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Archives of Dental Research

Journal homepage: https://www.adr.org.in/

Review Article

Role of oral microbiome in oral cancer: A review

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ARTICLE INFO

Article history:
Received 25-06-2022
Accepted 09-07-2022
Available online 28-07-2022

Keywords:
Oral malignant growth
Bacteria
Microbiota
Oral microbiome

ABSTRACT

Oral microbiota is among the most assorted in the human body. In excess of 700 species have been distinguished in the mouth, and new sequencing techniques are permitting us to find significantly more species. The life structures of the oral cavity is not the same as that of other body locales. The oral cavity has mucosal surfaces (the tongue, the buccal mucosa, the gingiva, and the palate), hard tissues (the teeth), and exocrine organ tissue (major and minor salivary organs), all of which present novel elements for microbiota organization. Oral squamous cell carcinoma is quite possibly the most well-known danger and is the main source of dismalness and mortality.

1. Introduction

Oral malignant growth is a multifactorial illness. Have hereditary qualities and ecological elements assume a part in the causation of this sickness. Tobacco, liquor utilization, betel quid biting and human papillomavirus (HPV) contaminations are notable risk factors. A potential etiology could be ascribed to the organisms in around 15% of oral disease patients without known risk factors.[1] The squamous epithelium of the oral cavity is persistently presented to an assortment of microbial difficulties, at both cell and molecular levels.[2] Bacterial dysbiosis in the adult oral cavity can cause, e.g., gum disease, periodontitis, dental caries (tooth decay), and endodontic abscesses. In any case, because of the nonstop transaction among microbiota and the human host's safe reaction, intense diseases in the oral cavity are somewhat uncommon thinking about the dense microbial colonization.[3–6] Oral squamous cell carcinoma (OSCC), an obtrusive epithelial neoplasm with various degree of differentiation, represents around 90% of oral cancer. It begins with the collection of hereditary changes and explicit hereditary varieties in oncogenes and suppressor genes.[7] The high-risk regions are the floor of the mouth and the ventrolateral tongue, while the low risk lie in the palatal mucosa and the tongue dorsum.[8]

2. Etiology

Tobacco smoke contains many poisonous synthetics. Ordinary smoking is known to build people's risk of OSCC and other cancers.[9] What's more, smoking straightforwardly influences oral mucosal sites and in this manner additionally the oral bacterial diversity. Smoking has been distinguished to lessen bacterial diversity (α variety), particularly in the buccal mucosa, and by changing the bacterial composition leaning toward R.mucilaginosa, Streptococcus salivarius, and S. mitis.[10,11] Besides, more elevated levels of Prevotella, Veillonella, and Leptotrichia have been seen in current smokers.[12] Then again, lower levels of F.nucleatum and Leptotrichiahave been distinguished in patients who smoke and have OLP[13]. Raised degrees of R.mucilaginosa, Veillonella,

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https://doi.org/10.18231/j.adr.2022.001

Streptococcus, and Leptotrichia have been associated with OSCC autonomously, without the presence of smoking.[12,13] Regardless, the degrees of S.mutans and Lactobacillushave been seen to be unaltered even with regular smoking.[14]

3. Location of Oral Microbial Habitat

Tongue, buccal mucosa, supragingival and subgingival surfaces of the teeth, soft and hard palates, and saliva of OC might address different biological specialties or habitats.[15] The warm and sodden climate and host-inferred supplements, like saliva proteins, gingival crevicular liquid (GCF), and glycoproteins, favor the development of microorganisms in the OC.[16]

Dominating Microbial Communities in Oral Cavity and Oropharyngeal Region[17]

Table 1:

