REVIEW ARTICLE

RECORDS OF PHARMACEUTICAL AND BIOMEDICAL SCIENCES

Gut Microbiota and its Implication in Disease: A Mini-Review

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Abstract

The gut microbiota is critical to human health and has been increasingly implicated in a number of diseases. This mini-review sought to summarize the associations with gastrointestinal disorders, hepatic disorders, metabolic disorders, cardiovascular disorders, immune disorders, and neuropsychiatric disorders. Dysbiosis is linked to disorders such as IBS, IBD, NAFLD, obesity, diabetes, CVD, asthma, and neurological disorders, by mechanisms such as altered fermentation, immune activation, and loss of barrier function. Although global studies report these associations, there are fewer data from Egypt. Localized aspects such as diet and environment likely promote unique microbiota, and there is a need for additional relevant studies.

Keywords: Gut microbiota - Dysbiosis - Metabolic disorders - Inflammatory bowel disease - Gut brain axis.

1. Introduction

The human gut contains trillions of microorganisms, which together make up a dynamic and complex ecosystem known as the gut microbiota **(Afzaal et al., 2022)**. This microbial community has key roles in digestion, immune activity, metabolism, and the biochemical processes that ultimately drive higher process signalling **(Wan et al., 2023)**. The past few years have seen an explosion of research linking the composition and function of gut microbiota to the pathogenesis of disease **(Khalil et al., 2024)**. Dysbiosis **(Figure 1)** (microbial imbalance) is increasingly implicated in the pathogenicity of gastrointestinal, hepatic, metabolic, cardiovascular, immune-mediated, and neuropsychiatric disorders **(Hrncir, 2022)**. Dysbiosis may play a role in disease through a variety of mechanisms (e.g., unintended effects of altered fermentation and SCFA production, altered intestinal barrier integrity, systemic inflammation, and dysfunction of host-microbe signaling mechanisms, including the gut–brain axis **(Weiss & Hennet, 2017)**.

With the increased emphasis on microbiome science, this mini-review presents a concise summary of the most recent and relevant findings relating gut microbiota to the pathogenesis of common chronic and non-communicable diseases, as well as global perspectives, but recognizing the need for more local studies, especially from under-represented regions

like Egypt, where distinct dietary patterns and environmental exposures are expected to lead to distinct microbial profiles.

Since the gut microbiota has a remarkable impact on host physiology **(Bajinka et al., 2020)**, it is not surprising that changes in gut microbiota have been associated with many chronic diseases. The gastrointestinal tract and liver diseases have been widely studied due to their association with dysbiosis and gut microbiota changes which has been shown to alter mucosal function, immune tolerance, and metabolic interactions **(Toor et al., 2019)**. These chronic gastrointestinal diseases often serve as models for understanding the impact of dysbiosis on disease, illustrating how the gut-brain microbiome is capable of contributing to local and systemic pathogenesis **(Kandpal et al., 2022)**. The examples presented in the following sections will focus on representative examples of diseases that affect the gut and are connected with gut microbiota changes, starting with irritable bowel syndrome (IBS) and other gastrointestinal and hepatic diseases.

