

International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
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ARTICLE TITLE	THE IMPACT OF CHRONIC STRESS ON THE GUT-BRAIN AXIS: A REVIEW
DOI	https://doi.org/10.31435/ijitss.4(52).2026.6866
RECEIVED	17 August 2026
ACCEPTED	23 October 2026
PUBLISHED	30 October 2026
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THE IMPACT OF CHRONIC STRESS ON THE GUT-BRAIN AXIS: A REVIEW

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ABSTRACT

Aim of the study: The aim of this article is to review the literature on the relationship between the gut–brain axis and chronic stress, with particular emphasis on the key components and mechanisms of this system.
Materials and methods: This study is a narrative review based on current literature retrieved primarily from the PubMed database. Original research articles and review papers published in English were included. Studies were selected according to their relevance to chronic stress and its impact on gut–brain axis functioning and associated mechanisms.
Results: Chronic stress disrupts the gut–brain axis by altering gut microbiota composition, increasing intestinal permeability, and promoting systemic inflammation. These changes impair bidirectional communication between the gut and the brain, leading to neuroendocrine and immune dysregulation and increasing susceptibility to neuropsychiatric disorders such as depression. Microbial metabolites, particularly short-chain fatty acids, play an important role in these processes and may represent potential therapeutic targets.
Conclusions: Gut–brain axis disorders result from complex interactions between the nervous, immune, and metabolic systems, with gut microbiota playing a key regulatory role. Although evidence supports its influence on stress responses and neuropsychiatric vulnerability, the underlying mechanisms are not yet fully understood. Further research is needed to clarify causal relationships and to develop targeted therapeutic strategies integrating microbiota modulation with stress regulation.

KEYWORDS
Gut Microbiota, SCFAs, Hypothalamic-Pituitary-Adrenal Axis, Neuroimmune Interactions, Dysbiosis

CITATION
Marciszewska, A., Popczyk, N., Tymchenko, H., Świątecka, M., Kaźmierczyk, J., Buczkowska, E., Jopek, J., Popczyk, M., Piechowicz, A., & Cionżyńska, W. (2026). The impact of chronic stress on the gut-brain axis: A review. *International Journal of Innovative Technologies in Social Science*, 4(52). https://doi.org/10.31435/ijitss.4(52).2026.6866

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Introduction

Stress is an inevitable part of our daily lives. Whether it stems from work, family circumstances, or unexpected situations, each type of stress can have negative health consequences. Chronic stress, on the other hand, refers to prolonged exposure to stressors, manifesting as a feeling of constant overwhelm. Behind the feeling of general unease, fear, and anxiety lies the activation of stress response mechanisms. This involves the hypothalamic- pituitary-adrenal (HPA) axis, as well as reactions from the nervous system. Our body attempts to adapt to incoming stimuli by mobilizing the HPA axis, the immune system, the nervous system, and molecular processes occurring in organs that are triggered by hormones. The phenomenon of physiological adaptation to stress-inducing factors acting on the body is called allostasis. When the balance of these systems is disrupted due to excessive activation of mediators, pathophysiological processes are triggered [1].

Elements involved in gut-brain axis

In recent years, scientists have described the gut-brain axis as a bidirectional communication system between the digestive tract, which is partially colonized by microorganisms, and the central nervous system. Information is transmitted both from the gut to the central nervous system and in the opposite direction, enabling dynamic adaptation of physiological responses to changing internal and external conditions. This process involves, among others, the immune system, the neuroendocrine system, the enteric nervous system (ENS), the circulatory system, and the vagus nerve, which together form a network of functional connections between the gut and the brain. The integration of systemic signals is facilitated by a range of molecules, including metabolites produced by the gut microbiota [2]. The function of the digestive system goes beyond mere digestion and absorption of nutrients. One of its primary roles is also to maintain the body's dynamic balance through the proper functioning of the gut microbiota. This microecosystem is composed mainly of bacteria, but also includes fungi, protozoa, and yeasts. Within this environment, multilevel communication occurs between microorganisms and the host organism, which plays a key role in regulating metabolic, immune,

and signaling processes. The main bacteria that colonize the human gut after birth are *Prevotella, Bacteroidetes, Firmicutes, Actinobacteria*, and *Proteobacteria* [3-5].

The gut microbiota constitutes a complex and dynamic population of microorganisms inhabiting the human gastrointestinal tract, recognized as an integral part of human physiology. Its impact on health and disease development stems primarily from the continuous interactions between microorganisms and the immune system of the intestinal mucosa. Despite a growing body of research, the mechanisms underlying this relationship are not yet fully understood. Increasing evidence suggests that metabolites and other products produced by the microbiota play a significant role in maintaining intestinal homeostasis and the proper functioning of the gastrointestinal tract. Abnormalities in the composition or activity of the microbiota, referred to as dysbiosis, can lead to an imbalance in the gut and contribute to the development of various pathological conditions [6,7].

