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The Association Between Periodontitis and Respiratory Diseases: A Comprehensive Review

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Abstract

Periodontal disease is a significant factor in periodontal destruction, primarily initiated by a multi-bacterial infection that progresses through a cascade of chronic inflammatory responses. This persistent inflammation not only affects periodontal tissues but also has systemic implications, heightening the risk of various systemic diseases. Among these, respiratory diseases—which are closely linked to inflammatory processes, are of particular concern. Conditions such as asthma, pneumonia, chronic obstructive pulmonary disease (COPD), and lung cancer have been increasingly associated with the chronic inflammatory state induced by periodontitis. The aim of this review was to elucidate the mechanisms underlying the association between periodontitis and respiratory diseases. Chronic periodontitis creates a state of systemic inflammation that can disseminate through the bloodstream, impacting distant organs, including the lungs. Emerging evidence from recent studies highlights the role of periodontitis in both the initiation and exacerbation of respiratory diseases. Microbial pathogens associated with periodontitis, such as *Porphyromonas gingivalis*, can be aspirated into the lower respiratory tract, leading to either direct infection or modulation of the local immune response. This can increase susceptibility to respiratory conditions such as pneumonia and exacerbate pre-existing diseases like COPD and asthma. In summary, the relationship between periodontitis and respiratory diseases is complex and multifaceted, involving direct microbial interactions, systemic inflammation, and immune modulation. Understanding these connections is crucial for developing integrated therapeutic strategies that address both oral and respiratory health.

Keywords: inflammation, periodontitis, respiratory disease

Introduction

Periodontitis is a chronic inflammatory disease resulting from an imbalance between pathogenic microorganisms and the host's defense mechanisms, leading to the destruction of the tissues surrounding the tooth. Clinically, periodontitis is characterized by gingival inflammation, loss of connective tissue and alveolar bone, increased probing depths, formation of periodontal pockets, and gingival recession.[1] Periodontitis has been recognized as a global public health burden, contributing significantly to tooth loss in the adult population worldwide.[2] The prevalence of total periodontitis (including mild, moderate, and severe forms) was reported to be 42%, based on full-mouth examinations conducted among the adult U.S. population during the 2009–2014 cycles of the National Health and Nutrition Examination Survey (NHANES).[3] Several predisposing factors associated with periodontitis have been identified, including tobacco smoking, poor oral hygiene, hormonal changes in females, age, genetic predisposition, and systemic diseases.[4] Recent studies have reported a bidirectional association between periodontitis and various systemic diseases, including cardiovascular disease, metabolic syndrome, osteoporosis, type II diabetes, gastrointestinal and colorectal cancer, as well as respiratory diseases.[5] It has been suggested that the presence of periodontal-related pathogens and their metabolic by-products in the oral cavity may trigger the host immune response beyond the oral cavity, potentially contributing to the development of life-threatening systemic diseases.[6]

Respiratory diseases have been recognized as a leading cause of increased morbidity and mortality in populations worldwide.[7] A recent study using data from the Sixth Korea National Health and Nutrition Examination Survey (KNHANES) conducted in 2014 evaluated the prevalence of periodontitis and investigated its association with reduced pulmonary function. The findings suggest that approximately 60% of patients with periodontitis are affected by respiratory conditions.[8] Furthermore, the severity of these respiratory diseases correlates with the progression of periodontal infection.[9]

It has been suggested that the continuity between the oral cavity and the respiratory tract may serve as a potential reservoir, where bacteria harbored in the mouth can be transmitted to the airway and lungs. Although the precise mechanisms underlying the transmission of periodontitis-associated bacteria to the respiratory tract remain debated, two potential mechanisms have been proposed: 1) direct bacterial transmission via hematogenous spread and aspiration[10] and 2) indirect inflammatory responses of the respiratory system caused by periodontitis-associated bacteria and their by-products.[11] Therefore, oral microorganisms can readily impact the respiratory system, potentially leading to the development of airway infections and exacerbating lung inflammation.

This review article examined the relationship between periodontitis and four major respiratory diseases. This review also addressed the inflammatory mechanisms that play a crucial role in respiratory diseases, the pathophysiological mechanisms of periodontitis, and the impact of periodontitis-related microorganisms and their by-products on the pathophysiology and severity of asthma, pneumonia, chronic obstructive pulmonary disease (COPD), and lung cancer.

