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Narrative Review of Microbiome and Non-Communicable Diseases

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Abstract

Non-communicable diseases (NCDs), including obesity, diabetes, cardiovascular disease, cancer, and chronic respiratory disorders, are the leading global causes of morbidity and mortality. Recent advances in microbiome research have revealed its central role in the onset, progression, and potential management of NCDs. The microbiome contributes to host metabolism, immune regulation, gut–brain signaling, and barrier integrity, with dysbiosis linked to systemic inflammation, metabolic dysfunction, and tumorigenesis. Mechanistic pathways include altered production of microbial metabolites such as short-chain fatty acids, bile acids, and trimethylamine-N-oxide, as well as disruption of immune tolerance and epithelial barrier functions. Diet is a major determinant of microbial composition and diversity, influencing NCD susceptibility and progression. Evidence supports that both Westernized and fiber-deficient diets promote dysbiosis, while plant-rich dietary patterns enhance microbial resilience and anti-inflammatory potential. Emerging therapeutic strategies, including probiotics, prebiotics, dietary modulation, and fecal microbiota transplantation (FMT), offer opportunities to restore microbial balance and improve health outcomes. However, methodological challenges, inter-individual variability, and limited longitudinal data complicate translation into clinical practice. This review synthesizes current evidence on the microbiome's role in NCDs, highlighting mechanisms, dietary influences, and microbiome-targeted interventions, while outlining future directions for precision medicine.

Keywords: Microbiome**,** Non-communicable diseases**,** Dysbiosis**,** Gut–brain axis, and Probiotics and prebiotics.

INTRODUCTION

The human microbiome is the collective genomes of bacteria, archaea, viruses, and eukaryotic microbes within the human body or a part of it [1]. This microbiota performs many important functions within the body that may influence some disease pathologies. This narrative review highlights the role of the microbiome in the onset and progression of common non-communicable diseases by discussing observational studies focused on these diseases and advances regarding available interventions [1].

Understanding the Microbiome

The microbiome refers to the collective community of microorganisms, including eukaryotes, bacteria, archaea, and viruses, that inhabit the human body, with persistent microbial colonization comprising an estimated 38 trillion bacterial cells [2]. Traditionally, understanding of microbial diversity at the species-level relied on culture-dependent methods, which accounted for fewer than 20 per cent of bacteria; however, recent advances in culture-independent techniques indicate a considerably more complex, individualized, and dynamic microbiome. Nevertheless, the number of species shared by many individuals remains quite limited. Each anatomical site harbours a distinctive microbial community that reflects the different selective pressures in local habitat conditions [2, 3]. Oral and skin microbiomes are particularly diverse owing to the variation in habitat conditions within these sites, and there is a strong selection imposed by host physiology that determines colonization according to nutrient availability and the physicochemical conditions. In each location, therefore, a unique microbiome with characteristic taxonomic abundances and compositions is maintained. Due presumably to its nutrient-rich nature

and low oxygen environment, the gut supports a particularly large bacterial population, estimated at 1011–1012 cells per gram of content at the colon, but also shows substantial variation in other parts of the tract with a distinct saliva microbiome [3]. The dominant bacterial members of the human microbiome include the phyla Bacteroidetes, Firmicutes, and Actinobacteria.

Definition and Components

The microbiome encompasses the collective assemblage of microorganisms residing across numerous regions of the human body, including bacteria, archaea, viruses, and fungi. These commensal microorganisms inhabit the oral cavity, skin, vagina, lungs, and particularly the gut. Functions of these microorganisms and their genetic material have also been considered within the definition of the host-associated microbiome. Microbial diversity involves the concepts of alpha diversity and beta diversity, reflecting richness and evenness [2]. The evaluation of the diversity of microorganisms can be carried out based on their relationship constructs, such as symbiotic, mutualistic, commensalistic, antagonistic, or parasitic associations. One of the first methods for microbial analysis was the identification of species and their quantification using 16S rRNA gene sequencing and biomarkers. However, this technique only accesses microbial aspects at the order and genus level. Currently, technologies such as shotgun metagenomics provide information about the species inhabiting a niche through the identification of their DNA. Additionally, analytical tools like metabolomics reveal the metabolites present in the niche or system. Collectively, these approaches offer valuable insights into the functional role of the microbiome in specific environments [3].

Microbiome Diversity

The microbiome comprises the collective genomes of microbes residing within an ecological niche, such as the human body [1]. A common misconception equates the microbiome solely with microorganisms themselves, yet it specifically denotes all microbial genes. The microbiota refers explicitly to the microorganisms inhabiting a defined environment like the gut, oral cavity, or skin. Prior to the dawn of metagenomics, understanding microbial diversity was hindered by the inability to culture many species in laboratory conditions [1]. Advances in metagenomics, bioinformatics, high-throughput sequencing, and culture-independent techniques have now facilitated the identification of a wide spectrum of microbes, their genes, and concurrent functional and metabolic activities across diverse human populations and tissues. Research consistently demonstrates that greater gut microbial diversity correlates with improved health, whereas monolayer microbiota associates with disease states. Consequently, numerous therapeutic strategies aimed at restoring gut homeostasis advocate increasing microbial diversity [1].

