Oral Health and Respiratory Infection

(Santé buccodentaire et infection respiratoire)

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Sommaire

La cavité buccale est depuis longtemps considérée comme un réservoir potentiel pour les pathogènes respiratoires. Divers mécanismes d'infection ont été mis en cause, notamment l'aspiration dans les poumons de pathogènes buccaux pouvant causer la pneumonie, la colonisation de la plaque dentaire par des pathogènes respiratoires suivie d'une aspiration, ou encore la facilitation de la colonisation des voies respiratoires supérieures par des pathogènes pulmonaires sous l'effet des pathogènes parodontaux. Plusieurs bactéries anaérobies de la poche parodontale ont été isolées de poumons infectés. Chez les patients âgés vivant en établissements de soins de longue durée, la colonisation de la plaque dentaire par des pathogènes pulmonaires est fréquente. Tant dans les maladies parodontales que dans l'emphysème pulmonaire, on observe une réponse exagérée du processus inflammatoire qui mène à la destruction du tissu conjonctif, et cette réponse exagérée pourrait expliquer le lien entre les maladies parodontales et la bronchopneumopathie chronique obstructive, quatrième cause de décès aux États-Unis. Ces résultats font ressortir l'importance d'améliorer l'hygiène buccale des patients à risque et des résidents des établissements de soins de longue durée.

Mots clés MeSH : *aged; periodontal diseases/epidemiology; pneumonia, aspiration/epidemiology; pneumonia, aspiration/prevention & control*

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The anatomical continuity between the lungs and the oral cavity makes the latter a potential reservoir of respiratory pathogens. Yet an infective agent must defeat sophisticated immunological and mechanical defence mechanisms to reach the lower respiratory tract. The defence mechanisms are so efficient that, in healthy patients, the distal airway and lung parenchyma are sterile, despite the heavy bacterial load (10^6 aerobic bacteria and 10^7 anaerobic bacteria per millilitre) found in the upper airway. An infection occurs when the host's defences are compromised, the pathogen is particularly virulent or the inoculum is overwhelming. The micro-organisms may enter the lung by inhalation, but the most common route of infection is aspiration of what pneumologists have long referred to as oropharyngeal secretions.[1] Therefore, it is plausible that oral micro-organisms might infect the respiratory tract. However, only recently has the role of the oral flora in the pathogenesis of respiratory infection been examined closely.

Epidemiology of Pulmonary Disease: Significance of the Problem

Pneumonia is usually classified as community-acquired pneumonia (CAP) or nosocomial (hospital-acquired) pneumonia. The distinction is important because the pathogens implicated and the preventive measures taken are very different for the 2 types. CAP is a frequent illness, with an incidence rate estimated at about 8 cases per 1,000 inhabitants per year in industrialized countries. The mortality rate is about 7% in hospitalized patients.[1] *Streptococcus pneumoniae* and *Haemophilus influenzae* are the main causative organisms (accounting for 40% to 60% of cases).[1] Until now, the only authors who have looked at the association between acute respiratory diseases (including CAP) and oral health markers did not find any association between them.[2]

Nosocomial pneumonia is the second most common infection (after infections of the urinary tract[3]) in long-term care institutions. It accounts for approximately 10% to 15% of all hospital-acquired infections, and 20% to 50% of affected patients will die because of the infection.[4] The etiologic agents are mainly gram-negative bacilli and staphylococci. The frequency of infection with anaerobic organisms is uncertain because of the technical difficulty associated with anaerobic culture and the possibility of contamination by anaerobic oral flora during sampling. Because of these difficulties, anaerobic bacteria are not cultured in routine analysis of pulmonary

microbiological samples. In one frequently cited study, 35% of nosocomial infections were due to obligate anaerobes.[5] Aspiration pneumonia refers to bacterial infection occurring in association with a condition predisposing to aspiration, such as stroke, Parkinson's disease or alcoholism. The bacteria involved are indigenous oral flora or they are acquired in hospital. As for aspiration pneumonia, the infections occurring during mechanical ventilation are closely associated with anaerobic bacteria.[6]

Because anaerobes are implicated in pneumonia, a role for the oral cavity in the pathogenesis of this disease has been suspected since the beginning of the 20th century. Yet it was only in the 1970s that the role of anaerobic bacteria in pulmonary infections came to be investigated more extensively.[7]

The oral flora may also be implicated in pulmonary diseases affecting airflow. The most prevalent is chronic obstructive pulmonary disease (COPD). In the United States, COPD affects 14 million people, and in 1991, it was the fourth leading cause of death, a toll that has been increasing over recent years.[1] The main etiological factor is tobacco smoking, but bacteria (including oral bacteria) may play a key role in progression of the disease.

