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Commentary

Brief Note on Causes of Sickle Cell Anemia

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DESCRIPTION

Sickle cell anemia is a genetic disease where red blood cells expose a sickle shape when seen under an ordinary microscope. It is a genetic disorder, the very first to be linked to a specific genetic mutation. A single base change in the DNA sequence of the gene producing the beta chain of hemoglobin, the protein that carries oxygen in red blood cells, was identified as the cause of sickle cell anemia.

Sickle cell anemia affects only people who inherit two copies of the sickle mutation (one from their mother and the other from their father). These people have a reduced life expectancy if they aren't treated; therefore it's not surprising that this mutation is rare in human populations. This is however, a long way from being the situation. Sickle mutation is highly chosen in people from places of the world where malaria is common, with up to 40% of the population bearing the mutation.

People with only one copy of the sickle mutation (inherited from either the father or the mother) have been reported to avoid developing sickle cell anemia and to live relatively normal lives. However, it was discovered that these same individuals, who were thought to have the sickle cell trait, were actually strongly protected against malaria, explaining the high occurrence of this mutation in malaria-endemic areas.

Symptoms of sickle cell anemia

- Jaundice
- Hands and feet swelling
- Recurring infections
- Chest, back, arm, or leg aches and pains
- Bedwetting due to kidney problems

These findings have led to widespread belief in the medical community that understanding the mechanism by which sickle cell trait protects against malaria would provide crucial insight into developing treatment or a possible cure for this deadly disease. Several studies have suggested that sickle hemoglobin may obstruct the Plasmodium parasite from infecting red blood cells, hence lowering the amount of parasites that actually infect the host and offering some protection against the disease.

Researchers revealed that mice genetically altered to produce one copy of sickle hemoglobin, comparable to sickle cell trait, cannot succumb to cerebral malaria, replicating what happens in humans. Despite the presence of the parasite, researchers studied the brains of these mice and established that the abnormalities associated with the development of cerebral malaria were absent. In these mice, sickle hemoglobin provides protection without interfering with the parasite's capacity to infect the host red blood cells. Sickle hemoglobin makes the host tolerant to the parasite.

Researchers had the option to show that the major participant in this protective effect is heme oxygenase-1 (HO-1), an enzyme whose expression is highly stimulated by sickle hemoglobin, through a series of genetic tests. This enzyme, which creates the gas carbon monoxide, confers protection against cerebral malaria.

CONCLUSION

The mechanism they discovered for sickle cell trait could be a generic mechanism at work in other red blood cell hereditary illnesses that have been shown to protect humans from malaria. The sickle mutation may have been naturally chosen in tropical regions, where malaria is endemic and one of the leading causes of death, due to its protective impact against malaria. Other clinically silent mutations may have been selected for their capacity to provide a survival advantage against Plasmodium infection through the evolution.

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