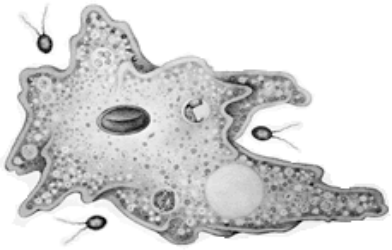


Q1: If genetic information were encoded in the living structure of cells, rather than in the nucleotide sequences within DNA molecules, Griffith's studies on the transformation in bacteria ..

□ A. would have produced exactly the same result

✓ B. would not have worked at all □ no idea

□ C. would have identified proteins as the genetic material

Explain (below) why the <u>correct</u> answer is correct

Because killing the S (virulent) cells would have lead to the loss of all that information. The information in DNA is in a stable chemical form, although to be read, it relies on the mechanisms (transcription and translation) found in living cells.

Q2. In his studies, Griffith found that S-strain (smooth + virulent) bacteria grown in culture very occasionally gave rise to R-strain (rough + avirulent) bacteria (a change from S → R).
Can you predict the relative frequency of a R → S mutation rate?

□ A. The same as the S → R rate □ B. Much higher than the S → R rate

✓ C. Much lower than the S → R rate □ D. impossible to say □ no idea

Explain the logic of your answer

Presumably the original S → R mutation disabled a gene. There are many more ways to break something than there are to fix it (in fact there may be only one way to fix a broken gene, reverse the original mutation). so the R → S rate is expected to be much lower.

Q3: A mutation occurs that leads to higher mutation rates in actively dividing cells, but which has no obvious effect on DNA in non-dividing cells. You would be justified in assuming that the original mutation inactivated ...

□ A. DNA-dependent DNA polymerase

✓ B. DNA polymerase's proof-reading activity

□ C. DNA-dependent, RNA polymerase (primase)

□ D. the repair of mutations due to the demethylation of C's □ no idea

Explain the logic behind your answer:

The effect is associated with cell division, which involves DNA replication. Replication occurs so that primase and the DNA polymerase must be ok. The demethylation reaction occurs in both dividing and non-dividing cells, so that is ruled out. That leaves the proof-reading function of the DNA polymerase, a defect in that activity would increase mutation rates in dividing cells (but not non-dividing cells).

Q4: PICK THE WRONG ANSWERS Which of the following statements is correct about DNA replication?

☐ A. DNA synthesis of the daughter strand always proceeds from 5′ to 3′ ☐ no idea

✓ B. DNA synthesis of the daughter strand always proceeds from 3′ to 5′

✓ C. DNA synthesis can occur in either direction depending on which strand is to be replicated

Explain the logic behind your answer (Hint: Draw a picture with labels and arrows indicating synthesis directionality for full credit) …

The correct response is that polymerization involves adding a new nucleotide to the 3′ end of an existing polynucleotide strand.

Q5: The YUM gene is normally expressed only in the skin cells of an organism. In your studies, you discover a mutant allele that leads to the expression of the normal YUM gene product in all cells of the organism. Which is the most plausible explanation?

✓ A. the mutation is in the regulatory region of the YUM gene ☐ no idea

☐ B. the mutation is within the coding region of the YUM gene

☐ C. the mutation alters DNA synthesis, leading to defect in primer synthesis

Explain the logic behind your answer

There is likely to be mutation in a regulatory region that normally acts to inhibit gene expression in non-skin cells.

Q6: As the percentage of GC in a double-stranded DNA molecule increases, what would you be completely and totally confident will occur?

☐ A. The rate of DNA synthesis will increase ☐ no idea

☐ B. The mutation rate will increase

☐ C. The separation of two strands of the DNA molecule, due to thermal motion, will increase

✓ D. The percentage of A in the DNA would decrease

Explain the logic behind your answer

Because %GC + %AT = 100% if GC increases AT (and so A) decreases.

Q7: A mutation occurs that leads to very high numbers of single stranded breaks in the replicated strands of a double-stranded DNA molecule, but with no obvious effects on the parental strands. 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 DNA ligase

☐ C. DNA-dependent, DNA polymerase

☐ D. topoisomerase I ☐ no idea

Explain the logic behind your answer:

During DNA synthesis, RNA primers are synthesized, start DNA synthesis, are then removed and replaced. DNA strands linked to together by ligase reaction; inactivation of ligation would leave strands as discrete (breaks).

