In the notes under conjugation, and frequency, it says \"Copies of plasmids are transferred more often than copies of parts of the chromosome, so genes on plasmids are relatively easily transferred from one bacterium to another.\"  So is it correct to say that F+ transfers happen more frequently than Hfr transfers?  If so, why?  Is it because it\'s more likely that the plasmid will exist in future generations?  As I understand it, Hfr stands for high frequency of recombination, so recombination happens more frequently with Hfr, but the transfer of genetic material happens less overall?\r\n&&

1543    A: Hfr means high freq. of transfering bacterial (chromosome) genes -- relative to without the integrated F. Not high freq. rel. to a plasmid gene.\r\nA gene on a plasmid is likely to be\r\ntransfered every time conjugation occurs, because the whole plasmid is copied. Any particular gene on the

chromosome is less likely to be transferred unless it happens to be right near the origin of the copying.\r\nPerhaps that wasn\'t clear enough.\r\n',NULL,1773,3,1),(###-11-26&&

1544    Q: I\'m a little confused about banding---if you have 2 different homologs, will they show different banding patterns?\r\n&&

1544    A: No. All normal homologs have the same banding pattern. Your maternal #1 and your paternal #1 have the same pattern. Different chromosomes (1 vs 2) have different patterns. There are pictures of karyotypes in your texts and on the web. That might make it clearer.\r\n',NULL,1774,4,1),('1008-12-09&&

1545    Q: What is the difference between a squash and a karyotype?\r\n&&

1545    A: A karyotype has the chromosomes arranged in order, in pairs. See texts, such as Becker fig. 19-23. A squash is just that -- a picture of the chromosomes spread out on the metaphase plate.\r\nIf you go to Google images and put in both terms I am sure you will get some nice typical pictures.\r\n',NULL,1775,4,1),(###-12-09&&

1546    Q: What is the difference between a squash and a karyotype?\r\n&&

1546    A: A karyotype has the chromosomes arranged in order, in pairs. See texts, such as Becker fig. 19-23. A squash is just that -- a picture of the chromosomes spread out on the metaphase plate.\r\nIf you go to Google images and put in both terms I am sure you will get some nice typical pictures.\r\n',NULL,1776,4,1),(###-12-12&&

1547    Q: When a population is NOT at genetic equilibrium, the allele frequency cannot be calculated from the genotype frequencies (and vice versa). Is there anything that can be determined about a gene pool using the HW equation if the population is not at equilibrium? Can other pertinent calculations be made?\r\n&&

1547    A: No. All you can figure out (using the H-W law) is that the population is NOT in g.e.',NULL,1777,4,1),(###-12-12&&

1548    Q: If there is nondisjunction in M1, do the homologous chromosomes that didn\'t disjoin originally ALWAYS separate in M2?\r\n\r\nAnd if so, do they ALWAYS split into two pairs of sister chromatids, or is it possible for them to split 3:1?\r\n\r\nIn other words, is it possible for there to be ND in M1 and partial ND in M2, resulting in 2 gametes with 0 copies of a chromosome, 1 gametes with only 1 copy, and 1 gamete with 3 copies?\r\n&&

1548    A: Yes. They aren\'t completely joined anyway -- just paired up.\r\n\r\nND is almost always a one time event, so if ND occurs at M1 it doesn\'t usually occur at M2 and vice versa.\r\n\r\nIs it possible? I suppose yes, but extremely unlikely. Remember the proteins that hold homologs together are different from the ones that connect sister chromatids, so double ND isn\'t a single failure -- it\'s two \r\nseparate mechanisms failing to work. The answer to \"does anything ALWAYS happen?\" or \"is anything \r\nimpossible?\" is always \"no.\"  But highly improbably, yes.',NULL,1778,4,1),(###-12-12&&

1549    Q: I had a quick question about 8-14 in the problem book. It says that the zygote in G1 and the skin cell in G1 have 46 chromatids, but I thought that in interphase, the DNA exists as chromatin. Can the DNA be considered chromatids also?\r\n&&

1549    A: You are correct that chromatids and chromosomes, strictly speaking, don\'t exist in interphase. However there has to be some way to say that the DNA per chromosome equivalent (stretched out chromosome?) is doubled in S, and that there is one chromatid equivalent (one double stranded DNA molecule per stretched out chromosome) before S, and two chromatid equivalents (2 DNA molecules) afterward. That\'s what the question is getting at.\r\n',NULL,1779,4,1),(###-12-12&&

1550    Q: Question (8-2) states that a diploid cell contains 2N chromosomes. After interphase, doesn\'t the cell contain 2N chromosomes, instead of 4N?  I understand that there are now 4 chromatids because of replication, but doesn\'t (N) refer to the number of different genetic material?  I would to be clear of the difference between (c) and (N) because this could be problematic.\r\n&&

1550    A: I think you are confusing chromosomes and chromatids. The number of chromatids, not the number of chromosomes, increases during interphase. A chromosome with two chromatids counts as one chromosome. The DNA content doubles, but the number of chromosomes remains the same. That\'s why C doubles, but not the number of chromosomes.\r\n',NULL,1780,4,1),(###-12-12&&

1551    Q: I\'m having a little trouble with problem 8-8, the second part of part A. The question asks which items are identical, and I don\'t see how you can distinguish whether the X shaped chromosome is cut along a vertical or horizontal axis because the centromere is not shown.  Are we supposed to assume that it is cut vertically down the middle because that is clearly the case with chromosomes III & IV, and since this is a karyotype we assume that all chromosomes are lined up in a similar way?\r\n\r\nIf this were not a karyotype but rather a chromosome squash would there be anyway to tell which way the chromosomes are joined without seeing the chromatids?\r\n&&

1551    A: You got it. When chromosomes are lined up in a karyotype they are always drawn with sister chromatids on the right and left, not on the top and bottom. (This follows, as you notice, from looking at the upside down V shaped chromosomes III & IV.)\r\n\r\nIf the chromosomes aren\'t banded, then in a squash you can\'t tell what an \'X\' shaped chromosome represents -- whether the sister chromatids are on the right and left or top and bottom. However when squashes are made, the chromosomes are almost always stained to show bands, and therefore it is usually quite obvious which arms are homologous.',NULL,1781,4,1),(###-12-12&&

1552    Q: I\'ve been having trouble with one of the concepts from meiosis. I know that c is equal to the DNA content per cell, but I don\'t quite understand what is meant by \"DNA content.\" Does c have a different value for different types of cells?\r\n\r\nIs it safe to say that the multiple of c (4c, 2c, etc.) is equal to the number of chromatids per cell?\r\n&&

1552    A: Each species has a unique value of c, based on the DNA content of the genome = content of DNA in a gamete.\r\n\r\nNo. The multiple of c = number of copies of each chromosome (ploidy) X # chromatids per chromosome. A haploid cell with 1 chromatid per chromosome has 1 X 1 = 1 C of DNA. A diploid cell with 1 chromatid per chromosome has  2 X 1 = 2C of DNA and so on.\r\n',NULL,1782,4,1),(###-12-15&&

1553    Q: With respect to Barr bodies, how does one tell the difference between a male with Klinefelters and a normal female?  From what I understand in the notes, the Barr bodies are seen at

interphase...and since everything else is non-distinguishable in the chromatin, wouldn\'t these two individuals\' cells appear the same?  Wouldn\'t they both have one inactive X-showing up?\r\n&&

1553    A: From Barr bodies, they\'d be the same. But the karyotype would be different. The male would have 3 sex chromosmes and a normal female has only 2. No Y in a normal female. If you suspect anything, you\'d do both a karyo and a Barr body count.\r\n',NULL,1785,4,1),(###-12-15&&

1554    Q: For question 15-6B in the problem book, I thought the answer should be that the enzyme is an allosteric inhibitor.  If the inhibitor is competitive, and the change to the substrate binding site was so great that the inhibitor no longer functioned, wouldn\'t that change also be so great that the enzyme would not bind substrate?  I thought that a change to the allosteric site would render the inhibitor ineffective, but could also affect the substrate binding site to some degree through changes in the overall enzyme structure, thus raising the Km in the process.  Is this reasonable?\r\n&&

1554    A: You are changing only 1 base, and presumably only 1 AA. That AA is in the substrate binding site of the enzyme. If you change the substrate binding site, even subtly, you are likely to change binding of a competitive inhibitor. This is the most parsimonious explanation. Your explanation requires that a change of one amino acid change two different sites on the enzyme. It is made even less likely by the fact (which I think wasn\'t stressed in this\r\nclass) that most allosteric enzymes have a substrate binding site on one subunit and an allosteric inhibitor/activator site on a different subunit.\r\nSo generally changing one site doesn\'t affect the other.',NULL,1786,4,1),(###-12-12&&

1555    Q: I am having conceptual trouble with the idea of recombination frequency... If one recombination event produces two recombinants (i.e. reciprocal), why do we count both recombinants when determining recombination frequency? It seems as though we should either count only one of the two possible recombinants or divide the final RF by two. The way it is done now seems to imply that recombination happens twice as often as it actually does. Am I understanding this correctly or am I just way off base here?\r\n&&

1555    A: Each recombination event generates two recombinants. However by convention we calculate map distance and RF by counting the total number of recombinants, not the number of recombination events. (This is true for both historical and practical reasons.) For small distances, the number of events and the number of recombinants are proportional. Since we want relative distances, whether we use RF or the number of recombination events doesn\'t matter, and what we call one map unit is arbitrary anyway. \r\nThere are ways to calculate the # of events from the number of recombinants, and vice versa, but we are leaving that for more advanced courses.\r\n',NULL,1784,4,1),(###-01-30&&

1556    Q: My question is regarding spindle fibers and microtubules. The Lecture 2 notes say that during anaphase \"MT slide \'out\' to give longer spindle fibers and elongated spindle.\" Does this mean that spindle fibers are microtubules or that the microtubules\' sliding out causes spindle fibers, which are not microtubules, to elongate?\r\n\r\nThe \"note\" below seems to specify that they are different fibers than the microtubules, but the solution to 1-11 says that spindle fibers are, in fact, made of tubulin.\r\n&&

1556    A: spindle fibers are made of MT\'s. They slide past each other to move chromosomes.\r\n\r\nThe MT\'s seen during interphase are made of the same tubulin monomers that make up the spindle fibers. The same monomers/Mt\'s  are rearranged to serve a different function during mitosis and interphase.\r\n',NULL,1787,1,2),(###-01-30&&

1557    Q: I was just reviewing lecture two and I am confused to how scientists recognized that the \"permanent\" structures like muscle would contain the same proteins (actin and tubulin) to make antibodies to probe for the antigen with in the cell in search of cytoskeleton, furthermore the same proteins to probe for the temporary structures during mitosis. Was this done on a chance basis knowing that the function of these proteins could be needed elsewhere

1557    A: I think the rationale was that maybe \'ordinary cells\' had some tubulin and/or actin during interphase. Ordinary cells (no cilia or flagella, and not muscle) certainly had MT & some MF during mitosis. So that\'s what led scientists to make the antibodies and add them to interphase cells. Also it is very common for the same (or similar)  proteins to be used by different cell types to do different things. \r\n',NULL,1788,1,2),(###-02-02&&

1558    Q: I\"m a little confused about the basolateral vs. apical regions of epithelial cells. Are they just defined by where the tight junctions are?\r\n\r\nAlso, the distinction between which side is basal and which side is lateral; the basal section will also be connected to the basal lamina?\r\n&&

1558    A: Yes, but you could look at it the other way -- the tight junction is where the boundary is.\r\n\r\nSometimes the distinction between \'basolateral\' and basal or lateral matters; we\'ll get to some examples. At this point, we\'re not distinguishing the two parts, although clearly the basal is (primarily) facing the body side, and the lateral is (primarily) facing the neighboring cells. However the cells are rounded, and they two surfaces fade into each other around the corners.\r\n',NULL,1789,1,2),(###-02-09&&

1559    Q: I was just curious about the situation with endo/exocytosis for plant cells? Obviously the cell walls are a barrier to this; what do they do instead?\r\n&&

1559    A: From a cursory check, I think plants do some endo and exo, although it is more limited than in animal cells. However there is plenty of vesicle traffic inside the cells.\r\n',NULL,1790,1,2),(###-02-09&&

1560    Q: I have a question about 2-1 in the problem set book.  The answer key says that experiment 1 shows us that it is passive transport because the concentration of the X in the cells appears to plateau around 10mM, which is the initial concentration on the outside of the cell. But, wouldn\'t the concentration on the outside of the cell be lower than 10mM now since the compound has moved from the outside of the cell to the inside?  Looking at this, I was sure that the concentration on the inside of the cell would be higher than the outside and that it was active transport.  How does the concentration remain at 10mM as time progresses?\r\n&&

1560    A: The volume inside the cells is so low that the concentration overall doesn\'t change very much.\r\n',NULL,1791,1,2),(###-02-09&&

1561    Q: The answer to problem 1-20 is talking about glycoproteins and it says \"oligosaccharides are added enzymatically in the ER and Golgi.\r\nTherefore *oligosaccharides can only be added to domains that are exposed to the enzymes inside the lumens of the organelles--the extracellular domains.*\"\r\n\r\nCould you clarify what this means? Does a glycoprotein get its saccharides attached in ER/Golgi and then gets sent to the membrane?\r\n\r\nOr does it get its saccharides while in a vesicle in the ER/golgi so only the part of it that is in the lumen of the vesicle gets modified?\r\n&&

1561   A: Yes, that\'s right. We\'ll explain this week or next.\r\n\r\nOnly the part in the lumen of the ER or Golgi is modified. There are no vesicles inside the ER or Golgi -- vesicles bud off or join parts of the ER or Golgi.\r\n',NULL,1792,1,2),(###-02-09&&

1562   Q: Are the domains of a transmembrane protein numbered starting from their amino end?\r\n&&

1562   A: Yes. The \'start\' end of a protein is its amino end and the start end of a nucleic acid is the 5\' end (For a gene, which is double stranded, you count from the 5\' end of the sense strand.)\r\n',NULL,1793,1,2),(###-02-10&&

1563   Q: I have a question about Basal Lamina.  On this week\'s recitation problems, specifically problem 3B-1, we are asked what happens if you stick a transcytosing vesicle on its way from the BL side to the apical side into a solution of glucose; specifically, which direction the glucose will flow.  The answer was that it can be either way, implying that it depends on the concentration of the glucose solution and the vesicle.\r\n\r\nI am wondering why the concentration of glucose in the vesicle isn\'t 0. The vesicle is coming from the BL side, implying that it budded off from the BL.  Since glucose is constantly flowing from the BL to the capillaries, wouldn\'t this imply that the BL\'s glucose concentration is a steady-state zero, and thus that all solution glucose concentrations are larger?  In short, I am asking, what are the BL steady-state glucose concentrations?\r\n&&

1563   A: We are ignoring the question of what is inside the vesicle. However it would probably have some glucose in it, because the steady state on the BL side is not zero. Glucose is flowing from the BL side of the cell into the capillaries, so the level must be signif. about zero.\r\n',NULL,1794,1,2),(###-02-10&&

1564   Q: I was a little confused on the difference between cadherins and integrins. Both are transmembrane proteins involved with cell-cell adhesion. So how does one determine whether an integrin or cadherin is involved in cell-cell linkages?\r\n&&

1564   A: Cadherins are involved in cell-cell connections, but integrins are involved in cell-ECM connections. See the diagrams on your handouts and in the books.\r\n',NULL,1795,1,2),(###-02-10&&

1565   Q: I have a quick question regarding one of the problems from 2R-10 (B). Te Amino end and the Carboxyl end are in the cytoplasm and the protein has 10 transmembrane regions.  If the drug Ouabain (drug B) affects binds to the extracellular region, why is the region between transmembrane 1 and 2 not a correct answer.  I figured the protein would look somewhat like the below figure (sorry for the graphics).\r\nThe answer key states the drug will only bind to regions btw (TM 3&4) and (TM 5&6); however, I thought the possible locations should be between TM (1&2), (3&4), (5&6), (7&8), and finally (9&10).  &&

1565   A: You have to look at the diagram in the problem and see that not every domain that sticks out on the correct side is needed to bind ouabain. \r\n(Look at the +++ and - symbols on the right.) You only get binding if the regions between 3&4 and 5&6 are from the Ouabain sensitive enzyme.\r\n',NULL,1796,1,2),(###-02-10&&

1566   Q: I am not understanding 2R-5. The questions says \"if the intracellular Na+ concentration is in the normal range (relative to the outside), then this exchanger (H+/Na+) could be used to...\" The answer is: \"Raise cell pH. (H+ leave, so [H+] goes down.\"\r\n\r\nBy normal range does it mean the

[Na+] that is established  by Na+/K+ pump? This would make sense because in this circumstance the large [Na+] from outside the cell could go into the cell thereby pushing H+ out of the cell which would raise cell pH.\r\n&&

1566    A: You\'re right. The Na+/K+ pump is assumed to run all the time and maintain hi [Na+] outside the cell and low [K+] inside.\r\n',NULL,1797,1,2),(###-02-11&&

