Australian National University

Plasmodium tRNA synthetases as antimalarial drug targets

Thursday 19 September 2013 1 – 2pm

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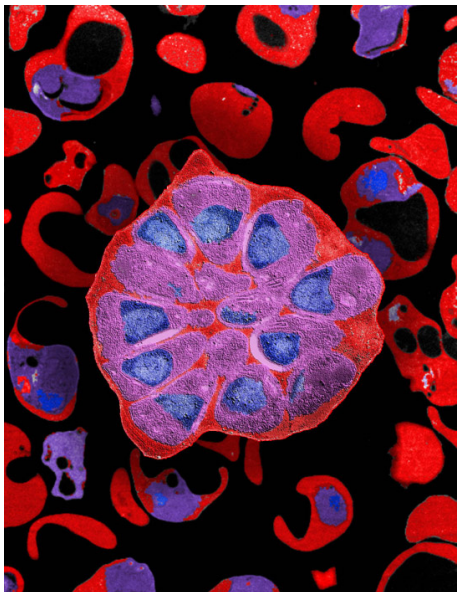
Slatyer seminar room R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU



Protein translation occurs in three compartments within *Plasmodium* parasites: the cytosol, the mitochondrion and a relic plastid called the apicoplast. Aminoacyl tRNA synthetases must charge tRNA for each of these compartments but *Plasmodium* encodes too few tRNA synthetases to allow a unique enzyme for each amino acid in all compartments. We have shown that several tRNA synthetases are dual targeted to the *Plasmodium falciparum* cytosol and the apicoplast, either by alternate translation initiation or alternate splicing. No mitochondrial localisation is apparent for any Plasmodium tRNA synthetase Because *Plasmodium* parasites depend on efficient translation, this process is a promising drug target. We have taken two approaches to drug discovery against *Plasmodium* tRNA synthetases – one is to inhibit the bacterial-like aaRSs that service the apicoplast based on existing anti-bacterial inhibitors, the other is to pursue novel and existing inhibitors of the dual targeted aaRSs that serve functions in both the cytosol and the apicoplast. Inhibitors of dual targeted tRNA synthetases, in particular, should have a rapid mode of action that blocks parasite proliferation, as well as mopping up surviving parasites by leading to loss of apicoplast.

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