Erythrocyte membrane remodelling and destruction by malaria parasites

Helen Saibil
Institute of Structural and Molecular Biology, Birkbeck College London

In the clinical phase of malaria infection, the parasite cells (merozoites) invade their host erythrocytes and create an intracellular vacuole, inside which they replicate. In addition, they modify the erythrocyte membrane to create sites of adhesion to endothelia, the cause of severe clinical disease in \textit{P. falciparum} cases. When the daughter parasites are mature, after about 48 h, they need to break through both vacuole and erythrocyte membranes in order to invade new erythrocytes. The process by which they escape (“egress”) is a highly ordered sequence of secretion, activation and proteolytic events, culminating in explosive release of the new parasites for the next round of infection.

We have used video microscopy, electron and X-ray tomography along with mutants and pharmacological blockers of different steps in egress, to study membrane disruption and breakage during the process of egress. This work has revealed new steps in egress and an unexpected role for the major merozoite surface complex MSP1.

