ABSENCE OF FC RECEPTORS ON AEDES ALBOPICTUS C6/36 CELLS PERMISSIVE FOR DENGUE VIRUS 2

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Over the last few years considerable interest has developed in the idea of genetically modifying arthropod vectors of infectious agents deleterious to human health. Work in this area is well summarized (1, 2, 3). The mosquito, Aedes aegypti, co-habits with approximately half the world's population and, in addition to a number of other arboviral diseases, is responsible for the transmission of Dengue virus and the associated disease states of Dengue haemorrhagic fever/ Dengue shock syndrome. At least one mode of entry for Dengue virus particles into mammalian cells is thought to depend on Fc receptor mediated adhesion of opsonized viruses, internalization via clathrin pits and membrane fusion in association with the E-glycoprotein due to the acidic pH of endocytic vacuoles (4, 5). The difficulty of producing an efficient polyvalent vaccine against Dengue virus infection makes genetic manipulation of the mosquito host a potentially attractive alternative control measure. In the event that a suitable means of transfecting Aedes aegypti becomes available, then the choice of an appropriate gene to be modified will become paramount. It appeared to us, that the Fc receptor, if present in vector species and active in the infectious process of Dengue, would prove an excellent choice for genetic manipulation. Elsewhere, Fc receptors are known to occur among several Gram-positive bacteria, Trypanosoma, Schistosoma, shark and mammalian species.

Initially, we wanted to test for the presence of Fc receptors on *Aedes albopictus* C6/36 cells grown at 28 °C, a cell line originally selected for its permissiveness for Dengue virus 1, 2, 3 and 4 (6). The same cell stock had previously been utilized to grow Dengue virus 2 in our laboratory. Flow cytometry and rosette assay using sensitized sheep crythrocytes (7), demonstrated the absence of Fc

receptors for either IgG_{1/2}, IgE or IgM.

Although non-conclusive for the situation *in vivo*, it would appear unlikely that Dengue virions utilize Fc receptors to infect mosquito cells. Thus, although the choice of the Dengue virus receptor remains valid as a potential site for molecular intervention, the nature of this receptor has yet to be determined. We report these negative results to help avoid other groups from following the same path, but also to stimulate interest in the infective cycle of Dengue virus in its invertebrate host where little is known.

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