Periodontal disease

Periodontitis is a chronic inflammatory disease attributed to hyper-inflammatory responses elicited by dysbiotic bacterial communities or dental biofilms in conjunction with host immune reactions. The microbial etiology of periodontitis is currently explained by the polymicrobial synergy and dysbiosis (PSD) model. In this model, the gingival crevice is populated by diverse, compatible microorganisms that form heterotypic communities or biofilms. Under homeostatic conditions, these communities maintain equilibrium with the host, allowing the host to regulate bacterial virulence factors, such as proteases and toxins, produced by these microorganisms. However, when keystone pathogens, including *Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia, Fusobacterium nucleatum, Prevotella intermedia,* and *Aggregatibacter actinomycetemcomitans,* colonize the site—even at low abundance—they promote the growth of other bacteria, alter microbiota composition, and modify metabolic activities. This disruption ultimately shifts the microbial balance towards dysbiosis.[12] Dysbiosis is a state in which previously commensal microbiota act as proinflammatory pathobionts. As these dysbiotic communities proliferate, they can impair host immune surveillance, ultimately leading to the destruction of periodontal tissues.[13]

Porphyromonas gingivalis is recognized as a key

causative bacterium in periodontal disease, and its abundance can be used to predict the progression of chronic periodontitis. When the periodontium is colonized by dental biofilms, keystone pathogens such as *Porphyromonas gingivalis* produce various virulence factors, including capsules, fimbriae, proteases, and lipopolysaccharides (LPS), which contribute to the onset of dysbiosis.[14] LPS is a crucial component of the outer membrane of Gram-negative bacteria, playing a significant role in activating the innate immune response in damaged gingival cells. LPS induces the release of cytokines such as IL-1, IL-6, IL-10, IL-12, and TNF-α. The elevated levels of LPS-inducible cytokines stimulate the recruitment of neutrophils and macrophages from the gingival epithelium, leading to immune-inflammatory destruction of periodontal tissues.[15]

It is well established that chronic periodontitis indirectly induces the release of a variety of inflammatory mediators, including cytokines, histamine, prostaglandins, substance P, matrix metalloproteinases (MMPs), bacterial toxins, and enzymes from both host and bacterial cells. These mediators trigger inflammatory responses to combat periodontal infections. Additionally, several studies have reported that these inflammatory mediators can be transmitted directly or indirectly from the oral cavity to distant organs, adversely affecting various systemic diseases, such as diabetes, cardiovascular disease, pulmonary disease, and chronic kidney disease.[16] Moreover, there is evidence suggesting a possible association between periodontitis and an increased risk of malignancy, both in nearby organs (e.g., oral cancer) and in distant organs (e.g., pulmonary and pancreatic cancers).[17]

Inflammation and respiratory diseases

Inflammation is a physiological response of the immune system that can be triggered by various harmful factors, including pathogens, damaged cells, and toxic compounds. This inflammatory response is crucial in several airway pathologies, including asthma, pneumonia, and COPD.[18] Additionally, inflammation plays a critical role in the progression of lung cancer, contributing to the neoplastic process by promoting tumor proliferation, invasion, migration, and metastasis.[19]

The lungs, as organs frequently exposed to the microbiome through both direct and indirect pathways, exhibit distinct microbial profiles. Studies utilizing culture-independent techniques have shown that a healthy lung predominantly harbors phyla *Bacteroidetes* and *Firmicutes*. In contrast, pathological conditions such as asthma, pneumonia, COPD, and malignancy are associated with alterations in the lung microbiome. These changes are driven by dynamic shifts in the microbial ecosystem, influenced by factors such as alterations in oxygen tension, blood circulation, and the deposition of effector inflammatory cells. Notably, the altered microbiome in the airways can trigger inflammatory responses and contribute to the pathogenesis of respiratory diseases.[20]

In the context of respiratory inflammation, various types of inflammatory cells are involved. Dendritic cells and macrophages form the first line of defense by recognizing a wide range of pathogens. Once a dendritic cell identifies, ingests, and processes an antigen, it migrates to the lymph nodes, presenting the antigen to resident T cells and thereby initiating an immune response.[21] Macrophages play a crucial role in regulating both acute and chronic inflammatory responses. Alongside dendritic cells, they are capable of phagocytosing bacteria, particulates, and apoptotic cells. Additionally, macrophages are a primary source of cytokines, chemokines, and other inflammatory mediators that either promote or inhibit the immune response.[22] Neutrophils serve as the second line of defense. During pulmonary infections, they migrate from the pulmonary capillaries into the air spaces, where they phagocytose pathogens. Following phagocytosis, neutrophils utilize reactive oxygen species, antimicrobial proteins (such as bactericidal permeability-inducing protein and lactoferrin), and degradative enzymes (such as elastase) to eliminate ingested microbes.[23] Lymphocytes are distributed throughout the airways and lung parenchyma. T lymphocytes are responsible for cell-mediated immunity, while B lymphocytes contribute to humoral immune responses through the synthesis of antibodies. T lymphocytes are divided into two major subsets: CD4+ and CD8+ cells. CD4+ T lymphocytes, also known as T helper cells, are further classified into type 1 T helper cells (Th1) and type 2 T helper cells (Th2). Th1 cells are involved in cellular immune responses targeting intracellular antigens, secreting cytokines such as interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α). In contrast, Th2 cells mediate humoral immune responses to extracellular antigens, releasing various cytokines including IL-4, IL-5, IL-9, and IL-13.[18] An imbalance

in the concentrations of these cytokines can exacerbate immune responses and contribute to the pathogenesis of several respiratory diseases.

