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Review Article

General and microbiological chronic diseases into humans

Mahesh Chandra*, Ansar Ahmad Paray

Centre for Interdisciplinary Biomedical Research Adesh University, Bathinda, Punjab, India

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***Correspondence:**
Mahesh Chandra,
E-mail: mahesh0231997@yahoo.com

ABSTRACT

Chronic illnesses including cancer and cardiovascular disease (CVD) are among the world's major causes of death and have become more prevalent in the last ten years. Microbial agents and the emergence of chronic diseases (CDs) have been linked in the past, and new links are presently being investigated. Researchers are employing new technology to investigate the connection more thoroughly and precisely between infectious pathogens and chronic illness.

Keywords: Chronic disease, Autoimmune disease, Gut microbiome, Periodontitis, Cardiovascular disease, Chronic kidney disease

INTRODUCTION

In every country, non-communicable and CDs are currently the leading cause of adult mortality, and over the next ten years, this number is expected to rise by an additional 17%.[1] About one-third of persons worldwide suffer from several chronic illnesses.[2] Previous Indian research show that at least one chronic illness affects 21% of India's elderly population. Chronic disease affects 29% of older people in urban settings and 17% of senior people in rural areas. All chronic disorders, hypertension, and diabetes account for over 68%. Kerala (54%) has the highest rate of chronic disease prevalence, followed by Andhra Pradesh (43%) West Bengal (36%), and Goa (32%). In the US, four out of ten persons have two or more CDs, and six out of ten adults have one or more.[3] An estimated 35 million of the 58 million fatalities that occurred in 2005 were predicted to be caused by chronic illnesses.[1] Several unhealthy behaviors, including smoking, non exercising, eating poorly, and drinking too much alcohol, are significant causes of the most common chronic illnesses.[4] In developed nations, the most common chronic illnesses are CVD and arthritis. heart attacks and strokes, diabetes, cancer (breast, colon, and epilepsy), obesity, and issues with the mouth. Senior citizens are afflicted with all these ailments. The increase in CDs is a grave concern for public health as well as the impacted societies and economies. The effect and profile of CDs were usually underappreciated until recently.[5]

Microbes and humans interact in a variety of intricate ways. Since we now know that infectious agents can cause chronic illnesses and that the human microbiome plays a significant role in both health and disease, our understanding of the interactions between humans and microorganisms has undergone a fundamental shift. We are discovering that chronic disorders previously believed to be unrelated to infectious processes can also be caused by infectious agents. The world's most prevalent, expensive, and avoidable health issues, CDs have a staggering toll. Seven out of ten deaths annually are caused by CDs, which are also the major cause of disability and mortality. Each year, cancer, and CVD cause over half of all deaths.[6]

Many CDs are not caused by infections; instead, their causes can be hereditary, environmental toxins, the combination of several behavioural and other risk factors, or an unidentified factor. Numerous mechanisms exist for infectious pathogens to induce chronic illness. CDs including Chlamydia infection and CVD can be brought on by inflammation brought on by infections or the immune system's reaction to infections.[7] Early infection

can potentially result in long-term, irreversible impairments or deficiencies (e.g., paralysis following poliovirus infection). An indirect risk factor for chronic illness is infection; for example, a mother's sickness can put her offspring at risk for psychological problems. Finding the source of an infection that causes a chronic illness is crucial because it may enable early laboratory diagnosis, treatment, and significant potential for prevention.[8-12]

GUT MICROBIOME AND AUTOIMMUNE DISEASE

The causes of autoimmune disorders (AIDs) involves both genetic predispositions and environmental influences, with a disturbed gut flora garnering more interest. Numerous autoimmune disorders have been linked to functional and compositional alterations in the gut microbiota, and mounting data indicates that disruptions in this area may play a role in the immunopathogenesis of these conditions.[9]

