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Innate defense re-direction as a mechanism to control deadly zoonotic viral infections

Zoonotic viruses enter foreign hosts without the benefit that co-adapted viruses and their respective hosts have selected. The result is that some zoonotic viruses override specific host innate defenses that would have made it possible initially for both host and virus to enjoy a long-term relationship. When this happens, the infection is dead ends as a result of the death of the host. B virus is one such example. Virus specific evasive strategies to circumvent innate defenses are common, however, using these strategies to achieve opposite effects in different species has rarely been explored to better understand zoonotic viruses. Humans infected with B virus succumb to infection in weeks in the absence of untimely treatment with a mortality rate of ~80%. Survivors suffer life-long infection with the fear of unpredictable reactivation. Strategies to control B virus in humans may become readily identified by modulating innate defenses, specifically MAPK stress pathways and PI3K activation of Akt, that play dynamic roles in B virus replication. Conclusions from experiments using macaque and human cell culture model systems have demonstrated the stark differences between innate defense engagement in natural versus foreign hosts revealing that B virus replication is controlled during natural host infection via p38 MAPK, MNK-1, and eIF4e, as well as PI3K activation of Akt. These same pathways are exploited in human cells to facilitate enhanced B virus replication, which is blocked effectively by inhibition of these pathways, while the same inhibition in macaque cells enables B virus to replicate more effectively. Learning how to control zoonotic viral infections can be advanced by identification of opposite natural versus foreign host dynamics engaged following virus entry into each susceptible host.

Biography

Julia Hilliard is a Georgia Research Alliance Eminent Scholar in Viral Immunology and Professor & Director of the Viral Immunology Center at Georgia State University in Atlanta. She received her Ph.D. at Baylor College of Medicine in Pharmacology studying the functions of DNA methylation. Her research on the immunology of herpesvirus infections began with a postdoctoral fellowship at Baylor College of Medicine focused on host responses to specific HSV-1 and HSV-2 polypeptides and glycoproteins. Subsequently, her research interests moved toward enhancing the understanding of host: pathogen relationships in zoonotic B virus infections to enable better understanding of why and how this neurovirulent simplex virus behaves so differently in foreign hosts versus natural hosts. Some of these studies resulted in the first diagnostic assay that enabled immunologic differentiation of B virus from HSV-1 and HSV-2, and subsequently, the first complete sequence of the B virus genome. More recently, her interests have included studying the roles of the innate and adaptive responses of natural and foreign hosts to deadly viruses to determine how emerging and re-emerging viruses may unexpectedly redirect, or be redirected in, foreign host defenses. These insights ultimately inform designs for therapeutic and/or protective vaccines and diagnostics for molecular control of high consequence infections.

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