Molecular analysis of malaria parasite and host cell responses to co-adhesion interactions

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POSTER ABSTRACT

The interaction between *Plasmodium falciparum* infected erythrocytes and endothelial and cells is thought to play a key role in the pathogenesis of cerebral malaria (CM). These interactions between different repertoires of receptors/ ligands are thought to mediate downstream effects on both the host and the parasite. These can influence protection and susceptibility to disease. The purpose of the present study was to understand how the malaria parasite can alter the behavior of human brain microvascular endothelial cells (HBMEC) responses via co-adhesion interactions.

To investigate this phenomenon, Illumina next generation sequencing was used to profile the transcriptional changes of HBMEC in response to A4 parasite isolate in the presence of tumor necrosis factor (TNF) at 0h and 6h. The study identified 88 genes differentially expressed; of them, 15 upregulated genes and 73 downregulated genes. The gene functional annotation analysis illustrated that adhesion of the malaria parasite with HBMEC induced the expression of genes involved in inflammation and apoptosis, such as PLA2G4A. However, it reduced expression of other genes involved in NOTCH signalling, for example HES1. The expression of selected genes was validated by RT-qPCR.

Overall, the outcomes from the study facilitate a greater understanding about changes in host responses after cytoadherence with the malaria parasite, identifying pathways with potential pathogenic or protective roles.