Rheumatoid arthritis

Joint destruction is a symptom of rheumatoid arthritis (RA), it is a systemic autoimmune inflammatory disease. Recent research has shown that a variety of environmental factors, the most significant of which are nutrition, smoking, and infections, play a role in the development of both intestinal/oral dysbiosis and the onset and course of arthritis.[9-11] Patients with RA also exhibit an increased abundance of *Prevotella species* at the compositional level, including *Prevotella copri.*[12,13] RA patients have lower levels of *Faecal bacterium*, a bacterium that is widely acknowledged to be helpful. Furthermore, it was discovered that RA patients had a higher relative abundance of *Collinsella.*[14] It is interesting to note that severe arthritis is produced when mice sensitive to collagen-induced arthritis (CIA) are vaccinated with *Collinsella. Collinsella aerofaciens* is thought to be a potential arthritogenic bacterium in the human gut based on in vitro investigations that revealed the bacteria increases gut permeability and stimulates the release of IL-17A, a crucial cytokine involved in the pathogenesis of RA.[14] In conclusion, patients with early RA have a gut microbiota dominated by *Prevotella copri* and *Collinsella*, which may have a role in the pathophysiology of the disease.[15] Short-chain fatty acids (SCFAs) have been linked recently to autoimmune arthritis in mice, and it has been shown that SCFAs are crucial for reducing inflammation in RA.[16,17] In RA models, mice lacking SCFA receptors displayed increased inflammation.[18] One of the most prevalent SCFAs, butyrate, has been demonstrated to reduce inflammation in animal models of RA and other inflammatory illnesses. It functions as an endogenous histone deacetylase (HDAC) inhibitor.[19] In mice with CIA, and maybe in patients with RA as well, recent study has demonstrated the importance of intestinal barrier function, particularly for zonulin, peptide that regulates epithelial tight junction permeability.[20] Elevated zonulin levels have been linked to inflammation, dysbiosis, and leaky intestinal barrier. Restoring the intestinal barrier in time leading up to development of clinical arthritis may help postpone disease's onset and lessen its severity. This can be achieved using pharmacological treatments like zonulin antagonists/dietary supplements containing SCFA butyrate.

Table 1: Autoimmune diseases summary table of key findings.

Disease group	Specific disease	Gut microbes	Main findings
Autoimmune diseases	RA	*Prevotella copri*.,	Increased in abundance[16,17]
		Collinsella sp.,	Increased in abundance[16]
		Faecalibacterium sp.	Decreased in abundance, links to SCFA production[21]
	Type-1 diabetes	*Dialister invisus, Gemella sanguinis., Bifidobacterium longum*	Increased in abundance (children), gut permeability[21]
		F. prausnitzii	Decreased in abundance (children), butyrate (SCFA) production[22]
	Atopic eczema	*Clostridium difficile*	Increased in abundance[23]
		Escherichia coli	Increased in abundance, eosinophilic inflammation[23]
		Staphylococcus aureus	Increased in abundance[23]
		Bifidobacteria spp.	Decreased in abundance[23]
		Bacteroidetes spp.	Decreased in abundance[24-26]
		Coprococcus eutactus	Decreased (in children), linked to butyrate production[27]
		F. prausnitizii	Increased in abundance, SCFA production[28]
	Atopic asthma	Various-*Clostridium, Pediococcus, Escherichia, Klebsiella, Morganella, and Proteus spp.*	Increased in abundance, increased bioamine (histamine) production[29]
		Enterococcus faecalis, Streptococcus spp., Bifidobacterium bifidum., Lactobacillus spp.	Increased in abundance, increased bioamine (histamine) levels, increased epoxide hydrolase production of oxylipins[7,8]

