

Role of *Selenomonas sputigena* Micro-Organism in Periodontal Diseases

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Abstract- *Selenomonas sputigena* progressively increase periodontal disease from simple gingivitis to severe periodontitis. Some specific bacterial species are considered to be agents that initiate and exacerbate human periodontal diseases. The role of the oral microbiota in the aetiology of periodontal diseases has been well established, and specificity may exist among certain bacterial species or groups and the various forms of periodontal disease. This review is done to discuss the nature and characteristics of *Selenomonas* species and to understand the etiopathogenesis of the periodontal disease.

Keywords- *Selenomonas sputigena*, bacteria, periodontal disease, co-aggregation, micro-organism

I. INTRODUCTION

Periodontal diseases are infections of the structures around the teeth which include the gingiva, cementum, periodontal ligament and alveolar bone. In the earliest stage, gingivitis was most common which affects the gingiva. It is now well accepted that various types of bacteria in dental plaque are the major causes to initiate the periodontal disease. In recent years, periodontal disease has been linked to a number of other health problems (1). The complexity and diversity of the periodontal microbiota has been confirmed by numerous studies (2). In recent studies it shows the group *Selenomonas* was the main focus to find out the periodontal disease in the oral microbiological research.

Description of bacteria:

Antonie van Leeuwenhoek documented *Selenomonas* species histologically in 1683. *seleno* means moon, and *musa* means banana. *Selenomonas* belongs to family *Spirillaceae* and it is a gram negative rods, obligately anaerobic, motile, non-spore forming bacteria. *S. sputigenum* was studied in pure culture by Dobell that the organism was transferred to the bacterial genus *Selenomonas* (3). Lessel and Breed countered, suggesting that the genus *Selenomonas* be recognized and that *Spirillum sputigenum* be listed as one of the three species of this genus, but that the latter's name be changed to *Selenomonas sputigena* (4).

Selenomonas has a characteristic crescent shape, with flagella inserted on the concave side (5). Robinow showed that the chromatin bodies of *Selenomonas palpitanis* are in close proximity to the flagellar tuft (6) [Figure no. 1; PC : ALAMY STOCK PHOTO -49162765]



In 1683, the features and movements of living crescent-shaped microorganisms from the human mouth were first described by Antonie van Leeuwenhoek. *Selenomastix* was named because the group was found to be protozoans in the earlier period.

The unique cell morphology of certain large selenomonads are bean shaped with bilateral symmetry along the long axis — an unusual property for prokaryotes [figure no. 2]. The large motile crescents found in the warm anaerobic nutrient-rich micro ecosystem provided by ruminant rumen, guinea-pig caecum (*S. palpitanis*) and even pockets in the human gingiva (*S. sputigena*) (5).

The large crescents have flagella which are quite differently inserted into the concave side of the cell from those of the smaller species of *Selenomonas*. Another interesting feature is the refractile body located beneath the massive flagella bundle characterising the large crescents. It is not related morphologically to the ciliate blepharoplast (6). A number of workers studied selenomonad growth, cell walls and their relationship to flagella (7).

II. CULTURE CHARACTERS

The most important prerequisite for culturing selenomonads is anaerobic technique both during the preparation of media and during the transfer of the organisms.

The common medium used are rumen fluid-glucose cellobiose agar (RGCA) medium and grown anaerobically in the presence of carbon dioxide at 37°C. The optimum temperature for growth is 30 to 37°C. There is moderate growth at 45°C and no growth or only very slight growth at 25°C (8).

S. sputigena grew well in MPB medium. The MPB medium was adopted for several reasons: (i) it was clear and almost colourless when reduced; (ii) with most strains this medium gave the highest yield of growth as judged by measurements of optical density; (iii) it was a reproducible, well-standardized medium; (iv) it was comparatively easily prepared. All selenomonads were grown in rubber-stoppered test tubes containing 10 ml of MPB broth.

