

GHI Floor Seminars

Special seminar by invited speaker

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“Prison Break”

Toxoplasma egress from infected cells is a tightly programmed event controlled by a lipid mediator

The phylum Apicomplexa groups obligate intracellular parasites responsible for severe veterinary and human diseases. The most ubiquitous member of the phylum, *Toxoplasma gondii* establishes a life-long chronic infection in human and animals. Gliding motility, a substrate-dependent form of locomotion, powered by an actomyosin system, assists invasion and egress from the infected cells, two key steps in the lytic cycle of the Apicomplexa. Egress from infected cells is a temporally orchestrated process underpinned by the release of the apical secretory organelles termed micronemes. At any point during intracellular replication, *T. gondii* tachyzoites are able to egress in response to deleterious environmental changes by activation of the cGMP-dependent protein kinase. This leads to a raise in intracellular calcium and production of intracellular phosphatidic acid, two key mediators of microneme adhesins exocytosis and their connection to the actomyosin system. We recently uncovered that in absence of extrinsic (alarming) signals, a diacylglycerol kinase secreted into the parasitophorous vacuole, produces phosphatidic acid, which dictates programmed egress after five to six cycles of parasite multiplication. Phosphatidic acid acts as an intrinsic signal that elicits natural egress upstream of an atypical guanylate cyclase, which is uniquely conserved in alveolates and ciliates and composed of a P4-ATPase and two guanylate cyclase catalytic domains. This study proposes a newly mechanism by which guanylate cyclase activated to govern natural egress.

Host: Gisou van der Goot

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12:15, SV 1717