Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025

NEUROBIOCHEMICAL DISORDERS AND THEIR ASSOCIATION WITH DISEASES

Rayimqulov Firdavs Azimjon ogli
2nd-Year Student, Faculty of Medicine, Alfraganus University
ORCID ID: 0009-0004-0348-2962

Abdumannonova Muborakxon Abdupulat qizi
2nd-Year Student, Faculty of Medicine, Alfraganus University
Scientific supervisor: Professor, Doctor of Chemical Sciences (DSc)

Jalilov Fazliddin Sodiqovich
Head of the Department of Pharmaceutical Chemistry, Alfraganus University
ORCID ID: 0000-0001-7379-8445
Alfraganus University, Tashkent, Uzbekistan

Abstract
This article analyzes the disorders of neurobiochemical processes and their relationship with various neurological and psychiatric diseases. It is emphasized that imbalances in the level of neurotransmitters, changes in the activity of ion channels, and disorders of protein metabolism play an important role in the development of nervous system diseases. The participation of neurobiochemical mechanisms in the pathogenesis of diseases such as depression, schizophrenia, Alzheimer's, Parkinson's, and epilepsy is reviewed based on scientific literature. The importance of neurobiochemical markers in diagnosis and treatment is also highlighted.

Keywords: Neurobiochemistry, neurotransmitter, dopamine, serotonin, glutamate, GABA, parkinson's, Alzheimer's, epilepsy.

Introduction

Neurobiochemical processes play a crucial role in the human body by generating and transmitting nerve impulses, ensuring inter-neuronal communication, and maintaining the stability of brain function. Any disruption of these processes leads to significant changes in the central nervous system and underlies many neurological and psychiatric disorders [1].

The activity of the nervous system is primarily regulated by neurotransmitters such as dopamine, serotonin, glutamate, gamma-aminobutyric acid (GABA), and acetylcholine. Their excessive production or deficiency contributes to the development of various diseases. For instance, dopamine deficiency leads to Parkinson’s disease, serotonin imbalance causes depression, and glutamate excess predisposes to epilepsy [2].

In recent years, it has been revealed that a deeper understanding of neurobiochemical mechanisms can help better explain the pathogenesis of nervous system diseases and improve treatment methods. For example, in neurodegenerative disorders (Alzheimer’s, Parkinson’s), pharmacological agents have been

Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025

developed to restore neurotransmitter levels, while in mental disorders (depression, schizophrenia), drugs are applied to normalize serotonin and dopamine metabolism [3].

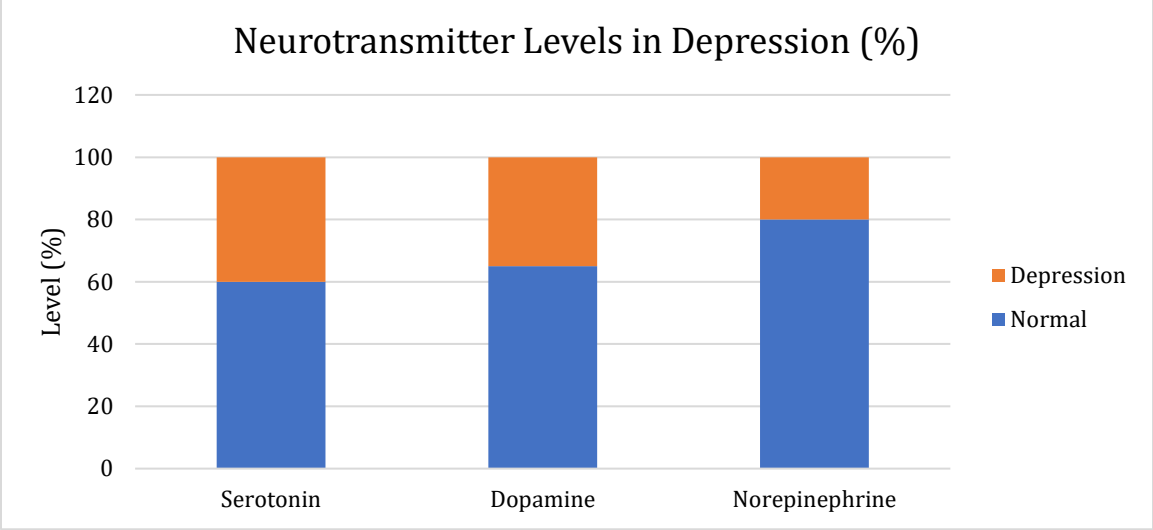
Moreover, the study of neurobiochemical markers allows for the early detection of such diseases. For example, measuring the levels of beta-amyloid and tau proteins in cerebrospinal fluid enables the early diagnosis of Alzheimer's disease [4]. This further emphasizes the importance of neurobiochemical research in clinical practice.

