# Engineered resistance in *Anopheles stephensi* to antimalarial effectors-Effect on *Plasmodium* development and evolution.

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Malaria transmitted by *Plasmodium falciparum* progresses through various developmental stages within the *Anopheles sp.* and can be transmitted to humans by female *Anopheles* mosquitoes. In view of the efforts towards the global eradication of malaria, population modification of the vector mosquito populations by efficient introgression of antimalarial gene(s) is being explored.

A previously characterized dual-effector, transgenic *Anopheles stephensi* mosquito line expressing single-chain antibodies (scFvs), namely m2A10 and m1C3 targeting the *Plasmodium* antigens, Circumsporozoite protein and Chitinase, was challenged with the rodent parasite *Plasmodium berghei* chimeric for the *Plasmodium falciparum* CSP antigen. Further these infected transgenic mosquitoes were used to infect a naïve mouse and the reinfected parasite was harvested at every infection cycle for four subsequent generations. Across the four generations, there was a delay observed in the appearance of the parasite upon reinfection.

The study successfully establishes an *in vitro* experimental evolution model, which can be used to evaluate the risk associated with mosquito transgenesis for the probable genetic changes that could occur in the gene encoding *Plasmodium* antigen(s), upon the multi-generational interactions with the scFvs expressed in the transgenic *Anopheles stephensi.*