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IMPACT OF CHRONIC STRESS ON GASTROINTESTINAL PHYSIOLOGY AND HORMONAL HOMEOSTASIS

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ABSTRACT

Background: The gastrointestinal tract is an essential component of systemic physiological regulation, participating in nutrient sensing, hormonal secretion, immune surveillance, and communication with the central nervous system. Increasing evidence indicates that chronic stress may influence these functions through interactions among the autonomic nervous system, hypothalamic–pituitary–adrenal axis, intestinal microbiota, and enteroendocrine signaling pathways. Alterations in this integrated network may affect gastrointestinal motility, epithelial barrier function, appetite regulation, glucose metabolism, and inflammatory activity. **Objective:** This review aims to examine the physiological relationships between chronic stress and the gut–brain–endocrine axis, emphasizing the regulatory roles of intestinal hormones, vagal signaling, microbial metabolites, and gastrointestinal barrier integrity. **Materials and Methods:** A narrative synthesis of contemporary peer-reviewed literature was undertaken, focusing on the physiological mechanisms connecting stress responses with gastrointestinal and endocrine regulation. The analysis considered neural, hormonal, microbial, metabolic, and epithelial signaling pathways and distinguished established physiological mechanisms from emerging experimental findings. **Results and Discussion:** The literature synthesis indicates that communication between the gastrointestinal tract and central nervous system is mediated through coordinated neural, endocrine, immune, and microbial mechanisms. Chronic stress may influence gastrointestinal motility, intestinal permeability, microbial metabolic activity, and enteroendocrine signaling. Glucagon-like peptide-1, peptide YY, ghrelin, serotonin, and microbial metabolites contribute to the regulation of appetite, gastrointestinal function, and energy homeostasis. Stress-associated disturbances may modify the integration of these signals, although their magnitude and direction depend on individual characteristics, dietary factors, and the duration of exposure. **Conclusion:** The gut–brain–endocrine axis represents an important physiological interface through which stress-related signals interact with gastrointestinal and metabolic regulation. Understanding these interactions may contribute to a more comprehensive interpretation of stress-associated physiological disturbances and provide a foundation for future research into endocrine and gastrointestinal adaptation.

KEYWORDS: Gut–brain axis; Chronic stress; Enteroendocrine regulation; Gastrointestinal physiology; Intestinal microbiota; Vagus nerve; GLP-1; Ghrelin; Serotonin; Metabolic homeostasis.

1. INTRODUCTION

The maintenance of physiological homeostasis requires continuous communication among organ systems responsible for nutrient acquisition, energy utilization, neural integration, and endocrine regulation. Although the gastrointestinal tract has traditionally been examined primarily in relation to digestion and nutrient absorption, contemporary physiological research recognizes its broader role as a sensory, endocrine, immune, and metabolic organ.

The gastrointestinal system contains specialized epithelial cells, intrinsic neural networks, immune populations, and microbial communities that collectively

participate in the regulation of systemic biological functions. Through communication with the central nervous system and peripheral endocrine organs, the intestine contributes to appetite regulation, energy balance, glucose homeostasis, gastrointestinal motility, and behavioral responses to internal physiological conditions.

These interactions are commonly described through the gut–brain axis, a bidirectional communication system involving neural, hormonal, immune, and metabolic signaling pathways. The inclusion of intestinal microbial communities has expanded this concept into the microbiota–gut–brain axis, emphasizing the contribution of microbial metabolites and host–microbial interactions to physiological regulation.[1]

Stress represents an important modifier of this regulatory network. Physical and psychological stressors activate central neural circuits and neuroendocrine pathways that influence autonomic activity, gastrointestinal function, and peripheral metabolism. These responses may alter intestinal motility, secretion, visceral sensitivity, and epithelial barrier regulation.

