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Malfuction through single point mutation in an odorant receptor of the major disease vector, *Aedes aegypti*

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The *Aedes aegypti* (*Ae. aegypti*) is a primary vector for dengue virus, the most widespread vector-borne viral disease. *Ae. aegypti* also acts as a vector of yellow fever, chikungunya and zika virus. Although odorant receptors of *Ae. aegypti* (*AaOrs*) has been identified 131 including co-receptor (*AaOrco*) by bioinformatics analysis, there is a lack of studies about function and structure. We have previously reported that *AaOr8* and *AaOr49* in the mouth part of mosquito play a major role in mosquito blood-feeding behavior by as detecting the volatile compounds in blood. In this study, we found the ligand binding site of *AaOr8* and *AaOr49* using SYBYL-X 2.1 packages and homology protein model by the computer program. For demonstrated ligand binding site, we also performed calcium imaging using Sf9 cells with site-directed point mutation *AaOrs*. *AaOr8*-S167A in which serine residue in position 167 amino acid was substituted to non-polar and hydrophobic amino acid, alanine, represented a loss of binding affinity to 1-octen-3-ol and cyclohexanol, while *AaOr49*-V168M where valine in the position 168 amino acid residue was replaced with methionine and *AaOr49*-Y190A where tyrosine of the position 190 amino acid was substituted to alanine showed complete loss of binding affinity to 2-ethyl-1-hexanol with intact responses to cyclohexanone and benzyl alcohol. Taken together, We identified major active residues of *AaOr8* and *AaOr49* using both *in silico* and *in vitro* approach, which should be useful in future research about odorant receptors and control of disease vector insects.