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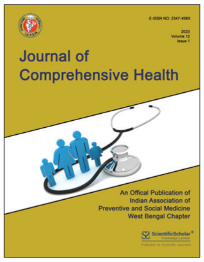

Review Article

Role of Gastrointestinal Tract (GUT) Health in Reducing Non-communicable Disease Risk

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ABSTRACT

The term “gut microbiota” refers to the group of bacteria in the human digestive system that exhibits functions essential to human life. The primary objective of the present review is to identify specific microbial patterns and mechanisms through which gut health influences these conditions. The review will also analyze clinical trials and observational studies to determine the best practices for utilizing gut health strategies as preventive measures for non-communicable diseases. According to recent research, maintaining a healthy lifestyle, eating well, and engaging in physical activity (like sports) are crucial for preserving a functional physiological microbiota that supports overall human health. Exercise has been demonstrated in numerous studies to alter the composition of gut microbiota (GM), enhance intestinal mucosal immunity, raise the *Bacteroidetes–Firmicutes* ratio, modify the bile acid profile, and enhance the production of short-chain fatty acids. Moreover, evidence suggests that low-intensity exercise may lower the incidence of gastrointestinal disorders, supporting the idea that skeletal muscle and GM are strictly correlated.

Keywords: Cardiovascular diseases, Exercise, Gut microbiota, Health, Healthy diet, Hypertension, Obesity: Metabolic syndrome, Osteoarthritis, Physical activity

INTRODUCTION

Non-communicable diseases (NCDs), also known as chronic diseases, encompass a range of health issues that do not stem from infectious agents or spread between individuals. These conditions typically manifest gradually, influenced by a blend of genetic, behavioral, environmental, and metabolic factors.[1-3] According to the World Health Organization’s 2022 data, NCDs are responsible for 74% of deaths globally, causing 41 million deaths each year.[4,5] In India, the burden of NCDs has significantly increased, with these diseases accounting for 66.7% of all deaths in 2019, compared to 37.9% in 1990, as reported in the Global Burden of Disease Study 2019.[1-6] Diabetes, cancer, chronic respiratory diseases, and cardiovascular diseases (CVDs) are the four primary NCDs. Four behavioral risk factors – bad diet, inactivity, alcohol and tobacco use, and use of dangerous substances – are shared by these four diseases. Human gut bacteria comprise mainly *Firmicutes* (60–80% of all gut bacteria) and *Bacteroidetes* (20–40%), in addition to a small amount of Proteobacteria and Actinobacteria, but their comparative abundance varies with anatomical setting among individuals.[1-7]

The population of gut microflora is largely stable, even though antibiotic use, food, and other environmental factors can cause rapid changes in its conformation. The host’s health is primarily influenced by the biological balance that exists between the host and the gut microbiota (GM).

The host requires the GM to provide various functions of the gut: Nutrient metabolism, mutagen, and carcinogen neutralization; expansion and role of the immune system; shield from pathogens; enterocyte and intestinal epithelium growth; and short-chain fatty acid (SCFA) production.[6] SCFAs initiate enterocyte propagation and mucin secretion, which greatly impact the tightness of the intestinal barrier.[8-10] The structure of the microbiota, particularly the presence of the above-mentioned bacteria, impacts the absorptivity of the toxic metabolites from the gut barrier.[11-13]

According to published studies, modest exercise has a beneficial effect on intestinal porousness, absorption and accommodation of electrolytes and nutrients, and the rate of excretion of toxic metabolic products.[12,14] On the other hand, increasing the amount of time spent exercising or the level of physical exertion may have an adverse effect on the digestive system and result in symptoms such as diarrhea, vomiting, colic, flatulence, and abdominal pain. Under this context, "exercise-induced gastrointestinal (GI) syndrome" refers to a variety of normal physiological responses to exercise that disrupt and impair the integrity and function of the GI tract.

This syndrome is believed to affect 70% of athletes and arises 1.5–3 times more frequently among qualified athletes than among laypersons.[14] It follows two different pathways: Cardio–gastrointestinal and neuroendocrine–GI signal trails. The previous grounds a reorganization of the blood flows to the working muscle and peripheral circulation, while the latter is associated with increased sympathetic initiation and the subsequent reduction in the functional volume of the GI tract.[15,16] It was suggested by Camilleri that physical activity could damage the digestive tract's lumen and upset the immune system, leading to GI symptoms and an augmented inflammatory response. Camilleri further proposed that changes in the composition of the GM, as evidenced by an increase in alpha diversity and the abundance of several dominant bacteria, including *Bacteroides*, raise intestinal permeability. According to published research, changes in the intestinal microflora's composition are linked to both acute and chronic illnesses, not just those affecting the digestive system. "Dysbiosis" is the forfeiture of commensal bacteria with possible valuable metabolic activity and the overgrowth of resourceful pathogens, as well as reduced biodiversity.[17-19]

