Seminar

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Effect of Type I Interferon on West Nile Virus specific CD8 T cells: An intricate phenomenon

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496 Seminar Hall, THSTI
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Abstract:

Development of potent anti-viral vaccines has always been a challenge for the viral immunologists since the proper understanding of immune memory depends not only on the diverse immune status of the individuals but also on the virus in question. One of the important contributors that help in programing the immune response is the Type I Interferon (IFN). Previous work from our laboratory has shown that the induction and requirement of Type I IFNs on CD8 T cells for their clonal expansion and memory generation depends largely on the pathogen. In this context, there is very little known about the effect of Type I IFNs on the CD8 T cell development during West Nile Virus (WNV) infection, which in humans the clinical manifestations can range from feeble fever to fatal meningoencephalitis. Here, using a murine footpad infection model we addressed two questions: (1) what happens to virus-specific CD8 T cell responses if the infected host is devoid of IFN-I signaling, (2) what happens to virus-specific CD8 response if only CD8 T cells lack IFN-I signaling. Our results show that IFN-I signaling on the infected host have opposing results on the quantity and quality of CD8 T cell response. The lack of IFN-I signaling in the host lead to increased susceptibility to WNV infection and exaggerated CD8 T cell clonal expansion with poor effector function, while lack of IFN-I signaling only on the CD8 T cells showed a diminished clonal expansion. While, in a host that has uncompromised IFN-I signaling, the IFN-I mediated innate viral control inhibits the exaggerated CD8 T cell clonal expansion with fitter effector differentiation. Together our results suggest that the IFN-I mediated innate viral control is important for determining the measure of CD8 T cell effector and memory differentiation. This knowledge has implications in the development of vaccine strategies against the neurotropic WNV infection.

Brief Bio sketch:

Education

2009 Ph.D. (Jadavpur University) from the Dept. of Infectious Diseases & Immunology, Indian Institute of Chemical Biology, CSIR, Kolkata, India

Thesis Title: Complete Soluble Antigen from a UDP-Galactose:N-Acetylglucosamine β 1-4 Galactosyltransferase expressing Leishmania donovani promastigote induces complete protection in an experimental model of visceral leishmaniasis

2004 M. Tech. (Bio-technology) Jadavpur University, Kolkata, India

Thesis Title: Infectivity and Attenuation Of Leishmania donovani parasites: Galactose terminal glyco-conjugates and the developmentally regulated galactosyl transferases

2002 B. Pharm. (Pharmaceutical Technology) Jadavpur University, Kolkata, India

Thesis Title: Antilipidemic and Anticholesterolimic Drugs

Research Experience

2014-present Research Associate at Emory Vaccine Center, Emory University, Atlanta, USA

Supervisor – Dr. Murali-Krishna Kaja

Research projects:

Understand the role of Type-I Interferon in early innate immune activation and bystander action following a flavivirus infection.

Immune regulation on viral infection in aged individuals.

Cholesterol 25-Hydroxylase and its effect on CD8 T cell memory differentiation.

RIG-I activator as influenza vaccine adjuvants in protection against lethal challenge with flu virus in aged individuals.

Research interest: One of main research interest is to continue translational research in understanding the TCR and Type I Interferon signaling cross-talk that would help develop better and fitter antigen specific memory T cells that can be have long-term implications in the field of vaccine development