“FOXO and HSF-1 reverses immunosenescence in C. elegans via down-regulating insulin-like peptide endocrine signaling”

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Time and Date: at 15:30 ~ 16:30 on 21 Dec, 2018 (Fri)

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

<Reference for Seminar>

One of the hallmarks of aging is immunosenescence, a decline in immune functions, which appeared to be inevitable. Surprisingly, here we show that lifespan-extending DAF-2/insulin/IGF-1 receptor mutations drastically delay immunosenescence and even improve immunocompetence in old C. elegans. We showed that p38 mitogen activated protein kinase (PMK-1), a key determinant of immunosenescence in wild-type, was dispensable for this enhanced immunocompetence. We then found that INS-7/insulin-like peptide and ZIP-10/bZip transcription factor, which were up-regulated by aging in wild-type but not in daf-2 mutants, contributed to immunosenescence. Moreover, we showed that longevity-promoting DAF-16/FOXO and heat-shock transcription factor 1 (HSF-1) increased immunocompetence in old daf-2(-) animals by decreasing the expression of ins-7, which in turn further down-regulated insulin/IGF-1 signaling. Our study suggests that reduced insulin/IGF-1 signaling can bypass normal immunosenescence via up-regulating anti-aging transcription factors that regulate an endocrine insulin-like peptide. Since insulin/IGF-1 signaling is evolutionarily conserved across species, the strategy for immune rejuvenation may be exploited for preventing immunosenescence in humans.

1. Lee, S.J.V. et al., Prefoldin 6 mediates longevity signaling from heat shock factor 1 to FOXO in C. elegans. *Genes Dev.* (Accepted)

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