The GM is concerned with the host's digestive, metabolic, and immune processes. The physiological and pathophysiological functions of the intestinal microbiota depend on its composition, and numerous individual or external aspects may be associated with the presence of some categories, phyla, or species of microorganisms in the gut.[15] More recently, it has been suggested that a correlation between intestinal microbiota and exercise, counting strong competitive sports activities, may help to explain the advantages of physical activity on overall body health.[14,15] Except for the brain and circulatory system, this microbial population is present throughout the body, primarily in the skin, intestinal tract, and oral cavity. Exercise guards against several chronic illnesses and many of these protective benefits may be mediated by the GM. Physical exercise alters the composition of intestinal microbes by promoting homeostasis and energy regulation. There is no study work described or discussed regarding the role of gut health in reducing NCD risk. Therefore, the primary objective of the present review is to identify specific microbial patterns and mechanisms through which gut health influences these conditions. The review will also analyze clinical trials and observational studies to determine the best practices for utilizing gut health strategies as preventive measures for NCDs.

METHODOLOGY

This systematic review was conducted according to preferred reporting items for systematic reviews and meta-analyses (PRISMAs) guidelines. A comprehensive literature search was performed across multiple electronic databases including PubMed/MEDLINE, Scopus, Web of Science, and EMBASE from inception through December 2023. The search strategy employed combinations of key terms including "gut microbiota," "gut microbiome," "intestinal flora," "coronary heart disease," "cardiovascular disease," "obesity," "dyslipidemia," and "noncommunicable diseases." Studies were included if they were original research articles published in peer-reviewed journals, focused on human subjects, were available in English, and investigated the relationship between GM and specified NCDs. Exclusion criteria encompassed review articles, animal studies, conference abstracts, and non-English publications. Two independent reviewers screened titles and abstracts, followed by a full-text assessment of potentially eligible studies. The extracted data were synthesized narratively, with quantitative meta-analysis performed where appropriate. Figure 1 illustrates the PRISMA flow diagram of the study selection process.

DISCUSSION

Obesity

Obesity and overweight conditions represent significant public health concerns, characterized by excessive fat accumulation that presents substantial health risks. The World Health Organization defines these conditions using body mass index (BMI), with overweight classified at BMI ≥25 and obesity at BMI ≥30. A striking global trend shows that childhood and adolescent obesity rates have seen an unprecedented rise, with worldwide prevalence among ages 5–19 increasing more than fourfold between 1975 and 2016, from 4% to 18%.[17] The global distribution of obesity presents a complex picture. While historically considered a high-income

