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Drosophila melanogaster as a model for studying genetics and epigenetics of aging

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We review our data on genetics and epigenetics basis of longevity on Drosophila model. Lifespan varies dramatically among species, but the biological basis is not well understood. Previous studies in model organisms revealed the importance of nutrient sensing, mTOR, NAD/sirtuins, and insulin/IGF1 signaling in lifespan control. By studying life-history traits (lifespan, length of the developmental period, imago body weight, and fecundity, response to oxidative stress, hyperthermia, starvation, and ionizing radiation) and transcriptomes of 14 Drosophila species differing more than six fold in lifespan, we explored expression divergence and identified genes and processes that correlate with longevity. We studied the life extension effect of neuronal overexpression of the Gclc gene and investigated its influence on the age-depended dynamics of transcriptome and biological functions such as fecundity, spontaneous locomotor activity and circadian rhythmicity, as well as on the resistance to oxidative, proteotoxic and osmotic stresses. It was shown that Gclc overexpression reduces locomotor activity in the young and middle age compared to control flies. Gclc overexpression slowed down the age-dependent decline of locomotor activity and circadian rhythmicity, and resistance to stress treatments. Gclc level demonstrated associations with the expression of genes involved in a variety of cellular processes including Jak-STAT, MAPK, FOXO, Notch, mTOR, TGF-beta signaling pathways, translation, protein processing in endoplasmic reticulum, proteasomal degradation, glycolysis, oxidative phosphorylation, apoptosis, regulation of circadian rhythms, differentiation of neurons, synaptic plasticity and transmission.

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