Actomyosin motors and malaria parasite invasion of the host cell

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This thesis is submitted in part fulfilment of the requirements of University College London for the degree of Doctor of Philosophy

June 2013

Abstract

In the blood stream the merozoite is the invasive form of the malaria parasite that must enter a new erythrocyte. Merozoites lack structure for locomotion such as cilia and flagella, but use an actomyosin motor complex that provides the force to drive them into the cell. This molecular motor is located at the pellicle membrane of the parasite. The motor protein complex or glideosome includes a class XIV myosin heavy chain (MyoA), Myosin Tail Interacting Protein (MTIP) and two anchoring proteins known as Glideosome Associated Protein 45 (GAP45) and GAP50. A ternary complex of MyoA-MTIP-GAP45 is first formed and this later associates with GAP50. Using a GFP-tagged MyoA transgenic parasite line the expression of MyoA was examined in both *Plasmodium falciparum* and *P. knowlesi*. The protein was expressed at the periphery of the parasite and associates with MTIP, GAP45 and GAP50. In time course studies it was shown that recruitment of GAP50 to the complex occurs late relative to the onset of GAP50 synthesis, likely reflecting a role for GAP50 in cellular processes prior to host cell invasion. A second class XIV myosin, Myosin B (MyoB) was also tagged with GFP in a transgenic parasite line. Whilst the timing of MyoB expression followed that of MyoA, with clearly detectable protein in segmenting schizonts at 40 hours post invasion, MyoB did not associates with MyoA or any of the other invasion complex proteins. The localisation of MyoB-GFP in *P. falciparum* was at the apical end of the merozoite, in front of other apical organelle markers such as Rhoptry neck protein 4 (RON4). During erythrocyte invasion, MyoB -GFP remained in the same location, whilst RON4 moved out onto the parasite surface as part of the moving junction and migrated to the rear of the parasite. Although the exact role of MyoB is unknown, it is suggested that it may be involved in the invasion process but within its own protein complex that is distinct from the glideosome.