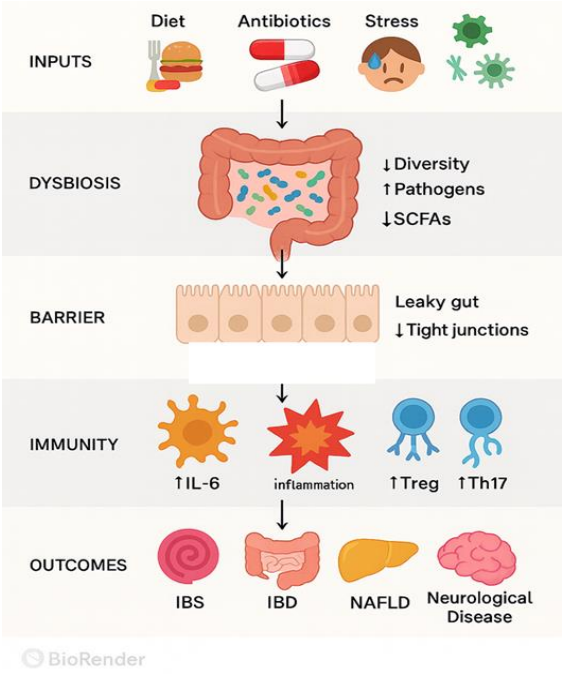

Figure 1: Consequences of gut microbiota dysbiosis on host immunity and disease.
Various environmental and clinical factors such as diet, antibiotics, stress, and infection contribute to gut microbial imbalance, leading to compromised epithelial barrier integrity and immune dysregulation. These changes promote the development of conditions such as irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), non-alcoholic fatty liver disease (NAFLD), and neuropsychiatric disorders. *This figure was created by the authors using BioRender.com.*

2. Gastrointestinal and hepatic diseases

2.1 Irritable bowel syndrome (IBS)

Irritable bowel syndrome (IBS) is a functional disorder characterized by abnormal bowel movements, flatulence, and abdominal pains. Increased fermentation and gas formation can be triggering factors for the symptoms. Higher concentrations of SCFA in IBS induce release of serotonin inside the intestine and subsequently increase intestinal transit **(Chen et al., 2022)**. Although it is not a life-threatening condition, it has been estimated that IBS somehow affects between 10 and 15% of the population**,** strongly related to the gut-brain axis **(Campbell et al., 2025)**. Changes in the gut microbiota could in fact be either predisposing or causative for IBS, with both qualitative and quantitative alterations having been identified **(Carco et al., 2020)**. Dysbiosis may enhance harmful bacterial adherence to the gut wall **(Meng et al., 2020)**. Studies report increased Firmicutes—especially *Clostridium*, *Ruminococcus*, and *Dorea*—and decreased *Bacteroides fragilis* and *Ruminococcus albus* in IBS patients **(Altomare et al., 2021; Surdea-Blaga et al., 2024)**.

2.2 Inflammatory bowel disease (IBD)

Inflammatory bowel disease (IBD) comprises long-term, immune-mediated inflammatory disorders of the digestive system **(Bezzio et al., 2022)**. It is thought that both genetics and the environment put together are involved in the development of IBD: stress, ever so slight variations in sleep, antibiotics, all things hygiene, diet, and smoking. The host genome also affects microbiota composition in the gut. The two main types are Crohn's disease and ulcerative colitis **(D.-Y. Kang et al., 2023)**.

While UC is characterized by continuous inflammation involving the colon and rectum, CD goes on to potentially affect an entire patchy gut (Kudelka et al., 2016). Microorganism–diet correlations are strong in IBD risk **(Weng et al., 2019),** and a fruit- and vegetable-rich diet increases SCFAs and reduces CD risk **(van der Merwe, 2021)**.

IBD is linked to a weakened mucosal barrier that permits microflora penetration and inflammation. Changes in abundance have mainly been linked to dysbiosis of the intestinal microbiota, such as the decreases of Firmicutes and Bacteroidetes **(Barbara et al., 2021)**, while reductions of *Roseburia* and *Faecalibacterium prausnitzii* have been recorded in UC and CD patients **(Mohebali et al., 2023).**

3. Liver diseases

Due to some of its factors and for some of its forms, liver disease has become rather prevalent throughout human history; evidences have clearly shown the crucial aspect of the gut microbiome in disease initiation and progression. These fatty liver diseases are associated with alcohol, metabolic syndrome, and obesity, with the microbiota, lifestyle, and diet influencing their course. Non-alcoholic fatty liver disease (NAFLD) is produced by the accumulation of triglycerides in hepatocytes and is carcinogenic to hepatocytes and cirrhogenic to liver parenchyma. Although NAFLD's pathophysiology is not fully understood, gut microbiota alterations are believed to contribute significantly **(Hrncir et al., 2021)**. Studies report elevated *E. coli* levels and reduced Firmicutes and Bacteroides in fatty liver disease. NAFLD may arise from increased energy harvest due to high SCFAs and a low Bacteroidetes-to-Firmicutes ratio **(Ghoshal et al., 2020)**. Dysbiosis also reduces butyrate production, which creates increased triglyceride accumulation via lipoprotein lipase activity **(Zwartjes et al., 2021)**. Chronic alcohol consumption supports the development of Gram-negative bacteria, which decrease gut barrier integrity, paving the way for the translocation of lipopolysaccharide and microbial metabolites to the liver, and subsequent inflammation and injury **(Dukić et al., 2023)**.

