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## Memory CD4 T cell regulation of innate lymphoid cells during protective responses against influenza A virus

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The primary goal of vaccination is to protect individuals from the morbidity and mortality of pathogen exposure by generating immunological memory. Our current understanding is that memory CD4 T cells, commonly regarded as the regulators of memory cytotoxic CD8 T cells and/or antibody-producing B cells, provide protection by hastening pathogen clearance through 'faster, bigger and better' immune responses. It is now clear, however, that many different subsets of CD4 T cells, many with specialized functions other than providing 'help', are generated during immune responses against pathogens. In studies designed to ascertain the full functional potential of memory CD4 T cells responding against influenza A virus (IAV), we found that virus induced T<sub>H</sub>1-like, as well as *in vitro* polarized T<sub>H</sub>1 or T<sub>H</sub>17 memory CD4 T cells, enhance early innate inflammatory responses that correlate with better and earlier control of IAV in infected lungs. In further studies, we found innate lymphoid cells (ILC), which are not normally associated with anti-viral responses but rather associated with healing and wound repair, are also mobilized by memory CD4 T cells during the early stage of the response against IAV. Unexpectedly, the protection afforded by memory CD4 T cells against lethal IAV challenge is significantly compromised when ILCs are depleted. These findings reveal a previously unappreciated beneficial role of memory CD4 T cells in regulating tissue homeostasis during recall responses against pathogens through the regulation of ILC subsets.

## **Biography**

Tara M Strutt has received her PhD from the Department of Microbiology and Immunology at the University of Saskatchewan. Her studies focused on the signals required to activate CD4 T cells during immune responses. She continued to study CD4 T cells during her Postdoctoral studies at the Trudeau Institute in the laboratory of Dr. Susan L. Swain. While at the Trudeau Institute, she uncovered novel protective functions mediated by memory T cells during influenza virus infection and that different protective functions are mediated by memory cells in different organs. In 2010, she relocated to the University of Massachusetts Medical School as an Instructor, Faculty Member of the Department of Pathology. Her research focus centered on understanding how the tissue environment dictates T cell function and on defining how the adaptive immune system can control innate inflammatory responses. In 2015, she started her own independent research laboratory within the Immunity and Pathogenesis Division of the Burnett School of Biomedical Sciences in the College of Medicine at the University of Central Florida where she is continuing to study how adaptive immune responses regulate innate immunity.

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