Periodontal Disease and Diet in Domestic Pets

Cecilia Gorrel

17 Burnt House Lane, Pilley, Nr Lymington, Hants SO41 5QN, UK

ABSTRACT Periodontal disease is the most common oral condition seen in domestic pets. In addition to the discomfort caused in the affected animal, there is strong circumstantial evidence to show that a focus of infection in the mouth may cause disease of distant organs. Consequently, prevention of periodontal disease is of paramount importance for the general health and well-being of pets. The presence of plaque on the tooth surfaces is the primary cause of periodontal disease. However, the mechanisms by which disease develops are by no means fully understood. Dietary texture does have an effect on the accumulation of dental deposits and consequently on disease development and progression. Daily toothbrushing remains the single most effective means of removing plaque, thus preventing periodontal disease. On the basis of current knowledge, the best way to maintain healthy periodontal tissues in our pets is frequent toothbrushing. The use of a dental hygiene chew and/or a diet designed to reduce dental deposits can be useful adjunctive measures and should be recommended. J. Nutr. 128: 2712S–2714S, 1998.

KEY WORDS: • periodontal disease • plaque toothbrushing • dental hygiene chew

Periodontal disease is the most common oral condition seen in small animals. Indeed, it is probably the single most common disease in small animal practice (Gorrel and Robinson 1995). Periodontal disease is not a new problem; it has been identified in domestic pets for at least 70 years. Also, there is no documentation to show that the prevalence of the disease is increasing. Suggestions that the prevalence is increasing may well represent an increased awareness, rather than an increased occurrence.

Periodontal disease is a collective term for a number of plaque-associated inflammatory conditions that affect the periodontium of the tooth. Gingivitis is inflammation of the gingiva. Periodontitis is the term used when the inflammatory reactions also involve the periodontal ligament, root cementum and alveolar bone. The end result of periodontitis is loss of the tooth due to progressive destruction of its attachment apparatus.

The disease often causes discomfort to the affected animal. Moreover, there is strong circumstantial evidence that a focus of infection in the oral cavity may cause disease of distant organs (DeBowes et al. 1996). Consequently, preventing periodontal disease is important for the general health and well-being of companion animals.

PLAQUE: THE PRIMARY CAUSE OF PERIODONTAL DISEASE

The primary cause of both gingivitis and periodontitis is the presence of plaque on the tooth surfaces. Dental plaque is composed of aggregates of bacteria and their by-products, salivary components, oral debris and occasional epithelial and inflammatory cells. It accumulates rapidly on a clean tooth surface. Plaque may accumulate supragingivally, i.e., on the clinical crown of the tooth, but also below the gingival margin, i.e., in the subgingival area of the sulcus or pocket. Differences in the composition of the subgingival flora have been attributed in part to the local availability of blood products, pocket depth, redox potential and pO2. Therefore, the question whether the presence of specific micro-organisms in patients or distinct sites may be the cause or consequence of disease continues to be a matter of dispute.

Studies in which animals were fed by intubation (Egelberg 1965b) have demonstrated that the formation of dental plaque has very little to do with food debris. In a 4-y study using the Beagle dog (Lindhe et al. 1975), it was shown that with no oral hygiene, plaque accumulated rapidly along the gingival margin with gingivitis developing within a few weeks. Dogs that were fed an identical diet under identical conditions but were subjected to daily toothbrushing developed no clinical signs of gingivitis. Loss of periodontal attachment occurred at 6 mo of undisturbed plaque accumulation and then progressed slowly. By 4 y of study, the individual teeth of dogs had lost an average of ~3 mm of attachment. Loss of alveolar bone as measured in radiographs became apparent at 24 mo after the start of the study, and at the end of the 4 y was ~2 mm. In the control group, the dogs whose teeth were brushed daily showed no signs of gingivitis or periodontitis, as determined at the end of the 4 y by both clinical and radiographic examination. It should be noted that two individuals in the test group, i.e., those not receiving daily toothbrushing, did not progress from gingivitis to periodontitis.

In summary, undisturbed plaque accumulation will result in gingival inflammation within a few weeks. In some, but not all individuals, untreated gingivitis will progress to periodontitis.
However, regular removal of plaque will prevent development of gingivitis and periodontitis.