Given the high prevalence of lower respiratory tract infection (RTI), if even a small percentage of these cases are caused or facilitated by oral flora, the total number of pulmonary infections attributed to these organisms would be significant.

Mechanism of Infection

Two routes exist for oral micro-organisms to reach the lower respiratory tract: hematogenous spread and aspiration. Hematogenous spread of bacteria is an inevitable adverse effect of some dental treatments and may occur even after simple prophylactic procedures. Nonetheless, this route of infection seems rare, and only 2 well-documented case reports could be found in the literature. In both cases hematogenous spread was the most likely source of pulmonary infection with periodontal anaerobes.[8,9] In contrast, aspiration of material from the upper airway occurs in 45% of healthy subjects during sleep and in 70% of subjects with impaired consciousness.[10] It is probably the main cause of nosocomial infection along with aspiration of gastric contents. Three mechanisms of infection related to aspiration of material from the upper airway can be envisioned. First, periodontal disease or poor oral hygiene might result in a higher concentration of oral pathogens in the saliva. These pathogens would then be aspirated into the lung, overwhelming the immune defences. Second, under specific conditions, the dental plaque could harbour colonies of pulmonary pathogens and promote their growth. Finally, periodontal pathogens could facilitate the colonization of the upper airways by pulmonary pathogens.

Microbiological Similarities Between Organisms Infecting the Lungs and Oral Flora

The vast majority of pulmonary diseases are due to aerobic bacteria that are found in the oral flora but are not related to any oral diseases.[11] In contrast, the list of anaerobes (facultative or obligate) that are implicated in the destruction of periodontal tissues and that have also been isolated from infected lungs is quite long.[11] For example, *Actinobacillus actinomycetemcomitans* and *Fusobacterium nucleatum* have both been isolated from infected lungs[7,9] whereas *Pseudomonas aeruginosa*, a known pulmonary pathogen, has been isolated from patients with "refractory" periodontitis.[12] The pulmonary pathogenicity of *Bacteroides gingivalis* has been confirmed in an animal model simulating aspiration.[13]

It seems that commensal oral micro-organisms such as *Streptococcus intermedius* may also become opportunistic pathogens under specific circumstances. The colonization of the oral flora by respiratory pathogens has specifically been investigated in 4 studies. Lindemann and others[14] isolated strains of *P. aeruginosa* from the tongues of 14 of 20 patients with cystic fibrosis. None of the 20 age-matched healthy patients had these bacteria, and none of the 40 plaque samples (from patients with cystic fibrosis and controls) were colonized by these strains either. However, in a study conducted in a critical care ward, Scannapieco and others[15] isolated several known pulmonary pathogens (e.g., *Klebsiella pneumoniae* and *Serratia marcescens*) from dental plaque. In contrast, none of the plaque samples collected from patients in a dental clinic harboured these species. In a similar study, Fourrier and others[16] found a high bacterial concordance between dental plaque, saliva and tracheal samples. In about 40% of their intensive care patients, dental plaque was colonized by aerobic respiratory pathogens. The colonization of dental plaque was highly predictive of concurrent or subsequent nosocomial infection. In the most recent study,[17] plaque colonization in subjects from a chronic care facility was compared with plaque colonization in age-matched outpatients from a dental clinic. In both groups, about a quarter of the samples were colonized, but the concentration of bacteria was much higher among the subjects from the chronic care facility.