1567    Q: 1) Normally focal adhesions are found in cells on the go? (like leukocytes).  One would not normally find focal adhesions on the basal side of an epithelial cell.  Becker shows focal adhesions in the book, but that is just to ?put things together,? correct?\r\n\r\n2) The plaques in desmosomes refer to the anchor protein(s) that act as the middle man between the cytoskeleton (IF) and cadherin.  The plaques do not refer to the IF, correct?\r\n\r\n3) The basal lateral side is the lateral side of an epithelial cell that includes the entire lateral side after the tight junctions, correct?\r\n\r\n4) The basal lamina serves as a structural support that maintains tissue organization and acts as a permeability barrier that regulates the movement of molecules as well as cells.  Because it is a structure (a thin sheet), the basal lamina has a top, side and bottom.  What is the naming convention for referring to the various parts of the basal lamina?\r\n\r\n5) Are Na+/K+ pumps found on both the apical and basal lateral side of an epithethlial cell, or only on the basal lateral side?\r\n\r\n6)When you mentioned that iron \"diffuses\" out an endosome and into the cytoplasm of the cell, did you mean that iron diffuses through a channel with H+, and H+ is providing the gradient?  This would be a similar set-up H+at the Na+/glucose symport and therefore be an example of secondary active transport?  I am not sure about this because my first thought was that the concentration of iron in the cytoplasm is low (we really did it? electron transport chain?) and therefore both H+ and Fe2+ would be going down their gradient from the endosome into the cytoplasm of the cell.  But I do not know when iron changes from an oxidation state from Fe2+ to Fe3+.  Can you please clarify if this occurs in the DMT1 channel or inside the endosome?\r\n\r\n7) What did you mean by some of the transport proteins have? enzymatic activity? and which transport protein that we talked about have? enzymatic activity? I think you were referring to a permease, but if you could further clarify that would be really helpful.\r\n\r\n8) In a freeze fracture, you can not see peripheral proteins on the E face or the P face, since you are looking ?head on? into the middle of the bilayer?\r\n&&

1567    A: 1) Yes.\r\n\r\n2) Yes.\r\n\r\n3) Also the bottom.\r\n\r\n4) It is so thin no one bothers.\r\n\r\n5) BL only.\r\n\r\n6) Diffuses is the wrong word. It requires a transporter. Whether it is active or passive I don\'t know for sure. Probably active.\r\n\r\n7)You can consider a transporter to catalyze Xout to X in or vice versa.\r\n\r\n8)Right.\r\n',NULL,1798,1,2),(###-02-12&&

1568    Q: I was just wondering where the GLUT 4 protein is stored when it is brought into the cell

1568    A: In vesicles, possible special ones and possible part of endosomes.\r\n',NULL,1799,1,2),(###-02-12&&

1569    Q: Problem 1-22.  When the spectrin/ankyrin/actin web was first introduced in class, we discussed it as a form of minimal cytoskeleton just under the plasma membrane of RBCs.  In this problem, however, we are seeing a spectrin/actin network in a colon epithelial cell. Are spectrin/ankyrin/actin networks found supporting the plasma membranes of most cells, or just RBCs?\r\n\r\nConversely, do RBCs also have normal MT/MF/IF based cytoskeletons like most other cells?\r\n\r\nor only the spectrin network?\r\n\r\nAnd if it is the case that some cells have both types of skeletons (spectrin + MT/MF/IF), how do the two skeletons work together to hold everything in

place -- i.e. how are the skeletons connected to each other, and how are the membrane proteins connected to them?\r\n&&

1569    A: Something similar is supposed to be in most cells. I should have made it clear that this is like RBC spectrin, but not exactly the same.\r\n\r\nAbsolutely not.\r\n\r\nYes.\r\n\r\nGood question. I don\'t know how it\'s all connected. It\'s hard to investigate the spectrin part when the regular cytoskeleton is there.\r\n\r\n',NULL,1800,1,2),(###-02-12&&

1570    Q: My current understanding is that \"structural\" transmembrane proteins (cadherins/integrins) are attached directly to filaments (for greater structural strength), while \"non-structural\" TM proteins (e.g. connexons, ENaC) are attached to the weaker spectrin network via ankyrins, and the spectrin network then attaches indirectly to the strong MT/MF/IF network through some other means (?).  Is this picture more or less correct?\r\n\r\nAlso, why is ENaC bound to spectrin directly in this problem, instead of to ankyrin?  I thought the ankyrin middle-man was required.&&

1570    A: Not sure about the last bit. Connexons etc. are connected to the cytoskeleton, but I assume it\'s indirect. Whether it\'s exactly as you say, I don\'t know. We can look it up if you like.\r\n\r\nThis problem doesn\'t rule out a middle man (although it is implied there is none). Glycophorin is linked to the spectrin network by 4.1, not ankyrin. So a middle man doesn\'t have to be ankyrin or even exist. Other proteins can bind to spectrin.\r\n',NULL,1801,1,2),(###-02-12&&

1571    Q: Problem 2R-3, part C.  Some pictures in Becker show the nucleus with bound ribosomes (see p. 539, 7th ed), just like the rough ER.  If this \r\nis true, why isn\'t Nucleus listed as an answer here? What\'s going on?\r\n&&

1571    A: There are no ribosomes inside the nucleus according to most. There are some on the outer nuclear membrane (as in picture in Becker. The outer nuclear membrane is continuous with the rough ER, and usually considered part of it. The question asks if the nucleus CONTAINS functional ribosomes. It is correct they can be attached outside, but they aren\'t inside.\r\n',NULL,1802,1,2),(###-02-20&&

1572    Q: Does a co-transport imply that both molecules or ions are being transported in the same direction, or does it depend on the concentrations, as in all other secondary active transports? Are all co-transports secondary active or could they be facilitated diffusion?\r\n&&

1572    A: I think there is disagreement on this issue -- whether to call certain transporters 2nd act. transp. or facil. diffusion. If the usual biological function is to push one substance up its gradient, while the other flows down (providing the energy), then transport is called secondary active, whether it is symport or antiport. If the function depends on the concentrations, as with the anion exchanger, there are different opinions on what to call it.\r\n',NULL,1803,1,2),(###-02-23&&

1573    Q: I\'m having some trouble visualizing the start/stop transfer sequences on a multi-pass protein and how they would get inserted into the membrane. When the 1st stop transfer/hydrophobic region has been translated, the rest of the protein is made in the cytoplasm.  Then when we hit another hydrophobic/start sequence, how does this get back into the translocon? Does the translocon open and close in the middle of this

1573    A: It\'s a good question, and I don\'t know the answer. Presumably, the hydrophobic sequence \'gates\' or opens the translocon to the cytoplasmic side. (Which implies, as you say, that the translocon closes once a hydrophobic \'stop\' region gets to it, or slides out of it. (Which doesn\'t

explain how the chain gets started in the first place.) Perhaps a hydrophobic region opens it if it\'s closed, and closes it if it\'s open! The main point I am trying to make is that chains go through in successive loops, and don\'t wiggle across the membrane and back, one end first.\r\n',NULL,1804,2,2),(###-02-23&&

1574    Q: I\'m a little bit confused about problem 3-3 part B.  When you describe the 3rd condition of bacterial vesicles, you talk about using right side out vs. wrong side out PM vesicles.  I assumed when doing the problem that right side out meant what a vesicle would look like for endocytosis, with the extracellular domain on the inside, but the answer key said that right-side out meant keeping the cytoplasmic region on the inside.  I was just wondering if you could give me any advice about what to assume when using the \"right\" orientation

1574    A: No, you have it backwards. Endocytosis turns the membrane \'inside out.\' \r\nUsually \'right side out\' means that you break the membrane into pieces, and reseal it with the same curvature. If it was a vesicle, what was inside is still inside. If it was a piece of plasma membrane, what was sticking out of the cell is still sticking out.\r\nWe try to be clear in writing problems. Perhaps it\'s better to use other terminology, such as lumen side in or cytoplasmic side in or out. \r\n',NULL,1805,2,2),(###-02-23&&

1575    Q: Is the presence of an additional localization sequence the only difference between a default vesicle and a secretory vesicle, since both will release their proteins into the ECM?  What\'s the point of that additional localization sequence if it would have been secreted regardless?  And, can both vesicles carry transmembrane proteins

1575    A: Both vesicles can carry TM proteins. Some vesicles hold onto their contents until the cell gets a signal, usually from the outside, that the contents are needed in the blood stream or elsewhere, outside the cell, or the TM proteins need to be inserted in the plasma membrane. When the signal arrives, the vesicle fuses with the plasma membrane. This allows fast action in response to the signal. A special sequence or patch is needed in the corresponding protein for it to end up in a vesicle that will \'wait\' and not fuse immediately with the plasma membrane.\r\n',NULL,1806,2,2),(###-03-02&&

1576    Q: I\'m a little bit confused about proximal control elements.  Do basal TFs or regulatory TFs bind to them

1576    A: Regulatory. If it\'s called a \'control element\' it\'s a binding site for regulatory factors.',NULL,1807,2,2),(###-03-02&&

1577    Q: I\'m not exactly clear on what the difference is between micrococcal nuclease and DNase I. Does micrococcal nuclease always cut nucleosomes in the same spot

1577    A: These two enzymes have different specificities. Micrococcal cuts in the same spot in the linker every time, if you use a little of it. DNase I cuts exposed DNA in general, and shows no preference for any particular part of the linker. DNase I is usually used to distinguish higher orders of folding from each other.\r\n',NULL,1808,2,2),(###-03-03&&

1578    Q: When you have a particular gene that isn\'t being transcribed in a cell, do you expect nucleosomes/histones to be associated with their regulatory sequences, sequences that in other cells that are transcribing the gene would be hypersensitive

1578    A: Yes.',NULL,1809,2,2),(###-03-09&&

1579    Q: What exactly is a hormone?  Is it a protein

1579    A: hormone = endocrine. There are many different hormones, and they differ in chemical composition. Only some are proteins. I suggest looking \'hormone\' up in a text or online.\r\n',NULL,1810,2,2),(###-03-09&&

1580    Q: One of the animations makes it seem as if the G protein is floating around in the cytoplasm when it is inactivated instead of in the membrane--which is usually the case

1580    A: It\'s attached to the membrane by a lipid tail, according to my sources. You can always look it up yourself to see if your sources agree. (I recommend using the Pubmed bookshelf. See  the useful web sites page for\r\ninstructions.)\r\nAnimations often differ in details, sometimes on purpose (to simplify and/or include details we ignore)  and sometimes through misunderstanding by the artist, or technical difficulties in making the animation. So you always have to view them with a dose of caution. \r\n',NULL,1811,2,2),(###-03-09&&

1581    Q: So even though proximal control (cis-acing) elements bind TFs that can inhibit or promote transcription, they are never referred to as enhancers or silencers?\r\n\r\nAlso, is it in general only the hormone receptors that bind to the proximal control elements?\r\n&&

1581    A: Those terms are generally reserved for distal control elements. But never say never!\r\n\r\nI think other things may too. Hormones\r\ngenerally bind to proximal, not distal, control elements.\r\n\r\n',NULL,1812,2,2),(###-03-09&&

1582    Q: Is it the same enzyme that adds the poly A and cleaves the primary transcript

1582    A: The process of cutting and adding polyA is carried out by a large complex containing many peptides. Different peptides do the cleavage and addition, but they all have to bind at once for anything to happen. For details see the texts by Lodish or Alberts.\r\n',NULL,1813,2,2),(###-03-09&&

1583    Q: In question 4R-6, the answer describes the sequence that directs the enzyme to go to the intermembrane space of the mitochondria as a signal peptide--I didn\'t think that these LSs were referred to as signal peptides--could you clarify this for me

1583    A: The term \'signal peptide\' is sometimes used in a very broad sense to mean any sequence that is a localization signal. That would include transit peptides. It is also used in a more narrow sense to mean the LS that directs proteins to the translocon complex. I have tried to be careful to use the term in its more specific sense but have sometimes used it in the broader sense. It should be clear from context what is meant. I\'ll check the answer and see if it needs editing.\r\n',NULL,1814,2,2),(###-03-09&&

1584    Q: My question is about ligand gated channel signaling.  I understand that the ligand binds to its receptor and the result is that some ligand gated channels open, but is the receptor the actual channel and the change in shape is simply the opening of the channel?  Or is the receptor separate from the channel but it somehow triggers the channel\'s opening?\r\n&&

1584   A: Some receptors are part of a channel. This gives very fast, direct signaling. Other receptors are separate from the channel, and cause opening of the channel using a G protein or other intermediary (or by binding to it themselves).\r\n',NULL,1815,2,2),(###-03-09&&

1585   Q: My question is about lipid soluble signals.  Since the only cis acting regulatory element we talked about in reference to the intracellular receptors were hormone response elements, I was wondering if most of the lipid soluble signals are hormones or is that just what you decided to focus on?  Are there other binding sites for these receptors?\r\n&&

1585   A: I think all the lipid soluble long distance signals are hormonal. \r\n(Steroids or thyroid hormone.) Prostaglandins are fatty acid derivatives, but they act locally, and by a different pathway. So probably I should have said all the lipid soluble endocrines (hormones) act this way.\r\n',NULL,1816,2,2),(###-03-09&&

1586   Q: Looking over my notes, I was having difficulty understanding why DNA cut by Micrococcal Nuclease produces a ladder pattern when on a \r\nstained gel. I would have imagined that there would be a single, but very thick, band at 200 bp.\r\n&&

1586   A: You just add a little and it doesn\'t cut at every possible site (ever 200 bp). If you add enough to cut every 200 pb it cuts in additional places and degrades the linkers.\r\n',NULL,1817,2,2),(###-03-12&&

1587   Q: Would intracellular receptors ever be referred to as activators or repressors

1587   A: Some people reserve the term repressor for bacterial operon repressors, but others use repressor for any protein that turns off transcription and activator for anything that turns it on. They also use the terms in even more general ways to say a \'translational repressor\' etc.\r\n',NULL,1818,2,2),(###-03-12&&

1588   Q: A) I\'m a little confused about the terms \"alternative splicing\" and \"alternative processing\". I had gotten the impression from lecture that they\'re not the same, but one of the problems and one of the Q&As seem to suggest that they are the same. Can you please clarify this?\r\n\r\nB) Also, just to make sure I get this, does it make sense to say that the primary transcript of an alternatively spliced gene is sort of the \"ancestor\" of all mRNAs that result?\r\n\r\nC) That is, whatever different versions of the protein you get from alternative splicing, they are all traced back to the same primary transcript. On the other hand, for alternative processing, the different poly A additions give rise to different primary transcripts so that all different versions of the protein cannot be traced back to the same original primary transcript?\r\n\r\nD) In a similar vein, can alternative processing and alternative splicing occur in the same gene?\r\n\r\nE) That is, can a gene give rise to two different primary transcripts due to different poly A additions with those primary transcripts then spliced in different ways?\r\n\r\nF) Lastly, can you explain what you mean in the solution to 4-12 when you say that, \"if the amount of mRNA stays the same, but the rate of protein synthesis changes, you assume translation is being regulated\"?\r\n\r\n&&

1588   A: A) They are usually used interchangeably. It depends on the fine points of the case which is a better term.\r\n\r\nB) Yes. The term usually used is precursor, but it\'s the same idea.\r\n\r\nC) Anything that modifies a transcript counts as processing. That includes polyA addition and splicing. It\'s true that once you add polyA, that limits the splicing options thereafter.\r\n\r\nD) Yes.\r\n\r\nE) Yes.\r\n\r\nF) I don\'t have the book in front of me, so I hope this is clear and the issue you meant. If

you have more mRNA, you have more translation. But that counts as transcriptional regulation since you made more mRNA, and the faster translation was just a consequence of that. If you have the same amount of mRNA, but get more protein out of it, then translation (= protein synthesis) per se is going faster, so translation is being regulated, that is, speeded up. More ribos are reading the same mRNA more often or something like that.\r\n',NULL,1819,2,2),(###-03-12&&

1589    Q: I have a question about core promoters and basal transcription factors. If basal transcription factors are the same in all cells, does that mean that the core promoters are as well?\r\n&&

1589    A: Promoters are not all the same, but the promoter of any particular gene is the same in all cells. There are different basal TFs, and not all the same ones are probably used on all promoters. But every cell is thought to have all the basal TF\'s needed to transcribe all its genes.\r\n',NULL,1820,2,2),(###-03-12&&

1590    Q: A) You mentioned that as an alternative to splicing, different proteins can be made from the same gene if transcription starts at different points on the gene. Does this imply that there may be multiple promoters for a single gene?\r\n\r\nB)Would these promotors be in introns?  For such a case, would one promotor have a higher affinity for regulatory proteins? \r\nMust there also be multiple sites that code for initiation of translation?\r\n\r\nC) Does every gene have only one stop translation codon, but possibly several start translation codons (because that\'s just AUG for met anyway, right?)?\r\n\r\nD) If there is only one stop translation codon, then should we assume that the \"last\" exon containing this codon is always included in any variation of final transcript?\r\n\r\n&&