The significance of calculus. Calculus or tartar is mineralized plaque. It is not in itself thought to be an irritant; in fact, it has been shown that under certain circumstances a normal attachment may be seen between the junctional epithelium and calculus (Fitzgerald and McDaniel 1960). It has also been shown that autoclaved calculus may be encapsulated in connective tissue without causing marked inflammation (Allen and Kerr 1965). The primary consequence of calculus is that it acts as a retention surface for plaque. The consensus is that supragingival calculus per se is not directly involved in the etiology or pathogenesis of the disease, and is mainly of cosmetic significance if plaque removal is adequate (Lang et al. 1997).

PATHOGENESIS OF DISEASE

The pathogenesis of periodontal disease is by no means fully elucidated. The plaque microbiota as well as the inflammatory reactions of the host contribute to the destruction of the periodontium.

Direct injury by plaque microorganisms. Although numerous microbiological studies have been performed, the association between specific periopathogens and periodontitis remains to be conclusively proven, and it is as yet not possible to state whether the microbiota found in deep pockets are the cause or effect of periodontitis.

Plaque microorganisms produce a large variety of soluble enzymes to digest host proteins and other molecules and thereby produce nutrients for growth. Among the enzymes released by the bacteria are proteases capable of digesting collagen, elastin, fibronecin, fibrin and various other components of the intercellular matrix of epithelial and connective tissue. The microorganisms in dental plaque also release numerous metabolic products such as ammonia, indole, hydrogen sulphide and butyric acid. Periodontal disease is initiated and sustained by factors produced by the subgingival microbiota. Some of these factors can directly injure host cells and tissues.

Host response. Many microbial products have little or no direct toxic effect on the host; instead, they possess the potential to activate nonimmune and/or immune inflammatory reactions. It is these inflammatory reactions that actually cause the tissue damage.

The view that the tissue destruction is mediated by the host response to plaque rather than by direct injury by the microorganisms is supported by the findings that anti-inflammatory drugs do not affect the microbiota but still reduce attachment loss (Nyman et al 1979, Williams et al 1989), and that sterile granulation tissue degrades collagen in the absence of bacteria (Asman et al 1988).

The assumption of a specific host response is further supported by epidemiologic studies in humans (Hugosson et al 1992, Yoneyama et al 1986), which show that the frequency of moderate-to-severe periodontitis is in the order of 5–10% in most parts of the world, independent of oral hygiene measures. In areas with little oral hygiene, higher amounts of plaque and dental calculus and higher frequencies of gingivitis have been reported, but the majority of the population still do not develop periodontitis (Baelum et al 1986 and 1988). Moreover, in families with a high frequency of early onset periodontitis (Michalowicz et al 1991, van der Velden et al 1990), there is a genetic coupling, indicating that periodontitis is a patient-associated specific host response.

Nonimmune inflammatory responses. Lipopolysaccharides (endotoxins) of gram-negative microorganisms may be responsible for a spectrum of inflammatory events. They interact with and modulate the behavior of leukocytes and platelets and induce changes in endothelial cells.

It is also known that specific proteins or polysaccharides produced and released from plaque bacteria activate endogenous mediators of inflammation (Kinane and Lindhe 1997). Specific factors belonging to most categories of endogenous mediators of inflammation have been identified in gingival exudates. It is not possible at present to assign a more important role to one category of mediators compared with the others. Many different mediators are present in the lesion simultaneously and contribute to the vascular and cellular phases of the disease process. Moreover, several mediators are linked to one another via positive feedback systems that amplify their biological potentials. Indeed, most mediators have more than one inflammatory function and many have physiologic effects unrelated to their proinflammatory effects. After being released or activated, mediators are usually rapidly inactivated in the local area (Kinane and Lindhe 1997). This is an important step in the control of the inflammatory process. It is conceivable that an abnormality in one or more of these control mechanisms contributes to the development of disease.

Immune reactions. Most of the substances produced and released by plaque microorganisms are antigenic and elicit both cell-mediated and antibody-mediated immune reactions. It has been shown that both systemic and local antibody synthesis are activated in response to many plaque antigens. Local antibody synthesis by lymphoid cells may well be more relevant in gingival defense than the systemic immune response. In support of this, it has been shown that gingival exudates usually contain higher concentrations of antibodies against specific plaque organisms compared with serum from the same patient (Taichman and Lindhe 1992).

Other conditions such as physical or psychological stress (Green et al. 1986) and malnutrition (Enwonwu 1994) may impair protective responses such as production of antioxidants and acute phase proteins; as such, they can aggravate periodontitis but not cause destructive tissue inflammation per se.

