Review

Systematic Review of the Association Between Respiratory Diseases and Oral Health

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Background: The purpose of this review was to investigate evidence for a possible etiological association between oral health and pneumonia or other respiratory diseases.

Methods: The following data sources were used: Ovid MEDLINE (In-Process & Other Non-Indexed Citations, Daily Update, and OLDMEDLINE); Cumulative Index to Nursing & Allied Health Literature; Evidence Based Medicine of Cochrane Central Register of Controlled Trials; Cochrane Database of Systematic Reviews; Database of Abstracts of Reviews of Effects; EMBASE; Health and Psychosocial Instruments; HealthSTAR; International Pharmaceutical Abstracts; PubMed; and Google Scholar from the earliest record until July 2005. Studies were selected from randomized controlled clinical trials and longitudinal, cohort, case-control, and epidemiological studies. Searches were limited to English language and human studies.

Results: A total of 728 articles were searched for relevancy, determined by article title, abstract, and full copy, resulting in a yield of 19 studies that met our inclusion criteria. These articles were read and scored independently by the reviewers to obtain the evidence for this review: 1) the potential risk factors for pneumonia were identified as the presence of cariogenic and periodontal pathogens, dental decay, and poor oral hygiene in five studies; 2) a weak association between periodontal disease and chronic obstructive pulmonary disease (COPD) was identified in four poor to fair studies; and 3) 10 studies were retained providing evidence that interventions aiming to improve oral health reduced the progression or occurrence of pneumonia.

Conclusions: 1) There is fair evidence (II-2, grade B recommendation) of an association of pneumonia with oral health (odds ratio [OR] = 1.2 to 9.6 depending on oral health indicators). 2) There is poor evidence of a weak association (OR <2.0) between COPD and oral health (II-2/3, grade C recommendation). 3) There is good evidence (I, grade A recommendation) that improved oral hygiene and frequent professional oral health care reduces the progression or occurrence of respiratory diseases among high-risk elderly adults living in nursing homes and especially those in intensive care units (ICUs) (number needed to treat [NNT] = 2 to 16; relative risk reduction [RRR] = 34% to 83%). *J Periodontol 2006;77:1465-1482.*

KEY WORDS

Dental plaque; oral health; oral hygiene; periodontal diseases; pneumonia; pulmonary disease, chronic obstructive.

Since the publication of the United States Surgeon General's report,[1] there has been increased interest in determining whether oral health is a risk factor for systemic diseases. One such area of interest is the link between respiratory diseases such as chronic obstructive pulmonary disease (COPD) and pneumonia, both community-acquired (CAP) and especially hospital-acquired (HAP or nosocomial pneumonia).

COPD, characterized by chronic blockage in the airflow and breathing-related problems, includes two lung diseases, chronic bronchitis and emphysema,[2] and sometimes asthma.[3] It is the fourth leading cause of death in the United States, resulting in 124,816 deaths in 2004, which represents a mortality rate of 43.3 per 100,000 individuals.[4] The main risk factor for COPD is smoking, and since 2000, the prevalence of the disease in women has surpassed that in men as more women have taken up smoking. Air pollution, secondhand smoke, history of childhood respiratory infections, genetic factors, and heredity are among other risk factors for COPD.[2]

Pneumonia, an acute condition, manifests as the "gradual onset of cough with little or no fever. Less

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common presentations are pharyngitis, laryngitis, and sinusitis. The spectrum of illness can range from asymptomatic infection to severe disease."[5]

CAP is a prevalent illness, with an incidence rate of 11.6 per 1,000 adults per year.[6] Each year, 500,000 pneumonia-related hospitalizations occur in the United States,[5] with outpatient and inpatient costs of approximately $385 million and $8.4 billion, respectively.[7]

HAP is a serious, life-threatening pneumonia that accounts for 15% of all hospital-acquired infections (diagnosed on the basis of the Centers for Disease Control and Prevention [CDC] surveillance definition of nosocomial pneumonia) and is the second most common type of nosocomial infection after those of the urinary tract.[8,9] Most nosocomial pneumonia cases occur among infants, young children, and persons older than 65 years of age, as well as persons who have had severe underlying disease, immunosuppression, depressed sensorium and/or cardiopulmonary disease, or thoracoabdominal surgery.[9] Nosocomial pneumonia incidence has been estimated to be 25 to 44 per 10,000 individuals in people >60 years of age with mortality rates of 21% to 70% in intensive care unit (ICU) patients, 0.009% in low-risk people >65 years of age, and 1% or higher in high-risk populations.[8] HAP generally occurs at least 48 hours after patients have been admitted to medical or surgical general wards.[10,11] Among the ICU patients, those being mechanically ventilated are particularly susceptible to pneumonia.[11] The latter is defined as ventilator-associated pneumonia (VAP) and is reported to be the most common hospital-acquired infection among patients requiring mechanical ventilation. The incidence for VAP is reported as high as 78%.[12] There are high morbidity and mortality rates even with adequate treatment.[13] Each case of HAP/VAP has been associated with increased health care costs of $5,800 to $20,000.[10] Although the main causative agents of CAP are *Streptococcus pneumoniae* and *Haemophilus influenzae*,[14] for HAP, the most frequently associated microbial agents are *Staphylococcus aureus* and *Enterobacter*.[7]

There are four possible routes of contamination of the lower airways by microorganisms: aspiration of oropharyngeal secretions, food, or gastric contents; inhalation of infectious aerosols; spread of infections from contiguous sites; and hematogenous spread from extrapulmonary sources of infection.[15] However, the primary mechanism of entrance of these bacteria to the lung is the aspiration of colonized secretions from the oropharynx into the upper airway, which can then be aspirated to the lower airway and adhere to the bronchial or alveolar epithelium via specific adhesion-receptor interactions (Fig. 1).[8,9,16] The process of infection is initiated through the transmission of a signal to the epithelial cell through either direct signal transduction via the adhesion receptor or the release of biologically active molecules such as lipopolysaccharides.[16]

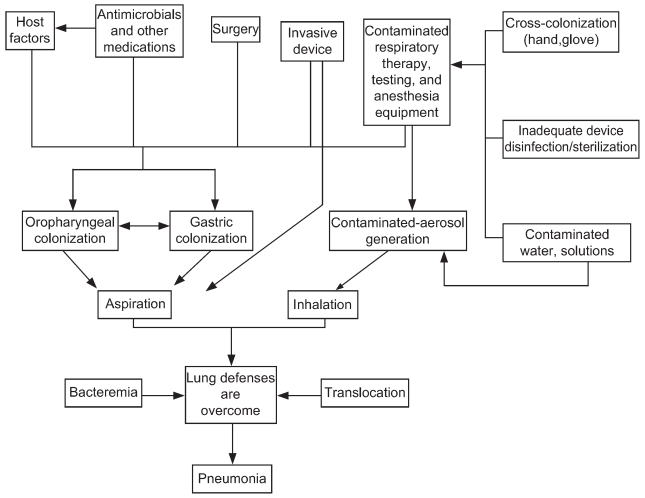

Figure 1.
Pathogenesis of nosocomial bacterial pneumonia (source: reference 9).

