Australian National University

Malaria goes bananas before sex

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Slatyer Seminar Room, R N Robertson Building (#46)

Survival of the human malaria parasite *Plasmodium falciparum* in the circulation of the host relies on its ability to drastically alter its host red blood cell (RBC) through the large-scale export of host cell modifying proteins. This remodelling allows asexual trophozoites to sequester away from the circulation and thus avoid clearance in the spleen. Sexual stage gametocytes also adhere during early development stages but re-enter the blood stream as crescent-shaped mature stage gametocytes and can be taken up by mosquitoes. Development of this specialized form of the parasite relies on the systematic expression, trafficking and assembly of a series of multi component protein complexes. These complexes include an elaborated basket of microtubules with proteinaceous cross-links, as well as actin structural elements and myosin-based anchoring elements. The complexes are assembled together with an ER-derived system of flattened cisternal membrane compartments underneath the parasite plasma membrane. In this work we define the processes in the orchestrated assembly of these structures and complexes and show that the cellular elongation is likely need by the parasite to survive in the circulation of the host. The elongation process changes the deformability properties of the parasite allowing it to avoid mechanical clearance within the spleen; this will facilitate disease transmission by making the circulating gametocytes available to feeding mosquitos.

Matthew Dixon is a NHMRC Early Career Fellow and an associate member of the ARC Centre of Excellence for Coherent X-ray Science. In 2010 he was the recipient of a Young Investigator Award at the Lorne Conference on Protein Structure and Function. He has also been awarded a Ramaciotti Foundation start-up grant and an ANZ Trustees Equipment Grant.

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