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,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	THE GUT–BRAIN AXIS IN THE DIGITAL ERA: CAN PSYCHOLOGICAL STRESS FROM ONLINE ENVIRONMENTS ALTER THE MICROBIOME?
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5441
RECEIVED	13 February 2026
ACCEPTED	28 April 2026
PUBLISHED	06 May 2026
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THE GUT–BRAIN AXIS IN THE DIGITAL ERA: CAN PSYCHOLOGICAL STRESS FROM ONLINE ENVIRONMENTS ALTER THE MICROBIOME?

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ABSTRACT

The gut–brain axis constitutes a complex, bidirectional communication network integrating neural, endocrine, immune, and metabolic signaling pathways between the gastrointestinal tract and the central nervous system. A growing body of evidence demonstrates that psychological stress can significantly alter gut microbiota composition and function, contributing to the pathophysiology of gastrointestinal and neuropsychiatric disorders. Concurrently, modern digital environments—characterized by pervasive social media use, continuous connectivity, and high levels of information exposure—have emerged as novel and sustained sources of psychological stress.

This review aims to evaluate whether stress associated with digital environments may influence the gut microbiome through established gut–brain axis mechanisms. A structured narrative synthesis of the literature was conducted, focusing on studies examining stress-induced microbiome alterations, neuroendocrine responses, and the physiological consequences of digital stress. Evidence indicates that chronic activation of the hypothalamic–pituitary–adrenal axis leads to sustained cortisol release, which may disrupt intestinal barrier integrity, modulate immune responses, and reduce microbial diversity.

Although direct empirical studies linking digital stress to microbiome alterations remain limited, converging evidence from psychoneuroendocrinology and microbiome research supports a biologically plausible pathway. This review proposes an integrative conceptual framework connecting digital stressors with gut microbiome dysregulation.

The findings underscore the importance of considering digital behavior as a potential environmental determinant of physiological health. Future research should incorporate objective measures of digital exposure alongside longitudinal microbiome analyses to clarify causal relationships and support the development of targeted preventive and therapeutic strategies.

KEYWORDS

Gut–Brain Axis, Gut Microbiome, Psychological Stress, Digital Stress, HPA Axis, Microbiota Dysbiosis

CITATION

Wiktoria Urowska, Magdalena Kossmann, Amin Abdulgater, Paulina Osuch-Tomaszewska, Laura Szalewska, Jędrzej Łysiak, Izabela Kazubek-Fuksiewicz. (2026) The Gut–Brain Axis in the Digital Era: Can Psychological Stress from Online Environments Alter the Microbiome? *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5441

1. Introduction

The human gut microbiome has emerged as a critical regulator of host physiology, influencing metabolic processes, immune function, and central nervous system activity. Over the past two decades, growing evidence has established the importance of the gut–brain axis as a bidirectional communication system linking the gastrointestinal tract and the brain through neural, endocrine, immune, and metabolic pathways. This complex network enables gut microbiota to influence brain function and behavior, while central nervous system activity can, in turn, modulate gastrointestinal physiology and microbial composition.

The gut microbiome plays a central role in this interaction by producing a range of bioactive compounds, including short-chain fatty acids, neurotransmitter precursors, and immune-modulating molecules. These microbial metabolites can influence neural signaling, regulate inflammation, and alter host stress responsiveness. Disruptions in microbial composition, commonly referred to as dysbiosis, have been associated with a wide range of conditions, including irritable bowel syndrome, depression, anxiety disorders, and metabolic diseases.

Psychological stress is one of the most significant factors influencing gut microbiota composition and function. Both acute and chronic stress have been shown to alter microbial diversity, increase intestinal permeability, and promote inflammatory responses. These effects are primarily mediated through activation of the hypothalamic–pituitary–adrenal (HPA) axis, a central component of the stress response system. Activation of the HPA axis leads to the release of cortisol, which exerts widespread effects on gastrointestinal physiology, including modulation of gut barrier integrity, immune activity, and microbial balance.

Experimental and clinical studies have consistently demonstrated that stress-induced activation of the HPA axis can result in significant alterations in the gut microbiome. For example, reductions in beneficial bacterial genera such as *Lactobacillus* and *Bifidobacterium* have been observed under stress conditions, alongside increased abundance of potentially pathogenic taxa. These changes may contribute to both local gastrointestinal dysfunction and systemic inflammatory processes, highlighting the importance of stress as a modulator of gut health.