Edentulous subjects participated in 2 of these studies. It seems that some species, such as *A. actinomycetemcomitans* and *Porphyromonas gingivalis,* cannot be found after dental clearance,[18] but other putative pulmonary pathogens such as *Prevotella* spp. are present in the oral microbiota of edentulous patients.[19] If denture hygiene is inadequate, anaerobic bacteria count is inversely proportional to the cleanliness of the denture.[20] Also worth mentioning is the fact that *Candida albicans* has been isolated from transtracheal aspirates from patients with pleuropulmonary infection[6] and other pulmonary samples.[21] Nevertheless, it seems that RTI is less frequent among edentulous patients.[22,23]

Mucosal Colonization and Periodontal Pathogens

Fibronectin, a protein that coats the oral mucosa, is probably involved in the ecology of the mucosal flora by providing binding sites for oral streptococci while inhibiting adhesion of more virulent bacteria.[6] The competition for colonization

seems to be modulated by the ability of bacteria to degrade fibronectin.[24] The protease activity (including degradation of fibronectin) of periodontal pathogens has been extensively studied and is correlated with poor oral hygiene.[25] Thus, fibronectinolytic activity of the crevicular fluid could play a role in pneumonia by promoting the adherence of anaerobic gram-negative bacteria to the epithelium of the upper airway.[6] Scannapieco and Mylotte[26] proposed a model to explain the colonization of the oral cavity by respiratory pathogens.

Linking Respiratory Infections and Oral Disorders

Epidemiological indications that oral micro-organisms might be implicated in lung infections are still scant. Pneumonia is most frequent in elderly people, and this segment of the population would seem a logical target for such research. However, pneumonia in elderly people has specific features that make it difficult to study. For example, the traditional clinical signs used to diagnose pneumonia (cough, fever and abnormal auscultation) lack sensitivity and specificity. Chest radiography is the gold standard for diagnosis, but it is rarely available. Also, elderly patients often have comorbidities or risk factors that could confound the relationship between oral disorders and pneumonia. For example, poor nutritional status and mediocre functional status are risk factors for pulmonary disease,[27] but they are also associated with poor oral health.[28,29] Smoking is also an obvious confounder with its profound detrimental effect on respiratory and periodontal condition. Unfortunately, it is difficult to obtain accurate smoking history for very old patients.

Three retrospective studies reported on the pneumonia and oral health in elderly people. Terpenning and others[22] found a trend toward a lower prevalence of pneumonia among edentulous elderly patients and an association between xerostomia and the development of pneumonia. In a cross-sectional study of frail elderly people living in a long-term care facility, the medical records of the residents were reviewed to identify episodes of RTI that occurred during the one-year period preceding an oral examination.[23] The prevalence of RTI was significantly greater among dentate than edentulous subjects (40% vs. 27%) and was also greater among subjects with selected oral disorders than those without such conditions (**Table 1**). The presence of selected oral disorders interacted with the degree of dependency of the subjects or their nutritional status to increase the prevalence of RTI. Also, the dentate subjects with a history of RTI had higher plaque scores than those who had not experienced an episode of RTI. The difference in RTI between edentulous and dentate subjects cannot be interpreted as an indication for removal of the remaining teeth. Indeed, even the observed difference in prevalence of 13% cannot be used to justify such a radical act, given the known negative consequences of edentulousness on nutrition and quality of life.

In the most recent study,[30] dental decay, cariogenic bacteria and periodontal pathogens emerged as potentially important risk factors for aspiration pneumonia after adjustment for established medical risk factors.

COPD and Other Chronic Pulmonary Conditions

As mentioned earlier, Scannapieco and others[2] found no evidence to support an association between acute respiratory diseases and poor oral health in the adult U.S. population. However, they did report poorer oral hygiene among patients with COPD. In a recent analysis based on a more recent and detailed survey, Scannapieco and Ho[31] found a significant association between COPD and periodontal attachment loss. The likelihood of COPD increased with severity of attachment loss, and lung function appeared to diminish as the amount of attachment loss increased. In a longitudinal study,[32] alveolar bone loss was measured with periapical radiographs at baseline and up to 25 years later in 1,118 healthy men (about 45 years old). About a quarter of the subjects developed COPD over the subsequent 25 years, and these had greater bone loss at baseline. When all the other factors that might be associated with COPD were taken into account, the risk of COPD was 1.8 times greater among the 20% of subjects with the worst alveolar bone loss (the worst quintile).