1590    A: A) Depends on what you call a promoter. Could call it same promoter with different start points.\r\n\r\nB) Don\'t know about this. A different pt. for starting transcription could just alter the 5\' UTR or it could change where translation starts.\r\n\r\nC) Could be alternative stops (for translation) depending on splicing etc.\r\n\r\nD) I think this is usually the case, but I\'m not sure. Just about anything you can think of happens!\r\n',NULL,1821,2,2),(###-03-26&&

1591    Q: When the action potential is fired, there is a influx of Na+ toward the cell which makes the cell to depolarize and the outflux of K+ results in cell repolarization. So how is it possible that the ion concentration does not change after this?\r\n&&

1591    A: Because the total numbers of ions is very large, relative to  the number of ions that move. Overall, the concentration is not significantly changed.',NULL,1822,3,2),(###-03-31&&

1592    Q: Why in Parasympathetic (PS)nerves, the first neuron is longer and the second one is shorter and it is the opposite case in sympathetic (S) nerves?\"\r\n&&

1592    A: I don\'t know the answer to that; as far as I\'m concerned, it\'s just the way it is! If I find out any rationale, I\'ll let you know.\r\n',NULL,1823,3,2),(###-04-06&&

1593    Q: I\'m a little confused about problem 8-15: do we have to know the specific pathways for the different senses?  This problem seemed to assume (unless I\'m missing something) that we\'re familiar with the pathway for smell reception.&&

1593    A: You are NOT supposed to know how smell works. However, you should be able to do the problem, once you look up the details of the pathway.\r\n\r\nI forgot that the problem assumes that

you know. We don\'t cover exactly the same things every year, so this sometimes happens. Next time I\'ll put a note in the book.\r\n',NULL,1824,3,2),(###-04-06&&

1594   Q: For the post synaptic region of a neuron in a two neuron system, are there any voltage gated ion channels present, or are there only ligand gated ion channels?  And then the voltage gated ion channels are found further \"down\" the neuron - past the axon hillock or something?\r\n&&

1594   A: The voltage gated channels are not at the synapse, but at the axon hillock and the axon.\r\n',NULL,1825,3,2),(###-04-07&&

1595   Q: When talking about the methylation of H3\'s lysines for the MRGs, are these for the nucleosomes that hold all of the gene\'s transcribed DNA, or the promoter regions?  \r\n&&

1595   A: The methylation of the H3 lysines of the master regulatory genes is on the nucleosomes that are wrapped by the promoter regions of the master regulatory genes.',NULL,1826,3,2),(###-04-07&&

1596   Q: I am looking over notes for lecture 15.  It seems paradoxical to me that the mechanism of visual phototransduction should be metabotropic, rather than ionotropic.  Metabotropic transduction is \"slow\", but vision strikes me as fairly \"fast.\"  How slow is \"slow\" exactly?  Also, how is it possible that a complex, multistep metabolic process such as regeneration of the \r\n11-cis-retinal chromophore can keep pace with the innumerable number of photons hitting the retina every second?\r\n\r\nI just have trouble imagining how a sluggish, chemically mediated process that involves molecules fortuitously bumbing into each other can produce a fast and ongoing sensation such as sight.\r\n&&

1596   A: The answer to this good question is unfortunately not that straightforward, but let me give you at least part of the answer.  In fact the second messenger signaling system in photoreceptors is much faster than most GPCR systems.  Fro one the second messenger, cGMP, directly operates a channel rather than initiating a downstream chemical cascade involving kinases and so forth.  So while it\'s chemical ti can work very quickly to produce an ion flow and an electrical signal.  The main advantage of using a chemical system is that there can be tremendous amplification and this, among other processes, is what allows us to be sensitive to light intensities that cover 6 orders of magnitude of luminosity.  a reasonable tradeoff for a few microseconds of speed.\r\n\r\nThe second  reason for a receptor based chemical signaling system helps to answer your second question.  In fact there is so much retinal in the retina, even in a single photoreceptor, that the brightest light, what we call a bleaching flash, nonetheless activates less than 1% of the available opsin ( and retinal).  It is virtually impossible to saturate the response of photoreceptors, although \"blinding\" flashes of light can saturate other parts of the visual processing circuitry.\r\n\r\n',NULL,1827,3,2),(###-04-16&&

1597   Q: On handout 19C, you emphasized that action potentials in cardiac muscle are different from those in skeletal muscle.  (The longer refractory period prevents cardiac tetanus.)  However one of the points Dr. Firestein emphasized is that action potentials are invariant -- every AP is the same as every other.  Is this a contradiction?\r\n&&

1597   A: All AP\'s in the same tissue are the same all the time. But cardiac muscle AP\'s are different from other AP\'s.',NULL,1828,3,2),(###-04-20&&

1598   Q: I\'m a little bit confused about the arrangement of actin and myosin in smooth muscle--are the dense bodies different than the actin and myosin bundles

1598   A: The dense bodies are the anchors for the actin in the bundles.\r\n',NULL,1829,3,2),(###-04-20&&

1599   Q: I had a few questions about the role of calcium in various muscle cells. In smooth muscle cells, the main source of calcium during contraction is from the outside because smooth muscle APs involve voltage-gated calcium channels. Since both pacemaker and contractile cardiac muscle cells use voltage-gated calcium channels, is the calcium for cardiac muscle contraction also mainly from the outside?\r\n\r\nYou mentioned that the IP3 pathway does not play a role in skeletal muscle contraction. Is this because skeletal muscle just doesn\'t use this pathway/lack the right G protein, or is it because IP3-induced calcium release is insignificant compared to how much calcium is actually needed for contraction?\r\n&&

1599   A: The Ca++ probably comes from the outside through other channels as well as the AP (in smooth muscle). For cardiac muscle contraction, most of the \r\nCa++ is from the SR/ER as in skeletal muscle.\r\n\r\nI don\'t think skeletal muscle has receptors for hormones that generate IP3. I would have to check an advanced text to be sure.',NULL,1830,3,2),(###-04-20&&

1600   Q: I think I understand what a twitch is, but I\'m less sure what counts as \'contraction\' or when the units of contraction (Twitch) add up to 1 full contraction. Does this have to do with each time the myosin does a power stroke? or each time the bridge cycle is completed?\r\n&&

1600   A: twitch = single contraction/relaxation cycle in a muscle fiber.\r\nContraction = process by which a muscle creates force = net result of multiple twitches (summed).',NULL,1831,3,2),(###-04-20&&

1601   Q: Dr. Firestein told us about the law of dynamic polarization. He did say there were exceptions. Is the case in problem 8-6 such an \r\nexception? Or is this a hypothetical?\r\n\r\nI just want to make sure I understand correctly from the answer to that problem that Action Potentials can \'move\' in either direction.\r\n\r\nDynamic polarization means APs can only *fire* signals in one direction.\r\n\r\nDoes an AP\'s movement in a given direction mean that the depolarization is spreading in that direction?\r\n&&

1601   A: This is an artificail, experimental situation.\r\n\r\nYes, but they don\'t normally get a chance to move in the \'other\' \r\ndirection. They are triggered from one end only, and can\'t go backwards because of the refractory period.\r\n\r\nUnder normal conditions, this is correct.\r\n\r\nYes. More correctly, the depolarization in one region is triggering another in the next region.',NULL,1832,3,2),(###-04-20&&

1602   Q: How does autonomic input affect cardiac cells? Do the PS and S neurotransmitters bind to pacemaker cells or cardiac muscle cells? And why is this input needed if pacemaker cells automatically depolarize to make a stable heart beat?\r\n&&

1602   A: The neurotransmitters bind to receptors on the pacemaker cells.\r\nYour heart beats at a steady rate without any additional input, but you need to slow or speed up that rate to adjust to what you are doing -- running, sleeping, sitting, etc. \r\n',NULL,1833,3,2),(###-04-20&&

1603   Q: In skeletal, there are nicotinic cholinergic receptors at the endplate.  How do these receptors start off an EPP in the sarcolemma? My understanding is that the nACHRs act as ligand-gated Na+

channels, \r\nallowing the myofibril to reach threshhold, thus triggering separate voltage-gated Na+ channels, exactly as in neurons. Is this correct? (Wikipedia says the nACHR is a non-selective ion channel, and allows Na+, K+ and Ca++ to flow -- which seemed totally counter-intuitive to me, since allowing BOTH Na+ and K+ to flow would have no effect -- the two would cancel each other out.)\r\n&&

1603    A: Wiki is right, but remember that K+ is close to its equil. pot, and Na+ is far from it. So if you let them both move, it\'s mostly Na+ that moves. \r\n(I\'m not sure about the Ca++ part, but it also would move in.)\r\n',NULL,1834,3,2),(###-04-20&&

1604    Q: Is it correct that the nACHR is the only significant input into skeletal muscle?\r\n\r\nWhat about hormones/other neurotransmitters/etc.?\r\n&&

1604    A: To trigger contraction, yes.\r\n\r\nHormones yes, but they don\'t affect contraction, No other NT\'s.\r\n',NULL,1835,3,2),(###-04-20&&

1605    Q: From 16B I see that the receptors on smooth muscle can be either Epinephrine, AcChol/Muscarinic, or Norepinephrine. I understand how epinephrine transduces its signal (through IP3-->Ca++ or cAMP-->MLCK, depending on which type of adrenergic receptor is present), but how do the norepinephrine and muscarinic AcChol receptors work? My understanding is that norepinephrine binds to the same receptor as \r\nepinephrine, and that the mACHR is a G-protein coupled receptor that comes in both contractatory (IP3-->Ca++) or relaxatory (cAMP-->MLCK) flavors. Is this correct?\r\n&&

1605    A: There are multiple receptors for both Epi, Norepi, and Ac Chol. Some activate Adenyl cyclase, some inhibit it, and some activate PLC.\r\n',NULL,1836,3,2),(###-04-20&&

1606    Q: It seems to me that smooth muscle isn\'t really \"electrically excitable\" in the true sense, since all of the Ca++ release seems to be chemically induced (IP3) rather than voltage-induced. None of the above receptors seem to set off polarization/depolarization waves in the membrane, instead they just activate a G-protein. Am I missing something?\r\n&&

1606    A: Some smooth muscle generates an AP; has voltage gated Ca++ channels.',NULL,1837,3,2),(###-04-20&&

1607    Q: In the heart, pacemaker cells generate regular polarization/depolarization waves, and this stimulates contraction in the contractile cells. How exactly does this occur? My understanding is that the pacemaker cells spontaneously generate regular pacemaker potentials (using Ca++ as the cation, instead of Na+) which travel along the membrane and spread to adjacent cells\' Na+membranes via gap junctions. The wave travels along to the contractile cells \r\n(which use BOTH Ca++ AND Na+ as cations in their wave), where the sudden influx of cations causes voltage changes that then trigger FURTHER release of Ca++ from the SR. The Ca++ then binds to troponin to trigger contraction. Is this correct?\r\n\r\n&&

1607    A: Sounds right. AP in contractile starts with Na+ and then Ca++.\r\n',NULL,1838,3,2),(###-04-20&&

1608    Q: Am I correct in thinking that there are 2-stages of Ca++ release, first from membrane channels to CAUSE depolarization (or prolong it), and second from SR channels, in RESPONSE to the

depolarization?  This is confusing!\r\n\r\nHow is it that Ca++ released by membrane channels can \"prolong the refractory period\" (i.e. prevent further contraction) when Ca++ is the very cation used to TRIGGER contraction?  Isn\'t this somewhat paradoxical?\r\n&&

1608    A: Yes, but that\'s how it is. Note we did NOT go into this in class. \r\nPartially because there is \'Ca++ induced Ca++ release.\" All of this is explained clearly in more advanced texts. \r\n\r\nCa++ allows myosin and actin to form bridges. Ca++ also allows the AP in\r\nheart to last longer. What\'s the contradiction?? Ca++ does both. If it turned OFF contraction, and prolonged it, THAT would be a contradiction.\r\n',NULL,1839,3,2),(###-04-20&&

1609    Q: Would the methylation of k3 and k4 of certain master regulatory genes in developing tissue be referred to as epigenetic changes?  thank you!\r\n&&

1609    A: Yes.',NULL,1840,3,2),(###-04-20&&

1610    Q: Only the pacemaker cells of cardiac muscle are innervated by the autonomic nervous system

1610    A: Yes. Only the SA node.',NULL,1841,3,2),(###-04-21&&

1611    Q: When you have the AP that travels down and causes the opening of Ca++ channels in the bouton of a neuron, is the voltage change at the bouton an AP or a graded potential?  Because after the AP leaves the axon, it can no longer be regenerated, right?  so wouldn\'t you have to refer to the potential that opens the Ca++ channels as a graded potential

1611    A: The Ca++ channels are opened by voltage changes. The AP dies out at the end of the axon when it reaches the last voltage gated Na+ channel.\r\nThe AP probably ends abruptly, so the potential at the end is not considered a graded potential.\r\n',NULL,1842,3,2),(###-04-21&&

1612    Q: Do all muscarinic receptors use the same G-protein and the same second\r\nmessenger?\r\n\r\nCan a particular receptor be coupled to different G-proteins in different cells?\r\n&&

1612    A: No.\r\n\r\nI don\'t think so. I think there is a match (in actual physical fit) between the receptor and the G protein.\r\n',NULL,1843,3,2),(###-04-21&&

1613    Q: I have a quick question about Ca++ in the Smooth Muscle. It is my understanding that most of the Ca++ in smooth muscle comes from \r\noutside the cell.\r\n\r\nYet, the only pathway that I can think of that\r\nreleases Ca++ in smooth muscle is the IP3 pathway. I thought that the IP3 binds to receptors on the ER, causing it to release Ca++.\r\n\r\nI guess I\'m wondering: \r\na) Am I misunderstanding the IP3 pathway?\r\n\r\nb) Is there a different pathway that causes Ca++ to be released from outside?\r\n&&

1613    A: Yes.\r\n\r\nThat\'s correct. We did not discuss how Ca++ could enter smooth muscle from the outside, because there are many ways to do it and I wanted to avoid the issue. However you are right to be mystified! I can see that next year I will have to say something to make this all clearer (you\'re not the only one to realize something is left out!).\r\n\r\na) No.\r\n\r\nb) Yes. (It can be a consequence of the Ca++ released by the IP3 pathway or channels in plasma membrane can be opened by other means.)\r\n',NULL,1844,3,2),(###-04-21&&

1614    Q: If all Somatic neurons are only excitatory, do we consider interneurons to be part of the somatic system?\r\n\r\nOr is the somatic system only motor neurons?\r\n\r\nIf that is the case, does it matter that we cannot really classify interneurons?\r\n&&

1614    A: Motor neurons are all excitatory. Interneurons can be otherwise. Note that the interneurons are in the CNS, the spinal cord, not in the peripheral NS.\r\n\r\nYes.\r\n\r\nSee above.',NULL,1845,3,2),(###-05-04&&

1615    Q: Can you please provide specific examples of a single hormone that is secreted by more than one gland (besides sex hormones being secreted by the gonads and adrenal cortex)

1615    A: Somatostatin is secreted by the pancreas, HT, and stomach. I\'m sure there are others, but that one comes to mind. The sex hormones secreted by the gonads are actually different (& more potent) than those secreted by the AC, but they are very similar.\r\n',NULL,1846,4,2),(###-05-06&&

1616    Q: In the notes it says that there are multiple types of MHC protein made per cell in a person-- are all these types recognized by CD4 or CD8 proteins

1616    A: Yes. Due to complications we did not discuss, your T cells only recognize your own MHC\'s.',NULL,1847,4,2),(###-05-07&&

1617    Q: Do T cells go through class switching

1617    A: No.',NULL,1848,4,2),(###-05-11&&

1618    Q: From everything I have read, it seems as though Tc cells can attack and do away w/infected cells w/o the help of Th cells. (Tc recognizes the MHCI-Ag complex, attaches, injects toxins, etc.). So in order for an infected cell to be killed, why do we need Th? In other words, are the Th\'s there only for the aftermath (clean-up, etc.)? Or is there something that I\'m missing (do the Th cells do something that then allows the Tc to bind to and toxify the infected cell)?\r\n&&

1618    A: Helper T\'s are needed to activate cytotoxic T\'s. We did not explain how, because it is complex. But they are absolutely necessary. \r\n',NULL,1849,4,2),(###-05-11&&

1619    Q: I\'m rereading the lectures for the final and came across something I had a question about. In Lecture 20, it is written that the IC raises the body\'s set point for temperature to cause a fever because \"high temperatures prevent bacteria from obtaining iron from host and improve immune function.\"  I was just wondering why people take tylenol, advil, etc. to reduce their fever and why doctors use cooling blankets, the Arctic Sun, etc. to reduce fever if it is actually helping fight the basteria or disease? I\'m sure very busy writing exams and such, but if you have time to answer this I\'d really appreciate it!\r\n&&

1619    A: Some people feel that taking aspirin etc. is a mistake for a mild fever, and you should wait it out in order to wallop the bacteria better. However this is not based on clinical results, but on evolutionary reasoning. \r\nSince people feel miserable with a fever, they (and their doctors) usually feel it isn\'t worth the suffering. It certainly is important to cool off if the fever gets dangerously high -- then the fever can damage the patient as well as the bacteria. If someone ever does a clinical study and proves that patients with a mild fever get better faster without aspirin, then doctors may change their advice.\r\n',NULL,1850,4,2),(###-05-11&&

