Research Article

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The Oral Microbiome And Oral and Upper Gastrointestinal Diseases

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1. Abstract

tions are significant. Microbiomes and the human body coexist peacefully under normal conditions, but disruptions in this balance can result in a number of illnesses. Numerous gastrointestinal and dental disorders are influenced by the oral microbiota and are influenced in their development. The association between oral microbiomes and disorders of the mouth and upper gastrointestinal tract is the main topic of this review, along with therapeutic approaches that might offer useful insights for clinical prevention and therapy. Techniques: We performed searches with no date constraints using terms like “oral microbiome,” “oral flora,” and “gastrointestinal disease” in PubMed, Google Scholar, and Web of Science to find pertinent studies. The publications that were recovered were then the focus of a narrative review. Findings: According to this analysis, there is a strong correlation between oral microbiomes and gastrointestinal and dental disorders including such as upper gastrointestinal tumors (mostly malignant ones), reflux esophagitis, gastritis, periodontitis, and dental caries. Saliva and buccal mucosa are examples of oral samples that are easier to obtain and have better sample stability than gastrointestinal tissues. Therefore, before conducting an endoscopic examination, investigation of the oral microbiome may prove to be an effective preliminary screening tool for high-risk groups. Additionally, therapies based on oral microbiomes may help in the early detection and management of these illnesses. In conclusion: Oral and gastrointestinal disorders are largely dependent on oral microbiomes. Treatments focused on the oral microbiomes may make it easier to identify and treat these illnesses early on.

Important messages
1. The mouth cavity contains a variety of microbiomes.
2. Oral bacteria and human health normally coexist peacefully, but once

2. Introduction

The ratio of the microbiome to human cells is around 1:1, making it an essential part of the human body [1]. Furthermore, the overall number of genes in the human genome is 150 times smaller than the total number of genes in the microbiome. Microbiomes and human bodies usually live in harmony with each other. Any disruption to this delicate balance, however, can result in a number of illnesses [2–5]. The relationship between the human body and its microbiome has garnered a lot of interest in recent years. The complex microbiome found in the human oral cavity is a result of its unique anatomical characteristics, interaction with the outside world, and humidity levels [2]. It has the greatest microbial species variety and density in contrast to other bodily parts [6]. The primary components of the oral microbiome are viruses, bacteria, fungus, archaea, and protozoa [2]. These microbes are common in entire saliva and mostly colonize the dental and oral mucosal pellicle [7]. Studies have verified that an individual’s oral microbiome undergoes variations in diversity throughout the course of their lifetime, following the initial colonization by early-life bacteria [2, 8]. There has been a great deal of research done on the connection between oral microbiome dysbiosis and the ensuing effects on human health. A number of oral disorders, most notably dental caries and periodontitis, have been linked to an imbalance in the oral microbiota. Additionally, new data contrasted with other bodily regions [6]. Bacteria, fungi, viruses, archaea, and protozoa make up the majority of the oral microbiome [2]. Whole saliva will have a high concentration of these bacteria, which mostly colonize the dental and oral mucosal pellicle [7]. Studies have verified that the variety of the oral microbiome varies over the course of an individual’s life after the early colonization by the first bacteria [2,8]. Many studies have been conducted on the relationship between oral microbiome dysbiosis and the ensuing effects on human health.

Many oral illnesses, including dental caries and periodontitis, have been linked to an imbalance in the oral microbiota. Moreover, new data Oral microbiome and oral diseases Periodontitis The immune system is activated by bacteria that cause an inflammatory response in the periodontal tissues as the disease progresses. The immune system then releases inflammatory mediators, such as chemokines and cytokines, which draw neutrophils and other immune cells to the location of inflammation. The activation of these immune cells results in the breakdown of tissue and triggers more inflammatory responses, which in turn propels the progression of periodontitis. Significant clinical features of periodontitis include irreversible tissue degradation, alveolar bone loss, gingival bleeding, and loss of periodontal attachment [45]. Periodontitis is an inflammatory illness. These signs set periodontitis apart from gingivitis, which only affects the gums' outer layers and is typically curable [46].

The identification of these permanent indicators is necessary for the diagnosis of periodontitis, which essential for managing and assessing healthcare cases. Tannerella forsythia, Porphyromonas gingivalis, and Treponema denticola have been identified as the pathogenic trio [47, 48] associated with periodontitis by traditional culture-based techniques. The list of pathogens has been expanded by molecular techniques to include species in the genera Prevotella, Desulfobulbus, and Synergistes, as well as Gram-negative members of the Bacillotaphylum (Dialister spp., Megasphaera spp., and Selenomonas spp.) and Grampositive bacteria like Filifactor alocis and Peptoanaerobacter stomatis [49,50]. This finding emphasizes the role that a variety of oral microbes play in the development and genesis of periodontitis. Thus, it is essential to look into the pathophysiology and treatment approaches of periodontitis from the perspective of oral microbe-host interactions. Dental plaque buildup is the primary cause of the common site-specific inflammatory disease known as gingivitis. Edema and gingival erythema are its defining features. without the periodontal attachment being lost. Gingivitis is often painless, although it can occasionally result in spontaneous bleeding that happens when cleaning and probing the teeth. Even though gingivitis is curable, periodontitis can develop from it if treatment is not received. A more severe form of periodontal disease called periodontitis is characterized by gingival inflammation as well as loss of bone and attachment of connective tissue, which can eventually cause tooth loss and movement [51, 52].

