

Internal Seminar

Systematic development of small protein inhibitors for disrupting *P. falciparum* AMA1- RON2 interaction

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Malaria is a mosquito-borne infectious blood disease, caused by the Protozoan parasite, *Plasmodium Spp.* Widespread resistance against the front line antimalarials is very concerning, and warrants immediate alternative approaches to develop novel therapeutics. It is known that the Apical Membrane Antigen 1 (AMA1) - Rhoptry Neck protein (RON2) interaction is a critical step in parasite invasion of erythrocyte. Recent studies show that disrupting *pf*AMA1- RON2 interaction is enough to inhibit parasite invasion.

Here, I will present two strategies, both focuses on interrupting *pf*AMA1- RON2 interactions. In the first project, I will brief our ongoing effort to develop D-protein inhibitors against both *pf*AMA1 and *pf*RON2 by using Mirror-image phage display. I will also present some of our attempt to develop a methodology for peptide thioester synthesis, which is very essential for segment-wise chemical synthesis of larger proteins (e.g. AMA1). The second work, a collaborative work with David Baker's Lab, deals with the chemical synthesis of their computationally designed protein inhibitors for *P. falciparum* AMA1. We resolved the challenges in its synthesis, and achieved required amount of the folded protein to perform various biophysical experiments.

Monday, Oct 16th 2017

02:00 PM (Tea/Coffee at 01:45 PM)

Auditorium, TIFR (FReT-B)