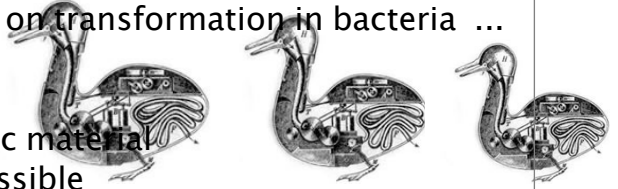1. If genetic information were encoded in the living structure of cells, rather than in the sequence of DNA molecules, Griffith's studies on transformation in bacteria ...
- ❏ would not have been effected
- X would not have worked
- ❏ would have identified proteins as the genetic material
- ❏ would have proved that evolution was impossible

a: is wrong because killing the bacteria would destroy the structure of the cell, so there would have been no information to transfer to the R cells.

c: is wrong proteins are not the "living structure of cells".

d: is wrong hereditable information could still be present in the living cell, and so this experiment would have no implications for evolution

2. A mutation occurs that leads to higher rates of mutation in actively dividing cells, but has no obvious effect in non-dividing cells. You would be justified in assuming that the mutation ...
- ❏ inactivated the DNA-dependent DNA polymerase
- X inactivated the proof-reading activity associated with DNA polymerase
- ❏ inactivated DNA-dependent, RNA polymerase
- ❏ inactivated DNA repair enzymes

since division requires DNA replication, the higher rate of mutation must influence some part of the replication machinery.

a: is wrong because this would block DNA replication completely, no DNA replication, no division.

c: is wrong because it would block transcription but without obvious effects on mutation rates.

d: is wrong because it would effects dividing and non-dividing cells, increasing the mutation rate in both.

3. Non-sense suppressor mutations ...
- X alter a tRNA's anti-codon
- ❏ alters the rate that DNA-dependent, RNA polymerase synthesizes RNA
- ❏ alter the ability of a ribosome to recognize an messenger RNA
- ❏ alters the specificity of an aminoacyl-tRNA synthetase

since the mutation is in a gene encoding a tRNA,

b: is wrong because the mutation would not alter RNA synthesis

c: is wrong because the ribosome would still recognize the mRNA; only if the non-sense suppressor mutation alter the anticodon of a methionine tRNA (which recognizes a translation start codon) and only if a single methionine tRNA gene were present in the cell would there be effects on ribosome-mRNA interaction.

d: is wrong because the synthetase does not "read" the anti-codon.

4. To say that a protein has a short half-life means….
- ❏ it is rarely synthesized
- ❏ it is inactive except in the presence of an allosteric effector
- ❏ it is short
- X it is rapidly degraded   ❏ no idea

half-life is a measure of stability, which is determined by degradation rate

a: is wrong because how often a protein is synthesized does not determine how rapidly it is degraded.

b: is wrong because allosteric factors typically regulate activity, not stability (although an allosteric factor could influence a protein's half-life.

c: is wrong because the length of a protein tells us nothing about its stability.

5. A mutation occurs in the region of a gene that is recognized by a negatively-acting transcription factor; such a mutation would most likely ….
- ❏ increase the rate of transcription in all genes
- ❏ decrease the rate of transcription of the mutant gene
- ❏ decrease the rate of translation of the mutant mRNA
- X increase the rate of transcription in the mutant gene   ❏ no idea

since this is a mutation in a region of a gene's regulatory region that normally binds a negatively acting transcription, it is likely to lead to the failure of that factor to bind, so the gene's transcription rate will increase.

a: is wrong because the mutation's effect would (most likely) be restricted to the mutated gene.

b: is wrong because the mutated region binds a negatively acting transcription factor

c: is wrong because the mutation would be unlikely to influence translation

6. You have two genes, _aya_ and _bub_. Both encode cytoplasmic polypeptides (AYA and BUB). The rates of transcription and translation are similar. The BUB polypeptide is 10 times longer than the AYA polypeptide. PREDICT the overall rate of AYA synthesis (# of polypeptides made per minute) compared to the BUB synthesis.
- ❏ both rates are similar
- ❏ AYA synthesis is 10 time faster than BUB synthesis
- X AYA synthesis is more than 10 time faster than BUB synthesis
- ❏ the BUB synthesis rate is faster than that for AYA

the question asks for the effect on # of polypeptide made per minute. which is a function of both the time required for transcription (a function of transcript length) AND the time required for translation (again a function of mRNA length).

a: is wrong because it takes ~10X longer to make the BUB mRNA and ~10X longer translate it. Both rates are involved in the total polypeptide synthesis rate within a cell

b: this would be the case if only translation were considered

d: this cannot be true, since it BUB is longer than AYA, and so takes longer to synthesize

7. A mutation occurs in the region of a polypeptide that, in the normal case, is buried within the molecule's interior. The mutation replaces a hydrophobic amino acid with a positively charged amino acid. You would be justified in predicting that such a mutation would …