These epidemiological findings seem to be supported by biological clues. According to Travis and others,[33] pulmonary emphysema and periodontal disease share a similar mechanism of tissue destruction. Neutrophils are recruited to inflammatory sites either because of the presence of foreign materials (e.g., smoke particles) or because of chemotactic factors activated by bacteria. In both diseases, degranulation of neutrophils occurs during attempted phagocytosis (so-called "frustrated phagocytosis"), which releases proteolytic enzymes. Proteins from the connective tissue are degraded, which results

Table 1 **Prevalence of respiratory tract infection in a nursing home (adapted, with permission, from Mojon and others[23])**

	Degree of dependence[a]		Serum level of albumin[b]	
Selected oral health disorders[c]	Semidependent BI > 20	Dependent BI ≤ 20	≥ 35 g/L	< 35 g/L
Absent	23%	22%	17%	29%
Present	35%	46%	32%	52%

[a] *According to the Barthel index (BI) of functionality, where a score of 100 indicates no limitation in activities of daily life and 20 is the limit between total dependency and semidependency. No resident of the nursing home was completely independent.*

[b] *Level < 35 g/L indicates protein-energy deficiency*

[c] *In edentulous patients: denture with defective base (porous resin, old temporary reline, missing parts) or generalized stomatitis; in dentate patients: presence of visible calculus, generalized gingivitis, teeth with pulpal exposures, or root tips*

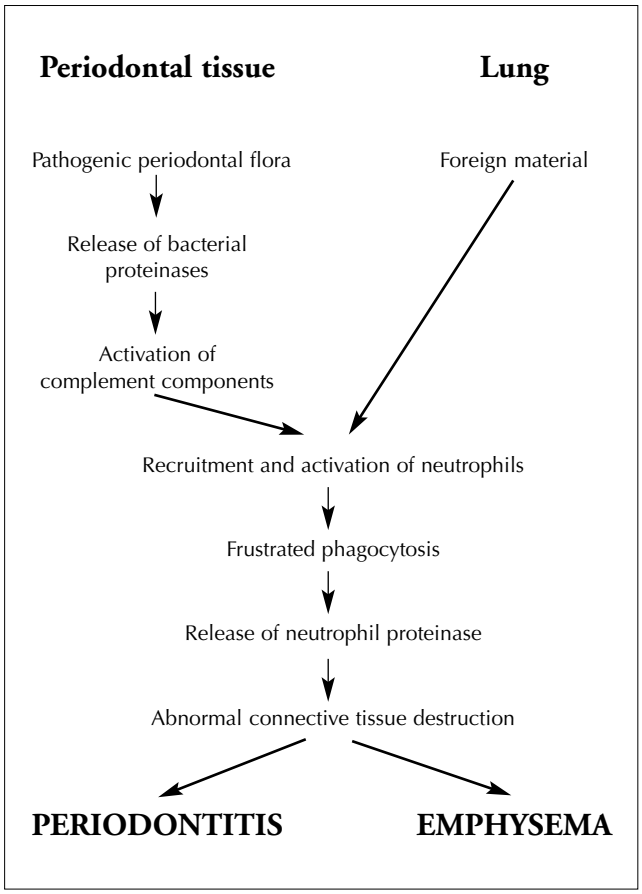

Figure 1: *Mechanism of tissue destruction in periodontal disease and emphysema (Adapted, with permission, from Travis[33])*

in the destruction of the pulmonary alveolus or the periodontal attachment (**Fig. 1**).

For cystic fibrosis, the only information available is the work of Lindemann and others,[14] as discussed earlier.

