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ACLEUS STANSLAUS MALINZI RUTTA : CYTOKINE RESPONSE AND
GENETIC REGULATION IN CHILDREN AND ADULTS WITH CEREBRAL
MALARIA DISEASE. THESIS ADVISORS: SANSANEE CHAIYAROJ, Ph.D.,
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Plasmodium falciparum is the most lethal form of human plasmodia. It induces various immunological responses that enable it to evade the immune system of the immunocompetent host and induce disease pathology. Death is usually due to cerebral complications arising from a number of phenomena. Several theories exist to explain the pathogenesis of cerebral malaria including the central role of cytokines in the modulation of the immune response in resolution and pathology to *Plasmodium falciparum*. In addition, various cytokines have been implicated in malaria associated immunosuppression. Related to disease susceptibility/resistance is also the involvement of the factors of HLA which play a crucial role by contributing to recognition of self and non-self, to the immune responses, to antigenic stimuli and to coordination of cellular and humoral immune responses.

In this study, I have explored the pathogenesis of cerebral malaria with particular attention to the possible relationship between susceptibility or resistance to cerebral malaria and profile of cytokine production /secretion pattern in children, and in groups of adults with cerebral, severe and uncomplicated malaria. The possible association of some HLA factors in susceptibility to cerebral malaria was also analyzed. I investigated the possible interaction and associations among cytokines by determining their levels in cerebral malaria patients.

I found significant elevation of IL-10, IFN- γ , TNF- α and immunoglobulin E in cerebral malaria patients compared to their matched controls. TGF- β , an immunosuppression cytokine, was statistically significantly decreased in cerebral malaria patients compared to controls, severe and uncomplicated malaria. There was no significant association among cytokines suggesting that the immunomodulatory action of one cytokine is independent of another. There was no interaction among cytokines in the modulation of the disease. My study also demonstrated no significant association between HLA-DQB1 and HLA-DRB1 gene products and susceptibility to cerebral malaria.