Association between periodontitis and respiratory diseases

Given the continuity between the oral cavity and the respiratory tracts, exploring the relationship between the oral microbiota and the lungs can provide insight into the pathophysiology of lung diseases in contrast to healthy states. There is an increased risk of respiratory disease associated with poor oral health. Improving oral health has been linked to a reduction in respiratory events among high risk elderly patients in nursing homes and intensive care units.[24] The most common bacterial strains in the oral cavity include *Streptococcus, Lactobacillus,* and *Prevotella,* with *Porphyromonas gingivalis* being the key pathogen typically associated with periodontal infections.[6] Oral microorganisms have also been detected in the respiratory tract. A microaspiration study using bronchoalveolar lavage (BAL) samples from healthy lungs revealed that oral microorganisms such as *Prevotella* and *Veillonella* are enriched in lower airway samples, and these microbiota are linked to increased numbers of lymphocytes and neutrophils.[25] Similarly, a study has demonstrated that the oral and lung microbiomes are largely homogenous. The finding revealed that the bacterial communities of healthy lungs share a significant overlap with those of the mouth.[26]

Current studies have demonstrated that chronic periodontitis is associated with not only respiratory diseases but also a range of other systemic conditions, including cardiovascular diseases, metabolic syndrome, osteoporosis, type II diabetes, and malignancies of various organs. It is clear that periodontitis-induced immune responses contribute to these systemic diseases through both hematogenous dissemination and trans-tracheal routes (Figure 1). Through the hematogenous route, periodontitis-related microorganisms and their by-products can travel from the oral cavity to the respiratory tract via the bloodstream, directly triggering the pathophysiology of the lungs.[27] In the transtracheal route, high levels of periodontitis-related microbiota in saliva can be aspirated into the lungs, leading to pulmonary bacterial

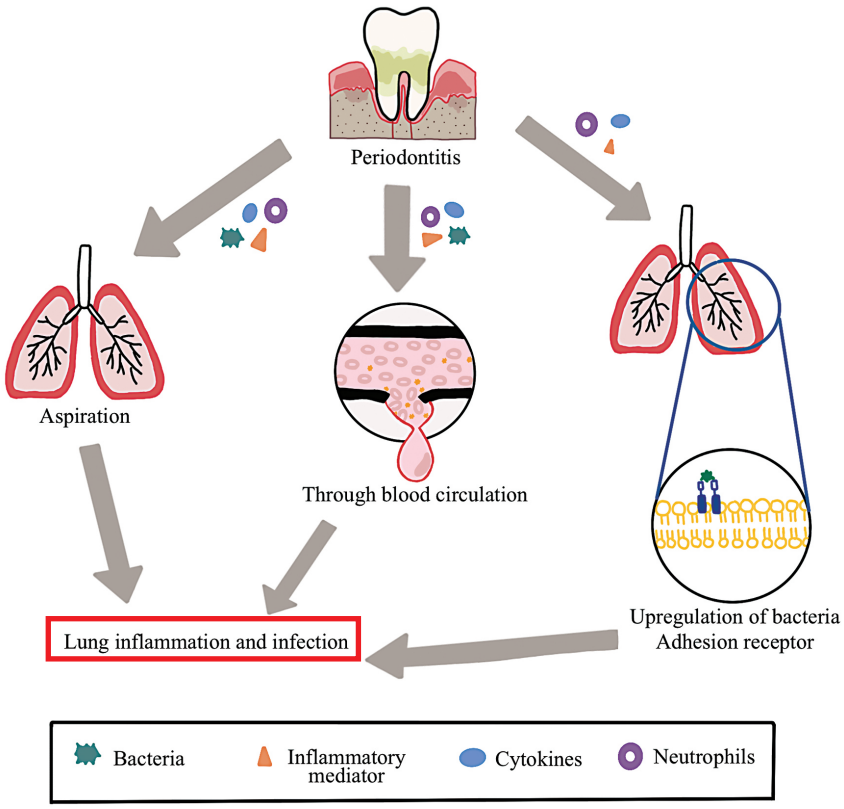

Figure 1: The routes through which periodontal pathogens can reach the respiratory tract include aspiration, hematogenous spread, and indirect mechanisms involving alterations in the respiratory epithelium and the upregulation of adhesion receptor.

colonization.[28] In addition to the direct bacterial transmissions mentioned, periodontitis-related bacteria and their by-products can indirectly induce alterations in the respiratory epithelium, thereby increasing susceptibility to bacterial infections.[29] In cases of untreated periodontal disease among high-risk individuals, a wide range of cytokines and other biologically active molecules continuously released from periodontal tissues and peripheral mononuclear cells may modify the respiratory epithelium. This can facilitate colonization by respiratory pathogens through the upregulation of adhesion receptor expression on mucosal surfaces, ultimately leading to infection.[30]

