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EDITORIAL

Bidirectional relationship between gastrointestinal cancer and depression: The key is in the microbiota-gut-brain axis

Bryan Adrian Priego-Parra, Jose Maria Remes-Troche

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Abstract

In this Editorial, we review the recent publication in the *World Journal of Gastroenterology*, which explores the complex relationship between depression and gastric cancer and offers perspectives. Key topics discussed include the microbiota-gut-brain axis, dysbiosis, and the influence of microbial metabolites in homeostasis. Additionally, we address toxic stress caused by hypothalamic-pituitary-adrenal axis dysregulation, psychological assessments, and future research directions. Our Editorial aims to expand the understanding of the bidirectional relationship between depression and gastrointestinal cancer.

Key Words: Gastric cancer; Depression; Psychogastroenterology; Microbiota-gut-brain axis; Mood disorders; Dysbiosis

Core Tip: Gastrointestinal cancer and depression are intricately linked in a complex, bidirectional relationship, likely mediated by the microbiota-gut-brain axis through partially understood mechanisms. Chronic inflammation, microbiome alterations, and disruptions to intestinal and blood-brain barriers are pivotal in this interaction. Depression may emerge as a consequence of cancer diagnosis and treatment, while also accelerating cancer progression through neuroendocrine and inflammatory pathways. Early psychological assessments, combined with interventions, such as cognitive-behavioral therapy and microbiota-based therapies, can optimize clinical outcomes, enhance treatment adherence, and improve quality of life. Tailored, culturally sensitive strategies are crucial for advancing precision medicine.

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INTRODUCTION

We read with great interest the Editorial "Depression weights in patients with gastric cancer: Bibliometric analysis as a weapon to chart the future of research" by Pellegrino and Gravina[1]. This article offers compelling insights into Liu *et al*'s groundbreaking bibliometric study exploring the bidirectional relationship between gastric cancer and depression[2].

Globally, gastric cancer is one of the most common malignant tumors, ranked as the fifth most frequent malignancy worldwide and the fourth leading cause of cancer-associated death. The incidence of gastric cancer varies across countries, and like most cancers, the majority of cases are diagnosed at advanced stages[3]. Known risk factors for gastric cancer include *Helicobacter pylori* infection, smoking, alcohol consumption, exposure to chemicals, and dietary patterns, among others. However, recent research has highlighted the role of mental disorders in increasing the odds of developing gastric cancer and colorectal cancer. Hypotheses suggest that chronic inflammation, oxidative stress, and lifestyle patterns associated with mood disorders, such as inadequate diet, substance abuse, and sedentary behavior, may contribute to this relationship[4].

Depression and related mood disorders constitute a global crisis. Their alarming growth over the past few decades, especially after the coronavirus disease 2019 pandemic, has profoundly impacted physical health and strained healthcare systems. These disorders lead to increased rates of disability, absenteeism, presenteeism, and comorbidities, resulting in significant economic burdens on healthcare systems and a diminished quality of life[5,6].

Understanding depression is crucial, not only as a psychological condition, but also as a multifaceted biopsychosocial phenomenon that could both precipitate and result from various adaptive mechanisms and pathologies. Beyond its psychological manifestations, depression encompasses social, genetic, epigenetic, and neurostructural elements. These components are influenced by interactions within the microbiota-gut-brain axis (MGBA), highlighting the complex interplay between the microbiome, mental health, and physical health states[7-9].

MGBA

The gastrointestinal (GI) tract and the central nervous system are interconnected through the vagus nerve. This communication is mediated by cross-kingdom interactions involving diverse species of bacteria, viruses, bacteriophages, fungi, parasites, archaea, and other protozoans, along with the metabolites they produce[10,11].

One significant group of microbial metabolites is short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate, which result from the fermentation of dietary fiber. SCFAs have far-reaching systemic effects, impacting immune responses, inflammation, and neurological functions[12]. Beyond SCFAs, other microbial metabolites, including neurotransmitters (*e.g.*, serotonin, dopamine, and gamma-aminobutyric acid), bile acids, and vitamins, play crucial roles in gut-brain communication[13]. Additionally, bacterial metabolites and endotoxins, such as lipopolysaccharides, can influence inflammatory pathways and disrupt immune homeostasis. The gut microbiota can influence the permeability of both the intestinal epithelial barrier and the blood-brain barrier (BBB), thereby modulating the central nervous system's exposure to various compounds.

