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Dietary Influences on Periodontal Health in Dogs and Cats

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Oral disease is common in dogs and cats, and factors related to oral health are the most common diagnoses in dogs and cats of all age categories. Oral diseases can be subdivided into conditions that affect the tooth, the periodontal apparatus, or other oral tissues. Nutrition, through nutrient composition and kibble esthetics, plays a key role in tooth development, gingival and oral tissue integrity, bone strength, and the prevention and management of oral and dental diseases. Periodontal disease is the principal cause of tooth loss in dogs and cats and is the focus of this article. The major oral health success story of the past 50 years is that periodontal disease can be prevented by a combination of individual and professional measures.

ORAL ANATOMY AND FUNCTION

Wild canids and felids depend on teeth for survival. Loss of teeth results in an inability of the animal to catch and prepare food and to defend itself. Although domesticated pets need not rely on capturing prey for survival, teeth serve a variety of functions and are important in eating, grooming, defense, and behavior. Different teeth provide different functions in dogs and cats. The incisor teeth are used for grasping and nibbling. The canine teeth are used for capturing and puncturing prey. The premolar and molar teeth are used for shearing, grinding, and chewing. The carnassial teeth, designated as the upper fourth premolar and the lower first molar, are the teeth primarily used for chewing [1].

Although the teeth of dogs and cats vary in size, shape, and function, the components and structure of all teeth are similar. A normal mature tooth has a crown and one to three roots. The junction of the crown and the root is termed the *cementoenamel junction* (CEJ). The crown is the portion of the tooth above the CEJ and is covered by dense smooth enamel.

The root or roots are the portion of the tooth below the CEJ and serve to anchor the tooth in the alveolar bone as well as to provide the neurovascular

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port (apical delta). A thin layer of cementum, the calcified structure in which the periodontal fibers are embedded, covers the root.

The dentin underlies the enamel and the cementum. Dentin is primarily collagen and inorganic hydroxyapatite. There are three types of dentin: primary, secondary, and tertiary. Primary dentin is present during formation of deciduous and permanent teeth. As the animal ages, primary dentin is replaced continuously by secondary dentin. Tertiary dentin is laid down as a reparative substance–a response by odontoblasts to trauma or excessive wear. The internal layer of the tooth, the pulp cavity, contains blood and lymphatic vessels, nerves, and odontoblasts supported in a connective tissue matrix. The tissues that support and protect the tooth comprise the periodontium, including the gingiva, periodontal ligament, cementum, and alveolar bone [2].

The gingiva is an extension of the oral mucosa and consists of keratinized epithelial tissue that attaches to the alveolar process and extends to the neck of the tooth. The gingivae are divided into the attached gingiva and the free gingiva. Normal attached gingiva extends from the mucogingival line to the CEJ. Normal free gingiva surrounds the neck of the tooth without attachment. The coronal edge of the free gingiva is termed the *marginal gingiva*. The space between the free gingiva and the tooth surface is the gingival sulcus or crevice [3].

The periodontal ligament is composed of collagenous connective tissue fibers that attach the teeth to the alveolar bone. The periodontal ligament acts as a cushion, allowing slight movement of teeth during mastication to prevent trauma to teeth from occlusal and root-to-alveolar bone contact.

Thin dense alveolar bone lines the tooth socket (lamina dura) and surrounds the root, providing attachment for the periodontal ligament and passage of blood and lymphatic vessels. Alveolar bone is surrounded and supported by trabecular and compact bone, which varies in thickness depending on the anatomic location. The alveolar process is a relatively active tissue that responds to external forces and systemic influences by resorption and remodeling [4].

DENTAL FORMULAS

Dogs and cats are diphyodont, erupting two dentitions termed *deciduous* (primary or baby) teeth and *permanent* teeth. Deciduous teeth begin erupting at approximately 3 weeks of age in dogs and cats. Breed, environment, nutrition, hormones, and season may influence eruption times.

Puppies have 28 teeth, and adult dogs have 42 teeth. Normal dental formulas for dogs are as follows [5]: deciduous: 2 (I3/3, C1/1, and P3/3) and permanent: 2 (I3/3, C1/1, P4/4, and M2/3).

Kittens have 26 teeth, and adult cats have 30 teeth. Normal dental formulas for cats are as follows: deciduous: 2 (I3/3, C1/1, and P3/2) and permanent: 2 (I3/3, C1/1, P3/2, and M1/1).