Communication pathways in the gut-brain axis

The autonomic nervous system (ANS), comprising the sympathetic (SNS) and parasympathetic (PNS) divisions, plays a key role in regulating bodily functions and the stress response. Activation of the SNS, particularly under stressful conditions, leads to increased secretion of catecholamines. In contrast, at rest, PNS activity predominates, its action is antagonistic and promotes regenerative processes [8]. The enteric nervous system (ENS) is a specialized network of neurons responsible for controlling gastrointestinal functions, including motility, blood flow, and secretion. By interacting with the autonomic and central nervous systems, it facilitates the integration of signals originating from the gut. These signals, which include hormones, neurotransmitters, and immune mediators, are transmitted to the brain and may influence the maintenance of homeostasis, behavioral regulation, and the body's response to stress [9].

The hypothalamic-pituitary-adrenal (HPA) axis is a key system involved in regulating the body's response to stress. Stressors trigger the hypothalamus to release corticotropin-releasing hormone (CRH), which stimulates the pituitary gland to produce adrenocorticotropic hormone (ACTH). ACTH, in turn, stimulates the adrenal cortex to release cortisol. An increase in cortisol levels inhibits further secretion of CRH and ACTH through negative feedback [10,11]. Cortisol plays a crucial role in the body's response, it has anti-inflammatory effects, supports metabolic processes (e.g., gluconeogenesis), and prevents excessive activation of the immune system. However, HPA axis activity is not limited solely to stress, it also participates in the regulation of the circadian rhythm, which manifests as the highest cortisol concentration in the morning. These hormones prepare the body for the adaptive response known as "fight or flight," while simultaneously influencing numerous physiological processes, including the functioning of the immune, metabolic, and gastrointestinal systems [11]. The microbiota plays an important role in regulating this axis and in modulating the immune response at both the local and systemic levels. Of particular importance are bacterial metabolites, especially short-chain fatty acids (SCFAs), which exhibit immunomodulatory properties and can influence the nervous system, among other things, by activating G protein- coupled receptors and modulating the activity of the autonomic nervous system. Studies conducted on rodents confirmed the positive effect of SCFAs on microglial cell maturation and demonstrated beneficial anxiolytic and antidepressant effects via the regulation of tryptophan metabolism [13,14]. SCFAs also influence the secretion of gut hormones by enteroendocrine cells, participating in the regulation of hormonal signaling between the gut and the brain [15,16]. Furthermore, the gut microbiota is capable of producing numerous neuroactive compounds, such as dopamine, histamine, and acetylcholine, and is also involved in the regulation of bile acid metabolism, which may affect blood-brain barrier function and HPA axis activity [15,16]. The immune system is one of the critical elements regulating communication within the gut-brain axis, acting as a mediator in maintaining the balance between these two systems.

Its role stems primarily from its numerous connections with the neuroendocrine and nervous systems, including the hypothalamic-pituitary-adrenal axis, the autonomic nervous system, and the enteric nervous system [17]. The proper functioning of the intestinal barrier is crucial for this communication. Damage to the barrier, known as "leaky gut," leads to increased permeability of the intestinal epithelium, allowing bacteria and their components, particularly Gram-negative bacteria, to enter the systemic circulation [18]. As a result, the immune system is activated and the production of inflammatory mediators, such as interleukin-6 and interferon γ, is intensified [19]. These processes are considered a major component of the pathogenesis of microbiota–gut–brain axis disorders, as well as central nervous system diseases, including depression and addiction [20]. An increasing volume of data also points to the involvement of immune system cells in

neurodegenerative processes. It has been shown that interleukin-17-producing T cells can lead to neuronal damage and accelerate the development of lesions characteristic of Parkinson's disease [21]. Furthermore, the gut microbiota and its metabolites influence the differentiation of T cells toward the Th17 phenotype, which may exacerbate neuroinflammatory processes observed, for example, in models of autoimmune diseases of the central nervous system [22].