Methods of Microbiome Analysis

An amplified transcriptome, increased inter-tissue covariance, and decreased inter-individual variation in response to diet were observed for those molecular features associated with reaction norms that plateaued or declined upon entering the richer medium [4]. The wholesome experimental profile captured both the genomic constraint(s) upon molecular plasticity and the (additional) physiological strain(s) enacted by some of the dietary constituents. Several mathematical tools and visualisation techniques facilitate the exploration of links between complex interaction networks and observed physiological outcomes, providing not only a basis for informed hypothesis generation, but also a means to benchmark subsequent genome-scale metabolic models [2]. The plethora of data generated by microbiome studies has attracted increasing interest from the mathematical sciences on the front of microbiome analytics [3]. Whilst core methods have existed for over a decade, much of the proposed framework has been adapted from other 'omics domains and is not well evaluated for microbiome data; therefore, many common approaches are either statistically flawed or operate under cruder assumptions than are necessary. The field remains dominated by empirical or data-driven approaches, providing useful initial insights which in turn support the formulation of process-based models that can inform potential therapeutic interventions [2]. Several issues complicate 16S- and shotgun metagenomic datasets, including compositionality, sparsity, biological variability, and high dimensionality. Further confounding features arise from differences in laboratory methods, sample processing, and contamination that contribute to systematic batch effects, making the extraction of true biological signal from statistical noise troublesome. These problems pose unique challenges in identifying the taxa responsible for a phenotype or the interconnectivity between taxa, eliciting functional or metabolic shifts, and disentangling driver from passenger species, and the rapid progression of techniques for microbiome analytics has yet to convince clinical or industrial researchers of their utility [3]. Research concerns predominantly 16S rRNA datasets from western populations, focusing on microbial responses from faecal samples, with limited longitudinal profiling and very small sample sizes. This restricts the potential for advances both in microbiome analytics and in interpreting the microbiome's contribution to health and disease, emphasizing the need for more rigorous, regularised, and model-driven approaches that can link microbial composition to function and behaviour. The role of microbes in resilient disease states, dysbiosis, multi-factorial disease pathways, and host-environmental

interplay requires validation in experimental or industrial models and presents an opportunity for mathematical modelling and systems medicine to make a transformative contribution [3]. Ultimately, clinical translation will rely upon the ability to engineer microbial communities and/or understand what groups of microbes can be used as robust biomarkers, an ambition only achievable through thorough characterisation and clear depiction of microbial interactions [3].

Non-Communicable Diseases Overview

Non-communicable diseases (NCDs), or chronic diseases, are medical conditions that are non-transmissible between people [1]. Their major types include cardiovascular disease (heart attacks and stroke), cancer, chronic respiratory disease (chronic obstructive pulmonary disease and asthma), and diabetes. In 2019 alone, it was estimated that 41 million deaths, accounting for 74% of all global deaths, were due to NCDs, three-quarters of which occurred in low- and middle-income countries, and many of which occurred before the age of 70. The most common causes of NCD deaths include cardiovascular disease (17.9 million, 32%), cancer (9.3 million, 17%), respiratory disease (4.1 million, 7%), and diabetes (1.5 million, 3%) [1].

Definition and Types

An infectious (communicable) disease is caused by the transmission of an infectious agent from one individual to a susceptible host. Conversely, non-communicable diseases (NCDs) have no transmissible causative agents [1]. Emerging evidence reveals that many NCDs are associated with contagious pathogenic risk factors, thereby blurring the distinction between communicable and non-communicable diseases [1]. Reliable estimates indicate that NCDs account for approximately 70% of total deaths worldwide and are the leading cause of premature adult mortality in many countries 3. NCDs are classified into four main types based on their nature, aetiology and organ system they affect: (i) metabolic syndrome with a cluster of risk factors such as insulin resistance, high blood pressure and obesity; (ii) neurodegenerative disorders including Alzheimer's and Parkinson's diseases; (iii) autoimmune diseases such as multiple sclerosis and systemic lupus erythematosus; (iv) allergic diseases like asthma [5].

Global Burden of Non-Communicable Diseases

Non-communicable diseases (NCDs), also called chronic diseases, tend to be of long duration and are the leading cause of death and disability worldwide [1]. The four main types of NCDs are cardiovascular diseases (e.g., heart attacks and stroke), cancer, chronic respiratory diseases (e.g., chronic obstructive pulmonary disease and asthma), and diabetes. Approximately 41 million people die each year from NCDs, equivalent to 71% of all deaths globally. Of these, 15 million people die prematurely between the ages of 30 and 69, as a consequence of NCDs. This global health crisis has a huge negative impact on economies and has now become a barrier to sustainable development [1].

