Mechanisms controlling motility and host cell invasion by the Apicomplexans

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Members of the phylum of Apicomplexa are important protozoan parasites that infect humans and animals, causing lethal or debilitating diseases worldwide. *Plasmodium falciparum* is the etiologic agent of malaria, which kills over a million people every year whereas *Toxoplasma gondii* infect all warm-blooded animals with one third of the human population being chronically infected. This parasite can cause brain and eye defects in the unborn fetuses of infected women and cerebral toxoplasmosis in immunocompromized patients.

Apicomplexans lack normal appendages for locomotion such as cilia and flagella and instead the invasive stages of these parasites use gliding motility to power their migration across biological barriers and to actively invade host cells and egress from infected cells. The molecular machinery that generates motion, the "Glideosome", is composed of signalling molecules, myosin motor complexes, regulators of actin dynamics, complexes of adhesins and proteases that all act in a concerted action.

Activation of these invasion factors needs to be tightly controlled in time and space and some of the key players of the machinery are regulated by posttranslational modifications such as phosphorylation and acylation. Interestingly, S-palmitoylation, which is a reversible addition of palmitate to proteins at cysteine residues through a thioester linkage, is frequently observed as PTM on the components of the glideosome. The Apicomplexans possess a dozen of genes coding for putative palmitoyltransferases and a few putative acyl protein thioesterases presumably capable of removing the thioester-linked palmitate from palmitoylated proteins. Enzymes involved in phosporylation and acylation of the components of the glideosome are currently investigated as potential novel drug targets against the Apicomplexa.