Research Article

pISSN: 2345-5357
eISSN: 2423-4214

OPEN ACCESS

APPLIED FOOD BIOTECHNOLOGY, 2022, 9 (3):195-204
Journal homepage: www.journals.sbmu.ac.ir/afb

Relationships between the Salivary Microbial Composition and Gastrointestinal Diseases

Asal Noruzpour [1,2], Hossein Dabiri[1], Fahimeh Sadat Gholam-Mostafaei[2], Mohammad Rostami Nejad[3*]

1- Department of Microbiology, School of Medicine, Shahid Beheshti University of Medial Science, Tehran, Iran

2- Basic and Molecular Epidemiology of Gastrointestinal Disorders Research Center, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran

3-Gastroenterology and Liver Diseases Research Center, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences

Abstract

Background and Objective: Salivary microbiota, including bacteria shed from oral surfaces, is individualized, temporally stable and affected by the environmental factors such as diet, lifestyle and oral hygiene. Salivary microbiota is nonpathogenic and commensal; however, if microbiota colonizes into the intestines, they could alter gastrointestinal microbiota and result in development of several chronic inflammatory diseases, foremost autoimmune diseases and gastrointestinal cancers. There are few studies that assessed salivary microbiota in autoimmune gastrointestinal disorders. This may help researchers find novel personalized therapeutic approaches for the gastrointestinal diseases. Therefore, the aim of this review was to discuss alterations of salivary microbiota composition in gastrointestinal disease progress, including celiac disease, inflammatory bowel disease and gastrointestinal cancers.

Results and Conclusion: This study suggested that oral microbiota composition is linked to chronic inflammatory diseases by changing the immune system responses through increasing the production of inflammatory cytokines and mediators. Investigation of saliva microbiota is becoming an important part of diagnosing gastrointestinal diseases and changes in the composition of oral microbiota can predict risks of disorder progression in high-risk individuals.

Conflict of interest: The authors declare no conflict of interest.

Article Information

Article history:
-Received 31 Jan 2022
-Revised 13 Mar 2022
-Accepted 9 May 2022

Keywords:
- Autoimmune diseases
- Gastrointestinal cancers
- Gastrointestinal disorders
- Salivary microbiota

***Corresponding author:**

Mohammad Rostami Nejad*****
Gastroenterology and Liver Diseases Research Center, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences

Tel: +98-21-22432518
Fax: +98-21-22432517
E-mail:
m.rostamii@gmail.com

How to cite this article

Noruzpour A, Dabiri H, Gholam-Mostafaei FS, Rostami Nejad M. Relationships between the Salivary Microbial Composition and Gastrointestinal Diseases. Appl Food Biotechnol. 2022; 9 (3): 195-204. http://dx.doi.org/10.22037/afb.v9i3.37524

1. Introduction

Human microbiota in composed of ecological, commensal, symbiotic and pathogenic microorganisms, including bacteria, archaea, Protista, fungi and viruses in the human body [1]. Oral microbiota is one of the most diverse populations in the human body [2]. After gut with more than 700 species, the oral cavity includes one of the most diverse and unique communities of microbes [3-5]. Microbiota is a normal part of the oral cavity and vital function. It can protect the body against colonization of extrinsic bacteria, affecting systemic health and maintaining health [6]. Only 57% of this microbial community are officially named and nearly 43% of these species are either unowned or uncultivated. [7]. Although microbiota composition varies from person to person based on the host genetics and environmental factors such as lifestyle, diet and personal oral hygiene [8]; however. Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria and Fusobacteria are the most common bacteria found in the oral cavity [9,10]. It is not surprising if individuals' health statuses are claimed to closely depend on oral and gut microbiota, as the vital interactions between these micro-organisms and the immune system guarantee appropriate stimulation of innate and adaptive immune responses

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

[11,12]. The best example that can verify this claim includes correlations between the population of oral microbiota and occurrence of autoimmune diseases (Table 1). It has been described that removing oral microorganisms can induce immune imbalance and thereby increase risks of autoimmune diseases [13]. Individuals' genetic reservoirs can be affected by the environmental factors. It has also been reported that society industrialization may diminish the oral bacterial population, exposing the immune system to neo-antigens. This is the reason why the risk of autoimmune diseases is rising in industrial societies [14]. Although this is still controversial, oral bacteria are accused as developers of systematic diseases (Figure 1). For example, correlations between the increased levels of *Neisseria* spp. and Bacteroides and decreased levels of *Rothia* spp., *Streptococcus* spp. And *Prevotella* spp. have been reported in patients with celiac disease (CD) [15-17]. Few studies have been published on salivary microbiota and autoimmune gastrointestinal (GI) disorders. Therefore, the aim of the current review was to discuss importance of oral microbiota in development of chronic GI and systematic diseases, ranging from auto-immune diseases to human cancers.

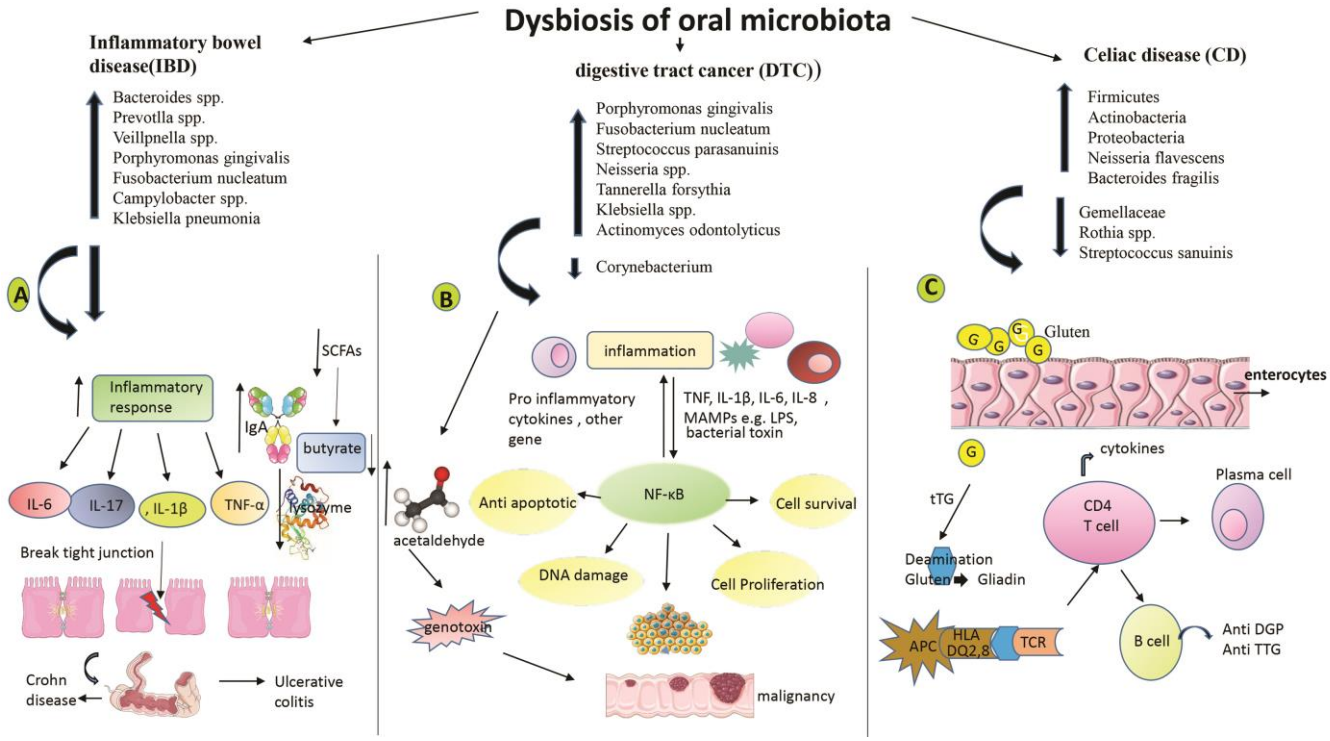
2. What is the Salivary Microbiota?

