NCGG SEMINAR

"Novel insights into the molecular mechanism of age-associated decline of autophagy"

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Time and Date: at **14:15 ~ 15:15** on **21 Dec, 2018 (Fri)**

Room: **The 2nd floor Conference Hall Small in the 1st Research building, NCGG**

<Reference for Seminar>

Recent genetic studies using model organisms reveals that autophagy, an evolutionally conserved cytoplasmic degradation system is one of convergent downstream targets of different longevity pathways. However, our knowledge of the molecular mechanism regulating autophagy in this context is still limited. One the other hands, the activity of basal autophagy, has been shown to decrease with age in many organisms. Yet, the underlying mechanism is completely unknown.
By RNAi based genetic screening and transcriptome analysis using the round worm, C. elegans, we have revealed the novel interdependent bHLH transcription network essential for longevity and autophagy. The network is especially critical for switching between reproduction and survival. Moreover, we have recently identified the key autophagy regulator which shows an age-dependent increase in worms, fly and mouse tissues in an evolutionarily conserved manner. Interestingly knockdown of this regulator activates autophagy and extends lifespan in worm and fly and ameliorates several age-associated phenotypes in mice. We have also addressed the tissue specific function of this regulator and upstream possible regulatory mechanism. These observations suggest an age-dependent increase of this key autophagy regulator could be one of causes for an age-dependent autophagic decline.

1. Nakamura S. et al., Autophagy and Longevity. ***Mol. Cells***. 41, 65-72, (2018)
2. Nakamura S. et al, Mondo complexes regulate TFEB via TOR inhibition to promote longevity in response to gonadal signals. ***Nat. Commun.*** 7, 10944, (2016).

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