Aspiration pneumonia occurs in more than 200,000 cases annually, leading to more than 15,000 deaths per year in the United States.[17] It is reported that 45% of healthy adults aspirate during sleep.[9] In a healthy person, forceful coughing, active ciliary transport, and normal humoral and cellular immune mechanisms lead to the clearance of infectious material without sequelae.[14] Nevertheless, the impairment of these mechanical or cellular defense mechanisms or aspiration of large amounts of secretion (as can be seen in patients with neurologic dysphagia, disruption of the gastroesophageal junction, anatomical abnormalities of the upper aerodigestive, or swallowing dysfunction) may lead to aspiration pneumonia.[14]

PLAUSIBLE ROLE OF ORAL BACTERIA

Oral bacteria have been implicated in the occurrence of HAP. Scannapieco[18] describes four possible mechanisms of the presence of oral bacteria in the pathogenesis of respiratory infractions.

First, dental plaque could be colonized by pulmonary pathogens. Several studies have documented that the oral cavity might be a reservoir for the respiratory pathogens responsible for aspiration pneumonia in high-risk patients.[19-22] Researchers examining transtracheal aspirates from infected lung sites have provided further evidence of the involvement of periodontal organisms in aspiration pneumonia.[23-25]

Second, periodontal disease-associated enzymes may facilitate the adherence of respiratory pathogens

to the airways. Among the possibilities of this adherence are the following: 1) mucosal epithelium alteration by elevated levels of proteolytic bacteria of periodontal disease and their specific enzymes, such as mannosidase, fucosidase, hexosaminidase, and sialidase; 2) the loss of surface fibronectin, protein which coats oral mucosa and masks mucosal surface receptors; 3) the removal of surface fibronectin by hydrolytic enzymes such as salivary fibronectin; and 4) the release of cytokines.

Third, hydrolytic enzymes of periodontal disease-associated pathogens may destroy protective salivary pellicles such as mucin, resulting in fewer non-specific host defense mechanisms in high-risk subjects. Travis et al.[26] reviewed a common pathophysiologic process common to both pulmonary emphysema and periodontal disease as characterized by tissue destruction as a result of uncontrolled proteolysis of connective tissue proteins by proteinases derived from human neutrophils.

Fourth, in untreated periodontal disease, a large variety of cytokines and other biologically active molecules are continuously released from the periodontium and peripheral mononuclear cells. Upon saliva

Table 1.

Search Strategy

Category	Key Words	N Articles Found in Database Group A	N Articles Found in Database Group B	N Articles Found In Database Group C
Key words related to special patient category	Intensive care unit or ICU or nursing home or hospitalized patient or institutionalized elder or long-term care facility or susceptible individual or normal individual	219,093	85,316	
Key words related to respiratory diseases	Chronic lung disease or chronic obstructive pulmonary disease or lower RTI or bacterial pneumonia or nosocomial pneumonia or aspiration pneumonia or hospital-acquired pneumonia or HAP or community-acquired pneumonia or CAP	46,555	24,317	
Key words related to oral health	Oral health or oral hygiene or periodontal disease or gum disease or dental plaque or plaque index or oral pathogen or oral bacteria or bacterial species or fungal species or bacterial cultivation or respiratory pathogen or *S. aureus* or *Pseudomonas aeruginosa* or *Acinetobacter baumannii* or *Enterobacter cloacae*	283,837	134,665	
Combination (limited to English language, local holding, and human subject)		728	272	19
Total		1,019		
Removing the duplicates		728		
Relevant articles at the title stage		68		
Relevant articles at the abstract stage		38		
Hand searching the references for new relevant articles		6		
Scored and included articles		19		

Note: Database groups
A) Ovid MEDLINE In-Process & Other Non-Indexed Citations, Ovid MEDLINE Daily, Ovid MEDLINE and Ovid OLDMEDLINE (dating from January 1950 to July 2005), CINAHL - Cumulative Index to Nursing & Allied Health Literature (dating from 1982 to July 2005), Evidence Based Medicine (EBM) of Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects (up to 2005), EMBASE (dating from 1980 to July 2005), Health and Psychosocial Instruments (dating from 1985 to June 2005), HealthSTAR/Ovid Healthstar (dating from 1975 to June 2005), and International Pharmaceutical Abstracts (dating from 1970 to July 2005).
B) PubMed.
C) Google Scholar and World Wide Web.

aspiration in high-risk patients, these cytokines could upregulate the expression of adhesion receptors on the mucosal surface, resulting in respiratory pathogen colonization.

PREVIOUS REPORT

Scannapieco et al.[27] conducted a systematic literature review to examine whether the rate of pneumonia in high-risk populations is reduced by interventions that improve oral hygiene. They found 11 case-control and cohort studies and nine randomized controlled trials (RCTs) and performed a meta-analysis on five of these studies to determine the relationship between oral hygiene intervention and the rate of pneumonia in institutionalized patients. They found an association between periodontal disease and pneumonia and a potential association between periodontal disease and COPD in several studies. Also, they found that the incidence of nosocomial pneumonia was reduced by an average of 40% through mechanical and/or topical chemical disinfection or antibiotics.

This report set out to update the report on the question addressed by Scannapieco et al.[27] and attempts to answer the following research question: Is there evidence of an etiological association between oral health indicators and pneumonia or other respiratory diseases?

STRUCTURE OF THIS REPORT

The structure of this evidence-based report is based on the template proposed at the Royal College of Dental Surgeons of Ontario and Community Dental Health Services Research Unit Workshop[28] and covers the following areas: 1) search strategy; 2) inclusion criteria; 3) summary of evidence; 4) comparison of outcomes; 5) evidence-based recommendations and any minority views; and 6) comments or suggestions for further research.

SEARCH STRATEGY AND INCLUSION/EXCLUSION CRITERIA

The literature search for relevant articles was performed using Ovid MEDLINE In-Process & Other Non-Indexed Citations, Ovid MEDLINE Daily, Ovid MEDLINE, and Ovid OLDMEDLINE (dating from January 1950 to July 2005), Cumulative Index to Nursing & Allied Health Literature (CINAHL) (dating from 1982 to July 2005), Evidence Based Medicine of Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects (up to 2005), EMBASE (dating from 1980 to July 2005), Health and Psychosocial Instruments (dating from 1985 to June 2005), HealthSTAR/Ovid Healthstar (dating from 1975 to June 2005), International Pharmaceutical Abstracts (dating from 1970 to July 2005), and PubMed. In addition to the above, searches of the World Wide Web were conducted using Google Scholar. Table 1 shows the key words and combinations of the key words used.

