AP® BIOLOGY
2008 SCORING GUIDELINES

Question 1

1. The physical structure of a protein often reflects and affects its function.

(a) **Describe** THREE types of chemical bonds/interactions found in proteins. For each type, **describe** its role in determining protein structure. **(6 points; 1 point for bond/interaction description, 1 point for description of role)**

Bond/interaction	Description	Role associated to bond/interaction
Covalent/ peptide	sharing electrons **OR** linking amino acids together	amino acid sequence **OR** primary structure (no credit for chain or polypeptide alone)
Disulfide/ covalent	disulfide, S–S bond (bridges); sulfur-containing R group bonding	tertiary or quaternary structure
Hydrogen	H–O or H–N interactions	α helix, β sheet; secondary, tertiary, or quaternary structure
van der Waals	unequal electron clouds in R group; dipole moments	tertiary or quaternary structure
Hydrophobic	nonpolar R groups	tertiary or quaternary structure
Ionic	charged R groups	tertiary or quaternary structure

(b) **Discuss** how the structure of a protein affects the function of TWO of the following. **(3 points maximum)**

Muscle contraction **(1 point for each bullet; 2 points maximum)**

- Actin (thin filaments) and myosin; cross-bridges OR filamentous proteins slide past each other.
- Troponin/tropomyosin interaction blocks binding of myosin to actin.
- Ca^{2+} changes troponin shape/binding of troponin-tropomyosin to actin altered.
- ATP/ADP changes myosin structure.

Regulation of enzyme activity **(2 points maximum)**

- Shape change caused by **(1 point for each bullet)**
  - Binding of allosteric or noncompetitive inhibitor.
  - Binding of allosteric activator.
  - Feedback control.
  - pH or temperature changes.
  - Cleavage of pre-enzyme (e.g., zymogen).
  - Cooperativity; coenzymes; cofactors.
  - Covalent modification (e.g., phosphorylation).
- Competitive inhibitors binding in the active site prevent substrate binding.

NOTE: The active site regulating enzyme activity is not enough to earn a point.

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Question 1 (continued)

Cell signaling **(2 points maximum)**

- Receptor-ligand binding **(1 point for each bullet)**
  - Event: Ligand binds specifically to receptor.
  - Result: Receptor structure altered by binding, transducing signal through membrane.

  Examples may include hormones, neurotransmitters.
- Enzyme-linked receptors: binding of ligand causes enzyme to catalyze reaction.
- Gap junctions: shape of junctions allows for passage of regulatory ions or molecules.
- Ligand-gated channel: binding of ligand opens channel.
- Immune signaling: leads to activation of cells.

(c) Abnormal hemoglobin is the identifying characteristic of sickle cell anemia. **Explain** the genetic basis of the abnormal hemoglobin. **Explain** why the sickle cell allele is selected for in certain areas of the world. **(3 points maximum)**

Genetic basis **(2 points maximum)**

- Point mutation in DNA; base substitution leading to a different amino acid in the hemoglobin.
- Changing glutamate (glutamic acid) to valine (in β-globin).

Selection **(2 points maximum)**

- Sickle cell condition protects against or resists malaria.
- Changed hemoglobin leads to oxygen-deprivation minimizing malarial infection.
- Heterozygotes maintain a reproductive advantage/success.

NOTE: Stating that sickle cell confers immunity to malaria does not earn a point.

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BIOLOGY
SECTION II
Time—1 hour and 30 minutes

1A1

Directions: Answer all questions.

Answers must be in essay form. Outline form is not acceptable. Labeled diagrams may be used to supplement discussion, but in no case will a diagram alone suffice. It is important that you read each question completely before you begin to write. Write all your answers on the pages following the questions in this booklet.

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   - Cell signaling

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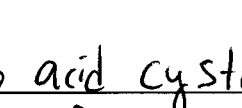
a) i) the Amino acid cysteine  Contains a SH (gonmasulfur) this sulfur may form a covalent bond with another cysteine to form a disulfide bridge. This has a tremendous stabilizing ~~effect~~ on the conformation because of the strength of a covalent bond. As such, ~~disulfide~~ disulfide bridge ~~(S-S)~~ will cause suffurs to orient near each other in the tertiary & Secondary Structures. Disulfide bridges also stabilize protein structure (and can help ~~confer~~ such things as Therme Stability)

ii) ~~Vander~~ Van der Waals interactions are also a common interaction in proteins. Van der Waals attractions result from the temporary realignment of electrons in the orbitals of two nearby atoms. As a result, large molecules w/many electrons often experience greater Van der Waals forces (also called London dispersion forces). This is one reason why amino acids w/large carbon side chains like Leucine, Isoleucine, Valine, & Phenylalanine agglomerate near each other, they mutually exert large VDW forces on each other because of this agglomerative tendancy (and their hydrophobicity) these nonpolar amino acids clump together on the interior of proteins. The agglomeration also plays a part in the formation of some proteins ~~structure~~ quaternary

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structures.