Therefore, the purpose of this article is to review the main mechanisms of neurobiochemical disorders and analyze their relationship with common neurological and psychiatric diseases.

Depression and Serotonin-Dopamine Imbalance

Depression is one of the most common disorders affecting mental health, with more than 280 million people worldwide diagnosed with it [5]. The monoamine hypothesis plays a leading role in its pathogenesis. According to this hypothesis, the main cause of depression is a decrease in serotonin, dopamine, and norepinephrine levels or an imbalance in their receptor activity. A reduction in serotonin is associated with low mood, sleep disturbances, and appetite changes, while dopamine deficiency leads to loss of pleasure (anhedonia) and reduced motivation [6].

In addition, modern studies show that not only monoamines but also dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis is important in the development of depression. Elevated cortisol levels, neuroinflammation, and decreased BDNF (Brain-Derived Neurotrophic Factor) are also considered part of the biochemical basis of depression [7]. Therefore, antidepressant therapy is aimed not only at normalizing serotonin and dopamine but also at restoring neurotrophic factors.



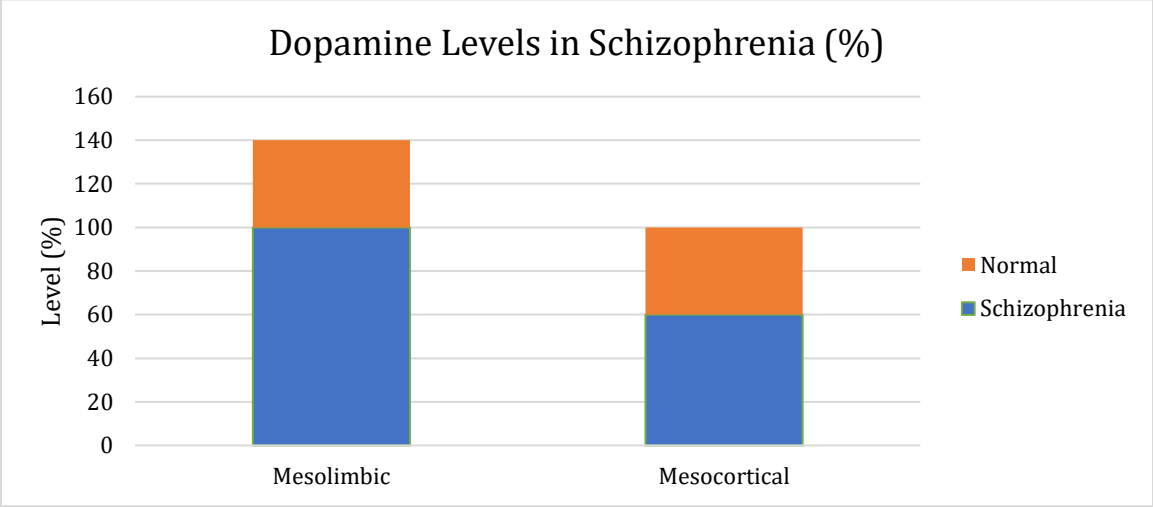
Note: The diagram illustrates the decreased levels of the main neurotransmitters — serotonin, dopamine, and norepinephrine — in the development of depression. These substances play a key role in mood regulation. Their deficiency leads to impaired memory, insomnia, reduced motivation, and general emotional instability [5][6][7].

Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025

Schizophrenia and the Dopamine Hypothesis. Schizophrenia is a chronic disorder with a complex pathogenesis, affecting more than 20 million people worldwide [8]. The classical dopamine hypothesis was proposed to explain this disease: an increase in dopamine in the mesolimbic pathway leads to positive symptoms (hallucinations, delusions), while a decrease in dopamine in the mesocortical pathway results in negative symptoms (apathy, social withdrawal).

In recent years, this hypothesis has been expanded with the glutamate hypothesis. A decrease in NMDAR (N-methyl-D-aspartate receptor) activity increases neuronal excitability and further exacerbates dopamine imbalance [9]. In addition, insufficient inhibition of GABAergic neurons contributes to enhanced cortical imbalance. Clinical observations also confirm disrupted glutamate metabolism in patients with schizophrenia [10]. These new mechanisms serve as a foundation for developing future therapeutic agents targeting the glutamate and GABA systems in the treatment of schizophrenia.



Note: The diagram illustrates the dual disruption of the dopamine system in schizophrenia: increased dopamine in the mesolimbic pathway — leading to positive symptoms (hallucinations, delusions), and decreased dopamine in the mesocortical pathway — leading to negative symptoms (apathy, social withdrawal). In addition, disturbances in the glutamate system are also associated with the pathogenesis of schizophrenia [8][9][10].

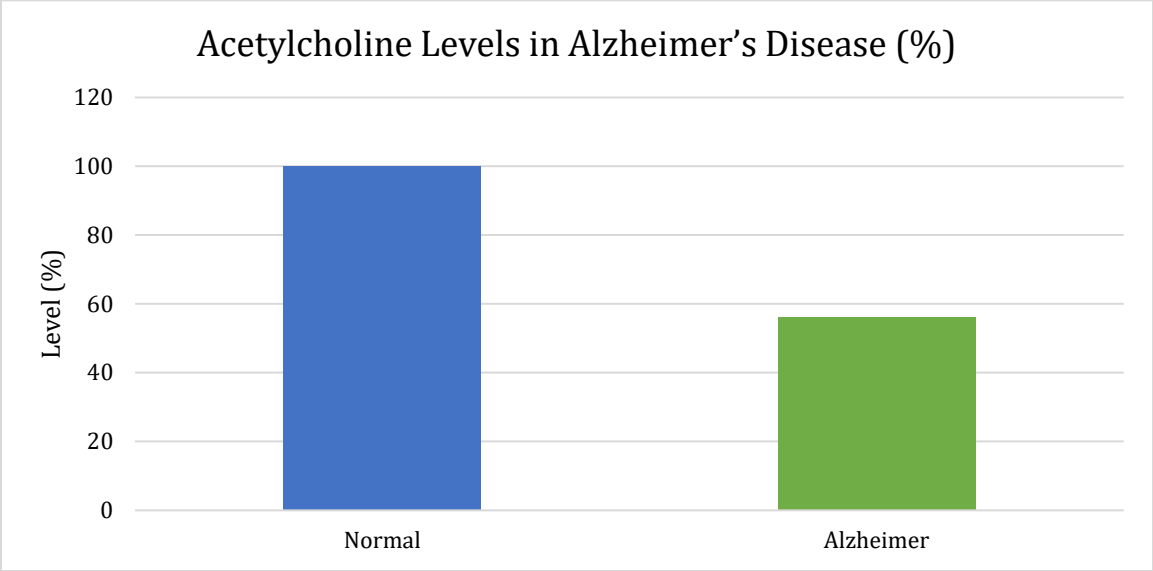
Alzheimer's Disease and Cholinergic Dysfunction. Alzheimer's disease is the most common type of dementia worldwide, affecting more than 55 million people globally [11]. Several important processes underlie the neurobiochemical basis of this disease. One of the main hypotheses is the cholinergic hypothesis, which suggests that degeneration of cholinergic neurons in the cerebral cortex and hippocampus leads to impaired cognitive functions. A reduction in acetylcholine levels results in disturbances in attention, memory, and learning processes. For this reason, acetylcholinesterase inhibitors (donepezil, rivastigmine, galantamine) are widely used in clinical practice [12].

