Neuroscience & Non-Communicable Diseases Seminar Series

All welcome

Friday 22 July 2022, 3pm

"Arming memory B cells to tackle RNA virus diversity"

Speaker: Dr Rowena Bull, The Kirby Institute, UNSW

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.

Abstract:

Induction of an immune response that can prevent re-infection is the basis behind all vaccines. But induction of a protective immune response against many of the RNA viruses that circulate with large genetic diversity has been problematic. We studied, in a rare prospective cohort, 15 subjects that were naturally followed through multiple infections of hepatitis c virus (HCV), the B cell and antibody characteristics that correlated with protection against chronic re-infection. HCV will develop chronic disease in 75% of patients and so we were able to compare individuals that were able to clear their second infection to those that developed a second or third chronic infection. We performed ELISAs, pseudoparticle and cell culture derived assays to measure autologous binding and neutralising breadth and also phenotyped and single cell RNA sequenced HCV-specific B cells. We found that the breadth of the antibody response did not necessarily protect from chronic re-infection, with evidence of antigenic sin from the conservation of the epitope hierarchy and clonal recall. However, a higher ratio of IgD+ to IgG+ HCV E2 positive cells and also a less 'activated' transcriptomic profile did associate with protection from chronic disease. Understanding what constitutes a protective response for highly variable RNA viruses is extremely important for both vaccine design and also for evaluating vaccine efficacy.

All welcome!

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