



Autophagy: Chapter 9. Role of Autophagy in Cellular Defense Against Inflammation

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Innate immune responses provide the first line of defense against invading pathogens or damaged self-molecules. However, both dysregulation and hyperactivation of immune responses are linked to inflammatory or autoimmune diseases. Autophagy is a component of primary host defense that mediates elimination of pathogens, particularly intracellular microbes. Recent advances have shown that autophagy plays an important role in the regulation of inflammation, thus providing new insights into the relationship between autophagy and inflammation. During innate immune responses, nuclear factor- κ B signaling plays an important role in regulation of the autophagy pathway. Two key energy/growth sensors, the AMP-activated protein kinase and the mammalian target of rapamycin, play important roles in the autophagy-regulated signaling network connected with inflammation. Abnormalities or defects in autophagy appear to lead to pathologic inflammatory status with defective microbial killing, as in cases of immunopathologic responses in Crohn's disease, lung inflammation, sepsis, or other inflammatory diseases. Furthermore, basal autophagy can play an important dual role in the regulation of inflammasome activation, thus shaping overall immune homeostasis, and in the unconventional secretion of a leaderless cytokine interleukin-1 β . Numerous human pathologies and diseases related to inflammasome activation are impacted by defective or dysregulated autophagy. In this overview, we describe recent advances in the roles of autophagy in the regulation of chronic inflammatory conditions and pathological reactions in autoimmune diseases.

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