

Autophagy: Chapter 5. Ubiquitin and p62 in Selective Autophagy in Mammalian Cells

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Macroautophagy is mainly considered to be a mechanism for the bulk degradation of the cytoplasm in times of nutrient depletion. However, growing evidence suggests that macroautophagy is more substrate-specific than originally considered. Numerous cytosolic components are selectively degraded by macroautophagy, including aggresomes, damaged mitochondria, peroxisomes, ribosomes, midbodies, and bacteria and viruses. Although the specific molecular components may differ for each substrate, the general mechanism of selective macroautophagy involves the targeted ubiquitination of the substrate and the recruitment of autophagy receptors. Autophagy receptors are proteins that act as an interface between the substrate and the nascent autophagosome, the double-membrane structure that sequesters the cytoplasm for delivery to lysosomes for degradation. In this chapter we will describe the general mechanism of selective autophagy in the mammalian system, focusing on the most described autophagy receptor, p62. The emerging data suggest that selective autophagy is not only necessary for cell survival during nutrient starvation, but also plays a critical role in cell development, cellular responses to oxidative stress, and innate immunity.

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