

Article Title

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Abstract

There's nothing here yet

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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, red blood cells represent an ideal location for parasite proliferation. The vascular system facilitates movement of the parasite to diverse locations throughout the body, and the cells' lack of an MHC (Major Histocompatibility Complex) system renders intracellular parasites immunologically invisible [?]. However, the specialised nature of the RBC also means that cells are not immediately suitable as a host and lack organelles required for protein export. Infected cells are regularly detected and killed in the spleen.

1.1 The *Plasmodium falciparum* Exportome

In order to transform the RBC into a suitable environment for proliferation, *P. falciparum* exports a range of proteins collectively termed the **exportome**.

The PEXEL Motif
PNEPs

1.2 Protein Structure Prediction

Disorder Prediction (metaPrDOS)
Coiled Coil Prediction (Coils)
Transmembrane Prediction (TMHMM)
Combined Approaches (Phyre2 and InterPro)

2. Implementation

2.1 Automating Sequence Submission

2.2 Collating output formats

2.3 Visualisation

3. Limitations and Further Work

Acknowledgments

References

- [1] World Health Organisation. *World Malaria Report 2013*. World Health Organization, 2013.
- [2] Qijun Chen, Martha Schlichtherle, and Mats Wahlgren. Molecular Aspects of Severe Malaria. *Clinical Microbiology Reviews*, 13(3):439–450, July 2000.