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Evasion of Host Immune System by Malaria Parasite, *Plasmodium falciparum*, at an Asexual Stage

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Malaria parasite has been evolved molecular mechanisms for evading the host immune guard that are the major obstacles for developing effective malaria vaccine. The SERA of *P. falciparum* is one of the vaccine candidate antigens. The immunization of Saimiri monkeys with a recombinant SERA (SE47') has shown that antibody titers against SE47' in two immunized monkeys were stimulated after the parasite challenge. The observed increase in SERA ELISA titers following challenge infection correlated with the degree of protection. However, all of the control monkeys did not develop antibodies against the recombinant SE47' after the parasite challenge suggesting that SERA molecule is masked from the host immune response. A survey of natural diversity within SERA protein revealed that more than 95% of amino acid sequence is conserved among alleles examined and that the majority of diversity is localized to the 47 kDa domain. Unlike the C-terminal 19 kDa fragment of MSP-1 or AMA-1 whose diversity is mainly point mutations, a majority of the diversity of 47 kDa segment of SERA is caused by deletion/insertion events. These observations strongly suggest the feasibility of further development of a malaria vaccine based on SERA protein. Such a vaccine formulation may only need to contain a limited number of allelic forms of the SERA gene.

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