4. Metabolic Diseases

4.1 Obesity

Obesity is defined as a BMI > 30 kg/m², and an estimated 2 billion people globally are overweight **(Lean, 2023)**. Obesity is associated with metabolic syndrome, which is characterized by a cluster of conditions such as diabetes, osteoarthritis, dyslipidemia, liver steatosis, insulin resistance, and cancer **(Mili et al., 2021)**. There are many causes for obesity, ranging from environmental and behavioral factors to genetics. The gut microbiota is critical to the initiation and progression of obesity **(Cheng et al., 2022)**. Obese people have a higher degree of fermentative ability and energy-harvesting capabilities than non-obese humans. An increased Firmicutes: Bacteroidetes ratio drives increased SCFA production and higher levels of microbial genes that drive polysaccharide degradation and carbohydrate metabolism **(Sutoyo et al., 2020)**. Obesity is also associated with higher levels of *Porphyromonas*, *Campylobacter*, *Bacteroides*, *Staphylococcus*, *Parabacteroides*, *Dialister*, and *Ruminococcus*, and lower levels of *Methanobrevibacter*, *Coprococcus*, *Bifidobacterium*, *Faecalibacterium*, *Akkermansia*, and *Lactobacillus* **(Murga-Garrido et al., 2023)**.

4.2 Diabetes

Type 1 diabetes results from immune-mediated destruction of insulin-producing pancreatic β-cells and typically appears early in life. This condition is linked to gut microbial organization, emphasizing its role in immune system development. The immune system–microbiota interaction influences type 1 diabetes risk **(Yau & Danska, 2024)**. Individuals with type 1 diabetes exhibit high Bacteroidetes levels, reduced lactate- and butyrate-producing bacteria, and decreased bacterial functional diversity **(Yuan et al., 2022)**.

Type 2 diabetes is a chronic medical condition characterized by insufficient insulin secretion or impaired ability of glucose to be utilized in the body, whether due to insulin resistance or loss of receptor responsiveness. The composition of gut microbiota are influenced by lifestyle factors, including host genetics, as well as the likelihood of developing type 2 diabetes **(Muscogiuri et al., 2025)**. Diabetic patients show higher concentrations of Proteobacteria, Bacteroidetes, and Firmicutes than healthy patients **(Ahmed et al., 2019)**.

LPS from Gram-negative bacteria are involved in the promotion of metabolic endotoxemia through their promotion of the secretion of pro-inflammatory cytokines as well as other variations of inflammatory mediators. Specifically, a high-fat diet, can increase blood levels of LPS and contribute to changes in existing gut microbiota **(Malesza et al., 2021)**.

Short-chain fatty acids (SCFA) created by gut microbes stimulate energy metabolism, by affecting the degradation of polysaccharides. Butyrate significantly improves insulin sensitivity by obstructing endotoxin translocation **(Figure 2)**. SCFA in metabolically appear to contribute significantly as substrates to gluconeogenisis and lipogenesis **(Zou et al., 2018)**.

Therefore, we see the change in the composition of microbiota in both types of diabetes, with each diabetes type including varying levels and alterations in the composition of gut microbiota. In type 1 diabetes the microbial composition is altered and varied at infancy or life stages hindered and diversity appeared reduced, while type 2 shows loss of microbial diversity drive by lifestyle factors across major phyla of bacteria **(Le Chatelier et al., 2013; Muscogiuri et al., 2025)**.