While traditional stressors—such as physical threats, social conflict, or major life events—have been extensively studied, modern environments introduce new forms of psychological stress that differ in both intensity and temporal pattern. In particular, digital technologies have become deeply integrated into daily life, fundamentally altering the way individuals interact, communicate, and process information. Social media platforms, continuous connectivity, and high levels of information exposure create conditions that may promote persistent psychological arousal and cognitive overload.

A growing body of research suggests that digital environments are associated with increased levels of stress, anxiety, and depressive symptoms. Mechanisms such as social comparison, fear of missing out, and exposure to emotionally salient content contribute to heightened psychological vulnerability. Additionally, the anticipation of notifications and constant accessibility may lead to a state of continuous partial attention, characterized by sustained cognitive engagement and reduced capacity for recovery.

Unlike traditional stressors, which are often acute and time-limited, digital stress is typically chronic, low-grade, and pervasive. This pattern of exposure may result in prolonged activation of stress-related physiological systems, including the HPA axis and the sympathetic nervous system. Such sustained activation has the potential to produce cumulative effects on physiological regulation, including alterations in immune function, endocrine signaling, and gastrointestinal homeostasis.

Despite extensive research on the effects of psychological stress on the gut microbiome, the potential role of digital stress as a modulator of gut–brain axis function remains largely unexplored. Most existing studies focus either on traditional stressors or on the psychological consequences of digital technology use,

with limited integration between these domains. As a result, there is a critical gap in understanding how modern digital environments may influence physiological systems through established biological pathways.

The present review aims to address this gap by examining whether psychological stress originating from digital environments can influence the gut microbiome via gut–brain axis mechanisms. By synthesizing evidence from microbiology, neuroscience, and digital behavior research, this study proposes an integrative conceptual framework linking digital stressors to microbiome alterations.

To our knowledge, this is one of the first reviews to explicitly integrate digital stress into the gut–brain axis model, highlighting the importance of considering contemporary environmental factors in the study of human health. Understanding this relationship may have significant implications for both clinical practice and preventive medicine, particularly in the context of increasing digitalization and its impact on lifestyle and well-being.

2. Materials and Methods

2.1 Study Design

This study was conducted as a structured narrative review aimed at synthesizing current evidence on the gut–brain axis, psychological stress, and gut microbiome alterations, with a particular focus on the emerging role of digital stress. Although not designed as a formal systematic review or meta-analysis, this study followed established principles of structured literature synthesis, including predefined search strategies, eligibility criteria, and thematic categorization of findings.

The objective was to integrate evidence from multiple research domains—including microbiology, neuroscience, psychoneuroendocrinology, and digital behavior research—to evaluate the biological plausibility of a pathway linking digital stress to microbiome alterations.

2.2 Data Sources and Search Strategy

A comprehensive literature search was conducted using the electronic databases **PubMed**, **Scopus**, and **Web of Science**. These databases were selected due to their broad coverage of biomedical, psychological, and interdisciplinary research.

The search included studies published between **2004 and 2025**, reflecting the period during which research on the gut–brain axis and microbiome has expanded significantly, as well as the emergence of digital stress as a relevant research topic.

Search queries were constructed using combinations of keywords and Boolean operators. The primary search terms included: “gut–brain axis”, “gut microbiome AND stress”, “microbiota AND psychological stress”, “HPA axis AND gut permeability”, “stress AND intestinal microbiota”, “digital stress”, “social media AND stress”, “online environment AND mental health”, “notification stress”, “information overload AND stress”.

Search strategies were adapted for each database to ensure optimal retrieval of relevant studies. In addition, manual screening of reference lists from key review articles was performed to identify further relevant publications.

2.3 Eligibility Criteria

Studies were selected based on predefined inclusion and exclusion criteria.

Inclusion Criteria

Studies were included if they:

- Were published in peer-reviewed scientific journals.
- Were written in English.
- Investigated at least one of the following domains:

a. gut–brain axis mechanisms,
b. effects of psychological stress on the gut microbiome,
c. neuroendocrine or immune pathways linking stress and gut function,
d. physiological or psychological effects of digital environments.

- Included human participants, animal models, or were high-quality narrative or systematic reviews.