Periodontitis and asthma

According to the World Health Organization, asthma affected approximately 262 million individuals in 2019 and was responsible for 455,000 deaths. The prevalence of comorbidities is higher among older adult patients with asthma.[31] Asthma is a chronic respiratory disease triggered by pathogens, irritants, or environmental factors, leading to bronchospasm. It is characterized by reversible airway obstruction, with symptoms including wheezing, shortness of breath, and chest tightness. These symptoms can vary in intensity over time. This condition results in symptoms such as chest tightness, wheezing, and shortness of breath. Upon exposure to stimuli, Th2 cells are activated and release interleukins IL-4, IL-5, IL-9, and IL-13. Additionally, these stimuli promote eosinophilic inflammation and the recruitment of immunoglobulin E (IgE), which further induces the production of inflammatory mediators such as histamine, and cysteinyl leukotrienes. These inflammatory mediators contribute to bronchoconstriction, pulmonary edema, and mucous secretion, which constitute the symptoms of asthma.[32]

Several studies have reported the positive relationship between periodontitis and asthma. A case-control study reported an association between chronic periodontitis and severe asthma, indicating that the risk of asthma induced by periodontitis was three times higher in patients with periodontitis compared to healthy individuals.[33] The associations between subjective oral health status and allergic diseases in adult Korean were also examined. In this study, the adjusted odds ratio (OR) for asthma was 1.48 in individuals with poor oral health compared to those with good oral health.[34] Similarly, another study found that patients in the asthma group had more severe periodontitis, with a greater proportion suffering from stage IV periodontitis. In contrast, stage II and III periodontitis were more prevalent in the non-asthma group. Increased tooth loss, attachment loss, and bone loss were also found among patients using anti-asthmatic drugs.[35] In contrast, A recent meta-analysis found no significant association between periodontal disease and asthma.[36]

Although the mechanism by which periodontitis induces asthma remains unclear, several plausible explanations have been proposed. First, *Prevotella intermedia* is a key periodontal pathogen involved in dysbiotic biofilms. *Prevotella intermedia* and its associated pathogens contribute to periodontitis and stimulate the release of proinflammatory cytokines, hydrolases, collagenases, and MMP, thereby exacerbating periodontal breakdown.[37] Second, certain MMPs, including MMP-1, MMP-2, MMP-3, MMP-8, and MMP-9, are prominently present in allergic and asthmatic patients.[38] These MMPs play a role in mediating cell trafficking and remodeling of the pulmonary tract.[39] And lastly, the prevalence of gingivitis in individuals with asthma is likely related to alterations in immune responses and habitual mouth breathing. Although the role of allergies in periodontal disease is not yet fully understood, IgE-mediated mechanisms contribute to periodontal tissue destruction. Notably, similar cytokines are involved in both periodontal disease and airway inflammation, with elevated levels of IL-5 and IL-6 observed in patients with asthma and periodontitis. The inflammatory processes in periodontal disease and asthma exhibit similar pathophysiological mechanisms, which may partially explain the high frequency of periodontal inflammation in individuals with asthma.[40] The continuity between the oral cavity and the trachea allows bacteria and periodontopathogens to reach the airways through both blood circulation and aspiration. The release of cytokines, mediators, and toxic by-products sensitizes the pulmonary epithelium, leading to local inflammatory responses (Figure 2).

Periodontitis and pneumonia

Pneumonia is an acute pulmonary inflammation caused by various infectious microorganisms, including fungi, viruses, and bacteria. Bacterial pneumonia is the most common and life-threatening form of the disease, particularly in adults aged 65 years and over, and children aged up to two years. According to the Global Burden

of Disease Study 2015, lower respiratory tract infection is the leading cause of infectious disease death, and the fifth most common cause of death overall and those with comorbidities.[41]

Pneumonia is characterized by symptoms such as cough, shortness of breath, and sharp or stabbing chest pain, particularly during deep breathing. The most common microbial pathogens responsible for pneumonia include *Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae,* and *Klebsiella pneumoniae*.[42] In response to bacterial infections, alveolar macrophages, which serve as a primary defense mechanism in the respiratory system, engulf pathogens through endocytosis. This process subsequently triggers the release of proinflammatory cytokines, such as IL-1, IL-8, and TNF-α, leading to localized inflammation at the site of infection. This respiratory defense mechanism is thought to induce localized lung inflammation, which may progress into pneumonia.[43]

It is well established that there is an association between periodontitis and pneumonia, with dental biofilms contributing to the increased incidence and progression of pneumonia. Periodontitis and pneumonia share common risk factors, including tobacco smoking, stress, and aging. Aspiration is the primary route through which periodontitis contributes to pneumonia.[7] Initially, subgingival microorganisms such as *Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia*, and *Fusobacterium nucleatum* are transmitted to the respiratory tract through aspiration. These periodontitis-associated pathogens colonize the respiratory system, becoming a source of infection that can lead to bacterial pneumonia. Additionally, proinflammatory biomolecules, including IL-1 and TNF-α, present in gingival crevicular fluid from periodontitis, can diffuse into saliva and subsequently be aspirated into the respiratory tract.[44] As a result, periodontitis increases the risk of pneumonia and exacerbates the inflammatory processes that contribute to its progression (Figure 3).