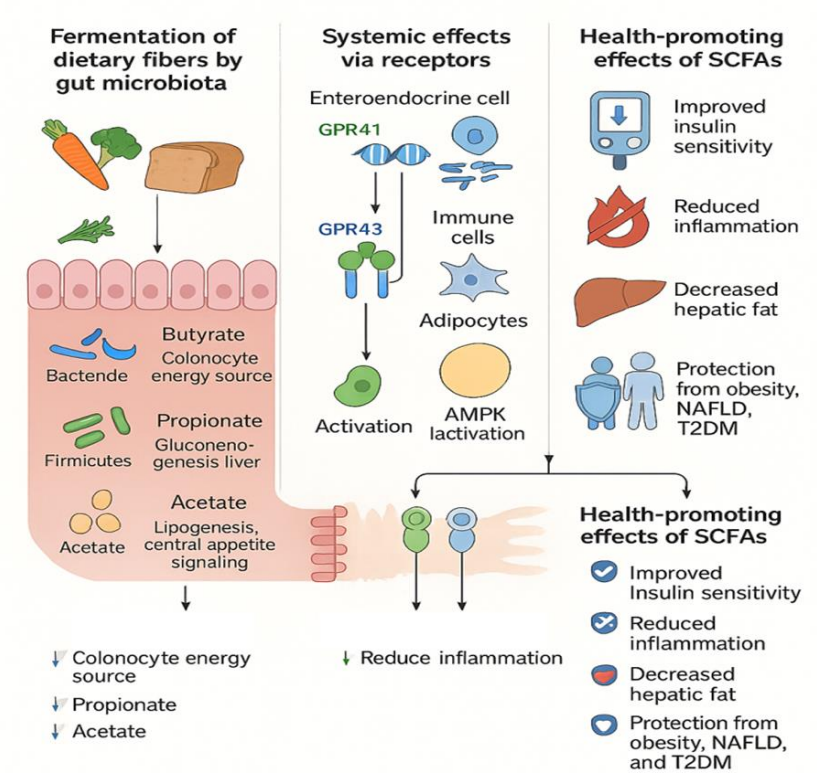

Figure 2: Short-chain fatty acids (SCFAs) and their metabolic effects on host physiology. SCFAs primarily acetate, propionate, and butyrate—are produced by gut microbial fermentation of dietary fibers. These metabolites play key roles in regulating glucose and lipid metabolism, improving insulin sensitivity, modulating immune responses, and maintaining gut barrier integrity through signaling via G-protein-coupled receptors (GPR41/43) and systemic endocrine pathways. Disruption in SCFA production is associated with metabolic disorders such as obesity, type 2 diabetes, and NAFLD. *This figure was created by the authors using BioRender.com.*

5. Cardiovascular diseases

Obesity, and type 2 diabetes, are among the key traditional and classical risk factors for cardiovascular disease (CVD) **(Roman & Stoian, 2021)**. Microbial metabolism of dietary choline and carnitine, which are abundant in the typical western diet, are also being contributed to **(Estruch et al., 2018)**. These compounds are converted in the liver to trimethylamine N-oxide (TMAO), which is associated with atherosclerosis and interferes with bile acid metabolism and cholesterol transport, further elevating CVD risk **(Thomas & Fernandez, 2021)**.

Atherosclerotic patients have less favorable butyrate-producing bacteria such as *Roseburia* and *Eubacterium*. The gut microbiota in CVD patients exhibit a proinflammatory profile, which can be measured by reductions in phytoene dehydrogenase, as well as increased genes responsible for peptidoglycan synthesis **(Pan et al., 2024)**.

6. Immune-related diseases

Asthma, atopic eczema, and allergic rhino conjunctivitis are among the most common allergic diseases **(Lee & Bak, 2011)**. Gut microbiota dysbiosis, along with genetic and environmental factors, contributes to the development of these conditions **(Parkin et al., 2021)**. During early infancy, a notable reduction in Bacteroidetes and other butyrate-producing bacteria—important for immune system development—has been observed. At the onset of allergic symptoms in children, *Bifidobacterium adolescentis* levels were elevated, while overall bacterial diversity, especially *Faecalibacterium prausnitzii*, *Clostridium*, and *Staphylococcus aureus*, was reduced **(Di Costanzo et al., 2021)**.

Cohort studies from various regions identified key taxa such as *Lachnospira*, *Akkermansia*, and *Faecalibacterium* in infants predisposed to asthma **(Durack et al., 2018)**. A protective molecule known as A20 (TNFAIP3), which regulates immune homeostasis and is expressed by lung epithelial cells, was found to be reduced in asthma patients, making them more susceptible due to failed LPS-induced responses **(Stokholm et al., 2018)**. These findings support the concept of the gut–lung axis and

demonstrate that gut microbiota can significantly influence asthma risk in children.