The Role of the Microbiome in Health

Not only is the human microbial community and its habitat, the microbiome, significant in determining the health of human beings, but all flora and fauna form a habitat for microbes that contribute to the ecosystem. Metabolic activities closely linked to the disease and health status of the plant and animal kingdom are also controlled by microbes [1,2]. The microbiome performs essential functions in metabolism, the immune system, the gut–brain axis, and even in cardiovascular health and brain functions. Healthy individuals harbor a diverse and resilient microbiome. The composition can vary based on geography, diet, ethnicity, and the possibility of vertical transfer from mother to child. Vertical transfer is the seeding of the maternal flora into the fetus during labor. This flora stabilizes within 3 years. There is an increasing recognition that the ecosystem contributed by the microbiome can either assist in the prevention or development of disease [1]. The wrangling of the ecosystem to alter the disease susceptibility, or modulating its course, has now become a major focus of research. The advancement in instrumental methodologies has added a different dimension to the ability to study the microbiome. Once defined on the basis of the biological specimens, cultures for isolation, and functional assays, the study of the microbiome has shifted to molecular diagnostics for a better level of specificity. It can now provide information on the abundance of different microbes, the composition of the community, and the overall ecology of the flora in the community [3]. It can also assess the relation of these parameters with the general health of individuals and populations and their association with various diseases in order to understand the pathogenesis and modulation of disease course. The spearheading techniques towards this research are NGS and microarray-based studies that provide enormous information on the phylogenetic and functional assessment of the microbial population in a state of disease or health. The most commonly reported taxa in metabolic disease are the members of phylum Firmicutes and Bacteroidetes [1,4].

Metabolic Functions

The importance of the gut microbiome to human health and disease was first reported in the early 20th century. Its roles in helping digestion, training the immune system, and regulating the central nervous system have been gradually recognized [1]. Alterations of the microbiome were discovered in different non-communicable diseases (NCDs), including obesity, diabetes, cardiovascular disease, cancer, and chronic respiratory diseases. Inflammation is believed to be a shared feature of these NCDs [2]. Microbiome-mediated bile acid and short-chain fatty acid metabolism affect systemic inflammation through the engagement of G-protein-coupled receptors or directly modulating key inflammation-associated pathways such as the nuclear factor-kappa B, Janus kinase, and signal transducer and activator of transcription, interleukins, and tumor necrosis factor. Changes to gut permeability may cause gut-derived endotoxins such as lipopolysaccharides to enter systemic circulation, eliciting systemic low-grade inflammation and insulin resistance. Recent findings highlighted the importance of gut microbiome profiling, which can improve the prediction of risk for NCDs [4].

Immune System Modulation

A finely tuned relationship between the microbiota and the immune system is of paramount importance. A disturbance of the microbiota, in particular of the gut microbiota, appears to be a potential feature of obesity and/or type II diabetes mellitus, reflected in a dysregulated immune response. The perturbation of this relationship might be the initial trigger for developing an impaired immune response towards changes in glycemic levels [1]. The microbiota–immune association is well investigated in inflammatory bowel diseases that are considered the result of a loss of tolerance towards microbiota [2]. Immune system stimulation and disease amplification via Toll-like receptor activation are caused mainly by translocated bacteria and metabolites. Experimental studies show that depletion of gut microbiota in mice by antibiotic administration leads to the absence of an immune response and makes them more susceptible to chemically induced colitis. On the other hand, colonization by commensal microbiota establishes an appropriate immune response. Dysbiosis appears to activate immune system components, which further enhance existing inflammation [2]. The presence of gut inflammation and obesity has some reciprocal influences that may be aggravated by microbiota dysbiosis. On the contrary, preclinical data show that transfer of healthy gut microbiota from lean donors reduces inflammation in diet-induced obesity [3].

Gut-Brain Axis

Through the gut-brain axis (GBA), the gastrointestinal tract has a bidirectional connection with the central nervous system (CNS) [1]. The GBA entails numerous intertwined communicating networks such as the neuroanatomic pathways and central nervous systems, neuroendocrine-hypothalamic–pituitary–adrenal axis, gut immune system, entero-endocrine system, microbial metabolite, and intestinal microbiota. Microbial colonization promotes the maturation and development of microglias that originate in the mesenchyme of the yolk sac and migrate to the CNS during early embryogenesis [1]. Microglia also mediates the inflammatory response in the CNS through the toll-like receptor, as demonstrated in some in vitro studies. Microbial butyrate independently regulates microglia-dependent amyloid-β clearance. The improvement of microglial function through a proper gut microbiota composition then represents a potential approach to slowing the development of Alzheimer's disease (AD). Entero-endocrine L-cells synthesize peptide YY and glucagon-like peptide-1 from proglucagon polypeptides following stimulation of SCFAs [3]. Serotonin biosynthesis is increased by the gut microbiota through increased enterochromaffin cell number. Intestinal serotonin supplements the circulating serotonin pool and mediates postprandial gastrointestinal motor and secretory reflexes [4]. The gut microbiota induces stress responsiveness via the HPA axis by decreasing the production of corticosterone and adrenocorticotropic hormones; impairment of the HPA axis in germ-free (GF) mice leads to anxiety-like behavior.