Although the major attention is focused on intestinal microbiota, it is noteworthy that oral cavity microbiota is the second most diverse bacterial community in the body [18]. However, it is not surprising as many microorganisms enter the mouth via foods and air flows. After microbiota are transferred to other organs, a group of the microbiota reside into the oral cavity, constituting the oral or salivary microbiota (SM). Even though saliva should be sterile when released, it includes a significant number of microorganisms [19].

Table 1. Correlations between the population of oral microbiota and occurrence of autoimmune diseases

Disease	First Author	Article title	Year of published	method	country	Age Group	Result of frequency
Celiac	Ruggiero Francavilla	Salivary Microbiota and Metabolome Associated with Celiac Disease[15]	2014	pyrosequencing 16S rRNA	Italy	children	Increase in T-CD children *Bacteroidetes*, *Porphyromonas* sp., *Porphyromonas endodontalis* and *Prevotella nanceiensis*. Decrease in T-CD children *Actinobacteria*. *Streptococcus thermophilus*
Celiac	Simona Panelli	Comparative Study of Salivary, Duodenal and Fecal Microbiota Composition Across Adult Celiac Disease[73]	2020	PCR	Italy	adult	Increased *Neisseria*, in active CD patients
IBD	YingQi	High-throughput sequencing provides insights into oral microbiota dysbiosis in association with inflammatory bowel disease[59]	2021	16S rRNA	Japan	adult	significantly increased in CD patients *Saccharibacteria* (TM7), *Absconditabacteria* (SR1), *Leptotrichia*, *Prevotella*, *Bulleidia* and *Atopobium*
IBD	Heba S.Said	Dysbiosis of salivary microbiota in inflammatory bowel disease and its association with oral immunological biomarkers[58].	2014	NGS	Japan	adult	Increase of *Prevotella*, decrease of *Neisseria* and *Haemophilus* in IBD.
Intestinal cancer	Shinya Kageyama	Characteristics of the Salivary Microbiota in Patients With Various Digestive Tract Cancers[65].	2019	16S rRNA	Japan	adult	*Porphyromonas gingivalis* was more abundant in the saliva of all groups of DTC patients. *Corynebacterium* species and *Neisseria* species was more abundant gastric cancer patients. *Fusobacterium nucleatum*, *Streptococcus parasanguinis* II and *Neisseria* species were significantly higher in tongue/pharyngeal cancer patients *Actinomyces odontolyticus* was significantly higher in CRC
Intestinal cancer	Kun Huang	Salivary Microbiota for Gastric Cancer Prediction: An Exploratory Study[74].	2021	16S rRNA. PCR	China	adult	*Haemophilus*, *Neisseria*, *Parvimonas*, *Peptostreptococcus*, *Porphyromonas and Prevotella*, *Haemophilus*, increased in gastric cancer

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.



Legend for figure 1. A) Dysbiosis of oral microbiota lead to increased inflammatory response of the host immune cells such as; IL-6, IL-17, IL-1β, TNF-α and increasing IgA and decreased lysozyme enzyme and short-chain fatty acids for examples; butyrate, disrupted gut homeostasis, impaired mucosal barrier and tight junction finally triggered the induction or progression of IBD. B) Dysbiosis of inhabitant microbes in oral cavity linking with gut microbiota can stimulation of chronic inflammation of host, this process by MAMPs e.g. LPS and other bacterial toxins activated to induce the expression of pro inflammatory cytokines such as; TNF, IL-1β, IL-6, IL-8, chemokines and other genes lead to activation of the NF-κB signaling pathway as a central regulator of host responses can be activated following DNA damage activated NF-κB also affects cellular proliferation and apoptosis and contribute to digestive tract cancer. C) Some gluten peptides cross the intestinal epithelium and can be deamidated by the tissue transglutaminase (tTG), in adition dysbiosis of oral microbiota and following them gut microbiome, which increases their ability to bind the HLA-DQ2/8 molecules of APC and triggered an adaptive and native immune response and provoked inflammatory cascade and the production of CD antibodies. Immune response by interacting with epithelial cells immunogenic peptides from gluten and by mediating host-microbe interactions which could influence to the mucous membrane of the intestinal and lead to damage them. Tumor Necrosis Factor Alpha: TNF-α, Interleukins: IL, Immunoglobulin A: IgA, Microbe-associated molecular pattern: MAMPs, Lipopolysaccharide: LPS, nuclear factor kappa-light-chain-enhancer of activated B cells: NF-κB, deamidated gliadin peptide: DGP, Tissue Transglutaminase: TTG, Antigen-presenting cell: APC, T-cell receptor: TCR, Human Leukocyte Antigen: HLA, short-chain fatty acids: short-chain fatty acids.

The SM composition is unique. A part of the microorganisms in SM is almost similar in every population [20]. *Streptococcus* spp., *Streptococcus mutans*, *Porphyromonas* (*P.*) *gingivalis* and *Lactobacillus* spp. are the most detected bacteria in the oral cavity [21]. The *F* is the major bacterium of the oral microbiota [22] and it is one of the major pathogens responsible for dental caries [23]. The *P. gingivalis* is another common, Gram-negative anaerobic bacteria in the oral cavity that (if untreated) can cause teeth fall [24]. *Lactobacillus* spp. is another group of bacteria present in the oral cavity. By converting sugar to lactic acid, they can increase risks of dental caries [24]. Although these bacteria can be detected in the saliva of every individual, populations of microorganisms can be unique in individuals, formed as a result of the environmental factors, diets [25,26] and health statuses [27,28,29]. For example, it has been described that climate change can affect the composition of SM [30]. Furthermore, it has been reported that the long residence in the Antarctic can change the composition of SM [31]. Even at the early stage of life, SM composition is affected by the mode of delivery, breastfeeding length and antibiotic treatment, as the population of microorganisms in the oral cavity can differ between dentate and edentulous individuals [32]. Moreover, it has been reported that full-mouth extraction could change the SM population [33]. Oral cavity diseases such as periodontitis, gingivitis and dental caries can change the population of microbiota, suggesting that assessment of SM population can reflect the health status of the individuals [34]. Relatively, it has been reported that the identification of *Prevotella* spp. and *Veillonella* spp. in

