IMCB Invited Speaker

Speaker : **Dr. Daniel J. Cua**
Merck Research Labs, Palo Alto, USA

Date : 3 November 2014, Monday

Time : 2:00PM - 3:00PM

Venue : Level 5, Multi-function Room 1, Matrix, Biopolis

Host : A/Prof. John Connolly

Seminar :

IL-23 regulation of adaptive and innate immunity

Following the discovery of IL-23-dependent T cell immunity, the past decade has witnessed a major revision of the T-helper subset paradigm and substantial progress has been made in deciphering the molecular mechanisms of T cell lineage commitment and function. I will present our recent work on the transcriptional control of TH17 cell development and highlight the protective vs. pathogenic roles of IL-17 and IL-22 in mucosal tissues. I will also discuss the emerging clinical data showing that antibody-mediated neutralization of IL-17 and IL-23 are remarkably effective for treating immune-mediated inflammatory diseases.

About the Speaker :

Daniel J. Cua is Senior Principal Scientist at Merck Research Labs, Palo Alto (Formerly DNAX Research Institute). The focus of his laboratory is discovery of novel cytokines that regulate the immune system during health and disease. He has contributed to medical literature with numerous original research articles of which 6 landmark studies were referenced by 1000 to 2500 scientific papers. In 2014, he was listed by Thomson Reuters Intellectual Property and Science as one of the "World's Most Influential Scientific Minds". He is also principal investigator / co-investigator of more than 15 US and international patent applications for the use of biologic agents to treat unmet medical needs.

One of his research interests over the past decade focused on understanding the roles of IL-23 and IL-17 in immune-mediated inflammatory diseases such as multiple sclerosis, rheumatoid arthritis, ankylosing spondylitis, and inflammatory bowel disease. His work led to the initial discovery of a novel T cell subset, called Th17 cells that are highly pathogenic in autoimmune inflammatory conditions. His team demonstrated that IL-23 is required for the in vivo function of Th17 cells and that blocking this immune pathway can reverse brain, joint, and intestinal autoimmune disorders. Importantly, this work has provided the bases for successful clinical development of therapeutic compounds for the treatment of autoimmune disorders.

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