Microbiome and Chronic Diseases

Non-communicable diseases (NCDs) are the leading causes of death globally. The growing prevalence of NCDs may be partly explained by rapid and multifaceted changes in the exposome, including perspectives that consider the internal microbial ecosystem of the human body [1]. This internal microbial ecosystem is crucial to maintaining human health by influencing metabolism, immune function, and the gut–brain axis. These key features of the microbiome are involved in numerous chronic diseases: obesity, type 2 diabetes mellitus, cardiovascular diseases, cancer, and chronic inflammatory and allergic diseases of the respiratory organs [4]. The microbiome is implicated in the etiopathogenesis of various diseases. Rapid and substantial changes in the human lifestyle and environment may lead to disruption of the balance between host and microbiota. This disruption can be either a cause or a consequence of many pathological conditions and illnesses of the digestive system. Microbial imbalances favoring pathological bacteria may result in the transfer of toxins through the damaged intestinal wall,

triggering excessive immune responses and chronic inflammatory processes in the body. Such a pathomechanism appears common in obesity, insulin resistance, and cardiovascular diseases [4,5].

Obesity

The prevalence of many non-communicable diseases (NCDs) has increased tremendously over the last decades. This is particularly true for obesity and metabolic disorders associated with health conditions such as diabetes, cardiovascular disease, and even some types of cancer [5]. Microbial metabolism links bacteria with obesity development. Genomic analysis of the gut microbiome detected a reduction of gene richness in obese subjects. However, the link between the faecal microbiome and body weight remains to be confirmed. Obese individuals were found to have a reduced abundance of health-associated bacterial species such as Akkermansia muciniphila and F. prausnitzii. Furthermore, different groups of patients stratified by their Firmicutes to Bacteroidetes ratio showed distinct metabolic capabilities, i.e., a reduced production of acetate but increased synthesis of the SCFAs propionate and butyrate in obese individuals [6]. These findings indicate that the human gut microbiome is associated with energy metabolism and that obesity is a condition related to dysbiosis.

Diabetes

Substantial evidence from clinical studies and microbe-based therapies reveals a significant association between the gut microbiome and diabetes mellitus. Insulin sensitivity demonstrates a positive correlation with the relative abundance of phylum Firmicutes and genera such as Clostridium, Subdoligranulum, Eubacterium, and Faecalibacterium, alongside an inverse association with phylum Bacteroidetes. Moreover, the incidence of Type 2 Diabetes Mellitus (T2DM) corresponds positively with the presence of phylum Bacteroidetes and genera Ruminococcus, Fusobacterium, and Blautia, while associating negatively with phylum Firmicutes and genus Bifidobacterium [5]. Dysbiosis within the gut microbiome leads to hyperglycemia, systemic inflammation, body-weight alterations, and insulin resistance. Elevated blood glucose levels and systemic inflammation cause insulin receptor malfunctions and diminished insulin production in pancreatic beta cells, resulting in chronic hyperglycemia characteristic of diabetes. Additional contributing factors include elevated serum lipopolysaccharide (LPS) levels, reduced populations of butyrate-producing bacteria, impaired integrity of the gut epithelial barrier, and metabolic endotoxemia [6].

Cardiovascular Diseases

The past decade has brought to light new knowledge demonstrating a crucial role of gut microbes in cardiovascular diseases. For instance, the gut microbiome plays a role in the development and progression of atherosclerosis and regulates the variation of risk factors of cardiovascular diseases, such as lipid levels and hypertension [5, 6]. Furthermore, bacteria and bacterial products have been found in diseased atherosclerotic plaque; however, how the microbiome causes coronary artery disease remains unclear. Recent studies suggest that host-microbiome metabolic interactions contribute to atherogenesis [5]. Vascular metabolite trimethylamine-N-oxide (TMAO), derived from dietary choline and carnitines through gut microbial metabolism, is known as an independent risk factor of atherosclerosis and cardiovascular disease. Nineteen gut microbiota genera are independently associated with blood pressure variations in humans and explain 4.4% of the variance in blood pressure levels. Hypertension is associated with an increased Firmicutes-to-Bacteroidetes ratio and reduced microbial diversity and gene richness. Lactobacillus species may play an important role in hypertension and salt-sensitive hypertension via anti-inflammatory actions. Studies in germ-free rodents confirm observations in human studies that the gut microbiome can affect blood pressure [6]. Colonizing germ-free rats with a normal microbiota increases blood pressure, indicating the involvement of the microbiome in hypertension.