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

the saliva microbiota reveals poor oral health, high body mass index (BMI) and old age [35]. In contrast, predominance of *Neisseria* spp. in saliva indicates healthy periodontal conditions. Although SM composition can partially be affected by external factors, it is noteworthy that population of SM does not change conveniently unlike fecal microbial community, which is highly vulnerable to antibiotic exposure and short-term hospitalization [36]. Due to the easy access of the oral cavity to other organs such as respiratory tract and gastrointestinal tract (GIT), the oral microbiota can enter other organs. Since these microbiota members are not communal, they can cause systemic diseases [7]. The gnotobiotics include conditions of life; in which, well-known types of organisms are present [37]. These conditions have often been purported to enable animals to enjoy improved physiological health, even leading to increased life spans with absence of microorganisms, and improving quantity and quality of the peoples' lives [38,39]. Nevertheless, unexpected consequences may occur due to the gnotobiotics, including enlarged cecum and decreased organ sizes of heart, lungs and liver lymph nodes [40]. This status may include neglected consequences such as decreases in serum immunoglobulins and leukocytes and affections on mental health [41]. This illegal transportation can cause several inflammatory diseases as well, in particular inflammatory bowel disease (IBD) and digestive tract cancer (DTC) [1]. In the following section, it has been discussed how salivary microbiota may be linked to these inflammatory diseases.

3. Interactions or Relationships between Foods and Microbiota

The oral cavity is the first part of GIT, which mechanically breaks down foods [42]. Saliva mixed the foods, resident microflora, enzymes and metabolites by digestive juices begin the process of digestion [43]. Another critical role of the oral cavity includes maintaining of oral homeostasis and colonization of microflora. In another word, it is a microbial gatekeeper and primary contributor to the intestinal microbiota. A few studies investigated associations between eating habits and saliva microbiota. A study by Collado et al. in 2018 demonstrated that changes in meal timing included lethal effects on a diversity of salivary microbiota. Dietary and consumption of foods can increase pro-inflammatory cytokines and risks of intestinal inflammatory-linked diseases [44]. Furthermore, healthy diets and lifestyle are essential parts of an individual's health. Good nutrients in diets promote health and decrease risks of GI diseases. Diets have shaped and changed microbial compositions during human evolution and the microbial community affects development of intestinal immunity and health. In mice models, studies have shown that cause of 57% of changes in the gut microbiota is diet, whereas host genes account for nearly 12% of changes [45].

Roles of diets on intestinal microbial homeostasis are complex because of interactions between the intestinal epithelia, immunity and gut microbial composition of the hosts. Imbalanced diets can change intestinal microbiota and dysregulate host immunity, becoming susceptible to inflammation and intestinal disorders such as CD and IBD. Although, diets affect microbiome and metabolome in GIT, triggering several metabolites and GI disorders. A few studies have assessed relationships between the saliva microbial composition and inflammatory diseases. Studies on associations between eating habits and saliva microbiota in 842 adolescents showed that eating breakfast was associated to increased saliva microbiota diversity. In another study on the timing of food intakes and relationships of salivary microbiota demonstration, significant daily rhythms were reported in diversity and relative abundance of the bacteria (e.g., TM7 and *Fusobacteria*) of salivary microbiota in early and late eating meals [46,47].

4. Salivary Microbiota and the Autoimmune Gastrointestinal Diseases

4.1. Salivary Microbiota and Celiac Disease

As a well-known multifactorial autoimmune disease targeting the small intestine, CD is developed when the immune cells react to gluten and propagate intensive inflammatory responses in the lamina propria and the epithelia of the small intestine [48,49]. Clinical manifestations of CD vary from asymptomatic to multiple organ involvement. Gluten-free diets seem as the most effective strategy for curing the disease, while it is truly hard to follow the whole life. Although genetic factors contribute to CD susceptibility and HLA DQ2 and DQ8 haplotypes have been detected in 40% of the patients, several factors such as timing of gluten administration in infancy and breastfeeding can trigger onsets of the disease [50]. Breastfeeding was associated to overall phylum distribution. A major part of the maturation of oral microbiome occurs during the first two years of life. Based on a previous study, this development of oral microbiota increased relative abundances of *Porphyromonas* spp. and *Fusobacterium* with increased age after six months of life. Subsequent decreases of *Firmicutes* (largely attributed to members of streptococci) were accompanied by increases in other phyla, including *Proteobacteria* [5].

In-depth analyses revealed footprints of aberrant SM composition in development of CD, as it was verified that these bacterial taxa could stimulate local and systemic immune responses in the small intestine of CD patients [51]. For example, it has been reported that increases in the number of salivary bacteria such as *Gemella* spp. and *Porphyromonas* spp. together and diminishes in the number of aerobic bacteria can increase risks of CD in susceptible individuals [16,52]. Another study demonstrated that

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

decreases in Actinobacteria population and hence increases in proportion of Firmicutes and Proteobacteria are associated to the higher possibility of developing CD in infants. Numbers of Gemellaceae, Lachnospiraceae and *S. sanguinis* showed higher frequencies in the saliva of children with CD treated with gluten-free diets, compared to healthy individuals. Iaffaldano et al. reported that *Proteobacteria* and *Neisseria* were the most abundant phyla and genera in various types of saliva bacteria in CD patients [53]. Later, another study on 18 healthy individuals, 11 active CD patients and 16 gluten-free diet patients showed that the population of *Neisseria* spp., especially *Neisseria flavescens*, was higher in the oropharyngeal part of active CD patients, compared to healthy individuals or inactive CD patients. *In vitro* investigation showed that *Neisseria flavescens* could stimulate inflammatory responses and disturb mitochondrial respiratory chain, leading to metabolic imbalances in the endothelial cells. This species of *Neisseria* could recruit inflammatory dendritic cells to the duodenum mucosa, potentiating pathogenesis of CD (42). The *Bacteroides fragilis* is another saliva commensal bacterium that affects CD development. The produced polysaccharide A by this bacteria provokes $CD4^+$ T-lymphocytes to produce T-helper 1 lineage, a lymphocyte responsible for producing inflammatory cytokines in lamina propria [54]. Of these bacteria, species of *Rothia* might be further involved in in development of CD. *Rothia* species of *Rothia mucilaginosa* and *Rothia aeria* are a group of bacteria that produce glutamine endoprotease in saliva to digest gluten and proline. In-depth analysis results also suggested that glutaminase produced by *Rothia* spp. could target the immunogenic epitope of gluten, which is indeed responsible for CD development [13]. Generally, *Rothia*-produced glutaminase, activated at pH 4-10, cleaves the protein at XPQQ and LPYQ sites. A study by Zamakhchari et al [16] showed that decreasing the number of *Rothia* spp. in oral cavity could increase risks of CD in genetically susceptible individuals. Therefore, these species of bacteria could be recruited for therapeutic perspectives as their produced glutaminase could decrease immunogenicity of gluten in CD patients. These findings suggested that CD could be classified as a dysbiosis disease and thereby treatment of this disease with use of gluten-free diets would be possible if population of some bacterial species could be restored into the oral cavity.

