Computational analysis of Plasmodium falciparum RNA-Seq data reveals PPIs that might be implicated in the invasion of the RBCs

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Abstract:
In this study, differentially expressed genes for the trophozoite and schizont stages of Plasmodium falciparum's life cycle were extracted from a time series RNA-Seq gene expression experiment. About 28% of the 5,270 genes used in the experiment were found to show significant expression at these stages. Enrichment analysis using Gene Ontology implicated a total of 62 functions as highly enriched from the list of differentially expressed genes (DEGs). Some include; protein targeting to membrane, protein import, establishment of proteins localization to organelle, ribonucleic protein complex, nucleotide-excision repair and processes related to the mitochondria. A protein interaction network (PIN) for the DEGs at the schizont stage was extracted from experimental data of protein-protein interactions and supplemented with data from a protein interaction database. We predicted a number of protein-protein interactions in Plasmodium falciparum that may be implicated in invasion of the human red blood cells (RBCs). Some of these predictions are consistent with those from previous studies while quite a number of them are novel. We also identified 16 protein complexes from the PIN using the Molecular Complex Detection (MCODE) algorithm. The functional enrichment of the identified protein complexes showed functions related to gene expression, translation, RNA transport and metabolic/biological processes which have been

identified to be important in the invasion process. The result from this study is meant to provide better insight into disease at hand.

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I. Introduction

The invasion of the erythrocytes is critical to the survival and pathogenesis of the popular malaria parasite, Plasmodium falciparum (P.f.). The preliminary interaction between the merozoite and the red blood cell is a vital key since the parasite must separate between erythrocytes competent for invasion and other cell types [1], [2]. Therefore, a tight junction must be established between the parasite and the host red cell membrane before the parasite can gain entrance into the red blood cells. This tight junction moves from the apical to the posterior end of the merozoite in a complex turn of activities powered by the parasite actin-myosin motor [3], [4]. A structure called the parasitophorous vacuole is created when the parasite makes its way into the host cell and encloses itself against the cytoplasm of the host cell to create a hospitable environment for its development. Invasion by the parasite thus causes vast changes in the host RBCs particularly, the increase in the rigidity of the membrane which assists the parasite to survive within the cell of its host contributing to an increase in disease malignity [5]

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