At first, limiting the searches to holdings at the University of Toronto, articles in English, and human subjects retrieved 1,019 articles. After removing duplicates, 728 articles were searched for relevancy, determined by article title, resulting in a yield of 68 articles. The abstracts of these articles were independently reviewed for relevancy, resulting in a yield of 38 potential articles that were selected for retrieval. The abstraction sheets for annotated references, i.e., with citation; author/date; population and representative population; age; gender; location; intervention, or test treatment (number studied); control treatment (number studied); outcome; critical appraisal comments; conclusion; strength of evidence and classification, were printed, read, reviewed independently to determine relevance, and scored to obtain the evidence for this review. Reference lists were checked to identify any other articles relevant to the research question that may have provided additional information. Most of these were found in the original searches, but those which had not been found were retrieved. Also, four articles, not available from the holdings at the University of Toronto, were requested from the interlibrary loan services of the Faculty of Dentistry Library.

SUMMARY OF EVIDENCE

To establish a possible cause-and-effect relationship, one must consider the following questions:[29] 1) Did the cause precede the effect? 2) Was the estimate of risk beyond chance and large? 3) Was there a dose-response relationship? 4) Was reversibility demonstrated? 5) Is the cause consistently observed in different times and places? 6) Is the cause biologically plausible? 7) Is the cause specific to that disease? and 8) Is the cause analogous to another established disease/exposure?

Usually, because of ethical issues, observational studies are used to establish a causal relationship. Well-designed cohort and case-control studies are generally the standard study designs that minimize known confounding, selection, and measurement biases.[30] Although RCTs are not ordinarily used to assess a cause-and-effect relationship, well-conducted double-blinded RCTs with adequate sample size, limited or no loss to follow-up, and carefully standardized methods of measurement and analysis may establish reversibility as strong, yet not infallible,[30] evidence of a causal relationship.

Because CAP and HAP are acute diseases, one would expect a short time interval between exposure and onset of the disease, resulting in a more accurate measurement of the risk of exposure. However, due to the chronic nature of COPD and the likelihood of

Table 2.

Canadian Task Force on Preventive Health Care: Quality of Evidence and Grades of Recommendations

Quality of Published Evidence	
I	Evidence from at least one proper RCT.
II-1	Evidence from well-designed controlled trials without randomization.
II-2	Evidence from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.
II-3	Evidence from comparisons between times or places with or without the intervention. Dramatic results in uncontrolled experiments could also be included here.
III	Opinions of respected authorities based on clinical experience; descriptive studies or reports of expert committees.
Grades of Recommendations	
A	Good evidence to support the recommendation that the condition be specifically considered in a PHE.
B	Fair evidence to support the recommendation that the condition be specifically considered in a PHE.
C	Poor evidence regarding inclusion or exclusion of a condition in a PHE, but recommendations may be made on other grounds.
D	Fair evidence to support the recommendation that the condition be specifically excluded from consideration in a PHE.
E	Good evidence to support the recommendation that the condition be specifically excluded from consideration in a PHE.

Source: reference 34. PHE = periodic health examination.

repeated exposures over a long time, accurately measuring the exposure and thereby estimating the true risk of periodontal diseases for COPD is difficult.

We identified nine case-control (of which two are epidemiological studies) and cohort studies that examined the association of pneumonia or COPD with oral health indicators. Also, we found nine clinical trials that examined the efficacy of improvement of oral health indicators in reducing the incidence and occurrence of pneumonia. Of these, four were dated later than Scannapieco et al.[31-33] The best available evidence was summarized in evidence tables listing the studies in decreasing order of strength, according to the level of evidence classification system developed by the Canadian Task Force on Preventive Health Care[34] (Table 2) and checklists for appraising evidence in health care.[29]

ASSOCIATION BETWEEN PNEUMONIA AND ORAL HEALTH (TABLE 3)

Oral health was associated with pneumonia in four prospective cohort studies and one case-control study;[17,19,32,35,36] therefore, the evidence is of level II-2.

However, in two of these studies,[32,36] no bivariate analyses were reported, and in another,[35] no model fit statistics were reported, thus limiting their validity. Also, the results could not be verified from the published data in one study.[19]

The first study in the table[17] investigated the importance of medical and dental factors in relation to pneumonia among 358 older veterans who were followed for 9 years. Dental decay, presence of cariogenic bacteria, and periodontal pathogens were shown to be significant risk factors for aspiration pneumonia. The next study[35] followed 189 elderly subjects for a period of 4 years. The investigators confirmed the risk associated with decayed teeth. They also found that dependence on caregivers for oral care was associated with pneumonia. However, no fit statistics related to the logistic regression model were provided in the article.

Overall, potential risk factors for pneumonia were identified as the presence of cariogenic and periodontal pathogens in saliva and dental plaque (odds ratio [OR] = 4 to 9.6) and dental decay (OR ~1.2 per decayed tooth).[17,35,36] Higher plaque scores were also shown to be associated with a previous history of respiratory tract infection (RTI).[36]

ASSOCIATION BETWEEN PERIODONTAL DISEASE AND COPD (TABLE 4)

Oral health indicators were associated with COPD in two case-control[20,37] and two cross-sectional studies.[38,39] Ordinarily, for case-control and cross-sectional studies, the evidence is of level II-2 and II-3, respectively. However, the quality measure was lowered to II-3 in one case-control study[20] as a result of small sample size and poor control selection. All four studies in this set showed a potential association between periodontal disease and COPD. The first study in this series[37] analyzed the data from a Veterans Affairs (VA) Dental Longitudinal Study, a prospective 25-year cohort study of aging and health in male veterans who were medically healthy at the baseline. Periodontal status, as assessed by radiographic measures of alveolar bone loss (ABL), was found to be associated with an increased risk for COPD. However, ABL was only measured at the baseline, which would not likely be a good marker of the exposure to active periodontal diseases over every one of the 25 years. The next two studies[38,39] analyzed the association

Table 3.