GUT MICROBIOME-GUT INFLAMMATION OR BOWEL DISORDERS

Changes in bowel patterns and pain and discomfort in the abdomen are the main symptoms of syndrome of the irritable bowel (IBS). While the cause of the condition is complex, changes in the normal gut microbiota may contribute to the low-grade intestinal inflammation linked to it, according to new research on the pathophysiology of IBS.[30] The pathophysiology of IBS is assumed to be influenced by microbial dysbiosis in the gut. A recent study found a distinct difference in the gut microbiota of IBS patients compared to controls. Firmicutes, notably *Ruminococcin*, *Clostridium*, and *Dorea*, were found in greater abundance in IBS patients, but beneficial bacteria like *Bifidobacterium* and *Faecal bacterium* spp were significantly reduced.[31] Additionally, comprehensive reviews have shown that people with IBS have potentially hazardous microbiota, such as members of the genus Bacteroides (phylum *Bacteroidetes*), family *Lactobacillus*, phylum *Proteobacteria*, and family *Enterobacteriaceae*. Numerous dangerous bacteria, including *Salmonella*, *Escherichia*, *Shigella*, and *Campylobacter*, are members of the *Enterobacteriaceae* family.[32] These may indicate a shift in the intestinal environment or a history of intestinal illness in these people. Abdominal pain, bloating, and diarrhoea-three of the characteristic symptoms of IBS-have been linked to buy products from these potentially dangerous bacteria. The uncultured *Clostridial* group was the most consistently detected potentially "protective" bacterial group in IBS patients. Even though the relationship is not causative and it is unknown how a protective impact on IBS symptoms works. Gut mucosal health has been linked to the genus *Faecal bacterium*, particularly *Faecal bacterium prausnitzii*, which shares an order with the uncultured *Clostridia*. This bacterium was thought to be the primary butyrate-producing and anti-inflammatory organism.[33] It also preserved the integrity of the intestinal barrier and decreased the symptoms of IBS in rats by mediating the expression of IL-17.

Moreover, independent of the IBS subtype, there was a significant decrease in the genus *Bifidobacterium* in IBS patients. As a result, it was yet another intriguing candidate genus for reducing IBS symptoms. IBS symptoms are reduced by *Bifidobacterium*-containing therapies, which are not observed in products that include *Lactobacillus* alone, according to a comprehensive evaluation of probiotics in IBS.[34]

Restricting fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) can help manage the symptoms of irritable bowel syndrome. The low-FODMAPS diet has been clinically proven to be effective in lowering symptoms of IBS.[35] Since FODMAPs can alter microbial composition and microbial metabolite production, one effect of this dietary intervention is on the composition of the gut microbiome.[36] Because not every IBS patient responds, and putting a low-FODMAPS diet into practice could be difficult. There has been a growing focus on the potential role of the gut microbiome in predicting the efficacy of the low-FODMAPS diet due to the role of the microbiome in metabolizing poorly absorbed carbohydrates, which depends on an individual's microbiome composition.[37] New data suggests that there may be baseline differences in microbiome activity and composition that can distinguish between low-FODMMAP diet responders and non-responders. Inflammatory bowel disease (IBD) is primarily characterized by chronic inflammation and colon ulceration, which are also characteristics of ulcerative colitis (UC) and Crohn's disease (CD). The hallmark of IBD, which includes ulcerative colitis and Crohn's disease, is recurrent, chronic inflammation of the GI tract. It is generally accepted that a widespread microbial dysbiosis in the gut, rather than a particular causative organism, is what causes the beginning of both illnesses.[38] Numerous studies have suggested a role for gut microorganisms in the manifestation of IBD, and the gut microbiota is believed to be a crucial component in the formation of mucosal lesions. Previous research has demonstrated changes in the gut microbiota's functionality and composition in individuals with IBD as opposed to those without the disease. A reduction in the microbiota's stability and diversity is a common feature of microbial dysbiosis in IBD.[39,40]

In particular, the most consistent finding from IBD microbiome investigations is a rise in *Proteobacteria* taxa and a decrease in *Firmicutes*. Additionally, compared to healthy control samples, a typical indicator of microbial dysbiosis in IBD patients, particularly in (active) CD, is the decreased number of *Firmicutes* bacteria from the families *Ruminococcaceae* and *Lachnospiraceae.*[41] Since most butyrate-producing bacteria in the human gut are members of these families, they are significant functional components of the gut microbiota. Thus, the observed disruption on a functional level, such as a reduced ability of the IBD microbiota to produce butyrate, can be associated with the depletion of these bacterial groups in IBD.[42] Furthermore, a study utilizing metagenomic and proteomics techniques in the ileal CD microbiota revealed a decrease in metagenomic reads and proteins of significant butyrate producers, *Faecalibacterium prausnitzii* and *Roseburia* spp., as well as an underrepresentation of genes involved in SCFA generation.[43] Because it provides colonocytes with their primary energy source, improves the integrity of the epithelial barrier, and reduces inflammation, butyrate offers therapeutic potential in the treatment of IBD. Consuming bacteria that produce butyrate to boost in situ butyrate production is an alternative probiotic strategy that has been the subject of recent observational and interventional investigations [44]. This may imply that addressing microbial dysbiosis using butyrate-producing bacterial supplements could help IBD patients regain gut homeostasis and overall health.