Q8: Which is correct? the binding of a transcription factor to DNA …

☐ A. has no effect on the direction of transcription ☐ no idea
☐ B. determines exactly where translation begins
☐ C. determines where in the cell the encoded polypeptide will end up
✓ D. determines which strand will be used to generate an RNA
☐ E. determines when and where RNA primers are synthesized

Explain what will happen to the transcript (RNA) made if you were able to remove, rotated 180°, and reinsert back into to DNA the transcription factor's binding site (a diagram could be useful).

Transcription factors interact, directly or indirectly, with RNA polymerase, which position the polymerase on the DNA and the strand of DNA copied (into RNA). The other choices involve processes distinct from transcription.

Q9: Consider a cell. Which of the following processes are absolutely required to produce a functional transcription factor?

☐ A. DNA replication ☐ no idea
☐ B. transcription
☐ C. translation
✓ D. both transcription and translation

Explain the logic of your answer.

Transcription factors are proteins, protein synthesis involves information in DNA (genes), read out by transcription (RNA synthesis). These RNAs direct the synthesis of the polypeptides (translation) that form (alone or in multi-polypeptide complexes) to form the active transcription factor.

Q10: A protein has a short half-life, meaning that ….

☐ A. it is rapidly synthesized ☐ no idea
☐ B. it is rarely synthesized
☐ C. the mRNA that directs its synthesis is unstable
✓ D. it is rapidly degraded after it has been synthesized

Explain the logic of your answer.

Half-life involves the rate at which a molecule is degraded. Synthesis rates are normally constant, although the number of molecules synthesized per unit time can vary (from high to low). That said, if turn-over (degradation) rates are low, the protein will be stable, that is, have a long half-life, which is the time it takes for 50% of the molecules at time = 0 to disappear.

Q11: You are asked to genetically engineer an organism so that it now incorporates a new type of amino acid (not one of the normally used set of amino acids). Which molecule or molecular complex would you NOT need to change?

☐ A. one of the genes encoding a tRNA ☐ no idea

✓ B. the genes that encode the ribosome

☐ C. the gene that encodes the enzyme that adds the new amino acid to the tRNA

☐ D. the genes encoding the enzymes involved in synthesizing the new amino acid (assuming that it is not normally made by the organism)

Explain the logic of your answer.

The basic reaction (adding an amino acid to a polypeptide chain) remains the same, so the ribosome, which acts as a catalyst, of that reaction does not need to change. BUT you need a mutant tRNA and a mutant tRNA amino-acid synthetase that adds the non-biological amino acid to the mutated tRNA - of course the mutant tRNA needs to recognize a codon (perhaps a previously "non-sense" codon, so its anti-codon loop might also need to be mutated. Alternatively, you could re-specify a rarely used codon to encode the new a-biological amino acid.

Q12: We discussed a type of mutation that allows a stop codon to be read as an amino acid. Such a mutation would occur in a gene that encodes a …

☐ A. ribosomal RNA ☐ no idea

☐ B. messenger RNA

✓ C. transfer RNA

☐ D. a gene's regulatory region

Explain the logic of your answer (and why the other choices are wrong).

Because tRNAs read codons. Stop codons are stops to translation because normally no tRNA is around that can read them. If you mutated a tRNA gene's anticodon to match a stop codon, that tRNA could read the stop and insert the amino acid attached to it. As above the ribosome is just a catalyst and the message is read by tRNAs, it is the tRNAs that specify its meaning.

Q13: The time between the synthesis and degradation of particular RNA or protein is noisy (stochastic), like radioactive decay, because …

✓ A. it depends upon random collisions between molecules ☐ no idea

☐ B. it is determined by the molecule's structure

☐ C. it is based on radioactive decay

☐ D. it can be regulated by other factors

Explain the logic of your answer.

For any particular molecule, its degradation will depend upon a random collision with the enzyme that catalyzes the reaction. Which molecule will collide (productively) next occurs at random.

Q14: You isolate total tRNA from a cell and analyze its base composition (i.e. the ratio of the various nucleotides). This ratio will be …

☐ A. A = U ☐ no idea
☐ B. A = G
☐ C. the same as the bulk composition of the cell's DNA (but with Us instead of Ts)
✓ D. impossible to know based on the information supplied

Explain the logic of your answer.

because, while there are double-stranded regions of a tRNA, there are parts that are not double stranded (loops), and their composition is not constrained by base pairing. the region of DNA that encodes tRNAs is a minor percentage of total RNA (so C is wrong). A=G is never necessarily true in either DNA or RNA.