Cancer

Cancer is the world's second leading cause of death, accounting for 9.6 million deaths in 2018. Increasing evidence has identified a human gut microbial dysbiosis in colorectal and other epithelial cancers, with distinct taxa associated with colorectal cancer observed in CRC 6. The mechanism(s) linking these observations remain unclear, but recent evidence supports a role for microbial-driven epithelial–mesenchymal transition (EMT) and oncogenic inflammation across multiple cancers [5]. CRC is the single most prevalent gastrointestinal malignancy, and causality has been assigned to individual 'oncobacteria' like Fusobacterium nucleatum and enterotoxigenic Bacteroides fragilis [7]. However, F. nucleatum and altered anti-tumour immune profiles also contribute to carcinogenesis in other tumours. Synthesis of epidemiological, experimental, and metagenomic data indicates that reciprocated microbe–cancer cell interactions emerge during tumour development and maintain cancer progression in multiple tumour types [7]. The current chemotherapy and radiation therapies for these cancers are only partially effective, and additional strategies are required [7].

Chronic Respiratory Diseases

Chronic respiratory diseases (CRDs) represent a significant burden to healthcare systems and remain a leading cause of death worldwide. CRDs refer to persistent diseases of the airways and other structures of the lung. The most common CRDs include chronic obstructive pulmonary disease, asthma, occupational lung diseases, and pulmonary hypertension[8]. In 2020, 3.23 million people died from CRDs, accounting for 6% of all global deaths. The respiratory tract is colonized by diverse microbiota (bacteria, fungi, and viruses) that collectively create a unique microbial niche. The lung has long been recognized as vulnerable to colonization by microbial species [8]. A study of bronchial microbial communities in healthy and asthmatic subjects demonstrated significantly increased levels of Haemophilus, Moraxella, and Neisseria in asthmatics compared with controls. The links between the microbiome and respiratory diseases have also been widely studied. The respiratory microbiome is relatively depleted in alpha diversity (a measure of species diversity within an ecological community) in chronic respiratory diseases. This is supported by a number of observational studies reporting variable reductions in alpha diversity across chronic respiratory diseases [8].

Mechanisms Linking Microbiome to Non-Communicable Diseases

Alterations in the human microbiome contribute to the development and progression of various non-communicable diseases (NCDs) [1]. These alterations promote inflammation, alter the production of important metabolites, and disrupt the integrity of the gut barrier. The gut microbiota produces numerous metabolites—such as short-chain fatty acids, bile acids, and vitamins that modulate the immune system via signaling receptors. Dysbiosis during NCDs changes the levels of many of these microbially derived compounds, thereby contributing to immune dysregulation and chronic inflammation [1]. This interplay between altered bacterial metabolite secretion and immune modulation forms the basis of the germ-organ theory, which posits that host control over microbial ecosystems plays a key role in NCD pathogenesis. The theory is supported by modified Koch's postulates tailored for NCDs, which state that dysbiotic microbiota are present in affected individuals, can be cultured, induce disease when transferred to healthy hosts, and can be re-isolated from those hosts. These mechanisms highlight the microbiome as a potential target for therapeutic interventions aimed at restoring immune homeostasis and mitigating NCD risk [1].

Inflammation

Growing evidence indicates that chronic systemic inflammation is a fundamental cause of a wide variety of NCDs. Interactions with the intestinal microbiota seem to have a crucial role in the primary and secondary maturation of the immune system, as well as in regulating peripheral immune responses in adult life. Therefore, overt or subtle changes in the composition of the microbiota can generate inflammatory responses with a systemic impact [2]. The possibility that diet, the substrate for the metabolism of gut bacteria, influences the composition of the microbiota and, consequently, the systemic inflammatory response has received considerable scientific attention [5]. The bacterial families Bacteroidaceae, Prevotellaceae, Porphyromonadaceae, and Rikenellaceae have been associated with the production of proinflammatory cytokines such as interleukin (IL)-1β, IL-6, and tumor necrosis factor (TNF)-α. Conversely, bacteria such as Akkermansia muciniphila or Faecalibacterium prausnitzii have been associated with the expression of IL-10 or transforming growth factor (TGF)-β, which play important anti-inflammatory functions. Some bacteria can modulate proinflammatory mediators such as nuclear factor κβ (NF-κβ) or protect intestinal barrier function by maintaining the integrity of tight junction proteins. Microbial metabolites can also influence the inflammatory response; for example, SCFAs can promote a hypomethylated state of the gene promoter for Foxp3, which results in enhanced differentiation of naive T cells into regulatory T cells [6].

