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COMMENTARY

Oral biofilms, periodontitis and pulmonary infections

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Pneumonia is one of the commonest serious infections, causing significant morbidity and mortality both in healthy and debilitated subjects. Any method of preventing pneumonia should have a substantial benefit and important implications for the delivery of health care. In this issue of *Oral Diseases*, Paju and Scannapieco (2007) review whether colonization of oral or dental biofilms with respiratory pathogens is one cause of pneumonia that could be prevented by improving oral hygiene.

Cases of pneumonia are divided into two broad categories that have a different range of likely causative pathogens. Pneumonia acquired by subjects at home is called community acquired pneumonia (CAP), and is usually caused by microaspiration of nasopharyngeal commensuals such as *Streptococcus pneumoniae* and *Haemophilus influenzae* that are capable of causing pneumonia in the normal host, or by droplet spread of viral or fastidious bacterial pathogens such as *Mycoplasma pneumoniae*, *Chlamydia pneumoniae* and *Legionella pneumophila*. Hence CAP in previously relatively fit individuals is unlikely to be influenced by oral or dental biofilms. In contrast to CAP, pneumonia occurring in a patient hospitalized for another medical problem, termed hospital acquired pneumonia (HAP), is frequently caused by a range of Gram-negative enteric organisms, multi-resistant environmental bacteria such as *Pseudomonas* and *Acinetobacter* species, and *Staphylococcus aureus*. These organisms are thought to reach the respiratory tract by microaspiration from the oropharnyx (American Thoracic Society, Infectious Diseases Society of America, 2005). The term ventilator acquired pneumonia (VAP) describes HAP occurring in patients undergoing mechanical ventilation, and pneumonia developing in patients who are chronically debilitated and live in nursing home environments is often initiated by microaspiration and is caused by a range of organisms similar to HAP.

As HAP and pneumonia in nursing home residents is probably caused by microaspiration of oropharyngeal contents, the contents of the bacterial flora of the oral and dental biofilms could affect the pathogens involved and potentially be a risk factor for the development of pneumonia. Measures to improve oral hygiene are relatively simple, and it is therefore important to define the relationship between the bacterial flora of the oral cavity and pneumonia. However, in order for any association between oral biofilms and periodontitis with pneumonia to be convincing, the following should be demonstrated: (1) that pathogens causing HAP and CAP in nursing home residents are present in oral biofilms, (2) that the level of bacterial contamination of oral biofilms and severity of periodontitis is associated with an increased incidence of pneumonia, and (3) that interventions to decrease the amount of pathogenic bacteria found in oral biofilms and the severity of periodontitis result in a lower incidence of pneumonia. The review by Paju and Scannapieco (2007) is a useful summary of existing knowledge for these three requirements, all of which need further research.

The data available at present implicate oropharyngeal colonization with potentially pathogenic organisms as the source of bacteria causing HAP, whereas for CAP oral organisms only rarely cause disease (American Thoracic Society, Infectious Diseases Society of America, 2005). This difference between the aetiology of CAP and HAP probably reflects a combination of physical and bacterial factors. Greater levels of recumbence and debilitation amongst hospitalized patients and nursing home residents may result in increased microaspiraton of oral flora, impaired pulmonary clearance of aspirated material and reduced mucosal immunity in the lung. Antibiotic treatments and impaired physical and immunological defences of the oral cavity leads to the overgrowth of normal mouth flora with larger numbers of potentially pathogenic, often resistant, organisms, so that microaspiration results in the delivery of a more virulent bacterial bolus to the lungs than in a healthy individual. Which anatomical part of the oropharynx (the pharynx, oral cavity and teeth) is likely to contrib-

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ute to the development of HAP is not clear and will depend on the organisms present in each location. The pharynx is physically closest to the lower respiratory tract and is very likely to be a major source of HAP pathogens, but the contiguous nature of the upper and lower respiratory and oral tract suggests that the bacterial flora of the oral cavity and dental plaque biofilms could also be a potential source of pneumonia. Pathogenic organisms such as Gram-negative enteric bacteria, *S. aureus* and *Pseudomonas* that commonly cause HAP are found both in the oral cavity and in dental plaque, supporting these areas as a potential source of infection (Preston *et al*, 1999; El-Solh *et al*, 2004). However these data are rather limited, and further studies are required. Information is needed on the changes in numbers and types of pathogenic bacteria found in the pharynx, oral cavity and dental plaques over time in different groups of hospitalized patients, and on the relationship of these changes with the incidence and severity of oral and dental disease. In particular, using molecular epidemiology techniques it should be possible to demonstrate whether strains of pathogenic bacteria prospectively isolated from oral biofilms or dental plaques are identical to strains of the same organism isolated from the lungs in patients developing HAP. If so, this would provide powerful evidence that these sites could act as sources of bacterial pathogens that then cause HAP.

As discussed by Paju and Scannapieco, the data on the relationship between poor oral hygiene and an increased risk of pneumonia are conflicting. On balance, there probably is an association but the level of risk and exactly which patient groups are affected requires much further research. The relationship between periodontal disease and pneumonia is even less clear-cut, but the data suggest that poor dental hygiene is also associated with an increased incidence of pneumonia in hospitalized patients or nursing home residents (Paju and Scannapieco, 2007). Meta-analysis of intervention studies actually provides quite strong support for an association between poor oral hygiene and pneumonia, with treatments that improve oral hygiene decreasing the incidence of pneumonia by up to 40% (Scannapieco *et al*, 2003). This is a striking observation which, if converted into common clinical practice, could have a marked effect on mortality and morbidity resulting from HAP and potentially CAP in patients from residential nursing homes. However, there have been negative results for some interventional studies, the most effective regimens have not been defined, and there is a lack of clarity about exactly which patient groups benefit the most from oral interventions to prevent pneumonia, and as a consequence improving oral hygiene to prevent pneumonia is not routine practice. In addition, the large gaps in basic knowledge on the relationship between the potential sources of oropharyngeal infection and pneumonia discussed above make the design of intelligent interventional studies difficult. Once the microbiology has been defined properly, then randomized controlled trials of oral hygiene methods in specific patient groups can be designed. To understand fully a possible role for oral biofilms in the development of HAP, these trials should include surveillance of the effects of treatment interventions on the bacterial flora of the oropharynx.

In summary, Paju and Scannapieco have reviewed the existing patchy state of knowledge on the relationship between oral biofilms and dental hygiene with pneumonia. This is a potentially very important field as it offers the possibility that relatively simple interventions could prevent pneumonia in debilitated patients. However, considerably more detailed research in this area is required to define fully whether improving oral hygiene could significantly reduce the mortality and morbidity associated with HAP, VAP and pneumonia in nursing home residents.

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