Exclusion Criteria

Studies were excluded if they:

- Were conference abstracts, editorials, commentaries, or non-peer-reviewed sources. - Focused exclusively on unrelated gastrointestinal, neurological, or psychiatric conditions without addressing stress or microbiome-related mechanisms.
- Lacked sufficient methodological detail or scientific rigor.
- Were duplicate records identified across databases.

2.4 Study Selection

The study selection process was conducted in two stages. First, titles and abstracts retrieved from database searches were screened for relevance. Second, full-text articles were assessed based on the predefined eligibility criteria.

Priority was given to:

- studies with strong methodological design,
- frequently cited foundational articles,
- recent publications reflecting current scientific understanding,
- studies providing mechanistic insights into stress–microbiome interactions.

Due to the limited availability of direct studies examining digital stress and microbiome alterations, indirect evidence was also included. This encompassed studies investigating traditional psychological stress, HPA axis activation, gut permeability, immune responses, and microbiome dynamics, as well as research on digital stress and its physiological effects.

2.5 Data Extraction and Synthesis

Relevant data were extracted from selected studies, including:

- study design and methodology,
- population characteristics or experimental model,
- type and duration of stress exposure,
- microbiome-related outcomes (e.g., diversity, composition),
- physiological mechanisms (e.g., HPA axis activation, immune responses),
- relevance to digital stress or behavioral factors.

The extracted data were synthesized using a thematic approach. Studies were grouped into four main categories:

1. Mechanisms of the gut–brain axis
2. Effects of psychological stress on the microbiome
3. Digital environments as sources of stress
4. Integration of digital stress into gut–brain axis models

This approach enabled the identification of converging evidence across different research domains and supported the development of a conceptual framework linking digital stress to microbiome alterations.

2.6 Quality Assessment

Given the narrative nature of this review, a formal quantitative quality assessment was not conducted. However, methodological quality was considered during study selection. Preference was given to:

- peer-reviewed studies with clear experimental design,
- studies with adequate sample sizes,
- research employing validated measurement tools,
- high-impact and frequently cited publications.

This approach ensured that the included literature reflected current scientific standards and contributed meaningfully to the synthesis. As a structured narrative review, this study was not intended to provide a quantitative synthesis of effect sizes, but rather to identify converging evidence and generate a hypothesis-driven conceptual framework.

3. Results

3.1 Mechanisms of the Gut–Brain Axis

The gut–brain axis is mediated by a complex network of neural, endocrine, immune, and metabolic pathways that enable bidirectional communication between the gastrointestinal tract and the central nervous system. These interconnected systems allow for the integration of physiological signals and contribute to the regulation of both gastrointestinal and neurobehavioral processes.

3.1.1 Neural Pathways: The Role of the Vagus Nerve

The vagus nerve represents a primary neural pathway within the gut–brain axis, facilitating rapid bidirectional communication between the gut and the brain. It transmits afferent sensory signals from the gastrointestinal tract to the central nervous system while also regulating efferent responses that influence gut motility, secretion, and inflammatory activity.

Microbial metabolites, including short-chain fatty acids (SCFAs), have been shown to influence vagal signaling either directly or indirectly through interactions with enteroendocrine cells. These interactions enable gut microbiota to modulate central nervous system function, including behavioral and emotional responses.

Experimental studies have demonstrated that disruption of vagal pathways can attenuate or abolish microbiota-induced behavioral effects, indicating that intact neural signaling is essential for microbiota–brain communication. Additionally, alterations in vagal tone have been associated with changes in emotional regulation and stress responsiveness.

Chronic stress has been reported to impair vagal tone, reducing the efficiency of gut–brain communication. This impairment is associated with dysregulation of gastrointestinal function and may contribute to shifts in microbial composition.

3.1.2 Endocrine Pathways: Activation of the HPA Axis

Endocrine signaling within the gut–brain axis is primarily mediated by the hypothalamic–pituitary–adrenal (HPA) axis. In response to psychological stress, the hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates the secretion of adrenocorticotropic hormone (ACTH) from the pituitary gland. This process leads to the release of cortisol from the adrenal cortex.

Cortisol exerts systemic effects on metabolism, immune function, and gastrointestinal physiology. Within the gut, elevated cortisol levels have been associated with increased intestinal permeability, often referred to as "leaky gut." This increased permeability allows microbial components, including lipopolysaccharides, to translocate into systemic circulation, contributing to inflammatory responses.