7. Neurologic and psychiatric diseases

For both humans and animals, the gut-microbiota-brain axis is essential. Numerous neurological and mental illnesses have been linked to disruptions in the microbiome-brain-gut axis. These illnesses include neurodevelopmental conditions like autism, stress-related conditions like anxiety and depression, and neurodegenerative conditions like Alzheimer's disease (AD) **(Bicknell et al., 2023)**.

7.1 Anxiety

Anxiety is an emotional condition influenced by immunologic, endocrine, and neurological mechanisms, and can be intensified by environmental or biological stressors. The gut-brain pathway with its neurotransmitters and immune factors is central to the regulation of anxiety **(Jiang et al., 2024)**. While intestinal dysbiosis due to pathogenetic bacteria, can induce or aggravate anxiety through both immune and metabolic pathways, Campylobacter jejuni influences anxiety behaviors through neuronal activation from c-Fos protein and without increasing proinflammatory cytokines **(Bharwani, 2019)**.

Probiotic bacteria, particularly *Lactobacillus* and *Bifidobacterium*, are associated with an anti-anxiolytic impact in part through influencing the gut–brain axis. *Lactobacillus rhamnosus* influences the central GABA receptor expression and the anxiety- and depression-related behavior through vagus nerve signalling. *Bifidobacterium adolescentis* is also a GABA producer; supporting the notion of the gut–brain axis influencing a role for the *Lactobacillus* and *Bifidobacterium* for the alleviation of anxiety behaviours **(Ansari et al., 2023)**.

7.2 Depression

Depression is a mood disorder linked to immune system dysregulation and tryptophan metabolism deficiencies. Numerous observational studies demonstrate that the gut microbiota and depression interact in both directions **(Schwarcz et al., 2012)**. An alteration in the gut-brain axis that results in inflammation is linked to depression. Certain Gram-negative bacteria, like *Enterobacteriaceae*, are translocated across the barrier and the inflammatory process is triggered when intestinal permeability shifts and barrier integrity is compromised **(Doney et al., 2022)**. Another study by **Zhou et al., (2023)** verified that the development of depression may be caused by the gut microbiota. Transplantation of fecal microbiota from depressed subjects into microbiota-depleted rats affected tryptophan metabolism, resulting in observations of anhedonia and anxiety-like phenotypes in the animals.

7.3 Autistic spectrum disorder

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that causes impairments in behavior and social communication, along with associated and/or comorbid intestinal dysfunction and compromised integrity of the intestinal barrier **(Rosenfeld, 2015)**. Previous studies involving microbiota transfer therapy produced hopeful outcomes; for example, 18 children with ASD were reported as improving neurological and gastrointestinal symptoms **(Kang et al., 2017)**. Initial hypotheses linking ASD to the gut microbiota proposed that Clostridium species impacted the disorder through neurotoxic effects **(Argou-Cardozo & Zeidán-Chuliá, 2018).** Those with ASD exhibit a significantly lower abundance of Bacteroidetes and higher Firmicutes-to-Bacteroidetes ratio, suggesting a specific microbial imbalance that may be involved in influencing gut and brain function.

7.4 Alzheimer’s disease

Alzheimer’s disease (AD) is the leading cause of dementia globally and is a progressive, degenerative disorder that affects the central nervous system (CNS) **(Hibberd et al., 2017)**. Multiple studies have confirmed the crucial role of gut microbiota in the development of AD. The condition involves the gut-brain axis and its bidirectional communication **(Figure 3)**. Dysbiosis is believed to increase the permeability of both the gut and the blood-brain barrier **(Jiang et al., 2017)**.

Gut microbiota in AD contributes to the production of lipopolysaccharides (LPS), amyloids, and inflammatory cytokines, all of which are associated with the disease. Akkermansia and Allobaculum are lower, and Bacteroidetes and Firmicutes are higher in the mouse models of AD when compared to healthy controls **(Verhaar et al., 2022)**.

