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Genes and Pathway Regulating Longevity in Model Organisms

Guest Editor:

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Message from the Guest Editor

Is aging avoidable? Can life be disentangled from aging? Although we so far have no definitive answer to these questions, Comparative Longevity (CL) has revealed significant differences between mammalian species, suggesting that minor genetic differences may be responsible for the notable differences in lifespan often observed. The example of dogs, whose size, life expectancy and susceptibility to disease can vary greatly even between breeds, suggests that few alleles may have a significant effect on longevity and age-related disease. The identification of the genes and mechanisms regulating longevity has utilized simple model organisms. Worms, yeasts, fruit flies and mice have aided in determining the role of Igf1, Tor, Ras and PI3K as major regulators of longevity and genomic instability, epigenetic derangement, the nutrient response pathway, as well as the rate of telomere shortening, which could be associated to accelerated or delayed aging. Owing to their low cost, genetic resources and limited ethical constraints, model systems can play a significant role in identifying substances, alleles, and the molecular mechanisms capable of modulating aging.



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Special Issue



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