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The Contribution of Anaerobic Bacteria that Causes Periodontitis in the Pathogenesis of Chronic Obstructive Pulmonary Disease (COPD)

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During the past decade, several studies reported that dental plaque and poor oral health have been associated with nosocomial pneumonia and Chronic Obstructive Pulmonary Disease (COPD). It is generally accepted that unhygienic oral conditions that causes periodontitis could negatively affect the general health of individuals. A cause and effect relationship between the health condition of the oral cavity and some systemic diseases is attributed to the presence of dental plaque and periodontal infection [1]. Periodontitis is a chronic inflammatory reaction that affects the tooth supporting structures and bone, mainly caused by bacteria of dental plaque and calculus. It is well known that teeth, gingival margins and periodontal pockets are places that could harbor bacterial colonization. Accordingly it may serve as persistent eco-system for potential pathogens associated with respiratory tract infections. One cubic millimeter of dental plaque contains about (100 million) bacteria [2-4]. In the dento-gingival cavity, plaque accumulation leads to the growth of bacteria such as *Porphyromonas gingivalis* and *Fusobacterium nucleatum* [5].

Chronic Obstructive Pulmonary Disease (COPD) is a serious respiratory infection affecting a significant segment of the population and considered as the sixth cause of mortality [6]. The condition is characterized by chronic obstruction to the airflow with excess production of sputum resulting in breathing related problems including two lung diseases, chronic bronchitis and emphysema and sometimes asthma [6-9]. The factors responsible for the initiation or exacerbation of the two conditions are not completely known, although investigators thought it to be provoked in part by bacterial infection [10]. Recent evidence now supports the oropharyngeal region as the likely source of the causative bacteria it appears that teeth may serve as an eco-niche of respiratory infection in which severe anaerobic lung infections can occur following aspiration of salivary secretions especially in patients with oral infection [7,8]. It is completely clear that the bioactive molecules when released from periodontal tissues into the secretions and then aspirated, they may modify the pathophysiology of respiratory mucosa facilitating the adhesion of respiratory pathogens [8,9]. Also enzymes within the aspirate may expose receptors that permit further adhesion and colonization of respiratory pathogens, and may destroy protective molecules such as mucins. The resulting bacterial products or host cytokines including C-reactive protein, IL-8 and Tumor Necrosis Factor- α (TNF - α) in the aspirate may induce more cytokine production from respiratory epithelial cells with subsequent recruitment of inflammatory cells that aid in the alterations of the respiratory epithelium making it more susceptible to infection [8,11].

It should be understood that poor oral health alone is not responsible for COPD rather poor oral health may work in concert with other factors such as continued smoking, environmental pollutants, viral infections, allergy, and genetic factors where all might contribute to exacerbation and progression of respiratory diseases including COPD and pneumonia [8,12]. Although the pathology of periodontitis and COPD is complex and involves many cell types such as CD8 positive cells and macrophages, both conditions are characterized mainly by neutrophilic inflammation.

Increasingly, there is evidence that the two conditions are underpinned by similar pathophysiological processes, especially centered on the functions of the neutrophil. These include a disturbance in protease/anti-protease and redox state balance [13]. Thus it is convincible that improved oral health especially infections is crucial and may help in the prevention of progression of COPD.

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