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THE DOMAINS OF APOPTOSIS AND INFLAMMATION

The domains of apoptosis and inflammation

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Cell death signaling and Immune-related signaling events are mediated by dynamic protein networks created by protein interaction domains including the death domain (DD) superfamily, BIR domain, TIR domain, BH domain and CIDE domain. Viruses also have proteins containing those domains incorporated into their genomes, enabling their escape from the host immune system and death in infected cells by direct interaction with host proteins. Because those domain-mediated signaling events are associated with many human diseases, such as cancer, autoimmunity and many immune disorders, studies in these fields are of the utmost biological importance. Among the protein interaction domains, especially, the death domain (DD) superfamily is one of the largest classes. It comprises four subfamily, death domain (DD) subfamily, death effector domain (DED) subfamily, caspase recruitment subfamily (CARD), and pyrin domain (PYD) subfamily. Critical caspase activating complexes in the apoptosis and inflammation signaling pathways are assembled via the DD superfamily-mediated interactions. These domains are also involved in recruiting downstream effectors for immune cell receptor signaling, intracellular pathogen sensing, and response to DNA damage. TIR domain is involved in mediating interactions in the Toll-like receptor (TLR) and interleukin-1 (IL-1) signaling pathways. Many TIR domain-containing proteins including TLRs and adaptor proteins (MyD88, MAL, TRIF, TRAM, and SARM) play critical roles in the inflammation and innate immune response by TIR domain-mediated specific interactions.

The past decade has seen an explosion of biochemical and structural studies of proteins and protein interaction domains involved in these signaling pathways. In this special issue of "APOPTOSIS", we introduce articles that explore biochemical and structural aspects of protein interaction domains and their interactions in apoptosis, inflammation, necrosis, and immune response. In addition, we also introduce articles showing how virus proteins containing protein interaction modules similar to host proteins can be used to escape the host immune system and apoptosis process. This special issue will help us understand and summarize the Lego block-like assembly of the code involved in key cellular process such as apoptosis and inflammation.

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