RNA interference (RNAi) has generated much excitement as a mechanism of gene regulation and as a laboratory tool for experimental gene silencing. In plants and insects, RNAi is a primary antiviral defense response, recognizing harmful nucleic acid and silencing its expression. In mammals, there are conflicting views as to whether RNAi represents a meaningful antiviral defense. In this work, we present data that address the role of RNAi in cultured mammalian cells. Specifically, we probe the interactions of host and viral RNAi machinery and/or effectors during infection. Our results uncover unanticipated activities of the RNAi machinery that strongly suggest that RNAi is not an antiviral response in at least some types of mammalian cells. On the contrary, some mammalian viruses, including members of the herpesviruses, polyomaviruses, retroviruses and anelloviruses have evolved the ability to purposely engage the host RNAi machinery via microRNAs. An emerging model is that diverse viruses utilize both host and viral miRNAs to optimize persistent infections.

Biography

Chris Sullivan received his PhD from the University of Pittsburgh and conducted postdoctoral training at the University of California at San Francisco. He is an Associate Professor at the University of Texas at Austin where his lab focuses on understanding the role of noncoding RNAs in the regulation of virus infection and the antiviral host response. He serves on the editorial boards of prestigious virology journals, has published over 40 articles in reputed journals and has received a Burroughs Wellcome Investigator of Infectious Disease Award, an NSF Career Award and was named a Kavli Fellow.

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