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Virus-host interaction: *the battle for supremacy*

Presented by

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Abstract

The 21st century has been marked with an increasing number of viral epidemics, even prior to the emergence of SARS-CoV-2. Between 2011 and 2017 alone there were more than 1350 epidemics across 172 countries. Many of these epidemics were caused by RNA viruses resulting in >100 million deaths/year. Currently, no vaccine or specific treatment is available for many of these infections. The major hurdle in developing vaccines against RNA viruses is their large genetic diversity. Hepatitis C virus (HCV) is one of the most genetically diverse RNA viruses that urgently needs a vaccine to control the ongoing epidemic globally. The relatively recent discovery that RNA viruses use multiple mechanisms to ensure their persistence has demanded sophisticated and multi-facetted research strategies, as it is evident that the simple vaccine designs successfully applied to many of the DNA viruses, such as hepatitis B, will not be effective. *My research objective is to combine cutting edge virological and immunological techniques, with novel bioinformatic analysis tools to investigate the interactions between* HCV *and the host during transmission and early disease progression.* HCV is a relatively unique pathogen in that only about 25% of infections will be cleared with the remainder developing into chronic infection with a high rate of progression towards liver failure or hepatocellular carcinoma if left untreated. Therefore, understanding the factors that drive successful clearance early in infection are highly relevant to vaccine design. Recent work by my team has been investigating which viral and immunological factors early in infection are associated with varied disease outcome. One of the key findings is that despite the donor having a large genetically diverse pool of viral variants, new HCV infections are on average initiated by only 1-3 unique 'founder' variants. This reduction in genetic diversity may be a vulnerable period in HCV infection. We have also investigated how rapidly, and under what selective pressure, these founder viruses evolve and discovered that both T cell- and antibody- driven selection pressure influence disease outcome. We have also investigated how this need to escape immune pressure is balanced against fitness costs for viral replication. The combined findings from these studies suggest that an early co-ordinated cellular and humoral immune response is critical to prevent the emergence of immune escape and to facilitate viral clearance.

Biography

Dr Rowena Bull is an Associate Professor and current NHMRC Investigator at The Kirby Institute, University of New South Wales, Australia. She completed a PhD in Medical Microbiology and Immunology in 2007 in the Faculty of Science, UNSW. Her thesis was focussed on examining the evolutionary mechanisms Norovirus used to cause periodic epidemics and pandemics. Over the last decade her research program has focused on understanding the transmission dynamics and pathogenesis of selected RNA viruses with local and global high health burden, by developing and applying cutting edge genomic methods to generate clinically relevant research outcomes. Most recently she has been using antigen-specific B cell sorting combined with single cell RNAseq to examine the optimal features of the antibody response and memory B cell response that contribute to sustained and robust responses against reinfection. A/Prof Bull is the Treasurer for the Australian Centre for Hepatitis Virology (ACHV) and Vice President for the Australasian Virology Society (AVS).

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