Gut Microbiota in Egypt

The study of gut microbiota among Egyptians studies is limited, but, it has been suggested due to diverse diets and environmental exposures that the gut microbiota in Egyptians is different from other populations. Traditional Egyptian diets which are rich in plant-based foods and low in saturated fats have a different profile than Western diets that are largely processed foods with higher fat content which have the potential to be health-promoting, increasing the growth of SCFA produced by bacteria which are

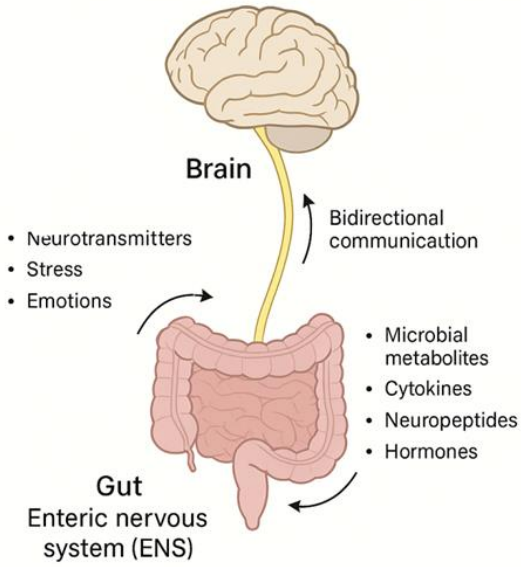

Figure 3: Schematic representation of the gut-brain axis and its bidirectional communication. Microbial metabolites, cytokines, neuropeptides, and hormones produced in the gut influence central nervous system function via neural pathways including the vagus nerve, while brain-derived signals such as neurotransmitters, stress, and emotions modulate gut physiology and microbiota composition. *This figure was created by the authors using BioRender.com.*

beneficial to gut and metabolic health **(Shankar et al., 2017)**. There are limited studies exploring gut microbiota between rural, semi-urban, and urban Egyptian populations. Environmental factors, including pollution, and exposure to pathogenic species, may also contribute to microbial diversity in Egypt. There are data from the Middle East and Africa that highlight the differences in diets and environments on gut microbiota. Individuals with obesity, and diabetes have been shown to have increased levels of Bacteroidetes and Firmicutes **(Hassan et al., 2022)**.

Comparisons of diets observed in Mediterranean vs. Western diets support diet's influence on microbial profile, when compared, there are few studies that provide context based on location or specific to Egypt. Environmental influences such as urbanization, pollution, and dietary shifts—well documented in other African countries—are underexplored in Egypt. Given the nation's unique environmental challenges, including water quality and agricultural practices, more studies are needed. Recently, a pilot study began addressing this gap by examining gut microbiota variations among healthy Egyptians across different regions **(Hassan et al., 2025)**.

8. Conclusion

The gut microbiota has a significant impact on the physiology and disease of the host, with mounting evidence for its involvement in the onset and progression of disease states that range from gastrointestinal, hepatic, metabolic, cardiovascular, immune-related, and neuropsychiatric disorders. Dysbiosis can cause disease mechanisms through altered microbial composition, altered barrier function and integrity, immune dysregulation, and altered metabolism. While global studies have broadened our knowledge of the associations of gut microbiota on health and disease, there is a lack of information on specific population, like Egyptians. Local dietary regimen, environmental exposure and lifestyle, may have a considerable impact on the composition and function of gut microbial diversity. Clearly, research to track local microbiota profiles is urgently needed in order to properly inform regionally appropriate personalized treatments and also regionally appropriate public health interventions. Understanding the role of the microbiome in health and disease could lead to new diagnostic, preventative, and treatment methods based on individual or on population-based microbial signatures.

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Competing interests

The authors declare no competing interests.

Author Contributions

Kareem Talaat Mohamed conceived and wrote the mini-review. Amro Hanora contributed to the conceptual framing and supervised the development of the manuscript. Nora Fahmy, Sarah Shabayek, and Mahmoud Mohamed Tawfick critically reviewed and provided intellectual input during manuscript preparation. All authors read and approved the final version of the manuscript.

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