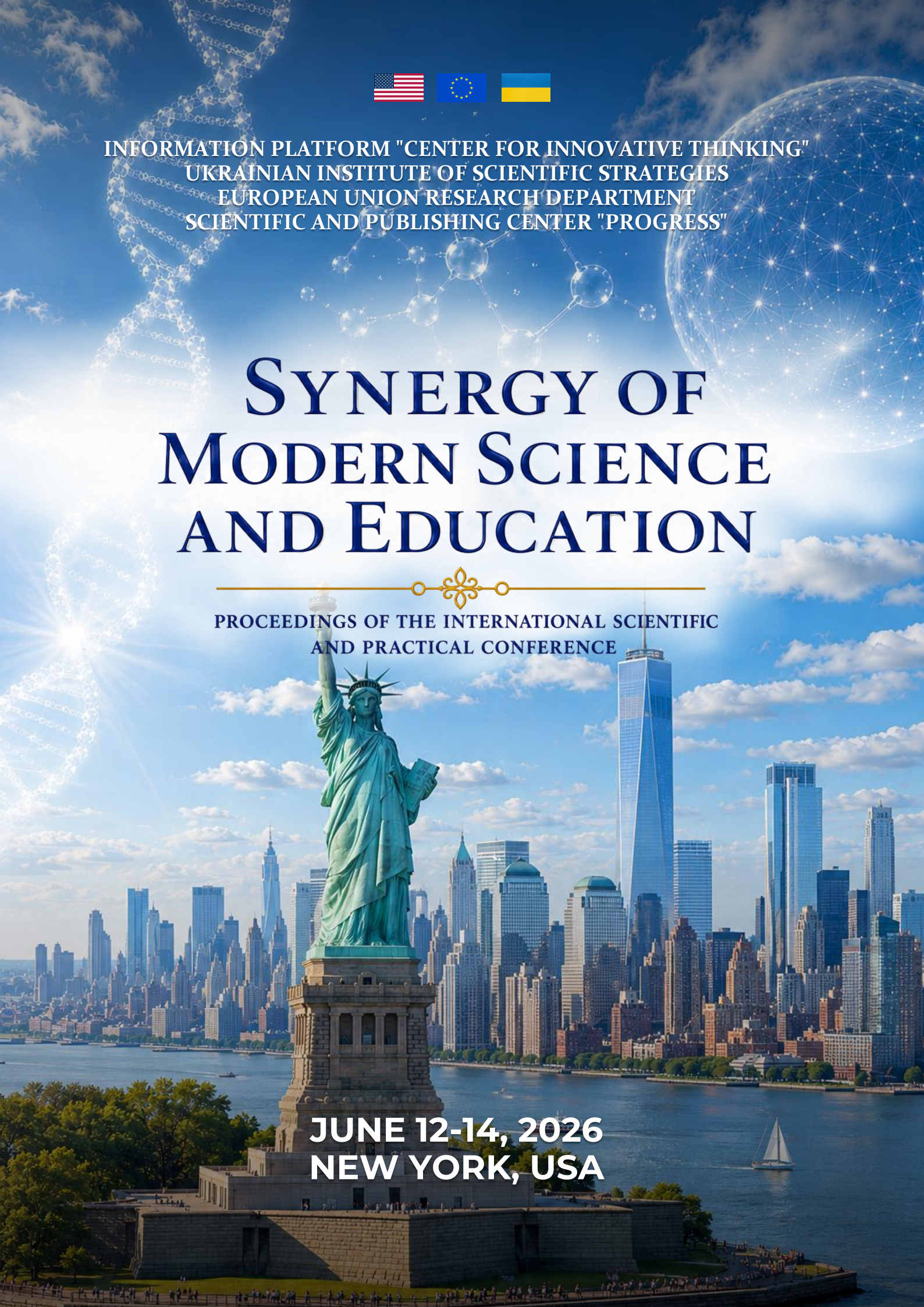

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SYNERGY OF MODERN SCIENCE AND EDUCATION

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THE ROLE OF EARLY-LIFE MICROBIOTA PROGRAMMING AND AGE-RELATED NEUROENDOCRINE HOMEOSTASIS REMODELING IN THE MODULATION OF COGNITIVE AGING

Abstract. The role of early-life microbiome programming and age-related neuroendocrine homeostasis remodeling in the development of cognitive aging is considered. Current data on the influence of the gut–brain axis, intestinal microbiota, neuroinflammation, insulin resistance, and hormonal changes on neuroplasticity, the functional state of the central nervous system, and the development of neurodegenerative processes are summarized. It is shown that early dysbiosis, impairment of the intestinal barrier function, chronic low-grade systemic inflammation, and age-related deficiency of neuroprotective hormones may represent important factors contributing to cognitive decline and the acceleration of neurodegenerative changes during aging.

