Australian National University

PUBLIC SEMINAR

P. falciparum induces the formation of novel chaperone/co-chaperone complexes in the infected erythrocyte

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Slatyer Seminar Room RN Robertson Building (46), ANU

Following invasion of the human erythrocyte, *Plasmodium falciparum* parasites traffic a large number of proteins to the cytosol and membrane of the host cell. Although the function of many of these proteins is still not fully understood, it appears that these parasite proteins are involved in remodelling of the host cell, including both biochemical and structural alterations of the erythrocyte.

To carry out these functions, these proteins must be transported to, and through, the cytosol of a denucleated eukaryotic cell (the erythrocyte) which is, to our knowledge, lacking most of the cellular machinery normally required for protein transport.

Amongst the "exportome" of the parasite are a large number of proteins related to both chaperones, and co-chaperones. These molecules are, in other systems, involved in protein folding, degradation, and trafficking processes. Thus, our working hypothesis that these parasite proteins are exported to the host cell as part of an "extracellular" parasite-derived secretory system.

We have previously shown that parasite-encoded co-chaperone proteins are trafficked to the host cell, where they reside in specialised structures referred to as J-dots. Here we present the results of our recent studies. Expression of a dominant negative version of one specific exported co-chaperone protein resulted in a much reduced parasite growth rate, this phenotype could be reversed by co-expression of the wild-type protein. Transport of various marker proteins was inhibited in the dominant negative expressing parasite line, suggesting a role for this co-chaperone in protein export processes. Additionally, we have identified further components of the J-dots, which support our hypothesis that these structures contain a functional chaperone/co-chaperone complex.

Taken together, our data suggest that chaperone complexes induced by the parasite play a vital role in both host cell modification and parasite survival, probably due to their involvement in protein transport processes.

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