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Autophagy: A Lysosome-Dependent Process with Implications in Cellular Redox Homeostasis and Human Disease.

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Abstract

SIGNIFICANCE: Autophagy, a lysosome-dependent homeostatic process inherent to cells and tissues, has emerging significance in the pathogenesis of human disease. This process enables the degradation and turnover of cytoplasmic substrates via membrane-dependent sequestration in autophagic vesicles (autophagosomes) and subsequent lysosomal delivery of cargo. Recent Advances: Selective forms of autophagy can target specific substrates (e.g., organelles, protein aggregates, and lipids) for processing. Autophagy is highly regulated by oxidative stress, including exposure to altered oxygen tension, by direct and indirect mechanisms, and contributes to inducible defenses against oxidative stress. Mitochondrial autophagy (mitophagy) plays a critical role in the oxidative stress response, through maintenance of mitochondrial integrity.

CRITICAL ISSUES: Autophagy can impact a number of vital cellular processes including inflammation and adaptive immunity, host defense, lipid metabolism and storage, mitochondrial homeostasis, and clearance of aggregated proteins, all which may be of significance in human disease. Autophagy can exert both maladaptive and adaptive roles in disease pathogenesis, which may also be influenced by autophagy impairment. This review highlights the essential roles of autophagy in human diseases, with a focus on diseases in which oxidative stress or inflammation play key roles, including human lung, liver, kidney and heart diseases, metabolic diseases, and diseases of the cardiovascular and neural systems.

FUTURE DIRECTIONS: Investigations that further elucidate the complex role of autophagy in the pathogenesis of disease will facilitate targeting this pathway for therapies in specific diseases. Antioxid. Redox Signal. 00, 000-000.

KEYWORDS: autophagy; lysosome; mitophagy; oxidative stress; selective autophagy

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