

## SUJATHA SUNIL

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Sujatha Sunil obtained her B.Sc and M.Sc (Botany) in 1994 at Madras University, Chennai and Ph.D (Basic Medical Sciences) at the M.G.R Medical University, Chennai in 2000 with specialisation in cancer genetics. After her Ph.D, she headed the HLA unit, Genetic Medicine department at Sir Ganga Ram Hospital, New Delhi and following a stint as Research Scientist at the All India Institute of Medical Sciences, she pursued her research in malaria at National Institute of Malaria Research (NIMR), New Delhi and International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi. Sujatha gained industrial exposure at Clininvent Pvt. Ltd as Head of Genomics, Sequencing, and served as adjunct faculty at Institute of Molecular Medicine (IMM), New Delhi. With the Ramanujan Fellowship, she joined International Centre for Genetic Engineering, New Delhi as Research Scientist in July 2010.

### RESEARCH DESCRIPTION

#### Understanding oxidative stress in *Anopheles* during *Plasmodium* development

*Plasmodium* lives in a pro-oxidant environment that contains oxygen and iron and its redox and antioxidant systems are well developed to combat stress in both hosts, namely human and mosquito. In spite of this, the parasite numbers dwindle at the time of invasion in *Anopheles*. Of the several species of *Anopheles*, not all are vectors and there are several strains that are refractory to *Plasmodium* infection. Morphological analysis of the midgut of the refractory and susceptible strains reveals differences in pericardial cells that are active in detoxification and neutralization of ROS. These morphological differences suggest systemic deficiency in ROS detoxification may contribute to refractoriness in mosquitoes. Our lab is involved in studying parasite-vector interactions during the development of the parasite within the midgut and salivary glands of the vector with special emphasis on understanding the redox state regulation using transcriptomics, miRNA and systems biology approaches.

#### Divergence of Chikungunya virus and its host pathogen interactions with the vector

Chikungunya virus (CHIKV), a positive-stranded alphavirus, causes epidemic febrile infections characterized by severe arthralgia. Usually transmitted by *Aedes aegypti*, CHIKV has emerged as a pathogen of concern due to its adaptability in an atypical vector, namely *Aedes albopictus* in the latest epidemic affecting millions. It has been shown that mutations in the structural envelope proteins altered its vector preference. Our lab is focusing in studying the evolution of the virus in our country vis-à-vis mutations in the viral genome. Our preliminary data has revealed the emergence of a new molecular signature in the Indian isolates. Work is underway to study the effect of these mutations on the pathogenicity of the virus and its virulence.

During the course of infection, CHIKV encounters numerous bottlenecks, constituted by host cell functions both essential as well as inhibitory for viral propagation. However, once inside the vector, the virus evades the vector's surveillance mechanism and builds up infective titre to infect subsequent individuals. Our long term goal is to understand the basis of virulence and the host factors that may be involved in this process.

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