Stress as a factor disrupting intestinal homeostasis

Chronic stress is one of the significant risk factors for the development of neuropsychiatric disorders, including depression, which manifests itself, among other things, as a depressed mood and an inability to derive pleasure from previously enjoyed activities [23,24]. A growing number of studies suggest that a vital element linking stress to the pathophysiology of these disorders is the gut-brain axis, in which the gut microbiota plays a significant role. It participates in regulating the integrity of the intestinal barrier, and its dysregulation can lead to increased intestinal permeability and the development of chronic, low-grade inflammation. Dysbiosis, by increasing intestinal barrier permeability, allows inflammatory mediators and pathogens to enter the systemic circulation. Consequently, an immune response is activated, which may affect the central nervous system and contribute to the development of mental disorders [25]. Inflammation is considered one of the main mechanisms linking microbiota dysfunction to increased brain susceptibility to diseases such as schizophrenia [26] and depression [27]. Studies in animal models indicate that the gut microbiota influences the body's response to stress [28]. Interventions such as using probiotics can modulate emotional and stress responses [29]. It has also been demonstrated that stress in adulthood leads to changes in the composition of the gut microbiota, including an increase in the abundance of *Clostridium* bacteria and a decrease in *Bacteroides* [30]. This is accompanied by increased bacterial translocation across the intestinal barrier and changes in immune system function, suggesting a significant role for the microbiota in modulating the immune response to stress [30] The sympathetic nervous system, which mediates communication between the brain and the gut, likely plays a role in these mechanisms [30]. Stress also affects the metabolic activity of the microbiota, leading to changes in the production of bacterial metabolites, particularly short-chain fatty acids, which play a significant role in maintaining immune and metabolic homeostasis, as well as the integrity of the intestinal barrier [31]. Animal studies have shown that chronic stress, whether associated with immobilization or social dysfunction, led to reduced levels of SCFAs such as propionic, butyric, and acetic acids [32]. Although the number of studies on long-term stress and its impact on the human gut microbiome remains limited, the available data suggest that its effects may resemble the changes observed in rodent models of chronic stress. In models of post-traumatic stress, it has been shown that the composition of the microbiota prior to stress exposure may determine the organism's susceptibility or resistance to its effects [12]. Importantly, although a single episode of severe stress does not always lead to increased microbial diversity, this parameter correlates with the severity of behavioral disturbances [12]. An analysis of the available research indicates that chronic stress affects the functioning of the gut-brain axis in multiple ways. It involves changes in the composition and activity of the gut microbiota, impairments in intestinal barrier integrity, and the activation of inflammatory mechanisms. These processes interact with one another, leading to dysregulation of communication between the gut and the brain and increasing the risk of developing neuropsychiatric disorders. Understanding these relationships may be crucial for developing new therapeutic strategies aimed at modulating microbiota and stress responses.

Discussion

Based on the data presented above, it can be concluded that chronic stress significantly affects the functioning of the gut-brain axis, simultaneously influencing the gut microbiota, the immune system, and neuroendocrine mechanisms. Key mechanisms include increased intestinal barrier permeability and progressive imbalances in the composition of the microbiota, which promote the activation of an inflammatory response. Consequently, the resulting inflammatory process may affect the central nervous system, influence emotional and cognitive functions, and increase the risk of neuropsychiatric disorders. Additionally, changes in the production of microbiota metabolites, such as short-chain fatty acids, which participate in the regulation of intestinal and immune homeostasis, play a significant role. Despite the growing number of studies, the mechanisms underlying these relationships are not fully understood, and one of the limitations remains the heterogeneity of research models and individual differences in microbiota composition. Importantly, the gut–brain axis represents a bidirectional communication system, which provides a rationale for microbiota-targeted

interventions, such as probiotics or dietary modifications. It has been shown that probiotics may modulate inflammatory responses, HPA axis activity, and neurotransmitter production, potentially alleviating symptoms of depression and anxiety. However, clinical findings remain inconsistent and require further validation [33,34]. This may be related to differences in study design, probiotic strains used, duration of intervention, as well as baseline microbiota composition of participants, which together may determine individual variability in therapeutic response. Therefore, future research should focus on identifying specific microbial signatures associated with treatment responsiveness and on standardizing clinical protocols to better evaluate the efficacy of microbiota-based interventions.

Conclusions

An analysis of the available research indicates that disruptions in this axis do not result from a single mechanism, but are the result of complex interactions between neural, immune, and metabolic components. A notable finding is the growing importance of the gut microbiota as an active regulatory element, rather than simply a passive participant in these processes. Its ability to influence the body's response to stress suggests that it may also play a role in shaping susceptibility to neuropsychiatric disorders. Despite significant progress in this field, there is still a need to integrate the results of experimental and clinical studies and to identify precise cause-and-effect mechanisms. It seems especially important to determine to what extent changes within the gut-brain axis are reversible and what factors determine their long-term consequences. A deeper understanding of these relationships could contribute to the development of more targeted therapeutic strategies that incorporate both the modulation of the gut microbiota and a comprehensive approach to regulating the stress response.

Contributions: Conceptualization was done by Aleksandra Marciszewska; methodology by Aleksandra Marciszewska and Natalia Popczyk; software by Hanna Tymchenko, checking by Martyna Świątecka; formal analysis by Jakub Kaźmierczak; investigation by Ewa Buczkowska; resources by Jakub Jopek; data curation by Michał Popczyk; writing-rough preparation by Agnieszka Piechowicz; writing-review and editing by Weronika Cionżyńska; visualization by Aleksandra Marciszewska and Jakub Kaźmierczak; supervision by Hanna Tymchenko; project administration by Agnieszka Piechowicz. All authors have read and agreed with the published version of the manuscript.

Funding statement: The study did not receive special funding.

Conflict of Interest Statement: The authors report that there is no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process: In preparing this work, the authors used Gemini for the purpose of language-related support, including grammatical and stylistic editing in English. After using this tool, the authors have reviewed and edited the content as needed and accepted full responsibility for the substantive content of the publication.

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