Evidence for Assessing Causation: Pneumonia

Author, Date	Population	Cases	Non-Cases
Terpenning et al., 2001[17]	Department of Veterans Affairs outpatient clinic, inpatient ward, and nursing home. Yearly follow-up visits for dental examinations and prospective medical follow-up for development of pneumonia. Nine-year prospective study. Age: ≥55 years. All males.	N = 50 subjects with aspiration pneumonia.	N = 308 subjects who did not develop any type of pneumonia as determined by a review of medical records and chest radiographs.
Langmore et al., 1998[35]	189 subjects from the outpatient clinics, inpatient acute care medical wards, and the nursing home care center at the VA Medical Center, Ann Arbor, Michigan, followed for 4 years. Age: ≥60 years. All males.	N = 41 subjects with pneumonia.	N = 148 subjects without pneumonia.
El-Solh et al., 2004[32]	49 critically ill residents of long-term care facilities in the critical care unit, in a hospital affiliated with the University of Buffalo, State University of New York at Buffalo, Buffalo, New York. F/M gender ratio: 27/22.	N = 35 HAP patients. Mean age: 80.0 ± 6.1 years.	N = 14 non-HAP patients. Mean age: 78.4 ± 5.8 years.
Mojon et al., 1997[36]	302 frail elders living in a medical care facility from 1993 to 1995. Mean age: 85 years. F/M gender ratio: 215/87.	N = 100 residents identified from medical records as having had RTI in the year prior to the study.	N = 202
Fourrier et al., 1998[19]	57 patients admitted to the ICU during the 3-month interval between June and September 1995. Mean age: 49 ± 18 years. F/M gender ratio: 27/30.	N = 21 subjects with nosocomial infection.	N = 36 subjects without nosocomial infection.

CI = confidence interval; F = female; M = male; CA = critical appraisal; RTI = respiratory tract infection.
* Unable to confirm the given results from the published table.

Table 3. (continued)

Evidence for Assessing Causation: Pneumonia

Outcome	Critical Appraisal and Conclusion
Significant dental risk factors for aspiration pneumonia in logistic regression analysis: 1) Dentate and edentulous patients: (pneumonia [+] = 50; pneumonia [−] = 308; model sensitivity: 0.66; model specificity: 0.71; *P* value 0.12). Presence of *S. aureus* (OR = 8.3; 95% CI = 2.8 to 24.7) in the saliva. No significant association with plaque index, gingival bleeding score, number of decayed teeth, number of functional (dental) units, or the presence of other pathogens. 2) Dentate patients only: (pneumonia [+] = 28; pneumonia [−] = 190; model sensitivity: 0.82; model specificity: 0.70; *P* value: 0.10). Number of decayed teeth (OR = 1.2; 95% CI = 1.1 to 1.4, for each additional decayed tooth). Number of functional (dental) units (OR = 1.2; 95% CI = 1.02 to 1.4). The presence of *S. aureus* (OR = 7.4; 95% CI = 1.8 to 30.5) and of *Streptococcus sobrinus* (OR = 6.2; 95% CI = 1.4 to 27.5) in the saliva and the presence of the periodontal pathogen *Porphyromonas gingivalis* in dental plaque (OR = 4.2; 95% CI = 1.6 to 11.3).	Authors' conclusion: dental decay, presence of cariogenic bacteria, and periodontal pathogens identified as potentially important risk factors for aspiration pneumonia. II-2. CA score: 9/13.
Significant dental risk factors for aspiration pneumonia in different logistic regression models: 1) All subjects: (N = 189) Dependent for oral care (OR = 2.83; 95% CI = 1.08 to 7.84). Tube fed before pneumonia (OR = 3.03; 95% CI = 1.00 to 9.16). 2) Only dentate subjects: (N = 101) Number of decayed teeth (OR = 1.23; 95% CI = 1.07 to 1.41). Multiple medical diagnoses (OR = 4.93; 95% CI = 1.43 to 16.95).	Authors' conclusion: dental decay was associated with pneumonia in the dentate subjects. Logistic regression analysis, but no model fit statistics. II-2. CA score: 8.5/13.
1) Trend toward a higher incidence of HAP in colonized group (RR = 1.88; 95% CI = 0.73 to 5.2). 2) 28 of all subjects (57%) had colonization of their dental plaques with aerobic pathogens. 3) *S. aureus* accounted for the majority of the isolates (45%), followed by enteric Gram-negative bacilli (42%) and *P. aeruginosa* (13%). 4) Of the 13 isolates recovered from positive bronchoalveolar lavage fluid, nine respiratory pathogens genetically matched those recovered from the corresponding dental plaques of eight patients. 5) Patients who had dentures (N = 11) had a lower plaque index than dentate patients (1.65 ± 0.29 versus 2.28 ± 0.43; *P* <0.001) and showed a lower frequency of colonization with respiratory pathogens (27% versus 66%; *P* = 0.04).	Authors' conclusion: aerobic respiratory pathogens colonizing dental plaques may be an important reservoir for HAP in institutionalized elders. No logistic regression analysis. II-2. CA score: 7/13.
1) Dentate subjects with a history of RTI had higher plaque score in comparison to dentate subjects with no RTI (2.9 versus 2.3; *P* = 0.02). 2) The incidence of RTI in edentulous subjects was 27% and for dentate subjects was 40%; resulting in a significantly increased OR of RTI of 1.7 (95% CI = 1.1 to 2.8) in dentate subjects. 3) OR of having had RTI with the presence of one or more oral disorders (wearing a denture with a defective base or having generalized stomatitis among edentulous subjects and the presence of visible calculus, generalized gingivitis, teeth with pulpal exposures, or root-tips) was 2.5 (95% CI = 1.5 to 4.1). RTI (+) / RTI (−) / Total One or more oral health problems: 65 / 86 / 151 None: 35 / 116 / 151 Total: 100 / 202 / 302	Authors' conclusion: the association between presence of actual oral health problems and a previous experience of RTI was more noticeable in those who had poor general health or were more debilitated. Improvement of oral health might reduce the risk of RTI among dependent elderly subjects. Poor oral hygiene and the presence of potential emergency could be major risk factors for RTI among frail elderly subjects. The diagnosis was based on just clinical signs. No laboratory or x-ray test or autopsies (low sensitivity); therefore, more false negatives. No logistic regression analysis. Contingency table was derived from published data. II-2. CA score: 6.5/13.
1) Dental plaque colonization on days 0 and 5 was associated with the occurrence of nosocomial pneumonia and bacteremia (RR: 9.6; *P* <0.001).* 2) In six cases of nosocomial infection, the pathogen isolated from dental plaque was the first identified source of nosocomial infection.	Authors' conclusion: dental plaque colonization by aerobic pathogens might be a specific source of nosocomial infection in ICU patients. No logistic regression analysis. II-2. CA score: 5.5/13.

CI = confidence interval; F = female; M = male; CA = critical appraisal; RTI = respiratory tract infection.
* Unable to confirm the given results from the published table.

Table 4.