The physiological consequences of stress are not uniform. Acute stress may temporarily modify gastrointestinal activity as part of a broader adaptive response, whereas prolonged stress exposure may contribute to persistent changes in neural signaling, microbial composition, and intestinal function.[2]

An important component of gut–brain communication is the enteric nervous system, which consists of interconnected neuronal networks located within the gastrointestinal wall. These networks regulate intestinal motility, secretion, local blood flow, and interactions with mucosal tissues.

Although the enteric nervous system can coordinate many gastrointestinal functions independently, it remains functionally connected to the central nervous system through autonomic pathways, particularly vagal and sympathetic signaling.

The vagus nerve provides an important communication route through which gastrointestinal sensory information reaches the brainstem. This information includes signals related to nutrient availability, mechanical distension, intestinal hormones, and local physiological conditions.

In parallel, central autonomic pathways influence gastrointestinal function by modifying motility, secretion, vascular activity, and interactions among epithelial, neural, and immune cells.[3]

The gastrointestinal tract also functions as a major endocrine organ. Enteroendocrine cells detect nutrients and other luminal signals and release biologically active substances that influence digestion, pancreatic endocrine secretion, appetite, and systemic metabolism.

Among these mediators, glucagon-like peptide-1 (GLP-1), peptide YY (PYY), cholecystokinin (CCK), ghrelin, and serotonin participate in complex regulatory networks that connect gastrointestinal activity with central and peripheral physiological responses.

Stress-related alterations in these pathways may influence feeding behavior, gastrointestinal motility, and metabolic homeostasis. However, the biological consequences depend on the characteristics of the stressor, nutritional status, microbial composition, and individual physiological responsiveness.

The intestinal microbiota represents another important component of this regulatory system. Microbial communities participate in the metabolism of dietary substrates and produce compounds capable of influencing intestinal epithelial function, immune signaling, and host metabolism.

Short-chain fatty acids, including acetate, propionate, and butyrate, are among the microbial metabolites that contribute to host physiological regulation. Their effects may involve intestinal epithelial cells, immune populations, and specialized receptors expressed in enteroendocrine and other tissues.

Stress-associated changes in gastrointestinal function may modify the intestinal environment and influence microbial activity. Conversely, microbial metabolites and host–microbial interactions may affect neural, immune, and endocrine signaling involved in stress responses.[4]

Despite increasing recognition of these relationships, important questions remain regarding the mechanisms through which chronic stress influences gastrointestinal endocrine regulation and the extent to which observed changes contribute directly to metabolic dysfunction.

The distinction between physiological adaptation and persistent regulatory disturbance is particularly important because not all stress-related changes in gastrointestinal activity represent disease.

The objective of this review is to examine the physiological mechanisms connecting chronic stress with the gut–brain–endocrine axis and to analyze how alterations in neural communication, enteroendocrine signaling, intestinal microbiota, and epithelial barrier function may influence systemic metabolic homeostasis.

2. MATERIALS AND METHODS

The present article was developed as a mechanism-oriented narrative review examining the interactions among gastrointestinal physiology, endocrine regulation, and stress-associated neural and metabolic responses.

The literature synthesis focused on contemporary peer-reviewed publications addressing the microbiota–gut–brain axis, intestinal neuroepithelial communication, gastrointestinal barrier regulation, stress-related inflammation, enteroendocrine hormones, and metabolic signaling.

The analytical framework was organized around five physiological domains: neural communication between the gastrointestinal tract and central nervous system; endocrine signaling originating from gastrointestinal tissues; microbial metabolism and host signaling; epithelial barrier and immune regulation; and the effects of stress-related regulatory changes on appetite and energy homeostasis.

Particular attention was given to the relationships among these domains rather than treating individual hormones, microbial metabolites, or neural pathways as isolated mechanisms.

The interpretation distinguished established physiological functions from associations observed in human studies and mechanisms primarily supported by experimental animal models.

A qualitative thematic synthesis was used to organize the evidence into interconnected physiological processes and to identify potential pathways through which chronic stress may influence gastrointestinal and endocrine function.