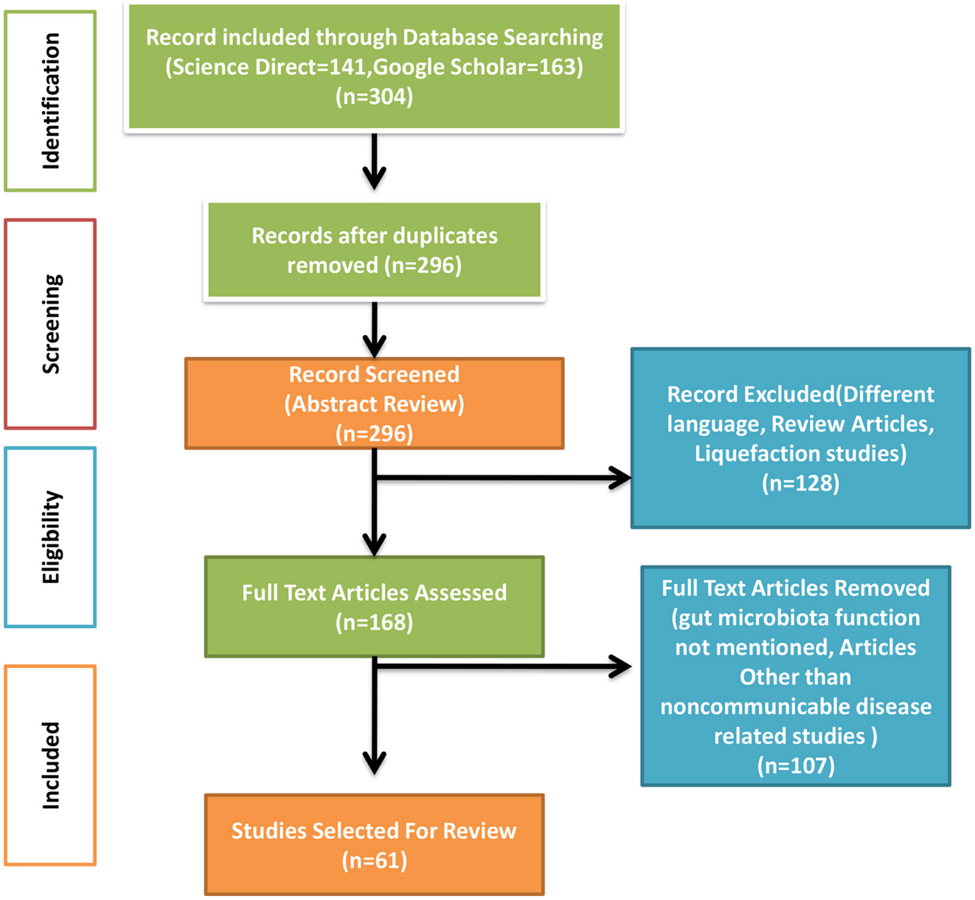

Figure 1: Details work flow regarding selection of article and literature review.

country phenomenon, obesity rates are now escalating rapidly in low- and middle-income nations, particularly in urban regions. This shift has been especially pronounced in developing countries, where the rate of increase has surpassed that of developed nations by more than 30%.[17] This trend contributes to what health experts term the "double burden of malnutrition," where obesity rates exceed underweight prevalence in most regions globally, except sub-Saharan Africa. The health implications of excess weight are extensive and severe. Obesity significantly elevates the risk of various chronic conditions, notably CVDs – the leading global cause of mortality. The relationship between obesity and diabetes is particularly concerning, given that diabetes prevalence has increased fourfold since 1980. Obesity's impact extends to musculoskeletal health, particularly osteoarthritis (OA), and shows strong associations with various cancers, including endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colorectal malignancies. Research indicates that even modest weight excess increases disease risk, with risk levels correlating directly with BMI increases.[15-20]

Dietary patterns significantly influence GM composition, representing a crucial environmental factor in physiological GM configuration. The intricate relationship between nutrition and microbial well-being affects both human health and the trillion-strong microbial communities residing in the gut.[21] Research demonstrates that high-fat dietary patterns correlate with reduced *Bacteroidetes* populations and increased opportunistic pathogens, potentially compromising intestinal barrier integrity and promoting systemic inflammation and insulin resistance.[20,21]

The role of nutrition in promoting healthy aging deserves special attention. Research supports the combination of appropriate dietary interventions, including vitamin D and amino acid supplementation, alongside physical activity in preventing age-related conditions such as sarcopenia.[20] The Mediterranean diet, renowned for its anti-inflammatory properties, has shown promising results in reducing OA risk. Evidence suggests that fiber-rich nutrition patterns, combined with adequate micronutrient intake and regular physical activity, may be fundamental for maintaining optimal health, particularly among older adults.[20,22] Adipose tissue accumulation involves complex interactions between environmental and genetic factors. This process, when imbalanced, can lead to metabolic syndrome, characterized by multiple conditions including hypertension, dyslipidemia, hyperglycemia, and hypertriglyceridemia. Recent studies have established correlations between BMI and GM composition. The inflammatory nature of obesity makes it a significant risk factor for OA and can compromise muscle mass and functionality in older populations. Animal studies demonstrate that high-fat/high-sucrose diets adversely affect muscle mass and function while promoting knee joint deterioration. These effects are associated with increased intestinal permeability and elevated serum lipopolysaccharide (LPS) levels, suggesting interconnections between gut dysbiosis, metabolic inflammation, and OA development [Table 1].[23,24]

Metabolic syndrome

Metabolic syndrome is a cluster of conditions that heighten the risk of heart disease, stroke, and type 2 diabetes. Key contributing factors include poor diet, physical inactivity, and genetic predisposition. Effective management often involves lifestyle interventions such as adopting a healthier diet, increasing physical activity, and achieving weight loss. In some cases, medications may be necessary to control cholesterol levels, blood pressure, or blood sugar.[24,25] Globally, metabolic syndrome affects an estimated 20–25% of adults. Individuals with this condition face a threefold increased risk of heart attack or stroke and are twice as likely to die from these events. Over the past two decades, its prevalence has risen markedly, particularly in countries experiencing higher caloric consumption and declining physical activity levels.[25] These statistics emphasize the urgent need for preventive measures and targeted interventions to reduce the growing burden of metabolic syndrome worldwide.

