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THE PROXIMAL CAUSES OF HUMAN AGING: RANDOM MOLECULAR DAMAGE OR HYPERFUNCTION OF THE DEVELOPMENTAL PROGRAMS?

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Summary

Currently, there are two main theoretical approaches to understanding of the ultimate causes of human senescence. These are deterministic views and stochastic models. Proximate theories of aging constitute a distinct group of conceptions, and they involve mechanistic causes of aging. However, recent experimental studies carried out on evolutionarily distant model organisms have shown that aging can be a consequence of evolutionarily programmed and conserved signaling pathways, including insulin/IGF-1 pathway and MTOR (mechanistic target of rapamycin), and does not result primarily from random accumulation of molecular damage. Based on this, an alternative and interesting theory of hyperfunction has been recently formulated, including the important "quasi-program" described by the "MTOR-centric" model of aging, rival to the disposable soma theory, and offering a completely different approach to numerous problems and paradoxes in current biogerontology, as well as allowing the prediction of entirely new relationships. The aim of the article is to present and compare the views of both parties in the dispute, based on the results of recent experimental biogerontological studies and the contemporary knowledge of selected major aspects of human aging and longevity, including findings on the relationship between body size and lifespan.