Autoinducer-2 Signaling: Modulates Cell-Cell Quorum Sensing in Oral Biofilm

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The principle of considering dental plaque as biofilm had emerged in the last 10-15 years that totally changes the concept of pathogenesis and control of periodontal diseases. A biofilm environment confers certain properties to bacteria that are not seen in the nomadic state, a fact that explains the importance of recognizing dental plaque as a biofilm and not as bacteria in the planktonic state [1]. Some of the unique properties of biofilm are dependent on the ability of the bacteria and micro-colonies within the biofilm to communicate with one another. Bacteria also harbor vast arrays of mechanisms to sense and respond to features of their environment, including the presence of other bacteria. Small diffusible molecules known as autoinducers are synthesized and released in accordance with cell number; their subsequent detection enables bacteria to synchronously regulate behaviors at the population level in a process known as quorum sensing [2].

Quorum sensing systems in bacteria have been generally divided into at least three classes:

a) LuxI/LuxR-type quorum sensing in Gram-negative bacteria, which use acyl-homoserine lactones (AHL) as signal molecules
b) Oligopeptide two-component-type quorum sensing in Gram-positive bacteria, which use small peptides as signal molecules
c) LuxS-encoded autoinducer-2 (AI-2) quorum sensing in both Gram-negative and Gram-positive bacteria

Each type of signal molecule is detected and responded by a precise sensing apparatus and regulatory network [3-8]. Many quorum sensing signals are species specific; however, production of and responses to one molecule, autoinducer-2 (AI-2), are observed throughout the bacterial kingdom and AI-2 produced by one species can influence gene expression in another, this signal can foster interspecies communication and enable bacteria to modify behaviors such as virulence, luminescence, and biofilm formation across different species [9].

Dental plaque will be a center point production of quorum-sensing signal molecules, due to the intricacy of the biofilm organization and the existence of putative pathogens which could lead to develop periodontal diseases. As Fusobacterium nucleatum is the major co-aggregation bridge organism that links early colonizing commensals and late pathogenic colonizers in dental biofilms via the accretion of periodontopathogens, it is proved that AI-2 of F. nucleatum contributes to this interspecies interaction, and interruption of this signaling could result in the inhibition of biofilm formation of periodontopathogens [10].

Research on microbial biofilms is being made in many dimensions, with specific focus on elucidation of the genes specifically expressed by biofilm-associated organisms, assessment of different control approaches for either preventing or remediating biofilm colonization of medical devices, and development of new methods for evaluating the efficacy of these treatments [11]. The central goal is to develop Anti-quorum-sensing strategies to combat periodontal diseases.

References

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