

MCAT Biology and Biochemistry 3 Homework

Passage 1 (Questions 1-6)

Passage Outline

Paragraph 1: Hypercholesterolemia = high levels of lipoproteins; LDL = risk of heart disease

Paragraph 2: LDLR gene function and mutations

Paragraph 3: ApoB = LDL/LDLR connection; ADH = LDLR protein binding LDL receptor

Figure 1: Interaction of ApoB, LDLR, ADH

Table 1: Plasma LDL profiles for FH

Q1.

Assess: This question presents a measurement about which we must draw conclusions using the data provided in the table, then expand upon with the described result of a liver transplant.

Plan: Ask the class what the specified LDL level indicates. Elicit- How would we have expected the liver transplant affect this child's condition? Why would the outcome be different?

Execute: This level of plasma LDL is characteristic of an individual with homozygous LDLR mutation (as can be seen from Table 1). Therefore, a liver transplant should be effective in providing the child with a liver that produces normal/wildtype LDLR protein. If this is not the case, then the high plasma LDL must be caused by another mutation if it is familial (which the questions says it is), then something outside the liver must be causing it. Choice A can be eliminated because mutant ADH proteins would also be replaced with the new liver. Choice B is wrong because gene crossover only occurs during meiosis and would not occur between cell types. The best answer is C, because ApoB proteins occur in the plasma and therefore replacing the liver would not necessarily help this phenotype. High expressivity is also a clue, since the table says the average plasma LDL for homozygous ApoB mutants is 250 and this child has a level twice that.

Answer: C

Q2.

Assess: This problem requires an understanding of the modes of genotype expression.

Plan: Elicit- What does it mean two types of proteins (normal and mutant) are being produced at the same time?

Execute: Here it is important to realize that half of the proteins are normal in these individuals, and the other half will be produced by the mutant allele. The individual will

express both proteins at once, therefore this is an example of co-dominance. You may be tempted to say incomplete dominance because of the data in Table 1 that shows the cholesterol levels are half in heterozygous compared to homozygous LDLR mutants. However, the question is asking about proteins, and since both kinds of proteins are present, it is co-dominance. In this case, the answer would be incomplete dominance if the individual LDLR proteins had both wildtype and mutant characteristics, but that is not the case, there are distinct normal proteins and distinct mutant proteins.

Answer: A

Q3.

Assess: This problem tests understanding of modes of inheritance.

Plan: Elicit- Which gene coded for the connecting protein between LDL and LDLR? Where can we find information about this gene in the passage? What type of mutation is it?

Execute: This question is asking about the ApoB gene. Paragraph 3 in the passage tells us this is an autosomal dominant mutation. Only one copy of the mutation is required, but 2 copies could end up with the same mutation due to the definition of autosomal dominant mutations.

Answer: C

Q4.

Assess: This question requires understanding of the genes described in the passage.

Plan: Review with the class information from the passage (paragraphs 2 and 3) which types of inheritance for the different genes would result in a normal phenotype. Check which statements describe a possible inheritance from two affected parents.

Execute: The patient could show no symptoms and be heterozygous for ADH given the results in Table 1. This could happen, too, if one parent was heterozygous for LDLR and one parent was homozygous for ADH. However, while heterozygotes for ApoB are non-symptomatic, this genetic makeup couldn't come from two affected parents. Finally, if I is true, III can't be true.

Answer: A

Q5.

Assess: This question requires understanding of the genes described in the passage and modes of inheritance.

Plan: Review with the class information from the passage (paragraphs 2 and 3) which types of inheritance for the different genes would result in a normal phenotype.

Execute: The key to this is that we recognize this is a family that has the recessive allele, and that ADH is the only recessive cause of FH that is mentioned in the passage. You may be tempted to think it is a dominant pedigree because so many are affected, however you need to remember that prevalence does not mean dominance.

Answer: B

Q6.

Assess: The key to this problem is to carefully consider all of the information presented in the question stem.