Studies have demonstrated that chronic activation of the HPA axis is associated with alterations in microbial composition, including reductions in beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*, as well as increased abundance of potentially pathogenic taxa.

3.1.3 Immune System Interactions

The immune system represents a critical interface between the gut microbiome and host physiology. Gut microbiota interact with immune cells to regulate inflammatory processes and maintain intestinal homeostasis.

Psychological stress has been shown to increase the production of pro-inflammatory cytokines, which can disrupt gut barrier integrity and contribute to microbial imbalance. Stress-induced immune activation may therefore play a key role in mediating microbiome alterations.

Additionally, immune signaling molecules, including cytokines, can influence central nervous system function, providing a pathway through which gut microbiota may affect mood and behavior.

3.1.4 Microbial Metabolites and Neurotransmitters

Gut microbiota produce a wide range of metabolites that contribute to gut–brain communication. These include short-chain fatty acids, neurotransmitter precursors, and bioactive compounds such as gamma-aminobutyric acid (GABA).

Approximately 90% of the body's serotonin is synthesized in the gastrointestinal tract, highlighting the role of gut microbiota in regulating neurotransmitter systems. Microbial metabolites can influence neural activity, immune responses, and endocrine signaling pathways.

Alterations in microbial composition may therefore have significant effects on both physiological and psychological processes.

3.2 Psychological Stress and the Gut Microbiome

Psychological stress has been consistently identified as a major factor influencing the composition and function of the gut microbiome. Both acute and chronic stress have been shown to induce measurable changes in microbial diversity, abundance, and metabolic activity.

3.2.1 Evidence from Animal Studies

Animal studies provide strong evidence for stress-induced microbiome alterations. Exposure to stressors such as restraint stress, maternal separation, and social defeat has been associated with reduced microbial diversity and significant shifts in bacterial composition. These changes include decreases in beneficial bacterial taxa, particularly *Lactobacillus* species, and increases in potentially pathogenic microorganisms. Stress exposure is also associated with increased intestinal permeability and inflammatory responses.

In germ-free animal models, the absence of microbiota has been shown to alter stress responsiveness, indicating that the microbiome plays an active role in regulating the stress response. These findings highlight the bidirectional nature of the gut–brain axis.

3.2.2 Evidence from Human Studies

Human studies support the association between psychological stress and microbiome alterations, although variability exists due to differences in study design and measurement methods.

Clinical observations indicate that individuals experiencing chronic stress exhibit reduced microbial diversity and increased inflammatory markers. For example, stress during academic examination periods has been associated with changes in gut microbiota composition and increased gastrointestinal symptoms.

Patients with stress-related conditions, including depression and anxiety, have been shown to exhibit altered microbiome profiles, suggesting a relationship between psychological stress and dysbiosis.

3.2.3 Mechanistic Pathways Linking Stress and Microbiome

Several mechanisms have been identified through which psychological stress influences the gut microbiome:

- activation of the HPA axis and cortisol release
- increased intestinal permeability
- altered gastrointestinal motility
- immune system activation

These processes contribute to microbial imbalance and reduced diversity, characteristic of dysbiosis.

3.3 Digital Environments as a Source of Psychological Stress

Digital environments have become an integral component of modern life, fundamentally transforming patterns of communication, information consumption, and social interaction. Alongside these changes, a growing body of research has identified digital environments as significant contributors to psychological stress. Unlike traditional stressors, which are often acute and time-limited, digital stress is typically characterized by continuous exposure, high frequency, and persistent cognitive engagement.

One of the defining features of digital stress is its chronic and low-grade nature. Digital platforms, including social media, messaging applications, and online information systems, create conditions in which individuals are repeatedly exposed to stimuli that demand attention, evaluation, and response. This sustained engagement may result in prolonged activation of cognitive and emotional processing systems, contributing to increased perceived stress and reduced psychological recovery.

Social media platforms represent one of the most extensively studied sources of digital stress. These platforms facilitate constant social comparison, often involving idealized or selectively curated representations of others' lives. Upward social comparison has been associated with negative emotional outcomes, including reduced self-esteem and increased anxiety. Additionally, the phenomenon of fear of missing out (FOMO) contributes to persistent engagement with digital content and heightened psychological distress. Exposure to emotionally salient content, including negative news or interpersonal conflict, further amplifies emotional reactivity and may contribute to sustained stress responses.

