**Literature Review:**

What is the effect of the structure of a red blood cell vs a sickle red blood cell?

 What goes on inside the tiny membranes of red blood cells? To get a closer look and become possibly in reach of a solution to the sickling blood cell; measures such as analyzing the structure of a normal red blood cell vs a sickle red blood cell must be observed for there may be key evidence inside the structure that could be of use. Beginning with the membrane of a normal, healthy erythrocyte, scientist have found that an erythrocyte is structured upon a flexible membrane, constantly bending and conforming to the tiny size of a vein and/or artery before springing back to its normal shape. However, in the membrane of sickle cell, the sickle shape is rigid and so far out of its normal shape that it is unable to effectively move through arteries without clogging the vein itself. On a molecular level, the sickling is due to a mutation in the beta globin chain. These results in insoluble polymers forming which in a significant concentration can eventually lead to the sickling of the erythrocyte (Marengo-Rowe, MD, Alain J.). The sickle erythrocyte becomes hard and sticky unlinke the characteristics of a normal, healthy blood cell which is round and soft.

 I f scientist were to take an approach such as fixing the genetic mutation, there would be at least two large factors to consider. The first factor is that scientist cannot predict when a genetic mutation will occur, no matter what disease there is. In sickle cell anemia, symptoms do not start to show until 4 to 6 months old because of another type of hemoglobin; hemoglobin F, or otherwise known as fetal hemoglobin. Fetal hemoglobin is produced as a child up until about 6 months however the exact time when Hemoglobin F switches to producing adult Hemoglobin A is unknown (Amuzu, Dominic). For this reason only is why the symptoms of sickle cell anemia do not show until at least 6 months after birth. The role of fetal hemoglobin in sickle cell will be later explored throughout this paper. Due to the unknown time where the cells begin to sickle, scientists are at a great disadvantage in preventing this sickling. The second factor to take into consideration concerns the fact that scientist have not found an effective way, if any way at all to revert a genetic mutation. Throughout history and the scientific theory of evolution, and primary succession, mutations in an organism cause that organism to be able to adapt to an environment in a more successful manner than members of their species will in turn benefit them and enable them to live to pass the mutation along to their children. Then the mutation is not seen as a mutation anymore, but an adaptation. Scientist trying to take on the task of changing a genetic mutation adaptation would equate to them, in a sense, trying to revert a physical adaptation; something that has never been proven among any scientists. Therefore, upon reviewing the structure of both a healthy erythrocyte and a sickle erythrocyte, a conclusion drawn is that taking an approach in which a scientist attempts to correct a genetic mutation will most likely result without success.