Table 2: Gut inflammation disorders summary table of key findings.

Disease group	Specific disease	Gut microbes	Main findings
Gut inflammation disorders	Irritable bowel syndrome	*Ruminococcus spp., Clostridium spp., Dorea spp.*	Increased in abundance[45]
		Bifidobacterium spp.	Decreased significantly in abundance (all IBS subtypes)[37,45]
		Faecal bacterium spp.	Decreased in abundance, anti-inflammatory, butyrate production[36,45]
		Enterobacteriaceae spp.	Increased, links to previous intestinal infection and pathogen byproducts[46]
		Lactobacillus spp., Bacteroides spp.	Increased in abundance[46]
	IBD	*Ruminococcaceae spp., Lachnospiraceae spp.*	Decreased in microbiome, butyrate production[42-44]
		Faecalibacterium prausnitzii, Roseburia spp.	Decreased in microbiome, butyrate production[45]

Chronic kidney disease

Research on the makeup of the gut microbiota in CKD patients and the processes by which gut dysbiosis advances the disease are of increasing interest. Both CKD and the gut microbiota have a reciprocal effect on the gut-kidney axis, with the former greatly altering the latter's composition and functioning.[47] On the other hand, through inflammatory, endocrine, and neurologic pathways, the gut microbiota can influence the processes that lead to the onset and progression of CKD. Therefore, by focusing on the gut microbiota, new therapies to stop the progression of CKD may be made possible by understanding the intricate relationship between these two organs. CKD has been linked to changes in the intestinal microbiota, such as a decrease in microbial richness, variety, and uniformity.[48] The intestinal levels of *Enterobacteriaceae*, specifically *Enterobacter*, *Klebsiella*, and *Escherichia*, as well as *Enterococci* and *Clostridium per fringes*, are higher in patients with CDK, while the colonization of *Bifidobacterium spp.*, *Lactobacillus*, *Bacteroides*, *Akkermansia*, and *Proteaceae genera* is lower.[49,50] A changed microbiota in CKD may increase chronic systemic inflammation, as suggested by the negative correlation found between plasma IL-10 levels and the decrease in the abundance of the essential probiotic *Akkermansia muciniphilla* in CKD patients. The development of chronic systemic inflammation is a significant risk factor for CKD. Dietary fibre consumption is reduced in CDK patients, which is a characteristic that is necessary for the formation of SCFAs. Reduced dietary fibre raises amino nitrogen levels, which the gut microbiota can convert to uremic toxins.[51] An imbalance favouring proteolytic microbiota over fermentative microbiome is characteristic of CDK patients. In addition to having negative consequences, imbalance in favor of proteolytic species is essential to development of CKD. Additionally, it was discovered that patients' serum and feces contained less of the major SCFAs, particularly butyrate, as their CKD progressed.[52] Nevertheless, more investigation is required to ascertain whether raising circulating SCFA levels might directly help CDK patients' clinical outcomes. Prebiotic, probiotic, and symbiotic supplementation has been found to have positive effects on the gut microbiota-renal axis in several experimental and clinical trials. These have come to light as a possible therapeutic intervention to correct dysbiosis of gut microbiota, lessen oxidative stress/inflammation markers, and regulate gut-derived uremic toxins, like trimethylamine N oxide (TMAO), indoxyl sulphate (IS), and PCS, which have been linked to advancement of CKD.[53-55]

Table 3: CKD summary table of key findings.