Dental formulas represent teeth that should normally be present in all dogs and cats. Anatomically, the maxillary first premolars and the mandibular first and second premolars are absent in cats. Thus, feline premolars are identified

as the maxillary second, third, and fourth premolars and the mandibular third and fourth premolars [6,7].

Individual dogs and cats may have abnormal numbers of teeth. Oligodontia and supernumerary teeth occur commonly in dogs and less frequently in cats. Missing teeth may predispose to soft tissue trauma from occluding teeth and may reduce the effect of oral cleansing, particularly in the carnassial area. Extra teeth may lead to overcrowding, which affects anatomic positioning and may increase plaque retention, decrease effectiveness of dietary cleansing, and require more aggressive oral hygiene to maintain gingival health.

NUTRITION AND TOOTH DEVELOPMENT

Puppies and kittens are born edentulous; however, the nutrition that the bitch or queen receives during gestation and lactation is critical to tooth development in the offspring. Maternal nutrients must supply the pre-eruptive teeth with the appropriate building blocks for proper development and formation. After eruption, nutrient intake continues to affect tooth development and mineralization, enamel strength, and the eruption patterns of remaining teeth. Local nutritional effects influence bacterial composition and dental substrate accumulation. Throughout life, nutritional intake and dietary form affect tooth, bone, and mucosal integrity; resistance to infection; and tooth longevity. Food texture and nutritional composition can affect the oral environment through maintenance of tissue integrity, metabolism of plaque bacteria, stimulation of salivary flow, salivary composition, and contact with tooth and oral surfaces.

NUTRITION AND PERIODONTAL DISEASE

Etiopathogenesis

As the gateway to the body, the mouth is challenged by a constant barrage of invaders–bacteria, viruses, parasites, and fungi. Periodontal diseases are infections caused by bacteria in the biofilm (dental plaque) that forms on oral surfaces. Periodontal disease occurs in all mammals and is a common and potentially serious condition. Periodontal disease has been observed in adult dogs and cats of various breeds and ages. Surveys representing data from several countries report prevalence rates of periodontal disease that range from 60% to more than 80% of dogs and cats examined [8–20]. As early as 1899, Eugene Talbot [114] described "interstitial gingivitis or so-called pyorrhea alveolaris" found in dogs at necropsy. A decade later, Lund and colleagues [21] reported that dental calculus and gingivitis were the most commonly diagnosed disorders in an epidemiologic survey of 31,484 dogs and 15,226 cats across 54 veterinary practices in the United States. It is commonly reported that most adult dogs and cats older than the age of 5 years demonstrate some degree of periodontitis. Perhaps more relevant is the statement that all dogs and cats need preventive or therapeutic periodontal care.

Periodontal diseases comprise a variety of conditions affecting the health of the periodontium, the tissues that surround and support the tooth. Periodontal disease commonly refers to gingivitis and periodontitis. Gingivitis is the

reversible stage of periodontal disease and can be appropriately treated and largely prevented with thorough plaque removal and effective supragingival plaque control. Periodontitis is more severe and requires advanced therapy and meticulous plaque control to prevent progression of the disease. Periodontal disease is a "silent" disease, often progressing without overt clinical signs to the pet or the pet owner. Left untreated, periodontal disease leads to oral pain and dysfunction and eventual tooth loss. The discomfort and effect on tooth function associated with the disease may lead to many behavior changes, ranging from changes in eating habits to general behavioral changes, such as reluctance to groom and socialization or subtle signs of "depression." Emerging evidence in people and in dogs supports the critical relation between oral health and systemic health. In people, an association has been demonstrated between periodontal diseases and diabetes, cardiovascular disease, stroke, and adverse pregnancy outcomes [22–25]. In dogs, it has been reported that increased periodontal disease severity is positively correlated with histopathologic changes in the myocardium and renal interstitium [26]. It is often reported that pets "act younger" or are "more energetic" after periodontal therapy. Periodontal disease may not be considered immediately life threatening; however, there are clinically significant effects that warrant preventive and therapeutic care.