In addition, the amyloid hypothesis and tau hypothesis are also common. The accumulation of beta-amyloid peptides in the extracellular space forms "amyloid plaques," which cause neuronal death.

Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025

Hyperphosphorylation of tau proteins leads to neurofibrillary tangles. These two mechanisms are considered the main pathological hallmarks of the disease and are also used as diagnostic markers [13].



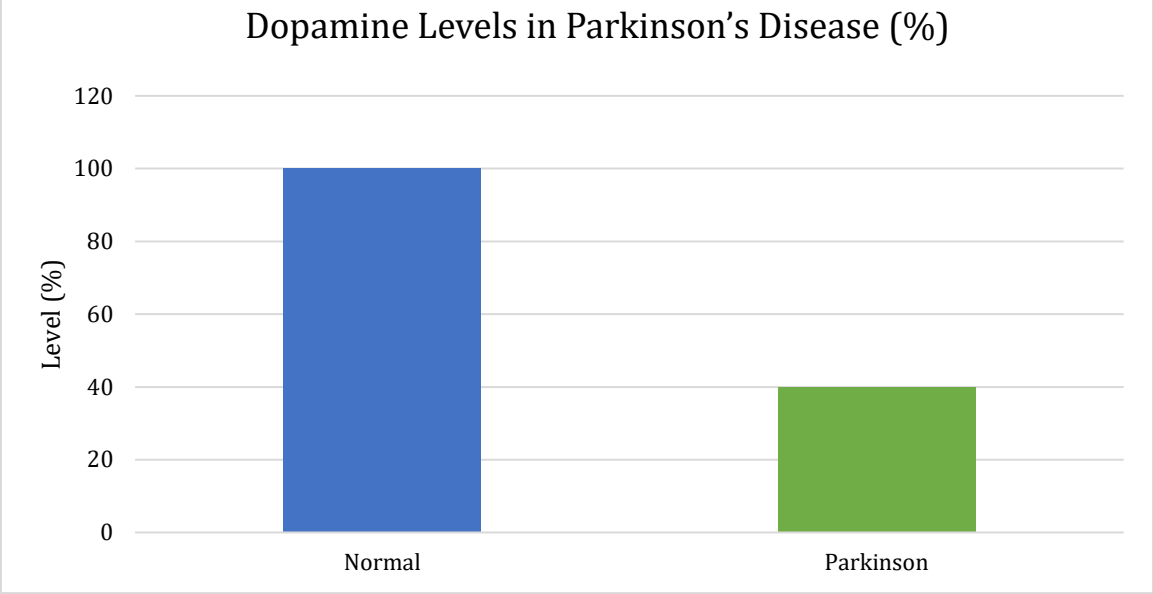
Note: The diagram shows the decrease in acetylcholine levels in Alzheimer's disease and the resulting cognitive impairments (memory loss, speech difficulties, problems with reading and reasoning). It also illustrates the accumulation of beta-amyloid plaques and tau proteins, which contribute to neuronal degeneration and synaptic disruption [11][12][13].

Parkinson's Disease and Dopamine Deficiency. Parkinson's disease is associated with the degeneration of dopamine-producing neurons in the substantia nigra and predominantly occurs after the age of 60. Globally, more than 10 million people are affected by this disorder [14]. Dopamine deficiency disrupts the neuronal circuits of the basal ganglia, clinically manifesting as tremors, muscle rigidity, and bradykinesia.

