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Changing the cytokine environment: Implications in mycobacterial immunity for adults and newborns

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Tuberculosis remains a major threat to global public health. The primary target cells for Mycobacterium tuberculosis (MTB) are macrophages of the lung. Human macrophages produce interleukin (IL)-27 following infection by MTB. Interestingly, IL-27 has been demonstrated to be involved in driving commitment of naïve T cells to a Th1 phenotype but also suppression of inflammatory responses. The goal of our work is to understand human macrophage responses to IL-27 and their impact during tuberculosis. IL-27 opposes macrophage-mediated control of MTB. Neutralization of IL-27 limits bacterial growth in human macrophages through a mechanism that is dependent on IFN-γ, TNF-γ, and IL-18 production. These cytokines interact to regulate expression and responsiveness of one another. Furthermore, in conjunction with IL-27 during infection these cytokines alter the macrophage gene expression profile and activation phenotype. We have also uncovered novel mechanisms by which the cytokine environment influences mycobacterial trafficking. We have additionally evaluated the biology of IL-27 in monocyte-derived macrophages from umbilical cord blood. IL-27 is expressed at an elevated level in these cells as compared with adult macrophages. The blockade of IL-27 in neonatal macrophages influences their stimulation of IFN-γ from CD4+ T lymphocytes in response to BCG. These findings enhance our understanding of innate immune responses that contribute to more effective control of MTB. Furthermore, they suggest that approaches to modulate expression of IL-27 in adults and newborns may have important implications in susceptibility to infectious agents and vaccination responses.

Biography

Cory M. Robinson completed his Ph.D in Microbiology from Miami University in 2004. This was followed by postdoctoral studies at the Uniformed Services University of the Health Sciences under the direction of Dr. Alison O'Brien and at the University of Pittsburgh School of Medicine under the direction of Jerry Nau. He is currently an Assistant Professor at the University of South Carolina School of Medicine.

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