1620    Q: I am confused about the way the releasing factor and tropic hormone trigger the AC to release Aldosterone, and how the activation of Angiotensin via secretion of Renin from the kidney fits in with this scheme?  Maybe I\'m getting stuck up on wanting the Aldosterone to be made by the AC if we say it\'s released by the AC...  So my question really is what is the role of the AC here when it seems like the sympathetic signal HT  -->  kidney  would be enough to produce Aldosterone, as the Angiotensin is already in the blood?\r\n&&

1620    A: Aldosterone is made by the AC. ACTH may have some influence, but the main trigger for synthesis and release of aldosterone is Angiotensin II. \r\nActive angiotensin II is NOT in the blood without renin. Renin is needed to activate a precursor.\r\n',NULL,1851,4,2),(###-05-11&&

1621    Q: Question about 12R-15 B-1: Why is the high [phosphate] in the ECF relevant to the question? Shouldn\'t only high [phosphate] in the filtrate saturate the co-transporters for\r\nNa+/Phosphate?\r\n&&

1621    A: Phosphate is a small molecule, and if it is high in the ECF it\'s high in the blood and therefore in the filtrate.\r\n',NULL,1852,4,2),(###-05-11&&

1622    Q: About DNA rearrangement/class swictching: Problem 13-10E) Answer Key reads: \"If its decendant cells make IgM after meeting antigen, they must be effector cells, not memory cells.\"  How does this imply that the decendants are effector cells?  Wouldn\'t the memory cell also produce IgM? Are effector cells, but not memory cells, able to undergo \r\nclass switching?\r\n\r\nDoes the final rearrangement of H and L chain genes contain alleles from both maternal and paternal chromosomes?\r\n\r\nIs this similar to crossover in meiosis?\r\n\r\nOnce the rearrangement is complete, is only one chromosome accessible for transcription, or are all the other unused alleles simply degraded?\r\n&&

1622    A: AS far as I know, most memory cells make secondary antibodies (A, G or\r\nE) not M. Effector cells the second time arise from memory cells from the first time, which have already switched. We are simplifying everything, so there are probably some complications we are ignoring.\r\n\r\nNo.\r\n\r\nNot really. Uses some of the same enzymes, but also different ones. Does not involve homologs.\r\n\r\nThe remaining DNA is inactive, like most of the DNA in chromatin. \r\n\r\n',NULL,1853,4,2),(###-05-11&&

1623    Q: So only pieces of proteins that are made inside the cell (not endocytosized or phagocytocized) can be put into MHC 1

1623    A: Generally yes. \r\nThere are exceptions to everything, but at this level, yes.\r\n',NULL,1854,4,2),(###-05-11&&

1624    Q: I\'m having a hard time understanding the mechanism of sweating.  If the Na+ concentration is high outside of the cell, why does it need to be pumped out of the epithelial cells into the lumen of the gland.  Why doesn\'t the Na+ from the ECF, that is higher in concentration, get transported into the lumen of the glands?\r\n&&

1624    A: The ECF isn\'t next to the lumen. To get to the lumen, the Na+ has to leave the ECF and pass through the cells.',NULL,1855,4,2),(###-05-11&&

1625    Q: If someone\'s BMR is low/high, does that mean that their actual blood temperature (body temp.) is lower/higher respectively than someone with > a relatively \"normal\" BMR?\r\n\r\nI\'m trying to figure out if it\'s the body temp. itself that is higher/lower or if it\'s only the critical temp. that is altered in \r\nsomeone with pathologically high/low BMR\r\n&&

1625    A: No. The BMR generates more or less heat, but the body adjusts cooling or heating mechanisms to compensate. \r\n\r\nBody temp itself is not changed except in cases of fever and other pathological states. However if BMR is generating unusual amounts of heat, other mechanisms will kick in more readily (or less) to compensate. See anwser to problem 5-9.\r\n\r\n',NULL,1856,4,2),(###-05-11&&

1626    Q: I\'m a little confused about the blood pressure effects of ADH and aldosterone.  Can they both actually constrict peripheral blood vessels, or is it just the retention of water that leads to a higher blood pressure?\r\n\r\nOne more question: when you talked about the osmolarity receptors that trigger the hypothalamus to produce ADH when your blood is too concentrated, you mentioned that thirst is also triggered--is this signaled through the HT

1626    A: Aldosterone is not vasoconstrictor. ADH is. Both lead directly or indirectly to water retention. \r\n\r\nHT signals both thirst and ADH release.',NULL,1857,4,2),(###-09-24&&

1627    Q: Since the amino acids we talk about are only in the L-forms, we should just write it out in the way that we learned in class. Is there a different form for R? It seemed that you didn\'t emphasize on the difference that much during lecture.\r\n&&

1627    A: Ignore it when writing an AA structure. Few people bother learning how to do it.\r\n',NULL,1858,1,1),(###-09-24&&

1628    Q: Regarding weak bonds between non-polar amino acids in the \"loops,\" you mentioned that hydrophobic force are created by the repulsion of the water molecules, and not necessarily a bond

1628    A: Non-polar molecules are pushed together mainly by hydrophobic forces which are not bonds. The forces arise from thermodynamics, systems tend to resolve themselves ito a situation that maximize entropy, all other things being equal.  The amino acids with apolar side chains get together for the same reason the octane molecules did.\r\nThe only true weak bonds between the apolar side gfroup will be Van der Waals, which is an electrical attraction. Bth these bonds are minor compared to the hydrophobic forces.\r\n',NULL,1859,1,1),(###-09-24&&

1629    Q:  Just to clarify, is an ion-dipole interaction also a polar interaction? One of my TAs said this, but I wanted to make this clear.\r\n&&

1629    A: Yes, it\'s a polar molecule bonding with a charged molecule (ion-dipole) . So one partner is (simply) polar while the other is a charged ion (and also very polar).\r\n',NULL,1860,1,1),('0000-00-00&&

1630    Q: Why we don\'t account for the ATP necessary to transport pyruvate into the mitochondrial matrix to start the Krebs Cycle, since we do subtract 1 ATP per NADH formed in glycolysis for that transport across the mitochondrial membrane

1630    A: There\'s a lot of transporting that must be done in and out of the mitochondria (ATP and ADP for example). Most of this transport is \r\nmediated by coupled entrance and exit flows. For example, the ticket for ATP to get out of the mitochondrion is a transport in of an ADP molecule. A specific transport protein, with binding sites for each of the two molecules,  mediates this \"antiport\" process, which is driven by the higher concentration of on or the other metabolite on one or the other side of the membrane. On the case of pyruvate the antiport partner \r\nseems to be OH- ions, which are in excess inside the mitochondrion due to the proton pumping of electron transport chain. Since the net accumulation of prtons outside the mitchondrion is compromised by this loss of OH- ions inside, so there is an indirect but real ATP cost. We can calculate it: pyruvate oxidation inside the mitochondrion will \r\nproduce 4 NADHs and 1 FADH2 which will pump about 10 protons per electron pair from each NADH, or 40, and let\'s say 7 from each FADH2, for a total of 47. The cost of bringing that pyruvate in is thus only 1/47 of the energy captured from the subsequent oxidation of the pyruvate. That said, there are many transports that use either OH- out \r\nnor H+ in to effect the transport of metabolites (e.g, aKG for amino avid biosynthesis, acetyl-Coa for fatty acid synthesis, etc. It has been estimated that 1 of 5 protons pumped out are used for these transport purposes, with only the other4 being used for ATP synthesis.\r\n',NULL,1861,2,1),('0000-00-00&&

1631    Q: Problem Set #1, Question 21-B, after you hydrolyze phospholipid of microorganism cell, why isn\'t \"several amino acids\" one of the possible answers?  I\'m thinking it could be serine or threonine, both of which have \"OH\" Groups, and would fit with ROH - side group of Phospholipid. &&

1631    A: I had never heard of phosphatidylthreonine as a component of phospholipids. And I don\'t agree with your reasoning that simply because the OH is there, and it is an amino acid, that it will be found in natural phospholipids. What about tyrosine, or fructose? The metabolites that are found in living things are there because they have been found to work, to take care of a useful task. It takes a lot to fashion a phospholipid from glucose, and each step is carefully orchestrated. So why just go off and make some phosphatidylthreonine to insert into a membrane.  That said, I Googled phosphatidylthreonine and came up with 275  hits, and learned that this compound is indeed found in some places. For comparison, phosphatidylserine yielded 733,000 hits.  So that does make 2 amino acids, but still one short of \"several.\" ;).',NULL,1862,1,1),('0000-00-00&&

1632    Q: Is there a limit to the size of the peptide that can be run on paper chromatography (like we can run oligopeptides but not polypeptides)? \r\n&&

1632    A: Yes. If it gets too long (>~ 25? ) it will precipitate in the presence of the organic solvent. ',NULL,1863,1,1),('0000-00-00&&

1633    Q: Problem 1-25 b, I don\'t understand you know that the helix will be looser than a starch helix.  Can you please explain? \r\n&&

1633    A: The looseness re starch is presented as an observation (true), and is included to help push you along the right path to the answer. The angle produced by the alpha-beta link between glucose molecules in starch was presented as pretty severe, but the angle caused by the turn in connecting an equatorial C1 to an equatorial C3 (all -OH\'s in glucose are equatorial except C1 of alpha glucose) in curdlan is milder. The diameter of the helix turns will therefore be larger due to the wider turns in curdlan.  This was a very difficult question. \r\n',NULL,1864,1,1),('0000-00-00&&

1634    Q: Problem 2-2 do we treat pH 6 like pH 7?  Doing this would give the right answer, but I\'m confused why you asked for the charge at pH 6 and not at pH 7, what is the significance? \r\n\r\n&&

1634   A: So you just don\'t spit back a memorized answer for \"pH7\".  If you remember that the pK of the carboxyl group is around 3, or remember that over 99% of the -COOH group is deprotonated at pH7, then you could figure out that changing the H+ ion concentration 10-fold toward protonation would not affect the answer. ',NULL,1865,1,1),('0000-00-00&&

1635   Q: When you denature a protein with SDS or urea can this change the charge?  (maybe by opening up R groups that are charged?) \r\n\r\n&&

1635   A: Opening up? What do you mean by that? The charge of a glutamate side group carboxyl is usually there, whether open (dangling out) or closed (forming an intramolecular ionic bond with a lysine for example). Wait for biochemistry to see exceptions.  Urea disrupts H-bonding, it is thought, nothing to do with charge. It is neutral, does not change the H+ ion concentration, so no effect on the tendency of charged groups state of protonation. SDS on the other hand, binds prttey tightly to all polypeptides, and since it carries a string (permanent) negative charge d from its sulfate groups, imparts a ~ uniform negative charge on every polypeptide, sothe polypeptide-SDS complex (held togheter by many weak bonds, is highly negatively charged. It does not change the the intrinsic charge the polypeptide had due to its particular side groups. But whatever those were, negative of positive, they are now insignificant compared to the negative charge brought in by the SDS. \r\n',NULL,1866,1,1),('0000-00-00&&

1636   Q: Can hydrogen bonding between backbone atoms contribute to quaternary structure? \r\n&&

1636   A: Yes.Especially easy for beta sheets, where secondary structure can contribute to interfacial bonds and therefore quaternary structure. The beta sheet transitions smoothly from one subunit to the other. \r\n',NULL,1867,1,1),('0000-00-00&&

1637   Q: Am I wrong in thinking that we can consider delta G only when we have two coupled reactions, or two reactions that are influencing one another as in the withdrawal of products/substrates?\r\n\r\n&&

1637   A: We can usefully consider delta Go and / or delta G without regard to any other reactions.\r\nFor instance, considered for a single reaction A to B , the delta Go can tell us whether we have a problem or we do not have a problem if our goal is to go from left to right as written. If it is highly positive, then the equilibrium lies far to the left and we have a problem. We can then try to figure out how nature has solved that problem for a living system in which the goal was important, as in a metabolic pathway synthesizing serine or catabolizing glucose to get ATP.  There are two general answers: 1) changing the reaction so it is not  A to B with a positive delta Go but rather A + X to B + Y with a negative delta Go and now therefore the equilibrium lies in the desired direction. 2) withdrawing product B so that the delta G is negative even if the delta Go is positive, another solution. But we identified the problem by considering first just the positive delta Go in isolation. \r\nThe delta G can also be considered for a single reaction without explicitly considering any other reaction. For instance if we measure the steady state levels of the reactants and products in a particular cell or tissue, then we can calculate the delta G and see whether the reaction is going to the right or to the left. We need to know the delta Go for the reaction, but that can be looked up in a book (derived there from someone doing an experiment in a test tube, letting the reaction run to equilibrium and measuring the equilibrium concentrations of the reactants and products).  See problem 4-11.\r\n',NULL,1868,2,1),('0000-00-00&&

1638    Q: Were the combinations of DNA in the 2nd generation 2HL\'s and 2LL\'s because it was grown in a 14N medium? ....Therefore there was no genetic material for H to be uptaken?\r\n&&

1638    A: There was no nucleotides with H to be used to make new strands.',NULL,1869,2,1),('0000-00-00&&

1639    Q: I thought the semiconservative 1st generation H-L helices would break up and copy themselves to make 4 H\'s and 4L\'s, and these would come together in different combinations in a 1:2:1  HH:HL:LL.\r\n&&

1639    A: No. You can\'t make any more H\'s, first of all. You have no heavy raw materials. You only have the 2 you started with. Secondly, there is no randomization of single strands. If a strand is a template strand, the partner stays with it.\r\n',NULL,1870,2,1),('0000-00-00&&

1640    Q: I was initially convinced that changing the pH wouldn\'t make a difference in the mutant mitochondria considering the condition that \"The yield of ATP produced per NADH2 oxidized is the same in the test tubes containing either type of mitochondria.\" My reasoning followed that if oxidative phosphorylation was working equally in both types, then changing the strength of the proton motive force wouldn\'t make much of a difference in increasing the rate of ADP phosphorylation. However, the solution suggests that oxidative phosphorylation is exactly the problem with the mutant mitochondria. I was wondering were my reasoning is incorrect in interpreting the condition to mean that oxidative phosphorylation, specifically in the oxidation of NADH2 to produce ATP along the ETC, is flawed.\r\n&&

1640    A: Oxidative phosphorylation has two parts, the oxidative part that drives the protons, and the phosphorylation part that uses the protons to make ATP. The mutant has a problem with part one, but part two is ok. I think you are lumping the two parts together.\r\n\r\nThe idea here is that per H+, the mutant gets the same amount of ATP made as normal. What is limiting, is the pumping of protons and the H+ gradient. The ETC is too slow, or inefficient, and there aren\'t enough H+ available to run through the ATP synthase. So if you add more H+, you get more ATP. It isn\'t the phosphorylation itself that is the problem, it\'s the oxidative part that drives the phosphorylation that is defective.\r\n',NULL,1871,2,1),('0000-00-00&&

1641    Q: In glycolysis we make ATP from ADP four times but make ADP from ATP only twice. So why doesn\'t this balance? Isn\'t this a \"loose end? Where do we get the ADP.\"\r\n&&

1641    A: The reason to run glycolysis is to to convert ADP to ATP, captruriing the energy from the catabolism of glucose to do so. The ADP is coming from the hundreds of coupled reaction where ATP is used to drive them to the  right as desired. All those biosynthetic pathways . . .  For instance, putting two amino acids together in a growing polypeptide chain takes several ATPs. Adding one deoxynucleotide to a growing DNA chain takes 2 ATPs.  Etc. \r\nAnd if the cell doesn\'t need any ATP, it simply shuts down glycolysis; it is highly regulated by feedback inhibition and feed forward activation.\r\n',NULL,1872,2,1),('0000-00-00&&

1642    Q: For rxn 1 on the handout regarding DNA synthesis: we argued that rxn one goes to the left b/c its \"standard\" delta G is positive. I thought we could only know the direction of the reaction from regular delta G. Does the fact that the \"standard\" delta G for this reaction is very positive allow us to

assume that the regular delta G will be positive(unless the reactant concentration exceeds the product concentration by an enormous amount)?\r\n&&

1642    A: If the standard delta G (delta G zero)  is that positive, it is virtually impossible to have a negative delta G. To get a neg. delta G in this case requires an unreasonable supply of reactant.',NULL,1873,2,1),('0000-00-00&&

1643    Q: I am getting a bit confused with the lexicon of 5\' and 3\'. These only refer respective to template? Are there any other conventions? Is \r\nthere a trick to not getting confused?\r\n&&

1643    A: Every DNA chain has a 3\' and 5\' end. The template and its complement or the strand being made are always antiparallel. This means the new strand is made 5\' to 3\' and the template is read 3\' to 5\'. This is indeed confusing, and I don\'t now any trick to it.\r\n',NULL,1874,2,1),('0000-00-00&&

1644    Q: I\'m confused about the different functions of the enzymes of DNA, particularly lygase and DNA polymerase (as an exonuclease), and especially as they apply to joining Okezaki fragments.\r\n&&

