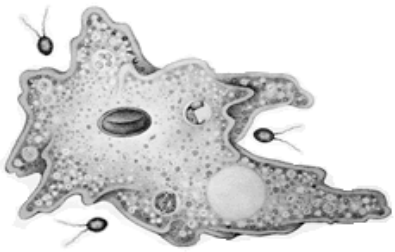
MCDB 1111 coreBio: Fundamentals Midterm 3 Name: _____

Directions: There are 10 questions, each worth 10 points. Remember, you can check "no idea" and you will receive 1 point (no reasoning is required). As before, in some cases you are asked to select the wrong answer or multiple correct answers. READ CAREFULLY to determine what the question asks you to do.



Q1: Both the replication of DNA and the transcription of RNA involve the synthesis of nucleotide polymers. **PICK ALL OF THE CORRECT** statements (there may be more than one).

☐ A. Both require a primer to start

✓ B. Both synthesis reactions proceed in the same direction.

✓ C. Both DNA and RNA polymerases depend on DNA to determine the sequence of the newly synthesized polymer.

✓D. Both DNA strands and RNA molecules have a distinct directionality (that is, there two "ends" differ from one another

☐ E. Both require a "ligase" to form the final molecule ☐ no idea

Explain why the wrong responses are wrong…

A: RNA polymerase does not require a primer and

E: RNA strands are not ligated together (while the lagging strands generated in DNA synthesis are. (n.b. while there is splicing of RNA, this involves a different mechanism).

Q2: A mutation occurs that leads to the presence of regions of RNA in newly replicated DNA molecules. A plausible model for this effect would be to assume that the mutation inactivated ...

☐A. the proof-reading activity associated with DNA polymerase

☐✓ B. the RNA exonuclease activity associated with DNA polymerase

☐ C. the DNA ligase

☐ D. the DNA-dependent, DNA polymerase

☐ E. topoisomerase I ☐ no idea

Explain the logic behind your answer and why the wrong answers are wrong:

DNA polymerase requires an primer to start the polymerase reaction. As the DNA-polymerase continued, when a lagging strand encounters an "upstream" RNA-DNA molecule (or a leading strand for that matter), an RNA exonuclease associated with this complex removes the RNA. The DNA-DNA junction is joined by the ligase. A mutation in the RNA exonuclease would leave the RNA in place.

Q3: A molecule has a short half-life; you can assume that that means that the molecule ….

☐ A. is rapidly synthesized ☐ B. is rarely synthesized

☐ ✓ C. is rapidly degraded ☐ D. is inherently unstable ☐ no idea

Explain the logic of your answer and explain why organisms would want to have molecules with short half-lives.

Half-life refers to populations of molecules, in which there is a high probability that a molecule will be rapidly degrade soon after synthesis. SO, if synthesis stops the population will disappear quickly. Since the behavior of organisms (cells) depends upon the molecules they contain, using molecules with a short half-life enables an organism rapidly change its behavior to suit changes in its environment.

Q4: The YMY gene is normally expressed only in the brain. In your studies, you discover a mutation that leads to the loss of YMY gene expression in the brain, although the brain forms apparently normally. The mutation affects only a single gene, but you are not sure that the mutation is in the YMY gene. Where else could it be?

☐ A. in the gene that encodes the DNA-dependent RNA polymerase required for transcription
☐ B. in any of the genes encoding tRNAs or rRNAs required for mRNA translation ☐ no idea
☐ ✓ C. in the gene that encodes the transcription factor that activates YMY gene expression
☐ D. in a gene that encodes a transcription factor that normally represses YMY gene expression

Explain the logic behind your answer and why the wrong answers are wrong:

The expression of the YMY protein in the brain is must be turned up specifically in the brain, which implies a gene for transcription factor that is expressed in the brain. Such a mutation would effect brain expression of YMY, but not lead to its expression elsewhere. If D were true, a mutation in the gene encoding the repressor would like to universal expression of YMY outside the brain, but would live brain expression normal. A and B would disrupt the expression of all genes, and the translation of all mRNAs, so you would not expect normal brain formation (of even that the original would be viable, mutations in such genes would be expected to be lethal).

Q5: A mis-sense mutation occurs in a highly (evolutionarily) conserved region of a polypeptide. Which type of mutation will likely have the **LEAST SEVERE** effect on the organism, a mutation ...

☐ A. that replaces a hydrophilic with a hydrophobic amino acid ☐ no idea
☐ B. that replaces a hydrophobic with a hydrophilic amino acid
☐ ✓ C. that replaces a hydrophilic amino acid with a different, similarly sized hydrophilic amino acid.
☐ D. that replaces a large hydrophobic amino acid with a small charged hydrophilic amino acid
☐ E. that generates a stop codon up-stream of the conserved region

Explain the logic of your answer and why the incorrect answers are wrong or irrelevant.