No original patient data, experimental measurements, or statistical analyses were generated. The present work is a narrative review and does not claim to represent an exhaustive systematic review or meta-analysis.

3. RESULTS AND DISCUSSION

3.1. The Gut–Brain–Endocrine Axis as an Integrated Physiological Network

The literature synthesis identifies the gut–brain–endocrine axis as a multidirectional regulatory system linking gastrointestinal sensory activity, neural integration, hormonal secretion, and metabolic function.

Communication within this system involves the enteric nervous system, vagal afferent pathways, autonomic efferent signals, intestinal endocrine cells, circulating mediators, and microbial metabolites.

Under normal physiological conditions, these pathways contribute to the coordination of digestive processes with systemic energy requirements.

Nutrient exposure in the gastrointestinal lumen activates specialized epithelial and neural mechanisms that communicate information about nutrient availability to peripheral organs and central regulatory networks.

These signals influence gastric emptying, pancreatic secretion, intestinal motility, satiety, and subsequent feeding behavior.

The gut–brain system is therefore not simply a pathway through which the intestine communicates with the brain. It represents an integrated physiological mechanism coordinating nutrient processing with metabolic and behavioral responses.

Recent research emphasizes the importance of neuroepithelial interactions in transmitting intestinal sensory information to neural circuits involved in systemic regulation.[5]

3.2. Chronic Stress and Autonomic Regulation of Gastrointestinal Function

The autonomic nervous system provides a major pathway through which stress-related central signals influence gastrointestinal physiology.

Sympathetic and parasympathetic pathways regulate gastrointestinal motility, secretion, vascular activity, and interactions among intestinal neural and epithelial structures.

During acute stress, increased sympathetic activity may modify digestive function as physiological resources are redistributed toward systems involved in immediate adaptation.

Changes in gastrointestinal activity may include altered motility, modified secretion, and changes in visceral sensory processing.

However, the direction of these responses is not identical throughout the gastrointestinal tract. Stress may inhibit some motor functions while enhancing others, depending on the region, neural pathways, and characteristics of the stimulus.

Prolonged stress exposure may influence autonomic regulation and contribute to persistent alterations in gastrointestinal function.

These effects may interact with intestinal immune activity, microbial metabolism, and endocrine signaling.

The physiological significance of autonomic regulation is therefore determined by its ability to coordinate gastrointestinal activity with broader systemic requirements rather than by maintaining constant digestive function under all conditions.[2,3]

3.3. Enteroendocrine Cells and Hormonal Communication

Enteroendocrine cells are specialized sensory and secretory epithelial cells distributed throughout the gastrointestinal tract.

Although they represent a relatively small proportion of intestinal epithelial cells, they participate in the detection of luminal nutrients and the regulation of multiple digestive and metabolic functions.

Different enteroendocrine populations release hormones and signaling molecules that influence gastrointestinal motility, pancreatic endocrine secretion, appetite, and nutrient absorption.

GLP-1, secreted predominantly by intestinal L cells, contributes to glucose-dependent insulin secretion, suppression of inappropriate glucagon secretion, delayed gastric emptying, and regulation of food intake.

PYY participates in postprandial satiety signaling and gastrointestinal regulation, whereas CCK contributes to pancreatic enzyme secretion, gallbladder contraction, and nutrient-related satiety responses.

Ghrelin, produced mainly by gastric endocrine cells, participates in appetite regulation and communicates information related to energy availability.

These hormones interact with central and peripheral neural pathways, including vagal afferent signaling.

The physiological importance of this system lies in its capacity to coordinate nutrient availability with endocrine and behavioral responses.

Emerging research indicates that intestinal endocrine cells can participate in rapid neuroepithelial communication, providing a functional link between luminal conditions and neural circuits.[5]

3.4. Vagal Signaling and the Regulation of Appetite

The vagus nerve is a major component of gastrointestinal sensory communication.