Several studies have demonstrated that periodontitis increases the incidence and accelerates the progression of pneumonia. Pathogenic microorganisms that colonize the oral microbiome have been linked to aspiration pneumonia, particularly in older individuals residing in long-

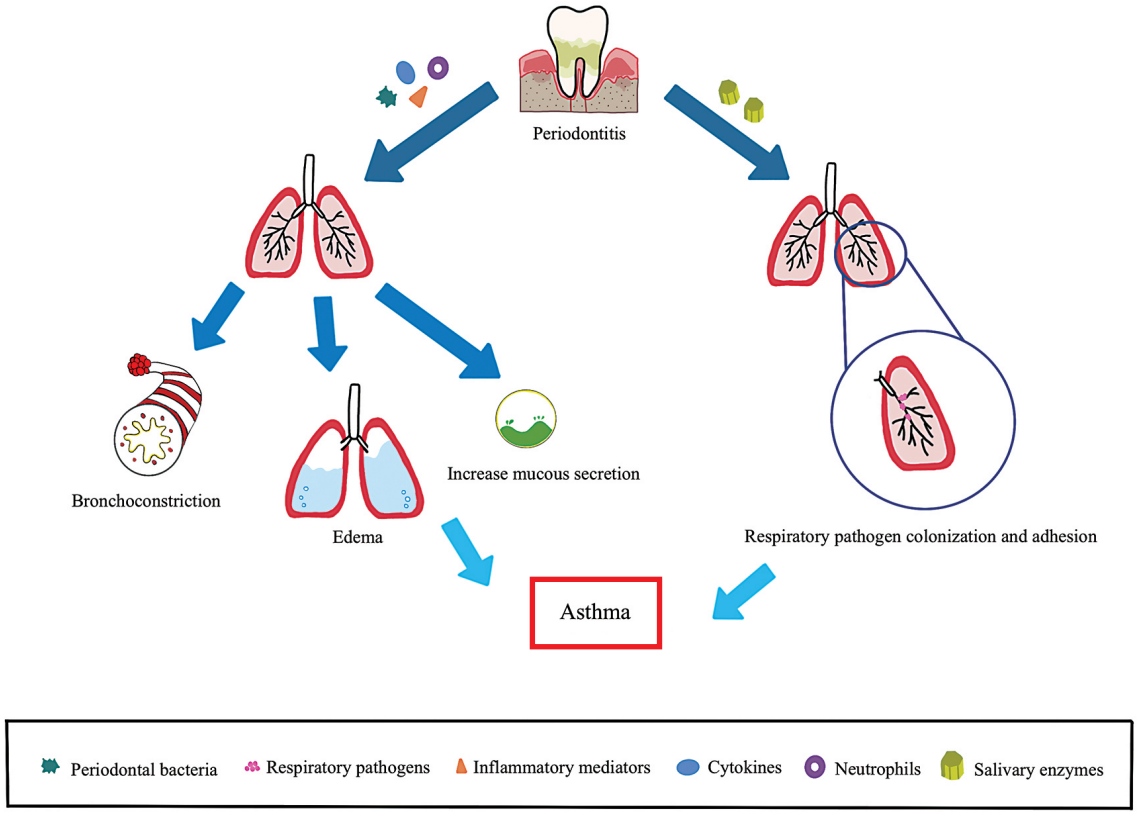

Figure 2: Periodontal and respiratory pathogens, along with inflammatory cells and mediators or salivary enzymes from patients with periodontitis, can reach the respiratory tract through aspiration, hematogenous spread, and indirect mechanisms. These pathways can facilitate the colonization and adhesion of respiratory pathogens, adversely affecting lung health and exacerbating asthma severity.

term care facilities.[44] A nationwide population-based study from 2001-2012 in Taiwan indicated that patients who received periodontal treatment exhibited a significantly lower risk of developing pneumonia compared to the general population.[45] The previous study assessed the impact of periodontitis on pneumonia progression by comparing C-reactive protein (CRP) levels between pneumonia patients and healthy individuals. The CRP levels serve as an indicator of host immune responses, reflecting periodontal tissue destruction. This study have shown that CRP levels were significantly higher in pneumonia patients compared to the control, further confirming the association between periodontitis and pneumonia.[46]