C would result in the smallest change hydrophilic amino acid for hydrophilic amino acid of a similar size. The others change amino acid type (A,B,D), changes more likely to change protein structure; while E disrupts (deletes) the conserved region, which because it is conserved is likely to deleterious.

Q6. DNA repair systems can recognize a mismatched base pair, the absence of a base, or a single-stranded break in a double-stranded DNA molecule because such mutations ...

☐ A. alter the polypeptide that the mutated gene encodes ☐ no idea
☐ B. alters the binding of transcription factors to the regulatory region of the mutated gene
☐ ✓ C. alters the structure of the DNA molecule
☐ D. generates a chemical signal

Explain the logic of your answer and why the incorrect answers are wrong or irrelevant.

The normal structure of DNA involves 1) continuous backbone (deoxyribose-P-deoxyribose) strands and AT and GC base pairs, which a extremely similar in physical dimensions. All of the changes mentioned change the structure of the DNA, a change that can be recognized by repair proteins. The effects on encoded polypeptides, or transcription factor binding are indirect; they influence selection of the organism. There is no obvious mechanism by which a change in DNA structure directly leads to the generation of a signal (although it is possible at activation of repair mechanisms themselves, might)..

Q7: In prokaryotes, the cell's DNA occurs as a single circular molecule, this means that DNA replication ...

☐ A. involves only leading strands ☐ no idea

☐ ✓ B. does not require telomerase

☐ C. does not require type I topoisomerase, only type II topoisomerase

☐ D. does not require DNA ligase

Draw a diagram of the replication of a circular DNA molecule and use it to explain the logic of your answer and why the incorrect answers are wrong or irrelevant.

No ends, no need for telomerase. The process still involves leading and lagging strands, and the other proteins mentioned**.**

Q8: You are asked to genetically engineer an organism so that it now incorporates a new (third) type of base pair in its DNA. What physical properties would **NOT** be required of such a base pair …?

☐ A. the same length as A=T and G=C base pairs ☐ no idea

☐ B. hydrophobic upper and lower surfaces of the base pair

☐ ✓ C. covalent bonds that link the bases to one another

☐ D. the ability to be linked to the deoxyribose group of the nucleotide backbone

Explain the logic of your answer (you can include a drawing if that helps, but it is not required).

A, B, and C are required for the new base pair to be compatible with an existing DNA molecule, they are like the properties of the A, T, C, and G bases and the AT and GC base pairs. Covalent bonds between the new bases would make them extremely difficult to break, and would like block both RNA transcription and DNA synthesis. It is more likely that these are H-bonds or van der Waals interactions.

Q9: The wild type (normal) ACAT gene encodes the 354 amino acid long ACAT polypeptide. A mutation occurs that changes the original translation start codon into a stop codon. What is (are) the most likely effect(s) of such a mutation (pick **ALL** that apply) …

☐ A. the mutant ACAT polypeptide will be longer than the wild type polypeptide ☐ no idea

☐ ✓ B. the mutant ACAT polypeptide will be shorter than the wild type polypeptide

☐ C. the mutant ACAT polypeptide will be the same length as the wild type polypeptide

☐ ✓ D. the mutant ACAT polypeptide may have a completely different amino acid sequence than the wild type polypeptide

☐ ✓ E. the mutant ACAT polypeptide that is made may have an amino acid sequence that is exactly the same as an amino acid sequence found in the wild type polypeptide.

☐ F. the mutation will inhibit the gene's transcription

For each possibility, make a schematic of the wild type and mutant gene/RNA/polypeptide and explain how it could occur.

For B, there may be another "in-frame" start codon downstream of the wild type start codon, leading to a shorter polypeptide

For D, the down-stream start codon is in a different reading frame frame the original start codon, leading to a much shorter polypeptide with a different amino acid sequence

For E, essentially the same answer as B.

Q10. **In his studies, Griffith found** that S-strain (smooth/virulent) bacteria grown in culture occasionally gave rise to R-strain (rough/avirulent) bacteria. How did he know that this was a **genetic** change?

Basically, because once it occurred, it was stable - R bacteria gave rise to R bacteria.
This is similar to the case with S bacteria, which the vast majority of the time gave rise to S bacteria, and only rarely to R (due to a mutation).

One could add (not necessary) that the ability of the trait to be transferred from dead S to living R was another piece of evidence for the genetic nature of the change.

Predict the relative probability of a R → S mutation compared to that of a S → R mutation?

☐ A. same as S → R ☐ B. much higher than S → R

☐ ✓ C. much lower than S → R ☐ D. impossible to say ☐ no idea

Explain the logic of your answer.

We presume that the S factor is active and that there are more ways to break (inactivate) the factor. SO S to R is (relatively) high. The probability of a random event fixing the broken S factor found in R cells is low, so the probability of such an event is lower.