There are many risk factors associated with the prevalence and severity of periodontal disease. The primary cause results from bacterial colonization and subsequent inflammation and infection. Several materials accumulate on tooth surfaces and participate in the pathophysiology of dental and periodontal disease. These substances are commonly referred to as tooth-accumulated materials or dental substrates and include enamel pellicle, plaque, materia alba, calculus, and stain. These substrates accumulate in a dynamic process, and the influence of nutrition and diet for maintaining oral health is best understood by appreciating this continuum of periodontal disease.

Enamel pellicle is a thin film or cuticle composed of proteins and glycoproteins deposited from salivary and gingival crevicular fluids. Pellicle begins formation immediately on a cleaned tooth surface and initially provides a protective and lubricating layer. Studies have demonstrated that within minutes after polishing, approximately 1 million organisms are deposited per square millimeter of enamel surface [27]. As pellicle ages, modifications occur and additional bacterial components are incorporated, which provides a framework for bacterial colonization. The bacteria colonizing the mouth are known as the oral flora. They form a complex community that adheres to tooth surfaces in a gelatinous mat, or biofilm, commonly called dental plaque. Aggregates of bacteria combine with salivary glycoproteins, extracellular polysaccharides, and occasional epithelial and inflammatory cells to form a soft adherent plaque that covers tooth surfaces. Plaque deposits begin within 24 hours of a prophylaxis procedure. Dental plaque is a biofilm and has a specific microbial composition and structure that changes with time [28]. Supragingival plaque and subgingival plaque are distinct compositional masses that influence the inflammatory reaction of gingival tissues. There is an organized progression of

microbial colonization and growth that leads to the development of mature pathogenic dental plaque. Hundreds of bacterial species have been identified in normal and infected mouths of dogs and cats [29]. Supragingival plaque, which forms above and along the gingival margin, is composed primarily of gram-positive aerobic organisms. Growth and maturation of supragingival plaque is necessary for subsequent colonization of subgingival plaque [30]. Subgingival plaque, which forms within the gingival sulcus, is composed of gram-negative anaerobic organisms. The inflammation and destruction that accompany periodontal disease result from the direct action of bacteria and their byproducts on periodontal tissues as well as from the indirect activation of the host immune response. Bacterial plaque is the most important substrate in the development of periodontal disease [31–33]. Other soft accumulations, termed *material alba*, occur on and between the teeth and do not demonstrate the organized structure or the adherence of dental plaque. Materia alba is a soft mixture of salivary proteins, bacteria, desquamated epithelial cells, and leukocyte fragments. Additionally, dogs and cats (particularly dogs) are prone to deposition of oral debris, including impacted hair, food, and foreign materials related to chewing behaviors. Any foreign body impacted in the oral tissues provides a nidus for bacterial accumulation and incites an inflammatory reaction by the host.

Dental calculus, or tartar, is a hard mineralized shell of plaque. Existing plaque is exposed to salivary and crevicular calcium and phosphate ions and undergoes mineralization. Calculus can occur within 48 hours of plaque deposition and is also located supragingivally and subgingivally. Calculus formation is influenced by the alkalinity of the oral environment and dietary composition [34]. It has been reported that in the presence of plaque control, calculus deposits are primarily cosmetic. Although plaque bacteria are the primary cause of periodontal disease, calculus has a contributory role because of its roughened surface, which enhances bacterial attachment and further plaque development and also irritates gingival tissues. Dental stain refers to a discoloration of the tooth surface and may be extrinsic, which is discoloration of any of the previously described substrates, or intrinsic, which is discoloration of dentin or enamel because of some influence during tooth development or poster-uption injury. Dietary factors and chewing behaviors may affect dental stain. Stain is nonpathologic but is often a key signal to the pet owner of a tooth abnormality.

Bacterial-laden plaque induces inflammation in adjacent gingival tissues. Without plaque removal or control, gingivitis progresses in severity and local changes occur, allowing subsequent bacterial colonization of subgingival sites. Inflammatory mediators damage the integrity of the gingival margin and sulcular epithelium, allowing further infiltration of bacteria. The immune response of the host attempts to localize the invasion of the periodontal tissues; the result may be further destruction of local tissues because of cytokines released from inflammatory cells [35–37]. Although progression and severity are dependent on a variety of factors, periodontal disease, left untreated, leads to increased

destruction of the periodontal apparatus, resulting in tooth mobility and eventual tooth loss.