Evidence for Assessing Causation: COPD

Author, Date	Population	Cases	Non-Cases
Hayes et al., 1998[37]	1,118 men from VA Dental Longitudinal Study, a long-term study of aging and health in veterans who were medically healthy at baseline. All males.	N = 261 men with COPD. Mean age: 45.06 ± 9.7 years.	N = 857 men without COPD. Mean age: 42.18 ± 9.1 years.
Scannapieco et al., 2001[38]	13,792 subjects ≥20 years of age with at least six natural teeth selected from the cross-sectional, retrospective study of NHANES III of Americans randomly selected from 1988 to 1994.	N = 810 with COPD. Mean age: 51.2 ±17.9 years. F/M gender ratio: 506/304.	N = 12,982 without COPD. Mean age: 43.9 ± 17.7 years. F/M gender ratio: 6,821/6,161.
Scannapieco et al., 1998[39]	Data from NHANES I were analyzed. Age was evenly distributed from 25 to 74 years. >50% female.	N = 41 subjects with a confirmed chronic respiratory disease.	N = 193 subjects without a respiratory disease.
Russell et al., 1999[20]	Goldwater facility nursing (chronic care) wards of Coler-Goldwater Memorial Hospital, a 1,000-bed public hospital in New York, New York.	N = 28 elderly CCF residents. Mean age: 75.9 years. F/M gender ratio: 14/14.	N = 30 age-, gender-, and race-matched outpatient control subjects from undergraduate clinics of the New York University College of Dentistry, New York, New York. F/M gender ratio: 18/12.

CCF = chronic-care facility; CI = confidence interval; F = female; M = male; MAL = mean attachment loss; OHI = oral health index; DOC = dental outpatient control.

Table 4. (continued)

Evidence for Assessing Causation: COPD

Outcome	Critical Appraisal and Conclusion
1) The mean whole-mouth ABL scores were greater in the cases than in the control group. 2) Over the study follow-up period of 25 years, those who subsequently developed COPD had greater bone loss at baseline. RR for the association between COPD and baseline bone loss (adjusted for age, smoking, education, and height): 1) Whole-mouth bone loss scores at baseline: RR = 1.6; 95% CI = 1.2 to 2.0. 2) ABL status at baseline was an independent risk factor for COPD, with subjects in the worst population quintile of bone loss (mean ABL >20% per site) found to be at a significantly higher risk (OR = 1.8; 95% CI = 1.3 to 2.5).	Authors' conclusion: periodontal status, as assessed by radiographic measures of ABL, is associated with an increased risk for COPD. Bone loss was measured only at baseline. Exposures (ABL) measured too far in advance of disease onset. II-2. CA score: 9.5/13.
OR for the association between COPD and attachment loss (adjusted for age, gender, race and ethnicity, education, income, number of dental visits, pack-years of smoking, alcohol consumption, and diabetes mellitus): 1) Subjects with MAL ≥3.0 mm had a higher risk of COPD than those having MAL <3.0 mm (OR = 1.45; 95% CI = 1.02 to 2.05). 2) A trend was noted in that lung function appeared to diminish with increasing periodontal attachment loss.	Authors' conclusion: association between periodontal disease and COPD. No logistic regression model fit statistics. II-3. CA score: 8/13.
Logistic regression analysis (controlling for age, race, gender, and smoking): 1) Subjects having the median OHI value were 1.34× (95% CI = 1.01 to 1.77) more likely to have a chronic respiratory disease relative to those with an OHI value of 0. 2) Subjects with the maximum OHI value were 4.50× (95% CI = 1.06 to 18.99) more likely to have a chronic respiratory disease than those with an OHI value of 0.	Authors' conclusion: OHI has a residual effect on chronic respiratory disease of both practical and statistical significance. No evidence was found to support an association between the advanced periodontal destruction and respiratory disease. No model-fit statistics. II-3. CA score: 8/13.
<table><tr><th></th><th>COPD (+)</th><th>COPD (−)</th><th>Total</th></tr><tr><td>Colonization(+)</td><td>3</td><td>4</td><td>7</td></tr><tr><td>Colonization(−)</td><td>1*</td><td>20*</td><td>21</td></tr><tr><td>Total</td><td>4</td><td>24</td><td>28</td></tr></table>* Approximate value to avoid zero cell value. Oral colonization with respiratory pathogens in CCF subjects (N = 28) was associated with the presence of COPD (OR = 15; 95% CI = 1.59 to 130.23) and higher plaque scores (2.6 ± 0.4 versus 2.2 ± 0.5; *P* <0.05). Plaque scores on teeth and dentures were significantly higher in the CCF subjects than in the DOC subjects (2.3 versus 1.2; denture plaque: 1.4 versus 0.3; *P* <0.001). Colonization with respiratory pathogens: no subjects in the DOC group; 14.3% (4/28) of the CCF subjects.	Authors' conclusion: CCF patients exhibited markedly higher plaque scores than dental outpatient control subjects. Deficient dental-plaque control and the presence of COPD may be related to respiratory pathogen colonization of dental plaque in CCF residents. Contingency table for CCF subjects was derived from the published data. Small sample size from a single CCF. Not necessarily generalizable to other CCF due to, e.g., the difference among facilities, training, and demographic characteristics. No periodontal history obtained or periodontal exam undertaken. Poor control. II-3. CA score: 6/13.

CCF = chronic-care facility; CI = confidence interval; F = female; M = male; MAL = mean attachment loss; OHI = oral health index; DOC = dental outpatient control.

Table 5.

Evidence for Efficacy (reversibility)

Author, Date	Population	Intervention	Control Treatment
Bergmans et al., 2001[40]	165 patients from three ICUs from September 1994 to December 1996. ICU 1 and ICU 2 are located in the University Hospital Maastricht, Maastricht, The Netherlands; both harbor a mixed population of medical, surgical, trauma, and neurologic patients. ICU 3 is located in the University Hospital Groningen, Groningen, The Netherlands, and is a surgical and trauma ICU.	N = 87 patients. Mean age: 56.6 ± 19.0 years. F/M gender ratio: 28/59. Intervention: topical antimicrobial prophylaxis (gentamicin/colistin/vancomycin 2% in orabase, every 6 hours) in the oropharynx.	Group A: N = 78 patients were studied in the presence of test group. Placebo: orabase without antibiotics. Mean age: 58.1 ± 16.4 years. F/M ratio: 25/53. Group B: N = 61 patients were studied in an intensive care unit with no test group. Mean age: 58.7 ± 16.7 years. F/M gender ratio: 14/47.
Fourrier et al., 2000[42]	60 patients consecutively admitted in a 16-bed adult ICU in a university hospital with a medical condition suggesting an ICU stay of 5 days and requiring mechanical ventilation. Mean age: 51 ± 16 years. F/M gender ratio: 22/38.	N = 30 patients. Mean age: 51.2 ± 15.2 years. F/M gender ratio: 11/19. Intervention: dental plaque decontamination with 0.2% CHX gel, three times a day during the ICU stay.	N = 30 patients. Mean age: 50.4 ± 15.5 years. F/M gender ratio: 11/19. Placebo: standard oral care including mouth rinsing with bicarbonate isotonic serum followed by suctioning four times a day during their whole ICU stay.
Yoneyama et al., 2002[45]	366 patients from 11 nursing homes in Japan were randomly assigned to an oral care group or a no oral care group in September 1996 and were investigated for 2 years. 51 lost to follow-up (death not due to pneumonia).	N = 184 subjects. Mean age: 82.0 ± 7.8 years. F/M gender ratio: 148/36. Intervention: nurses or caregivers cleaned patients' teeth with a toothbrush for ~5 minutes after each meal or swabbed with povidine iodine (1%) without rinsing in some cases; weekly professional care such as plaque and calculus control as necessary by dentists or dental hygienists.	N = 182 subjects. Mean age: 82.1 ± 7.5 years. F/M gender ratio: 145/37. Placebo: no oral care; some patients performed toothbrushing by themselves once a day or irregularly, but none of them requested oral care from caregivers.
Fourrier et al., 2005[46]	N = 228 non-edentulous patients (January 2001 to September 2002) requiring endotracheal intubation and mechanical ventilation; anticipated length of stay ≥5 days.	N = 114. Age: 61.0 ± 14.7 years. 72.8% male. Intervention: antiseptic decontamination of gingival and dental plaque with a 0.2% CHX gel, three times a day, during the entire ICU stay.	N = 114. Age: 61.1 ± 14.9 years. 64% male. Decontamination of gingival and dental plaque with a placebo gel, three times a day, during the entire ICU stay.

