Diet and disease in companion animals

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INTRODUCTION

Canine nutrient requirements have probably changed little since dogs were first domesticated, but our knowledge and understanding of these requirements and their applications have changed dramatically (Anon 1985a). The same can be said for cats. An obvious change in application has been the trend away from home-prepared rations towards commercial pet foods, a process which has occurred rapidly over the last 30 years in Australia. Between 1967 and 1977, for example, the proportion of meals fed as commercial foods increased for dogs from 18% to 49%, and for cats from 8% to 39% (Wheatley 1977). By 1992, calories fed as commercial prepared foods were 50% of the total for dogs and 66% for cats (Anon 1992).

The convenience of pre-packaged pet food was probably a major reason for the change, but it is likely that the overall nutrition of domestic pets was improved at the same time. Although prevalences of diet-related diseases have probably altered as a result, relevant data are lacking. Assessment of this is difficult because many diseases caused by nutritional deficiencies or excesses produce low-grade signs of ill-health, and diagnoses are often difficulty to verify. However, review of diagnoses of three easily-recognised, nutrition-related diseases (nutritional secondary hyperparathyroidism in dogs and cats, hypervitaminosis A and thiamine deficiency in cats) at the Sydney University Veterinary Teaching Hospital for 1975 to 1993 showed a substantial reduction in frequency over these 19 years - the overall occurrence in the second half of the period was about one-quarter of that in the first (Figure 1).

Along with likely benefits of modern pet foods, there have been some problems. Difficulties have arisen from time to time because knowledge of the requirements for

specific nutrients is still evolving and errors can develop during the processing of foods. Several diet-related disorders have "emerged" in the last decade, including taurine deficiency retinopathy and cardiomyopathy in cats (Pion and Kittleson 1990), chronic renal disease in cats fed a diet high in protein and acid but marginal in potassium (DiBartola and others 1993), and metabolic acidosis and bone demineralization in cats fed acidifying diets for lower urinary tract disease (Dow and others 1990, DiBartola and Buffington 1993). Suspicions have also been raised about associations between acidifying feline diets and oxalate urolithiasis (Buffington 1993) and between increased feeding of canned food (or decreased dry and semi-moist foods) and feline hyperthyroidism (Scarlett and others 1988). It is interesting that problems have been associated mainly with feline diets, presumably reflecting the more specialised nutritional needs of cats and the preoccupation that some veterinarians and pet food manufacturers have with struvite crystalluria (Watson 1993).

Except for feline hyperthyroidism, these conditions have not been important in Australia. By contrast, there has been much discussion recently in this country about the relation between pet foods and the development of periodontal disease. For pragmatic reasons, it is this topic which forms the focus of the present review.

DIET AND PERIODONTAL DISEASE

The material reviewed was identified from textbooks, personal files, recent review papers, and material provided by interested colleagues, with selective back-referencing from these sources. Finally, two computer searches of the literature (CAB abstracts 1984 to 1993, Medline 1966 to 1993) were used to identify additional relevant articles. The

information presented is collated under several headings, each related to aspects of periodontal disease raised in recent discussions in the Australian veterinary community.

Periodontal disease is not a new problem in small animals

Periodontal disease has been identified as a problem in domestic pets for at least 70 years. Gray (1923) foreshadowed most of the current concerns about this condition, its causes and consequences. He noted that the disorder:

1. was worse in smaller dogs

2. progressed with age, becoming severe in middle age

3. caused loosening and loss of teeth

4. occurred in dogs fed soft diets with insufficient dental activity "in cutting and tearing raw flesh, breaking or crunching bones, and using their teeth in ratting, rabbiting, etc."

5. was very common in animals eating soft foods and that "such animals, if they live long enough, have pyorrhoea to the extent of 100%"

6. could lead to a variety of secondary diseases affecting other organs and tissues.

An even earlier study (Talbot 1899) identified periodontal disease in 75% of dogs between four and eight years of age and 25% of those one to four years old.

Periodontal disease also has a long history in the cat. Harvey and Alston (1990) found evidence of moderate or severe periodontal disease in about 25% of skulls from 80 cats (*Felis catus* and *Felis sylvestris*) that died before 1960: 75 of the skulls dated from 1841-1958 AD and two were from Pharaonic Egypt.

Periodontal disease is common in dogs and cats

Various surveys have shown that periodontal disease is common in pet dogs and cats in many areas of the world.

In the USA, Talbot (1899) identified periodontal disease as a naturally occurring disorder in 75% of an unspecified number of autopsied dogs between four and eight years of age. Amongst 125 colony Beagles, approximately 95% had heavy calculus deposits after 26 months of age, and gingival inflammation developed concomitantly (Rosenberg and others 1966). In another colony of > 2000 beagles, almost all animals had gingivitis, although only 15 were regarded as having periodontal disease (Page and Schroeder 1981). A study of 63 dogs, older than one year and anaesthetised for reasons other than oral disease, showed almost all had some degree of gingivitis and 53% had evidence of periodontitis (Golden and others 1982). In the largest North American survey, involving 1355 dogs, extensive calculus deposition was said to be common, but prevalence figures for calculus and periodontal disease were not given (Harvey 1992).

