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Riboflavin utilisation by the malaria parasite as a target for novel antimalarials

Thursday 6 September 2012 1pm

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Slatyer Seminar Room R N Robertson Building (#46), ANU

The intraerythrocytic malaria parasite *Plasmodium falciparum* requires an external supply of several essential nutrients, including riboflavin (vitamin B_2). In this study, riboflavin transport and metabolism in the infected erythrocyte and isolated parasite were characterised. Two genes encoding putative riboflavin-metabolism enzymes in the parasite's genome were investigated for gene function and localisation. The antimalarial activity of several riboflavin analogues was studied, with the compounds shown to target both the parasite and the host erythrocyte. I also present data demonstrating that a familial erythrocyte flavin deficiency prevalent in Ferrara, Italy, inhibits growth of the malaria parasite.

This study provides significant advances in knowledge regarding riboflavin utilisation by *P. falciparum*. It also demonstrates that riboflavin analogues may provide potent antimalarials with a dual mechanism of action, potentially reducing the parasite's ability to develop drug resistance. The results of this investigation may lead to identification of novel chemotherapies targeting parasite utilisation of riboflavin.

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STAFF LECTURE