Overview of the Minireviews on Autophagy

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After coining the term ‘autophagy’ by Dr. Christian De Duve in 1967 during a study using glucagon-perfused liver (Deter and de Duve, 1967), researches on autophagy was slow. The discovery of autophagy genes using a yeast model revolutionized the field and ushered into the era of molecular autophagy (Noda et al., 1995). In recognition of this discovery, Yoshinori Ohsumi was awarded the Nobel Prize in Physiology or Medicine 2016. Because of extensive effort and contribution by other investigators, we now know that autophagy is indeed critical for the maintenance or rejuvenation of cellular organelles and for energy homeostasis from yeast to human. Thus, many aspects of physiological or pathological phenomena are influenced by autophagy and its dysregulation. Dysregulated autophagy appears to participate in the development of multitudes of diseases such as neurodegeneration, cancer, metabolic diseases, cardiovascular diseases, inflammatory disorders, and aging.

Considering enormous implication of autophagy in cellular physiology and in the pathogenesis of diverse diseases at the organismal level, the current minireview series was organized. Nakamura and Yoshimori (2018) summarized the role of autophagy in longevity and aging. Aging could be considered either as a physiological or pathological process, and would be affected by autophagy regardless of grouping. Shimizu (2018) reviewed the topic of non-canonical autophagy. In addition to classical Atg5/Atg7-dependent macroautophagy, several other types of autophagy have emerged, including unconventional autophagy that does not involve certain classical Atg genes. Fukuda and Kanki focused on mitophagy in yeast system, while Yoo and Jung (2018) summarized recent findings regarding mitophagy in mammalian system. Mitophagy is an example of selective autophagy that has implications in several important diseases such as Parkinson’s disease. Cho et al. (2018) reviewed another type of selective autophagy - pexophagy, autophagy of peroxisome which is critical in very-long chain fatty acids oxidation. While the role of lysosome in autophagy execution is well known, fate of lysosome after execution of autophagy is not well recognized. Chen and Yu (2018) addressed this point and discussed the molecular mechanism of autophagic lysosome reformation (Yu et al., 2010). Kim et al. (2018) reviewed the role of autophagy in the development of diabetes associated with obesity and also of human-type diabetes. Human diabetes is different from murine diabetes in that islet amyloid is found in > 90% of patients with human diabetes. Since amyloid-prone protein is a well-recognized substrate of autophagy, efficient autophagy could be important for normal islet function and viability. Namkoong et al. (2018) discussed about autophagy in obesity, and particularly focused on the reciprocal interaction between obesity and autophagy. Autophagy has a crucial role in the clearance of microbes through a specific phenomenon named xenophagy. Kwon and Song (2018) reviewed interaction between autophagy machinery and bacterial products from the structural perspective.

These minireviews illustrate recent progress in understanding of the molecular mechanism of autophagy, and also potential application of this knowledge to the development of autophagy modulators that can be employed for treatment of the aforementioned diseases. Since many investigators and pharmaceutical or biotech companies are making great efforts to develop such autophagy enhancers or blockers,
depending on the target diseases or processes, novel therapeutic agents that modulate autophagy will become a reality in the near future.

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