JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS

VOLUME 04, ISSUE 11
MOPNTHLY JOURNALS

ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

BLOOD–BRAIN BARRIER: STRUCTURE, FUNCTION, AND NEUROLOGICAL CONSEQUENCES OF ITS DISRUPTION

Petrova Yurita Yurievna

Assistant Professor, Department of Biomedical Sciences, EMU University.

Annotation: The blood–brain barrier (BBB) is a highly specialized physiological structure that regulates the exchange of substances between the systemic circulation and the central nervous system. It plays a fundamental role in protecting the brain from toxins, pathogens, and fluctuations in blood composition while maintaining neural homeostasis. The BBB is formed by endothelial cells connected by tight junctions, supported by pericytes, astrocytes, and the basement membrane, collectively known as the neurovascular unit. Disruption of BBB integrity is a critical pathological mechanism in a wide range of neurological disorders. This article reviews the structural and functional characteristics of the blood–brain barrier and examines the consequences of its dysfunction in major neurological conditions, including stroke, neuroinfections, neurodegenerative diseases, and brain tumors. In ischemic and hemorrhagic stroke, BBB breakdown contributes to cerebral edema and secondary neuronal injury. In neuroinfections, increased permeability facilitates inflammatory infiltration and neural damage. Chronic BBB impairment is implicated in the progression of neurodegenerative disorders, while tumor-associated vascular abnormalities lead to localized barrier disruption. Understanding the mechanisms underlying BBB dysfunction is essential for the development of targeted therapeutic strategies aimed at preserving or restoring barrier integrity and improving neurological outcomes.

Key words: Blood–brain barrier; neurovascular unit; brain homeostasis; BBB disruption; stroke; neuroinflammation; neuroinfections; neurodegenerative diseases; brain tumors; cerebral edema; neurological disorders**.**

Introduction

The blood–brain barrier (BBB) is a highly specialized and dynamic interface that separates the circulating blood from the central nervous system (CNS). It plays a crucial role in maintaining the structural and functional integrity of the brain by tightly regulating the passage of ions, nutrients, metabolites, and cells between the bloodstream and neural tissue. By preserving a stable biochemical environment, the BBB ensures optimal conditions for neuronal signaling and synaptic activity.

Structurally, the BBB is formed by brain microvascular endothelial cells interconnected by complex tight junctions and supported by pericytes, astrocytic end-feet, and the basement membrane, together constituting the neurovascular unit. This unique organization distinguishes cerebral capillaries from peripheral vasculature and underlies the selective permeability of the barrier.

Disruption of BBB integrity has emerged as a critical pathological event in a wide range of neurological disorders. Acute conditions such as ischemic and hemorrhagic stroke are characterized by rapid breakdown of the BBB, leading to cerebral edema and secondary neuronal injury. Inflammatory and infectious diseases of the CNS compromise barrier function through cytokine-mediated mechanisms, while chronic BBB dysfunction contributes to the progression of neurodegenerative diseases. Furthermore, pathological angiogenesis in brain tumors results in localized barrier disruption, influencing disease progression and therapeutic efficacy.

Understanding the structure, functions, and mechanisms of BBB disruption is essential for elucidating the pathogenesis of neurological diseases and for the development of targeted therapeutic strategies aimed at protecting or restoring BBB integrity.

Purpose of the study

The purpose of this study is to analyze the structural and functional characteristics of the blood–brain barrier and to evaluate its role in protecting the brain and maintaining central nervous system homeostasis. Particular emphasis is placed on the mechanisms underlying blood–brain barrier disruption and the resulting neurological consequences. The study aims to examine the involvement of blood–brain barrier dysfunction in the pathogenesis of major neurological disorders, including stroke, neuroinfections, neurodegenerative diseases, and brain tumors. By synthesizing current scientific evidence, this work seeks to highlight the clinical significance of blood–brain barrier impairment and to underscore its importance as a potential therapeutic target in neurological disease management.

Materials and methods

This study is based on a comprehensive narrative review of current scientific literature focused on the structure, function, and pathological alterations of the blood–brain barrier. Relevant peer-reviewed articles, systematic reviews, and experimental studies were identified through electronic databases including PubMed, Scopus, and Web of Science. The literature search was conducted using key terms such as blood–brain barrier, neurovascular unit, BBB disruption, stroke, neuroinfections, neurodegenerative diseases, and brain tumors.

Publications written in English and published within the last 10–15 years were prioritized to ensure the inclusion of up-to-date and clinically relevant data. Additional classical and highly cited studies were included to provide foundational background on blood–brain barrier physiology. Articles were selected based on their relevance to BBB structure, molecular mechanisms of barrier dysfunction, and associated neurological outcomes.

Data from the selected studies were analyzed qualitatively and synthesized to identify common mechanisms, pathological patterns, and clinical implications of blood–brain barrier disruption across different neurological conditions. No original experimental or clinical data were collected, and ethical approval was not required for this review-based study.

Results and discussion

Analysis of the reviewed literature demonstrates that the blood–brain barrier plays a central role in maintaining cerebral homeostasis and that its disruption is a common pathological feature across a wide spectrum of neurological diseases. Structural and functional alterations of the blood–brain barrier were consistently associated with disease severity, progression, and clinical outcomes.

Structural and Functional Alterations of the Blood–Brain Barrier

he reviewed studies indicate that damage to endothelial cells and degradation of tight junction proteins, such as claudins and occludins, represent key mechanisms of blood–brain barrier dysfunction. Loss of tight junction integrity results in increased paracellular permeability, allowing plasma proteins, immune cells, and neurotoxic substances to enter the brain parenchyma. In addition, alterations in pericyte coverage and astrocytic end-foot signaling further compromise barrier stability and regulation of cerebral blood flow.

