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New Advance in Chaperone-Mediated Autophagy

Guest Editor:

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Message from the Guest Editor

Dear Colleagues,

Chaperone-mediated autophagy (CMA) is a selective proteolytic pathway in the autophagic lysosomal protein degradation system. CMA substrates are delivered to a lysosomal CMA receptor, lysosome-associated membrane protein 2A (LAMP2A) and transported to lysosomal lumen via a translocon complex formed by oligomerization of LAMP2A. CMA is considered to be related to the maintenance of intracellular protein homeostasis. Indeed, CMA has been focused as the regulator of physiological functions and disease pathogenesis. In various organs, the lysosomal protein degradation in kidney is mainly mediated by CMA, because macroautophagy is not active in kidney tissues. Regarding diseases, impairment of CMA participates in the pathogenesis of Parkinson's disease, because CMA is mainly involved in the degradation of α-synuclein, which is highly accumulated in Lewy bodies found in affected neurons of Parkinson's disease patients. In addition, Hsc70 and LAMP2A are decreased in lymphocytes and tissues of Parkinson's disease patients.

This special issue focuses on the recent advances on the roles and mechanisms of CMA.

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