Different Sites in Oral Cavity and Oropharyngeal Region	Bacterial Species
Tongue	Veillonella atypica
	Porphyromonas gingivalis
	Selenomonas subspecies
	Aggregatibacter actinomycetemcomitans
	Prevotella intermedia
	Capnocytophaga
	Enterococcus faecalis
	Eikenella corrodens
	Streptococcus pyogenes
Oropharynx	Streptococcus pneumoniae
	Haemophilus influenzae
	Haemophilus parainfluenzae
	Streptococcus mutans
	Streptococcus salivarius
	Streptococcus anginosus
Tooth Surface	S mutans
	Actinomyces
	Eubacterium
	Peptostreptococcus
	Streptococcus viridans
Tonsil	H influenzae
	Neisseria species
	Staphylococcus
Gingival crevice	Fusobacterium
	Prevotella
	Porphyromonas
	Streptococcus mitis
	Streptococcus sanguinis
	Propionibacterium acnes
	Leptotrichia buccalis
	Actinomyces odontolyticus
	Veillonella parvula
Dental Plaque	Actinomyces
	Rothia
	Microbacterium
	Mycobacterium
	Propionibacterium
	Corynebacterium
	Bifidobacterium

4. Role of Bacteria

Malignant growth has been depicted as a molecular disease of cell membrane glycoconjugates. Certain glycoconjugates are receptors for distinct microbes. Ongoing examinations support the studies that changes in the colonization of various disease cells are linked with noticable changes in cell surface receptors. It is currently distinguished that microscopic organisms tie to and colonize the mucosal surfaces in an exceptionally specific way by means of a "lock and key" instrument. There is a particular restricting between adhesins on microbes to reciprocal receptors on the mucosal surfaces of the host.[18] Periodontal pathogenic microbes have been related with a higher risk for OSCC and Fusobacterium, Peptostreptococcus, Filifactor, Parvimonas, Pseudomonas, Campylobacter, and Capnocytophaga were accounted for high overflow in OSCC patients. Also, proinflammatory substances discharged by periodontal pathogenic microbes, for example, lipopolysaccharide (LPS) were advanced in malignant growth samples.[19]

Microorganisms might induce carcinogenesis by the accompanying mechanisms:[20]

1. Stimulation of chronic inflammation
2. Cell proliferation
3. Inhibition of cellular apoptosis
4. Promotion of cellular invasion
5. Production of carcinogenic substances[20]

5. Oral Micobiota and OSCC

Altogether more elevated levels of Peptostreptococcus, Fusobacterium, Prevotella (particularly P.melaninogenica), Porphyromonas, Veillonella (basically Veillonella parvula), Haemophilus, Rothia, and Streptococcushave been distinguished in OSCC tests.[21,22] OSCC can be isolated into various disease stages by the TNM (growth, node, metastasis) classification.[23] These TNM stages of OSCC have been seen in essentially unique oral bacterial microbiota composition. Porphyromonas gingivalis speeds up movement through the S-period of the cell cycle by forestalling action of p53 growth silencer gene.[24] Induced expression of B7-H1 and B7-DC receptors on OSCC cells and primary GECs by P gingivalis has been accounted before. expression of B7-H1 receptor restrains effector T cells through instigating administrative T cells. expression of B7-H1 receptor prompts immune evasion in oral cancers.[25] ERK1/2-Ets1, p38/HSP27, and PAR2/NF-KB pathways are actuated by P.gingivalis contamination to induce the expression of favorable to MMP-9. Gingipains

(cysteine proteinases) from P.gingivalis changes over supportive of MMP-9 into MMP-9 advances cell relocation intrusion and metastasis in OSCC.[26]

6. Conclusion

The Human body is a host to various microorganisms. These microorganisms alongside their hereditary material structure a critical part of the human body which is known as the microbiome. Adjustments in the creation of the ordinary microbiome are reffered to as dysbiosis which prompts sick states. Organisms have been related with oral sicknesses like dental caries, periodontal illnesses, and numerous others including oral disease. During the development of OSCC, the abudance of oral microbes changes and various microscopic organisms show distinct alterations. A few microbes show essentially high overflow in OSCC patients, while certain microscopic organisms make up a smaller proportion of the microorganisms in OSCC tissues and present a higher overflow in healthy examples. In view of the compositional changes of oral microorganisms in OSCC, a couple of mixes of various microscopic organisms have been viewed as markers for oral disease analysis.

7. Source of Funding

None.

8. Conflicts of Interest

None.

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Cite this article: Johny J, Sadiq A, Anamika, Sreedhar S, Fida A, Rishna A. Role of oral microbiome in oral cancer: A review. *Arch Dent Res* 2022;12(1):1-4.