1644    A: Ligase is used to join two adjacent fragments with no gap between them. DNA polymerase, acting as a 5\' to 3\' exonuclease, is required to remove primer. DNA polymerase, acting as a polymerase, is need to extend a chain and fill the gap left by removal of the primer. Once the gap is filled, ligase can join the two chains. Problem 6-14 is a good way to review all of this.\r\n',NULL,1875,2,1),('0000-00-00&&

1645    Q: For noncompetitive inhibitors,  Vmax decreases.  As the inhibitor binds to a nonactive site, some of us think it effectively \'removes\' the enzyme, so there is a smaller apparent [E] which leads to the lowered Vmax (Vm = k3[E]).  However, k3 IS present in the equation, so k3 is basically a measure of how well the enzyme can change ES to E + P (the \'enzymatic power\').  So with this information, is k3 affected by noncompetitive inhibitors?  In Vmax = k3[E], is the decreasing [E] the only reason for the decrease of Vmax? \r\n&&

1645    A: Seems to me you could think of it either way. I believe noncompetitive inhibition is basically defined via the kinetics curve. From the pictures I drew you could say the same amount of grams or moles of enzyme is present at the k3 has been affected in some of them at the concentration illustrated (always marginal). On the other hand, in terms \"active\" enzyme some is there is 100% functional and the rest could be considered 0% functional. Then there\'s a gray area: suppose the noncompetitive inhibitor just inhibits 80% even when bound . . . . so all bets are on. I would not worry about as long as you understand what\'s going on, which you obviously do. \r\n',NULL,1876,2,1),('0000-00-00&&

1646    Q: I am getting very confused by the different terms used to describe nucleotides such a s dXMP or dXTP. In one of the problems (6-10) it \r\nsays ddCTP. I\'m having trouble understanding what that means and also where to find the structure for that.&&

1646    A: I believe the problem (6-11) describes ddCTP thoroughly. It\'s very similar to dCTP -- you have to figure out first what that looks like. Please take a look at handouts 10-1 & 10-2. The structure of C is on 10-1 and AMP, ADP, and ATP are drawn on 10-2. In dXTP or XTP, the \'d\' means the sugar is deoxyribose. If there is no \'d\' the sugar is ribose. The MP means monophosphate, DP diphosphate, etc. X means any of the 4 bases (either A, T, G, or C -- if we are making DNA, or OR A, T, G or C if we are making RNA).',NULL,1877,2,1),('0000-00-00&&

1647 Q: We previously learned that DNA can be renatured by cooling it (after it was denatured by heating). Why then does DNA not renature in PCR during the cooling steps?\r\n&&

1647 A: Because of the concentrations involved. There is a very high conc. of primer, so the DNA is much more likely to hybridize with primer than its full complement.\r\n',NULL,1878,2,1),('0000-00-00&&

1648 Q: In PCR, does the DNR primer remain in the newly formed chain, or does the DNA primer get removed by DNA polymerase as the RNA primer does in regular DNA replication?\r\n&&

1648 A: Not removed. Why bother if it is DNA? Besides, the enzyme used can\'t remove primer. If the primer is DNA, you can\'t tell the primer from the rest of the molecule anyway. \r\n',NULL,1879,2,1),('0000-00-00&&

1649 Q: We\'ve discussed using centrifugation to separate DNA and using gel electrophoresis w/o SDS to separate PCR products. Does this mean that all the separation techniques we learned for proteins apply to nucleic acids (- can we also separate DNA molecules w/ gel filtration and SDS PAGE)?\r\n&&

1649 A: Most of the separation procedures work on more than one type of macromolecule. However there are variations depending on what you want to separate. For example, you don\'t use SDS PAGE for nucleic acids -- you don\'t need the SDS.\r\n',NULL,1880,2,1),('0000-00-00&&

1650 Q: 6-2 Isn\'t it still possible that the DNA is double stranded but the strands are not perfect complements? I thought you said with \r\nhybridization there can be some difference and the parts that line up with H bonds will be strong enough to hold the two strands together. Is there too much difference here?\r\n&&

1650 A: We are talking about a natural molecule, not a hybrid made in a lab. There are viruses with all 4 types of DNA or RNA, but as far as I know, nothing natural is improperly paired. (When you replicate it you have to make an almost perfect copy.)\r\n',NULL,1881,2,1),('0000-00-00&&

1651 Q: 6-4 What makes the hybridization perfect under a high T?\r\n&&

1651 A: High T breaks H bonds. If there aren\'t enough of them, the 2 strands separate. A perfect hybrid has more than an imperfect one.\r\n',NULL,1882,2,1),('0000-00-00&&

1652 Q: 6-15, A-4 since the new DNA made is H bonding to circular DNA won\'t this make the new DNA circular?\r\n&&

1652 A: Not unless you have an enzyme to join the end to the beginning of the new strand.\r\n',NULL,1883,2,1),('0000-00-00&&

1653 Q: Does the DNA circle have to be cut in order for\r\nreplication?\r\n&&

1653 A: No.',NULL,1884,2,1),('0000-00-00&&

1654 Q: C- how do you know for sure none of them are the same length? Isn\'t it possible that a promoter starts all the way at the end so the \r\nwhole strand is replicated?\r\n&&

1654    A: There are no promoters here. This is DNA replication. You mean primer, I think. If you do PCR the whole point is to selectively replicate a shorter piece.\r\n',NULL,1885,2,1),('0000-00-00&&

1655    Q: 6-16  A to totally break down DNA to its nucleotides don\'t you also need to break the H bonds between base pairs?\r\n&&

1655    A: Any condition that is harsh enough to break covalent bonds will break H bonds. There will be no H bonds left.\r\n',NULL,1886,2,1),('0000-00-00&&

1656    Q: My question is, considering that we have three strands of three different lengths, full length (lets say L), approximately half length (L/2), and some much shorter length from the Okazaki fragments lets say for simplicity (L/4). also I am aware that the mass difference difference between the N-14 and N-15 is an extra neutron, so N-14+ 1 neutron, not double.\r\n\r\nNow I\'ll get closer to my question. Density is Mass/ Volume. I would think any density comparison should look at both the Mass and Volume between the respective entities. I do not know precisely what volume formula is best to use for the DNA denatured strands or fragments, but I\'m just guessing and will use a cylinder, Volume= (Pi) x r^2 x Length. In that case could there be three lines?  If not, I am having trouble reconciling the different lengths. I figure, that there would be 1 lowest line for the short strand which has the highest density (N-14 + 1n)/ ((L/4) x Pi x r^2)= Density.  Then, another, middle line for the 1/2 Length strand where Density= (N-14 + 1n)/ ((L/2)Pi r^2). Finally one for the last strand, the full length strand, where Density= (N-14/ ( (L) x Pi x R^2). This strand would be the highest.\r\n&&

1656    A: All the DNA is collapsed here, so the length of chains doesn\'t matter. \r\nAlso, usually the DNA is broken down to shorter lengths first, before these experiments are done. (Not always deliberately, but long DNA gets very tangled and easily broken.) So it\'s only the density of the isotopes used that matters.',NULL,1887,2,1),('0000-00-00&&

1657    Q: Why don\'t we need pyrophosphatase in PCR?\r\n&&

1657    A: The concentrations of substrate are so high that the reaction is driven to the right.\r\n',NULL,1888,2,1),('0000-00-00&&

1658    Q: In reaction 6 of the glycolytic pathway, where do the two Hs come from that reduce NAD? One from the phosphate and one from G3P?\r\n&&

1658    A: It is not the H\'s that reduce NAD, it is the electrons. The H+ ions can come and go, released to the aqueous environment or picked up from them.\r\nThe glyceraldehyde-3 phosphate dehydrogenase reaction is quite complex, and involves a transient covalent binding between the substrate and the enzyme. There is a transfer of the aldehyde H to the NAD+. However, this level of mechanism is more appropriate to a biochemistry course. You should focus on the provenance and destination of the electrons, which are important for the strategy the cell uses for getting energy out of chemical reactions.\r\n',NULL,1889,2,1),('0000-00-00&&

1659    Q: With regard to translation, where and when does the loading reaction occur? Does it occur close to the mRNA? is it occurring as the ribosome is translating the mRNA or is it prior to this?\r\n&&

1659    A: Loading occurs first. We think of it as occuring independently in time and space. \r\n',NULL,1890,3,1),('0000-00-00&&

1660   Q: With regard to translation, where and when does the loading reaction occur? Does it occur close to the mRNA? is it occurring as the ribosome is translating the mRNA or is it prior to this?\r\n&&

1660   A: Loading occurs first. We think of it as occuring independently in time and space. \r\n',NULL,1891,3,1),('0000-00-00&&

1661   Q: Are the loading enzymes, activating enzymes, and AA-tRNA synthase enzymes all different enzymes w/ different functions or are they different functions of the same enzyme?\r\n&&

1661   A: Same enzymes. Just different ways of thinking about what they do.\r\n',NULL,1892,3,1),('0000-00-00&&

1662   Q: When AA~AMP if formed, does the AA bond with the AMP using its carboxyl end (like it does in AA-tRNA) or amino end?\r\n&&

1662   A: Same end -- bonds with carboxyl end.\r\n',NULL,1893,3,1),('0000-00-00&&

1663   Q: Is the repressor considered a part of the operon in general?\r\n&&

1663   A: The repressor gene and protein are required for the operon to function. \r\nThe repressor gene does not need to be located in the same section of DNA as the rest of the operon.\r\n',NULL,1894,3,1),('0000-00-00&&

1664   Q: Is it true that the only way recombination can occur between a plasmid and a chromosome is when the plasmid integrates itself into the chromosome and subsequently breaks off from the chromosome again but this time with a bit of the chromosome? Or can recombination occur \r\nthe way it does with fragments, i.e. 2 cuts on the plasmid and 2 cuts on the chromosome and swap?\r\n&&

1664   A: Recombination can occur either way.',NULL,1895,3,1),('0000-00-00&&

1665   Q: Can we say that a recombinant made from 2 ds linear DNA\'s always only requires ONE cut and rejoin event or is it just in the example you demonstrated with only two segments?\r\n&&

1665   A: I meant that you can do a successful recombination between two linear DNAs with one cut and rejoin. Each recombinant molecule is functional. However, with a circle and a linear DNA, one cut and rejoin leads to generation of a nonfunctional chromosome. (The circle is cut open.) You are absolutely correct that it can take more than one cut and rejoin to generate certain new combinations.\r\n',NULL,1896,3,1),('0000-00-00&&

1666   Q: Is the recombination/ integration that occurs when the F-factor gets integrated into the chromosome and forms the Hfr cell the same as the recombination on handout 16A bottom with letters d, b, a? I\'m wondering b/c it seems that there is exchange of equivalent genetic \r\nmaterial in the latter, whereas in the former, new genes are added to the chromosome?\r\n&&

1666   A: You have only one crossover event (cut and rejoin the two DNAs once) for integrating the F factor. It takes two cut and rejoin events to replace a section of the chromosome with a section from the fragment.\r\n',NULL,1897,3,1),('0000-00-00&&

1667    Q: With regard to making a probe: B/c a cDNA probe is complementary to the mRNA, will it hybridize to the sense strand, whereas b/c the \r\noligopeptide probe is made using the expected codons of the AA [and thus contains the sense strand sequence], will it hybridize to the template strand?\r\n&&

1667    A: If you make ss cDNA or a probe to match the code, the two probes will hybridize to different strands. However, you can make ds cDNA which hybridizes to both strands, or an oligo for either strand. When you are looking for a sequence, it is usually ds but denatured, so it doesn\'t matter which strand you will hybridize to.\r\n',NULL,1898,3,1),('0000-00-00&&

1668    Q: I am reviewing conjugation and have come across an issue I can\'t seem to figure out.  When an F+ cell donates a copy of it\'s plasmid to an F- cell, does the copy go through the pilus in a circular form or a linear form?\r\nDoes the copy link back up into a circle some how?  Or is the copy linear in the recipient cell?\r\n&&

1668    A: Linear. I have been avoiding the details on purpose. The DNA then recircularizes in the recipient.',NULL,1899,3,1),('0000-00-00&&

1669    Q: Can you please check the answer for problem #1 in the recitation problems #8 for part C. The answer states that tRNA with met is in the A site of the ribosome unit. I thought that the initiator tRNA with met only fits in the P site. And then a peptide is formed between met and the ser that is in the A site. Is the answer written in the website correct

1669    A: There are two different tRNA\'s for met. Here you are dealing with the regular one, because you are adding met to a growing chain, not using it and the special initiator tRNA to start.\r\n',NULL,1900,3,1),('0000-00-00&&

1670    Q: For problem 12R-5, why is it that the plasmid can only integrate at certain points in the DNA (in between A&B or E&F, but not elsewhere)? Is it true that cut+ rejoin/ crossing over can only occur at specific parts/ sequences cut+ of the DNA?\r\n&&

1670    A: Normally homology between the two sequences is required to get crossing over. (If you take advanced courses you will find out about the exceptions, but we are ignoring them.)\r\n',NULL,1901,3,1),('0000-00-00&&

1671    Q: A lot of the problems in problem set 11 deal with polypeptides. Is this generalization true: 1 polypeptide= 1 gene?\r\n&&

1671    A: In one definition of gene (= cistron), yes. To be exact, 1 polypeptide per cistron (if we are talking about protein coding genes). There are also cistrons or genes coding for tRNA, rRNA etc.\r\n\r\nGiven the existence of introns, alternative splicing etc., it is hard to know exactly how to define gene. That\'s why people use different terms such as cistron, locus, etc.\r\n',NULL,1902,3,1),('0000-00-00&&

1672    Q: I think I understand from the notes and problems that all organisms basically, including ones normally called \"haploid\", have several copies of genes coding for a given tRNA. Does the term \"diploid\" or \"partially diploid\" only refer to organisms with duplicate copies of genes that code for protiens? Also, for the term \"diploid\" to apply, do the duplicate genes have to lie on a seperate chromosome?\r\n&&

1672    A: Diploid means two complete sets of the genome, as if you have two complete encyclopedias. Multiple copies of any particular gene, such as the gene for a particular tRNA or protein, means that each genome contains multiple copies of that particular gene.  Sometimes all the copies are on the same chromosome, sometimes not. It\'s as if there are multiple articles on that one subject in each set of encyclopedias.\r\n',NULL,1903,3,1),(###-09-21&&

1673    Q: In the lecture notes, you mention what the Haworth projections tell us (the relative positions of -OH and -H). What do the straight-chain projections tell us

1673    A: The same thing. The straight chain figures we have been looking at are called Fischer projections. By convention vertical bonds are placed so that they go away from the viewer, back behind the plane of the sheet of paper or screen. For a hexose such as glucose this means they are constantly curling further and further away if you would start the the top (C1) and follow the vertical bonds always going away from you. If one adopts this rule for the vertical lines, then because the 4 bonds of carbon are tetrahedral in directions, the side constituents, held by horizontal bonds, will always be coming out toward the viewer relative to their central carbon. With these severe constraints, the hydroxyls and hydrogens will take unique positions that are characteristic of the stereoisomer being shown (e.g., D-glucose, L-glucose, mannose  galactose. ) \r\n\r\n',NULL,1904,1,1),(###-09-21&&

1674    Q: Besides, below or above, can we know if an -OH group or -H group is axial or equatorial from a Haworth projection

1674    A: Figuring this out without some reference point is not easy without the aid of a real 3-dimensional model set (where it would be easily evident). But if you know just one such reference point, such as that the C4 in galactose is axial and up, the rest should follow, since the direction of the axial bond alternates between up and down as you go around the ring.  ',NULL,1905,1,1),(###-09-27&&

1675    Q: I understand why the choice is gel electrophoresis without SDS but why can it not also be fingerprinting. In a different question, the logic you gave in the answer key was \"separation based on charge, therefore different amino acids, and therefore different fingerprints.\" Why is this reasoning not applied for the answer in this question

1675    A: Your goal here is to \"separate the two types of chains,\" presumably to then characterize them further. You want to purify them, to have one in one tube and the other in another tube. Fingerprinting is an analytical method that destroys the chains by cutting them up at trypsin-sensitive linkages. Fingerprinting is not a purification or \"separation\" method. \r\n',NULL,1907,1,1),(###-09-28&&

1676    Q: Can two strands from two separate polypeptides in one protein form a beta-pleated sheet

1676    A: There are instances of such a combination of secondary and quaternary \r\nstructure. \r\n\r\n\r\nSee: http://nar.oxfordjournals.org/content/26/5/1337/F2.medium.gif \r\nfor a drawing from the crystal structure of the\r\nbacteriophage MS2 coat protein dimer. \r\n',NULL,1908,1,1),(###-09-30&&

1677    Q: If a protein with disulfide bonds  is treated with SDS but no mercaptoethanol, can it be considered to be undenatured