Metabolite Production

The human microbiome influences non-communicable diseases via multiple mechanisms, including inflammation, metabolite production, and gut barrier integrity. Metabolites secreted by gut bacteria participate in key physiological host processes, contributing to health or disease during dysbiosis [3]. Altered bacterial metabolites thereby drive immune dysregulation and chronic inflammation common in many non-communicable conditions. Identification of microbial metabolites that modulate host immunity, together with elucidation of their underlying mechanisms, offers prospects for targeted microbial therapeutics. A combination of experimental and in silico approaches, including multi-omics datasets, systems biology, and modelling of in vitro or animal experiments, facilitates mechanistic discovery [6]. Contextualised metabolic models in particular enable prediction of host-microbiome-disease interactions, which can be experimentally tested. Integrative use of metabolomics, other -omics, bioinformatics, and systems biology delivers a trans-omics strategy through which such complex challenges may be met and clinically relevant interventions developed [3]. This approach further supports precision medicine by allowing the prediction of individual responses to drugs and diet. Microbial genome-scale metabolic models (GEMs) combined with comprehensive host metabolic reconstructions, such as Recon 3D, enable analysis of

metabolic interplay between microbial communities and the host [3]. This integration supports personalised modelling of microbe–microbe and microbe–host interactions, clarifying the causal role of microbiome alterations in disease pathology and revealing corresponding metabolic adaptations in gut and host environments [5].

Gut Barrier Function

The gut barrier influences intestinal permeability, allowing the passage of microorganisms, toxins, and molecules both from inside and outside of the intestinal lumen into the systemic circulation. Consequently, gut barrier dysfunction leads to microbial translocation and low-grade inflammation, with the induction of several cardiometabolic disorders, including insulin resistance, dyslipidemia, and atherosclerosis [6]. Furthermore, gut barrier dysfunction is related to the development of tumorigenesis processes and the activation of immune responses in the lungs. Specifically, metabolite-related mechanisms affecting gut barrier function include those associated with lipopolysaccharides (LPS) and trimethylamine N-oxide (TMAO), which is related to subclinical atherosclerosis and venous thromboembolism, both conditions with an inflammatory-based tissue injury [5]. The effects of retinol, spermidine, and putrescine on the gut barrier may be associated with a reduced risk of gastrointestinal cancer through physiological modulation of the intestinal barrier and intestinal microbiota [7].

Dietary Influences on the Microbiome

Dietary Intake as a Primary Determinant of Microbiome Composition. Dietary intake is one of the most effective factors in determining the structure and diversity of the gut microbiome [1]. Diet and the type of nutrients consumed have been linked to the development of NCDs, such as cardiovascular disease, T2DM, partly through their effects on the microbiome. In Europe in 2013, diet accounted for just over 15% of attributable deaths. Specific dietary compositions modulate the microbiome and its metabolic actions, contributing to the risk of NCD development [6]. Associations Between Dietary Intake, Microbiome, and Health. Studies have linked adverse health outcomes to complex dietary factors related to the microbiome, including habitual dietary patterns, insufficient intake of components such as whole grains, poor adherence to diets like the Mediterranean diet, and timing of eating 9. Dietary Patterns Versus Macronutrients in Microbiome Modulation. Current understanding suggests that dietary patterns exert stronger associations with the microbiome and disease states than individual macro- and micronutrients. Studies of specific dietary patterns have been reviewed in the context of microbiome and NCDs, underscoring the impact of overall dietary habits on microbial composition and function [5].

Impact of Diet on Microbiome Composition

Dietary components have an immense impact on the composition of the microbiome [9]. Dietary and nondietary components can be digested by the host or by the microbial communities in the gut. Because the metabolism of different components leads to a different collection of nutrients available for microbial growth, diet has a powerful effect on the microbiome [10]. Diet is the most straightforward and potentially achievable approach to alter the microbiome to promote health and prevent disease [11]. Increased knowledge about the role of the microbiome in the different NCDs could allow dietary strategies for prevention and treatment by modulating the microbiome.

Specific Dietary Patterns

Studying the interplay between diet, the microbiome, and health has advanced as a critical research area for preventing non-communicable diseases (NCDs). Dietary components influence necessary activities of intestinal microbes, such as spreading antimicrobial resistance and producing beneficial molecules like short-chain fatty acids [12]. Policy decisions, effective interventions, and improved human well-being depend on understanding how habitual diets modulate the microbiome. Westernized diets, high in fat and low in fibre, reduce species diversity, cause a loss of bacteria specialised in fibre fermentation, and increase gut exposure to pro-inflammatory and carcinogenic metabolites [12]. Experimental models confirm these alterations can be transmitted across generations and partially reversed by dietary fibre reintroduction. Ancestral diets rich in plant-based foods and fibre promote ecological diversity, supporting bacteria that metabolise undigested nutrients and maintain protective microbial functions. Activities generate up to 50% of body-fluid metabolites, including short-chain fatty acids such as butyrate, which strongly influence organs, including the brain. Diet's plasticity subsequently allows reversal of dysbiosis and restoration of healthy host-microbe interactions [12]. Dietary patterns consistently correlate with bacterium groups sharing functional roles in health and disease. Specific foods and nutrients are associated with species known to confer mucosal protection and anti-inflammatory effects. Microbial mechanisms thus provide a rationale for future intervention studies [13].