Periodontitis and chronic obstructive lung disease (COPD)

The airflow limitation that characterizes COPD results from a prolonged time constant for lung emptying, which is caused by increased resistance in the small conducting airways and increased lung compliance due to emphysematous destruction.[47] Two hypotheses have been proposed to explain the mechanisms underlying periodontitis-induced COPD (Figure 4). The first mechanism involves aspiration. Periodontitis-associated microorganisms reside in the oral cavity and can be aspirated directly into the respiratory tract. These periodontal pathogens may contribute to the exacerbation of inflammation, airflow limitation, and the degradation of lung structures. Specifically, *Porphyromonas gingivalis*[48] and *Fusobacterium nucleatum*[49] stimulate the gene expression of MUC5AC, a mucin core protein, in primary human bronchial epithelial cells, potentially leading to narrowing of the airway lumen. The second hypothesis suggests that periodontal pathogens act as reservoirs, spreading infection via the bloodstream.[50]

Several studies have reported the association between periodontitis and COPD. Poor periodontal health, as indicated by missing teeth and plaque index scores, was significantly associated with a lower quality of life in patients with COPD.[51] Patients with more severe periodontal infection exhibited a greater risk for COPD compared to those with mild periodontitis or healthy individuals.[52] A meta-analysis revealed a significant association between deteriorating periodontal health

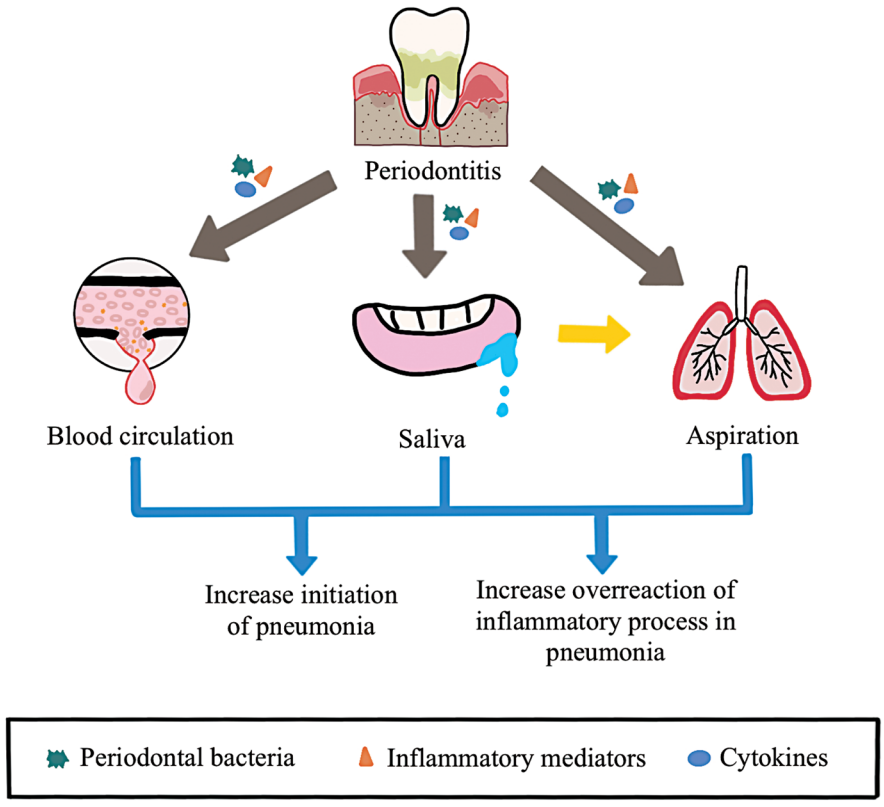

Figure 3: The routes for periodontal pathogens and inflammatory mediators in periodontitis patients to reach the respiratory tract can initiate pneumonia and exacerbate the inflammatory response, contributing to the progression of the disease.

and an increased risk of COPD, encompassing chronic bronchitis and emphysema, with ORs ranging from 1.45 to 4.50. The authors suggested that cigarette smoking may have confounded the findings, as periodontal status was linked to a heightened risk of COPD among current smokers (relative risk [RR] = 1.63, 95% confidence interval [CI] = 1.20–2.21), whereas no such association was observed in never-smokers (RR = 1.19, 95% CI = 0.71–1.98).[53] The recent cross-sectional study in Japan revealed mutual relationships among periodontal disease and its various factors. Probing pocket depth (PPD) was significantly associated with occlusal force in employees with moderate COPD.[54] In contrast, some studies indicate that smoking and aging may significantly influence the outcomes. Their findings revealed that the presence of dentures, missing teeth, oral mucosal disease, and a higher index for decayed, missing, and filled permanent teeth (DMFT) correlated with reduced maximal expiratory flow at 25%. Because periodontitis and DMFT were associated only with age and the amount of smoking, they concluded that much of the association between pulmonary function and poor oral hygiene could be explained by smoking and age.[55]