Microbiome and blood pressure

High blood pressure is a hallmark of metabolic syndrome and a significant risk factor for CVDs in both individuals with metabolic syndrome and the general population. Emerging evidence highlights the role of the gut microbiome in regulating blood pressure. For instance, inorganic nitrate, commonly found in green vegetables, has been shown to increase plasma nitrite levels and reduce blood pressure. Similarly, a meta-analysis of 14 randomized, placebo-controlled clinical trials demonstrated that probiotic-fermented milk significantly lowers blood pressure in pre-hypertensive and hypertensive patients.[26-29] The mechanism underlying this effect involves probiotic microorganisms, such as *Lactobacillus* and *Bifidobacterium*, which ferment milk proteins and produce peptides with angiotensin-converting enzyme (ACE) inhibitory activity. ACE, a component of the renin-angiotensin system, is crucial in blood pressure regulation. During fermentation, proteinases from probiotics release ACE-inhibitory peptides, which exert blood pressure-lowering effects. Such peptides have also been identified in fermented dairy products such as milk, yogurt, and cheese, as well as in soy products where fermentation enhances the production of ACE-inhibitory peptides.[27] In addition, estrogen plays a known role in regulating blood pressure. Probiotics can enhance the bioavailability of food-derived phytoestrogens and natural estrogen alternatives. Due to their structural similarity to mammalian estrogens, soy isoflavonoids, such as genistein and daidzein, may mimic estradiol's effects on vascular function. These isoflavonoids are hydrolyzed into bioactive aglycones by GM or probiotics, leading to faster and more efficient absorption. The combined action of probiotics and phytoestrogens offers the potential for managing hypertension more effectively.[28-41] These findings underscore the potential of dietary and probiotic interventions as natural strategies to manage hypertension, particularly in metabolic syndrome and related conditions [Table 1].

GM and astherosclerosis

Of the estimated 54 million annual deaths worldwide, CVDs remain the leading cause. Acute cardiovascular events, such as heart attacks, are primarily triggered by thrombus formation resulting from the rupture of atherosclerotic plaques. Recent research has identified a connection between atherosclerosis and the gut microbiome.[29] Dysbiosis, or an imbalance in microbial communities, often arises from shifts in microbial composition or disruption of mucosal barrier function. Common causes include high-fat diets, various illnesses, and excessive antibiotic use. Gut dysbiosis can affect gut permeability, allowing bacterial DNA, metabolites, and endotoxins to translocate into the bloodstream. In a healthy state, the intestinal barrier is a complex system composed of physical, biochemical, and immunological components. The primary physical barrier is formed by intestinal epithelial cells (IECs), which are linked by tight junction proteins. This barrier is reinforced by two layers of mucus and a healthy community of gut commensal microorganisms.[31,32] An increased *Firmicutes*-to-*Bacteroidetes* ratio, characterized by elevated acetate and reduced butyrate levels, has been associated with atherosclerosis and hypertension. Butyrate, a key SCFA, is essential for maintaining gut barrier stability as it serves as the primary energy source for IECs.[28] High-fat diets, a significant risk factor for atherosclerosis, cause notable alterations in GM composition. Dysbiosis resulting from various factors can lead to a "leaky gut," facilitating the translocation of bacteria and harmful microbial products. This, in turn, triggers abnormal immune responses and contributes to the development of atherosclerosis.[29] Addressing gut dysbiosis through dietary, microbial, or lifestyle interventions may offer a promising avenue for preventing or mitigating the progression of CVDs.

GM alteration as cure of OA

GM modulation can be achieved through specific diets, probiotics (live microorganisms), and prebiotics (indigestible

Table 1: Relevant research and limitations.

