

Research Theme:
Research Project Title: The plasmodium parasite exported protein interactome inside the host erythrocyte
Principal Investigator/Supervisor: Prof. Peter Preiser
Co-supervisor/ Collaborator(s) (if any):
Project Description
<p><i>Plasmodium</i> have a complex life cycle and all the symptoms of malaria are caused when they develop within the human erythrocytes. The parasite must refurbish its host-cell in order to stabilize the host-cell cytoskeleton, to acquire nutrients and to ultimately promote virulence. For this, <i>Plasmodium</i> export proteins in the host-cell cytosol which form an interaction network involving parasite and host proteins. Using export motif identified in silico, increasing data provided partial information on the identity of the malaria proteins exported in the host and highlighted that exported proteins are mostly unique to <i>Plasmodium</i> with no further functional annotation. To date, only a few exported proteins have been characterized and were proposed to be involved in functions that promote parasite virulence. Importantly, knock-out experiments reported that these proteins are mostly not essential for parasite development in vitro. The role of the remaining exported proteins is unknown but it is likely that a fraction of the exportome is involved in parasite survival via a complex network of functional interactions. Using proteomic approach, we have mapped the proteome of five <i>Plasmodium</i> species, revealing its exportome. This research project now aims at using these results, to identify 1) the subset of exported proteins conserved across the genus and essential for the parasite development, 2) the interaction networks of these proteins with parasite and host proteins and 3) identify the interactions essential for parasite survival. Reaching a fine understanding of the interactome in the host cell could provide new therapeutic avenues less prone to the resistance that has rendered current antimalarial treatment ineffective. Indeed, the blockage of essential interactions between Plasmodium and the host proteins with strategies that target the host protein could be the downfall of the parasite with a lower likelihood to develop resistance than approaches that would target parasite molecules.</p>
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