The integrity of the intestinal epithelial barrier, which selectively regulates molecular passage, is maintained by components, such as the mucus layer, desmosomes, interepithelial junctions, and various tight junction proteins. Disruptions in the microbial equilibrium, known as intestinal dysbiosis, can lead to increased intestinal permeability. This condition may enable the translocation of microbiota-derived metabolites into the peripheral circulation, triggering immune activation and systemic inflammation. Furthermore, alterations in membrane permeability could compromise the BBB, potentially allowing these metabolites to access the brain and induce astrocytic dysfunction, oxidative stress, and neuroinflammation[14]. These processes are implicated in the onset of various diseases, including GI disorders, neuropsychiatric conditions, and chronic metabolic diseases. They may drive a persistent proinflammatory cycle[15] that could lead to different types of cancers in a subset of individuals[16].

Moreover, the role of tight junction proteins in cancer pathogenesis, tumor growth, and metastasis has been described [17], offering new therapeutic opportunities or prognostic markers. Additionally, neurotransmitters synthesized by the gut microbiota can influence brain function directly, *via* the circulatory system, or indirectly, by modulating vagus nerve activity. Furthermore, the microbiota can shape the tumoral microenvironment, predict drug efficacy and toxicity, modulate the immunological response, and serve as a cancer-related biomarker[18,19].

TOXIC STRESS AND GI CANCER

Toxic stress refers to the prolonged and excessive activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system, triggered by chronic, severe, or recurrent exposure to stressors, without adequate emotional or social buffering. This persistent activation disrupts homeostasis, leading to long-term physiological dysregulation and increased disease risk.

A primary source of toxic stress is adverse childhood experiences (ACEs), defined as preventable, potentially traumatic events occurring during childhood or adolescence. ACEs encompass various forms of abuse, neglect, and household dysfunction, which disrupt normal developmental processes and initiate toxic stress responses. When these stressors persist or recur, they dysregulate the HPA axis, promoting chronic inflammation, metabolic disturbances, and gut dysbiosis. These physiological disruptions accumulate over time, contributing to systemic, cellular, and neurological damage. Furthermore, the physiological responses to ACEs may vary by sex, reflecting distinct biological mechanisms [20].

ACEs are increasingly recognized for their role in the pathogenesis and clinical presentation of several chronic conditions. For instance, ACEs have been linked to heightened risks for cancer, including GI malignancies, as well as mental health disorders and other neuropsychiatric conditions[21,22]. Survivors of GI cancers with a history of ACEs often report poorer quality of life, suggesting that early-life trauma can exacerbate disease outcomes[23].

Emerging research further links ACEs with accelerated biological aging, metabolic syndrome, and epigenetic modifications[24,25], underscoring the need for integrative approaches to healthcare. A psychoneuroimmunoendocrinological framework, which combines mental, neurological, immune, endocrine, and microbiome-related factors, offers a promising avenue for developing more effective therapeutic strategies. Such interdisciplinary approaches may enhance outcomes across multiple health domains.

Behavioral patterns related to diet, alcohol consumption, and tobacco use are often established during childhood and adolescence, periods that frequently coincide with the occurrence of ACEs. These behaviors, shaped by biological, social, and cultural influences, often function as maladaptive coping strategies in response to toxic stress, further increasing the risk of GI cancers.

The microbiome, which is also shaped by chronic stress and ACEs, plays a central role in regulating behaviors, such as social interactions[26], food intake, energy balance[27,28], and substance cravings[29-31], including those for alcohol, tobacco, fructose, and ultra-processed foods[32]. This bidirectional relationship between ACEs, behavioral patterns, and microbiome dynamics highlights how early-life adversities can predispose individuals to long-term health risks, including cancer.

The physiological consequences of toxic stress can persist across the lifespan[25]. Additionally, emerging evidence suggests that these effects can be transmitted across generations through complex epigenetic, biological, and psychosocial mechanisms[33]. Given the profound implications for individual and public health, promoting healthy childhood development and addressing the impact of ACEs should be prioritized worldwide, to reduce the burden of chronic diseases.

DEPRESSION AND GI CANCER

GI cancers frequently lead to dysbiosis due to such factors as tumor-related inflammation, changes in GI secretions, and the effects of treatments like chemotherapy and radiation. Dysbiosis can promote chronic inflammation, facilitating carcinogenic processes and compromising immune functions. Moreover, disruption in the microbial balance can influence the synthesis of neurotransmitters and other compounds that affect brain function and cognition. The inflammatory state induced by dysbiosis may enable inflammatory cytokines to permeate the brain, potentially causing depressive symptoms. Meanwhile, depression itself, by causing a chronic inflammatory state with dysregulation of catecholamines, may influence cancer progression[34]. Consequently, both GI cancer and depression trigger cascades that cause systemic dysregulation and mutually reinforce each other in a complementary manner.