Disease progression. In many instances, disease progression may be an episodic occurrence rather than a continuous process. Tissue destruction occurs as acute bursts of disease activity followed by relatively quiescent periods. The acute burst is characterized by rapid deepening of the periodontal pocket, detachment of periodontal fibers from root cementum, and loss of alveolar bone. The quiescent phase is not associated with clinical or radiographic evidence of further disease progression. The inactive or quiescent phase often lasts for extended periods of time.

The pathogenic basis of these active and inactive phases of periodontal tissue destruction are, as yet, unexplained. Theoretically, the burst may represent a transient impairment of the inflammatory response. Lowered resistance may also allow "new" organisms to infect the pocket. By contrast, it is possible that the host's inflammatory response is activated in such a way that it damages rather than protects the affected site.

In summary, it is not yet possible to give an entire account of the pathogenesis of periodontal disease. The dog has been used for the last 30 years as the experimental model for human periodontal disease. Despite this, we still do not fully understand the mechanisms involved in disease development in either species. It is now well accepted, however, that it is the host's response to the organisms rather than microbial virulence per se that actually causes the tissue damage. Further studies in dogs to gain information about the host response are required.

Effect of diet. Gross nutritional imbalances will affect the individual's ability to mount a protective inflammatory re-
response. At present, when most animals are fed nutritionally complete commercially prepared pet foods, such gross imbalances are uncommon. The main effect of diet on the initiation and progression of periodontal disease can thus be attributed to the texture of the diet, which affects accumulation of plaque.

Several studies have evaluated mechanical methods of reducing plaque accumulation via dietary texture. A coarse diet may reduce plaque accumulation on some teeth and on some tooth surfaces (Egelberg 1965a). A recent study (Logan 1996) performed over a 6-mo period that investigated oral cleansing by dietary means showed that dogs consuming a test diet (Canine t/d, Hill’s Pet Nutrition, Topeka, KS) specifically designed to promote dental hygiene accumulated significantly less plaque and calculus and developed less gingival inflammation than the control group. Similarly, the daily use of a specifically designed dental hygiene chew (Pedigree Rask/ Dentabone, Mars, McLean, VA) has been shown to reduce accumulation of dental deposits (plaque and calculus) and gingivitis in both short- and longer-term studies (Gorrel and Rawlings 1996a and 1996b, Gorrel and Bierer 1997). A study comparing the effect of feeding the test diet with that achieved by feeding a nutritionally complete dry meal supplemented with the daily addition of the dental hygiene chew, showed the two regimens to be equivalent (Rawlings and Gorrel 1997). Both regimens reduced the accumulation of dental deposits and the severity of gingivitis.

Control of periodontal disease. It has been shown by several studies that the single most effective way of removing plaque and thus preventing periodontal disease is regular, usually daily, toothbrushing (Lindhe et al 1975, Gorrel and Rawlings 1996b, Tromp et al 1986a and 1986b). As noted, other studies have evaluated mechanical methods of reducing plaque accumulation via dietary texture. However, it has yet to be demonstrated whether reducing the amount of dental plaque on a tooth via dietary texture, compared with regular removal by means of toothbrushing, will actually achieve the goal of controlling disease.

Antiplaque agents delivered from toothpastes, gels or mouthrinses can augment mechanical oral hygiene to control the formation of supragingival plaque and the development of early periodontal disease. Clinically effective antiplaque agents are characterized by a combination of intrinsic antibacterial activity and good oral retention properties. Agents that have been evaluated include chlorhexidine, essential oils, tricosan, sanguinarine, florides, oxygenating agents, quaternary ammonium compounds, substituted amino alcohols and enzymes. Of the above, the greatest effect on the reduction of plaque and gingivitis can be expected from chlorhexidine, essential oils, tricosan and substituted amino alcohols. It must be emphasized that none of these agents will prevent gingivitis on their own. Long-term studies with chlorhexidine (Hamp et al. 1973) have shown that although plaque accumulation is reduced, gingivitis, albeit at a reduced frequency, does still develop. Moreover, all of these agents are associated with adverse effects. These effects vary according to the chemical agent and include poor taste, burning sensation of oral mucous membranes, staining of teeth and soft tissues, and allergic reactions. The use of chemical antiplaque agents should be seen as adjunctive to the mechanical removal of plaque by means of toothbrushing.

SUMMARY

There is as yet no magic bullet that we can feed pets to prevent periodontal disease. Daily toothbrushing remains the single most effective way of maintaining clinically healthy gingivae. However, reduction of plaque by dietary texture is a useful adjunctive measure and should be recommended.

LITERATURE CITED


