

**A-27: Enhancement of cytoadherence of *Plasmodium falciparum* infected erythrocytes to CD36 by malarial paroxysm plasma**

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Recent studies have implicated Tumor Necrosis Factor alpha and parasite products released during a malarial paroxysm in the pathogenesis of malaria. The objective of the present study was to investigate the effect(s) of these plasma factors on expression of endothelial cell receptors CD36 and ICAM-1 which mediates adherence of *Plasmodium falciparum* infected erythrocytes (PfIE) to vascular endothelial cells.

Plasma collected from the *P. falciparum* infected patients during paroxysm, before and after the paroxysm (designated as pre- and post-paroxysm plasma respectively) and plasma collected randomly during infection were incubated

with target cells for 30 minutes at room temperature and assessed for infected cell adherence. Endothelial cell receptors CD36 and ICAM-1 expressed on Chinese hamster ovary cells (designated as CHO-CD36 and CHO-ICAM1 cells respectively) were used as target cells.

Incubation with paroxysm plasma of 5 out of 7 (71%) patients gave rise to 1.5-3.8 fold increase in PfIE-adherence to CHO-CD36 cells; there was no increase in adherence to CHO-ICAM1 and untransfected CHO cells. Incubation with pre- and post-paroxysm plasma did not affect adherence. Yet another effect was lysis to varying degrees (5-100%) of target cells when incubated with plasma for 24-48 h. The lysis was obtained with 65% of paroxysm plasma and with none of random plasma tested. These results indicate that plasma factors present during a malarial paroxysm enhance PfIE-adherence to CD36. Further, the effect of paroxysm plasma on lysis of target cells after 24-48 h. exposure could constitute a new assay for factors mediating pathogenesis of malaria.