1677    A: No. Breaking the disulfides often has NO effect on the tertiary or quaternary structure of proteins, which is mostly determined by the collaborative effect of a great number of weak bonds. Disrupting these weak bonds with a denaturing agent will destroy the native structure even if the disulfide bonds remain intact. However, the polypeptide chain(s) cannot adopt a truly random coil state until the disulfides are also reduced. A single disulfide bond would be sufficient to spoil a random coil state (unless the cysteines involved were very close to each other, e.g., adjacent.)  In cases where one or more  disulfides are indeed necessary to keep the protein in a native state, their reduction would still leave substantial secondary and tertiary stucture remaining. The number of disulfides necessary for such protein cannot be predicted.\r\n',NULL,1909,1,1),(###-09-30&&

1678    Q: How would the presence or absence of disulfide bonds in a protein affect its frictional coefficient

1678    A: The random coil state (completely linear, i.e., no disulfides) with no secondary or tertiary structure would have the most susceptibility to friction, as the composite positions in space of the atoms in these molecules would present the largest effective diameter. Any bonds that compromised this randomness (e.g., disulfides), or being in only a partially denatured state (e.g., low urea), would result in a lower frictional coefficient compared to the random coil.\r\n',NULL,1910,1,1),(###-09-30&&

1679    Q: Do denaturing reagents like SDS and urea break any covalent bonds

1679    A: No.  Denaturing agent break only the weak bonds.',NULL,1911,1,1),(###-10-04&&

1680    Q: I have some questions from pset #3.  For problem 3-5B, IF the question did say that there were equal amount of [E] for both enzymes, should we be able to predict which enzyme has a larger turnover number, even if we don\'t know exactly how much enzyme is being used to determine the Vmax?.\r\n&&

1680    A: If you know that equal moles of each enzyme were used you could conclude that the enzyme that exhibited the higher Vmax had the higher turnover number, just based on the simple relationship of k3 = Vmax/Eo.  To compare the two enzyme you could calculate the ratio of th ek3\'s; equal Eo\'s would cancel out, no matter what their actual numerical values.',NULL,1912,1,1),(###-10-04&&

1681    Q: For #3-9, the solution says enzymes lower the activation energy in both directions and so they affect the forward and backward rates equally. I don\'t really understand this because I thought in every reaction a certain direction (whether forward or backward) is favored more than the other...\r\n&&

1681    A: Yes ,a certain direction is favored but that direction depends only on the starting and ending states of the reaction (i.e., the reactants and products) and that difference is independent of the route by which reactants are turned into products. Any such route (e.g., with or without a catalyst, or via the formation of intermediate different\r\nmolecules) will be associated with different activation energies. In summary, activation energy is not related to the (eventual) directionality of the reaction; directionality is determined just by the nature of the reactants and the products, period.\r\n',NULL,1913,1,1),(###-10-14&&

1682    Q: I am trying to look at the equation $C6H12O6 + 6O2 \rightarrow 6CO2 + 6H2O$ .I know that the 6 C\'s in CO2 come from the 6 C\'s in glucose. What I\'m having trouble with is why there are only 6 H2O as opposed to 12. Because 12 NADH2 (actually 10 NADH2 and 2 FADH2) are used in the following way:

NADH2 + 1/2 O2 --> H2O . So if you use 12 NADH2, that calls for 6 O2 which we see in the overall equation, but then wouldn\'t that produce 12 H20? \r\n&&

1682   A: You are right to be disturbed. The reaction below represents the NET amounts of reactants and products, and the stoichiometry works out OK. \r\nBut an equation that is more revealing of some of the intermediate steps would be:\r\nC6H12O6 + 6O2  + 6 H2O  --> 6CO2 + 12H2O .\r\nThat is, there are several reactions in glycolysis + the Krebs cycle in which water is consumed or produced. The consumers outscore the producers to give a net consumption of 6 H2Os. After the production of 12 H2Os in respiration, we are left with only 6 net H2Os.\r\n',NULL,1914,2,1),(###-10-18&&

1683   Q: For  Prob. 4-13E I thought ATP at 0.001 mol would limit you to 0.001 moles of ATP produced. Why isn\'t this so

1683   A: You can make more than 0.01 mol of ATP because you have plenty of ADP that can be turned into ATP via the glycolytic pathway to pyruvate. However, there will be a limit on the running of that pathway as you cannot regenerate the NAD that you are using up to run the 2X glyceraldehyde-3-P oxidations. So when you run out of NAD, the path will stop. You have 0.01 mol of  NAD, so that you can run the glycolytic path to generate 0.01 mol of pyruvate and that running will result in 0.02 net moles of ATP from ADP per NAD or 0.01 moles per mole of glucose (since you need 2 NADs for every glucose utilized). As for the ATP requirement, you just need a little to get a few molecules of glucoes phosphorylated; once you start you generate plenty of ATP for further starts because you are making 2 net ATP molecules from ADP for each glucose molecule. So you could even start with 0.0001 mol of ATP.',NULL,1915,2,1),(###-10-26&&

1684   Q: I have a question about where net 38 ATP comes from. \r\n\r\n\r\nAccording to the class handouts, starting with 1 glucose molecule -- in glycolysis we net 2ATP and 2NADH. At the end of Kreb\'s cycle, we have 10NADH, 2 FADH and still 2ATP. After the ETC & oxidative phosphorylation, the 10 NADH yields 30 ATP, 2 FADH yield 4, and then in total we have 36 ATP.\r\n\r\n\r\nAccording to a chart I found online, 2 ATP (per glucose) is also produced in the Kreb\'s cycle. Which would result in the total of 38.\r\n\r\n\r\nIs this how I should think about it? I know that eukaryotes use 2 ATP in transporting pyruvate to the mitochondria, but I am just having trouble coming up with 38 in the first place.\r\n&&

1684   A: See the GTP from the Krebs cycle on the handout. It\'s equivalent to ATP.',NULL,1916,2,1),(###-10-26&&

1685   Q: During PCR, why is it that for additional cycles after the 3rd cycle the strand doesn\'t get even shorter such as after the 1st and 2nd cycle?\r\n\r\nHow does it know to copy just the target sequence at this point, and why not always copy just the target sequence? \r\n&&

1685   A: Because the primer fits right at the end of the target sequence, which is the end of the strand (after the 3rd cycle).',NULL,1917,2,1),(###-10-26&&

1686   Q: In question 7-21, the mRNA strand that is synthesized from the appropriate template is shown below (at least by my calculations)\r\n\r\n5\'CGAUGAGCGAGCGAGCGAUGUAGGG3\'\r\n\r\nThen the question asks, what is the amino acid sequence that you would get using this mRNA, and the answer given is (NH2) met-ser-glu-arg-ala-met (COOH).\r\n \r\nThis answer makes sense to me if you knew to start at the third reading frame...but how did you know this? My understanding is that \r\nyou know its this frame because the question states that the mRNA does in fact result in a functioning protein. What if there

had been another AUG that was not in the same frame? In this case, would you have to be told in the question which AUG to use?\r\n\r\nI guess what I\'m asking is, how do we know which frame to read it in? Can you assume that the first AUG that you see determines the frame? \r\n\r\nFinally, are there overlaps?  That is, can you have a single mRNA be read in two (or\r\n3) different ways, or is that particular mRNA only going to have one particular frame? \r\n&&

1686    A: I think I rigged it so there are no more AUG\'s in this message, so as to avoid the issues you raise. In bacteria, which AUG you use is dependent on other factors we have not discussed, so you would have to be told which one to use (or have more info). In eukaryotes, it\'s generally the first one.',NULL,1918,2,1),(###-10-26&&

1687    Q: In problem 7-12 D, the mRNA that is being used is 5\'GUAGCCUACCCAUAGG3\' The answer states that if you start with the 2nd base you can\'t translate the 1st codon (because it\'s a stop) but you can translate the subsequent ones. I was under the impression that you need an AUG to code for methionine to get the translation started. In other words, if there were a stop in the middle of the RNA, I thought this would stop translation from proceeding, and that you would need an AUG to get started again further. If you were reading the first reading frame or the third, I am under the impression that I should assume that there was an AUG \"earlier on.\" I put the words earlier on because I wanted to use the words upstream or downstream but I am confused about this terminology. In the above mRNA, if something were downstream of this mRNA, would it be to the right? or to the left? Is upstream\"ness\" a term that orients you relative to the 5 prime to 3 prime orientation of the thing that you are referring to? \r\n&&

1687    A: Note that in B it says no start codon is required. This applies to D also. Otherwise, it\'s a more complex question.  Upstream means read first. Toward the 5\' end of the sense strand or mRNA.\r\n',NULL,1919,2,1),(###-10-27&&

1688    Q: I understand the ratio of NAD-glucose-ATP (2 to 1 to 2); however, could you please explain why NAD is the limiting factor and not ATP?  I am confused because there is only 0.001 moles of ATP which would only produce 0.002 moles of final ATP, and then would run out.  Is ATP not the limiting factor because of the excess amount of ADP and we are to come to the conclusion that this ADP will be able to create more ATP

1688    A: You were OK with 0.001 moles of ATP being produced starting with only 0.001 moles of ATP. So where did the additional 0.001 moles of ATP come from? Well, from the gobs of ADP that are there, just waiting for some reaction that could turn them into ATP by transferring a Pi group. Such reactions are the heart of glycolysis (rxns 6 and 9). Glycolysis makes 2 NET ATP, starting from...ADP. The ATP moles are not limiting; you just need a smidgen to \"prime the pump\". In theory, you would need only 2 molecules of ATP to get one molecule of glucose running. After that you\'re making net ATP from the gobs of ADP (and free Pi, in reaction 6) so always have more than enough to start additional molecules of glucose down the glycolytic path.',NULL,1920,2,1),(###-10-27&&

1689    Q: In reviewing question 5.6 A, I am afraid that I need some clarification. Why is it that either ethanol/CO2 or lactate are possible products of bacteria on glucose in anaerobic conditions? Researching the question further, I see that this could either have to do with the enzymes that the specific bacteria contain OR could it have to do with the classification of yeast possibly being considered a bacteria (even thought I though that yeast was eukaryotic), or maybe something else altogether? \r\n&&

1689    A: Yeast is not a bacterium. It is a unicellular eukaryote. Different bacteria have different enzymes, so some make lactic acid and some make ethanol and CO2 from fermentation.\r\nYou are right, it is the enzymes that the specific bacteria contain that make the difference.\r\n',NULL,1921,2,1),('0000-00-00&&

1690    Q: I thought there were no U bases on Okazaki fragments because Okazaki fragments are only produced in DNA synthesis and U bases are only found in RNA?\r\n&&

1690    A: You forgot that DNA synthesis requires a primer, and the primer is usually RNA.',NULL,1922,2,1),(###-10-28&&

1691    Q: Could you please explain one problem to me?\r\n4-18 B i) how is the isocitric acid going to be made when we don\'t have oxaloacetic acid made? are we assuming that the the 0.001 moles of oxidized NAD is enough to go through one cycle? and even if i was enough, how do we start the cycle anyway without oxaloacetate? Are we assuming there is some present?\r\n&&

1691    A: It says the Krebs cycle is working, so there must be enough OAA present to run the cycle. Normally OAA is regenerated, so it isn\'t limiting. If there is a block after isocitric, then no OAA will be regenerated, and the enzymes will stop converting acetate to isocitrate when all the OAA is used up.',NULL,1923,2,1),(###-11-09&&

1692    Q: Part I: Dear Dr. Mowshowitz, The answer to problem 7-15 part C, says that one tRNA  can read AGU/C and one tRNA can read CAU/C. In both of these cases,  according to the wobble rule it would be a tRNA with Inosine \r\nin the first  position of the anticodon that could bond with these pairs of codons.\r\n\r\nPart II: If this is correct however, wouldn\'t the same tRNA code for AGU/AGC which are the same AA, as well as AGA which is a different \r\nAA. Same  scenario with the CAU/C?\r\n\r\n&&

1692    A: Part I: No, that is wrong. You want a tRNA with G in the wobble position, not I.\r\n\r\nPart II: That\'s exactly why you don\'t want I in the wobble position.',NULL,1924,3,1),(###-11-12&&

1693    Q: Part I: I\'m having trouble understanding Recombination. Do mutations in recombination occur in the same GENE but on different spots of that gene?\r\n\r\nPart II: Also, what is necessary to restore function or what impedes function?  How do we know that mutation means no function? And what is \"functioning\" (or not) when you ask the question \"if you put 2 defective copies \r\ntogether, can you ever get back function\" on your handout?\r\n\r\n&&

1693    A: Part I: Recombination can occur between mutations in the same or different genes. \r\n\r\nPart II: The assumption is that mutants don\'t function properly -- whatever the function is, they have trouble with it. They have a mutant (not normal) phenotype. \"get back function\' means the cell with two copies acts normal, not mutant.',NULL,1925,3,1),(###-11-29&&

1694    Q: Hi, I was wondering what exactly answer to problem set #12-5 means. The question asks if more or less DNA will end up in hybrid; the \r\nanswer says: less...DNA is the same. I Could you please clarify what exactly is less and if RNA is less in rich medium, why would DNA not be less but the same? Thanks.\r\n&&

1694    A: DNA in the cell is the same under all conditions. (The amount that is hybridized changes, but not the total amount per cell.) The number of different genes that are transcribed varies, depending on

the growth conditions. The more genes are transcribed, the larger the % of DNA that ends up in hybrid.',NULL,1926,3,1),(###-11-29&&

1695    Q: When an F factor integrates into the bacterial chromosome via a single cut and rejoin event, is it using restriction enzyme methods?\r\n\r\nIs the single cut and rejoin event facilitated by the presence of a restriction enzyme/ligase and if not, what enzyme is it?\r\n&&

1695    A: NO. The enzymes for recombination and for restriction/modification are totally different.',NULL,1927,3,1),('0000-00-00&&

1696    Q: After you make your library of chimeric plasmids and you wish to determine which of your colonies got the plasmid with the right\r\ninsert using colony hybridization, why is the use of a cDNA probe preferable to an mRNA probe? Is the cDNA probe sturdier because it\'s DNA not RNA?\r\n&&

1696    A: Yes.',NULL,1928,3,1),(###-11-30&&

1697    Q: I was reviewing material for the third exam, and I am a bit confused about  conjugation, specifically the F factor and Hfr.  Am I correct to say: When conjugation occurs between F+ and F-bacterial cells, the \r\nrecipient (F-) becomes F+ since the F+ genes are on a plasmid.  However, when a Hfr \"mates\" with F-, the recipient will most likely NOT be a donor since  replication of the chromosome starts in the middle of the integrated F. Unless the cell is given time to completely copy the chromosome, the F gene is \"broken.\"\r\n\r\nI suppose my question can be summarized as: When F+ and Hfr conjugate with  F-, does the F- become F+?\r\n\r\n\r\n&&

1697    A: Right. As you say, if donor is F+, recipient usually is too. If donor is Hfr, recipient usually stays F-.\r\n',NULL,1929,3,1),(###-11-30&&

1698    Q: I am figuring out a problem concerning complementation (11-14). The question says that you infect 2 mutants at the same time and look to see if they form small or large plaques.  Then in the table it shows strands A-A producing a small size of plaques.  Shouldn\'t they not produce any places at all if the mutation is in the same place on the same gene?How do they produce any plaques?\r\n&&

1698    A: The phenotype of the mutants to begin with is making small plaques, not being unable to replicate. If they don\'t complement, you get small plaques, not no virus.',NULL,1930,3,1),(###-11-30&&

1699    Q: How can one probe hybridize to 4 different fragments?  Is the same amount of probe present in each band

1699    A: If the probe overlaps a section of each fragment, one separate molecule of probe can hybridize to each fragment. ',NULL,1931,3,1),(###-12-03&&

1700    Q: Part I: If you have a cell that doesn\'t need lactose as an energy source (lets say because its being grown on glucose), then it doesn\'t need to break down lactose right?\r\nIn other words, from your notes, the way I understand this situation is that lactose will act as an inducer and turn the lac operon \"on\" only if it is present and NEEDED.\r\n\r\nPart II: If this is true, does that mean that\r\nif lactose is present but is not needed (because there is some other energy source), lactose will somehow not bind to its lac repressor protein, and the lac operon will remain off?\r\n&&

1700    A: Part I: Correct. We didn\'t go into it, but glucose blocks induction of the operon. \r\n(See catabolite repression in a text or on line.)\r\n\r\nPart II: See above. Other regulatory proteins are involved here beyond the ones discussed in class.',NULL,1932,3,1),(###-12-02&&

1701    Q: Say a plasmid has one restriction site for a particular restriction enzyme. You insert a fragment prepared with the same restriction enzyme. Do you now say that two restriction sites have been created in the plasmid by the insertion of the fragment (since an overhanging end from both the plasmid and the fragment contribute to each site), or do you say that the plasmid still has one and the fragment also carries one?\r\n&&

1701    A: You say the recombinant plasmid has two, which is the case, exactly as you describe it. ',NULL,1933,3,1),('0000-00-00&&

1702    Q: We learned about non-coding sequences in DNA and of introns in pre-mRNA. According to a cited reference on the \'Non-Coding DNA\' article on Wikipedia: \"More than 98% of the human genome does not encode protein sequences, including most sequences within introns and most intergenic DNA.[1]\". Does the existence and presence of such no-coding sequences represent potential human evolution? Perhaps such regions that in \r\nfuture progeny may indeed code for certain genes/proteins which can differentiate a \"future human\" from a \"modern (current) human\", much the same way man has become differentiated from other primates. Ultimately, can these non-coding regions allow for higher intelligence (seemingly\r\nalien) life forms of men?\r\n&&

