Bioregulatoren – Ultra-short Peptides – in der modernen Alternsforschung

Epigenetik, Inflammaging, mitochondriale Dysfunktion und Neurodegeneration zwischen molekularer Gerontologie und translationaler Longevity-Medizin

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Literatur

[1] Ahmed S et al. Telomerase does not counteract telomere shortening but protects mitochondrial function under oxidative stress. J Cell Sci. 2008.

[2] Blackburn EH et al. Telomeres and telomerase: the path from maize to human cancer and aging. Nat Med. 2006.

[3] Campisi J. Aging, cellular senescence, and cancer. Annu Rev Physiol. 2013.

[4] Couillard-Després S et al. Doublecortin expression levels in adult brain reflect neurogenesis. Eur J Neurosci. 2005.

[5] De Felice FG, Ferreira ST. Inflammation, defective insulin signaling, and mitochondrial dysfunction as common molecular denominators connecting type 2 diabetes to Alzheimer disease. Diabetes. 2014.

[6] Donato AJ et al. Cellular and molecular biology of aging endothelial cells. J Mol Cell Cardiol. 2011.

[7] Duewell P et al. NLRP3 inflammasomes are required for atherogenesis. Nature. 2010.

[8] Franceschi C et al. Inflammaging and 'Garb-aging'. Trends Endocrinol Metab. 2018.

[9] Harman D. Aging: a theory based on free radical and radiation chemistry. J Gerontol. 1956.

[10] Hardy J, Selkoe DJ. The amyloid hypothesis of Alzheimer's disease: progress and problems. Science. 2002.

[11] Heneka MT et al. NLRP3 is activated in Alzheimer's disease and contributes to pathology. Nature. 2013.

[12] Horvath S. DNA methylation age of human tissues and cell types. Genome Biol. 2013.

[13] Horvath S, Raj K. DNA methylation-based biomarkers and the epigenetic clock theory of ageing. Nat Rev Genet. 2018.

[14] Imai SI, Guarente L. NAD+ and sirtuins in aging and disease. Trends Cell Biol. 2014.

[15] Kennedy BK et al. Geroscience: linking aging to chronic disease. Cell. 2014.

[16] Kim EK, Choi EJ. Pathological roles of MAPK signaling pathways in human diseases. Biochim Biophys Acta. 2010.

[17] Khavinson VK et al. Mechanism of biological activity of short peptides. Biol Bull Rev. 2013.

[18] Khavinson VK, Anisimov VN. Peptide bioregulation of aging: results and prospects. Biogerontology. 2010.

[19] Khavinson, V.; Linkova, N. et al. EDR Peptide: Possible Mechanism of Gene Expression and Protein Synthesis Regulation Involved in the Pathogenesis of Alzheimer's Disease. Molecules 2021,

[20] Kirkland JL, Tchkonia T. Senolytic drugs: from discovery to translation. J Intern Med. 2020.

[21] Longo VD, Mattson MP. Fasting: molecular mechanisms and clinical applications. Cell Metab. 2014.

[22] López-Otín C et al. The Hallmarks of Aging. Cell. 2013.

[23] Madamanchi NR, Runge MS. Mitochondrial dysfunction in atherosclerosis. Circ Res. 2007.

[24] Mattson MP. Apoptosis in neurodegenerative disorders. Nat Rev Mol Cell Biol. 2000.

[25] Mattson MP, Arumugam TV. Hallmarks of brain aging. Cell Metab. 2018.

[26] Ocampo A et al. In vivo amelioration of age-associated hallmarks by partial reprogramming. Cell. 2016.

[27] Ota H et al. Sirt1 and vascular aging. Circ Res. 2007.

[28] Scarpulla RC. Metabolic control of mitochondrial biogenesis. Biochim Biophys Acta. 2011.

[29] Selkoe DJ, Hardy J. The amyloid hypothesis of Alzheimer's disease. EMBO Mol Med. 2016.

[30] Sinclair DA, Guarente L. Small-molecule allosteric activators of sirtuins. Annu Rev Pharmacol Toxicol. 2014.

[31] Shay JW, Wright WE. Telomeres and telomerase: three decades of progress. Nat Rev Genet. 2019

[32] Sun N et al. The mitochondrial basis of aging. Mol Cell. 2016.

[33] Tchkonia T et al. Cellular senescence and the senescent secretory phenotype. J Clin Invest. 2013.

[34] Ungvari Z et al. Mechanisms of vascular aging. Circ Res. 2018.