Plan: Ask the class to set up a Punnett Square based on what the question stem describes. Be sure to point out to them there is one possibility from this square that should not be taken into consideration.

Execute: The first key is to establish that the gene is recessive and recognize that both parents must be carriers. Also remember that the daughter is unaffected. She therefore cannot be homozygous recessive for the recessive FH allele. In the Mendelian cross, the parents would make 25% AA, 50% Aa, and 25% aa individuals. However, since this daughter is not aa (since we know that she doesn't have the disease), the probabilities change to 33% AA and 67% Aa (carrier).

Answer: D

Passage 2 (Questions 1-6)

Passage Outline

Paragraph 1: Intestines = digestion and absorption

Paragraph 2: Gap junctions

Paragraph 3: Ion movement in the large intestine

Paragraph 4: Cancer protection in the small intestine

Q1.

Assess: Notice that the answer choices are all simple statements.

Plan: Elicit- Why do gap junctions exist in certain areas?

Execute: You should remember from the passage (paragraphs 2 and 3) that the intestines are mainly involved in absorption. Hence the gap junctions allow the intestines to do their function.

Answer: B

Q2.

Assess: The question wants to know the consequence of low sodium.

Plan: Find where sodium is discussed in the passage.

Execute: Based on the passage we should realize that a low sodium diet would result in increased expression of epithelial sodium channels to allow reabsorption of sodium.

Answer: C

Q3.

Assess: The question wants to know the consequence of activating chloride secretory channels.

Plan: Elicit- What does the passage tell us about movement of Cl^- ions?

Execute: Based on the passage we know that Na^+ and water follow the Cl^- ions. Hence cholera causes loss of fluids from the body. The treatment of the disease involves replacing the fluids lost from the body.

Answer: D

Q4.

Assess: Another problem requiring an understanding of the gap junctions described in the passage.

Plan: Elicit- What does the passage tell us about the kinds of molecules that pass through gap junctions?

Execute: The passage states that only very small molecules can pass through gap junctions. Only C is small, the rest are large proteins.

Answer: C

Q5.

Assess: This problem asks us to draw a conclusion using information from the passage.

Plan: Elicit- What did the passage say about benzpyrene hydroxylase? What can we conclude about the relationship between this action and formation of cancer?

Execute: The passage states that benzpyrene hydroxylase breaks down polycyclic aromatic hydrocarbons and that benzpyrene hydroxylase protects against cancer through this action. Therefore, polycyclic aromatic hydrocarbons must contribute to cancer formation so having less of them decreases the risk of cancer.

Answer: A

Q6.

Assess: The key here is to apply the new information in the question stem to the function described in the passage.

Plan: Elicit- What did the passage describe as the function of the paracellular pathway? If this function is reduced, what will be the effects? How does this then impact the consistency of the material within the lumen?

Execute: The paracellular pathway controls movement of ions and water. Less function of the paracellular pathway means less Na^+ and water in the lumen which means the material will not have as much water in it and be less fluid.

Answer: D

Passage 3 (Questions 1-5)

Passage Outline

Paragraph 1: MHC intro and role in adaptive immunity

Paragraph 2: MHC II epitope interaction with T-cells

Paragraph 3: MHC I role in self-recognition by T-cells

Paragraph 4: MHC encoded by chromosome 6 genes

Figure 1: MHC I and MHC II complexes

Q1.

Assess: This question tests understanding of the passage information and knowledge of cell types.

Plan: Elicit- According to the passage what does it mean to be a part of the adaptive immune response through MHC complexes?

Execute: MHC complexes are either expressed on phagocytic cells or nucleated cells (paragraphs 2 and 3). The only example of a non-nucleated cell listed here is Choice B. All other answer choices are examples of nucleated cells, and are therefore capable of presenting epitopes and activating the specific immune response.

Answer: B

Q2.

Assess: A preview of the answer choices shows they all deal with Mendelian genetics.

Plan: Ask the class what it means for both alleles to be expressed equally.