Year of study	Author(s) of the study	Observation from the literature review	Gap identified in the literature review	Scope of research
2021[18]	Palmas *et al.*	Showed a negative correlation between *Bacteroidetes* taxa and body fatness and waist circumference, but a positive correlation between *Firmicutes* taxa and body fat and a negative correlation with muscle mass and/or physical activity level.	The research study is confined to a particular demographic in a particular location.	Further investigation is necessary to gain a deeper comprehension of the association.
2020[21]	Lane *et al.*	Studies show that including. Very low-energy diets adjunctive to comprehensive lifestyle interventions such as nutritional counseling, behavioral therapy, and exercise programs on gut microbiota and metabolic outcomes in individuals with obesity. However, outcomes were inconsistent, with some trials also showing decreases in *Bacteroides* taxa and increases in commensal microbiota, such as Lachnospiraceae and Bifidobacteriaceae.	Only a small portion of the population was surveyed.	Further rigorously designed trials, with more standardized microbiological sequencing techniques and detailed reporting, are required.
2015[22]	Hermes *et al.*	Obesity-associated metabolic disorders microbiota can be characterized by a potential pro-inflammatory composition, with less potential for the production of short chain fatty acids and butyrate in particular	Nevertheless, which bacterial species contribute to the onset of human obesity is still unidentified.	For a deeper comprehension of disease, further research in this area is required.
2008[23]	Berthold-Losleben and Himmerich	It has been demonstrated that modifications to the TNF-α system contribute to the emergence of psychiatric diseases and are also linked to variations in body weight, endocrine, and metabolic abnormalities in psychiatric patients.	There are just early research studies on these topics with small sample sizes.	An extensive investigation would be advantageous.
2019[55]	Miyamoto *et al.*	Recent findings observed the relationship between the metabolites of dietary omega-6-FAs and the host energy metabolism, providing insight into how to prevent and treat metabolic illnesses by focusing on the metabolites of gut microbiota.	PUFA supplementation increased the *Lactobacillus* and the PUFA-metabolite HYA in the gut microbiota.	Some more data-driven studies are required.
2009[56]	NIH HMP Working Group. Peterson *et al.*	The ultimate goal of the Human Microbiome Project is to show that human health can be enhanced by monitoring or modifying the human microbiome.	Although not entirely accomplished, attempts were made to assemble a sample that was quite diverse in terms of color, ethnicity, and other demographic characteristics.	Research gaps can be leveraged for future investigations.

TNF-α: Tumor necrosis factor-alpha, PUFA: Polyunsaturated fatty acid, TNF: Tumor necrosis factor, FA: Fatty acids, HYA: 10-hydroxy-cis-12-octadecenoic acid

components). Restoring GM balance has shown promise in managing dysbiosis-associated diseases. Probiotics such as "*Bifidobacterium*" and "*Lactobacillus*" contribute to gut health by regulating pH levels and competing for colonization, thereby supporting microbial balance.[31] Prebiotics, on the other hand, stimulate the activity and proliferation of beneficial bacteria in the GI tract. This promotes fermentation processes that generate SCFAs, key metabolites in maintaining gut health.[34] Studies involving germ-free and control mice have demonstrated GM's influence on hepatic triacylglycerol and glucose production. Gut microbial colonization has been found to suppress fasting-induced adipose factor (FIAF), a lipoprotein lipase (LPL) inhibitor, leading to increased LPL activity. This heightened LPL activity facilitates greater fatty acid uptake and triacylglycerol storage in adipocytes, illustrating the microbiota's role in host energy regulation.[26,27]

A key link between GM and OA appears to be chronic low-grade inflammation, forming the basis of a proposed "metabolic OA" phenotype. Shifts in the microbial community, influenced by factors such as antibiotic use, germ-free environments, or high-fat diets, have been implicated in OA progression. These findings suggest that targeted modulation of the microbiome could offer therapeutic potential in preventing or mitigating joint degeneration associated with OA.[29,30] In addition, dietary supplements, and nutraceuticals may enhance health outcomes in the elderly by inducing qualitative and quantitative changes in the GM, further highlighting the microbiome's central role in overall health and disease management.

Physical activity and microbiota: Some underlying mechanisms

Physical activity accelerates changes in GM through mechanisms such as the release of myokines, enhanced intestinal transit, and the secretion of hormones and neurotransmitters. However, defining a "healthy" GM remains challenging due to significant inter-individual variability and the plasticity of the gut microbiome. Factors such as diet, sleep patterns, antibiotic exposure, and comorbidities further complicate understanding the relationship between physical activity and microbiota composition.[33,34] A healthy GM plays a crucial role in immune system development, exhibiting anti-inflammatory effects partly by reducing histone deacetylase activity and regulating T cells through G protein-coupled receptors.[33,34] Physical activity-associated alterations in microbiota composition, particularly in sedentary individuals, have been linked to increases in SCFAs like n-butyrate. These SCFAs can influence host energy balance and enhance nutrient availability. Conversely, an inactive lifestyle is associated with reduced microbial diversity and a less intricate microbial network structure.[42] GM diversity has emerged as a potential indicator of overall health. Active individuals generally display a more diverse and stable microbial community, characterized by enhanced resilience, resistance to pathogenic invasions, and functional redundancy, which ensures efficient resource utilization. This contrasts with sedentary individuals, whose GM tends to exhibit diminished diversity and functionality.[42-45] Thus, physical activity not only supports GM diversity but also reinforces its role in promoting host health and resilience.

