The type of anaemia biology essay

Science, Biology

Sickle cell anaemia is inherited in an autosomal recessive disorder, which means both copies of the gene in cell do have the mutations. [3, 4] The allele that responsible for this autosomal recessive can be found on the short arm of the chromosome 11. It needs the presence of two defective genes (SS) for this disorder. [3] When each parent carries one sickle hemoglobin gene (S) one normal gene (A) with heterozygous genotype (HbAS) and, each child will has a 25% chance of inheriting two defective genes with homozygous sickle genotype (HbSS) and having a sickle cell anaemia, a 25% chance of inheriting two normal genes with a homozygous normal genotype (HbAA) and not having disease, and a 50% chance of being unaffected carrier like the parents. [3] This sickle cell anaemia follows Mendel’s law in the pattern of inheritance.[4] This genetic disorder inherited as simple recessive trait. [5]If individual who are heterozygote at a particular locus have greater fitness than do both kind of homozygotes, they exhibit the heterozygote advantage. This heterozygote advantage is defined in term of genotype not phenotype. Thus whether heterozygote advantage represents stabilizing or directional selection depends on the relationship between the genotype and the phenotype. In homozygous individual, a certain recessive allele at that locus causes sickle cell disease. The red blood cell of people with sickle cell disease becomes distorted in shape which can lead to serious complication, including damage to kidney, heart and brain. However heterozygotes are protected against the most severe effects of malaria although they are not resistant to malaria infection). This protection is important in tropical region where malaria is a major killer. In such region, selections favor heterozygotes over homozygous dominant individuals, who are more susceptible to

malaria, and also, and also over homozygous recessive individuals, who develop sickle cell disease. Sickle cell anaemia is an inherited disorder. The sickling occurs because of a mutation in the hemoglobin gene. The molecular basis of sickle cell disease is a point mutation. Point mutation can be divided to two general categories that is substitution and base pair insertion or deletion. But sickle cell anaemia is only cause by the substitutions. A base-pair substitution is the replacement of one nucleotide and its partner with another pair of nucleotides. They divide to silent mutation, missense mutation and nonsense mutations. Sickle cell anaemia is fall under missense mutations because the substitutions only change one amino acid to another. Humans normally make several types of the oxygen-carrying protein hemoglobin. Haemoglobins are made of 3 components that are heme, alpha globin, and beta globin. Sickle hemoglobin is the result of a genetic change in the beta globin component of normal adult hemoglobin. The beta globin gene is located on chromosome 11. Mutation in the hemoglobin genes that alter the protein composition but not the number of expression is known as qualitative mutations. The missense mutation in β-globin gene causing sickle cell anemia is a single nucleotide substitution (A to T) in the codon for amino acid 6. The change converts a glutamic acid codon (GAG) to a valine codon (GTG). The form of hemoglobin in persons with sickle cell anemia is referred to as HbS. The nomenclature for normal adult hemoglobin protein is HbA. In sickle cell anaemia, the problem is that the valine that to code the glutamic acid substitution results in hemoglobin tetramers that aggregate into arrays upon deoxygenation in the tissues. As the result , the sickle hemoglobin had a unique properties. In sickle cell anaemia, the havoc caused by the

abnormal hemoglobin, hemoglobin S (HbS), results from a change in just one of the 146 amino acids in a beta chain of the globin molecule. This alteration causes the beta chains to link together under low-oxygen conditions, forming stiff rods so that haemoglobin S become spiky and sharp. This in turn, causes the red blood cells to become crescent shaped when they unload oxygen molecules or when the oxygen content of the blood is lower than normal, as during vigorous exercise and other activities that increase metabolic rate. The stiff, deformed erythrocytes rupture easily and tend to dam up in small blood vessels. These events interfere with oxygen delivery, leaving the victims gasping for air and in extreme pain. Bone and chest are particularly severe, and infection and stroke are common sequels. Typically, the disease results in premature death. Because the genetic disorder is incompletely recessive, a person with only one SCA allele and one unaffected allele will have a " mixed" phenotype: The sufferer will not experience the ill effects of the disease, yet will still possess a sickle cell trait, whereby some of the red blood cells undergo benign effects of SCA, but nothing severe enough to be harmful. Those afflicted with sickle-cell trait are also known as carriers: If two carriers have a child, there is a 25% chance their child will have SCA, a 50% chance their child will be a carrier, and a 25% chance that the child will neither have SCA nor be a carrier. Were the presence of the SCA allele to confer only negative traits, its allele frequency would be expected to decrease generation after generation, until its presence were completely eliminated by selection and by chanceThe optimal care for patients with sickle cell disease (SCD), including preventive care, is best achieved through treatment in clinics that specialize in the care of SCD. During an acute crisis,

comfort measures, use of analgesics, patients regularly take over-the-counter (OTC) pain-relieving medications such as ibuprofen; during a crisis, stronger opioid analgesics, such as morphine or hydromorphone, are prescribed. In addition to opioids, ketorolac is effective in reducing pain stimulus and inflammation. As the children with SCD are placed on antibiotics as infants to prevent pneumonia. A cell-cycle-phase antineoplastic drug that is hydroxyurea that inhibits DNA synthesis, this medication reduces the frequency of painful crises. However, it can produce bone marrow suppression and worsen anemia. Nonpharmacologic measures include heat applications and limiting movement of the painful extremity. It's essential that the child be included in the pain management plan to ensure a sense of control and prevent psychological trauma associated with pain syndromes. Blood transfusions are often needed to treat anemia. A sudden worsening of anemia because of infection or spleen enlargement is an indicator for a blood transfusion. Blood transfusions improve tissue oxygenation and reduce sickling. However, frequent blood transfusions can lead to iron toxicity. Other treatment are like gene threrapy. Researchers are exploring the possibility of inserting a normal gene into the bone marrow of children with SCD to promote the production of normal hemoglobin. By using butyric acid as food additive can increases the amount of fetal hemoglobin in the blood. Allogeneic bone marrow transplantation (BMT) can cure SCD, but it is difficult to decide which patients should be offered BMT. Many risks are associated with BMT, and the risk-to-benefit ratio must be assessed carefully. With the advent of cord blood stem cell transplantation and with the development of less immunoablative conditioning regimens, perhaps BMT will gain wider

acceptance and use. The lack of availability of a matched donor may limit the utility of BMT. There are many more such clotrimazole and decitidine as the medicine, nicosan one of the herb use by Nigeria and also using the nitric acid gas. If patients with SCD crisis are being transported by emergency medical services (EMS), they should receive supplemental oxygen and intravenous hydration en route to the hospital. Lentivirus (lente-, Latin for " slow") is a genus of viruses of the Retroviridae family, characterized by a long incubation period. Lentiviruses can deliver a significant amount of viral RNA into the DNA of the host cell and have the unique ability among retroviruses of being able to infect non-dividing cells, so they are one of the most efficient methods of a gene delivery vector. HIV, SIV, and FIV are all examples of lentiviruses. Lentiviral gene vectors derive their high gene transfer efficiency and ability to modify non-dividing cells from their relationship to native lentiviruses such as HIV-1. For use in gene therapy, they are extensively modified to eliminate pathogenic components and are rendered incapable of self-replication. However, they retain their superior capability for gene transfer and thus represent a particularly effective platform technology for delivery of therapeutic genes potentially useful for the treatment of a wide variety of human diseases. Lentiviral gene vector transfers an anti-sickling variant of the faulty hemoglobin gene to the bone marrow, where it incorporates itself with unusually high efficiency into the stem cells that give rise to all red blood cells