Nutrients

Nutritional factors have the potential to affect all the various oral tissues during development, maturation, and maintenance. Mature enamel is a static tissue, but nutrition may affect its growth and maturation. The periodontal apparatus surrounds, protects, and supports teeth. Any negative influences affecting these structures may progress to tooth mobility and exfoliation. Oral mucosa has a high turnover rate; adequate nutrition is necessary to maintain tissue integrity. Table 1 provides general nutrient guidelines for foods designed to prevent periodontal disease.

The role of nutrients in periodontal disease is a topic that has not received extensive or recent attention in human or veterinary medicine. Periodontal disease develops slowly, and it is likely that studies investigating the influence of nutrients on various components of periodontal disease do not reflect lifetime nutritional status. Although the amount of available information is small and dated, the following information reviews specific nutrients of concern in the occurrence and management of periodontal disease.

Specific nutrients that have been investigated, at least in part, include water, protein, soluble carbohydrates, fiber, minerals, and vitamins. Most commercial foods provide adequate levels of nutrients to prevent deficiency diseases provided that the food meets levels recommended by the Association of American Feed Control Officials (AAFCO) and adequate amounts are fed to meet daily energy requirements [38,39].

PROTEIN

Protein deficiencies cause degenerative changes in the periodontium, specifically the gingivae, periodontal ligament, and alveolar bone in laboratory animals [40]. In 1962, Ruben and coworkers [41] investigated the effects of a soft-consistency protein-deficient food on the periodontium of 22 dogs over a 1-year period. Results included inflammatory and dystrophic changes in the gingivae, periodontal ligament, and alveolar bone. The study did not

Table 1
Key nutritional factors in foods designed to promote dental health

Nutritional factor	Adult dogs	Adult cats
Protein[a]	16%–35%	30%–50%
Digestibility	>80%	>80%
Calcium[a]	0.5%–1.5%	0.5%–1.5%
Phosphorus[a]	0.4%–1.3%	0.4%–1.3%
Texture	Fiber	Fiber
Kibble size	Increased	Increased

[a]Avoid deficiency or excess.

quantify the individual effects of food consistency and protein content. Protein deficiency, however, occurs rarely in dogs and cats and is not a practical consideration as a typical cause of periodontal disease in these species.

The role of soluble carbohydrates (sugars) in the development of dental caries has been well documented in people and rodents [42]. Dental caries, however, occurs infrequently in dogs and cats. One study demonstrated that dogs do not develop carious lesions even after long periods of consuming carbohydrate-rich foods [43]. Carlsson and Egelberg [44] reported that the addition of sucrose to a soft food resulted in no difference in plaque accumulation and gingival inflammation in a group of 12 mongrel dogs. Human studies have demonstrated that larger amounts of plaque were formed when sucrose was the primary sugar consumed [45,46]. Commercial and homemade pet foods typically contain large quantities of soluble carbohydrates, usually in the form of starch.

Fiber-containing foods have long been viewed as "nature's toothbrush." It is theorized that fibrous foods (1) exercise the gums, (2) promote gingival keratinization, and (3) clean the teeth. Fiber in foods, especially as it relates to texture, has been shown to affect plaque and calculus accumulation and gingival health in dogs and cats and is discussed in this article in relation to maintaining periodontal health [47–49].

MINERALS

Foods deficient in calcium and excessive in phosphorus may lead to secondary nutritional hyperparathyroidism and significant loss of alveolar bone [50,51].

Experiments in dogs have demonstrated resorption of alveolar bone after consumption of a low-calcium high-phosphorus food [52]. It has been proposed that periodontal disease results from a nutritional deficiency of calcium, an excess of phosphorus, or both [53,54]. Svanberg and colleagues [55] reported that nutritional secondary hyperparathyroidism occurred in a group of Beagles fed a food deficient in calcium. The food did not have any effect on the initiation or rate of progression of periodontal disease when compared with findings in a control group fed a nutritionally adequate food. Although secondary nutritional hyperparathyroidism may contribute to bone loss, and thus affect the progression of periodontal disease, there is little evidence to support the theory that it is the primary cause. Calcium deficiency is essentially unheard of in dogs and cats that consume commercial pet foods containing calcium levels that meet AAFCO allowances.

