Institute of Molecular and Cell Biology
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SEMINAR ANNOUNCEMENT

We would like to invite you to attend this seminar hosted by Dr. Vinay Tergaonkar (IMCB) & Dr. Alessandra Mortellaro (SIgN):

Date: 29 September 2014, Monday
Time: 2:00PM – 3:00PM
Venue: Level 3, IMCB Seminar Room 3-46, Proteos, Biopolis

Speaker: Dr. Kate Schroder, Laboratory Head, Institute for Molecular Bioscience, The University of Queensland, Australia
Title: Inflammation control by neutrophil inflammasomes

The innate immune system is the body's first line of defence against microbial attack. The immune system recognises such situations of cellular 'danger' via protein families that recognise invariant microbial structures and launch antimicrobial defence mechanisms. One such innate immune receptor pathway is a multiprotein signalling complex called the 'inflammasome', which controls the activity of pro-inflammatory caspases such as caspase-1. Macrophage inflammasomes drive potent innate immune responses against intracellular pathogens, by eliciting caspase-1-dependent pro-inflammatory cytokine production (e.g. interleukin (IL)-1β and pyroptotic cell death. However, the potential contribution of other cell types to inflammasome-mediated host defense has not been investigated in detail. We have found that neutrophils, typically viewed as cellular targets of IL-1β themselves activate the NLRC4 inflammasome during acute *Salmonella* infection, and are a major cell compartment for IL-1β production during acute peritoneal challenge in vivo. Importantly, unlike macrophages, neutrophils do not undergo pyroptosis upon NLRC4 inflammasome activation. The resistance of neutrophils to pyroptotic death is unique amongst inflammasome-signaling cells so far described, and allows neutrophils to sustain IL-1β production at a site of infection, without compromising the crucial inflammasome-independent antimicrobial effector functions that would be lost if neutrophils rapidly lysed upon caspase-1 activation. Inflammasome pathway modification in neutrophils thus maximizes host pro-inflammatory and antimicrobial responses during pathogen challenge.

Biography:
Kate Schroder's research centers on the interactions between host and pathogen during the initial stages of infection and the development of inflammation. Her PhD studies investigated cross-talk between innate immune signalling pathways (cytokines and Toll-like receptors) in macrophages (PhD in Immunology awarded 2005). Her subsequent postdoctoral position (2005-2008), focused on transcriptional programs triggered by macrophage differentiation and toll-like receptor ligation. Simultaneously, Kate was heavily involved in investigating macrophage gene regulation as part of a large international consortium, FANTOM4. During her NHMRC CJ Martin Fellow in Jürg Tschopp's group in Switzerland (2009-2011), Kate gained expertise in Nod-like receptor function and inflammasome signalling. She returned to Australia in 2011, and now heads the Inflammasome Laboratory of the Institute for Molecular Bioscience, as an ARC Future Fellow.

ALL ARE WELCOME *(No registration required)*