Another key dimension of digital stress is the anticipation of notifications. Unlike traditional stressors, notification-related stress does not require the presence of an external stimulus. Instead, the expectation of incoming messages or updates is sufficient to induce a state of heightened cognitive vigilance. This phenomenon has been described as "continuous partial attention," in which individuals remain cognitively engaged with potential stimuli while performing other tasks. Empirical evidence indicates that notification anticipation is associated with increased cognitive load, attentional fragmentation, and elevated stress levels

From a neurobiological perspective, notification anticipation and social media engagement are linked to activation of dopaminergic reward pathways, particularly under conditions of variable or unpredictable reinforcement. This pattern of reinforcement has been shown to promote habitual checking behaviors and sustained interaction with digital devices. Concurrently, these processes may activate stress-related neural circuits, including regions involved in emotional regulation and threat processing.

Information overload represents an additional and increasingly relevant component of digital stress. The rapid expansion of digital information has resulted in environments in which individuals are exposed to large volumes of data that exceed cognitive processing capacity. This overload has been associated with mental fatigue, reduced decision-making ability, and impaired attentional control

Cognitive overload is further exacerbated by multitasking and frequent task-switching, which are common in digital contexts. Studies have shown that individuals engaging in media multitasking exhibit reduced cognitive control and increased susceptibility to distraction. These effects may contribute to sustained cognitive strain and increased psychological stress.

Importantly, the physiological consequences of digital stress are increasingly recognized. Exposure to digital stressors has been associated with activation of the sympathetic nervous system and alterations in autonomic regulation, including reduced heart rate variability. These physiological responses are indicative of increased stress and reduced recovery capacity.

While direct evidence linking digital stress to gut microbiome alterations remains limited, the activation of stress-related pathways—including the hypothalamic–pituitary–adrenal axis—provides a potential mechanistic link. Chronic activation of these pathways has been shown to influence gastrointestinal function, immune responses, and microbial composition.

3.3.1 Social Media and Emotional Stress

Social media platforms have become a dominant component of digital environments and are increasingly recognized as significant contributors to psychological stress. A growing body of research has demonstrated associations between social media use and increased levels of anxiety, depression, and perceived stress, particularly among adolescents and young adults.

One of the primary mechanisms underlying these effects is social comparison. Social media platforms often present curated and idealized representations of others' lives, which may lead individuals to engage in upward social comparison. This process has been associated with reduced self-esteem, increased negative affect, and heightened psychological distress (Keles et al., 2020). In addition, the phenomenon of fear of missing out (FOMO) has been identified as a key psychological driver of stress, characterized by the persistent concern that others may be experiencing rewarding events in one's absence.

Exposure to emotionally salient or negative content represents another important factor contributing to stress. Continuous exposure to distressing news, social conflict, or emotionally charged interactions may lead to increased emotional reactivity and sustained psychological arousal. This type of exposure can contribute to chronic low-grade stress, particularly when individuals are unable to disengage from digital content.

Experimental studies have demonstrated that social media feedback mechanisms, such as "likes" and comments, can significantly influence emotional processing. Neuroimaging research has shown that receiving positive social feedback activates reward-related brain regions, including the ventral striatum, suggesting that social media interactions engage dopaminergic pathways associated with reward and reinforcement (Sherman et al., 2016). This reinforcement mechanism may promote repetitive engagement with social media platforms, increasing exposure to stress-related stimuli.

Furthermore, patterns of excessive or compulsive social media use have been associated with disrupted sleep, reduced attention, and increased emotional dysregulation. These factors may further contribute to physiological stress responses, including activation of the HPA axis and increased cortisol secretion.

Although the relationship between social media use and physiological outcomes remains complex, current evidence indicates that social media exposure constitutes a significant and persistent source of psychological stress, with potential downstream effects on broader physiological systems.

3.3.2 Notification Anticipation and Continuous Partial Attention

The anticipation of digital notifications represents a distinctive and increasingly studied component of psychological stress associated with modern digital environments. Unlike traditional stressors, which are typically discrete and time-limited, notification-related stress is characterized by persistent expectancy and continuous cognitive engagement. This phenomenon contributes to what has been described as "continuous partial attention," a state in which individuals remain cognitively alert to potential incoming stimuli while simultaneously engaging in ongoing tasks.

