CELL-MEDIATED IMMUNITY TO *PLASMODIUM VIVAX* INFECTION

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Immunity induced by *P. vivax* during acute infection leads to memory T cells recruitment of which will be activated during subsequent infection. This study therefore aims to verify the level of memory T cells during acute and convalescent periods. Memory T cells are recognized by the surface molecules $CD45RO^{+}CD27^{+}$ as an early stage and $CD27^{-}$ as a mature stage. The results showed significant [P<0.01] increase of mean percentage of $CD4^{+}$ memory T cells during acute infection when compared with those either from healthy donors and immune villagers. The *P. vivax*-induced memory T cells were maintained at high level until 60 days post treatment. The mean percentage of $CD8^{+}$ memory T cells was also significantly higher during acute infection until 60 days post treatment. Interestingly, the $CD8^{+}$ memory T cells was stably maintained at high level among the non-acute malaria immune villagers in contrast to the low level $CD4^{+}$ memory T cells in the same immune villager group. These results suggest that memory T cells particularly $CD8^{+}$ phenotype-play role in the development of naturally acquired protection against *P. vivax* infection. The ongoing study will investigate further whether or not the protection is mediated by the memory T cells of γδ-phenotype.

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THE DEFORMABILITY OF RED BLOOD CELLS PARASITIZED BY *PLASMODIUM FALCIPARUM* AND *P. VIVAX*

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Red blood cells (RBCs) must deform considerably during their multiple passages through the microvasculature and the sinusoids of the spleen. RBCs infected with *Plasmodium falciparum* (Pf-IRBCs) become increasingly rigid as they mature but avoid splenic clearance by sequestering in venules and capillaries. In contrast, RBCs infected with *P. vivax* (Pv-IRBCs) do not sequester. We compared the effects of *P. vivax* and *P. falciparum* infection on RBC deformability in a laminar shear flow system. Pf-IRBCs became more rigid as the parasite matured, but equivalent maturation of Pv-IRBCs resulted in a doubling of flexibility. Coincidentally, the IRBC surface area increased from 56.7 ± 1.3 μm^2 to 74.7 ± 0.6 μm^2 to 90.9+/-1.1 μm^2 in ring-, trophozoite-, and schizont-stage Pv-IRBCs, respectively, whereas Pf-IRBCs did not increase in size. *P. vivax* increases the deformability of IRBCs and thereby avoids splenic entrapment.

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