III. COAGGREGATION PROPERTIES OF SELENEMONAS SPECIES

Fusobacterium nucleatum and *selenomonas sputigena* are two gram negative bacteria that act together and bring progressively increase periodontal status from simple gingivitis to severe periodontitis (9). *Selenomonas sputigena* doesn't coaggregates with other selenomonads rather mostly binds with *Fusobacterium nucleatum* to increase the destruction in periodontium. *Selenomonas* isolates coaggregates with other species like *actinomyces* (10), *bacteroids*, *Capnocytophaga*, *Haemophilus* and *Streptococcus* species. Ruminant selenomonads that were successively subcultured and grown in MPB medium. The rate of cell division seems to be highest and lost their motility during exponential phase. When a active growth is over during decline phase they regained their motility (11). *S. sputigena* when sectioned it showed fine bacterial structure with number of flagellum. This flagellum helps in further motility and helps in binding with other organisms.

IV. VIRULENT CHARACTER OF SELENEMONAS IN ORAL MUCOSA

Selenomonas sputigena was the most frequently detected bacterial species, whereas high levels of other species of *Selenomonas* were often also found. A high prevalence of *Selenomonas* species has also been observed in subgingival samples from subjects with chronic periodontitis. This species was found at significantly higher levels and with a significantly higher prevalence with generalized aggressive periodontitis more than in periodontally healthy subjects. *Selenomonas sputigena* from the periodontal pockets of patients with advanced destructive periodontal disease been identified-in patients as a dominant member of the microbial microbiota of the periodontal pocket (12). The production of

antibody with high titer in patients against *S. sputigena* (13) and the bone resorption. *Selenomonas sputigena* seen in germ-free rats (14) suggest that this bacterium is a periodontopathic agent in humans. It has been proposed that a surface component of bacteria is a virulent factor that causes tissue destruction in patients with periodontal disease. *Selenomonas sputigena* were detected in deep pocket than shallow pocket. High count of this pathogen in association with generalised aggressive periodontitis.

V. ROLE OF SELENEMONAS IN BIOFILM

The type strain is ATCC 43541, isolated from the gingival crevice of an affected site in a person with rapidly progressive periodontitis (15). This species is isolated from the gingival crevice of persons with gingivitis or periodontitis and from the adjacent supra gingival plaque coronal to the sites affected with periodontitis. This species has been referred to as *Selenomonas* D-4 and D-12 in previous publications (16).

The type strain is ATCC 43527, isolated from the supra gingival flora coronal to an affected site in a person with localised periodontitis. The species is isolated from the gingival sulcus of persons with moderate periodontitis and from the supra gingival flora coronal to the sites affected with moderate and localised periodontitis and has been referred to as *Selenomonas* D-10 as a species negatively correlated with gingivitis (17) and that does not increase in numbers in diseased sites as compared with healthy sites. The type strain is ATCC 43928, isolated from the subgingival crevice of a person with localised periodontitis. This species is isolated from the gingival crevice of persons with gingivitis and periodontitis and has been referred to as *Selenomonas* D-14 in previous publications.

VI. VIRULENT CHARACTER OF LIPOPOLYSACCHARIDE FROM SELENEMONAS SPUTIGENA

Lipopolysaccharide isolated from *selenomonas sputigena* ATCC 33150, a possible causative agent of periodontal diseases. Lipopolysaccharide residing in the cell wall outer membrane of gram-negative bacteria is a heteropolysaccharide composed of three portions: lipid A, which is the centre of its endotoxic activity; a core oligosaccharide; and an O-polysaccharide chain that determines the serological O-specificity of bacteria. Lipopolysaccharide exhibits a wide variety of endotoxic activities such as B-cell mitogenicity, induction of the Schwartzman reaction and stimulation of cytokine production (18). Lipopolysaccharide has therefore been considered to be one of the virulent factors involved in

periodontal disease. Lipopolysaccharide may cause periodontal inflammation by expressing endotoxic activities, leading to progressive periodontitis. IL-1 α produced by murine macrophages was determined by ELISA. IL-1 α activity was determined by a standard curve. IL-6 activity was determined by measuring the in vitro growth of the IL-6-dependent hybridoma cell line 7TD1 (19) using methylthio tetrazolium bromide.