Keywords: gut microbiome, gut–brain axis, cognitive aging, neuroendocrine homeostasis, neuroinflammation, hypothalamic–pituitary–adrenal axis, neurodegeneration, insulin resistance, neuroplasticity.

The concept of early-life microbiota programming suggests that the pattern of primary intestinal colonization in infants under the influence of breastfeeding determines the architectonics and maturation of synapses in the central nervous system through the gut–brain axis [1, 2].

The microbiome is considered a complex metabolically active system capable of modulating immune, endocrine, and neural regulation. Of particular interest is the concept of the gut–brain axis, which describes the bidirectional interaction between the central nervous system and the intestinal microbiota through neural, humoral, and immune mechanisms [3, 4].

A key role in the modulation of cognitive status is played by bacterial metabolites, including short-chain fatty acids, tryptophan derivatives, bile acids, neurotransmitter-like compounds, and antioxidant polyphenol metabolites, which influence neuroinflammation, neuroplasticity, barrier functions, and brain energy metabolism. Age-related changes in the intestinal microbiota, accompanied by reduced microbial diversity and the development of chronic low-grade inflammation (inflammaging), may represent one of the pathogenetic factors contributing to cognitive decline in older individuals [2].

The composition of the microbiome in early life modulates the expression of genes responsible for the stress resilience of the hypothalamic–pituitary–adrenal (HPA) axis, thereby protecting the brain from premature aging. Alterations in the microbiome composition during early life, particularly due to antibiotic therapy, formula feeding, infections, or chronic stress, may affect HPA axis activity and increase cortisol secretion in response to stress stimuli. This is accompanied by changes in neurotransmitter metabolism, neuroinflammation, and impaired expression of genes associated with stress resistance and neuroplasticity, including brain-derived neurotrophic factor (BDNF). As a result, the risk of anxiety, depressive, and stress-related disorders later in life increases [5].

In addition, early dysbiosis may have long-term consequences for brain functional status, since alterations in the interaction between the microbiota and the HPA axis can influence cognitive functions,

emotional regulation, and the vulnerability of the nervous system to age-related neurodegenerative changes [5].

The concept of inflammaging is considered one of the key mechanisms underlying age-related diseases. Chronic low-grade systemic inflammation resulting from age-associated changes in immune, metabolic, and neuroendocrine regulation contributes to the development of neurodegenerative, cardiovascular, and metabolic disorders. Age-related dysbiosis may enhance pro-inflammatory processes, impair intestinal barrier function, and promote the progression of inflammaging, whereas a balanced microbiome is associated with reduced systemic inflammation and healthier aging [6].

Chronic systemic inflammation and increased intestinal barrier permeability contribute to the activation of microglia, the principal immune cells of the central nervous system. Prolonged microglial hyperactivation is accompanied by the development of neuroinflammation, neuronal damage, reduced neuroplasticity, and acceleration of age-related cognitive impairment [7].

Age-related remodeling of the endocrine system (neuroendocrine homeostasis) significantly affects cognitive functions [8, 9]. Chronically elevated cortisol levels in older individuals are associated with reduced hippocampal volume, a brain structure that plays a key role in memory, learning, and emotional regulation. Prolonged hyperactivation of the hypothalamic–pituitary–adrenal axis is accompanied by cognitive decline, impaired neuroplasticity, and disturbances in memory formation. Excessive glucocorticoid secretion is considered one of the mechanisms underlying age-related hippocampal neuronal damage. Chronic stress and impaired neuroendocrine regulation may contribute to suppressed neurogenesis, increased neuronal vulnerability to degenerative changes, and accelerated cognitive aging of the brain [10].

