

BIOENGINEERING

PRESENTS

How do Pattern Recognition Receptors, such as Toll-like Receptors, signal within living cells?



THURSDAY, MAY, 19 2016

1:00 PM – 2:00 PM

2101 ENGINEERING V

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ABSTRACT:

Innate immune responses are mediated by cell surface (Toll-like receptors (TLRs)), cytosolic (Nuclear Oligomerisation Domain Leucine Rich repeat receptors (NLRs)) and endosomal pattern recognition receptors (PRRs) which drive protective immunity against pathogens and optimise the development of adaptive immunity. Many diseases of man and animals, particularly those associated with chronic inflammation, are linked to dysregulation of innate immunity. We use multidisciplinary approaches to determine how PRRs signal and how the macromolecular innate immune signaling complexes they induce self-assemble within cells. We are particularly interested in how their molecular constituents change in response to different pathogens or ligands and the consequent functional impact on the host immune response to infection. There are profound differences within and between animal species in inflammasome genes and how these impact on inflammasome self-assembly and host innate immune responses is of particular interest to the Bryant lab.

BIOGRAPHY:

Dr. Clare Bryant is Professor of Innate Immunity at the University of Cambridge. She graduated in Physiology and Biochemistry, then qualified as a veterinary surgeon, but decided to focus on research rather than clinical work. After completing a PhD at the University of London she worked as a Wellcome Research Fellow with Professor Sir John Vane in London before moving to Cambridge. Her work is focused on understanding how bacterial pathogens are detected by host cell receptors. Her work involves collaborations with mathematicians, physicists and chemists to understand the fundamental molecular mechanisms involved in host-pathogen interactions.

Man, S.M., Hopkins, L.J., Nugent, E., Cox, S., Glück, I., Tourlomousis, P., Wright, J.A., Cicuta, P., Monie, T.P. and Bryant, C.E. (2014) Inflammasome activation causes dual recruitment of NLRC4 and NLRP3 to the same macro-molecular complex. *Proc Natl Acad Sci* **111**, 7403-8

Man SM, Ekpenyong A, Tourlomousis P, Achouri S, Cammarota E, Hughes K, Rizzo A, Ng G, Wright JA, Cicuta P, Guck JR, Bryant CE. (2014) Actin polymerization as a key innate immune effector mechanism to control Salmonella infection. *Proc Natl Acad Sci.* **111**:17588-93