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Perspective

WAYS FOR THE VIRUS TO STOP HOST CELL APOPTOSIS

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INTRODUCTION

Apoptosis is a type of cell death that can be triggered by external (external apoptosis) or internal (intrinsic apoptosis) cues. In multicellular organisms, this type of programmed cell death is essential for development and tissue homeostasis, and dysregulation is a root cause of many diseases. Members of the evolutionarily conserved B-cell lymphoma-2 (Bcl-2) family, which includes both pro- and anti-apoptotic members, control intrinsic apoptosis. Numerous viruses, including the pox virus subfamily chordopoxviridae, which is known to infect nearly all vertebrates, have also assimilated Bcl-2 genes. By mimicking the activity of their cellular counterparts, the viral Bcl-2 proteins, which are virulence factors, aid in the evasion of the immune system of the host. Structural studies have demonstrated that, despite the fact that they frequently share very little sequence identity with their cellular counterparts, viral Bcl-2 genes have nearly identical three-dimensional structures. This has been demonstrated to be essential for the survival of virus-infected cells. However, the ways in which they operate vary. The structural biology, molecular interactions, and detailed mechanism of action of poxvirus-encoded apoptosis inhibitors, as well as their impact on host–virus interactions, are the focus of this review. In the end, this makes it possible for viral infections to successfully infect and spread. A type of programmed cell death called apoptosis is triggered by either external (external apoptosis) or internal (intrinsic apoptosis) stimuli.

DISCUSSION

Apoptosis selectively removes unwanted, damaged, or pathogen-infected cells in multicellular organisms, where it plays a crucial role in development and tissue homeostasis. Apoptosis probably started out as a way to protect against pathogens. Later, it was used for other things like controlling how tissue grows during development. Subversion of apoptosis is the root cause of numerous diseases, including cancer and autoimmune disorders, despite the fact that it is essential for homeostasis and other regulatory functions. Numerous apoptotic regulatory genes have been assimilated by viruses as a result of the significance of controlling immune responses and host cell death. The viral Bcl-2 (vBcl-2) homologs, serpin protease inhibitors, dsRNA inhibitors, NF-B inhibitors, and Interferon (IFN) inhibitors that are used to manipulate apoptosis have all been captured by poxviruses in particular [1].

Bcl-2 proteins, caspases, and adaptor proteins are examples of homologous genes that regulateboth

intrinsic and extrinsic apoptosis in metazoans. The activation of cysteine aspartyl proteases (caspases), which degrade intracellular targets, controls both intrinsic and extrinsic apoptosis initiation. However, the manner in which the caspase cascade is initiated in intrinsic versus extrinsic apoptosis differs significantly. These DEDs work in a similar way with pro-caspase-8's DED to bring it into the DISC complex. In order to form the active caspase-8 homodimer, the inactive pro-caspase-8 undergoes proteolytic cleavage at the DISC, releasing the p18/p12 domain, which then proteolytically activates the executioner caspases (caspase-3, -6, and -7). Through its N-terminal DED domains, heterodimerization with the cellular FLICE (FADD-like IL-1_converting enzyme)-like inhibitory protein (cFLIP) regulates caspase-8 activation at the death receptor. Caspase-8 homodimerization, a crucial step in forming active caspase, is prevented by interaction with cFLIP, a protein that is structurally related to pro-caspase-8 but has a catalytically inactive caspase-like domain in addition to its two N-terminal DED modules [2,3].

Viruses have assimilated cFLIP-like genes to prevent pro-caspase-8 activation, which is necessary for cell survival. Active caspase-8 cleaves the cellular BH3 interacting domain (Bid) protein to activate this BH3-only protein for Bcl-2 initiated apoptosis, thereby establishing a link between intrinsic and extrinsic apoptosis. In addition, it activates the caspases. Host cells must initiate apoptosis as a first line of defense against invading pathogens. Poxviruses have developed a wide range of anti-apoptotic strategies to stop host cell suicide mechanisms because of their large genomes. In order to successfully infect hosts, poxviruses have developed both direct and indirect inhibition mechanisms of apoptosis. Viral Bcl-2-mediated mitochondrial apoptosis, inhibition of the caspase cascade, and deactivation of death receptor molecules are examples of direct apoptosis inhibition. The inhibition of dsRNA-induced apoptosis, mimicry of the Golgi anti-apoptotic protein, and Cu–Zn–SOD inhibition are all examples of indirect inhibition. The sophistication with which poxviruses target apoptosis is highlighted by these properties. There are currently insufficient direct interaction measurements or structural data to demonstrate the interaction of these molecules with essential host signaling proteins. In addition, it is evident that the viral Bcl-2 homologs inhibit apoptosis in multiple ways even when binding data are available [4-6].

CONCLUSION

There are a number of poxviral modulators of another crucial host defense system that counter interferon (IFN)-based responses. These are in addition to the numerous described poxviral encoded proteins that are used to disarm the numerous apoptosis-associated host responses to viral infection. Since these have recently been critically examined, we did not include IFN regulatory mechanisms in this review. Although recent research on the structure and interactions of anti-apoptotic proteins encoded by poxviruses has attempted to provide a more in-depth understanding of apoptosis inhibition in poxviruses, our comprehension of their modulation of host cell apoptosis remains largely incomplete.

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