A more realistic concern is the excessive levels of calcium and phosphorus present in many commercial pet foods. High levels of calcium and phosphorus are calculogenic in rats [56]. In people, the plaque deposits of heavy calculus formers contain significantly higher levels of calcium and phosphorus compared with deposits of slow calculus formers. Further research to define the role of dietary calcium and phosphorus is warranted; however, the role that these minerals have in calculus formation should be kept in mind when recommending a food as part of an oral care regimen.

Polyphosphates, such as hexametaphosphate (HMP), are sequestrants that bind salivary calcium, making it unavailable for incorporation into the plaque biofilm to form calculus [57,58]. HMP is delivered as a coating on various treats, dental chews, and foods. The purported benefits of polyphosphates are that they are released during chewing and remain in the oral cavity for prolonged periods. It has been demonstrated that the addition of HMP to the surface of baked biscuit treats, rawhide chews, and dry foods results in reduced calculus accumulation [57,59,60]. There is also evidence to support no significant differences in plaque or calculus accumulation in dogs fed dry foods plus HMP-coated biscuits [61]. Polyphosphates have no known direct effect on oral microflora populations or plaque accumulation, and an effective plaque control regimen should always be the primary recommendation for prevention or post-therapeutic care of periodontal disease. Zinc ascorbate, zinc gluconate, and other soluble zinc salts are found in a variety of oral cleansing gels, rinses, and dentifrices and have been reported to help control plaque accumulation because of their antimicrobial activity [62,63].

VITAMINS

Vitamins that have been studied in relation to periodontal disease include vitamins A, B, C, and D. Deficiencies in vitamin A have been reported to cause marginal gingivitis, gingival hypoplasia, and resorption of alveolar bone [64]. B-complex vitamin (including folic acid, niacin, pantothenic acid, and riboflavin) deficiencies have been associated with gingival inflammation, epithelial necrosis, and resorption of alveolar bone [65]. Vitamin C plays a key role in collagen synthesis. Ascorbic acid deficiencies have been reported to affect periodontal tissues adversely in people [66]. Vitamin D helps to regulate serum calcium concentrations. Vitamin D deficiencies affect calcium homeostasis and reportedly affect the gingivae, periodontal ligament, and alveolar bone [67]. Almost all commercial pet foods contain adequate levels of these vitamins. Furthermore, cats and dogs do not have a dietary vitamin C requirement because it is synthesized within the liver.

SUPPLEMENTAL INGREDIENTS

There are a variety of supplemental ingredients that have purported antibacterial or anti-inflammatory characteristics, and there are a variety of oral rinses, sprays, and additives marketed for use in pets. Many of these agents have demonstrated variable efficacy in reducing plaque bacteria and gingival inflammation when used as oral hygiene aids in people; however, comparable studies in pets are lacking. Dietary antioxidants have been reported to have beneficial effects in people with chronic inflammatory periodontal disease [68,69]. Essential oils, such as thymol, eugenol, menthol, and eucalyptol, have demonstrated efficacy in reducing plaque and gingival inflammation in human beings [70]. Chlorhexidine gluconate, a cationic bisbiguanide, has been shown to be effective in reducing plaque accumulation and gingival inflammation [71–77]. One study in dogs evaluated the addition of chlorhexidine to a dental hygiene chew

and demonstrated no significant difference in gingivitis or calculus accumulation but did report a significant reduction in plaque accumulation [78]. Xylitol, a sugar alcohol, has been associated with reduced salivary bacterial count and plaque accumulation in people [79]. Polyphenols and herbals, such as green tea, magnolol, and honokiol, have been evaluated for antimicrobial activity against caries and periodontal pathogens in people [80]. The evidence for efficacy as part of a nutritional regimen for these supplemental ingredients is lacking in dogs and cats. It is important to evaluate critically whether the addition of these agents is at a level that is safe and efficacious to provide the needed dental benefit and support the marketed claim or if the ingredients are added primarily as label dressing.

