A-26: Production of cytokines by peripheral blood mononuclear cells during malaria infections

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The changes in the cellular profile in the peripheral blood, the plasma cytokine levels and the cytokine production by different T-lymphocytes in the peripheral blood were studied in *Plasmodium vivax* (n=10) and *P. falciparum* (n=10) patients from the General Hospital Colombo and as controls in 10 healthy non-malarial individuals from Colombo.

Peripheral blood mononuclear cells (PBMC) were isolated from whole blood by standard density gradient methods. The phenotype of the cytokine producing cells were determined by staining with monoclonal antibodies (Mabs) against cell surface markers (monocytes, gamma-delta and alpha-beta)

and the same cells were then made permeable to anti-cytokine antibodies. The cells were stained with Mabs against the cytokines (TNF-alpha, TNF-beta, IL-1, and IL-6) by double immunofluorescent staining of the PBMCs. TNF-alpha and IL-6 levels were measured in patient plasma by an Enzyme Linked Immunosorbant Assay.

Malaria infected individuals had significantly elevated plasma TNF-alpha and IL-6 levels compared to non-malarial controls ($p < 0.05$). The proportion of total PBMCs producing TNF-alpha and IL-1 during infection were generally higher than in the control although it was not statistically significant. The monocyte count (but not other cell counts) were elevated in malaria patients compared to controls ($p < 0.05$).

In both patient and control groups, 90-100% of the gamma-delta T-cells which constituted less than 5% of PBMCs produced TNF-alpha and less than 10% produced IL-1 and IL-6. 40-50% of alpha-beta cells produced all four cytokines and 100% of monocytes produced IL-1 and TNF-alpha. Thus, during a malaria infection, the main source of TNF-alpha and IL-1 among PBMCs appears to be monocytes and alpha-beta cells, and of IL-6 and lymphotoxin was alpha-beta cells. No significant differences were detected either in the cytokine levels or their production by the different cellular subsets between *P. vivax* and *P. falciparum* infections.