Interventions Targeting the Microbiome

Non-communicable diseases (NCDs) such as obesity, type 2 diabetes, cardiovascular disease, cancer, and chronic respiratory diseases have emerged as major global health concerns. Concurrently, mounting evidence suggests that the microbiome may be causally linked to NCDs, creating new prospects for intervention [11]. The gut microbiome, the vast collection of microorganisms and their genetic material residing within the gastrointestinal

tract, demonstrates differential compositions in NCDs [12]; they also seem to play a direct role in the trajectory of metabolic disorders [5]. Thus, manipulating the microbiome by modulating host diet, administering probiotics, or performing faecal microbiota transplantation (FMT) to suppress predispositions to metabolic or immune-mediated NCDs is under intense investigation [5].

Probiotics

The administration of living but non-pathogenic microorganisms to impact a host's health favorably defines probiotic therapy. Although most evaluated probiotics belong to the genera Lactobacillus and Bifidobacterium, other bacteria, such as Streptococcus, Enterococcus, Bacillus spp., and even some yeasts have been used [13]. Though the full range of probiotic effects remains uncertain, potential benefits include regulating the intestinal microbial environment, inhibiting colonic carcinogenesis, reducing serum cholesterol, modulating the immune system, producing vitamins, improving lactose intolerance, and preventing Helicobacter pylori infection. In disease settings, various strains of Lactobacillus (L. rhamnosus, L. acidophilus, L. casei) and Bifidobacterium (B. longum, B. breve, B. bifidum) have been employed to treat or prevent gastrointestinal disorders such as lactose intolerance-related diarrhea, acute or persistent diarrhea, infantile colic, necrotizing enterocolitis, and Helicobacter pylori infections [11]. Confirmed probiotic effects encompass the treatment and prevention of diarrhea, modulating allergic conditions, and lowering total cholesterol levels. Nevertheless, the efficacy of probiotics depends on the specific strain used, the health condition considered, and individual patient characteristics [12].

Prebiotics

The concept of prebiotics was first introduced in 1995 with the aim of fine-tuning the composition and function of the gut microbiota to promote health and well-being [14]. Recently, the International Scientific Association for Probiotics and Prebiotics (ISAPP) defined a prebiotic as "a substrate that is selectively utilised by host microorganisms conferring a health benefit". Prebiotics represent a prominent class of bioactives already included in many foods. Many studies have investigated their health effects, revealing important roles in colorectal cancer, neurovegetative diseases, cognitive functions, intestinal diseases, obesity, type-2 diabetes, metabolic syndrome, osteoporosis, and immunity [15]. The term "prebiotic" provides a more meaningful and comprehensive description of various stimulating food components than the traditional term "fiber". The mechanisms underlying the selective stimulation of beneficial microbes include factors such as molecular structures and surface glycans, as well as the ability of certain species to utilise specific substrates [14, 15].

Fecal Microbiota Transplantation

Fecal Microbiota Transplantation Fecal microbiota transplantation (FMT), the transfer of intestinal microbiota from a healthy donor to the gastrointestinal tract of a recipient, is gaining attention for treating various microbiota-associated disorders [16]. Its approved use for antibiotic-refractory Clostridium difficile infection is supported by large randomized trials. Additional applications encompass chronic intestinal pseudo-obstruction, recurrent urinary tract infections, and the eradication of multidrug-resistant organisms. Immune-mediated conditions, including immune checkpoint inhibitor colitis and hepatic encephalopathy, are also under investigation. Protocols for assisted delivery, such as transendoscopic enteral tubing, facilitate administration. The contemporary regulatory landscape favors personalized microbiota-based therapies over simplistic fecal transplants [17]. Further research addresses unexpected outcomes, ethical considerations, and long-term efficacy across diverse medical conditions. The evolving understanding of the gut microbiome highlights FMT's potential yet underscores the need for continued exploration [16, 17].