1702    A: No one really knows. Changes in actual coding sequences and their regulation seem the most likely future directions. (Some of these regulatory sequences may be in the \'junk.\') We didn\'t have time to discuss it, but some of our junk DNA contains pseudo-genes -- copies of genes that have acquired so many mutations that they don\'t make anything or do anything anymore. It is thought that most of these will be lost, or become even more defective, but perhaps some of these will acquire even more mutations and produce different proteins. All that we are pretty sure of is that humans will continue to evolve, but whether it will be toward greater intelligence or not is an open question.',NULL,1934,4,1),('0000-00-00&&

1703    Q: 8-8F: Isn\'t meiosis II also possible? For mitosis and meiosis I, we would be  assuming a diploid, but wouldn\'t meiosis II work for a tetraploid organism? If this is indeed the case, nowhere in the problem does it specify \r\nthat the  cell must be from a diploid (or at least that I see).\r\n\r\n8-11C: At this point in the problem, how do we know to assume that N remains  constant, as opposed to thinking that a change in the ploidy of the organism  causes a change in the number of different types of chromosomes (N) given a  constant number of chromosomes (12)?\r\n&&

1703    A: 8-8F Ans: We are assuming this is a normal organism, so the cell is diploid or haploid. \r\n\r\n8-11C Ans: N is constant for a species. It does not change. Only the ploidy changes.',NULL,1935,4,1),(###-12-21&&

1704    Q: Q: I am going over problems that I had some difficulty with I re-encountered 8-8,F which asked, given the shown karyotype, in which \r\nof the  phases could the cell have been. I was able to deduce it definitely could  have been in meiosis I or mitosis but was not 100% clear as to why it could not have been in meiosis II. The shown karyotype pictures a 2N cell however,  as in the case of plants, this could been 4N cell now undergoing meiosis II. Are we to assume that we generally will not encounter cells greater than 2N  unless otherwise specified or am I simply missing something?\r\n\r\n&&

1704    A: Answer: We are assuming this is a normal situation, no tetraploids involved. Only haploids or diploids. Stick with the normal situation unless it says otherwise or there is no other solution. ',NULL,1936,4,1),(###-12-21&&

1705    Q: I don\'t know if others possibly have this problem but if  they were, this might help them. I was having a hard time understanding/picturing meiosis, and to  a lesser extent mitosis, in that I understood what was happening at each step and I got the bigger picture of  what each process did but I could not consistently understand what genetic  info would end up in each gamete. I realize my mistake was that I thought of  each chromosome as it would appear in the karyotype and would expect the  gametes to have such chromosomes - i.e., X-shaped - but I neglected the fact  that the chromosomes of the karyotype are in metaphase thus already doubled  and entered interphase as a single chromatid.\r\n\r\n\r\n&&

1705    A: I have tried to explain enough to head off this confusion, but somehow the whole chromosomes & chromatid business is hard to get straight. We draw the X shaped chromosomes at division to emphasize that DNA replication occurs before division, not during it.',NULL,1937,4,1),(###-12-21&&

1706    Q: Hi, I have a question regarding recitation problem set #12 question 1(B). Should we assume all ppl with HC have the genotype of HC/hc? Because if the condition is HC/HC then the percentage of offspring \r\nwill be different?\r\n&&

1706    A: I assume you mean problem 10-12. The answer is yes, because the condition is rare, and people who marry into the family are almost certainly homozygous normal. So there is no way someone can get two copies of the HC allele unless two related affected people marry.',NULL,1938,4,1),(###-02-22&&

1707    Q: I\'m having trouble rationalizing why the Na/K pump could ONLY be on the basolateral side in parietal cells but not on the apical side.  I \r\nanswered that it could be either. Is it because having both pumps pumping in K from stomach would deplete that area of K so you have to spread them out a bit?\r\n\r\n&&

1707    A: The pump is always on the BL side of everything. What\'s on the apical side depends on the cell type. There are various rationales, but I think the main one is that the main supply of ions and everything else for all cells is the blood, which is on the BL side.',NULL,1939,1,2),(###-02-22&&

1708    Q: Part I: In your lecture 5 audio recording, slightly before the midway point, you say that \"glucose ALWAYS goes down its gradient, but that whether it goes in or out of a cell depends on the relative concentrations.\" I \r\nthought that glucose, when entering an epithelial cell from the lumen of the gut, is traveling up its gradient.\r\n\r\nPart II: Also, you mention liver as one of the few tissues where glucose can go in AND out. I\'m confused about this. Doesn\'t glucose go in and out of an epithelial cell?\r\n\r\n&&

1708    A: Part I: I was talking about transport with GLUTS. Not with active transport.\r\n\r\nPart II: Not in and out to and from the blood.',NULL,1940,1,2),(###-10-25&&

1709    Q: How can we follow the carbons from glucose through step 5 of glycolysis if they are moved around during isomerization

1709    A: Carbons are not moved, so glucose carbon 1 carries the phosphate in DHAP and that is not changed after isomerization to G3P. Once the DHAP is isomerized to G3P it is exactly a molecule like the G3P pictured below it. You can line them up so that the newly born G3P has its phosphate on the bottom by just sliding the structure around on the paper or screen surface. We established that carbon 1 of glucose has the phosphate and it\'s now on the bottom of the new G3P. So reading top to bottom, with phosphates on the bottoms, the origin of the carbons on the two G3Ps after step 5 is:\r\n4    3\r\n5    2\r\n6    1\r\nreferring to the original glucose molecule. ',NULL,1941,2,1),(###-10-23&&

1710    Q: Since DNA replication is semi-conservative, and I came from a double strand\r\nof DNA (fused from the DNA of each of my parents), what are the chances that\r\nnone of the two DNA stands from my parents is still in my body? What are the\r\nchances that any of the molecules from the zygote (or DNA) from which I was\r\nformed are still in my body

1710    A: You do NOT come from a ds of DNA fused from both parents. If you did, how could you get different genetic info from each of them?? You received a ds DNA from EACH of your parents.\r\n However you are correct that the old DNA strands are always passed on, so baring cell death or destruction, you still have the DNA strands you inherited from your parents. However, since many cells die during development, you probably don\'t have the parental DNA any more.',NULL,1942,2,1),(###-10-09&&

1711    Q: How can a \"high energy\" phosphate bond be generated in reaction 9 of glycolysis just by rearranging atoms

1711    A: I do not know the organic chemical mechanism, but found this explanation satisfying:\r\nFrom: Biochemistry. 5th edition.,  Berg JM, Tymoczko JL, Stryer L., New York: W H Freeman; 2002.\r\n\r\n\"In the next reaction [reaction 9], an enol is formed by the dehydration of 2-phosphoglycerate. Enolase catalyzes the formation of phosphoenolpyruvate (PEP). This dehydration markedly elevates the transfer potential of the phosphoryl group. An enol phosphate has a high phosphoryl-transfer potential, whereas the phosphate ester, such as 2-phosphoglycerate, of an ordinary alcohol has a low one. The Î”GÂ°Â´ of the hydrolysis of a phosphate ester of an ordinary alcohol is -3 kcal mol-1 (- 13 kJ mol-1), whereas that of phosphoenolpyruvate is -14.8 kcal mol-1 (- 62 kJ mol-1). Why does phosphoenolpyruvate have such a high phosphoryl-transfer potential? The phosphoryl group traps the molecule in its unstable enol form. When the phosphoryl group has been donated to ATP the enol undergoes a conversion into the more stable ketoneâ€"namely, pyruvate.\"\r\n\r\nThus, the high phosphoryl-transfer potential of phosphoenolpyruvate arises primarily from the large driving force of the subsequent enol-ketone conversion. Hence, pyruvate is formed, and ATP is generated concomitantly. The virtually irreversible transfer of a phosphoryl group from phosphoenolpyruvate to ADP is catalyzed by pyruvate kinase. Because the molecules of ATP used in forming fructose 1,6-bisphosphate have already been regenerated, the two molecules of ATP generated from phosphoenolpyruvate are â€œprofit.â€\"',NULL,1943,2,1),(###-11-28&&

1712    Q: I understand why the mutation has to be in the same gene.\r\nSpecifically, mixed infection with mutants 1 & 10 led to no progeny\r\nphage - suggesting that no complementation took place. Therefore the\r\nmutations should be in the same complementation group (same gene). But\r\nhow does the fact that all progeny are mutant indicate that there is\r\nno recombination occurring? Could (in this case) recombination lead to\r\na wild-type phage? And since this doesn\'t happen here, recombination\r\nmust not have occurred? Is this right?\r\n\r\nFurther, IF recombination did occur,

and progeny phage WERE still\r\nmutant, then we could conclude that the mutations are in the same base\r\npair. Is line of reasoning correct

1712　A: If recombination could occur, some of the mutant pairs would have mutations in different base pairs, and would give wt recombinant progeny. Any one pair of mutants could fail to give wt progeny if recombination were allowed, but at least some pairs should give wt progeny. You don\'t expect all 82 different mutants to have mutations in the same base pair, so some combos should give normal progeny (if recombination were allowed). So you can rule out recombination.',NULL,1944,3,1),(###-11-16&&

1713　Q: When a a plasmid crosses over to join a host cell\'s chromosome to become an Hfr, does it \"cross over\" in the sense that it recombines (replaces a homologous section in the chromosome) or does it \"extend the loop\" of the chromosome by not replacing an existing section but by simply adding onto it?\r\n\r\nI\'m just confused by the simultaneous use of the tubing analogy and the word \"cross over\"&&

1713　A: Crossing over refers to the process of cutting and rejoining DNAs. Depending on the topology and the number of cross over events, you can combine circles or substitute one piece for another, etc. Any new combination is considered recombinant, whether you join or replace.',NULL,1945,3,1),(###-11-30&&

1714　Q: When 2 bacterial cells are from the same strain,\r\nthey cannot have bacterial sex... Why is that? Does \"strain\" mean the same\r\nthing as \"species\"? \r\nI was thinking that if this is the case, then the 2 bacterial genomes would\r\nhave different alleles for different genes, and thus they could exchange\r\nDNA.&&

1714　A: Strain means both F+, or both F- in addition to being the same species -- same genes in same order, etc.',NULL,1946,3,1),(###-11-30&&

1715　Q: When viruses assemble, do they enclose just 1 fragment of DNA into their\r\nprotein coat? I know they usually take in their complete set of viral\r\ngenetic information, but can they also take in the complete bacterial DNA\r\n(even though I assume bacterial DNA is much longer than viral RNA/DNA)

1715　A: Usually the bact. DNA substitutes for a viral piece of the same size.',NULL,1947,3,1),(###-11-30&&

1716　Q: Do we usually get plasmids\r\nintegrated into the chromosome? And what exactly happens? Do only the\r\nhomologous regions of the DNA get exchanged/replaced with one another? Or\r\ndoes the entire plasmid integrate (no segment is exchanged for another)? And\r\nwhat about the \"extra\" DNA on the plasmids (that aren\'t in the chromosomal\r\nDNA)...will they also come into the chromosome or will they get cut out as\r\nfragments and then eventually get degraded

1716　A: There is one crossover event between the plasmid and the chromosome. When this happens between 2 circles, you get one big circle, not exchanges.',NULL,1948,3,1),(###-01-22&&

1717　Q: If we are looking at animal cells through a microscope - any type of microscope - does this mean the cells are dead?  (If so, how do we look at living cells?)&&

1717　A: You can see living or dead cells in the light microscope, but only dead cells in the EM. Irradiation with light doesn\'t kill cells. However the light microscope is often used to look at preparations of cells that are dead and stained. See the appendix to Becker for details of all kinds of microscopy.',NULL,1949,1,2),(###-01-22&&

1718　Q: I have a question about immunofluorescene and the big picture. So if you are trying to find and tag the protein of a microtubule in a sample, would you inject the antigen (which is a the primary protein of the MT) into an animal, take the antibodies they produce and put it into the sample and then tag those Ab\'s with a secondary with the tag? Also is \r\nthis considered an in situ assay

1718　A: That\'s one way to do it. It\'s usually called indirect immunofluorescence, but it is identifying the protein of interest \'in situ.\' The term \'in situ assays\' is usually reserved for enzyme assays.',NULL,1950,1,2),(###-12-19&&

1719　Q: I was under the impression that \"parental\" always referred to phenotype. Can it be genotype, or must it be both geno and pheno.\r\n\r\nThe question that raises this problem for me is: Problem book question 10-18 E-1 1)\r\n\r\nI believed type 1 to be both recombinant and (since it has a matching parental phenotype) parental.&&

1719　A: You really care about genotype, and the trick is to set it up so you can deduce genotype from phenotype. It is impossible for something to be both recombinant and parental at once.',NULL,1951,4,1),(###-12-20&&

1720　Q: For problem set 10, question 8, I was having a tough time with understanding\r\n the situation. If the genes are linked, do you mean that they are\r\n completely linked, or that they can still cross over and form many different\r\n gametes? I put 3 as my initial answer for the genotypes, and I can\r\n understand why the answer is 8 since there\'s three traits and two alleles\r\n for each.&&

1720　A: \'Linked\' does not mean completely linked. It means only more parentals than recombinants. If there is complete linkage (no crossing over) it will say so. Even \'closely linked\' is not the same as \'completely linked.\'",NULL,1952,4,1),(###-12-20&&

1721　Q: For problem set 10, question 13, I assumed that we did not know the parent\r\n genotype and thus their gametes. Therefore, I answered that for A, I could\r\n not tell. However, I checked the answer key and that assumption is wrong.\r\n Should I have thought we know the parent genotypes for this question

1721　A: You can deduce the heterozygous parent\'s geno (AB/ab or Ab/aB) from the offspring if the genes are on the same chromosome.  If the genes are linked (close together) you\'ll get more of the parental combo than the recombinant combo for both crosses. If the genes are on the same chromosome, but aren\'t linked, you\'ll get equal numbers of parental and recombinants from (2), but all parentals from cross #1. If the genes are on separate chromosomes, you\'ll get equal numbers of parental and recombinant combos (independent assortment) from both crosses.',NULL,1953,4,1),(###-12-18&&

1722　Q: Something in unsettling about our discussion of why the recombination\r\n frequency is limited to 50 percent as the distance between gene loci\r\n increases.  I thought the argument was that many cross-over events happen at\r\n large distances and the net result of these multiple cross-overs is as\r\n likely to be a shuffling resulting in a new combination of alleles as a\r\n RESHUFFLING

BACK to the original combination of allele.\r\n\r\nWhile this makes\r\n sense, this argument would seem to only apply to the chromatids involved in\r\n recombination (i.e., the chromatids that line up), which is just 1 chromatid\r\n from each homolog.  (I have starred these lined-up chomatids from 2 homologs\r\n in the diagram below.)\r\n\r\n -----A----B-----\r\n -----A----B----- *\r\n\r\n ------a----b----- *\r\n ------a----b------\r\n\r\n This is what is unsettling: we seem to be ignoring the fact that there are 2\r\n chromatids that will ostensibly always result in PARENTAL haploid meiotic\r\n products since they are never involved in x-over events (i.e., the unstarred\r\n chromatids). So, the only way to get a RF 50% is for the lined-up chromatids\r\n (the starred ones) to ALWAYS shuffle their alleles.  But, by the argument\r\n above, they are as likely to shuffle as reshuffle the combination of\r\n alleles. \r\n\r\nSo, why isn\'t the limit RF 25%? The unstarred chromatids always generate\r\n parentals and half of the time the lined-up (starred chromatids) yield\r\n recombinant haploid products. This would suggest a RF frequency of 25% not\r\n 50%.  What is wrong with my logic

1722    A: All 4 chromatids can get in the act. Just only 2 in any one event. If you want a detailed explanation, look at the notes of 2010 or a textbook.',NULL,1954,4,1),(###-12-17&&

1723    Q: Sometimes it seems that\r\nthe term \"double crossover\" is used to describe a multiple (i.e.\r\ndouble) crossover event that puts the genes back into parental\r\ncombination (which then goes unreflected on the RF).  However, later\r\nin the lecture (on page 7 of the notes), \"double crossover\" is used to\r\ndescribe 2 separate cut and rejoin events that can be used to\r\nestablish gene order (because of the rarity of the double-crossover\r\nevent).  I gather that these two uses of \"double crossover\" do not\r\nrefer to the same thing, correct

1723    A: They both mean the same thing. Two cut and rejoin events. One is from the point of view of only A and B -- whether there was no cross over or two between them doesn\'t matter -- it comes out the same. The other case includes the state of a gene or marker in between A & B. In that case, you can tell two crossover from one.',NULL,1955,4,1),(###-12-16&&

1724    Q: I am having difficulty with problem set 10-8-B.  Can you explain why there are 8 different genotypes and phenotypes among F1 progeny?\r\n\r\nAren\'t the parents AABBCC x aabbcc.  So wouldn\'t there be only one different F1 genotype, AaBbCc