In the UK, Bell (1965) reported that 73% of 600 dogs presented as "dental cases" had periodontal disease. In Denmark, 62 mongrels, aged three months to 12 years, were examined and 97% of them had periodontal disease, soft debris and calculus - the only two unaffected were three and five months old (Gad 1968); however, the method of selection of these dogs was unstated. In Sweden, 162 randomly selected dogs, aged seven months to more than 12 years, were examined post-mortem: the prevalence of dental calculus and of periodontitis were 83% and 64% overall and both increased with age; the prevalence of periodontitis exceeded 80% in dogs six years or older (Hamp and others 1975, 1984). Meyer and Suter (1976) found 31% of 200 dogs examined in Switzerland had periodontitis, although the prevalence was higher (66%) in dogs aged 10 years or

older. Also in Switzerland, 58% of 200 cats brought to a veterinary clinic for other reasons had calculus deposits associated with periodontal disease (Schlup 1982).

In an enormous survey of pet animals in Japan, halitosis was identified in 21% of 2593 dogs and 17% of 738 cats, while calculus was noted in 38% of 2600 dogs and 33% of 737 cats (Anon 1985b). In both species calculus increased to involve >50% of individuals at three to five years and older, but about 20% remained unaffected when >10 years old. Another Japanese study involved 143 stray dogs and 108 pet dogs visiting veterinarians: periodontal disease and calculus deposition increased in both groups with age. Changes were generally more severe in strays, but in dogs aged five years or older calculus was more prevalent in pets (88%) than in strays (64%), while periodontal disease prevalences were not significantly different, at 55 and 79% respectively (Isogai and others 1989).

Similar population survey data are lacking in Australia, however, Wilson (1993) recorded that periodontal disease was present in 58% of 80 dogs and 36% of 67 cats presented for routine dental prophylaxis.

In addition to survey data, many clinicians have commented that periodontal disease is common in pet dogs and cats, and a prevalence rate of 80-85% or more is frequently mentioned, often attributed to other "sources". The age at which this rate applies is given variously as >1 year (Bennet and Pollard 1993), >2 years (Penman and Harvey 1990), >3 years (Beard 1991), >4 years (Hamlin 1991) and >5 years (Harvey 1993a). Other estimated prevalences are approaching 95% in animals >2 years (Harvey 1989) and almost 100% in dogs >5 years (Grove 1985). Though these are informal estimates, they presumably reflect the writers' conclusions based on personal experience, discussion and reading.

Some caution is warranted in assessing these surveys and estimates, because it is not always clear whether they include all forms of gingivitis, or only those forms considered part of periodontal disease. Some of the figures may overestimate the prevalence of periodontal disease.

Even so, the consensus is that gingivitis - calculus - periodontal disease is a common, ubiquitous and important problem in pet dogs and cats. However, there are several obvious gaps in the data on incidence and prevalence:

1. there is relatively little information relating to cats

2. data for Australian pets are few

3. there is no documentation on whether the prevalence is increasing - suggestions that this is so might reflect enhanced awareness rather than increased occurrence.

In contrast to the foregoing, periodontal disease may be uncommon in wild canids and felids, and suggestive evidence (alveolar bone destruction) was found in only 2% of 1157 canid jaw bone specimens examined by Colyer (Miles and Grigson 1990).

Dental plaque is a key factor in the genesis of periodontal disease

The development of periodontal disease is a complex process. Of prime importance in the aetiology is the accumulation of dental plaque and ensuing changes in the local oral microflora. The following description was summarised from several sources, which should be consulted for further details (Grove 1982, 1985, 1990, 1993; Addy and others 1992; Harvey 1993a,b; Sarkiala and others 1993).

On any clean tooth surface, as after scaling and polishing, an invisible glycoprotein layer, the *pellicle*, begins to form within a few seconds of exposure to saliva. Primary plaque-forming bacteria, especially *Actinomyces* spp and *Streptococcus*

spp which are part of the normal oral flora, adhere to the pellicle and proliferate. Within 24 hours a smooth layer of *plaque* covers the entire tooth, except where removed by natural dietary abrasion. The aerobic and facultative anaerobic plaque bacteria proliferate over the next few days, producing a rough surface to which more bacteria adhere. The mature plaque layer which forms is composed of bacteria in a matrix of glycoprotein, polysaccharide, epithelial cells, leucocytes, macrophages, lipids, carbohydrate, inorganic material and water.

As the plaque thickens and extends into the gingival sulcus, oxygen is depleted and anaerobic bacteria proliferate. Calcium salts in saliva deposited in the plaque produce *calculus* ("tartar"), which can form above and below the gingival crest (supragingival and subgingival calculus). Calculus provides a rough surface favouring accumulation and maturation of more plaque.

