``TRIMming the host interferon response: the role of ubiquitination in ZAP antiviral mechanism``

Research / Bio
My lab studies the host innate immune response to medically relevant mosquito-borne viruses such as alphaviruses and flaviviruses. Cellular sensing of viral pathogens triggers interferon (IFN) production and signaling, leading to expression of a wide array of IFN-stimulated genes (ISGs). However, the antiviral mechanisms of many of these ISGs are not fully characterized. In particular, zinc finger antiviral protein (ZAP) targets diverse viruses by recognition of CG dinucleotide, recruitment of cellular RNA decay machineries, and suppression of viral translation. We found that ZAP co-opts the host ubiquitination pathway to block viral translation, and some alphaviruses have evolved strategies to evade ZAP. Understanding how cellular and viral processes modulate ISG antiviral function will shed light on the molecular mechanisms driving viral virulence and pathogenesis.

Publications
Emily Yang, and Melody M. H. Li All About the RNA: Interferon-Stimulated Genes That Interfere With Viral RNA Processes Frontiers in Immunology. Dec 2020
