

Conclusions: These observations suggest that the effect of the association between lymphoproliferative response and virus load is established early during HIV-1 infection and does not increase over time and suggest that antigen-specific lymphoproliferative responses reflect the dynamic state of HIV-1 infection and are inversely associated with virus load.

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CELL MEDIATED IMMUNITY TO *PLASMODIUM VIVAX* INFECTION: THE PROFILES OF MEMORY T-CELLS ACTIVATION IN MALARIA PATIENTS OF THAILAND

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Immunity induced by *P. vivax* during acute infection may lead to memory T-cells recruitment which will be activated during relapsing period or subsequent infection. This study therefore aims to determine memory T-cells in acute and convalescent *P. vivax* infection. PBMC from 13 patients infected with *P. vivax*, 17 immune villagers lived in endemic area, and 15 healthy adults were separated by gradient centrifugation. Patients were followed up after antimalarial treatment on day 14, 28 and 60. Memory T-cell phenotypes were determined by using three-color flow cytometry. Memory T-cells are recognized by the expression of CD27⁺ in the CD45RO⁺ T-cells population as an early memory T-cells which will differentiate to CD27⁻ when mature. In this study mean percentage of CD4⁺CD45RO⁺CD27⁺ and CD27⁻ T-cells were increased during acute infection (34.13, 17.19%) when compare with those of healthy donors (12.67, 6.13%, P<0.01) and immune villagers (14.04, 9.30%, P≤0.02). In addition, CD27⁺ levels remained high up to day 14 (36.44%) and slightly decreased during day 28 and 60 (24.81 and 22.90%) after antimalarial treatment. However, the CD27⁻ T-cells were continuously decreased from day 14 to 28 and 60 (13.96, 11.82 and 10.68%). The mean percentage of CD8⁺CD45RO⁺CD27⁺ T-cells also showed higher level in acute infection (31.34%) when compared with those of healthy donors (7.01%, P≤0.05) and immune villagers (17.87%, P>0.05). This CD27⁺ phenotype was slightly decreased from day 14 (19.75%) to day 60 (18.96%) until nearly the same level as the immune villagers (17.97%). In conclusion, during acute *P. vivax* infection, both early stage and mature memory T-cells were triggered to yield effector cells. The memory T-cell phenotypes could be expanded by the parasites activation. Upon treatment and clearance of parasites from the circulation, the production of memory T-cells was slowed down. However, memory T-cells of both CD4⁺ and CD8⁺ populations were maintained in the immune villagers living in the endemic area. These suggest that memory T-cells play role in the development of cellmediated immunity against *P. vivax* infection. This work is supported by Royal Golden Jubilee Ph.D. Programme/Thailand Research Fund.

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