Vagal afferent neurons transmit information from the gastrointestinal tract to brainstem structures involved in autonomic and metabolic regulation.

These pathways respond to mechanical and chemical signals associated with food intake, gastric distension, intestinal nutrients, and gastrointestinal hormones.

Information transmitted through vagal circuits contributes to the regulation of meal size, satiety, gastric function, and energy balance.

GLP-1 and other intestinal mediators may influence these processes through direct and indirect interactions with neural pathways.

The functional relationship among vagal signaling, intestinal microbiota, and metabolic regulation has attracted increasing attention because disturbances in these pathways may be relevant to obesity and impaired glucose homeostasis.

However, the contribution of individual vagal mechanisms varies according to experimental conditions and the physiological process being investigated.

The available evidence supports the importance of vagal communication in gastrointestinal and metabolic regulation while also indicating that the specific mechanisms connecting microbial changes to human metabolic outcomes remain incompletely understood.[6]

3.5. Serotonin and Gastrointestinal Neuroendocrine Regulation

Serotonin is an important signaling molecule within the gastrointestinal tract.

A substantial proportion of peripheral serotonin is synthesized by enterochromaffin cells in the intestinal epithelium.

Within gastrointestinal tissues, serotonin participates in the regulation of intestinal motility, secretion, sensory signaling, and interactions between epithelial and neural structures.

Its physiological actions depend on receptor distribution and the specific cellular pathways involved.

Intestinal serotonin signaling is distinct from central serotonergic neurotransmission because circulating serotonin does not freely cross the intact blood–brain barrier.

Nevertheless, gastrointestinal serotonergic activity can influence gut–brain communication indirectly through neural, immune, and metabolic pathways.

Microbial metabolites and luminal conditions may modify intestinal serotonin-related signaling, providing another mechanism through which the intestinal environment interacts with host physiology.

Stress-associated changes in autonomic activity and gastrointestinal function may influence this regulatory network.

However, the relationship between chronic stress and intestinal serotonin activity is complex and should not be interpreted as a uniform increase or decrease in serotonin production across all physiological conditions.

3.6. Intestinal Microbiota and Microbial Metabolic Signaling

The intestinal microbiota contributes to physiological regulation through the transformation of dietary substrates and production of biologically active metabolites.

Microbial fermentation of certain dietary fibers produces short-chain fatty acids, including acetate, propionate, and butyrate.

These metabolites participate in intestinal epithelial metabolism and may influence immune signaling, enteroendocrine activity, and systemic metabolic pathways.

Butyrate is particularly important as an energy substrate for colonocytes and contributes to the maintenance of normal colonic epithelial function.

Short-chain fatty acids can also interact with host receptors involved in metabolic and inflammatory regulation.

The composition and metabolic activity of intestinal microbial communities are influenced by diet, medication exposure, environmental conditions, host physiology, and gastrointestinal transit.

Chronic stress may modify some of these factors through changes in autonomic signaling, intestinal motility, and host inflammatory activity.

Consequently, stress-associated microbial alterations may influence host physiology through several interacting mechanisms.

However, microbial composition alone does not establish functional consequences. Similar taxonomic changes may have different metabolic implications depending on the microbial ecosystem and host environment.

Current research therefore increasingly emphasizes microbial functional activity and metabolite production rather than relying exclusively on the relative abundance of individual bacterial groups.[4,7]

3.7. Stress, Intestinal Barrier Integrity, and Inflammatory Regulation

The intestinal epithelial barrier plays a fundamental role in separating luminal contents from internal tissues while allowing regulated nutrient and water transport.

Barrier integrity depends on epithelial cell function, intercellular junctional complexes, mucus production, immune surveillance, and interactions with resident microbial communities.

Tight junction proteins contribute to the regulation of paracellular permeability.

Under normal conditions, selective epithelial permeability supports nutrient absorption while restricting uncontrolled movement of potentially harmful luminal substances.