Table 5. (continued)

Evidence for Efficacy (reversibility)

Outcome	Critical Appraisal and Conclusion
VAP based on chest x-ray and three out of four clinical/laboratory criteria. 1) Comparing study and control A patients: RRR = 0.66 (95% CI = 0.34 to 0.83); NNT = 5. 2) Comparing study and control B patients: RRR = 0.55 (95% CI = 0.04 to 0.79); NNT = 8.	Authors' conclusion: targeted approach to prevent colonization at this site is a very effective method of infection prevention. No randomization. II-1. CA score: 14/16.
Nosocomial pneumonia based on six criteria: 1) Overall, the rate of nosocomial infection acquired in the ICU was significantly higher in the control group (χ^2 = 5.54; P = 0.018). 2) The nosocomial infection rate and the incidence densities related to risk exposition were lower in the test group compared to the control group (18 versus 33/1,000 days in the ICU and 10.7 versus 32.3/1,000 days of mechanical ventilation; P <0.05). 3) Preventive effect of the antiseptic decontamination (OR: 0.27; 95% CI = 0.09 to 0.80); RRR = 53%. 4) Trend to a reduction of mortality, length of stay, and duration of mechanical ventilation.	Authors' conclusion: antiseptic decontamination of dental plaque with a 0.2% CHX gel decreases dental bacterial colonization and may reduce the incidence of nosocomial infections in ICU patients submitted to mechanical ventilation. Single-blinded study. I-RCT. CA score: 13/16.
Diagnosis of new pneumonia, febrile days: 1) The RRR of patients for febrile days in the OC– group compared to those in the OC+ group was 2.45 (95% CI = 1.77 to 3.40). 2) RRR of diagnosis of new pneumonia in OC+ in comparison to OC– = 0.39 (95% CI = –0.004 to 0.631); NNT = 14. 3) RRR of patients dying due to pneumonia in OC+ compared to OC– groups = 0.54 (95% CI = 0.17 to 0.75); NNT = 11. 4) RRR of diagnosis of new pneumonia in OC+ compared to OC– in dentate subjects = 0.43 (95% CI = –0.11 to 0.71); NNT = 12. 5) RRR of patients dying due to pneumonia in OC+ in comparison to OC– in dentate subjects = 0.64 (95% CI = 0.23 to 0.83); NNT = 8.	Authors' conclusion: patients receiving oral care had fewer febrile days than patients not receiving oral care. The risk of pneumonia in patients in a long-term care facility followed up for 2 years was significantly reduced in patients receiving oral care. I-RCT. CA score: 12/16.
Primary: incidence of nosocomial bacteremia, bronchitis, and VAP acquired in the ICU, calculated in each group as the sum of these three infections and expressed in percentage and per 1,000 ICU days. Secondary: the incidence of VAP and bronchitis per 1,000 days of mechanical ventilation and 1,000 days of intubation; the incidence of VAP per 1,000 days of mechanical ventilation and 1,000 days of intubation; the mortality in the ICU; the length of stay per the total Ω score and Ω-day score (Ω-day score = total Ω score ÷ N days in ICU). 1) The antiseptic CHX decontamination did not reduce the incidence of nosocomial infections (18.4% in the test group versus 17.5% in the control group). 2) No significant difference in any of the primary or secondary endpoints of the study. 3) No difference and no trend toward difference for any of the endpoints of the study between the placebo and the study group. Bacteriology: 1) Significantly lower positive dental plaque cultures in the treated group versus placebo on day 10 (29% versus 66%). 2) Highly resistant *Pseudomonas, Acinetobacter,* and *Enterobacter* species identified in late-onset ventilator-associated pneumonia and previously cultured from dental plaque were not eradicated by the antiseptic decontamination.	Author's conclusion: efficacy was insufficient to reduce the incidence of respiratory infections due to multiresistant bacteria. Gingival and dental plaque antiseptic decontamination significantly decreased the oropharyngeal colonization by aerobic pathogens in ventilated patients. Power is not mentioned. Toothbrushing was not allowed in the trial. Two-thirds of the patients were considered infected at admission to the ICU. I-multicenter RCT. CA score: 12/16.

Table 5. (continued)

Evidence for Efficacy (reversibility)

Author, Date	Population	Intervention	Control Treatment
DeRiso et al., 1996[41]	Patients from cardiovascular ICU of a tertiary care hospital undergoing coronary artery bypass grafting, valve, or other open-heart surgical procedures.	N = 173 patients. Mean age: 64.1 ± 0.86 years. F/M gender ratio: 54/119. Intervention: 0.12% CHX gluconate oral rinse.	N = 180 patients. Mean age: 63.5 ± 0.84 years. F/M gender ratio: 57/123. Placebo: similar to the base of CHX.
Pugin et al., 1991[13]	52 patients admitted to the surgical ICU unit of University Hospital of Geneva, Geneva, Switzerland.	N = 25 patients. Mean age: 45 ± 20.2 years. F/M gender ratio: 6/19. Intervention: topical application of PNV.	N = 27 patients. Mean age: 46 ± 20 years. F/M gender ratio: 7/20. Placebo: topical application of 5% dextrose solution identical to PNV in aspect.
Houston et al., 2002[33]	561 patients from St. Luke's Episcopal Hospital, Houston, Texas. Age: not mentioned.	N = 270 F/M gender ratio: 73/170. Intervention: 0.12% CHX gluconate received twice a day for 10 days postoperatively or until extubation.	N = 291 F/M gender ratio: 61/230. Placebo: phenolic mixture received twice a day for 10 days postoperatively or until extubation, tracheostomy, death, or diagnosis of pneumonia.
Adachi et al., 2002[31]	141 elderly persons needing daily care and living in two nursing homes in Tokyo, Japan. Mean age: 84 years. F/M gender ratio: 104/37. Lost to follow-up: 37%.	N = 40 Intervention: POHC as defined by mechanical cleaning with scaling with hand scalers; brushing of the teeth with an electric brush with an automatic water supply, an interdental brush, and a sponge brush.	N = 48 Placebo: no POHC; either the patient or a staff member of the nursing home continued basic oral hygiene, mainly with swabbing with a sponge brush and denture cleaning.

