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Using viral innate immune evasion strategies to inform vaccine design

Thursday 27 June 2013 1.00 – 2pm

Dr Rebecca Sumner University of Cambridge, UK

Slatyer seminar room R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU



The innate immune system provides the first line of defence against viruses and its activation results in the production of soluble messengers such as interferons and proinflammatory cytokines that combat the infection and activate adaptive immunity. During their evolution viruses have developed numerous mechanisms to counteract these defences, and as such can be useful tools to study the immune system. The poxvirus vaccinia virus (VACV) encodes an interesting family of proteins with a Bcl-2 fold, the majority of which have evolved to antagonise host innate immune responses rather than apoptosis. A previously uncharacterised member of this family, C6, was found to be a broad inhibitor of interferon β production. Deletion of C6 from VACV resulted in significant attenuation in mouse models of infection and furthermore, vaccination with a C6 deletion virus provided better protection against subsequent lethal VACV challenge, indicating that

C6 contributes to immunogenicity. Recent data have demonstrated that while other members of this family of virus proteins inhibit innate immunity, deletion of only some of these lead to enhanced adaptive immune responses.

Characterising these differences will provide a better understanding of how innate immunity contributes to adaptive responses and can aid in the design of better vaccine vectors.

Biography: I completed my undergraduate degree in Biochemistry from Imperial College London in 2007, including a research year with Merck, Sharpe and Dohme in both the UK and Italy working on the design of an adenoviral-based malaria vaccine. Following this I did a Masters and a PhD, which was funded by a Wellcome Trust fellowship, at the St. Mary's Hospital Campus of Imperial College working with Geoff Smith on innate immune evasion strategies of vaccinia virus. I then moved with the Smith lab to the University of Cambridge in 2011 as a postdoc where I've continued my work on viral immunomodulation and its impact on designing more efficacious vaccines.

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