

Biochemistry essay

[Science](#), [Genetics](#)



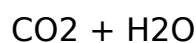
The differences between the oxygenated and deoxygenated states of hemoglobin

Erythrocytes of the blood act as carriers of hemoglobin; a four unit protein that through binding process with oxygen molecules, thereby forming oxyhemoglobin, transports oxygen to various part of an animal's body. Oxygenated and deoxygenated states of hemoglobin have different quaternary structure. The disparity in quaternary structures of these two kinds of hemoglobin is attributed to the binding of oxygen with hemoglobin. The beta chains in oxygenated hemoglobin are much closer to each other compared to the ones in the deoxygenated hemoglobin (Campbell & Farrell, 2009). In the same light, the quaternary structure is affected by the hemoglobin's acid-base properties. The oxygenated form of hemoglobin is a stronger acid than the deoxygenated form; thence it confirms the high affinity for H⁺ by the deoxygenated hemoglobin (Campbell & Farrell, 2009). The two states of hemoglobin have different crystal structure; the oxygenated hemoglobin contains scarlet needles, reducing space at the center while the deoxygenated hemoglobin contains plates, which creates a wide space at the centre. Russell, Wolfe, Hertz, Starr & McMillan (2008) affirm that the two kinds of hemoglobin contain different colors; the color of oxygenated hemoglobin is bright red while deoxygenated hemoglobin contains a dark red coloration. This is illustrated in fig. 1 below

Fig. 1

3. The Bohr Effect in the association and disassociation of oxygen and hemoglobin

Bohr Effect can be referred to as the change in the oxygen affinity attributed to the change in the plasma pH. It links and enhances the transportation of oxygen and carbon (CO₂) within the body tissues. In the lungs, the pH of the plasma is higher than the actively metabolizing tissues. This facilitates the binding of oxygen with hemoglobin. In the mean time, H⁺ ions are released by hemoglobin, which further reduces the pH. In actively metabolizing tissues, the high hemoglobin affinity for oxygen is reduced by the increase of carbon (IV) oxide (CO₂) concentration. The core mechanism of Bohr Effect is depicted through the formation of carbonic acid (H₂CO₃), a reaction between CO₂ and water (H₂O), catalyzed by carbonic anhydrase present in the red blood cells.



carbonic anhydrase H₂CO₃

The carbonic acid dissociates into HCO₃⁻ and H⁺. HCO₃⁻ ions, and then is transported to the plasma. Increase in H⁺ decreases the intracellular pH. Similarly, it causes the protonation of the key amino acids, which encompasses the N-Terminals of the alpha chains and His146 of beta chains, a condition favorable to the deoxygenated form of hemoglobin (Campbell & Farrell, 2009).

Fig. 2: Relationship between oxygen affinity and pH

Fig. 3a: Molecular Structure of hemoglobin

Fig. 3b: Molecular Structure of Myoglobin

Sickle cell Anemia

Difference between diseased cells and normal red blood cells in the context of their capacity to carry oxygen

Oxygen content; the amount of oxygen in the blood, and oxygen carrying capacities are the key components in the transportation of oxygen in the blood system, depend on the amount of hemoglobin in an individual's blood. Oxygen content varies directly as hemoglobin content. Mathematically, oxygen content can be expressed as follows

Oxygen Content = Hemoglobin Bound Oxygen + Dissolved Oxygen

Abnormal hemoglobin may cause abnormal red blood cell that contain a reduced surface area, necessary for the diffusion of oxygen in and out of the cell. In addition to this, abnormalities of skeletal proteins, integral proteins and anchoring proteins may result to abnormality of red blood cells, in tandem to their early destruction by the ability to maintain their surface area and volume (Yawata, 2003).

Similarly, the abnormalities in the blood cells may result to low levels of hemoglobin concentration, thence; there will be a decrease in the oxygen carrying capacity of the blood. This results to low transportation of oxygen from the lungs where there is high concentration of oxygen to the actively metabolizing tissues, where there is low concentration of oxygen (Hillman, Ault & Rinder, 2005).

Fig. 4: Picture showing the difference between sickle cells and normal red blood cells in the context of their capacity to carry oxygen

Source: www.pathologystudent.com

Molecular inheritance of sickle cell anemia

The sickle cell anemia is because of mutation caused by the substitution of a single nucleotide in the sixth codon of the beta-globin gene, changing a glutamic acid to a valine (Chowning, 2000). Basically, Sickle cell anemia is caused by the breakdown of red blood cells. The victims of the disease are subjected to long term chronic diseases of the heart, kidney and the brain among other principle organs of the body. The Sickle cell anemia is a recessive gene and it contains two-beta globin alleles important for the inheritance of sickle cell anemia; A and S. Homozygous individuals display two distinctive characteristics (Chowning, 2000). Individuals with two normal A alleles (AA) have normal red blood cells ascribed to the normal formation of hemoglobin. Individual who develop sickle cell anemia, contain the two mutant S alleles (SS). The heterozygous individuals (AS) for the sickle cell alleles produce both normal and abnormal hemoglobin. They are usually healthy with minor symptoms of sickle cell anemia, only predominant in conditions of low oxygen supply (Chowning, 2000). The major symptoms of the disease are jaundice and a pale yellow skin. Other sundry symptoms include; cold, constant headaches. In addition, victim suffer from acute bone pains. It is worth noting that sickle cell anemia destroys the spleen; an explanation of the increase of infections of sickle cell anemia patients especially young children who have relatively weak immune system.

Fig. 5a: A demonstration of molecular inheritance of sickle cell anemia from heterozygous parents

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Fig. 5: A demonstration of molecular inheritance of sickle cell anemia from homozygous parents

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