Table 5. (continued)

Evidence for Efficacy (reversibility)

Outcome	Critical Appraisal and Conclusion
Incidence of total RTIs: 1) RRR of the incidence of total RTIs in the CHX-treated group in comparison to the control group: 0.69 (95% CI = 0.22 to 0.88); NNT = 16. 2) RRR in the overall nosocomial infection rate: 0.65 (95% CI = 0.28 to 0.74); NNT = 12.	Authors' conclusion: oropharyngeal decontamination with CHX oral rinse reduces the total nosocomial respiratory infection rate and the use of non-prophylactic systemic antibiotics in patients undergoing heart surgery. No loss to follow-up mentioned. No baseline data of oral hygiene index or the level of Gram-negative bacteria as causative organism of nosocomial infections in patients of both groups. I-RCT. CA score: 12/16.
Pulmonary infection based on eight criteria, calculated as CPIS: 1) During the first 12 days of intubation, less tracheobronchial colonization by Gram-negative bacteria, *S. aureus*, and *S. pneumonia* in the PNV than in the placebo group (16% versus 78%; $P < 0.0001$). 2) No difference in hospital mortality. 3) Less prescription of systemic antibiotics. 4) No resistant microorganism in PNV group. 5) RRR in the PNV group versus the placebo group: 0.79 (95% CI = 0.56 to 0.91); NNT = 2.	Authors' conclusion: in these critically ill patients, topical oropharyngeal antibiotic application lowered the rate of ventilator-associated pneumonia by a factor of 5 and decreased the requirement for intravenous antibiotics. 34% overall loss to follow-up. I-RCT. CA score: 12/16.
Nosocomial pneumonia using the criteria established by the CDC: 1) 52% reduction (NS) in the overall rate of nosocomial pneumonia (4/270 versus 9/291; $P = 0.21$) in the 0.12% CHX gluconate–treated patients. 2) Cultures showed growth more often (NS) in 0.12% CHX gluconate group than in the phenolic mixture group (52/271 versus 44/291; $P = 0.19$). 3) 58% reduction (NS) in the rate of nosocomial pneumonia among patients intubated for more than 24 hours with positive microbial cultures and treated with 0.12% CHX gluconate (4/270 versus 9/291; $P = 0.21$). 4) 71% reduction in the rate of nosocomial pneumonia among patients intubated for more than 24 hours with heavy growth in the sputum samples and treated with 0.12% CHX gluconate (2/10 versus 7/10; $P = 0.02$).	Authors' conclusion: although rates of nosocomial pneumonia were lower in patients treated with 0.12% CHX gluconate than in patients treated with the phenolic mixture, the difference was significant only in those patients intubated more than 24 hours who had the highest degree of bacterial colonization. Power: 66%. I-RCT. CA score: 11/16.
Any Fevers ≥37.8°C; Prevalence of Fatal Aspiration Pneumonia; Number of *Staphylococcus* Species and *Candida albicans* in Swab Samples Feveral Month (+) / Feveral Month (–) / Total POHC (+): ~2 / ~38 / 40 POHC (–): ~4 / ~44 / 48 Total: 6 / 82 / 28 Approximate (~) value calculated from the published bar graph. 1) RR of patients for several months in the POHC (+) compared to those in POHC (–) was 0.60 (95% CI: –0.13 to 2.69), RRR: ~0.4 (95% CI: ~–1.69 to 0.86), NNT: ~30. 2) No significant difference between the two groups for the first 6 months of observation. 3) Reduction in prevalence of fever, ratio of fatal aspiration pneumonia, and numbers of *C. albicans* species, and NS reduction of number of *Staphylococcus* in samples obtained from the oral cavity after 6 months in POHC (+) versus POHC (–).	Authors' conclusion: POHC administered by dental hygienists to a group of elderly patients needing daily nursing care was associated with a reduction in prevalence of fever and fatal pneumonia. 37.5% overall lost to follow-up. Contingency table was derived from the published bar graph. I-RCT. CA score: 10.5/16.

Table 5. (continued)

Evidence for Efficacy (reversibility)

Author, Date	Population	Intervention	Control Treatment
Genuit et al., 2001[43]	All adult patients >18 years old admitted to the surgical ICU of the VA Maryland Health Care System, Baltimore, MD, over a time period of 10 months who required mechanical ventilation and followed until discharged from the hospital.	Phase 1: October 1998 to Feb 1999. N = 39. Mean age: 69.2 years. F/M gender ratio: 1/38. Intervention 1: using WP. Phase 2: March 1999 to July 1999. N = 56. Mean age: 65.7 years. All males. Intervention 2: CHX 0.12% oral rinse administered twice daily was added to the protocol, initiated on ICU admission in all intubated patients.	Control: Using the hospital and ICU databases, all patients (N = 39) requiring mechanical ventilation for ≥48 hours during the 5-month period immediately preceding the onset of this study were selected as historic controls. Mean age: 65.7 years. F/M gender ratio: 1/38.
Yoneyama et al., 1996[44]	46 subjects of a 100-bed nursing home located in the countryside in the middle part of Japan were divided into two groups: Group A: N = 21 subjects; mean age: 77 ± 7 years; F/M gender ratio: 14/7. Group B: N = 25 subjects; mean age: 79 ± 9 years; F/M gender ratio: 14/8.	Intervention: daily professional dental care by a dentist and a dental hygienist; mouth cleaning after each meal by gargling for a few minutes or swabbing with povidine iodine (1%) in some cases by nurses. The protocol was as follows: Period I (July 1, 1992 to December 31, 1992): no oral care for groups A and B. Period II (January 1, 1993 to June 30, 1993): oral care for group A but not for group B. Period III (July 1, 1993 to December 31, 1993): oral care for group B but not for group A.	Placebo: usual nursing management; no oral care.

NNT = number needed to treat; RRR = relative risk reduction; POHC = professional weekly oral health care; NS = not significant; F = female; M = male; WP = weaning protocol; OC– = non–oral care; OC+ = oral care; CPIS = clinical pulmonary infection score.

between COPD and oral health from cross-sectional studies of the National Health and Nutrition Examination Surveys (NHANES I and III). Controlling for possible confounders, an association between oral hygiene and chronic respiratory disease was identified. However, no statistics related to the fit of the logistic regression models were provided in these articles.