At the neurobiochemical level, reduced dopamine also disturbs the balance of other neurotransmitters. For example, increased glutamate activity and GABA system imbalance further impair motor function. In clinical treatment, levodopa remains the primary medication; however, long-term use may induce dyskinesias. Therefore, dopamine agonists, MAO-B inhibitors, and glutamate receptor antagonists are also used in combination therapy [15].

Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025



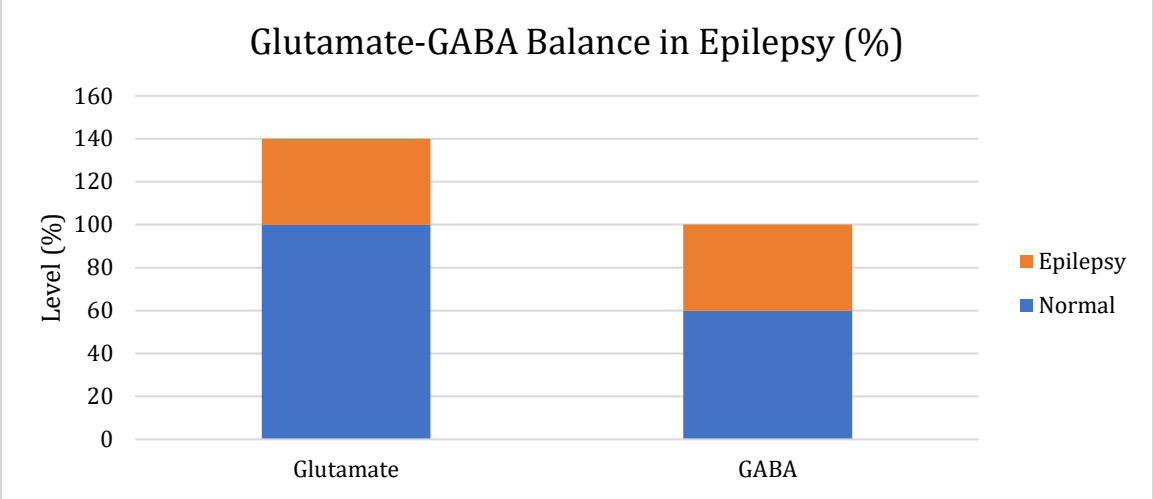
Note: The diagram illustrates the loss of dopamine-producing neurons in the **substantia nigra pars compacta**. As a result, dopamine levels in the basal ganglia sharply decrease, leading to clinical symptoms such as tremor, muscle rigidity, and slowness of movement (bradykinesia). The diagram also shows how levodopa and other medications alleviate symptoms by replenishing dopamine [14][15].

Epilepsy and the Glutamate-GABA Balance. Epilepsy is a disorder characterized by excessive synchronous neuronal activity in the brain and affects approximately 50 million people worldwide [16]. The main neurobiochemical mechanism is the disruption of the balance between excitatory glutamate and inhibitory GABA. Excessive production of glutamate leads to neuronal hyperexcitability, while reduced GABAergic inhibition results in insufficient inhibitory control.

Many antiepileptic drugs target these mechanisms. For instance, benzodiazepines activate GABA-A receptors, lamotrigine reduces glutamate release, and valproate enhances GABA metabolism [17]. In addition, neuroinflammation and ion channel dysfunction have also been found to increase epileptic activity. In recent years, particular attention has been given to ion channel mutations in genetic types of epilepsy.

Academicia Globe:
Inderscience Research

ISSN: 2776-1010 Volume 6, Issue 8, August 2025



Note: The diagram illustrates the disruption of the balance between the excitatory neurotransmitter glutamate and the inhibitory neurotransmitter GABA in the pathogenesis of epilepsy. An increase in glutamate or a decrease in GABA activity leads to excessive neuronal excitability and the onset of epileptic seizures. This mechanism serves as the basis for the development of many antiepileptic drugs [16][17].