Execute: This is a crossover question with their Mendelian genetics content. When the phenotype is expressed equally and simultaneously, it is a co-dominant trait. Therefore, the individuals would express two slightly different alpha 1 proteins, for example. It is not an example of incomplete dominance because the trait is not a mixture of the two proteins.

Answer: C

Q3.

Assess: This question tests for a knowledge of the steps for the immune response process.

Plan: Elicit- What happens immediately after a bacterial invasion? What then is the first type of cell to arrive on the scene?

Execute: The question is asking which is the most plentiful lymphocyte at the tissue level immediately after infection. Helper T cells are not normally located here, however Natural Killer cells are recruited very quickly to these sites. B cells are not recruited to

sites of infection, and macrophages are also quick responders but they are just leukocytes, not specifically lymphocytes.

Answer: B

Q4.

Assess: The key here is to be able to use all of the information presented, whether it is in the written passage or accompanying tables and/or figures.

Plan: Elicit- Where do we get an idea of where alpha 3 subunit is and the role it plays?

Execute: As seen in Figure 1, the alpha 3 subunit is responsible for anchoring the MHC I complex to the cell surface. Therefore, if this is absent from the complex the placement on the cell membrane will not happen. B is the best match. A and D would be more affected by the lack of expression of MHC II. The ability to produce lymphocytes should not be affected.

Answer: B

Q5.

Assess: Here we have to relate the information from the passage to our understanding of the immune response process.

Plan: Move through each of the answer choices looking for the one that uses support from the passage to explain a decreased immune response.

Execute: A mutation rendering the transmembrane domain of the MHC II complex nonfunctional will make it impossible for the MHC II complex to exist on the cell membrane where the complex needs to be to present pathogenic particles. This presentation initiates an immune response so if it doesn't happen, immune responses are decreased.

Answer: B

Passage 4 (Questions 1-5)

Passage Outline

Paragraph 1: Intro edema and its causes

Mechanism 1: High capillary pressure

Mechanism 2: Decrease plasma concentration

Mechanism 3: Lymphatic obstruction

Mechanism 4: High capillary porosity

Q1.

Assess: This problem requires us to compare the various mechanisms presented in the passage.

Plan: Elicit- Which of the mechanisms described something also tied to the immune response?

Execute: Mechanism III is all about lymphatic tissue. A block in the lymphatic system will also block immune function.

Answer: C

Q2.

Assess: After reading about various causes of edema, we must now identify a situation that would not cause the condition.

Plan: Since all of the described processes will cause edema, we are looking for the answer choice that is not one of the mechanisms in the passage.

Execute: Decreased fluid reabsorption by the kidneys leads to greater urine output. This means the body is losing fluid, which is the opposite of edema. It is also the only choice not connected to one of the described mechanisms.

Answer: A

Q3.

Assess: Here we have to compare two of the mechanisms from the passage.

Plan: Ask the class what the two mechanisms have in common, then discuss how this would affect fluid movement in the body between the plasma and interstitium.

Execute: As the question stem even points out, both mechanisms involve decreased plasma protein concentration. Since water follows ion movement, that means in cases of edema where the fluid moves towards the interstitium there must be more ions in the interstitium. Under normal circumstances, the reverse must be true: more ions would be found in the plasma.

Answer: C

Q4.

Assess: This problem tests the ability to make a conclusion about a proposed treatment using the information in the passage.

Plan: Elicit- Where in the passage is there a relationship described between protein synthesis and edema? How does increasing plasma proteins affect this relationship?

Execute: Mechanism II says edema can be caused by a loss of protein in blood, leading to an inability to retain water in capillaries. Increasing cortisol increases protein synthesis and more protein in the blood increases water flow into blood. This would definitely decrease edema.

Answer: A

Q5.

Assess: The key here is to simply focus on the information in the passage rather than get distracted trying to think about the inflammatory response.

Plan: Elicit- Where in the passage is histamine mentioned?

Execute: Mechanism IV says that histamine can make capillaries leaky, leading to edema or swelling.

Answer: C