Periodontitis is characterized by tooth mobility and possible tooth loss in addition to gum redness, swelling, bleeding, and halitosis. Young people are mostly affected by aggressive periodontitis, a rare but severe form of periodontal disease that causes extensive tooth destruction and accelerates the illness's course [53, 54]. Remarkably, individuals suffering from invasive periodontitis generally exhibit reduced buildup of plaque and calculus. in the adherence of microorganisms to dental surfaces. Consequently, sucrose Chemical substances connected to Streptococcus mutans are essential in promoting the development of tooth caries [68]. In reaction to a surge in cariogenic infections can quickly adapt by changing their metabolism when there are carbs in the diet [68]. Plaque pH levels have been observed to rapidly drop to 4.0 or below within a few minutes after carbohydrate stimulation [71]. The majority of native microorganisms cannot survive in such an acidic environment; when the pH falls below about 5.5, they stop growing or even perish. In order to adjust to this abrupt pH drop, acidogenic microbes produce acid metabolites such lactic acid, which encourage the demineralization of enamel. Consequently, the crucial pH value connected to the demineralization is around 5.5.

Dental caries in children and adults has historically been linked to important pathogens such as Streptococcus mutans and Lactobacillus spp. [72,73]. A more thorough evaluation of the complete oral microbiota has been made possible by recent developments in high-throughput genomic analysis, which have revealed a complex interplay of possible pathogens involved in the development of caries. Beyond what is commonly known, the "extended caries ecological hypothesis," which was presented in recent research [74], proposes that even though Streptococcus mutans may not be the most prevalent species in the oral microbiome, it has the highest cariogenic potential because of its special ability to produce acid, its ability to adhere to surfaces, and its ability to survive in an acidic environment. This theory is additionally reinforced by the caries progression succession model put out by van Ruyven et al. [75], which describes a dynamic change in the makeup of microbes along the course of dental caries formation. According to this hypothesis, dental plaques develop an acidic milieu due to the early colonizers, which are mostly aciduric and acidogenic microorganisms such as non-mutans streptococci and Actinomyces spp. More cariogenic organisms, such as Streptococcus mutans, might proliferate as a result of this environmental acidity, which intensifies the demineralization process and advances the caries lesion [76]. Even though these results are noteworthy, further research is necessary to create focused preventive measures and therapeutic approaches because of the complex interactions between these microbes and how they together affect the development of dental caries.

3. Barrett's esophagus

Barrett's esophagus (BE), defined as a unique intestinal metaplasia that replaces healthy mucosa [171], is thought to be a precancerous condition of esophageal cancer, and its incidence has been steadily rising in recent years [209,210]. The precise pathogenic mechanisms are still unknown, though. Changes in the esophageal flora and oral saliva, according to some experts, may have an impact on the onset and progression of BE [211]. Related research, however, have produced contradictory findings. Rather than using invasive and costly endoscopic procedures, early

detection of salivary microbes can help detect BE and stop it from progressing to esophageal cancer if oral microbiological changes in BE patients are specific. This is especially true for high-risk patients who are predisposed to developing BE. Patients with BE have quite different oral microbiomes [212]. This could be brought on by oral bacteria moving to the distal esophagus and creating a steady microenvironment [213,214]. Snider et al. demonstrated that whereas Neisseria, Lautropia, and Corynebacterium were less common in BE patients, Streptococcus, Veillonella, and Enterobacteriaceae were more common. In order to test for microbial makeup, swabs from the uvula and the endoscope itself were acquired in another investigation [215], and biopsies were taken from the proximal, middle, distal, and BE of the esophagus. Using models of the microbiome's metabolism, some researchers have forecasted major

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Abbreviations

CPR, radiation candidate phylum. The Human Microbiome Project, or HMP. PSD, dysbiosis, and polymicrobial synergy. gingival crevicular fluid, or GCF. Extracellular polymetric substances, or EPS. Behcet's disease, or BD. Tumor necrosis factor alpha, abbreviated TNF-α. Oral potential malignant diseases, or OPMD. Oral lichen planus, or OLP. Oral leukoplakia, or OLK. gastroesophageal reflux disease, or GERD. reflux esophagitis, or RE. EoE stands for eosinophilic reflux disease. Barrett's esophagus (BE). Nasopharyngeal squamous cell carcinoma, or ESCC. Esophageal adenocarcinoma, or EAC. Functional dyspepsia, or FD. intestinal metaplasia, or IM. Helicobacter pylori, or HP. GC stands for stomach cancer. Colorectal cancer, or CRC.

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