Future of Malaria Research Symposium

Monday, November 18, 2019

Johns Hopkins Malaria Research Institute

Call for Abstracts

Instructions & Format

The submission deadline for poster presentation and/or short talk consideration is September 18, 2019.
Please use the below format and email submissions to **malaria-conference@jhu.edu**

Please provide **title, authors, institution**, and a **brief abstract** of your research (300 words maximum).
Audience: Both researchers and lay persons.

Font: Times New Roman, Type size: 12pt, Format: MS Word
Title: Bold, capitalizing first word and proper nouns, as well as italicizing when appropriate
Authors: Please use full names, (first name last name), and use numeric symbols to designate institutions
Institution(s): Please order alphabetically and designate with numeric symbols

EXAMPLE

Characterizing the *Anopheles gambiae* late-phase immune responses that limit *Plasmodium* oocyst survival

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Mosquitoes of the genus *Anopheles* serve as the obligate vectors of the malaria parasite *Plasmodium*. During its development within the mosquito host, several factors and developmental bottlenecks limit parasite success, including two distinct phases of the mosquito innate immune response. Emerging evidence suggests that parasite numbers are largely influenced by an "early-phase" that targets ookinetes as they reach the basal lamina of the mosquito midgut through the exposure to complement-like components of the hemolymph and a "late-phase" response that limits oocyst survival through the production of nitric oxide mediated by the STAT pathway. Recently, we have identified a novel LITAF-like transcription factor (LL3) in *Anopheles gambiae* that is an important component of the mosquito response to *Plasmodium*. Following LL3-silencing, oocyst numbers are significantly increased and preliminary evidence suggests that LL3 is involved in the "late-phase" response by limiting oocyst survival. Experiments to determine the relationship between the "late-phase" response for LL3 and those previously described for the STAT pathway imply that the LL3 and STAT phenotypes occur through independent, yet closely related mechanisms involving hemocyte function. Current experiments aim to further dissect LL3 function to address a critical gap in our knowledge of the mosquito late-phase immune response and the mechanisms that limit oocyst survival in its mosquito host.

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