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## Systems biology studies to dissect the regulatory mechanisms of antiviral immunity

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The innate immune system is necessary to protect the host against infection; however, when not properly regulated, it can lead to inflammatory disease. Consequently, diverse and potent regulatory mechanisms have evolved to enable tight control over the protective innate immune responses. Our systems biology studies of antiviral immunity revealed that antiviral immune responses are controlled by gene regulatory circuits. In these circuits, dynamic interplay of transcription and epigenetic factors determines the temporal dynamics and expression profile of critical antiviral effectors and thus ensures appropriate host response to invading pathogens. Importantly, viral pathogens, including various influenza viruses, are equipped with a wide range of virulence factors to subvert host protective responses and to establish a niche for their survival and proliferation. Viral virulence factors dynamically interact with host transcription factors and chromatin remodeling proteins to rewire and reprogram host gene regulatory circuits. Elucidating the mechanisms and consequences of influenza virus-induced reprogramming of host gene regulatory networks is critically important for the discovery of novel antiviral therapies and rational vaccine design and development.

## **Biography**

Vladimir Litvak, PhD is an Assistant Professor in the Department of Microbiology and Physiological Systems at the UMass Medical School. He received his PhD from the Weizmann Institute of Science and completed the Post-doctoral training at the Institute for Systems Biology in the laboratory of Dr. Alan Aderem. His primary research goals are directed toward understanding the transcriptional and epigenetic mechanisms that regulate the development and function of macrophages in health and disease. The Litvak Lab employs a systems biology approach to define molecular pathways that govern the functional role of macrophages in virus-host interactions and in the pathogenesis of inflammatory disorders.

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