The expectation of notifications has been shown to induce heightened psychological arousal, even in the absence of actual stimuli. Experimental studies suggest that the mere possibility of receiving messages or updates can activate attentional and emotional processing systems, leading to increased cognitive load and reduced task efficiency. This anticipatory state may result in frequent attentional shifts and fragmented cognitive processing, which are associated with increased perceived stress and reduced cognitive performance.

From a neurophysiological perspective, notification anticipation is linked to activation of dopaminergic reward pathways, particularly in response to unpredictable or variable reinforcement patterns associated with digital communication. This intermittent reinforcement may strengthen habitual checking behaviors and contribute to sustained engagement with digital devices. At the same time, anticipatory processes may activate stress-related neural circuits, including the amygdala and prefrontal cortex, which are involved in emotional regulation and threat detection.

Frequent interruptions and notification exposure have also been associated with increased sympathetic nervous system activity, reflected in physiological markers such as elevated heart rate and reduced heart rate

variability. These changes are indicative of heightened stress responses and reduced autonomic recovery. Over time, repeated activation of these systems may contribute to chronic low-grade stress.

Importantly, notification-related stress differs from acute stress responses in that it lacks a clear onset and resolution, resulting in prolonged activation of stress pathways. This pattern may promote sustained activation of the hypothalamic–pituitary–adrenal (HPA) axis, leading to repeated or prolonged cortisol release.

3.3.3 Information Overload and Cognitive Fatigue

Digital environments expose individuals to unprecedented volumes of information, often exceeding the cognitive processing capacity of the human brain. This phenomenon, commonly referred to as information overload, has been increasingly recognized as a significant contributor to psychological stress and cognitive fatigue.

Information overload occurs when the amount of incoming information surpasses an individual's ability to effectively process, evaluate, and integrate it. In digital contexts, this is driven by constant exposure to news feeds, social media content, emails, and real-time updates. Empirical studies have shown that excessive information exposure is associated with increased mental fatigue, impaired decision-making, and reduced attentional control.

From a physiological perspective, sustained cognitive load associated with information overload has been linked to activation of stress-related neural circuits, including the prefrontal cortex and limbic system. This activation may contribute to increased sympathetic nervous system activity and elevated levels of stress hormones, including cortisol. Prolonged exposure to such conditions may result in chronic low-grade activation of the hypothalamic–pituitary–adrenal (HPA) axis.

In addition, information overload has been associated with disruptions in attentional regulation and working memory, which may further contribute to cognitive strain. Studies suggest that frequent task-switching and multitasking—common in digital environments—can impair cognitive efficiency and increase perceived stress levels.

Emerging evidence also indicates that cognitive fatigue and sustained mental effort may indirectly influence physiological processes, including immune function and inflammatory responses. Although direct links between information overload and gut microbiome alterations have not yet been established, the involvement of stress-related neuroendocrine pathways suggests a potential connection mediated by the gut–brain axis.

3.4 Evidence Linking Stress Pathways to Microbiome Alterations

Although direct studies examining the effects of digital stress on the gut microbiome remain limited, a substantial body of evidence supports the role of stress-related physiological pathways in mediating microbiome changes.

Activation of the HPA axis has been consistently associated with increased intestinal permeability, immune activation, and alterations in microbial composition. Additionally, stress-induced inflammatory processes have been shown to influence gut microbiota.

3.5 Quantitative and Emerging Evidence

Recent studies have begun to quantify the impact of psychological stress on gut microbiome composition using advanced sequencing techniques and diversity indices. Measures such as the Shannon diversity index and operational taxonomic unit (OTU) richness have been used to assess microbial diversity under stress conditions. Several studies have reported significant reductions in microbial diversity associated with both acute and chronic stress exposure.

In addition to diversity changes, alterations in the relative abundance of major bacterial phyla have been observed. Stress-related shifts in the Firmicutes-to-Bacteroidetes ratio have been reported, alongside reductions in short-chain fatty acid-producing bacteria. These changes are associated with impaired gut barrier function and increased inflammatory signaling.

Emerging research utilizing high-throughput sequencing and multi-omics approaches has provided further insight into the functional consequences of microbiome alterations. These studies indicate that stress may influence microbial metabolic pathways, including those involved in neurotransmitter production and immune modulation.