Q15: A mis-sense mutation can alter a polypeptide's 3D folding because ...

✓ A. a different amino acid is inserted at the site of the mutation ☐ no idea
☐ B. the polypeptide's synthesis stops prematurely
☐ C. any change at any position of a polypeptide will lead to misfolding
☐ D. it will alter the rate at which mRNA is synthesized

Explain the logic of your answer.

And the sequence of amino acids in a polypeptide determines a polypeptide's (protein's) 3D structure, particularly if there is a hydrophilic to hydrophobic (or vice versa) substitution).

Q17: For an organism to be able to survive a mutation that creates a non-sense suppressor, which must be true?

☐ A. the mutated gene must be relatively unimportant ☐ no idea
☐ B. the original mutation (the mutation that is suppressed) must be in a non-coding region
✓ C. there must be multiple genes encoding specific tRNAs
☐ D. the mutation must alter the region of the tRNA that determines which amino acid is attached to the tRNA

Explain the logic of your answer.

Otherwise, the codon recognized by the (original) mutated tRNA would be read as a stop, resulting in many, many, many mutations in many, many, many genes.

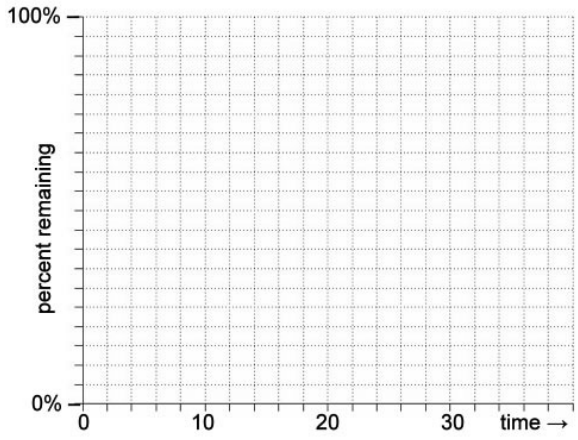
Q18: A non-sense mutation will always …

☐ A. lead to the production of a longer polypeptide ☐ no idea
✓ B. lead to the production of a shorter polypeptide
☐ C. lead to the production of a dysfunctional polypeptide
☐ D. generally have no effect on polypeptide function

Explain how the position of a non-sense mutation would be likely to influence polypeptide activity.

Because it leads to a new stop codon in the coding region, this stop codon has to be "up-stream" of the original one (leading to a shorter polypeptide).

Q16: You are studying a particular polypeptide; it has a half-life of 10 minutes. The cell contains 300,000 copies of this polypeptide.
At time 0 the synthesis of polypeptide stops completely.



Using a solid line draw a graph that represents the amount of polypeptide that remains as a function of time.

It will drop to 150,000 by 10 minutes.

How will your graph will change if there are only 10 copies of this polypeptide in the cell ☐ no idea

Given the small number of molecules, the graph will be step function, and will reflect the stochastic nature of the process.

How might a cell could benefit from making a protein with a short half life?

It enables the cell to change its behavior(s) rapidly, as the expression of one set of genes is turned off, their gene products will rapidly disappear, to be replaced by the new set of genes that come to be expressed.

Q19: You are studying the XUP gene of the speckled trout (a eukaryote). The XUP gene encodes a negatively acting transcription factor. You identify a mutation in the XUP gene and you find that the mutant Xup protein is secreted from the cell. Which is the most likely effect on the expression of genes whose transcription is directly regulated by the Xup protein?

☐ A. no effect, since it normally acts negatively ☐ no idea
✓ B. their expression would increase
☐ C. their expression would decrease
☐ D. the expression of all genes would increase

Explain the logic of your answer.

A negatively acting transcription cannot interact with the cell's DNA if it (the transcription factor) is secreted out of the cell… All genes normally repressed may be active.

Q25: Some genes are transcribed but not translated; pick the type of RNA that is both transcribed and translated.

✓ A. mRNAs ☐ no idea
☐ B. rRNAs
☐ C. tRNAs ☐ D. depends on the gene

Explain the logic of your answer (include why are the wrong choices wrong).

tRNAs and rRNAs are structure, no encoded polypeptide - only mRNAs are translated.

Q27: A mutation occurs that replaces an mRNA's normal start codon with a stop codon. Draw and explain what can happen ….

Another AUG (start codon), down-stream of the original start codon will be used. BUT the resulting reading frame may be stopped early (if it is the same as the original reading frame, the polypeptide will have an N-terminal deletion and may even be function.