Microbiota gut-brain alliance

The human GM comprises a vast and intricate ecosystem, hosting over 40,000 species of microorganisms and at least 1,800 microbial genera, functioning as a critical metabolic organ.[46-48] Among the numerous bacterial phyla present in the gut, the most prominent are *Firmicutes* – which include genera such as *Lactobacillus*, *Clostridium*, and *Enterococcus* – and *Bacteroidetes*, with *Bacteroides* as a primary genus.[48] The GM plays a significant role in the gut-brain axis, a complex bidirectional communication network that involves the central nervous system, autonomic nervous system, enteric nervous system, GI cells, and gut microorganisms. This axis influences mental health and is implicated in conditions such as depression and anxiety, often contributing to mood regulation.[37,47] For example, studies on germ-free mice have shown that transferring microbiota from individuals with major depressive disorder or healthy individuals leads to recipient mice exhibiting behaviors reflective of the donor's mental state.[44] In depressed individuals, higher abundances of Proteobacteria and Enterobacteriaceae have been observed, which can produce LPSs that may exacerbate inflammatory processes. The signaling between the brain and GM is mediated by neural, endocrine, and inflammatory pathways. GM-derived metabolites, such as SCFAs, secondary bile acids, tryptophan metabolites, folate, and gamma-aminobutyric acid, influence neurotransmitter production and breakdown. These microorganisms also impact the gut-brain axis by regulating the expression of peptides involved in energy metabolism, inflammation, and other vital biological functions.[43-45] The interplay between GM and the brain underscores its potential role in modulating both physical and mental health.

Exercise as an antidepressant through the microbiota–gut-brain axis

Regular exercise has long been recognized for its ability to reduce stress levels and is also considered an effective antidepressant strategy, though the exact mechanisms remain uncertain.[44,45] Research consistently suggests that exercise mitigates depression through neuromolecular pathways, including increased serotonin availability, regulation of hypothalamic-pituitary-adrenal axis activity, and the expression of neurotrophic factors.

It is now understood that the gut and brain communicate through these pathways, with the GM playing a pivotal role in this interaction. Consequently, it has been proposed that physical exercise alleviates stress and depression symptoms by modulating GM composition and function. This hypothesis is supported by studies conducted on rats, which confirm that exercise-induced changes in the microbiota can positively influence brain health.[47-52]

In humans, evidence highlights a strong association between fitness levels and microbiome diversity. Notably, improvements in physical fitness and mental health have been linked to alterations in specific gut microbial taxa.[52] Exercise has been shown to enhance the diversity and richness of GM (α-diversity) and to increase the abundance of beneficial bacterial species, further emphasizing its role in promoting mental and physical well-being [Table 2].[51-63]

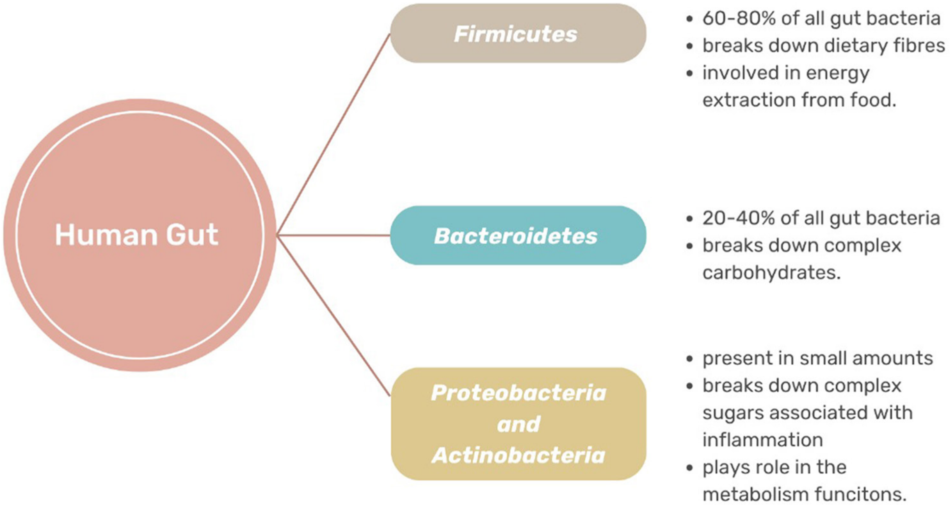

Figure 2: Classification of the assay of bacteria present in the human gut.