Although direct quantitative studies examining digital stress remain limited, advances in microbiome analysis and digital behavior tracking may enable more precise investigation of this relationship in future research.

3.6 Summary of Findings

The reviewed literature demonstrates that:

- the gut–brain axis is mediated by interconnected neural, endocrine, immune, and metabolic pathways
- psychological stress consistently influences gut microbiome composition and diversity
- digital environments contribute to chronic psychological stress through multiple mechanisms
- established stress-related pathways provide a biologically plausible link between digital stress and microbiome alterations

3.7 Integrating Digital Stress into the Gut–Brain Axis Model

The integration of digital stress into the gut–brain axis framework has emerged as a developing area of research, supported by converging evidence from studies on psychological stress, neuroendocrine signaling, and microbiome dynamics. Although direct empirical studies specifically examining digital stress and gut microbiome alterations remain limited, multiple lines of evidence indicate that digital stressors may engage established physiological pathways known to influence gut–brain interactions.

Digital stressors, including social media exposure, notification anticipation, and information overload, have been shown to activate psychological stress responses characterized by increased emotional arousal, cognitive load, and sustained attentional engagement. These responses are associated with activation of stress-related neurobiological systems, particularly the hypothalamic–pituitary–adrenal (HPA) axis.

Activation of the HPA axis leads to the release of cortisol, which exerts multiple effects on gastrointestinal physiology. Elevated cortisol levels have been associated with increased intestinal permeability, modulation of immune function, and alterations in gastrointestinal motility. These physiological changes create conditions that may influence gut microbial composition and diversity.

In parallel, stress-induced immune activation has been shown to increase the production of pro-inflammatory cytokines, which can further disrupt gut barrier integrity and contribute to microbial imbalance. Inflammatory processes have been linked to shifts in microbial populations and reduced microbial diversity, consistent with features of dysbiosis.

Neural pathways also play a role in mediating the effects of stress on the gut. Alterations in vagal signaling associated with chronic stress may affect gastrointestinal function and microbiota–brain communication. Reduced vagal tone has been associated with impaired regulation of inflammation and gut homeostasis.

In addition to direct physiological mechanisms, behavioral factors associated with digital environments may contribute to gut microbiome alterations. These include disrupted sleep patterns, increased sedentary behavior, and changes in dietary habits, all of which have been independently associated with changes in microbial composition.

Recent advances in microbiome research, including high-throughput sequencing and multi-omics approaches, have enabled more detailed characterization of microbial communities and their functional roles. These methods have demonstrated that stress can influence microbial metabolic pathways, including those involved in neurotransmitter production and immune modulation.

Taken together, the available evidence indicates that digital stressors may interact with established gut–brain axis pathways through neuroendocrine, immune, and neural mechanisms. These findings suggest that digital stress may be integrated into existing models of gut–brain axis function.

4. Discussion (Revised and Expanded Version)

The present review integrates evidence from microbiome research, psychoneuroendocrinology, and digital behavior studies to examine the potential role of digital stress as a modulator of gut–brain axis function. The findings synthesized in the Results section indicate that psychological stress consistently influences gut microbiota composition through well-established neural, endocrine, and immune pathways. At the same time, digital environments have emerged as pervasive and persistent sources of psychological stress. Taken together, these observations support the hypothesis that digital stress may represent a novel environmental factor capable of influencing gut microbiome dynamics.

One of the central observations of this review is that digital stress differs qualitatively from traditional stressors. While classical stressors are often acute and time-limited, digital stress is characterized by continuous exposure, high frequency, and persistent cognitive engagement. Mechanisms such as social comparison, fear of missing out, notification anticipation, and information overload contribute to sustained psychological

arousal. This pattern of exposure may result in prolonged activation of stress-related physiological systems, particularly the hypothalamic–pituitary–adrenal (HPA) axis.

Activation of the HPA axis and subsequent cortisol release have been consistently associated with changes in gastrointestinal physiology. These include increased intestinal permeability, modulation of immune responses, and alterations in microbial composition. The reviewed literature demonstrates that stress-related changes in gut barrier function and inflammatory signaling are closely linked to reductions in microbial diversity and shifts in bacterial populations. These findings provide a biologically plausible pathway through which digitally mediated psychological stress could influence gut microbiome homeostasis.