A recent systematic review and meta-analysis conducted by Cheng *et al*[35] demonstrated a 51% increased risk of depression following a colorectal cancer diagnosis, correlating with higher mortality and diminished quality of life. Meanwhile, Kouhestani *et al*[36] found a 37% pooled prevalence of depression among gastric cancer patients. These findings emphasize the critical need for integrated mental health interventions throughout the GI cancer care continuum.

Although a bidirectional relationship between GI cancer and depression is hypothesized, definitive confirmation remains challenging due to limited studies and numerous confounding variables. Observations indicate that individuals with gastric cancer, particularly women, have a heightened risk of developing depression[4,37]. Research has linked depression with an increased incidence of gastric adenocarcinoma and colorectal cancer[38] and suggests that depression may even expedite the progression of gastric cancer through various neuroendocrine mechanisms[39]. However, the controversy persists because no causal association between depression and GI cancer has yet been firmly established[40,41]. Nevertheless, depression could be the missing link between inflammatory mediators and cancer[42]. Despite the scarcity of studies, we need to acknowledge the dual role of depression in GI cancer, serving as a causative factor with clinical implications, as well as a consequence with direct and indirect mechanisms that are not fully understood, as elucidated by Christodoulidis *et al*[34].

PSYCHOLOGICAL ASSESSMENT AND FUTURE DIRECTIONS

Systematic psychological assessments should be integrated at all stages of GI cancer progression. Implementing psychological interventions has been shown to provide comprehensive health benefits. Studies have demonstrated that relaxation techniques[43] can enhance psychological, cognitive, social, and physiological aspects related to pain management and cancer-related therapy effects, whether conducted in person, with psychologists or trained healthcare professionals, or remotely, through cognitive-behavioral therapies facilitated by digital apps and virtual reality environments[44]. Beyond improving psychological well-being, these approaches can also trigger neurostructural alterations that reduce inflammation, thereby improving functional health and health-related quality of life[45,46].

We advocate for the continued investigation and validation of anxiety and depression scales across various types of GI conditions in different populations. Pellegrino and Gravina[1] have shared their experience using Beck's anxiety and depression inventory. In parallel, our group in Mexico has been working with the hospital anxiety and depression scale [47], which has been validated in diverse settings, both within hospitals and in broader contexts. It has proven effective in identifying anxious and depressive states in cancer patients[48]. However, there are other useful scales whose results and reliability may vary, depending on the study group and ethnicity. The cultural variation in how depression is experienced and understood is critical. Moreover, evolving social dynamics are reshaping public perceptions of depression. Future research should explore how these cultural shifts influence the presentation, diagnosis, and treatment of depression, as well as patient engagement with mental health services. Adapting screening tools and interventions to align with these changes will be essential for maintaining their relevance and effectiveness.

Innovative therapies, such as modulation of the MGBA through gut-brain therapies, prebiotics, probiotics, synbiotics, postbiotics, and psychobiotics, as well as fecal transplantation and nutritional interventions, hold promise not only for understanding underlying mechanisms, but also for pioneering holistic therapies that impact both mental health and cancer[49-51]. Novel techniques, such as artificial intelligence and new algorithms, promise to enhance the effectiveness and precision of predicting depression risk in cancer patients[52,53].

Furthermore, it is crucial to address health-related stigmas and increase awareness. The need to carry out further research is particularly pertinent in Latin American and African countries, where research is scarce, and cultural variations can influence the clinical presentations of cancer and depression. Tailoring interventions to meet specific cultural requirements will enhance patient outcomes across diverse populations.

Future research should broaden its scope to include measuring the impact of depression intensity and duration and incorporating advanced imaging techniques, functional assessments, and a wide range of psychological scales, thus enabling comprehensive head-to-head comparisons to identify the most effective tools for diverse patient demographics. Additionally, examining other cognitive processes, such as catastrophizing, neuroticism, hypervigilance, appraisal, coping strategies, resilience, and individual emotional and belief systems is important. This holistic approach, based on the biopsychosocial model[54], is key to ensuring optimal clinical outcomes, consistent treatment adherence, improved doctor-patient interactions, and enhanced overall quality of life for patients. By considering the cultural, biological, and psychosocial aspects of health, we can address the complex nature of illness and provide more personalized care.

CONCLUSION

The MGBA mediates the intricate relationship between GI cancer and depression, influencing immune response, neurodevelopment, inflammation, behaviors, substance abuse, dietary patterns, and lifestyle habits. Although the exact mechanisms are not fully understood, extensive global efforts aim to discover the molecular connections. The interaction between the microbiota and both the brain and digestive systems underscores the need for the international research community to continue investigating anxiety, depression, mood disorders, and cognitive states linked to GI diseases. This focused research is crucial for developing novel therapeutic and preventive strategies essential for the progress of precision medicine.

FOOTNOTES

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