4.2. Salivary Microbiota and Inflammatory Bowel Disease

In fact, IBD is the most common intestinal inflammatory disorder worldwide, divided into ulcerative colitis and Crohn's disease (CD). This chronic inflammatory disease includes alternative phases of remission and recurrence; however, the precise mechanisms leading to its pathogenesis have not been elucidated. Genetic susceptibility, environmental factors and disruption of immune homeostasis are possible drivers of IBD [55-57]. Recently, evidence showed that dysbiosis in oral and gut microbiota could exacerbate inflammatory responses in IBD [58,59]. Indeed, it has been reported that when opportunistic pathogens replaced the oral commensal bacteria, they could provide inflammatory backgrounds, which may harm intestine endothelial barrier. The *Streptococcus mutans*, *F. nucleatum*, *Campylobacter consensus* and *Klebsiella pneumoniae* are the most identified oral bacteria that could induce host immune system against the intestine endothelial cells via secretion of inflammatory cytokines. In a study on 35 IBD patients and 24 healthy volunteers, pyrosequencing of the bacterial 16S rRNA gene showed increases in the number of *Bacteroidetes* and decreases in the number of *Proteobacteria* in the saliva of IBD patients, compared to healthy people. Moreover, authors suggested positive correlations between *Streptococcus* spp., *Prevotella* spp. *Neisseria* spp., *Haemophilus* spp., *Veillonella* spp. and *Gemella* spp. of saliva and occurrence of IBD. It seems that production of inflammatory cytokines such as IL-1β, increases levels of immunoglobulin A, IL-6 and IL-8 and decreases in lysozymes and short-chain fatty acids (SCFA), which decrease butyrate levels, the SM bacteria can disturb the host immune system to degrees that it may lead to the development of IBD [58]. In another study, it was shown that ectopic colonization of *P. gingivalis* could be associated to IBD development or progression [60]. Results of further investigations showed that colitis alone could change the composition of SM, creating a vicious cycle that exacerbates conditions for IBD patients [60]. It is noteworthy that while the buccal microbiome seems affected by colitis (1.8%) hardly, composition of saliva could be altered by colitis up to 7.2%. These findings have demonstrated that dysbiosis in murine models of colitis is associated to changes in the composition of bacteria in oral cavity and saliva [60,61]. Overall, it seems that focusing on SM not only could revolutionize the current understanding of the pathogenesis of IBD, but also it could provide novel and valid biomarkers for rapid diagnosis of the disease.

4.3. Salivary Microbiota and Human Gastrointestinal Tract Cancers

It seems that alterations in the patterns of oral microbiota can lead to the development of malignancies in GIT [13], known as DTC and even extra GIT cancers such as oral squamous cell carcinoma, salivary gland tumors and cancers of the lymphatic tissues [2]. It has been reported that oral bacteria could locally activate carcinogens of tobacco (nitrosamines) and alcohol and thereby potentiating their carcinogenic activities [62]. It has also been reported that oral bacteria could convert less toxic ethanol to the genotoxic compound of acetaldehyde [63]. Its oncogenic effects have been well-established in *in vitro* and *in vivo* analyses [64]. Identification roles of oral microbiota in development of

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

several GI cancers suggest that possibly studying of salivary microbiota could shed further light on the etiology of various human cancers.

4.3.1 Salivary Microbiota and Digestive Tract Cancers

Results of the previous studies revealed that patients with DTC had more salivary bacteria than that their healthy individuals had. Moreover, it has been documented that of various operational taxonomic units, *P. gingivalis* includes the highest frequency in DTC patients. *Corynebacterium* spp. can abundantly be detected in all types of DTCs, except gastric cancer, which seems to include a small population. Increases in the population of operational taxonomic units corresponding to *F. nucleatum*, *S. parasanguinis* II and *Neisseria* spp. seem associated to tongue/pharyngeal cancer. *Neisseria* spp. and *Actinomyces odontolyticus* are abundant in gastric and colorectal cancers, respectively [65]. Periodontal disease-associated bacteria are suggested to increase risks of esophageal cancer. Based on a case-control study published by Peters et al. (2017), higher levels of *Tannella forsythia* and *P. gingivalis*, two important pathogens in gum diseases, were associated to increased risks of esophageal cancer and esophageal squamous cell carcinoma (ESCC), respectively [66]. A study by Ahn et al. on 122,000 participants reported that 106 individuals, who had periodontal pathogens in their saliva, developed esophageal cancer within the next ten years [66,67].

In another international cancer study, *Neisseria* spp. identified from the human oral cavity included an extremely high ADH activity within the bacterial species and produced acetaldehyde in culture media containing ethanol. This ability was more than 100-fold higher, compared to other genera in this study. In addition, ingestion of alcohol affected bacterial composition of the oral microflora, resulting in increased proportions of *Neisseria* spp. Although this genus is generally non-pathogenic oral microflora, this study suggested that *Neisseria* spp. could be sources of carcinogenic acetaldehyde in this region and possibly played essential roles in alcohol-linked carcinogenesis of the human upper GIT [68]. These findings clear tumorigenesis processed of GIT with the perspective of importance of the salivary microbiota [65]. How these oral bacteria could develop carcinogenesis? The answer to this question is still debatable and no effective mechanisms have been suggested for this association. However, there are suggestions. As previously stated, increases in *Neisseria* spp. in the oral cavity are associated to higher risks of tongue/pharyngeal, gastric, and colorectal cancers. It is suggested as an ability of *Neisseria* spp. to produce acetaldehyde from ethanol that may develop carcinogenesis [65]. Salaspuro et al. reported that increases in salivary acetaldehyde levels could reinforce risks of upper DTC [69]. Thus, it could be postulated that increases in the population of *Neisseria* spp. in the oral cavity could increase alcohol carcinogenic characteristics, thereby enhancing chances of DTC. It is noteworthy that the carcinogenic activity of *Neisseria* spp. has only been assessed *in vitro*. Therefore, behaviors of the bacteria might be different in *in vivo* analyses and at the presence of other bacterial communities [68]. Another mechanism suggested for the oral bacteria and their toxins in inducing carcinogenesis includes stimulation of the chronic immunologic responses [70]. The best example of this evidence includes the role of LPS (a lipopolysaccharide presented at the surface of Gram-negative bacteria) in propagation of human cancers. It has been suggested that LPS can increase activity of nuclear factor (NF)-κB in the target cells. This can increase secretion of inflammatory cytokines and upregulate expression of anti-apoptotic proteins [71]. In pancreatic cancer, it has been reported that produced IL-1β due to the bacteria exposures can exacerbate progression of the malignancy [72]. This evidence suggests that monitoring microbiota of the oral cavity can provide valuable clues about risks of cancer progression in patients [66].