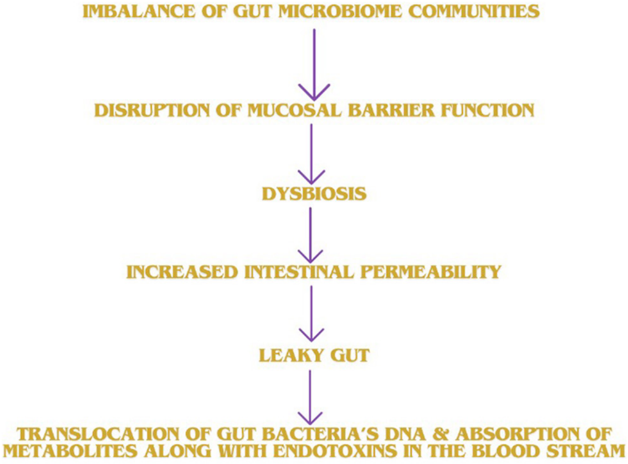

Figure 3: Impact of the imbalance of gut-microbiome communities.

Potential intervention in microbiota constitution

Antibiotic treatment has been shown to alter the composition of the GM in mice, leading to significant metabolic changes. In one study, antibiotic-treated obese mice experienced decreased body weight, improved glucose tolerance, and increased fasting glycemia. These findings suggest a potential role for GM in the development of metabolic disorders.[64] The mechanism underlying these effects appears to involve changes in inflammatory markers. Antibiotic treatment reduced levels of LPS, a bacterial endotoxin, and increased levels of adiponectin, an adipokine with anti-inflammatory properties. These changes may contribute to the improved glucose tolerance observed in the treated mice.[64] To further explore the potential of manipulating GM for therapeutic purposes, researchers have employed various strategies, including fecal microbiota transplantation and dietary interventions. Fecal microbiota transplantation involves transferring fecal matter from a healthy donor to a recipient, while dietary interventions, such as butyrate supplementation or oligofructose intake, can selectively modulate the growth of specific microbial populations [Figures 2 and 3]. By targeting the GM, we may be able to develop novel therapies for metabolic disorders, such as obesity and type 2 diabetes.[64]

GM and metabolism

Several studies have established specific connections between human metabolism and intestinal microbiota. Kau *et al.*[17] were pioneers in investigating GM as an environmental factor influencing obesity and fat storage. Their findings revealed that young, conventionally raised mice had 42% more total body fat and 47% more gonadal fat compared to germ-free mice. Further research on germ-free and control mice highlighted that GM significantly impacts glucose and triacylglycerol production in the liver. For instance, gut microbial colonization appears to suppress FIAF expression, which acts as an LPL inhibitor, thereby increasing LPL activity. This elevated LPL activity facilitates adipocyte fatty acid uptake and triacylglycerol accumulation.[57] Recent studies emphasize the intricate relationships between diet, lifestyle, and disease risk, suggesting the critical role of gut health in mitigating NCDs. While the cited studies may not directly address gut health, they provide relevant insights that contribute to a broader understanding of NCD prevention [Figure 4]. For example, Choudhury *et al.* (2024)[33] examined the cardiovascular risks associated with ready-to-eat food consumption among young adults in Kolkata, India. This work highlights how modern dietary patterns impact health outcomes, with potential implications for gut health, as diet significantly influences gut microbiome balance. Dysbiosis resulting from unhealthy dietary

Table 2: Relevant research on gut microbiota and atherosclerosis.

Year of study	Author(s) of the study	Observation from the literature review	Gap identified in the literature review	Scope of research
2004[57]	Li and Fang	Observed whether the stability of atherosclerotic plaque – whose rupture can result in a number of dangerous events such as shock, myocardial infarction, and acute heart failure – is influenced by the gut microbiota.	Regarding whether or not the bacteria will make the plaque rupture more readily, opinions are still divided.	Research gaps can be leveraged for future investigations.
2005[58]	Lehtiniemi *et al.*	Using the Br-PCR method to identify a broad range of oral bacteria from coronary tissues. According to the research, atheromas could function as mechanical sieves that gather bacteria from the bloodstream.	Regardless of the reason for death, bacterial rDNA sequences of oral infections were found in the coronary samples in every instance.	Additional research is required to achieve sufficient outcomes.
2019[59]	Brandsma *et al.*	Found that in HFC -fed Ldlr−/− mice, the Casp1−/− microbiota exacerbated atherosclerosis in the aortic root. Increased blood leukocyte counts, especially neutrophils and monocytes, increased neutrophil accumulation in atherosclerotic plaques, and decreased levels of SCFAs in the cecum were all associated with this. Proinflammatory plasma cytokines were also elevated. These findings suggest a causal connection between inflammation, atherosclerosis, and the makeup of the microbiota.	Cannot rule out the possibility that some effects on TMAO may have faded over the course of the investigation, even though they were not there at the time of sacrifice. Consistent with this, it is plausible that the inflammatory consequences have gradually diminished.	To rule out these possibilities, future research should incorporate earlier and more frequent time points.