(ii) Hydrogen bonds (H-bonds) are a type of polar bond between oxygen, nitrogen or fluorine & Hydrogen & result in a relatively strong bond. H-bonds are critical in the formation of helices as the carbonyl groups and the amine groups form hydrogen bonds. Beta sheets are likewise overridingly determined by the presence of H-bonds. Since these two secondary structures are vital to the tertiary structure as a whole, they play a pivotal role in structure.

b) Enzymes (a type of protein) have a particular location called an active site. This site is where substrate(s) bind to the protein & some reaction is carried out. The regulation of enzyme activity most often involves the inhibition of a substrate (S) reaching the active site. The site may be blocked by a competitive inhibitor which prevents substrate-binding because it is. A competitive inhibitor is some molecule which enters the active site and blocks it, leading to no enzyme activity. The protein may also have its active site deformed by a non-competitive inhibitor, a molecule that binds somewhere other than the active site but deforms the active site so that it no longer functions. Some proteins are allosteric having two forms but are stabilized in one form by the binding of another molecule. These two forms may have completely different functions.

ii) Many cell signal receptors are proteins. They too have an active site where a molecule may bind. In the case of a signal receptor protein, on the other hand, work is not done on the substrate, rather the substrate causes a change in protein structure, activating or inactivating it. In G-protein linked reception, the result is the phosphorylation of a G protein by GTP, setting of a phosphorylation cascade through protein kinases. Because the active site can be so distinguishing & selective, it allows only very specific molecules to bind to a given protein. Many signal proteins are transmembrane as well and so have two hydrophilic ends for the cytoplasmic & ECM sides and a hydrophobic region for the membrane.

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1A 3

ADDITIONAL PAGE FOR ANSWERING QUESTION 1

3) These abnormal hemoglobins are caused by a substitution mutation in the gene for one of hemoglobin's subunits. This mutation causes a histidine – leucine swap and radically changes the proteins structure However, the sickle-cell allele is selected for in some parts of the world like West Africa where malaria incidence is high. Sickle cell confer malaria resistance and so there is a selective influence for the allele.

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BIOLOGY
SECTION II
Time—1 hour and 30 minutes

1B1

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   - Muscle contraction
   - Regulation of enzyme activity
   - Cell signaling

   (c) Abnormal hemoglobin is the identifying characteristic of sickle cell anemia. **Explain** the genetic basis of the abnormal hemoglobin. **Explain** why the sickle cell allele is selected for in certain areas of the world.

a. The physical structure of a protein is affected by several types of bonds and interactions. One is the protein's primary structure, which is the order of the different amino acids in the protein, which are bonded together via dehydration synthesis. Another is the protein's secondary structure, which is a shape formed due to hydrogen bonds among the amino acids; the final shape is either an alpha helix or a beta pleated sheet. A third is the protein's tertiary structure, which is the combination of α helices and β pleated sheets within a protein. All three of these interactions affect a protein's overall physical structure/shape.

b. The structure of a protein greatly affects its function. In proteins that are enzymes, the shape affects the enzymatic activity and regulation. In the enzyme, the active site is where the substrate binds, which makes the shape of the active site vitally important for the enzyme to assist in reactions. Enzyme regulation is also affected by the shape

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ADDITIONAL PAGE FOR ANSWERING QUESTION 1

of the enzyme. Allosteric inhibition is when an allosteric inhibitor binds to an enzyme at a location other than the active site, thereby affecting the enzyme's ability to assist in a reaction. The shape of the allosteric site needs to fit the inhibitor for allosteric inhibition to work. Competitive inhibition is when an inhibitor competes with the substrate for the active site, so the shape of the active site is once again very important.

Cell signalling is also affected by the shapes of proteins. Signals are often relayed between cells by the sending and recieving of molecular signals. Signals are recieved by receptor proteins. In order for the receptor protein to be able to recieve the signal, the shape has to fit; therefore the shape of the protein is important. Signals are also relayed through channel proteins in cell junctions, in which case the protein needs to be the right shape to permit the passage of certain chemical or molecular signals, so the shape of the protein is vital.

c. Abnormal hemoglobin is [redacted] passed down by a sex-linked recessive allele. This abnormal hemoglobin is the identifying characteristic of sickle cell anemia, which is a disease characterized by the abnormal shape of hemoglobin cells. The shape of the hemoglobin protein is so important that it causes a disease, sickle-cell anemia. The shape of the protein is also why in some areas the gene is selected for, because protein shape is so important.