Disease group	Specific disease	Gut microbes	Main findings
CKD	-	*Bifidobacterium, Lactobacillaceae, Prevotellaceae*	Decreased in abundance, SCFA producers associated with anti-inflammatory cytokines.[56,57]
	-	*Enterobacter*	Increased in abundance.[57]
	-	*Klebsiella*	Increased in abundance, pathogen associated with inflammatory disease states including Crohn's.[56, 57]
	-	*Clostridium perfringes*	Increased in abundance, pathogen associated with intestinal diseases.[56]

MENTAL HEALTH DISORDERS

The "gut-brain axis" refers to the connections between gut microorganisms and neurological functions that have been demonstrated to affect each other.[58] Three main avenues exist via which the gut microbiota communicates with the brain: the neural pathway (by the vagus nerve and enteric nervous system), the immunological pathway (via cytokines), and the endocrine pathway (via the HPA axis and gut hormones). A compromised state of this relationship may result in the emergence of mental illnesses. Mental health illnesses may be exacerbated by common gut microbial species from the genera *Bacteroides* and *Bifidobacterium*, as well as the *phylum Firmicutes* and *Actinobacteria.*[59] The gut-brain axis is modulated by gut microbiota in a variety of direct and indirect ways. This includes generating a wide range of metabolites, such as neurotransmitters, SCFAs, and amino acids, and preserving gut permeability by adjusting the integrity of tight junctions in the gut epithelium. These metabolites from the gut can enter circulation and impact the brain, or they can operate locally on the enteric nervous system to affect the central nervous system. Additionally, changes in the levels of gut microbial metabolites, including tryptophan, histamine, ammonia, and SCFAs, have been linked to several neurological conditions, including Parkinson's disease (PD), anorexia nervosa (AN), Alzheimer's disease (AD), autism spectrum disorder (ASD), and chronic stress and depression.[60-62] These relationships may be direct or indirect. More research is necessary to determine if the breakdown of homeostasis in mental health disorders is the result of or the cause of changes in the gut microbiota and its functions.

Considering the available data, numerous investigations have been conducted to precisely target the gut microbiota using various therapeutic approaches, such as the administration of pre- and probiotics (psych biotics) to treat mental health conditions and their symptoms.[63,64] Probiotic combinations of lactobacilli and Bifidobacteria have been shown in human-intervention studies with psychotics to significantly reduce psychological distress.[65] improve communication and cognition in patients with ASD and AD. and alleviate symptoms in patients with PD.[66]

Table 4: Mental health disorders summary table of key findings.

Disease group	Specific disease	Gut microbes	Main findings
Mental health disorders	SCZ, ADHD	*Lactobacillus spp, Bifidobacterium*	Increased in abundance in specific disorders such as SCZ and ADHD.[67,68]
	General anxiety disorder (GAD)	*Bacteriodetes, Ruminococcus gnavus, and Fusobacterium bacteroidaceae, Enterobacteriaceae, and Burkholderiaceae*	Decreased in abundance in general anxiety disorder (GAD). Increased in abundance in GAD.[69,70]
	Post- traumatic stress disorder (PTSD)	*Actinobacteria, Lentisphaerae, and Verrucomicrobia*	Decreased in abundance in post-traumatic stress disorder (PTSD).[71]
	Depression	*Eggerthella., Holdemania, Turicibacter, Paraprevotella prevotella, Dialister*	Increased in abundance amongst individuals with depression.[72,73] Decreased in abundance amongst individuals with depression.[74]

CONCLUSION

The gut microbiota and CDs, such as inflammatory autoimmune disorders, gut inflammation-related disorders, and cardiometabolic diseases, have been clearly linked in the last ten years of research involving both human and animal investigations. It is becoming more and more evident that bacterial metabolites play a major role in the impact of the gut microbiome on human health, at least in part. Of these metabolites, SCFAs seem to be the most significant. Bacteria that produce butyrate have been linked to a decreased risk of irritable bowel syndrome, inflammatory autoimmune diseases, and cardiometabolic disorders.

Although there are several potential treatment approaches that target the gut microbiome, dietary modifications seem to be the most obvious, quick, non-invasive strategy to modify the makeup and function of the gut microbiome. Recent RCTs have demonstrated that certain dietary treatments have a consistent effect on both composition and function. When dietary fibre and unsaturated fat are consumed together or in a balanced diet like the Mediterranean diet, butyrate-producing bacteria are found in larger relative abundances. The resulting SCFAs and these bacteria lead to better health outcomes. distinct dietary fibre kinds cause distinct bacterial alterations and SCFAs. Within the next five years, it should be possible to create dietary interventions that are particularly aimed at raising specific bacterial metabolites to enhance the outcomes related to inflammation, metabolism, and cardiovascular health.

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