Since periodontitis and COPD share several predisposing factors, including smoking, age, obesity, socioeconomic status, and living conditions. Dental plaque containing bacteria may contribute to the risk of COPD; therefore, proper attention to tooth brushing and overall oral hygiene may help reduce this risk. Patients with COPD who are undergoing inhaled steroid treatment are advised to use a spacer and rinse their mouths with cold water afterward. Individuals susceptible to COPD should prioritize their oral hygiene, schedule regular dental check-ups, and refrain from smoking.[56]

Periodontitis and lung cancer

Lung cancer is a multifactorial disease influenced by various factors, including behavioral patterns, genetic predispositions, environmental exposures, infections, chronic irritation, and inflammation. Unlike other respi-

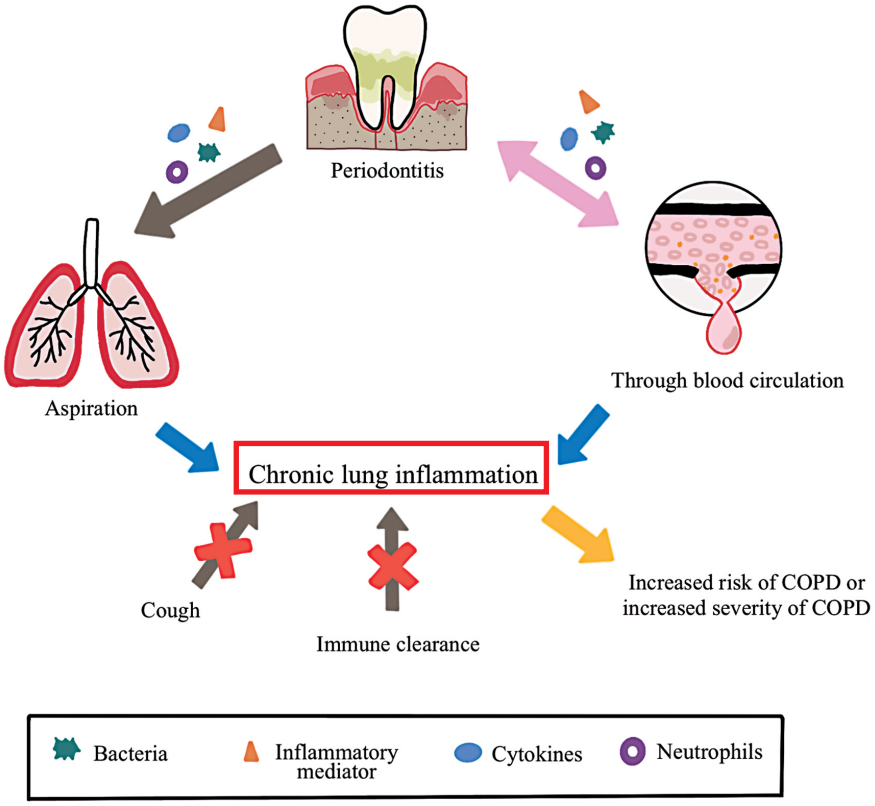

Figure 4: Periodontal pathogens can reach the respiratory tract through aspiration (indicated by brown arrows) and hematogenous spread (indicated by pink arrow), leading to chronic lung inflammation and an increased risk or severity of chronic obstrutive pulmonary disease (COPD). However, the body's natural reflexes, such as coughing and immune clearance, can help mitigate the inflammatory process.

ratory diseases, the relationship between periodontitis and lung cancer appears to be more complex. Current evidence suggests that periodontal pathogens may not directly cause lung cancer but instead contribute to tumorigenesis through inflammatory processes associated with disease progression. The chronic inflammation resulting from periodontitis can lead to systemic changes that create an environment conducive to cancer development. These environment contribute to exacerbated cell proliferation, invasion, and metastasis to other organs.[57] Although inflammation serves to protect the body from injuries, persistent or chronic inflammation can lead to several diseases, including diabetes, cardiovascular disease, musculoskeletal disorders, allergies, COPD, and malignancies.[58] Under normal conditions, inflammatory responses are regulated by a balance between proinflammatory cytokines (e.g., IL-1, TNF-α, IFN-γ) and anti-inflammatory cytokines (e.g., IL-1β, IL-10, IL-13). However, when this balance is disrupted, inflammatory cytokines may trigger neoplastic processes and stimulate the proliferation, survival, and metastasis of tumor cells.[59]

