

Transcriptome analysis of malaria parasite and endothelial cell responses to co-adhesion interactions

Basim Othman^{1,2}, Simon Wagstaff², Arnab Pain³ and Alister Craig².

¹ Department of Public Health, Faculty of Applied Medical Sciences, Al-Baha University, Al-Baha, Saudi Arabia Basim.Othman@lstmed.ac.uk.

² Liverpool School of Tropical Medicine, Liverpool L3 5QA, United Kingdom

³ Biological and Environmental Sciences and Engineering Division, King Abdullah University of Science and Technology, Thuwal, 23955-6900, Jeddah, Kingdom of Saudi Arabia

POSTER ABSTRACT

The interaction between *Plasmodium falciparum* infected erythrocytes and endothelial cells is thought to play a key role in the pathogenesis of cerebral malaria (CM). This interaction between different repertoires of receptors/ ligands could mediate downstream effects on both the host and the parasite and can also 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 microvascular endothelial cells responses via co-adhesion interactions.

To investigate this phenomenon, Illumina next generation sequencing was used to profile the transcriptional changes of HBMEC and HDMEC in response to IT4var14 parasite isolate in the presence of tumor necrosis factor cytokine (TNF) at 6h and 20h. The most highly significant gene of interacting IT4var14 parasite with HBMEC at 6h was C3, whereas; the ID2 gene was the lowly expressed gene. At 20h of interaction malaria parasite with brain cells, PRND was highly up-regulated gene, however; NOG gene was the most down-regulated gene. The gene functional annotation analysis illustrated that adhesion of the malaria parasite with HBMEC at 6h 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. Adhesion of malaria parasite with brain cells at 20h led to reduction in cells proliferation.

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.