Conclusion

Neurobiochemical processes form the foundation of central nervous system function. Their disruption plays a decisive role in the development of various neurological and psychiatric disorders. Examples include decreased serotonin and dopamine levels in depression, dopamine imbalance in schizophrenia, acetylcholine deficiency in Alzheimer's disease, dopamine reduction in the nigrostriatal pathway in Parkinson's disease, and disruption of the glutamate-GABA balance in epilepsy.

Recent studies have shown that a deeper understanding of these neurobiochemical mechanisms provides opportunities to improve diagnostic and therapeutic approaches. In particular, the identification of neurobiomarkers is of great importance for the early diagnosis of diseases and the development of personalized treatment strategies. Therefore, the study of neurobiochemical disorders remains a highly relevant field not only for clinical practice but also for fundamental research.

References

1. Nestler, E. J., Hyman, S. E., & Malenka, R. C. (2021). Molecular Neuropharmacology: A Foundation for Clinical Neuroscience. McGraw-Hill Education.
2. Purves, D., Augustine, G. J., Fitzpatrick, D., et al. (2018). Neuroscience (6th ed.). Oxford University Press.
3. Kandel, E. R., Koester, J. D., Mack, S. H., & Siegelbaum, S. A. (2021). Principles of Neural Science (6th ed.). McGraw-Hill Education.
4. World Health Organization (WHO). (2022). Neurological disorders: public health challenges. Geneva: WHO.

ISSN: 2776-1010 Volume 6, Issue 8, August 2025

5. Malhi, G. S., & Mann, J. J. (2018). Depression. The Lancet, 392(10161), 2299–2312. https://doi.org/10.1016/S0140-6736(18)31948-2
6. Cipriani, A., et al. (2018). Comparative efficacy and acceptability of 21 antidepressant drugs. The Lancet, 391(10128), 1357–1366. https://doi.org/10.1016/S0140-6736(17)32802-7
7. Pariante, C. M., & Lightman, S. L. (2008). The HPA axis in major depression: classical theories and new developments. Trends in Neurosciences, 31(9), 464–468. https://doi.org/10.1016/j.tins.2008.06.006
8. Howes, O. D., & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: version III. Schizophrenia Bulletin, 35(3), 549–562. https://doi.org/10.1093/schbul/sbp006
9. Javitt, D. C. (2010). Glutamatergic theories of schizophrenia. The Israel Journal of Psychiatry and Related Sciences, 47(1), 4–16. PMID: 20686195
10. Uno, Y., & Coyle, J. T. (2019). Glutamate hypothesis in schizophrenia. Psychiatry and Clinical Neurosciences, 73(5), 204–215. https://doi.org/10.1111/pcn.12823
11. Alzheimer's Disease International (ADI). (2021). World Alzheimer Report 2021. London: ADI.
12. Francis, P. T., Palmer, A. M., Snape, M., & Wilcock, G. K. (1999). The cholinergic hypothesis of Alzheimer's disease: a review of progress. Journal of Neurology, Neurosurgery & Psychiatry, 66(2), 137–147. https://doi.org/10.1136/jnnp.66.2.137
13. Selkoe, D. J., & Hardy, J. (2016). The amyloid hypothesis of Alzheimer's disease at 25 years. EMBO Molecular Medicine, 8(6), 595–608. https://doi.org/10.15252/emmm.201606210
14. Poewe, W., et al. (2017). Parkinson disease. Nature Reviews Disease Primers, 3, 17013. https://doi.org/10.1038/nrdp.2017.13
15. Olanow, C. W., Stern, M. B., & Sethi, K. (2009). The scientific and clinical basis for the treatment of Parkinson disease (2009). Neurology, 72(21 Suppl 4), S1–S136. https://doi.org/10.1212/WNL.0b013e3181a1d44c
16. World Health Organization (WHO). (2019). Epilepsy: a public health imperative. Geneva: WHO.
17. Treiman, D. M. (2001). GABAergic mechanisms in epilepsy. Epilepsia, 42(s3), 8–12. https://doi.org/10.1046/j.1528-1157.2001.042suppl.3008.x.