Impaired insulin signaling and reduced insulin sensitivity in brain tissues lead to deficient neuronal energy supply and contribute to the development of neurodegenerative processes. Alzheimer's disease is considered a form of "type 3 diabetes," characterized by insulin resistance, impaired glucose utilization, oxidative stress, and mitochondrial dysfunction within the central nervous system.

These disturbances are accompanied by the accumulation of β-amyloid and hyperphosphorylated tau protein, which are key molecular markers of Alzheimer's disease. Dysregulation of insulin signaling is also associated with the development of neuroinflammation, reduced neuroplasticity, cognitive decline, and the progression of age-related neurodegenerative changes [11, 12].

A decline in sex hormone and melatonin levels diminishes their neuroprotective effects, thereby accelerating cognitive deterioration. Estrogens play an important role in maintaining the structural and functional integrity of the brain. They are involved in the regulation of neuroplasticity, synaptic transmission, neuronal energy metabolism, and neurogenesis, and also exert antioxidant and neuroprotective effects. Estrogens are particularly important for the functioning of the hippocampus and cerebral cortex, brain regions associated with memory and cognitive functions.

Age-related decline in estrogen levels during menopause is associated with impaired cognitive functions, reduced neuroplasticity, and increased neuronal vulnerability to degenerative changes. Estrogen deficiency is considered one of the factors contributing to the development of neurodegenerative processes and the acceleration of cognitive brain aging [13].

Testosterone plays an important role in maintaining cognitive functions, neuroplasticity, and the normal functioning of the central nervous system during aging. Age-related reduction in testosterone levels is associated with deterioration of memory, attention, and executive functions, as well as with an increased risk of cognitive impairment and neurodegenerative processes.

Testosterone exerts neuroprotective effects through the regulation of synaptic plasticity, neuronal energy metabolism, antioxidant defense, and reduction of neuroinflammation. Testosterone replacement therapy in elderly men may positively influence certain cognitive functions; however, the effectiveness of this approach depends on age, baseline hormonal status, and the severity of cognitive impairment [14].

Melatonin is considered a multifunctional regulatory molecule involved in the coordination of circadian rhythms, neuroendocrine regulation, immune responses, and cellular metabolic processes. Melatonin has been shown to exert pronounced antioxidant, anti-inflammatory, and neuroprotective effects, contributing to the stabilization of mitochondrial function, reduction of oxidative stress, and protection of neurons against damage.

Age-related decline in melatonin production is associated with disturbances in circadian regulation, enhanced neuroinflammation, impaired neuroplasticity, and increased vulnerability of the nervous system to degenerative processes. Melatonin is regarded as one of the important factors supporting cognitive functions and slowing age-related neurodegenerative changes due to its ability to regulate apoptosis, oxidative balance, and cellular energy metabolism [15].

Thus, current evidence indicates a close relationship between early-life microbiome programming, age-related remodeling of neuroendocrine homeostasis, and the processes of cognitive aging. The intestinal microbiota participates in the regulation of immune, metabolic, neuroendocrine, and neuroinflammatory mechanisms that influence neuroplasticity, the functional state of the hippocampus, and the maintenance of cognitive functions throughout life.

Age-related dysbiosis, chronic systemic inflammation, impaired insulin signaling, hyperactivation of the hypothalamic–pituitary–adrenal axis, and deficiency of neuroprotective hormones are considered important pathogenetic factors of neurodegenerative processes and cognitive decline. Understanding the mechanisms underlying the gut–brain axis interaction opens perspectives for the development of new preventive and therapeutic strategies aimed at maintaining cognitive health and slowing age-related changes in the nervous system.

REFERENCES:

