D-35 Association of malaria with á1,3 galactosyl transferase inactivation in catarrhines

Rupika Rajakaruna[1,2], Ranjan Ramasamy[1]
([1]*Institute of Fundamental Studies, Kandy,*[2]*Present address: Dept. of Zoology, Univ. of Peradeniya*)

Humans and old world monkeys (catarrhines) lack Gal á1-3 Gal ß1-4 GlcNAc-R structures (á-galactosyl epitopes) and produce the corresponding antibodies (anti-gal), while new world monkeys (platyrrhines) and non-primate mammals possess á-galactosyl epitopes and lack anti-gal. We show that anti-gal binds to *Plasmodium falciparum* and inhibits its growth. This is consistent with the hypothesis that a *P.falciparum*-like malaria parasite may have selected for the inactivation of an á1-3 galactosyl transferase (1,3 GT) in catarrhines during the Miocene Period.

The effects of anti-gal antibodies on *P.falciparum* growth were examined *in vitro*, using anti-gal purified by affinity chromatography on Gal á-3 Gal ß1-4 GlcNAc oligosaccharide attached to silica beads.

Pooling results from different experiments, it was observed that anti-gal inhibited [3H]-hypoxanthine incorporation significantly. Visual determination

of the number of different blood stages indicated that inhibition of merozoite invasion was primarily responsible for this effect. Binding of anti-gal to *P.falciparum* was demonstrated by Enzyme-linked Immunosorbent assay (ELISA) and immunofluorescence assay on acetone-fixed late-stage parasites.

The binding of anti-gal to merozoite surface proteins may inhibit re-invasion by agglutinating merozoites or interfering with binding to rbc receptors of merozoites.

We postulate that during the early Miocene Period, a *P.falciparum*-like non-primate parasite adapted itself to infect primates in the old world leading to the inactivation of the á1,3GT gene. The driving force for inactivation would have been the ability to produce antibodies reactive with á-galactosyl epitopes of the parasite which then conferred significant protection against *falciparum*-like malaria without inducing auto-immunity.