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Poster presentation

IgM and IL-10 up regulation is related to parasite numbers and relapse parasitaemia during late stage disease in vervet monkeys infected with *Trypanosoma b. rhodesiense*

Maina Ngotho*[1,2,5], John Kagira[2], Henrik Jensen[3], Simon Karanja[4], Idle Farah[1] and Jann Hau[5]

Address: [1]Institute of Primate Research (IPR), PO Box 24481 00502, Karen, Nairobi, Kenya, [2]KARI-Trypanosomiasis Research Centre (KARI-TRC), PO Box 362, Kikuyu, Kenya, [3]Department of Veterinary Pathobiology, Faculty of Life Sciences, University of Copenhagen, Frederiksberg, Denmark, [4]Department of Biochemistry, Jomo Kenyatta University of Agriculture & Technology, P.O. Box 62000 00100 Nairobi, Kenya and [5]Department of Experimental Medicine, University of Copenhagen & University Hospital, DK-2200 Copenhagen N, Denmark

Email: Maina Ngotho* - ngothojm@yahoo.co.uk; mang@emed.ku.dk

* Corresponding author

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Human African trypanosomiasis is an infectious, neglected, vector-borne protozoan disease which has an early haemolymphatic phase followed by meningoencephalitis due to neuroinvasion of the central nervous system by trypanosomes. The determination of the earliest timing when the blood-brain barrier (BBB) is breached during neuropathogenesis of sleeping sickness has critical implications on the choice of therapy, without which the infection is fatal. Interleukin 10 (IL-10) and IgM have been proposed as potentially important biomarkers when this occurs. The vervet monkey is susceptible to experimental infection with same aetiological agent as that which causes HAT, providing a useful pathogenesis model. Fourteen vervet monkeys were experimentally infected with *Trypanosoma brucei rhodesiense*. The animals were treated with diminazene aceturate (DA) and melarsoprol (MelB) 28 and 140 days post infection (dpi), respectively. Serum and CSF samples were obtained at weekly intervals and assayed for immunospecific IgM and IL-10 by ELISA. There was no detectable immunospecific IgM in the CSF before 49 dpi. The CSF IgM increased progressively to peak levels that coincided with the appearance of relapse parasites in blood 140 dpi. The serum IgM levels increased significantly over control levels starting 21 dpi and peaked between 133 and 140 dpi. The serum IL-10 levels increased rapidly and were significantly elevated between 14 and 21 dpi. Following sub-curative treatment with DA the serum IL-10 levels declined and remained below detection limit until 127 dpi which coincided with relapse of parasitaemia. A significant rise in white cell counts in CSF was recorded starting 35 dpi. When curative treatment with MelB was carried out, there was a rapid decline of the three parameters leading to attainment of preinfection levels of IgM and white cell counts within 30 days. However CSF IgM levels remained above pre-detection levels to the end of the study. We were unable to measure/detect IL-10 in the CSF. Serum and CSF concentrations of immunospecific IgM as well as serum IL-10 changes follow a similar pattern that mimics the progression of the disease and may present reliable ad useful biomarkers of the disease stage.