Autophagy: Chapter 2. Mechanisms of Regulation of p62 in Autophagy and Implications for Health and Diseases

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Autophagy refers to bulk degradation processes responsible for the turnover of long-lived proteins, disposal of damaged organelles, and clearance of aggregate prone proteins. Aberrant autophagy causes the formation of cytoplasmic inclusion bodies, leading to liver injury and neurodegeneration. However, details of abnormalities related to impaired autophagy are largely unknown. The efficiency of the autophagy pathway relies on cargo receptors to identify the ubiquitinated targets destined for the degradation pathway. For instance, p62 promotes the formation of protein aggregates and their association with the autophagosome. Recent studies showed that murine p62 contains a highly conserved LC3 recognition sequence (LRS). Structural analysis of the LC3–LRS complex revealed an interaction between Trp340 and Leu343 of p62 and two hydrophobic pockets (hp1 and hp2) on the ubiquitin fold of LC3. The LRS motif of NBR1, autophagy receptor, presents differences to this classical LRS motif with a tyrosine residue and an isoleucine residue substituting Trp and Leu, respectively. NMR studies of NBR1–LRS complexed with GABARAPL, another Atg8 homologue, indicated that the presence of tryptophan residue in the LRS motif increases the binding affinity, but other substitutions have little effect on the binding affinity due to enthalpy–entropy compensation. The aforementioned results indicate that each autophagic receptor has a unique interaction form. Most recently, it has been demonstrated that the selectivity of the autophagy receptor NDP52 for LC3C is crucial for innate immunity. Other than those listed above, many autophagy receptors and Atg8 homologue binding proteins are reported. In vivo experiments showed that cells expressing p62 mutants lacking LC3 binding ability accumulate ubiquitin-positive inclusion bodies, instead of autophagosomes, as in hepatitis and neurodegenerative diseases. These data demonstrate that cellular levels of p62 are tightly regulated by autophagy through direct interaction with LC3, and that selective turnover of p62 via autophagy prevents inclusion body formation.

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