FOOD TEXTURE

Food texture and composition can directly affect the oral environment through (1) maintenance of tissue integrity, (2) alteration of the metabolism of plaque bacteria, (3) stimulation of salivary flow, and (4) cleansing of tooth and oral surfaces by appropriate physical contact. The physical consistency, or texture, of foods has long been thought to affect the oral health of dogs and cats. Historically, several studies have demonstrated that animals eating soft foods develop more plaque and gingivitis than animals fed fibrous foods [81,82]. The studies traditionally cited to substantiate those claims are old reports that used small numbers of animals, had varying evaluation methods, and did not report data analysis, making study comparison difficult. It has been considered conventional wisdom that typical dry crunchy commercial foods provide a dental benefit to cats and dogs. Many of the recommendations made about the effect of food texture on oral health are unsubstantiated, and several have turned out to be untrue when exposed to rigorous study. Although consumption of soft foods may promote plaque accumulation, the general belief that dry foods provide significant oral cleansing should be regarded with skepticism. It has been reported that a canned food performed similar to a dry food in the degree of plaque and calculus accumulation in dogs (Table 2) [83]. A large epidemiologic survey reported that in dogs, consumption of dry food alone did not consistently demonstrate improved periodontal health [84]. Plain baked biscuits have long been considered to have dental benefits. Studies in dogs have shown that plain baked biscuit treats provide little additional plaque and calculus reduction when compared with feeding dry dog food alone [82]. More recent research has clearly demonstrated that pet foods

Table 2
Comparison of moist versus dry foods on dental substrate accumulation in dogs

	Plaque index	Calculus index	Stain index
Canned A	10.12	6.28	4.37
Canned B	9.77	7.82	5.30
Dry A	10.19	6.90	5.63

can be specifically formulated and processed to provide an effective means of plaque and calculus control (Fig. 1) [89].

Many dry treats and chew aids have offered dental claims for many years, such as "promotes clean teeth, fresh breath, and healthy gums," "cleans teeth, freshens breath," or "scrubs away tartar buildup to help clean teeth and freshens breath." The AAFCO supports and recommends guidelines developed by the Center for Veterinary Medicine (CVM) of the US Food and Drug Administration for dental health claims [85]. These guidelines state that food products bearing claims to cleanse, freshen, or whiten teeth by virtue of their abrasive or mechanical action are not objectionable. Food products bearing claims for plaque or calculus reduction or for prevention or control of breath odor may be misbranded. Enforcement is a low priority for CVM and state regulatory agencies if these claims are made only with respect to the product's abrasive action, however. This has led to a wide availability of products that make some type of plaque or calculus control claim with little or no evidence to document their effectiveness. There is no single system for establishing quality evidence supporting dental claims. One method of recognizing effective products is through identification of the Veterinary Oral Health Council (VOHC) seal of acceptance. The VOHC recognizes products with proven efficacy for mechanical control of plaque or mechanical or chemical control of calculus through a data review system. Products approved by VOHC can display the VOHC seal for tartar control or plaque control on packaging and promotional materials. More information about the VOHC and a listing of products that carry the VOHC seal is available through the VOHC web site [86,87].

DENTAL FOODS

Most dogs and cats eat something everyday; thus, use of foods that provide dental benefits seems appropriate. Food texture can be an effective means of controlling dental plaque and, ultimately, periodontal disease. As a tooth penetrates a typical kibble or biscuit, the initial contact causes the food to shatter

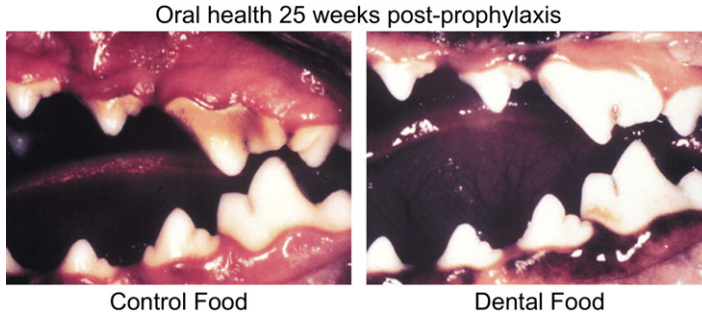

Fig. 1. Comparison of dental plaque and tartar in dogs fed a control food (Purina Dog Chow, Ralston Purina Co.) compared with a dental food (Prescription t/d Canine, Hill's Pet Nutrition, Inc.) for 6 months after dental prophylaxis. Note the extensive accumulation of plaque, tartar, and gingivitis in the oral cavity of dogs fed a control diet.

and crumble with contact only at the coronal tip of the tooth surface. To provide effective mechanical cleansing, a food should promote chewing and maintain contact with the tooth surface.