Stress-related neural and endocrine signals may influence epithelial barrier function through interactions involving autonomic pathways, immune mediators, and intestinal microbial activity.

Experimental and clinical evidence suggests that certain stress conditions may be associated with changes in intestinal permeability and inflammatory signaling.

However, these effects are influenced by the duration and intensity of stress, the characteristics of the study population, and the methods used to assess barrier function.

Alterations in intestinal permeability should therefore not automatically be interpreted as evidence of systemic disease.

Recent reviews emphasize that epithelial barriers are active physiological interfaces involved in communication among microbial, neural, and immune systems.[8]

3.8. Stress-Associated Inflammation and Gut–Brain Communication

Immune signaling provides an important connection between intestinal physiology and central stress-related regulation.

The gastrointestinal mucosa contains extensive immune populations that interact with epithelial cells, microbial communities, and local neural structures.

Under normal physiological conditions, these interactions contribute to microbial tolerance, tissue defense, and the maintenance of intestinal integrity.

Stress-related alterations in neural and endocrine signaling may influence mucosal immune activity.

In turn, changes in intestinal inflammatory signaling may affect neural communication and systemic physiological regulation.

Cytokines and other inflammatory mediators can influence peripheral neural activity and communicate with central regulatory systems through several pathways.

This bidirectional relationship creates the possibility that persistent disturbances in intestinal physiology may interact with stress-related neural and endocrine responses.

However, the available evidence does not establish that intestinal inflammation is a universal consequence of psychological stress.

The relationship is context-dependent and varies according to individual characteristics, microbial

activity, environmental factors, and underlying health conditions.

Contemporary research supports a model in which stress, intestinal barrier function, microbial activity, and inflammatory regulation form an interacting network rather than a single linear causal pathway.[2,9]

3.9. Gastrointestinal Hormones and Glucose Homeostasis

The gastrointestinal endocrine system contributes substantially to the regulation of glucose metabolism.

Following nutrient ingestion, intestinal hormones participate in the coordination of pancreatic endocrine secretion and nutrient handling.

GLP-1 is particularly relevant because it enhances glucose-dependent insulin secretion and contributes to postprandial glucose regulation.

These effects are integrated with changes in gastric emptying, glucagon secretion, and central appetite-related pathways.

The physiological response to nutrient intake therefore depends on interactions among gastrointestinal sensory mechanisms, endocrine secretion, pancreatic function, and neural regulation.

Chronic stress may influence glucose homeostasis through several pathways, including autonomic activation, altered feeding behavior, sleep disruption, and changes in peripheral insulin sensitivity.

These effects may interact with gastrointestinal hormonal regulation, although direct evidence linking chronic psychological stress to consistent changes in endogenous incretin secretion remains limited.

The relationship between the microbiota–gut–brain axis and metabolic disease is therefore best interpreted as a multifactorial physiological interaction rather than a single endocrine disturbance.[6]

3.10. Stress, Feeding Behavior, and Energy Balance

Feeding behavior is regulated through interactions among central neural networks, peripheral hormones, gastrointestinal sensory signals, and environmental influences.

Hunger and satiety are not determined by a single circulating hormone. Instead, they emerge from the integration of multiple signals reflecting nutrient availability, energy stores, gastrointestinal activity, and behavioral context.

Ghrelin contributes to hunger-related signaling, whereas GLP-1, PYY, and CCK participate in mechanisms associated with meal termination and satiety.

Stress exposure may influence feeding behavior through changes in neural processing, reward-related activity, autonomic function, and endocrine signaling.

However, stress does not produce a uniform feeding response. Some individuals experience reduced food intake, while others demonstrate increased consumption or changes in food preferences.

These differences may depend on stressor characteristics, previous experiences, dietary environment, and individual metabolic regulation.

Microbial metabolism may also interact with dietary factors and gut–brain signaling.

