Understanding how mitophagy regulates innate immune responses triggered by mitochondrial stress

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Video Byte

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Abstract

Mitochondrial stress is a key trigger of innate immune responses. Sources of stress include environmental changes, genetic mutations, and pathogenic infection. Mitochondria respond by releasing mitochondrial DAMPs and cytochrome c into the cytosol that induce inflammation and apoptosis through activating inflammasomes, cGAS and apoptotic caspases. One way cells manage mitochondrial stress is by eliminating dysfunctional mitochondria, a process known as “mitophagy.” Mitophagy regulatory pathways are classified as ubiquitin (Ub)-dependent or Ub-independent (receptor-dependent). Growing evidence shows that mitophagy can be induced by certain bacteria and viruses. Co-opting the mitophagy process enables these pathogens to evade hosts’ immune defense. Much remains to be learned about the mechanisms that pathogens employ to hijack host mitophagy. Understanding these mechanisms could point to new therapeutic strategies for fighting infection and related diseases.