Article Title

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Abstract
There's nothing here yet

Contents

1. Introduction

The apicomplexan parasite *Plasmodium falciparum* is the most virulent causative agent of malaria, and responsible for over 600,000 deaths annually [1]. Along with other members of the *Plasmodium* family, *P. falciparum* has a complex lifecycle, moving between several different tissues in both mammalian and arthropod hosts. Symptomatic disease in humans occurs when *P. falciparum* undergoes rounds of asexual reproduction inside human red blood cells (RBCs) [2].

In some respects, the intracellular environment of a red blood cell is an ideal location for parasite proliferation. The cells' lack of an MHC (Major Histocompatibility Complex) system, which would otherwise be used to identify intracellular pathogens to the host immune system, renders parasites immunologically invisible [3], whilst the vascular system allows the parasite to travel throughout the body. The highly specialised nature of RBCs, however, means that the intracellular environment also presents significant challenges to parasite survival.

Mature red blood cells lack protein production and export machinery, and are a nutritionally poor environment, with a proteome dominated by haemoglobin, which typically accounts for around 98% of the protein content of the cell [4]. *P. falciparum* is able to digest RBC proteins, however haemoglobin lacks several amino acids required for protein production. Red blood cells are also subject to regular 'quality control' in the spleen, where damaged or infected cells are killed and recycled [5].

In order to survive and proliferate inside RBCs, *P. falciparum* exports a range of proteins which radically transform the red blood cell, collectively termed the **exportome**. Many of these proteins are involved in setting up a parasite-derived protein export system, capable of directing exported *P. falciparum* proteins to sites both inside and outside the RBC. *P. falciparum* resides inside a parasitophorous vacuole, and exports proteins via structures termed Maurer's Clefts [6], which bear some similarity to golgi apparatus [7].

In order to avoid detection in the spleen, many exported proteins are associated with the formation of knobs, specialised structures which form at the RBC membrane and promote cytoadherence to epithelial cells, platelets and other red blood cells [8]. Severe forms of malaria, including cerebral malaria, are believed to be caused by sequestration of infected red blood cells in deep tissues, as well as overinduction of inflammatory cytokines [2]. Other exported components have been associated with increasing RBC membrane permeability to facilitate nutrient and waste exchange and strengthening the RBC cytoskeleton (Reviewed in [5]).

Recent estimates suggest that at least 10% of the *P. falciparum* genome is exported [9]

The Plasmodium Export Element (PEXEL) Motif

PEXEL Negative Exported Proteins (PNEPs)

1.1 Protein Structure Prediction

Disorder Prediction (metaPrDOS

Coiled Coil Prediction (Coils)

Transmembrane Prediction (TMHMM)

Combined Approaches (Phyre2 and InterPro)

2. Software Implementation

2.1 Automating Sequence Submission

2.2 Collating output formats

2.3 Visualisation

3. Limitations and Further Work

Acknowledgments

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