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Permissive and Restrictive Replication of Primate Lentiviruses in Small Ruminant Cells

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Abstract

Investigating cross-species transmission of viruses is crucial for control of infectious diseases thereby promoting a better health and well-being to living individuals. Indeed, a better knowledge of the factors that contribute to the emergence of lethal diseases, as well as the mechanisms by which viruses adapt to new hosts, can be determinant in the prevention of future outbreaks and the development of effective therapies and vaccines. We previously demonstrated that the lack of specific Caprine Encephalitis Arthritis Virus (CAEV) receptor(s) on the surface of human cells was the only barrier that prevent CAEV of their infection. In this study, we investigated whether primate lentiviruses (LVs) can replicate in small ruminant (SR) cells. Therefore, we examined the susceptibility of four sheep and goat cell lines to the HIV-1 and SIVmac viruses. Plasmid DNAs of infectious molecular clones of HIV-1 and SIVmac were used to transfect the cell lines. The permissive human TZM-bl cell line was used to detect and titrate infectious virus produced and released in the culture medium of treated cells. To further investigate viral replication/restriction in SR cells, pseudotyped HIV-GFP and SIV-GFP with Vesicular Stomatitis Virus G glycoprotein (VSV-G) were used to overcome virus entry via cell receptors. Our findings demonstrated that as expected, neither the HIV-1 nor SIVmac was able to infect any of the SR cells. Transfection of plasmid DNAs HIV-1 and SIV-mac in SR cells, on the other hand, resulted in the production and release of high viral titers. Surprisingly, SR cells inoculated with VSV-G-HIV-GFP were productively infected, producing HIV-GFP that efficiently infected the human indictor but not SR cells. These findings indicate that the only barrier stopping the virus from infecting SR cells is the lack of a functional HIV-1 receptor on their surface. In contrast, SR cells inoculated with VSV-G-SIV-GFP failed to show any sign of infection. This indicated that in addition to the lack of a functional SIVmac receptor on the surface of SR cells, there was a post-entry restriction. This restriction occurs in the early stages of viral replication before double-stranded DNA synthesis and transport to the nucleus. These findings highlight the possibility of HIV-1 infiltrating this seemingly weak species barrier and causing infection/adaptation in small ruminants. Our study demonstrates the need of intensification of Host/pathogen interactions in the setup of cross-species infection to implement further the One Health approach of control of infectious diseases

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HIV-1; SIVmac; small ruminant cells; restriction; replication.

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