Malaria effects an estimated 200 million people yearly. Of those infected by this mosquito borne parasite, 500,000 die annually. Most of the cases of Malaria are in Africa and the majority of those who die from it are children. Sickle cell anemia, a blood hereditary disorder, in some cases is a surprising advantage to those who may contract the Malaria parasite. Sickle cell anemia, can either be potentially fatal or beneficial. A person inherits two hemoglobin genes. They can both be normal, both be mutated or one be mutated and the other be normal. Hemoglobin is found in red blood cells and it allows them to carry oxygen. In the case of two normal hemoglobin genes the person does not inherit sickle cell disease. When two mutated genes are inherited the person inherits sickle cell disease. During periods of strenuous activity red blood cell will loss oxygen and change shapes from oval to sickle shaped, which become stuck in small vessels. This can cause tissue damage, which can be potentially lethal. A person who has one normal and one mutated hemoglobin gene has an advantage when the malaria parasite enters there blood. Their cells will turn sickle when infected with malaria and the spleen will eliminate the infected cells. Despite this resistance to malaria and the fact that it is preventable and curable many die from it each year. However, many lives are saved through research leading to treatment innovations and the efforts of the CDC. Just like we have evolved a resistance to malaria, through mutations in the hemoglobin gene, Malaria has done the same. The bacterium that causes Malaria, Plasmodium *falciparum,* which is transmitted through the bite of mosquito*,* has evolved to resist Malaria drugs. Drug resistant malaria strains have recently emerged in Southeast Asia and South America and are expected to spread in venerable regions. When the drug resistant Malaria parasite, known as the mutator phenotype, is genetically compared to the phenotype that does not confer drug resistance, researches can see that there is a difference in regulation of DNA repair. The mutator phenotype that has an accelerated resistance to multiply drugs (ARMD), has defective DNA repair machinery. The ARMD phenotype has mutations in proteins, involved in drug transport and drug targets. It also has mutations in the DNA repair genes. This phenotype appears to have an increased ability to tolerate mutations. Normally, when the phenotype without mutations experiences conditions that harm the DNA, such as exposure to methyl methanesulphonate, DNA packaging is modified and the genes that make DNA repair possible are expressed. The modification in DNA packaging allows DNA repair factors access to the damaged DNA. Without DNA repair mechanisms the ARMD phenotype experiences mutations. The ARMD phenotype has several DNA sequences of DNA repair genes, where mutations have occurred. When testing the two phenotypes using a DNA damaging agent, methyl methanesulphonate or other anti-malaria drugs, major differences in gene regulation were observed. For example, genes that regulate DNA repair mechanisms in the phenotype with mutations were expressed, but not in the ARMD phenotype. Furthermore, the ARMD phenotype had four DNA repair genes mutated in the strains shown to be resistant to artemisinin, an anti-malaria drug. There were also mutations of genes that sensed DNA damage in this phenotype. Both phenotypes experienced modification in DNA packaging when genes were damaged. Sometime mutations can be harmful, but other times they can be beneficial. Mutations in the hemoglobin genes allowed humans to become resistant to malaria. While mutations in the genes associated with DNA repair in Plasmodium *falciparum*, allowed resistance to malaria drugs. It is important that researchers better understand the ARMD phenotype so that they may develop a new drug or treatment can overcome the rapid rate of mutation allowing the drug resistance. Malaria already takes the life of so many people and with these discovered mechanisms it will only take more.

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