

Researchers unveil new approach to blocking malaria transmission

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University of Illinois at Chicago researcher Dr. John Quigley will describe a promising new approach to blocking malaria transmission during the American Society of Hematology's annual meeting in Orlando, Fla.

Quigley spoke at a press briefing Saturday, Dec. 4. His abstract, "Anopheline Orthologs of the Human Erythroid Heme Exporter, FLVCR, Export Heme: Potential Targets to Inhibit Plasmodium Transmission," will be presented at the plenary session Sunday.

The research focuses on potential targets to inhibit transmission of the [parasite Plasmodium](#) that causes malaria.

[Female mosquitoes](#) ingest large amounts of hemoglobin that serves as a food source required for mosquito egg development. When a mosquito ingests infected blood, Plasmodium reproduces in the mosquito gut. Plasmodium fertilized egg cells cross the lining of the mosquito gut and develop into oocysts. After maturing, the oocysts rupture and release thousands of parasites that allow the mosquito to transmit malaria when it bites another human.

Previous studies have shown that mosquitoes with increased oxidative stress in their midgut are resistant to Plasmodium transmission. Quigley and his colleagues hypothesize that if they can disrupt the function of a cell-surface transport protein called FLVCR that pumps heme out of the cell, it will increase the oxidative stress in the mosquito gut and hamper

Plasmodium at a crucial point in the parasite's life cycle.

The researchers isolated the FLVCR gene from two common malaria-transmitting [mosquitoes](#) and showed that the gene encodes a protein that exports heme and protects cells from oxidative stress. Using gene-silencing techniques, they were able to significantly reduce levels of FLVCR in the mosquito gut.

"If disruption of the function of the protein inhibits parasite transmission, then we can potentially use parts of the protein as an antigen to try to stimulate a vaccine in people," said Quigley, who is assistant professor of medicine at the UIC College of Medicine and senior author of the study. "So the antibody blocks FLVCR and increases [oxidative stress](#), and now the Plasmodium is not able to complete its life cycle, thus preventing the spread of malaria."

Quigley's research is ongoing, and future studies will focus on whether inhibiting FLVCR can block Plasmodium transmission. The research, he says, may be applicable to all blood-eating insects that cause a variety of diseases, such as West Nile Virus, dengue fever and leishmaniasis.

Provided by University of Illinois at Chicago

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