Future Directions in Microbiome Research

Research into the microbiome and its association with non-communicable diseases (NCDs) is a rapidly advancing field. Although the extant body of evidence is considerable, the role of the microbiota in the etiopathogenesis of NCDs is still not fully understood, and many challenges remain. Recent advances in dietary approaches connected to gut microbiota manipulation open promising perspectives for the prevention of NCDs. Despite progress, there remain many unexplored aspects of the function of different microbiomes in human body diseases [16]. Future perspectives encompass the application of artificial intelligence to forge new links between microbiota composition and diseases, the development of microbiota-targeted therapies, the implementation of personalized medicine, and the enhancement of public-provider and patient-centered education. With the accumulation of more microbiome-disease association data, novel treatment targets will emerge [17]. The support provided by artificial intelligence will aid in selecting personalized probiotic interventions tailored to patients' specific microbiota characteristics, thereby accelerating the clinical implementation of microbiota-directed diagnostics, prevention, and therapy for NCDs. Therapy aimed at rebalancing the intestinal microbiota holds promise for decreasing disease risk, mitigating symptoms, and reducing the disease burden [17].

Emerging Technologies

Studying the complex microbiome–host ecosystem demands appropriate technological and methodological tools. Techniques that can characterize specific features of microbial communities (e.g., anatomy, metabolic activity, species interactions) provide essential evidence for understanding alterations in certain diseases. Increased investigation into microbiome signatures has spurred efforts to transfer these signals into clinical applications as new strategies for prevention, diagnosis, and treatment of NCDs. Emerging technologies, most of which are evolving rapidly, often contend with incomplete data, knowledge gaps, inconsistent experimental conditions, and technical variability [18]. Despite these challenges, such technologies promise to restructure societal and individual visions of microbiota-associated diseases by facilitating the development of safe, effective, personalized microbiome-based medicines [3]. Additionally, microbiome research is invigorating artificial intelligence, generating an abundance of data and stimulating the creation of personalized systems, including intelligent wearables, real-time health monitoring devices, and self-adaptive drug carriers, all contributing to the advancement of personalized and precision medicine [18].

Personalized Medicine

Vancomycin-resistant Enterococcus (VRE) is an antibiotic-resistant bacterial pathogen prevalent in healthcare environments. Among patients who become VRE-colonized, a subset goes on to develop invasive disease. Using metagenomes obtained from 236 hospitalized patient rectal swab samples, three bacterial community states that vary in the abundance of Enterococcus (the VRE genus) and generally correspond to different stages of VRE-prevalence are identified [17]. In a subset of 74 samples from patients who tested VRE-negative upon hospital admission and later either became VRE-colonized or remained VRE-negative, a strong murine model association between Enterococcus expansion and a decrease in both community size and the anaerobic commensal Clostridiales is found. This study highlights the importance of community size in colonization resistance and identifies specific taxa whose enrichment is associated with VRE colonization [19]. Deciphering the intricate interactions within the microbial ecosystem is essential for optimizing human well-being, as the ecosystem encompasses one of the most sophisticated continua of symbiotic relationships on earth. Dietary preferences are among the most potent forces affecting the composition of our intestinal microorganisms, and this impact may change dramatically throughout the life cycle of an individual. Linking these interactions with gastrointestinal health opens opportunities for the design of personalized nutrition strategies in gastrointestinal health [18].

Public Health Implications

The ongoing SARS-CoV-2 crisis has diverted significant resources towards healthcare and also revealed vulnerabilities in public health systems. The pressures placed on economies, particularly the developed world, have been substantial. This is why the effects of the COVID-19 pandemic on other diseases have become a research priority, and how non-communicable diseases (NCDs) are much less researched than communicable diseases (CDs) [20]. However, the risks associated with NCDs are compounded by the current crisis; treatment interruptions resulting from the shutdown of pharmacies and drug supply chains leave patients with little choice but to withhold medication. Lockdowns signal the potential for increased stress and often lead to a reduction in physical activity and a shift in diet towards foods of lower nutritional value [20]. Interventions intended to control the COVID-19 pandemic may put many at higher risk of morbidity and mortality because of NCDs. Sick individuals appear to be at greater risk of severe COVID-19, but the mechanisms underlying this susceptibility are not clear and may involve multiple factors, including shared risk factors, the infection itself, or the resulting treatments [20].

CONCLUSION

The microbiome is emerging as a critical determinant in the development and progression of non-communicable diseases, bridging metabolic, immune, and neuroendocrine pathways. Dysbiosis contributes to chronic inflammation, metabolic dysfunction, and impaired barrier integrity, linking microbial alterations to obesity, diabetes, cardiovascular disease, cancer, and chronic respiratory conditions. Diet remains the most powerful and accessible modulator of microbiome composition, with plant-rich, fiber-based dietary patterns promoting microbial diversity and resilience, while westernized diets foster dysbiosis and inflammation. Interventions such as probiotics, prebiotics, and fecal microbiota transplantation show therapeutic promise, though their efficacy remains highly strain- and context-dependent. Future research must address methodological standardization, inter-individual variability, and the integration of microbiome analytics into longitudinal studies. Multi-omics approaches and systems biology hold potential to identify predictive microbial signatures and guide precision interventions. Ultimately, leveraging microbiome science for the prevention and treatment of NCDs could transform global health by enabling strategies that are preventive, personalized, and sustainable.

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