Lipopolysaccharide may cause periodontal inflammation by galactose, glucosamine and a small amount of mannose. It was not able that galactosamine was detected in lipid A to a level that was as high as that of glucosamine. The lipid A backbone of the most of gram-negative bacteria is a P(1-6)-linked glucosamine disaccharide. Therefore, the presence of galactosamine in lipid A suggested that the backbone of *S. sputigena* is quite different from that of most gram-negative bacteria, although the binding site of galactose in lipid A is not yet known (20). Lipid A of *S. sputigena* exhibited slightly weaker activity than that of lipopolysaccharide. Effects on the induction of IL-1 α and IL-6 production were examined by stimulating mouse peritoneal macrophages with lipopolysaccharide and the lipid A of *S. sputigena*. The production of IL-1 α and IL-6 in the peritoneal macrophages of C3H/HeN mice increased dose-dependently upon stimulation with lipopolysaccharide and lipid A at the dose range tested. Lipid A of *S. sputigena* stimulated the IL-1 α production and also induced the production of IL-6 in the macrophages, although to a slightly lesser extent.

VII. EFFECTS OF *SELENOMONAS SPECIES* TOWARDS PERIODONTAL DISEASES

The species of *Selenomonas* dominated the diseased sites of subjects with GAgP. *Selenomonas sputigena* was the most frequently detected bacterial species, present in nine of the 10 subjects, often at high levels of about 20% of the total bacterial population. This species (21) has been previously associated with necrotising ulcerative periodontitis, rapidly progressive periodontitis and active periodontitis lesions (22). Other predominant *Selenomonas* species were *Selenomonas* sp. oral clone EW084, *Selenomonas* sp. oral clone EW076, *Selenomonas* sp. oral clone FT050, *Selenomonas* sp. strain GAA14, *Selenomonas* sp. oral clone P2PA_80, and *Selenomonas noxia*. These species are also associated with oral infections. An increased number of motile bacteria have been observed in active periodontal disease sites (23). Therefore, the high prevalence and proportion of species of *Selenomonas* suggest a role for these species in the aetiology of aggressive periodontitis. These data suggest that species of *Selenomonas* may be associated with disease in GAgP subjects. Periodontitis is a polymicrobial disease characterised

by a complex interaction of host factors and a variety of different aetiological agents. When organisms targeted by the probe SELE were visualized in subgingival biofilms using FISH, they seemed to make a relevant contribution to the structural organization of these biofilms. Performing FISH and EM on periodontal carriers proved to be valuable for the topographic exploration of subgingival biofilms and can add valuable information to the interpretation of epidemiological data (24). *S. sputigena* in higher proportions from active sites as compared to inactive sites in patients with active destructive periodontal lesions.

VIII. INVESTIGATIONS

Previous investigations using the 16srRNA cloning and sequence showed that *selenomonas* species are present in higher proportion in the subgingival biofilms of chronic and aggressive periodontitis (25). Recent studies have reported contrasting studies regarding the potential of *selenomonas* species in periodontitis using ROQT test which is used to detect biofilms (26).

The studies showed significant results about *selenomonas* species and *selenomonas sputigena* was noted in higher proportions.

Many studies have proven that *S. sputigena* is found in active periodontal lesions and bleeding sites (27). Some authors concluded high prevalence rates between 70% and 100% for different *Selenomonas* isolates and clones (28). Other authors found highly significant differences between healthy and affected sites and suggested *Selenomonas* species to be appropriate diagnostic markers for active periodontal disease (29). *S. sputigena* may be associated with the pathogenesis of chronic periodontitis and therefore their role in the onset and progression of this infection merits further investigation.

IX. CONCLUSION

On reviewing the other studies, it clearly shows that *selenomonas sputigena* and other *selenomonas* species have significant role in periodontal diseases. The control and reduction of periodontal disease by *selenomonas* species should be investigated further for treating the periodontal problems. The products of *Selenomonas sputigena* involved in the colonization of the oral cavity and the mechanisms of inducing tissue destruction are unknown. Many authors research that culture of this *selenomonas* species is not easy to cultivate for other investigations.

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