Silencing mosquito gene could slow spread of disease

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A. aegypti mosquitos play a leading role in spreading diseases such as Zika and dengue viruses. (Photo from the Centers for Disease Control)

Next time you slap a mosquito consider this - Tulane researchers are testing a strategy to alter the genes of female *Aedes aegypti* mosquitos so they die soon after a blood meal. *A. aegypti* mosquitos play a leading role in spreading diseases such as Zika and dengue viruses. The results of this research were published in the June 2017 issue of *The FASEB Journal*, the journal of the Federation of American Societies for Experimental Biology.

The research team, led by Patricia Y. Scaraffia, assistant professor in the Department of Tropical Medicine and member of the Vector-Borne Infectious Disease Center at the School of Public Health and Tropical Medicine at Tulane University, found that they increased mosquito deaths and decreased egg laying by altering the way female mosquitoes use a crucial protein called xanthine dehydrogenase 1 (XDH1).

“XDH1 plays an essential role in blood-fed *Aedes aegypti* mosquitoes and that silencing of XDH1 gene promotes a blood feeding-induced adulticidal activity,” explains Scaraffia. By blocking this gene, the female mosquito can’t produce uric acid after a blood meal, which means the mosquito’s body can’t remove waste. As a result, mosquitos lay fewer eggs and die early.
Tulane University

“This novel finding can help researchers to design metabolism-based strategies to control populations of *A. aegypti* mosquitoes, vectors of diseases of public health significance,” says Scaraffia, adding that the team’s research has shown that either reducing the amount of XDH1 or blocking it entirely are effective ways to target mosquitos.

Co-authors include Jun Isoe, research scientist at the University of Arizona; Tulane post-doctoral fellow Natthida Petchampai; University of Arizona undergraduate Yurika E. Isoe; Tulane laboratory research scientist Katrina Co; and Stacy Mazzalupo, assistant scientific investigator at The University of Arizona. Financial support came from the Corine Adams Baines Professorship Award and a grant from the U.S. National Institutes of Health, National Institute of Allergy and Infectious Diseases Grant (NIH/NIAID).