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Influenza virus targets innate and adaptive immune cells, suppressing the immune response to homologous and heterologous antigens

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Influenza is one of the leading causes of morbidity and mortality worldwide, with the WHO estimating around 3 to 5 million serious cases of influenza globally, resulting in 250,000-500,000 deaths annually. Secondary infections further complicate influenza's morbidity and mortality, and significantly factored into the severity of the 1918 and more recent 2009 pandemics. The most common co-infections are bacterial, leading to bacterial pneumonia, though viral secondary infections may also occur. Previous studies have shown that influenza can target innate responses and damage affected tissues, opening the door for secondary infection. In this study we show that influenza virus targets not only innate immune responses but also the adaptive response specifically activated B cells, T cells and NKT cells. Finally, we explored the mechanism by which the virus targets these cells to suppress the immune responses to both homologous and heterologous antigens - potentially leaving the host more susceptible to co-infection.

Biography

Caitlin D Bohannon has received her PhD from Emory University in 2016 and is currently a Postdoctoral Fellow at the Centers for Disease Control and Prevention in Atlanta, Georgia. She has published several articles and works to expand our currently understanding of the role of adaptive immunity in influenza infection, as well as how the virus evades the immune response.

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