4. Conclusion

Oral microbiota, the second largest and the most diverse microbiota, plays vital roles in health and diseases of the hosts. Provided evidence in the present study support the importance of oral microbiota dysbiosis in formation of oral cavity-associated diseases and evolution of chronic inflammatory diseases by changing immune system responses through increases in production of inflammatory cytokines and mediators. Although bacteria dysbiosis seems to answer questions about the mystery of autoimmune diseases (e.g., IBD and CD) and human cancers, further analyses are needed to well describe correlations between the salivary microbiota and occurrence of diseases. The current study has suggested that alterations in oral microbiota compositions by next generation probiotics can further modulate GI diseases, including GI immune-associated diseases and cancers.

5. Acknowledgements

This study was supported by a grant from Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

6. Conflict of Interest

The authors report no conflicts of interest.

References

1. Zapata HJ, Quagliarello VJ. The microbiota and microbiome in aging: Potential implications in health and age-related diseases. J Am Geriatr Soc. 2015; 63(4): 776-781. https://doi.org/10.1111/jgs.13310

2. Tuominen H, Rautava J. Oral microbiota and cancer development. Pathobiol. 2021; 88(2): 116-126. https://doi.org/10.1159/000510979

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0). To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

3. Lamont RJ, Koo H, Hajishengallis G. The oral microbiota: dynamic communities and host interactions. Nat Rev Microbiol. 2018; 16(12): 745-759.
https://doi.org/10.1038/s41579-018-0089-x

4. Huttenhower C, Gevers D, Knight R, Abubucker S, Badger JH, Chinwalla AT, Creasy HH, Earl AM, FitzGerald MG, Fulton RS, Giglio MG. Structure, function and diversity of the healthy human microbiome. Nature 2012; 486(7402): 207.
https://doi.org/10.1038/nature11234

5. Kennedy B, Peura S, Hammar U, Vicenzi S, Hedman A, Almqvist C andolf E, Pershagen G, Dicksved J, Bertilsson S, Fal lT. Oral microbiota development in early childhood. Sci rep. 2019; 9(1): 1-14.
https://doi.org/10.1038/s41598-019-54702-0

6. Wade WG. The oral microbiome in health and disease. Pharmacol Res. 2013; 69(1): 137-143.
https://doi.org/10.1016/j.phrs.2012.11.006

7. Willis JR, Gabaldon T. The human oral microbiome in health and disease: From sequences to ecosystems. Microorganisms 2020; 8(2): 308
https://doi.org/10.3390/microorganisms8020308

8. Rinninella E, Raoul P, Cintoni M, Franceschi F, Miggiano GAD, Gasbarrini A, Mele MC. What is the healthy gut microbiota composition? A changing ecosystem across age, environment, diet and diseases. Microorganisms 2019; 7(1): 14.
https://doi.org/10.3390/microorganisms7010014

9. Sampaio-Maia B, Caldas I, Pereira M, Perez-Mongiovi D, Araujo R. The oral microbiome in health and its implication in oral and systemic diseases. Adv Appl Microbiol. 2016; 171-210.
https://doi.org/10.1016/bs.aambs.2016.08.002

10. Kamada N, Seo SU, Chen GY, Nunez G. Role of the gut microbiota in immunity and inflammatory disease. Nat Rev Immunol. 2013; 13(5): 321-335.
https://doi.org/10.1038/nri3430

11. Petrof E, Claud E, Gloor G, Allen-Vercoe E. Microbial ecosystems therapeutics: A new paradigm in medicine? Benef Microbes. 2013; 4(1): 53-65.
https://doi.org/10.3920/BM2012.0039

12. Piombino P, Genovese A, Esposito S, Moio L, Cutolo PP, Chambery A, Severino V, Moneta E , Smith DP, Owens SM, Gilbert JA, Ercolini D. Saliva from obese individuals suppresses the release of aroma compounds from wine. Plos one 2014; 9(1):1-11.
https://doi.org/10.1371/journal.pone.0085611

13. De Luca F, Shoenfeld Y. The microbiome in autoimmune diseases. Clin Experiment Immunol. 2019; 195(1): 74-85.
https://doi.org/10.1111/cei.13158

14. Chu E, Karr J. Environmental impact: Concept, consequences, measurement. Reference Module in Life Sciences. 2017.
https://doi.org/10.1016/B978-0-12-809633-8.02380-3

15. Francavilla R,D, Piccolo M, Vannini L, Siragusa S, De Filippis F, De Pasquale I, Di Cagno R, Di Toma M, Gozzi G, I Serrazanetti D, De Angelis M, Gobbetti M. Salivary microbiota and metabolome associated with celiac disease. Appl Environ Microbiol. 2014; 80(11): 3416-3425.
https://doi.org/10.1128/AEM.00362-14

16. Zamakhchari M, Wei G, Dewhirst F, Lee J, Schuppan D, Oppenheim FG, Helmerhorst EJ. Identification of Rothia Bacteria as Gluten-Degrading Natural Colonizers of the Upper Gastro-Intestinal Tract. PLOS ONE 2011; 6(9): e24455.
http://doi.org/10.1371/journal.pone.0024455

17. Boullerne AI, Adami GR, Schwartz JL, Skias D, Maienschein-Cline M, Green SJ, Feinstein DL. Deep DNA metagenomic sequencing reveals oral microbiome divergence between monozygotic twins discordant for multiple sclerosis severity. J Neuroimmunol. 2020; 343: 577237.
https://doi.org/10.1016/j.jneuroim.2020.577237

18. Kilian M, Chapple I, Hannig M, Marsh P, Meuric V, Pedersen A, M S Tonetti , W G Wade , E Zaura. The oral microbiome-an update for oral healthcare professionals. Br Dent J. 2016; 221(10): 657-66
https://doi.org/10.1038/sj.bdj.2016.865

19. Hasan NA, Young BA, Minard-Smith AT, Saeed K, Li H, Heizer EM, McMillan NJ, Isom R, Abdullah, AS, Bornman DM, Faith SA, Choi SY, Dickens ML, Cebula TA, Colwell RR. Microbial community profiling of human saliva using shotgun metagenomic sequencing. Plos One 2014; 9(5):1-10.
https://doi.org/10.1371/journal.pone.0097699

20. Baker JL, Bor B, Agnello M, Shi W, He X. Ecology of the oral microbiome: Beyond bacteria. Trends microbiol. 2017; 25(5): 362-74.
https://doi.org/10.1016/j.tim.2016.12.012

