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Interactions Between Viruses and Hosts During Tick-Borne Bunyavirus Infection

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ABSTRACT

The Bunyavirales order, which includes newly and reemerging human, plant, and animal pathogens, is the biggest grouping of RNA viruses. Bunyaviruses are found throughout the world, and many of them are spread by arthropods. In hosts of diverse phylogenies, they have evolved a variety of methods to interfere with the regulatory functions of the infected cell to facilitate their own replicative cycle. The study of virus-vector interactions is gaining popularity quickly. However, studies done in mammalian systems dominate current knowledge of the cellular interaction of tick-borne bunyavirus. In this succinct overview, we summarise our current knowledge of how important cellular pathways (innate immunity, apoptosis, and RNAi responses) in mammalian or tick cells are used by tick-borne bunyaviruses to promote virus replication.

Keywords: Bunyavirus, Host

INTRODUCTION

Since the Bunyaviridae family was elevated to the Bunyavirales order in 2016 and was subsequently divided into 13 viral families by the International Committee on Taxonomy of Viruses (ICTV), the taxonomy of bunyaviruses has undergone significant change. The negative- or ambisense RNA genomes of bunyaviruses are segmented, with each segment consisting of a small, medium, and large genome segment. For all recognised bunyaviruses, these regions encode orthologous structural proteins. The nucleocapsid protein is encoded by the S segment, the virion glycoproteins are by the M segment, and the RNA-dependent RNA polymerase is by the L segment (RdRp). Bunyaviruses' genomes can also encode non-structural proteins in a positive- or negative-sense orientation on the S segment (NSs) and/or the M segments, in addition to these structural proteins.

To aid in their spread or persistence, viruses have developed to control the activation or inhibition of apoptosis. The extrinsic or intrinsic routes, which are controlled by the sequential activation of caspase proteins, are separated into two interconnected sections of the apoptotic process. Cell death results from the convergence of pathways with the activation of executioner caspases.

According to reports, tick-borne bunyaviruses can continue to infect tick cell lines. In tick cell lines generated from Ixodes ricinus (IRE/CTVM19 and IRE/CTVM20) and Hyalomma anatolicum (HAE/CTVM8 and HAE/CTVM9), respectively, the viruses UUKV and HAZV survived.

To better comprehend these bunyavirus-induced antiviral responses, in-depth investigations are still needed to better understand the mechanisms underlying tick responses to bunyavirus infection. These studies and data are crucial for understanding the nature and effects of viral infection in ticks, as well as how this affects the infectiousness, transmission, and life cycle of bunyaviruses.