    ❏ alter the polypeptide's location within the cell
    ❏ have little effect on the polypeptide's three dimensional structure
    ❏ function normally since it only effects the polypeptide's primary structure
    X produce a dramatic change in protein folding and activity

such a change, from hydrophobic to highly hydrophilic, would be likely to dramatically disrupt the polypeptide's tertiary structure (which would influence it interactions with other polypeptides, if it were part of a multiple subunit protein, and its function).

a: is wrong because there is no reason to believe that the change would a "targeting" sequence

b: is wrong because such a dramatic change would be likely to influence 3D structure

d: is wrong because primary structure (sequence) determines 3D structure and function

8. Mutations can occur throughout the sequence of gene. Consider a non-sense mutation that occurs at codon 42 of a 544 amino acid long polypeptide, and consider a similar mutation that occurs at codon 536 of a the same polypeptide. You would be justified in predicting that …

    ❏ both mutations would have similar effects on polypeptide function
    X the mutation at codon 42 would have a more severe effect
    ❏ the mutation at codon 536 would have a more severe effect
    ❏ more information is required to answer this question

Given that the normal polypeptide is 544 amino acids long, a non-sense mutation at codon 42 would lead to a 41 amino acid long polypeptide, must of the polypeptide would be missing. It is very unlikely that this polypeptide would retain any biological function.

a: is wrong because the two mutations have dramatically different effects on polypeptide length, and so likely polypeptide function.

c: is wrong because it would produce a 535 amino acid long polypeptide, only 9 amino acids shorter than the wild type protein. This slightly shorter protein could retain some function.

d: is wrong because there is enough information to make an educated guess.

9. Assume an organism used single-stranded DNA (rather than double stranded DNA) as its genetic material. If you knew the percentage of A in the DNA of that organism, you would know
- ❏ the percentage of T
- ❏ the percentage of G
- ❏ the percentage of C
- ❏ the percentage of C + G
- X nothing else (or rather the percentage of T + C + G)

Since the DNA is single stranded, there are no constraints on nucleotide composition.
a, b, c, and d are wrong because there no constraints on nucleotide composition

10. Assume that mutations in a <u>single gene, encoding a polypeptide</u>, are responsible for the change from the disease causing (virulent) S strain of Streptococcus to avirulent R strains. Based on this information you would predict that ….
- X the rate of S to R mutation are more frequent than R to S mutations
- ❏ the rate of R to S mutation are more frequent than S to R mutation
- ❏ R to S and S to R mutations occurs with equal frequency
- ❏ only nonsense mutations at the start of the gene's coding region can explain the S to R phenotype

there are many ways to mutate a gene to produce a change a polypeptide that makes it non-functional. Once non-functional, however, there are many fewer ways to reverse the effect. The probably of mutating from functional to non-functional is higher than mutations that have the opposite effect
b: R to S involves a much smaller subset of possible mutation that does S to R
c: is wrong because the two events are distinct differ in terms of functional effects.
d: is wrong because non-sense mutations are not necessarily involved in either process

11. Through the studies of Avery et al, it became clear the molecules that carried genetic information in cells were nuclease sensitive. Knowing what we now know about <u>prion disease</u>, which type of enzyme would be most likely to destroy the "infectious" activity of a disease sample?
- X protease
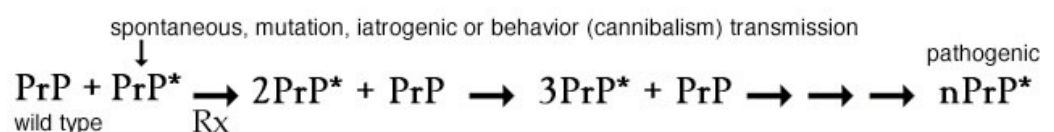- ❏ nuclease
- ❏ lipase
- ❏ reverse transcriptase

because the infectious agent appears to be a misfolded protein.
b: is wrong because nucleic acids do not appear to be involved in prion disease transmission
c: is wrong because lipids do not appear to be involved
d: is wrong because making a DNA based on an RNA does not appear to be involved in prion disease

12. In prion disease wild type PrP is converted into the

spontaneous, mutation, iatrogenic or behavior (cannibalism) transmission

$$\underset{\text{wild type}}{PrP} + PrP^* \underset{Rx}{\longrightarrow} 2PrP^* + PrP \longrightarrow 3PrP^* + PrP \longrightarrow \longrightarrow \longrightarrow \underset{\text{pathogenic}}{nPrP^*}$$

pathogenic PrP* form. This reaction can be influenced by mutation; alternatively the formation of PrP* can be initiated by surgical or behavioral "infection". Once present PrP* drives the transformation of PrP into PrP* (Rx). In the case of cannibalism-based disease vast majority of the PrP* found in the sick person differs from PrP...

❏ in its primary structure
X in its secondary and tertiary structure
❏ in the sequence of the gene that encodes PrP

Since this is cannibalism-based transmission, the victim (the cannibal) has the wild type protein, and the prion induces the misfolding of this normally normal prion.
a: is wrong because the host's protein's primary sequence is not altered
c: is wrong because the host's gene is not mutated