SCFA: Short-chain fatty acid, Br-PCR: Broad range polymerase chain reaction, rDNA: Recombinant DNA, HFC: Hydroflurocarbons, TMAO: Trimethylamine N-oxide

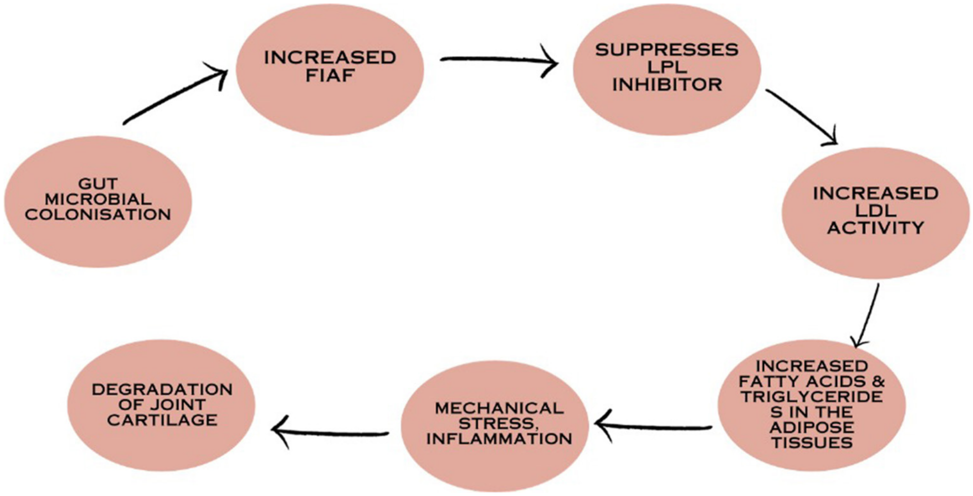

Figure 4: Gut microbiota and metabolism.

choices can elevate NCD risk. In addition, Ghosh (2024) reviewed the application of machine learning and artificial intelligence in addressing complications related to metabolic syndrome, proposing that these technologies could analyze gut microbiome data to predict NCD risk. Similarly, Shakil (2024) explored the link between nutritional status, mental health, and cognitive function among institutionalized and community-dwelling elderly women, underscoring nutrition's critical role in overall health and gut health. Ghosh (2024) also investigated artificial intelligence in detecting and predicting malnutrition risks among the elderly, with potential applications for assessing gut health and its impact on NCD prevention. Mukhopadhyay *et al*. (2024)[65] examined cytokine interactions in herpes simplex virus-induced skin infections, demonstrating the immune system's relevance to gut health. The gut microbiome's role in modulating immune responses is vital, influencing vulnerability to both infectious and NCDs. These diverse research findings collectively suggest that a comprehensive approach to reducing NCD risks should prioritize gut health. Future studies should directly explore the relationships between gut microbiome composition, dietary habits, and NCD risk across various populations. Integrating knowledge from nutrition, aging, immunology, and advanced technologies could lead to more effective strategies for promoting gut health and alleviating the global burden of NCDs.[64-69]

CONCLUSION

A growing body of evidence suggests that regular physical activity and sports can significantly influence the composition and function of the GM, thereby promoting host health and immunity. In this context, combining physical exercise with specific dietary interventions may be a promising strategy to restore microbial balance and potentially alleviate dysbiosis-related disorders. Numerous studies have explored the intricate relationship between GM and various physiological processes, including joint health. Obesity, in particular, is strongly linked to alterations in gut microbial composition. It is hypothesized that physical activity-induced changes in the microbiota may contribute to weight management. Given the complex interplay between diet, exercise, and the microbiota, it is crucial to investigate the impact of specific dietary components, nutrients, and supplements on gut microbial diversity. However, the responsiveness of the GM to physical exercise may vary depending on factors such as age and BMI. While the effects of exercise on the gut microbiome have been studied in various populations, research on older adults remains limited. Although existing studies suggest that exercise can positively influence the gut microbial composition of older individuals, identifying the specific microbial taxa that are most responsive to physical activity in this population requires further investigation.

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