Blood–Brain Barrier Disruption in Stroke

In ischemic and hemorrhagic stroke, acute hypoxia, oxidative stress, and inflammatory responses lead to rapid breakdown of the blood–brain barrier. This breakdown contributes to vasogenic edema, increased intracranial pressure, and secondary neuronal damage. Several studies highlight that the extent of blood–brain barrier permeability correlates with infarct size and neurological deficits, emphasizing its prognostic significance. Restoration or preservation of barrier integrity has therefore emerged as a promising therapeutic strategy in acute stroke management.

Role of the Blood–Brain Barrier in Neuroinfections

In neuroinfectious conditions, including bacterial meningitis and viral encephalitis, inflammatory cytokines and microbial factors directly impair blood–brain barrier function. Increased permeability facilitates leukocyte infiltration and the spread of inflammatory mediators within the central nervous system, exacerbating neuronal injury. While immune cell entry is necessary for pathogen clearance, excessive barrier disruption contributes to tissue damage and long-term neurological sequelae.

Blood–Brain Barrier Dysfunction in Neurodegenerative Diseases

Chronic and progressive impairment of blood–brain barrier integrity is a hallmark of several neurodegenerative disorders. In Alzheimer's disease, reduced clearance of β-amyloid peptides across the blood–brain barrier promotes their accumulation in brain tissue. In multiple sclerosis, immune-mediated disruption of the barrier allows autoreactive lymphocytes to infiltrate the central nervous system, driving demyelination and neuroinflammation. These findings suggest that blood–brain barrier dysfunction is not merely a consequence but an active contributor to neurodegenerative pathology.

Blood–Brain Barrier Changes in Brain Tumors

The reviewed evidence indicates that brain tumors induce abnormal angiogenesis, resulting in structurally and functionally compromised vasculature. Localized blood–brain barrier disruption facilitates tumor-associated edema and alters drug penetration. Although increased permeability may enhance delivery of certain chemotherapeutic agents, heterogeneous barrier disruption often limits treatment efficacy and contributes to therapeutic resistance.

Clinical and Therapeutic Implications

Collectively, the findings underscore the critical importance of the blood–brain barrier in neurological health and disease. Blood–brain barrier integrity represents both a diagnostic marker and a therapeutic target. Strategies aimed at modulating barrier permeability, reducing inflammation, and restoring tight junction function hold significant potential for improving outcomes in a variety of neurological disorders.

Conclusion

The blood–brain barrier is a critical physiological structure that ensures the protection of the brain and maintains central nervous system homeostasis. Its complex architecture, including endothelial cells, tight junctions, pericytes, astrocytes, and the basement membrane, underlies its selective permeability and regulatory functions. Disruption of the blood–brain barrier is a central feature in the pathogenesis of numerous neurological disorders, including stroke, neuroinfections, neurodegenerative diseases, and brain tumors. Acute injuries, inflammatory processes, and chronic pathological changes compromise barrier integrity, leading to cerebral edema, neuronal damage, and disease progression. Understanding the mechanisms of blood–brain barrier dysfunction provides valuable insights into disease pathophysiology and highlights its potential as a therapeutic target. Strategies aimed at preserving or restoring barrier function hold promise for improving clinical outcomes and mitigating neurological damage across a wide range of CNS

disorders. Maintaining the integrity of the blood–brain barrier is therefore essential for both neurological health and the effective management of brain diseases.

Bibliography:

1. Wu, D., Chen, Q., Chen, X., Han, F., Chen, Z., & Wang, Y. (2023). *The blood–brain barrier: Structure, regulation and drug delivery*. **Signal Transduction and Targeted Therapy**, 8, 217. Available from PMC. PMC
2. Sweeney, M. D., Zhao, Z., Montagne, A., Nelson, A. R., & Zlokovic, B. V. (2018). *Blood–Brain Barrier: From Physiology to Disease and Back*. **Physiological Reviews**, 99(1), 21–78. Available from PMC. PMC
3. Alahmari, A. (2021). *Blood–Brain Barrier Overview: Structural and Functional Correlation*. **Neural Plasticity**, 2021, 6564585. Available from PMC. PMC
4. "The role of the blood–brain barrier during neurological disease and infection". (2023). *Journal Article*, PubMed. PubMed
5. "The blood–brain barrier and blood-tumour barrier in brain tumours and metastases". (2019). *PubMed*. PubMed
6. Terstappen, G. C., Meyer, A. H., Bell, R. D., & Zhang, W. (2021). *Strategies for delivering therapeutics across the blood-brain barrier*. **Nature Reviews Drug Discovery**, 20(5), 362–383. (Cited in Litvinova et al.). RCSI Journals Platform
7. Trofimov, A. N., Litvinova, M. V., Schwarz, A. P., et al. (2022). *Molecular mechanisms of molecular transfer across the blood-brain barrier as a target for pharmacological action. Part 1: Structure, function and pathology of the BBB*. **Pharmacy Formulas**, 4(2), 60–69. Eco-Vector Journals Portal
8. Okonenko, T. I., Novikova, A. P., & Rumyantsev, E. E. (2024). *Blood-Brain Barrier: Defining Structures and Functions*. **Modern Issues of Biomedicine**. svbskfmba.ru
9. Mayer, M. G., & Fischer, T. (2024). *Microglia at the blood brain barrier in health and disease*. **Frontiers in Cellular Neuroscience**, 18:1360195. Frontiers
10. Wikipedia contributors. (2025). *Blood-brain barrier*. In *Wikipedia, The Free Encyclopedia*. (Use with caution; primarily as supplementary background).