1. Русалов В., Лагодич Т. Мікробіом кишечника як модифікатор когнітивних функцій та когнітивного старіння // Перспективи та інновації науки. 2026. № 5(63). С. 3555–3567. DOI: 10.52058/2786-4952-2026-5(63)-3555-3567.
2. Лагодич Т., Русалов В., Мельник С., Лазаренко В. Вплив грудного вигодовування на розвиток мікробіоти новонародженого // Перспективи та інновації науки. 2026. № 5(63). С. 3039–3055. DOI: 10.52058/2786-4952-2026-5(63)-3039-3055.
3. Diotaiuti P., Misiti F., Marotta G., Falese L., Calabrò G. E., Mancone S. The Gut Microbiome and Its Impact on Mood and Decision-Making: A Mechanistic and Therapeutic Review // Nutrients. — 2025. — Vol. 17, No. 21. — Article 3350. — DOI: https://doi.org/10.3390/nu17213350https://doi.org/10.3390/nu17213350
4. Miller B. C., Haggler J. A., Chaudhari D. S., et al. Gut microbiome signatures predict cognitive impairment in older cancer survivors // GeroScience. — 2025. — DOI: https://doi.org/10.1007/s11357-025-01917-xhttps://doi.org/10.1007/s11357-025-01917-x
5. Frankiensztajn L. M., Elliott E., Koren O. The microbiota and the hypothalamus-pituitary-adrenocortical (HPA) axis, implications for anxiety and stress disorders // Current Opinion in Neurobiology. 2020. Vol. 62. P. 76–82. DOI: 10.1016/j.conb.2019.12.003. PMID: 31972462.
6. Franceschi C., Garagnani P., Parini P. et al. Inflammaging: a new immune–metabolic viewpoint for age-related diseases // Nature Reviews Endocrinology. 2018. Vol. 14, № 10. P. 576–590. DOI: 10.1038/s41574-018-0059-4.
7. Thevaranjan N., Puchta A., Schulz C. et al. Age-Associated Microbial Dysbiosis Promotes Intestinal Permeability, Systemic Inflammation, and Macrophage Dysfunction // Cell Host & Microbe. 2017. Vol. 21, № 4. P. 455–466.e4. DOI: 10.1016/j.chom.2017.03.002. PMID: 28407483. PMCID: PMC5392495.
8. Лагодич Т., Русалов В. Вікові особливості обміну енергії та терморегуляції (огляд літератури) // Перспективи та інновації науки. 2025. № 1(47). С. 2297–2305. DOI: 10.52058/2786-4952-2025-1(47)-2297-2305.
9. Лагодич Т., Русалов В. Особливості фізіології центральної нервової системи та ендокринної системи у людей похилого віку (огляд літератури) // Перспективи та інновації науки. 2025. № 2(48). С. 2119–2130. DOI: 10.52058/2786-4952-2025-2(48)-2119-2130.
10. Lupien S. J., de Leon M., de Santi S. et al. Cortisol levels during human aging predict hippocampal atrophy and memory deficits // Nature Neuroscience. 1998. Vol. 1, № 1. P. 69–73. DOI: 10.1038/271. PMID: 10195112.
11. de la Monte S. M., Wands J. R. Alzheimer’s disease is type 3 diabetes–evidence reviewed // Journal of Diabetes Science and Technology. 2008. Vol. 2, № 6. P. 1101–1113. DOI: 10.1177/193229680800200619. PMID: 19885299. PMCID: PMC2769828.
12. Корильчук Н. І., Грищенко Г. В., Лагодич Т. С. Вплив кишкової мікробіоти на механізми розвитку інсулінорезистентності та підходи до її корекції препаратами про- і метабіотиків // Перспективи та інновації науки. 2026. № 5(63). С. 2951–2966. DOI: 10.52058/2786-4952-2026-5(63)-2951-2966.
13. Morrison J. H., Brinton R. D., Schmidt P. J., Gore A. C. Estrogen, menopause, and the aging brain: how basic neuroscience can inform hormone therapy in women // Journal of Neuroscience. 2006. Vol. 26, № 41. P. 10332–10348. DOI: 10.1523/JNEUROSCI.3369-06.2006. PMID: 17035515. PMCID: PMC6674699.

14. Hua J. T., Hildreth K. L., Pelak V. S. Effects of Testosterone Therapy on Cognitive Function in Aging: A Systematic Review // Cognitive and Behavioral Neurology. 2016. Vol. 29, № 3. P. 122–138. DOI: 10.1097/WNN.0000000000000104. PMID: 27662450. PMCID: PMC5079177.
15. Hardeland R., Cardinali D. P., Srinivasan V., Spence D. W., Brown G. M., Pandi-Perumal S. R. Melatonin—a pleiotropic, orchestrating regulator molecule // Progress in Neurobiology. 2011. Vol. 93, № 3. P. 350–384. DOI: 10.1016/j.pneurobio.2010.12.004. PMID: 21193011.