Receptor for Activated C-Kinase 1 (PfRACK1) is required for *Plasmodium falciparum* intra-erythrocytic proliferation

Emerging resistance to current anti-malarials necessitates a more detailed understanding of the biological processes of *Plasmodium falciparum* proliferation, thus allowing identification of new drug targets. The well-conserved protein Receptor for Activated C-Kinase 1 (RACK1) was originally identified in mammalian cells as an anchoring protein for protein kinase C (PKC) and has since been shown to be important for cell migration, cytokinesis, transcription, epigenetics, and protein translation. The *P. falciparum* ortholog, PfRACK1, is expressed in blood stages of the parasite and is diffusely localized in the parasite cytoplasm. Using a destabilizing domain to allow inducible knockdown of the endogenous protein level, we evaluated the requirement for PfRACK1 during blood-stage replication. Following destabilization, the parasites demonstrate a nearly complete growth arrest at the trophozoite stage. The essential nature of PfRACK1 suggests that the protein itself or the pathways regulated by the protein are potential targets for novel anti-malarial therapeutics.