21. Dzidic M, Collado MC, Abrahamsson T, Artacho A, Stensson M, Jenmalm MC, Mira M. Oral microbiome development during childhood: an ecological succession influenced by postnatal factors and associated with tooth decay. Isme J. 2018; 12(9): 2292-306.
https://doi.org/10.1038/s41396-018-0204-z

22. Gomez A, Espinoza JL, Harkins DM, Leong P, Saffery R, Bockmann M, Torralba M, Kuelbs C, Kodukula R, Inman J, Toby Hughes , Craig JM, Highlander SK, Jones MB, Dupont CL, Nelson KE. Host genetic control of the oral microbiome in health and disease. Cell host Microbe. 2017; 22(3): 269-278.
https://doi.org/10.1016/j.chom.2017.08.013

23. Hujoel PP, Hujoel MLA, Kotsakis GA. Personal oral hygiene and dental caries: A systematic review of randomised controlled trials. Gerodontol. 2018; 35(4): 282-289.
https://doi.org/10.1111/ger.12331

24. Tanner A, Kressirer CA, Faller LL. Understanding Caries From the Oral Microbiome Perspective. J California Dental Associat. 2016; 44(7): 437-446.

25. De Filippis F, Vannini L, La Storia A, Laghi L, Piombino P, Stellato G, Serrazanetti DI, Gozzi G, Turroni S, Ferrocino I, Lazzi C, Di Cagno R, Gobbetti M, Ercolini D. The same microbiota and a potentially discriminant metabolome in the saliva of omnivore, ovo-lacto-vegetarian and vegan individuals. Plos one 2014; 9(11): 1-9.
https://doi.org/10.1371/journal.pone.0112373

26. Hansen TH, Kern T, Bak EG, Kashani A, Allin KH, Nielsen T, Hansen T, Pedersen O. Impact of a vegan diet on the human salivary microbiota. Sci Rep. 2018; 8(1): 1-11.
https://doi.org/10.1038/s41598-018-24207-3

27. Hall MW, Singh N, Ng KF, Lam DK, Goldberg MB, Tenenbaum HC, Neufeld JD, Beiko RG, Senadheera DB. Inter-personal diversity and temporal dynamics of dental, tongue and salivary microbiota in the healthy oral cavity. NPJ Biofilms Microbiomes. 2017; 3(1): 1-7.
https://doi.org/10.1038/s41522-016-0011-0

28. Leake SL, Pagni M, Falquet L, Taroni F, Greub G. The salivary microbiome for differentiating individuals: Proof principle. Microbes Infect. 2016; 18(6): 399-405.
https://doi.org/10.1016/j.micinf.2016.03.011

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

29. Cameron SJ, Huws SA, Hegarty MJ, Smith DP, Mur LA. The human salivary microbiome exhibits temporal stability in bacterial diversity. FEMS Microbiol Ecol. 2015; 91(9): 1-9
https://doi.org/10.1093/femsec/fiv091

30. Li J, Quinque D, Horz H-P, Li M, Rzhetskaya M, Raff JA, Hayes MG, Stoneking M. Comparative analysis of the human saliva microbiome from different climate zones: Alaska, Germany and Africa. BMC Microbiol. 2014; 14(1): 316.
https://doi.org/10.1186/s12866-014-0316-1

31. Bhushan B, Yadav A, Singh S, Ganju L. Diversity and functional analysis of salivary microflora of Indian antarctic expeditionaries. J Oral Microbiol. 2019; 11(1): 1581513.
https://doi.org/10.1080/20002297.2019.1581513

32. Gazdeck RK, Fruscione SR, Adami GR, Zhou Y, Cooper LF, Schwartz JL. Diversity of the oral microbiome between dentate and edentulous individuals. Oral Dis. 2019; 25(3): 911-918.
https://doi.org/10.1111/odi.13039

33. de Waal YC, Winkel EG, Raangs GC, van der Vusse ML, Rossen JW, van Winkelhoff AJ. Changes in oral microflora after full-mouth tooth extraction: A prospective cohort study. J Clin Periodontol. 2014; 41(10): 981-989.
https://doi.org/10.1111/jcpe.12297

34. Mason MR, Chambers S, Dabdoub SM, Thikkurissy S, Kumar PS. Characterizing oral microbial communities across dentition states and colonization niches. Microbiome 2018; 6(1): 67.
https://doi.org/10.1186/s40168-018-0443-2

35. Yamashita Y, Takeshita T. The oral microbiome and human health. J Oral Sci. 2017; 59(2): 201-206.
https://doi.org/10.2334/josnusd.16-0856

36. Stahringer SS, Clemente JC, Corley RP, Hewitt J, Knights D, Walters WA, Knight R, Krauter KS. Nurture trumps nature in a longitudinal survey of salivary bacterial communities in twins from early adolescence to early adulthood. Genome Res. 2012; 22(11): 2146-52.
https://doi.org/10.1101/gr.140608.112

37. Luckey T. Germfree Life and Gnotobiology: 1st Edition, Elsevier; 2012.

38. MacDonald TH. Rethinking Health Promotion: A global Approach: Routledge; 2012.
https://doi.org/10.4324/9780203069875

39. Kirk RG. Life in a germ-free world: Isolating Life from the Laboratory animal to the bubble boy. Bull Hist Med. 2012: 86: 237-275
https://doi.org/10.1353/bhm.2012.0028

40. Stewart CL, Warner S, Ito K, Raoof M, Wu GX, Lu W-P, Kessler J, Kim JY, Fong Y. Cytoreduction for colorectal metastases: liver, lung, peritoneum, lymph nodes, bone, brain. When does it palliate, prolong survival and potentially cure?. Curr Probl Surg. 2018; 55(9): 330.
https://doi.org/ 10.1067/j.cpsurg.2018.08.004

41. Gilbert JA, Krajmalnik-Brown R, Porazinska DL, Weiss SJ, Knight R. Toward effective probiotics for autism and other neurodevelopmental disorders. Cell 2013; 155(7): 1446-1448.
https://doi.org/10.1016/j.cell.2013.11.035

42. Ogobuiro I, Gonzales J, Tuma F. Physiology, Gastrointestinal. StatPearls [Internet]. 2020.

43. De Haan P, Ianovska MA, Mathwig K, Van Lieshout GA, Triantis V, Bouwmeester H, Verpoorte E. Digestion-on-a-chip: A continuous-flow modular microsystem recreating enzymatic digestion in the gastrointestinal tract. Lab Chip. 2019; 19(9): 1599-609.
https://doi.org/10.1039/C8LC01080C

44. Collado MC, Engen PA, Bandín C, Cabrera-Rubio R, Voigt RM, Green SJ, Naqib A, Keshavarzian A, L Scheer FAJ, Garaulet M. Affiliations expand . Timing of food intake impacts daily rhythms of human salivary microbiota: A randomized, crossover study. FASEB J. 2018; 32(4): 2060-2072.
https://doi.org/10.1096/fj.201700697RR

45. Zhang C, Zhang M, Wang S, Han R, Cao Y, Hua W, Mao Y, Zhang X, Pang X, Wei C, Zhao GI. Interactions between gut microbiota, host genetics and diet relevant to development of metabolic syndromes in mice. ISME J. 2010; 4(2): 232-241.

