Dissecting Invasion In Plasmodium Falciparum: Reticulocyte Binding Protein Homologues Regulate Key Steps

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Abstract
Malaria disease severity and Plasmodium falciparum pathogenesis depends on the efficiency of erythrocyte invasion by merozoites. Invasion by merozoite is a multi-step process involving multiple ligand-receptor interactions. Two gene families, Reticulocyte binding like protein Homologues (RH) and Erythrocyte Binding like Ligands (EBLs) have been shown to play critical roles during this process. The differential expression of members of RHs and EBLs defines the invasion pathways utilized by the merozoites and serves as a mechanism in evading immune attack. P.falciparum RHs are located in the rhoptry of merozoite and are secreted during the invasion process to allow specific interactions with erythrocytes surface receptors. We have recently shown that the interaction of PfRH1 with its erythrocyte receptor triggers a key Ca++ signal in the merozoite that leads to the subsequent discharge of micronemal proteins and junction formation. Using a range of monoclonal antibodies we have now been able to show that another members of the RH family, RH2b mediates a similar function to RH1 during invasion. In contrast RH5 a unique member of the RH family that has been shown to be essential for parasite survival plays a distinct role during invasion and appears to trigger a crucial signal in the host cell that is required for successful invasion to occur. Overall, the data provides new insights on the different invasion steps that are regulated by RH.

Selected publications