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BIOLOGY
SECTION II
Time—1 hour and 30 minutes

1C1

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a Depending on the types of bonds in protein, a protein can be twisted into an alpha helix (secondary level), folded into a beta pleated sheet (secondary level), or folded into itself at the tertiary level. This alters the physical appearance of the protein.

b An enzyme has ~~primary~~ an active site which a substrate can bind to, according to the lock-and-key theory of enzymes. However, in competitive inhibition, another substance binds to the site, preventing the normal substrate from binding, and regulating the reaction. Enzymes may also contain allosteric site. An inhibitor can bind to the allosteric site in noncompetitive inhibition, changing the structure of the protein and preventing the substrate from binding. Or, the enzyme can be turned off by default, until ~~a~~ promoter binds to the allosteric site and changes the shape of the protein so that the substrate may fit.

Muscle contraction requires a sodium-potassium pump, a protein that aids in the exchange of sodium and potassium ions required for nerve impulses. The structure of this protein will need to allow sodium and potassium to bind to the protein in order to be properly exchanged.

c Sickle cell anemia is an autosomal trait caused by recessive alleles. It inhibits hemoglobin from functioning normally but it provides resistance to malaria. In certain areas of the world where ~~mosquitoes~~ malaria is a threat, ~~life in areas~~ such as in areas of Africa, the trait is selected for since individuals with it carry a resistance to malaria, which ~~is~~ poses a greater problem than the ~~effects of the~~ abnormal hemoglobin. in its heterozygous state

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AP® BIOLOGY
2008 SCORING COMMENTARY

Question 1

Overview

This question tested knowledge of protein form and function from the basic to the applied level. Students were asked to describe the bonds involved with protein structure and apply their knowledge of protein structure to specific functions, such as muscle contraction, cell signaling, and enzyme regulation. Students were expected to integrate their knowledge of molecular genetics, biochemistry, and population biology using sickle cell anemia as a model.

Sample: 1A
Score: 10

Part (a): 6 points were earned. The student provides an extensive description for disulfide bonds (1 point) and states that the bonds "stabilize protein structure," mentioning their involvement in tertiary structures (1 point). Two more points were earned for a thorough description of van der Waals interactions and their association in protein quaternary structure. A final 2 points were earned for an H^+ bond explanation (bonding between oxygen or nitrogen and hydrogen).

Part (b): 3 points were earned. The student earned 2 points for describing competitive and noncompetitive inhibitions relating to enzyme regulation. The third point was earned for the discussion of substrate (ligand) interactions, causing a change in the protein ("signal receptor protein") structure. The student goes further, providing details on G-protein signaling; however, no point was given since the student had already earned the maximum 3 points for part (b).

Part (c): 1 point was earned. The genetic explanation behind sickle cell anemia was given ("substitution mutation"), earning 1 point. No point was given for the amino acid change, since this was incorrect. The student states that this allele provides resistance to malaria, and therefore the "allele is selected for" in the population, but did not earn a point for this since the maximum 10 points for question 1 was already earned.

The student earned the maximum 10 points out of a possible 12 points.

Sample: 1B
Score: 6

Part (a): 3 points were earned. The student indicates that peptide bonds are involved in the primary structure and gives a description of peptide bonds as when amino acids are "bonded together via dehydration synthesis," earning 2 points. The description of the H^+ bond role in secondary structure earned 1 point. The student mentions that the tertiary structure is made from α helices and β sheets but gives no bond or interactions.

Part (b): 3 points were earned. The student earned 2 points for describing both allosteric and competitive inhibitions in relation to enzyme regulation. The receptor-ligand specificity point was earned for the discussion of cell signaling. The student provides a good description of cell junctions, but no point was earned since the 3-point maximum for part (b) was already reached.

Part (c): No points were earned because the student does not explain the genetics of sickle cell anemia, nor its association with malaria.

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Question 1 (continued)

Sample: 1C
Score: 3

Part (a): No points were earned. The student describes various protein structures but provides no bonds or interactions to account for them.

Part (b): 2 points were earned for the student's explanation of competitive and noncompetitive inhibition with regard to enzyme regulation. No points were given for the muscle contraction description.

Part (c): 1 point was earned for the student's explanation that sickle cell anemia "provides resistance to malaria."

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