New papers offer insights into process of malarial drug resistance

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Malaria, one of the oldest diseases known to man, has shown no signs of slowing down as it ages. More than 1 million children die from malaria in sub-Saharan Africa each year, and in areas along the Thailand/Cambodian border multiple drug-resistant strains of the disease are becoming commonplace.

With the previously mainstay antimalarial drug chloroquine nearly ineffective due to drug resistance and traditional public health approaches such as mosquito netting offering uneven results, two new papers by University of Notre Dame biologist Michael Ferdig suggest that the means of combating this old foe may lie in the new tools of genomics and bioinformatics.

In the papers, Ferdig points out that development of the malaria parasite Plasmodium falciparum in the blood is driven by a number of different genes expressed at different times and at different levels. Exactly what influences such transcriptional changes remains elusive, particularly in regard to important phenotypes like drug resistance.

Ferdig and his collaborators combined classical genetics with cutting-edge genomic methods to illuminate previously unrecognized transcriptional complexity and variation in Plasmodium falciparum and possibly master regulators within large copy number variants that contribute to the drug-resistant phenomena in malaria parasites.

By uncovering the genetic "architecture" of numerous drug responses

and identifying key regulators that control these responses, Ferdig hopes to map new approaches to conquering drug resistant malarial genes.

One paper from the Ferdig lab appeared in the journal PLoS Biology. The second, in collaboration with Tim Anderson at Southwest Biomedical Research Foundation, appeared in *PLoS Genetics*.

Source: University of Notre Dame