Intervention Studies

Many studies have reported on the effectiveness of decontaminating the digestive tract in the intensive care unit. Two intervention studies deserve attention because they used an oral rinse. The first study[34] was a controlled clinical trial in which a cocktail of antibiotics (polymyxin, neomycin and vancomycin) was administered to prevent ventilator-associated pneumonia in the intensive care unit. The solution was kept in the mouth for one minute and swallowed, or it was injected in the retropharynx when the patient was unconscious. The treatment was repeated every day. Only 4 (16%) of the 25 subjects in the study group developed ventilator-associated pneumonia, whereas 21 (78%) of the 27 subjects in the placebo group developed pneumonia. Unfortunately, there is no information on the number of subjects who received the oral solution in the retropharynx while unconscious. The other study[35] was also conducted in an intensive care unit and involved 353 patients undergoing cardiovascular surgery. The design was similar except that the oral rinse was 0.12% chlorhexidine gluconate. The incidence of nosocomial infection was reduced by 65% and of respiratory tract infection by 69%. Among the patients who developed a respiratory infection, gram-negative micro-organisms were more frequently isolated in the placebo group than in the study group. So far, only Japanese intervention studies have shown that improving oral hygiene reduces the incidence of pneumonia among patients at risk.[36,37] In the 1999 report,[36] the risk of pneumonia was 1.67 times higher in the control group than in the group in which oral hygiene measures were implemented, but the confidence interval suggested only a modest improvement (1.01–2.75). The other study[37] was a microbiological analysis of samples obtained from patients who received oral hygiene care and a group of controls after gargling with 5 mL of a saline solution. Colonization by various potential respiratory pathogens was more prevalent in the control group, but the study lacked baseline data and suffered from dubious statistical analysis.

Conclusion

This literature review highlighted at least 12 studies[2,14,15-17,23,30-32,34-36] (**Table 2**) that provide direct evidence of an association between pulmonary infection and oral diseases. The association seems to occur only in patients with severely compromised health, in frail elderly people and in patients with chronic pulmonary diseases. Accumulation of plaque on the teeth or dentures and some periodontal pathogens are the most likely culprits. Yet we cannot rule out that similar host susceptibility factors may lead to both periodontal and chronic pulmonary diseases. These results corroborate the suggestion made earlier[3,38] that improving oral hygiene might reduce the risk of pneumonia among subjects who are at risk. A more rapid intervention would be the use of an oral disinfectant, but studies on the long-term use of such medication are lacking. The treatment of periodontal diseases (either by repeated prescription of antibiotics or by clinical interventions) might be another way to reduce the incidence of pneumonia. This literature review underlines the necessity for regular recalls among "at risk" patients and the introduction of specific oral hygiene courses for caregivers in long-term care institutions. Nonetheless, a causal association has not been proven, and more studies, in particular intervention studies, are needed. ◆

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Table 2 Main studies showing direct evidence of an association between pulmonary infection and oral diseases

Authors	Date	Sample size	Study design	Main respiratory outcome	Major finding
Lindemann and others[14]	1985	40	C	Respiratory pathogens	Oral cavity is a potential reservoir for *Pseudomonas aeruginosa* in patients with cystic fibrosis
Pugin and others[34]	1991	52	RCT	VAP	Oropharyngeal decontamination by topical antibiotics reduces aerobic gram-negative bacteria and VAP in an intensive care unit
Scannapieco and others[15]	1992	59	C	Respiratory pathogens	Respiratory pathogens colonize dental plaque and oral mucosa
DeRiso and others[35]	1996	353	RCT	Nosocomial RTI	0.12% chlorhexidine rinse reduces occurrence of nosocomial respiratory infection in patients undergoing heart surgery
Mojon and others[23]	1997	302	C	RTI	RTIs are associated with greater plaque accumulation, specific oral health disorders and presence of teeth
Scannapieco and others[2]	1998	386	C	Respiratory diseases	No relationship exists between acute respiratory diseases and oral condition; poor oral hygiene is associated with chronic diseases
Fourrier and others[16]	1998	57	L	Nosocomial pneumonia	Respiratory pathogens in dental plaque are associated with nosocomial pneumonia
Hayes and others[32]	1998	1,118	L	COPD	Alveolar bone loss is associated with greater risk of COPD
Russell and others[17]	1999	58	C	Respiratory pathogens	Respiratory pathogens were not found in dental outpatients but were found in 14% of patients living in chronic care facilities
Yoneyama and others[36]	1999	366	CT	Pneumonia	Oral care decreases risk of pneumonia in frail, dependent elderly people
Scannapieco and Ho[31]	2001	13,792	C	COPD	Association exists between COPD and attachment loss
Terpenning and others[30]	2001	358	L	Aspiration pneumonia	Caries, cariogenic pathogens and periodontal pathogens are significant risk factors

C = cross-sectional
RCT = randomized clinical trial
VAP = ventilator-associated pneumonia
RTI = respiratory tract infection
L = longitudinal
COPD = chronic obstructive pulmonary disease
CT = non-randomized clinical trial

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