

Insights into the possible role of a Mycobacterial secretory protein in manipulating host function

Intracellular pathogens such as *Mycobacterium tuberculosis* (Mtb) have evolved with diverse strategies to manipulate host cell function. In this study, we identify a possible interaction of an Mtb secretory protein with the host nuclear pore complex (NPC), a critical structure regulating nucleocytoplasmic transport. Until now, through biochemical assays, we have demonstrated that this effector engages a specific NPC protein, suggesting a mechanism for Mtb to influence host nuclear signalling and immune responses. Our findings highlight the value of combining structural and host-pathogen biology to uncover hidden vulnerabilities in essential cellular machinery and possibly opens up new avenues for structure-based anti-TB small molecule discovery.



Sonanjali Aneja

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4:00 PM | THURSDAY | 19 JUNE 2025

●●● **AUDITORIUM, NII**

Characterization of phosphoinositide-binding proteins from malaria parasite

Phosphoinositides are phosphorylated derivatives of the structural membrane phospholipid phosphatidylinositol. They are enriched in several subcellular compartments and are involved in multiple cellular processes by interacting with specific phosphoinositide-binding domain-containing proteins. While phosphoinositides play a key role in the development of *Plasmodium falciparum*, the underlying molecular mechanisms need better understanding. Several putative phosphoinositide-binding proteins were identified in *Plasmodium in silico*, which are indispensable for parasite development. Two of these proteins were found to interact with phosphatidylinositol-3-phosphate (PI3P), which was critical for their subcellular localization in the parasite. The interaction of these proteins with PI3P seems to be critical for parasite development. Although further experimental validation is needed, these and other studies suggest that PI3P signaling via these proteins may be critical for asexual development of *P. falciparum*.



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●●● **AUDITORIUM, NII**