46. Moles L, Otaegui D. The impact of diet on microbiota evolution and human health. Is diet an adequate tool for microbiota modulation? Nutrients 2020; 12(6): 1-19.
https://doi.org/10.3390/nu12061654

47. Thaiss CA, Levy M, Korem T, Dohnalova L, Shapiro H, Jaitin DA, David E, Winter DR, Gury-BenAri M, Tatirovsky E, Tuganbaev T. Microbiota diurnal rhythmicity programs host transcriptome oscillations. Cell. 2016; 167(6): 1495-510. e12.
https://doi.org/10.1016/j.cell.2016.11.003

48. Cenit MC, Olivares M, Codoner-Franch P, Sanz Y. Intestinal microbiota and celiac disease: cause, consequence or co-evolution?. Nutrients 2015; 7(8): 6900-6923.
https://doi.org/10.3390/nu7085314

49. ROSTAMI NM, Ishaq S, Al Dulaimi D, Zali MR, Rostami K. The role of infectious mediators and gut microbiome in the pathogenesis of celiac disease. 2015; 18(4): 244-249
https://doi.org/015184/AIM.0010

50. Gholam Mostafaei FS, Rostami-Nejad M, Emadi A, Yadegar A, Asadzadeh Aghdaei H, Zali MR. Changes in the composition and function of the gut microbiota in celiac disease. koomesh. 2021; 23(3):301-316.
http://dx.doi.org/10.52547/koomesh.23.3.301

51. Poddighe D, Kushugulova A. Salivary microbiome in pediatric and adult celiac disease. Frontiers Cell Infect Microbiol. 2021; 11: 14.
https://doi.org/10.3389/fcimb.2021.625162

52. Gholam-Mostafaei FS, Didari T, Ramandi M, Vafaee R, Rostami-Nejad M. Gut microbiota, angiotensin-converting enzyme, celiac disease and risk of COVID-19 infection: A review. Gastroenterol Hepatol Bed Bench. 2021; 14: 24-31.

53. Iaffaldano L, Granata I, Pagliuca C, Esposito MV, Casaburi G, Salerno G, Colicchio R, Piccirillo M, Ciacci C, Blanco GDV, Guarracino MR, Salvatore P, Salvatore FD, Argenio V, Sacchetti L. Oropharyngeal microbiome evaluation highlights Neisseria abundance in active celiac patients. Sci Rep. 2018; 8(1): 1-10.
https://doi.org/10.1038/s41598-018-29443-1

54. Troy EB, Kasper DL. Beneficial effects of Bacteroides fragilis polysaccharides on the immune system. Frontiers in bioscience: A journal and virtual library. 2010; 15: 25.

55. Pereira MS, Munerato MC. Oral manifestations of inflammatory bowel diseases: Two case reports. Clin Med Res. 2016; 14(1): 46-52.
https://doi.org/10.3121/cmr.2015.1307

56. Taghipour N, Molaei M, Mosaffa N, Rostami-Nejad M, Aghdaei HA, Anissian A, Azimzadeh P, Zali MR. An experimental model of colitis induced by dextran sulfate sodium from acute progresses to chronicity in C57BL/6: Correlation between conditions of mice and the environment. Gastroent Hepatol Bed Bench.2016; 9(1): 45.

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

57. Asadzadeh-Aghdaei H, Mashayekhi K, Koushki K, Azimzadeh P, Rostami-Nejad M, Amani D, Chaleshi V, Mohammadian Haftcheshmeh S, Sahebkar AH, Zali MR. V617F-independent upregulation of JAK2 gene expression in patients with inflammatory bowel disease. J Cell Biochem. 2019; 120(9): 15746-15755.
https://doi.org/10.1002/jcb.28844

58. Said HS, Suda W, Nakagome S, Chinen H, Oshima K, Kim S, Kimura R, Iraha A, Ishida H, Fujita J, Mano S, Morita H, Dohi T, Oota H, Hattori M. Dysbiosis of salivary microbiota in inflammatory bowel disease and its association with oral immunological biomarkers. DNA Res. 2014; 21(1): 15-25.
https://doi.org/10.1093/dnares/dst037

59. Qi Y, Wu H-m, Yang Z, Zhou Y-f, Jin L, Yang M-f, Wang f-Y. New insights into the role of oral microbiota dysbiosis in the pathogenesis of inflammatory bowel disease. Dig Dis Sci. 2021: 1-14.
https://doi.org/10.1007/s10620-021-06837-2

60. Rautava J, Pinnell LJ, Vong L, Akseer N, Assa A, Sherman PM. Oral microbiome composition changes in mouse models of colitis. J Gastroenterol Hepatol.2015; 30(3): 521-527.
https://doi.org/10.1111/jgh.12713

61. Naseri K, Dabiri H, Rostami-Nejad M, Yadegar A, Houri H, Olfatifar M, Sadeghi A, Saadati S, Ciacci C, Iovino P, Zali MR. Influence of low FODMAP-gluten free diet on gut microbiota alterations and symptom severity in Iranian patients with irritable bowel syndrome. BMC gastroenterol. 2021; 21(1): 1-14.

62. Sami A, Elimairi I, Stanton C, Ross RP, Ryan CA. The role of the microbiome in oral squamous cell carcinoma with insight into the microbiome-treatment axisInt. J Mol Sci. 2020; 21(21): 8061.
https://doi.org/10.3390/ijms21218061

63. Stornetta A, Guidolin V, Balbo S. Alcohol-derived acetaldehyde exposure in the oral cavity. Cancers 2018; 10(1): 20.
https://doi.org/10.3390/cancers10010020

64. Karpinski TM. Role of oral microbiota in cancer development. Microorganisms 2019; 7(1): 20.
https://doi.org/10.3390/microorganisms7010020

65. Kageyama S, Takeshita T, Takeuchi K, Asakawa M, Matsumi R, Furuta M, Shibata Y, Nagai K, Ikebe M, Morita M, Masuda M, Toh Y, Kiyohara Y, Ninomiya T, Yamashita Y. Characteristics of the salivary microbiota in patients with various digestive tract cancers. Front Microbiol. 2019; 10: 1780.
https://doi.org/10.3389/fmicb.2019.01780

66. Peters BA, Wu J, Pei Z, Yang L, Purdue MP, Freedman ND, Jacobs EJ, Gapstur SM, Hayes RB, Ahn J. Oral microbiome composition reflects prospective risk for esophageal cancers. Cancer Res. 2017; 77(23): 6777-6787.
https://doi.org/10.1158/0008-5472.CAN-17-1296

67. Javanmard A, Ashtari S, Sabet B, Davoodi SH, Rostami-Nejad M, Akbari ME, Niaz A, Mortazavian AM. Probiotics and their role in gastrointestinal cancers prevention and treatment; an overview. Gastroenterol Hepatol Bed Bench. 2018; 11(4): 284.

