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Seeking *P. falciparum* sequestration strategies: Getting warmer with each bond

Lim Ying Bena
National University of Singapore, Singapore

Plasmodium (P.) falciparum infected red blood cells (iRBCs) adhere to host endothelial cells in their bid to avoid getting cleared by the spleen. Sequestration, or accumulation, of iRBCs occlude microvasculature and ultimately lead to multiple organ failure and cerebral malaria. The upregulation of CD36 and ICAM-1, two of the several endothelial receptors that iRBCs bind to, have been associated with severe disease pathology. Unlike CD36, ICAM-1 facilitates the rolling of iRBCs on endothelium and is also the only receptor known to correlate with cerebral malaria. Single bond force spectroscopy was conducted to probe the basic interactions of iRBC/ICAM-1 and iRBC/CD36 bonds. We found that iRBCs form catch bonds with ICAM-1 and slip bonds with CD36. These findings explain why ICAM-1 mediates iRBC rolling and sheds light on the possible reason why iRBC/ICAM-1 interactions are not affected by high amounts of soluble ICAM-1 in the brains of cerebral malaria patients. Since *P. falciparum* patients experience 48 hour febrile cycles, the next step is to investigate how sensitive these iRBC-receptor interactions are to temperature. We subsequently conducted single bond force spectroscopy for both iRBC/ICAM-1 and iRBC/CD36 at body temperature (37°C) and febrile temperature (41°C). Comparing the differences in behavior at room, body and febrile temperatures, we were able to gain deeper and more physiologically relevant insights on iRBC- receptor interactions at a fundamental level. Just by looking at single molecule interactions, we are now getting warmer in seeking the various biophysical strategies employed by the parasite in sequestration.

benalim@u.nus.edu