# *Plasmodium falciparum* proteins that interact with human erythrocyte spectrin

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Malaria is one of the most prevalent and lethal diseases affecting humans in Africa. The *Plasmodium* parasite spends the pathogenic stage of its life cycle in human erythrocytes, thereby making the interaction between erythrocyte and parasite proteins a key point of research. A *Plasmodium falciparum*-phage display library was screened against purified human erythrocyte spectrin. The *P.falciparum* cDNA inserts of interacting phage were compared to the PlasmoDB database and four interacting proteins were identified. Three of these are hypothetical and the fourth is a putative aminopeptidase. This aminopeptidase has a 30.7 % homology to a human aspartyl aminopeptidase which catalyses the release of N-terminal amino acids from a peptide. Twelve amino acids which are involved in the binding of two catalytic zinc ions in the active site are conserved in the *P.falciparum* aminopeptidase. The peptide fragment that bound to spectrin corresponds to a 33 amino acid fragment that is not found in the human aspartyl aminopeptidase. This suggests an evolutionary development of the parasite that allows the protease to bind to human spectrin. Microarray analysis from the PlasmoDB database shows the highest expression of the putative aminopeptidase in early and late trophozoite stages of the parasites erythrocytic life cycle. During this stage the parasite creates channels in the erythrocyte membrane for the transportation of nutrients and waste products. Therefore, the putative aminopeptidase could be responsible for the modification of the erythrocyte membrane during development of the trophozoite. It may also play a role in the release of schizonts from infected red cells.