1724    A: The parents are as you indicated, but this is a haploid organism. The zygote goes through meiosis to give spores, and it\'s those haploid spores you are looking at.  The \'F1\" is haploid, not diploid.',NULL,1956,4,1),(###-02-08&&

1725    Q: I was wondering if an exchanger (more specifically band 3) used energy, was\r\nan example of active transport. Wikipedia suggests that it is a\r\n\"protein counter-transporter\" that is not a pump and does not use energy.\r\n(The information on Wiki on counter-transporter proteins is not\r\nhelpful.)  In problem 2R 5 the solution says that the exchanger maintains a\r\ngradient that is then used for secondary transport. If it does not use\r\nenergy and it sets up a gradient, what is it and how can it work

1725    A: It uses the energy of the gradients of bicarb and chloride. No other source. Some consider it facilitated diffusion and some secondary active transport where the gradient of bicarb drives chloride.',NULL,1957,1,2),(###-02-08&&

1726    Q: Im a bit confused about lipid anchored integral and transmembrane integral proteins. Aren\'t they the same? It seems that the only difference would be that the integral proteins are lipid anchored

whereas the transmembrane proteins are either anchored to the cytoskeleton or float in the lipid bilayer.. Can transmembrane proteins not be lipid anchored? And can\'t lipid anchored proteins also be single and multipass? (I\'m referring to the diagram on 3A here). Also, in the list of characteristics of transmembrane proteins in the lecture outline, I was wondering-can\'t all those apply to lipid anchored proteins as well

1726    A: The terminology was invented before lipid anchored proteins were discovered. Some people consider them integral, and some consider them a separate class. Lipid anchored proteins are not transmembrane. They are on one side or the other.',NULL,1958,1,2),(###-02-16&&

1727    Q: In the lecture 7 notes in the Nucleolus section there is subsection\r\n\"b. Synthesis of rRNA & protein.\" I am confused, so can you please\r\nprovide some clarity?\r\n\r\nrRNA for cytoplasmic ribosomes (meaning just not mitochondrial?) is\r\nmade in the nucleolus. The rRNA must be then exported out of the\r\nnnucleus, since ribosomal proteins are made in the cytoplasm. Then the\r\nribosomal proteins are imported back into the nucleus and assembled\r\ninto subunits there, (presumably because the enzymes for subunit\r\nassemble are only in the nucleus, or nucleolus?)\r\n\r\nHow does mRNA fit into this scheme?  What exactly are ribosomal\r\nproteins and which come from rRNA and which come from mRNA...?\r\n&&

1727    A: Ribosomal proteins are made using mRNA in the usual way. rRNA is not mRNA. You have mixed them up. The rRNA is not exported from the nucleus until it is combined with the proteins into ribosomal subunits.\r\n\r\nThe mRNA for ribosomal proteins is exported. The rRNA stays in the nucleus.\'\r\n\r\nThe proteins assemble in the nucleolus with the rRNA, not the mRNA.',NULL,1959,2,2),(###-02-28&&

1728    Q: Here is question 4-1C:\r\n\r\nYou can digest the Sv40 mini-chromoesone briefly with DNase (microccal nuclease), isolate the DNA, and run the digested DNA on a gel. Alternatively, you can isolate the DNA first, treat it with DNase, and run the digest on a gel. Will the two gels look the same? Draw the expected results.\r\n\r\nWhenever you \'isolate DNA\', does that mean purify it? Basically take it from DNA + histone stage to just DNA? Is isolated DNA the equivalent of purified/naked DNA?\r\n\r\nSo in the first part of the question, the chromatin was essentially split into 200 bp parts and THEN stripped of all histones and related proteins. In the second part, it was FIRST stripped of all histones and related proteins and THEN cut (and therefore random pattern). Is this correct

1728    A: Yes to all.',NULL,1960,2,2),(###-03-08&&

1729    Q: Most proteins in the nucleus and peroxisomes are made on\r\nfree ribosomes; however, some membrane proteins of the nucleus and\r\nperoxisomes are made on the ER, then shipped over or slide over to the\r\nnucleus. Does that mean that they require a Signal Peptide to take them to\r\nthe ER first, making them exceptions (proteins made on attached ribosomes\r\nfor the nucleus and peroxisomes)

1729    A: Membrane proteins of nucleus are probably made on ER and slide over. Membranes of the peroxisome are probably made on free ribosomes and inserted into ER or perox. membrane after translation. They need a localization signal, but not a SP.',NULL,1961,2,2),(###-04-30&&

1730    Q: I have written down that heart cells are bi-nucleate. But in the chart I have comparing skeletal muscle to cardiac to smooth muscle, I have written that cardiac muscle is uni-nucleate and that

skeletal muscle cells are the only ones as multi-nucleate. Are these 2 different types of heart cells? If not, could you please clarify

1730    A: It is not clear about human cells, although mice heart cells are binucleate. Heart muscle cells are unusually large, and sometimes replicate their nuclei or DNA without further steps. The point is that the heart cells are NOT formed by fusion of multiple cells, unlike the case for skeletal muscle.',NULL,1962,4,2),(###-05-09&&

1731    Q: I\'m confused because\r\nwe\'re told that in the case of smooth muscle, most of the Calcium ions come\r\nfrom outside the cell.  But we\'re also told that smooth muscle goes through\r\nthe IP3 pathway, and that IP3 (I think) binds to receptors on the ER that\r\nopen channels (or maybe are channels themselves) and let Calcium flow down\r\nits gradient into the cytosol.  So in smooth muscle, how does most of the\r\ncalcium come from outside the cell while still using the IP3 pathway

1731    A: In some smooth muscle the Ca comes from the outside, and in some in comes from the ER, and in some it comes from both.\r\n Also smooth muscle  doesn\'t always use IP3 to release the Ca.',NULL,1963,4,2),(###-05-09&&

1732    Q: Can you clarify the correct usage of antibody and immunoglobulin.  Does Ig refer to the antibodies both secreted by the B cell as well as those expressed on the surface?  Is there a distinction between the two terms

1732    A: Membrane bound immunoglobulin is usually distinguished from humoral when it matters. When it doesn\'t, the terms antibody and immunoglobulin are often used interchangeably. The immunoglobulin on the membrane is often called a BCR instead of antibody (or membrane bound antibody) to avoid this confusion.',NULL,1964,4,2),(###-05-03&&

1733    Q: Is it an action potential that sweeps into t tubule or a graded potential?  Doesn\'t the action potential end at the presynaptic terminal

1733    A: An AP is generated in the muscle by NT from the presynaptic cell. It\'s the muscle AP, not the neuron AP, that enters the T tubule.',NULL,1965,4,2),(###-05-03&&

1734    Q: Are the Invaginations in the membrane the t tubules themselves or are the t tubules something separate from the invagination?  The diagram attached seems to show that they are different.&&

1734    A: The T tubules = the invaginations. ',NULL,1966,4,2),(###-04-25&&

1735    Q: In the explanation for problem 6-11, it indicates a single hormone can have more than one receptor and each may be associated with a different type of G-protein. Furthermore it states one receptor/G-protein combo could increase the level of cAMP and the one could decrease it.&&

1735    A: From the basic mechanism of G-coupled protein receptor pathway: I thought when the ligand binds the GPCR (the receptor), -> G-protein activated -> adenyl cyclase activated -> cAMP increases (made from ATP) -> activates PKA.',NULL,1967,4,2),(###-04-25&&

1736    Q: How can an activated G-protein then decrease the level of cAMP

1736   A: What you have written is only ONE possible pathway using a G protein. There are other G proteins that have different effects. There are multiple G proteins, and one of them inhibits adenyl cyclase.',NULL,1968,4,2),(###-09-11&&

1737   Q: I have a question concerning bonds from Lecture 2. The chart we were given says that hydrophobic forces only work with water, but it seems to me that the same thing would happen with any polar/non-polar mixture (naturally it would not be called HYDROphobic, but it seems that the basic idea would be the same). Is this present more with water than with other substances, or am I reading too much into the matter

1737   A: I hadn\'t thought about that possibility, being focused on biological systems, which consists of aqueous  (cytoplasm) and non-polar (interior of membranes) environments. It seems logical. A quick Google search got me this paper from 1999 in which the authors use modern methods (atomic force microscopy) to measure the attraction between two non-polar surfaces\r\n http://www.ncbi.nlm.nih.gov/pubmed/9878136\r\nThey tested two polar solvents: ethanol and ethylene glycol. As the proportion of the those solvents increase in a mixture with water, the attractive force indeed decreased markedly, but apparently did not go to zero.  So it seems you are correct in predicting \"ethanolophobic\" forces but water still wins in this limited sample. However, you probably are \"reading too much into the matter\" (albeit in an interesting way) in the sense that those other solvents are not relevant to understanding the effects of weak bonds in biology (on earth). ',NULL,1969,1,1),(###-09-10&&

1738   Q: You mentioned that hydrogen bonds can occur between 2 organic molecules, such as one with an amide and ethanol. You then mentioned that if the amide and ethanol are in water, this interaction might not occur because of competition with water. What exactly did you mean by this? How do we know if water is more likely to hydrogen bond with itself or with other polar molecules that might be inside of it, such as this amide and ethanol

1738   A: It just a matter of numbers.  Water in aqueous solution is close to the molarity of pure water: 55 M.\r\nMost individual organic molecules in a cell are at a much lower concentration, I\'d guess about 0.01 M at best.\r\nThus water molecules outnumber an ethanol molecules trying form a hydrogen bond to an amide molecule about 5000 to 1.',NULL,1970,1,1),(###-09-21&&

1739   Q: You said that there is not always a conservation of amino acid sequences and in proteins with different amino acid sequences, you can get the same domain and this domain will serve the same function in both proteins. If the primary structure is different for two regions of two different proteins, how could these different amino acid sequences give rise to the same domain

1739   A: Apparently there is more than one way to arrive at a given structure (and function). In most cases, a domain is inherited and then modified in the course of evolution and so an underlying similarity in primary sequence is evident.  But in rare cases (1%?) the same structural domain shows evidence of two independent origins, using amino acids in a different order, yet producing a very similar 3-dimensional structure. Since the two cases converge on the same structure such cases are called convergent evolution. They are rare, but striking, as they demonstrate the strength of functional selection in evolution.',NULL,1971,1,1),(###-09-21&&

1740   Q: You said students sometimes make the mistake of calling one flat ribbon a beta sheet, but in reality, the association of at least two flat ribbons is what makes a beta sheet. Does that mean that in

Fig. 3-9 of Becker (8th Ed.) the way the authors have labeled just one ribbon as \"beta sheet\" in figure b below is incorrect

1740    A: Not strictly incorrect. The labeling could be considered correct but misleading. If a section of the polypeptide is drawn as a ribbon with a arrowhead then it is certainly part of a beta sheet, and the thin pointing arrow could be interpreted as acknowledging that fact, i.e., that it is part of a beta sheet. But one should never see a single ribbon in a depiction of a beta sheet, because it makes no sense for the way a beta sheet is defined,a as an interaction between two (or more) strands situated side-by-side in 3-D space. The \"hairpin loop\" consists mostly of a beta sheet here consisting of two anti-parallel strands, both of which are part of the beta sheet and each of which consist of a single stretch of polypeptide chain. \r\n\r\nI would also differ with the characterization in the legend that the connecting loop can be considered a \"random coil.\"  It could be either a random coil with no defined structure (waving about) or it could be held in a defined 3-D structure (tertiary) albeit not a secondary structure. ',NULL,1972,1,1),('0000-00-00&&

1741    Q: I assume that generic hydrocarbon chains can be saturated or unsaturated if carbons form double bonds, what process results in cis/ trans orientations?  Is it simply random/ energy related?  &&

1741    A: If it is a simple (uncatalyzed) chemical reaction then it would be governed first by randomness and then by avoidance (bias against sterically clashing group. If it is an enzymatically catalyzed process then it will be strictly governed by the enzyme to be either 100 % cis or 100% trans.',NULL,1973,1,1),('0000-00-00&&

1742    Q: Regarding purification/ denaturation, should we assume that when mercaptoethanol or similar reagent is used with SDS, that the SDS is added first and then the mercaptoethanol?  If the mercaptoethanol is added first, will it tend to not reduce the disulfide bonds in tertiary structure

1742    A: In practice, the two are added together, because you want complete breakage of the disulfides bond and so want to have free and complete access to them even if they are located in the interior of the tertiary structure. If you want to find out if they are in the interior, or are examining a student\'s understanding of what structure means, you may want to treat the protein first with mercaptoethanol and then remove it and treat with SDS to see what you have accomplished (mapping the disulfides that are on the exterior (accessible in the 3-D state). ',NULL,1974,1,1),('0000-00-00&&

1743    Q: How can we picture the shape of proteins that are partially denatured (i.e. still have S-S bonds in tact, but no weak bonds), versus ones that still maintain their weak bonds for the purposes of purification.  I would imagine the size/ volume increases but MW obviously remains the same.  Is the size increase enough to effect(/ produce different bands) for filtration/ PAGE/ UCf?  Probably depends on the resolution of the gel?  But can you please clarify

1743    A: A single polypeptide protein that has its disulfides (one or many) intact would be expected to migrate differently from either the native (compact) protein and the fully denatured random coil. Its effective diameter would be in between these two extremes and it would be expected to migrate accordingly, e.g., more slowly than the native protein in the ultracentrifuge, but  more rapidly than the random coil.',NULL,1975,1,1),('0000-00-00&&

1744    Q: I\'d like to ask about glucose fermentation following glycolysis. In the notes you talk about the efficiency and yield, but I\'m rather confused as to their difference. Could you clarify that

1744 A: Yield is how much energy (how many ATPs) you can get from a particular metabolic (energy) pathway). We discussed 2 such routes: fermentation, yielding 2APs per glucose molecule metabolized and respiration, which yielded 38 ATPS per glucose molecule metabolized.\r\n Efficiency is calculated by considering the amount of free energy captured as chemical energy (ATPs) divided by the amount of free energy released by a metabolic pathway. For fermentation, for example, there is 45 kcal/mole of glucose released by its conversion to 2 lactates, and 14 kcal/mole captured in the form of 2 ATPS. SO the efficincy of capture is 14/45, or 31%.',NULL,1976,2,1),('0000-00-00&&

1745 Q: I wanted to ask about question 12Bi) of problem set 3. The answer key says that leaving out arg in the medium would cause the mutants to die because they can\'t make arg and hence protein. But isn\'t the point of the pathway itself to make arg? (Also, how come normal E. coli survive fine without arg in the medium?)&&

1745 A: Normal E. coli (wild type) have all the enzymes needed to transform some glucose into arginine, which they need for proten synthesis. Thus they are able to grow in a medium that lacks arginine. The 3 mutants in question each have lost the enzyme activity of one of the necessary steps in this pathway, either because the enzyme cannot be made, or because it is made, but is altered suchthat it is incapable of catalyzing its step. The point of the pathway is indeed to make arginine. But bad thind=gs happen, th ea mutant has become deficient in one of these activities and so now requires arginine to be added if it is to grow. The gene that specifies the protein that is the enzyme for this step has been changes such that it now codes for a defective protein, as you will soon learn about. So:\r\nWT: glucose --> --> -> -->C --> A --> B --> arg\r\nBut a mutant: --> --> -> -->C --> A --| (accumulates C + A and cannot grow without arginine added)',NULL,1977,2,1),('0000-00-00&&

1746 Q: In the notes for condensation of nucleotides, it says that two phosphodiester bonds of XTP can be broken to form one Phosphodiester bond. are two phosphodiester bonds broken or two anhydride bonds? Can you please explain it?\r\n&&

1746 A: You have to split two phosphates off of ATP to get them on XMP and make XTP, and then you have to split both Phosphates off from XTP to make the phosphodiester bond in a nucleic acid.',NULL,1978,2,1),('0000-00-00&&

1747 Q: Looking online, I've found examples of codominance where a heterozygous child of a white and a red flower gives a white flower with red patches or a red flower with white patches. They then use your example of white and red flowers giving pink flowers as the example of partial dominance. Wouldn't looking at the flower with patches from farther away make it appear pink?\r\n\r\nIs it codominant because different cells somehow pick an individual allele to express and not the other vs partial dominance where each cell would express both alleles? So under a microscope you see individual white and red cells with codominance, but only pink cells with partial dominance?\r\n\r\nSo with blood type, A and B code for two enzymes, which are both made by different cells. The red blood cells most likely come into contact with both of these enzymes and that's why they have a mix of sugars on them

1747 A: Partial and incomplete are not different -- they are synonyms. It\'s co-dominance that\'s different. \r\nBut the truth is all these terms overlap, and are used differently by different people. The real point is to understand the biochemical bases of the various phenotypes, and how it produces the given pattern of inheritance. \r\nThe patchy business is usually called mosaicism. In plants, it\'s isn\'t inactive X. I\'m not sure how it works. (Plants don\'t have sex chromosomes.) \r\nAll cells in an AB

person make both enzymes. The sugars are added INSIDE the cells before the proteins are sent to the surface. The ABO gene is on an autosome, and there is no inactivation like X inactivation.