Several complete and balanced adult pet foods are available that provide significant oral cleansing compared with typical dry, moist, or snack foods. The mechanism of action for these dental foods is based on the enhanced textural characteristics and kibble size that provide mechanical cleansing of the teeth. Combining increased fiber content with a size and pattern (texture) that promotes chewing and maximizes contact with teeth is critical to obtaining a dental benefit. Numerous short-term and long-term (6-month) studies have demonstrated that dental foods with enhanced textural characteristics provide significant plaque, calculus, and stain control in cats and dogs when used after dental prophylaxis [88–95].

NATURAL DIETS

Early literature reported that the natural diets of wild canids and felids had a plaque-retardant effect and that those wild canids and felids were not afflicted with the generalized form of periodontal disease seen in domesticated pets [96]. Pet food commercialization is often implicated as a contributing factor to the increased prevalence and severity of periodontal disease in domestic dogs and cats [97]. There are no published data that compare controlled populations of domestic dogs or cats consuming a natural diet with those consuming a commercial food. Reports have demonstrated that dogs and cats fed a natural diet had varying signs of periodontal disease as well as a high rate of tooth fractures [98–100]. Anecdotal reports suggest that feeding raw meaty bones improves oral health in cats and dogs [101]. Several potential health problems exist with feeding raw foods to cats and dogs, including increased dental fractures (Fig. 2) and nutritional and public health concerns associated with exposure to bacterial pathogens [102–106].

DENTAL TREATS AND CHEW AIDS

Studies suggest that dental treats and chew aids can be used as an adjunct to other dental home care techniques. It has been reported that dogs with access to chewing materials had less calculus accumulation, gingivitis, and periodontitis than those without any enhanced chewing activity. The study did not

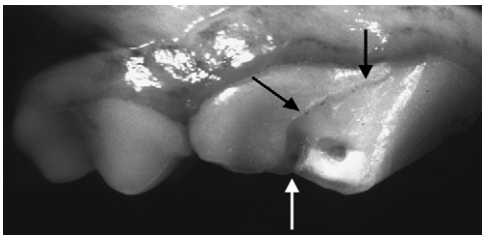

Fig. 2. Example of a slab fracture on a carnassial tooth in a dog (*arrows*). These are commonly seen as a result of chewing on excessively hard bones or treats.

measure how often or how long dogs chewed on their respective materials, however [107]. Consumption of cartilaginous materials, rawhide chew strips, and dental hygiene chews has been reported to reduce plaque and calculus accumulation and gingival inflammation [108]. Some veterinary dentists have expressed concern that hard dental treats may increase the risk for dental fractures (see Fig. 2) [109], whereas anecdotal reports of esophageal and intestinal obstruction have been associated with compressed vegetable protein chews.

DENTAL HOME CARE

Oral health is achieved through a combination of professional periodontal care as well as appropriate and effective client-provided dental home care. The primary goal of dental home care is effective plaque control that maintains oral hygiene and prevents the development of gingivitis and periodontal disease [110]. Appropriate home care recommendations that address the degree of oral pathologic change present and the ability of the client to provide frequent oral hygiene are critical. The key to effective home treatment is compliance. Compliance is a significant issue in veterinary medicine. A comprehensive study of compliance revealed that in the approximately 15.5 million dogs and cats with stage 2, 3, or 4 periodontal diseases, appropriate care was not received [111]. Reported compliance through pet owner surveys indicated that compliance with dental home care recommendations is 30% to 50% [112]. Although mechanical cleansing through frequent tooth brushing is an effective means of plaque control, compliance remains an issue. The simpler the required behavior, the more likely it is to be performed. If the complexity of the pet owner behavioral response is high, it is likely that the long-term effective compliance rate is going to be low [113]. These findings, coupled with evidence-based research, support the use of dental foods with textural characteristics as an appropriate effective means of daily plaque control and gingival health maintenance in dogs and cats.

SUMMARY

A pet cannot be healthy without oral health. Periodontal disease is a significant disease that has local and systemic ramifications. It has been stated earlier that effective plaque control prevents gingivitis. In human beings, 90% of periodontitis occurs as the result of progression of gingivitis, and this type of periodontitis can be completely prevented by plaque control. It is reasonable that dogs and cats react similarly and that effective plaque control could prevent a large percentage of periodontitis cases. Proper nutrition and effective oral hygiene are necessary components of oral health and should be jointly promoted in the management of oral disease in dogs and cats.

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