Importantly, the Results section highlights multiple converging mechanisms that may mediate this relationship. Neural pathways, particularly vagal signaling, play a critical role in microbiota–brain communication and are known to be affected by chronic stress. Endocrine responses involving cortisol release influence gut permeability and immune activity, while immune-mediated processes contribute to inflammatory changes that shape microbial composition. In addition, microbial metabolites, including short-chain fatty acids and neurotransmitter precursors, provide feedback signals that further integrate gut and brain function.

The role of social media as a source of psychological stress is particularly noteworthy. Social comparison, exposure to emotionally salient content, and reward-driven engagement mechanisms have been shown to influence emotional regulation and stress responses. These processes are associated not only with psychological outcomes but also with physiological changes, including activation of stress pathways that are known to affect gut function. Similarly, notification anticipation and continuous partial attention contribute to sustained cognitive load and sympathetic activation, while information overload imposes additional cognitive and emotional demands.

Another important finding is that behavioral factors associated with digital environments may act synergistically with physiological mechanisms. Disrupted sleep patterns, altered dietary habits, and reduced physical activity are commonly associated with excessive digital engagement and have independently been linked to changes in gut microbiota composition. These factors may amplify the effects of stress-related pathways, further influencing gut–brain interactions.

Despite these converging lines of evidence, several limitations must be considered. Most notably, direct empirical studies examining the relationship between digital stress and gut microbiome alterations remain scarce. The conclusions of this review are therefore based primarily on indirect evidence derived from well-established relationships between psychological stress and gut physiology, as well as emerging research on digital stress. As such, the proposed integration of digital stress into the gut–brain axis framework should be considered a hypothesis-generating model rather than a definitive causal explanation.

In addition, digital stress is a heterogeneous construct that encompasses multiple dimensions, including emotional, cognitive, and behavioral components. The lack of standardized definitions and measurement tools presents challenges for research in this area. Future studies will require more precise operationalization of digital stress, as well as the development of objective measures of digital exposure.

The findings of this review have potential implications for both research and clinical practice. From a research perspective, there is a need for longitudinal and experimental studies that integrate digital behavior metrics with microbiome analysis. Advances in wearable technology and digital tracking provide new opportunities for collecting real-time data on both physiological and behavioral variables. From a clinical perspective, digital behavior may represent a modifiable factor influencing gut health, particularly in conditions associated with stress and dysbiosis, such as irritable bowel syndrome and mood disorders.

To our knowledge, this review represents one of the first attempts to integrate digital stress into the gut–brain axis framework. By synthesizing evidence across multiple disciplines, this work highlights the importance of considering contemporary environmental factors in the study of human physiology. As digital technologies continue to shape modern life, their impact on biological systems—including the gut microbiome—warrants increasing scientific attention.

5. Conclusions

This review highlights the potential role of digital stress as an emerging environmental factor influencing gut–brain axis function and gut microbiome composition. The evidence synthesized in this study demonstrates that psychological stress consistently affects gut physiology and microbial balance through established neuroendocrine, immune, and neural pathways. At the same time, digital environments have introduced novel forms of stress characterized by continuous exposure, high frequency, and sustained cognitive engagement.

Although direct empirical studies examining the relationship between digital stress and microbiome alterations remain limited, converging evidence supports a biologically plausible link mediated by activation of the hypothalamic–pituitary–adrenal axis, immune system modulation, and alterations in gut barrier function. These findings suggest that digital behavior may contribute to physiological processes traditionally associated with stress-related disorders.

Importantly, this review proposes an integrative framework linking digital stressors to gut microbiome dynamics, highlighting a previously underexplored intersection between digital behavior and biological regulation. This perspective emphasizes the need to consider modern lifestyle factors, including digital exposure, as potential determinants of health.

Future research should focus on longitudinal and experimental studies that combine objective measures of digital behavior with microbiome profiling and physiological markers. Such approaches may help clarify causal relationships and support the development of targeted preventive and therapeutic strategies.

In conclusion, as digital technologies continue to shape human behavior, their impact on physiological systems—including the gut microbiome—represents an important and evolving area of scientific inquiry.

Funding: No external funding was received for the preparation of this manuscript.

Conflict of Interest: The author declares no conflict of interest.

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