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Programmed cell death and *Salmonella* pathogenesis: an interactive overview

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Programmed cell death (PCD) is the collective term for the intrinsically regulated death of cells. Various types of cell death are triggered by their own programmed regulation during the growth and development of organisms, as well as in response to environmental and disease stresses. PCD encompasses apoptosis, pyroptosis, necroptosis, autophagy, and other forms. PCD plays a crucial role not only in the growth and development of organisms but also in serving as a component of the host innate immune defense and as a bacterial virulence strategy employed by pathogens during invasion. The zoonotic pathogen *Salmonella* has the ability to modulate multiple forms of PCD, including apoptosis, pyroptosis, necroptosis, and autophagy, within the host organism. This modulation subsequently impacts the bacterial infection process. This review aims to consolidate recent findings regarding the mechanisms by which *Salmonella* initiates and controls cell death signaling, the ways in which various forms of cell death can impede or restrict bacterial proliferation, and the interplay between cell death and innate immune pathways that can counteract *Salmonella*-induced suppression of host cell death. Ultimately, these insights may contribute novel perspectives for the diagnosis and treatment of clinical *Salmonella*-related diseases.

KEYWORDS

programmed cell death, salmonella, apoptosis, pyroptosis, necroptosis, autophagy

1 Introduction

Programmed cell death (PCD) is a pervasive phenomenon in the growth and development of organisms, characterized by a genetically regulated process of active and organized cell death. Specifically, PCD involves the initiation of a protective mechanism in response to cytokine stimulation from either the internal or external environment. This mechanism entails the activation of gene coding and protein metabolism. Initially, apoptosis was acknowledged as the prevailing form of cell death in the early stages of PCD research, while necrosis was predominantly viewed as an incidental consequence of external stimuli leading to cell death. Necrosis is commonly defined as the uncontrolled death of a cell, typically occurring after a significant injury, leading to the release of cellular contents into neighboring tissues and subsequent harm (D'Arcy, 2019). As investigations into this genetically regulated, self-contained, and organized form of cell death progress, an increasing number of cell death modalities have been unveiled. In the context of pathogenic bacterial infections, these cell deaths are commonly classified into various prevalent forms of PCD, such as apoptosis, pyroptosis, necroptosis, autophagy, and ferroptosis, based on discernible morphological alterations, essential biochemical factors, immune pathway components, and effector proteins (Table 1).

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