Recent evidence emphasizes the importance of considering diet, intestinal microbial activity, and neural regulation together when examining the relationship between gastrointestinal physiology and behavioral responses.[10]

3.11. Physiological Adaptation Versus Persistent Gastrointestinal Dysregulation

An important distinction in stress physiology concerns the difference between temporary gastrointestinal adaptation and persistent functional disturbance.

Short-term changes in motility, secretion, appetite, or visceral sensitivity may represent coordinated responses to changing physiological demands.

Such alterations do not necessarily indicate pathological dysfunction.

However, persistent disturbances may interfere with nutrient handling, gastrointestinal comfort, and the normal coordination of digestive and metabolic functions.

The transition from adaptation to dysfunction may involve interactions among autonomic regulation, visceral sensitivity, epithelial barrier activity, microbial metabolism, and endocrine signaling.

Importantly, these mechanisms do not operate independently. Changes in gastrointestinal transit may influence microbial activity, while altered nutrient exposure may affect enteroendocrine secretion and neural signaling.

The physiological consequences therefore depend on the combined activity of several interacting pathways.

This integrated perspective may help explain why individuals exposed to apparently similar stressors can experience different gastrointestinal and metabolic responses.

3.12. Implications for Future Physiological and Endocrine Research

The reviewed literature supports the need for integrated investigations of stress-related gastrointestinal and endocrine regulation.

Studies focusing exclusively on individual hormones, microbial taxa, or isolated gastrointestinal parameters may not adequately capture the complexity of the gut–brain–endocrine axis.

Future research should consider simultaneous assessment of autonomic activity, gastrointestinal function, enteroendocrine signaling, microbial metabolic profiles, and systemic metabolic indicators.

Longitudinal studies may be particularly valuable for distinguishing temporary physiological adaptation from persistent regulatory disturbances.

Human studies should also account for dietary intake, medication exposure, sleep patterns, sex-related physiological differences, and pre-existing metabolic conditions.

The development of standardized methods for evaluating intestinal permeability, microbial functional activity, and endocrine responses may improve comparability across investigations.

An additional priority is the identification of causal relationships between stress-related changes in intestinal physiology and systemic metabolic outcomes.

Although experimental studies provide important mechanistic insights, findings from animal models require careful interpretation before being generalized to human physiology.

An integrated systems-physiology approach may therefore provide a more informative foundation for understanding the interactions among stress exposure, gastrointestinal function, and endocrine homeostasis.

4. CONCLUSION

The gut–brain–endocrine axis represents an important component of physiological homeostasis through which neural, hormonal, microbial, and metabolic signals are integrated.

The gastrointestinal tract contributes to systemic regulation not only through digestion and nutrient absorption but also through sensory communication, endocrine secretion, immune regulation, and microbial metabolic activity.

Chronic stress may influence this integrated network by modifying autonomic signaling, gastrointestinal motility, intestinal barrier function, and interactions among microbial and host-derived mediators.

Enteroendocrine hormones, including GLP-1, PYY, CCK, and ghrelin, participate in the coordination of nutrient availability, appetite regulation, and metabolic responses. Vagal pathways and intestinal serotonin signaling provide additional mechanisms linking gastrointestinal activity with neural regulation.

The physiological consequences of stress-associated changes depend on their duration, intensity, and interaction with individual biological characteristics.

Importantly, temporary alterations in gastrointestinal function may represent normal adaptation, whereas persistent disturbances may contribute to impaired regulatory coordination.

The present review highlights the need to examine stress-related gastrointestinal and endocrine responses as components of an interconnected physiological system rather than isolated biological phenomena.

Future investigations integrating gastrointestinal physiology, neuroendocrine regulation, microbial functional activity, and metabolic measurements may improve understanding of the mechanisms through which stress influences systemic homeostasis.

Such research may contribute to the identification of physiologically meaningful indicators of gastrointestinal and metabolic adaptation and support a more comprehensive understanding of the relationships among stress, endocrine regulation, and human health.

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