68. Yokoyama S, Takeuchi K, Shibata Y, Kageyama S, Matsumi R, Takeshita T, Yamashita Y. Characterization of oral microbiota and acetaldehyde production. J Oral Microbiol. 2018; 10(1): 1-8.
https://doi.org/10.1080/20002297.2018.1492316

69. Helminen A, Vakevainen S, Salaspuro M. ALDH2 genotype has no effect on salivary acetaldehyde without the presence of ethanol in the systemic circulation. Plos one 2013; 8(9): e74418.
https://doi.org/10.1371/journal.pone.0074418

70. Al-Hilu SA, Al-Shujairi WH. Dual role of bacteria in carcinoma: Stimulation and inhibition. Int J Microbiol. 2020; 8(9):1-15.
https://doi.org/10.3402/jom. v2i0.5195

71. Golchin S, Alimohammadi R, Rostami Nejad M, Jalali SA. Synergistic antitumor effect of anti-PD-L1 combined with oxaliplatin on a mouse tumor model. J Cell Physiol. 2019; 234(11): 19866-19874.
https://doi.org/10.1002/jcp.28585

72. Stasiewicz M, Kwasniewski M, Karpinski TM. Microbial associations with pancreatic cancer: A new frontier in biomarkers. Cancers 2021; 13(15): 3784.
https://doi.org/10.3390/cancers13153784

73. Panelli S, Capelli E, Lupo GFD, Schiepatti A, Betti E, Sauta E, Marini S, Bellazzi R, Vanoli A, Pasi A, Cacciatore R, Bacchi S, Balestra B, Pastoris O, Frulloni L, Corazza G R, Biagi F, Ciccocioppo R. Comparative study of salivary, duodenal and fecal microbiota composition across adult celiac disease. J Clin Med. 2020; 9(4): 1109.
https://doi.org/10.3390/jcm9041109

74. Huang K, Gao X, Wu L, Yan B, Wang Z, Zhang X, Peng L, Yu J, Sun G, Yang Y. Salivary microbiota for gastric cancer prediction: An exploratory study. Front Cell Infect Microbiol. 2021; 11: 1-10
https://doi.org/10.3389/fcimb.2021.640309

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0).
To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.

Research Article
pISSN: 2345-5357
eISSN: 2423-4214
OPEN ACCESS

APPLIED FOOD BIOTECHNOLOGY, 2022, 9 (3): 195-204
Journal homepage: www.journals.sbmu.ac.ir/afb

ارتباط ترکیب میکروبی بزاق و بیماری‌های گوارشی

عسل نوروزپور[۱و۲]، حسین دبیری[۱]، فهیمه سادات غلام مصطفایی[۲]، محمد رستمی نژاد[۳*]

۱- گروه میکروبیولوژی، دانشکده پزشکی، دانشگاه علوم پزشکی و خدمات بهداشتی درمانی شهید بهشتی، تهران، ایران

۲- مرکز تحقیقات اپیدمیولوژی پایه و مولکولی بیماری‌های گوارشی ، پژوهشکده بیماری‌های گوارش و کبد، دانشگاه علوم پزشکی و خدمات بهداشتی درمانی شهید بهشتی، تهران، ایران

۳- مرکز تحقیقات بیماری‌های گوارش و کبد، پژوهشکده بیماری‌های گوارش و کبد، دانشگاه علوم پزشکی و خدمات بهداشتی درمانی شهید بهشتی، تهران، ایران

چکیده

سابقه و هدف: میکروبیوتای بزاقی، شامل انواع باکتری های همزیست و پاتوژن موجود در سطوح دهان می باشد که این جمعیت باکتریایی، منحصربفرد و موقتا پایدار و تحت‌تاثیر عوامل محیطی مانند رژیم غذایی، شیوه زندگی و همچنین بهداشت دهان و دندان قرار می‌گیرد. میکروبیوتای بزاقی غیر بیماری زا و همزیست بوده و چنانچه این جمعیت میکروبی در روده کلونیزه شود، می‌تواند میکروبیوتای دستگاه گوارش را تغییر داده و منجر به ایجاد بسیاری از بیماری‌های التهابی مزمن، برخی از بیماری‌های خودایمنی و یا سرطان‌های دستگاه گوارش شود. مطالعات کمی میکروبیوتای بزاقی را اختلالات خود ایمنی دستگاه گوارش ارزیابی کرده است و این ممکن است به یافتن رویکردهای درمانی شخصی جدید برای بیماری های دستگاه گوارش کمک کند. بنابراین، هدف از این مطالعه مروری، بحث در مورد تغییر ترکیب میکروبیوتای بزاقی در پیشرفت بیماری های گوارشی مانند بیماری سلیاک، بیماری التهابی روده و چندین سرطان دستگاه گوارش بود.

یافته‌ها و نتیجه‌گیری: این مطالعه نشان داد که ترکیب میکروبیوتای دهان با تغییر پاسخ سیستم ایمنی از طریق افزایش تولید سیتوکین‌ها واسطه‌های التهابی، با بیماری‌های التهابی مزمن مرتبط است. بررسی میکروبیوتای بزاق در حال تبدیل شدن به بخش مهمی از تشخیص بیماری های دستگاه گوارش است و هرگونه تغییر در ترکیب میکروبیوتای دهان می تواند پیش بینی کننده خطر پیشرفت اختلال در افراد پرخطر باشد.

تعارض منافع: نویسندگان اعلام می‌کنند که هیچ نوع تعارض منافعی مرتبط با انتشار این مقاله ندارند.

تاریخچه مقاله

دریافت ۳۱ ژانویه ۲۰۲۲
داوری ۱۳ مارس ۲۰۲۲
پذیرش ۹ می ۲۰۲۲

واژگان کلیدی

- بیماری‌های خود ایمنی
- سرطان‌های دستگاه گوارش
- اختلالات گوارشی
- میکروبیوتای بزاق

***نویسنده مسئول**

محمد رستمی نژاد

مرکز تحقیقات بیماری‌های گوارش و کبد، پژوهشکده بیماری‌های گوارش و کبد، دانشگاه علوم پزشکی و خدمات بهداشتی درمانی شهید بهشتی، تهران، ایران

تلفن: ۰۲۱-۲۲۴۳۲۵۱۸+۹۸

فکس: ۰۲۱-۲۲۴۳۲۵۱۷+۹۸

پست الکترونیک:
m.rostamii@gmail.com

This open-access article distributed under the terms of the Creative Commons Attribution NonCommercial 4.0 License (CC BY-NC 4.0). To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/.