These three studies[37-39] found a weak association (OR/relative risk [RR] <2.0) between COPD and oral health measures.

EVIDENCE FOR REVERSIBILITY OF PNEUMONIA (TABLE 5)

Ten studies[13,31,33,40-46] were retained that examined the evidence that interventions aiming to improve oral health reduced the progression or occurrence of pneumonia. All were clinical trials and, therefore, of strong design (level I). However, three[40,43,44] were not randomized, which lowers the strength to II-1. Overall, the intervention methods examined in these studies are as follows: 1) professional dental care; 2) chlorhexidine (CHX) 0.12% mouthrinse; 3) CHX 0.2% topical gel; 4) topical application of a non-absorbable antibiotic solution (150 mg polymyxin B sulfate; 1 g neomycin sulfate; and 1 g vancomycin hydrocholoride [PNV]) in the oropharynx; and 5) topical antimicrobial prophylaxis (gentamicin/colistin/vancomycin 2% in orabase) in the oropharynx.

Among 87 ICU patients receiving topical antimicrobial prophylaxis in the oropharynx, the first study[40] found that the incidence of VAP was significantly lower compared to 78 placebo groups of orabase without antibiotics and 61 control patients receiving no treatment. The next study[42] reported on a prospective study of 30 ICU patients receiving oral decontamination with 0.2% CHX gel three times a day and 30 controls receiving mouthrinsing with bicarbonate isotonic serum followed by suctioning four times a day. They

Table 5. (continued)

Evidence for Efficacy (reversibility)

Outcome	Critical Appraisal and Conclusion
1) Mean duration of ventilation: WP versus control: a slight decrease in the incidence of VAP (11.9 versus 12.1) but 40% reduction in the median duration of mechanical ventilation (4.5 days versus 7.5 days; P <0.008) for WP. CHX + WP versus control: a significant reduction and delay in the occurrence of VAP (37% overall, 75% for late VAP; P <0.05) in CHX + WP. The median duration of mechanical ventilation in this group was similar to that of the WP group. There was no significant difference in the overall hospital or ICU length of stay between the groups. 2) Incidence of pneumonia: WP group versus control group: non-significant decrease (28.2 versus 31.3/1,000 ventilator days); WP + CHX versus control: 33% reduction (21.0 versus 31.3; P < 0.025).	Author's conclusion: improved oral hygiene via topical CHX application in conjunction with the use of a WP may be effective in reducing the incidence of VAP and the duration of mechanical ventilation in surgical ICU patients. No randomization. No blinding techniques. The treatment protocol for the control group (historic control) is not clear. II-1. CA score: 10/16.
Respiratory infection based on four markers. Febrile day, which was defined as one when body temperature rose above 37.5°C at any one of the three daily measurements. 1) Febrile days dropped in period II compared to the febrile days in period I. 2) Febrile days increased in period III compared to the febrile days in period I. 3) During oral treatment for 6 months, febrile days did not drop, but degradation of febrile days was prevented by oral care in a limited number of patients.	Author's conclusion: febrile days, but not other symptoms, were improved by oral care. Oral care associated with preventing increases in febrile days to a limited extent. Cross-over design but no washout period. No randomization/blinding. II-1. CA score: 9/16.

NNT = number needed to treat; RRR = relative risk reduction; POHC = professional weekly oral health care; NS = not significant; F = female; M = male; WP = weaning protocol; OC– = non–oral care; OC+ = oral care; CPIS = clinical pulmonary infection score.

found that 0.2% CHX gel decreased dental bacterial colonization and the incidence of nosocomial pneumonia in ICU patients submitted to mechanical ventilation.

Except for one study,[46] all studies showed that interventions reduced the incidence of pneumonia and/or the length of mechanical ventilation. However, none of the studies measured a decrease in plaque by the end of the trials, leaving us in the dark as to whether the interventions worked through reducing plaque or some other means.

One exception, the study by Fourrier et al.,[46] failed to demonstrate the efficacy of the use of a 0.2% CHX gel three times a day in reducing the incidence of VAP. However, the inclusion criteria permitted the enrollment of subjects with confirmed preexisting infections. Thus, on entry, 68% of the subjects had exacerbation of chronic bronchitis in chronic obstructive pulmonary disease and community-acquired pneumonia with acute respiratory failure. Thus, the study really demonstrated that 0.2% CHX does not reduce the incidence of nosocomial infections among those already infected.

Overall, the number needed to treat (NNT) varies from 2 to 16, and the relative risk reduction (RRR) varies from 34% to 83%. Therefore, intervention methods for reducing the colonies of respiratory pathogens in the oral cavity decreased mortality and morbidity with levels of evidence of I to II-1.

CONCLUSIONS AND EVIDENCE-BASED RECOMMENDATIONS

This report attempted to find evidence for a causal relationship between oral health and COPD and pneumonia. Based on the evidence, we made the following conclusions: 1) There is fair evidence of an association of pneumonia with oral health (II-2, grade B recommendation). The strength of the association varied from 1.2 to 9.6 depending on the oral health indicators examined. 2) There is poor evidence

supporting a weak association (OR <2.0) between COPD and oral health (II-2/3, grade C recommendation). 3) There is good evidence (I, grade A recommendation) that oropharyngeal decontamination with different antimicrobial interventions reduces the progression or occurrence of respiratory diseases (NNT = 2 to 16; RRR = 34% to 83%).

These findings are consistent with the only other systematic review by Scannapieco et al.[27] Oral hygiene and frequent professional oral health care are useful for reducing the occurrence of pneumonia among high-risk elderly adults living in nursing homes and especially those in ICUs.

Medically compromised patients in ICUs or in nursing homes, especially if they are dentate, are at risk of pneumonia, which can be prevented by oral hygiene interventions and frequent professional oral health care (I-A).

There is a necessity for oral health education and provision among the high-risk people of the community, nursing homes, and ICUs. This could result in significant cost savings in light of the economic burden of hospitalization. The ultimate goal will be improving the quality of life among the high-risk groups and decreasing hospital admissions and health care resource use.

IMPLICATIONS FOR FURTHER RESEARCH

Given that we have good evidence that oral care interventions are efficacious, the next requirement is for high-quality studies to evaluate the relative cost-effectiveness of prophylactic interventions. Following those, we would need demonstration projects to help decide on guidelines for such care and, as a special case, the appropriate interventions at the end of life. Accordingly, ethicists need to be prominent members of such investigative teams.

ARTICLES NOT INCLUDED IN THE EVIDENCE-BASED TABLES AND THE REASONS FOR REJECTION

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