Researchers identify new way the malaria parasite and red blood cells interact

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Virginia Commonwealth University Life Sciences researchers have discovered a new mechanism the malaria parasite uses to enter human red blood cells, which could lead to the development of a vaccine cocktail to fight the mosquito-borne disease.

Malaria is transmitted to humans through bites from mosquitoes. According to the Centers for Disease Control and Prevention, between 350 million and 500 million cases of malaria occur world-wide annually, and more than 1 million people, mostly children living in areas of Africa south of the Sahara, die each year from it.

For decades, researchers have known that a molecule called glycophorin B, which is found on the surface of human red blood cells, is important for invasion of the malaria parasite. However, the specific molecule by which the malaria parasite attaches itself to invade the host was not known until now.

The team examined how the malaria parasite, Plasmodium falciparum, interacts with red blood cells using a biochemical test that looks specifically at how the parasite and host bind to each other. The findings revealed that the EBL-1 molecule is the specific attachment site used by the parasite on glycophorin B.

The study was published online in the Early Edition of the *Proceedings of the National Academy of Sciences* the week of March 9.

"We have now identified how the parasite binds to glycophorin B on the red blood cells. Down the road, the EBL-1 molecule could be used as a vaccine target against malaria as part of a multivalent vaccine, or vaccine cocktail," said principal investigator Ghislaine Mayer, Ph.D., assistant professor in the VCU Department of Biology.

Additionally, Mayer and her team hypothesize that the malaria parasite may be the cause of the loss of the gene for glycophorin B in the pygmies of Ituri forest in the Democratic Republic of Congo.

According to Mayer, these findings suggest that the parasite may possibly be putting selective pressure on populations in malaria-endemic areas, such as the Democratic Republic of Congo. She said that there appears to be a disproportionate number of individuals in malaria-endemic areas with unusual or mutated red blood cell surface molecules.

"We think these changes on the surface of the red blood cell may lead to a decrease in the severity of malaria or resistance against malaria. For example, Africans are protected from a form of malaria caused by the *Plasmodium vivax* parasite because the molecule that the parasite recognizes is missing from the surface of their red blood cells because of a mutation," said Mayer.

Source: Virginia Commonwealth University