Several studies have identified chronic inflammation as a critical factor in the pathogenesis of lung cancer (Figure 5). They have reported the role of inflammation in pulmonary neoplasms. Inflammatory components, particularly the leukocyte population, promote tumor progression through their ability to release various cytokines, chemokines, and cytotoxic mediators, such as reactive oxygen species, MMPs, IFN, TGF-β, natural killer cells, macrophages, myeloid-derived suppressor cells, and mast cells. Consequently, these inflammatory components influence the proliferation of malignant cells, angiogenesis, and tumor metastasis.[60] The previous study demonstrated that *Porphyromonas gingivalis* LPS can synergistically induce the release of IL-6 and IL-8 in the human pulmonary mucoepidermoid carcinoma cell line (NCI-H292). The induced IL-6 and IL-8 activate the phosphorylation of the JNK signaling pathway in NCI-H292 cells, which may

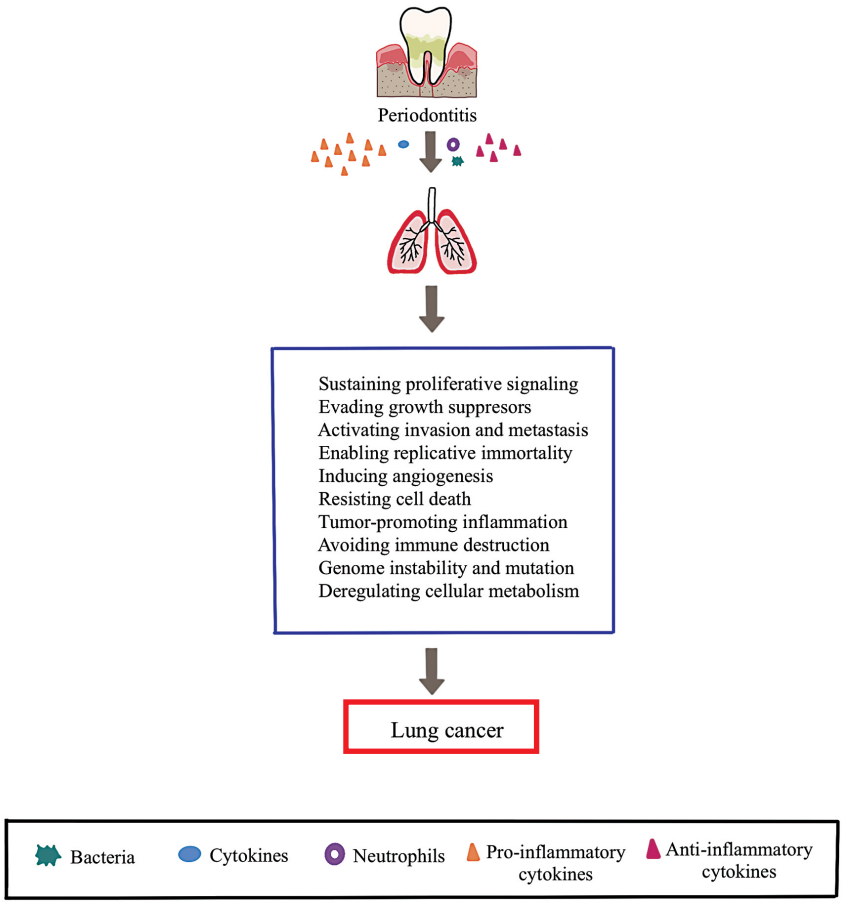

Figure 5: Periodontal pathogens, along with the inflammatory cells and cytokines can promote biological processes that may contribute to the development and progression of lung cancer.

contribute to the development of pulmonary neoplasms.[61] Similarly, another study examined the relationship between IL-6 and IL-8 and pulmonary cancer. The findings revealed that IL-6 and IL-8 were significantly associated with stage I lung cancer patients.[62] In addition, a recent study reported a 13% increase in the risk of total cancer among non-smoking men with periodontitis, while a 45% increase in the risk of total cancer was observed in men with advanced periodontitis.[63] A recent meta-analysis identified an increased risk of lung cancer in patients with chronic periodontitis compared to those without the condition.[64] Additionally, another study concluded that pocket depth may serve as a potential risk factor for lung cancer development. The incidence of lung cancer was observed to be twice as high in patients with increased pocket depth, even after controlling for age and smoking status.[65] It has been suggested that periodontitis may influence the risk of malignancy through systemic immune dysregulation.

Conclusions

This review highlights the positive association between periodontitis and several respiratory diseases, including asthma, pneumonia, COPD, and lung cancer. These conditions share common risk factors, such as smoking, age, and stress, which may influence their development. Periodontopathogens are thought to stimulate inflammatory processes that could increase the risk and progression of respiratory diseases. By elucidating these connections, we aim to enhance awareness among healthcare providers regarding the complexities of these associations. Understanding the relationship between oral health and systemic conditions is crucial for evaluating whether interventions for oral diseases can also prevent systemic diseases. A multidisciplinary approach to treatment is vital for extending healthy life expectancy, improving quality of life, and reducing healthcare costs. However, the precise mechanisms linking periodontal pathogens to respiratory diseases remain unclear, necessitating further research.

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