Ubiquitin is a 76 amino acid post-translational modifier that acts as a key signal regulating endocytic trafficking in mammalian cells. Endocytic adaptor proteins, which contain ubiquitin-binding domains (UBDs) recognize monoubiquitinated proteins and mediate their internalisation into endosomes.

In *Plasmodium falciparum,* the homologues of human endocytic proteins localise to the parasite's cytostome (the site of endocytosis) but do not contain canonical UBDs. Some parasite-specific proteins containing potential UBDs also co-localise. Although the cytostomal localisation of these proteins suggests mechanistic conservation with human endocytosis, their ability to bind ubiquitin and biological functions have not yet been determined.

The endocytic pathway in *Plasmodium* is essential for haemoglobin internalisation during the intraerythrocytic stage of infection. Artemisinin (ART), a frontline antimalarial, interacts with intraparasitic haem for its own activation, and therefore the activity of the endocytic pathway is strongly associated with ART resistance. Inactivation of those *Plasmodium* cytostomal proteins have also been shown to reduce ART susceptibility in *Plasmodium*. Defining ubiquitin-dependent mechanisms at the cytostome may therefore reveal tractable points of vulnerability relevant to ART response.

This project aims to (i) test whether cytostomal proteins bind ubiquitin, (ii) map the regions required for ubiquitin binding, and (iii) identify interacting partners, to clarify their roles in endocytosis and explore potential implications for ART susceptibility.