The development of *gingivitis* is closely related to the presence of bacterial plaque at the neck of the tooth. Under appropriate conditions, acute gingivitis can develop in about a week. Gingivitis can become chronic if plaque remains undisturbed. Gingivitis may persist without progressing into more severe disease, or *periodontitis* may ensue. Periodontitis results from the persistence of anaerobic bacteria and their products around the teeth, aided by inflammation and the immune responses of the host. The inflamed gums may become hyperplastic or undergo recession. With destruction of supporting connective tissues and loss of adjacent bone, teeth become loose and may be lost.

Periodontal disease refers to the whole process affecting the gingiva, the periodontal ligament (connective tissue between the tooth root and its socket) and the alveolar bone.

Several points are worth emphasising:

1. plaque formation occurs rapidly and is, to some extent, unavoidable

2. once plaque is deposited, it can only be removed by mechanical abrasion provided by diet, toothbrush, dental instruments or similar agents

3. periodontal disease is caused by bacterial plaque - while other factors (impaction of hair, crowding of teeth, mechanical effects of calculus, trauma to gums, mouth-breathing and mucosal drying) may contribute, the role of plaque is crucial

4. periodontal disease is not inevitable, but becomes highly likely if plaque formation is undisturbed.

Development of periodontal disease is facilitated by soft diets and impeded by hard foods

Informal observations. Many authors have reported that dogs and cats fed soft foods only, in clinical or research settings, have a high prevalence and, or, severity of periodontal disease; many also identified protection provided by incorporating some dietary component with form, texture or dimension requiring vigorous oral-dental activity during ingestion (Gray 1923, 1934; Colyer 1947; Andersen and Hart 1955; Whitney 1960; Brown and Park 1968; Attstrom and Egelberg 1971; Studer and Stapley 1973; Lindhe and others 1973, 1975; Heijl and Lindhe 1979; Penman and Harvey 1990).

Experimental studies. A number of experimental studies examined the effects of hard and soft diets on oral health in small animals. Burkwasser and Hill (1939) fed biscuits of hard consistency, or the same food ground and mixed with water, to two groups of dogs for 14 months. While numbers were small, dogs on hard food retained essentially normal teeth and gums, and those on soft food developed gingivitis, plaque and calculus. Krasse and

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Brill (1960) compared a diet requiring mastication (biscuit plus boiled bovine trachea) with the same food minced and mixed into a mush. Each was fed for one month to four dogs. With solid food, gums appeared normal and most gingival crevices remained free of bacteria, whereas with soft food gingivitis occurred, more crevices gave positive cultures, and the resulting microflora resembled that associated with periodontal disease. In another study (Ruben and others 1962), periodontal disease developed in dogs on a soft semi-synthetic diet but not in dogs given commercial dry dog food and occasional bones, however, the soft food was also low in protein, which complicated interpretation.

Egelberg (1965a) compared feeding raw whole bovine trachea with attached oesophagus, muscle and fat (plus vitamins and minerals), to the same stuff minced finely. Plaque accumulation and gingival exudation (an index of gingival inflammation) were both increased in dogs on minced food. The addition of sucrose to both diets did not modify plaque accumulation or gingival exudation (Carlsson and Egelberg 1965). In the third study in this series (Egelberg 1965b) the frequency of eating the minced food (once or five times per day) did not alter exudation or plaque accumulation. When dogs were given the same food by gastric intubation, by-passing the mouth, plaque accumulation and gingival exudation were not reduced, showing food did not need to be present in the mouth to induce these changes. On the contrary, gingival exudation tended to increase during tube feeding, suggesting that even the minimal chewing required with minced food had some cleansing or protective effect.

Another study compared Purina Dog Chow fed dry and in a ground, wet form. Dogs fed the dry form acquired less "soft debris" on their teeth, but there were no differences in gingivitis, calculus or periodontal measurements (Saxe and others 1967).

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Plaque accumulation over a four month period was about 50% less in two timber wolves fed hard dry food than in two fed a soft meat-based diet (Vosburgh and others 1982).

Zetner (1983) demonstrated the plaque-preventing effect of chewable "collagenic sticks" in 14 dogs fed soft food: plaque formation was reduced 56% in a three week period. Calculus and gingivitis were also reduced.

In a large experiment with Beagle dogs fed canned food, 20 dogs were given also 10 medium-sized regular dog biscuits each day, 20 received 10 medium-sized "tartar control" biscuits, and 20 had no biscuits (Samuelson and Cutter 1991). The teeth were cleaned initially then "tartar" accumulation was assessed weekly. Both biscuit types retarded accumulation, but the "tartar control" biscuits were more effective.

Finally, Harvey (1993c) referred to an unpublished study which showed large biscuits were more effective than small biscuits in keeping dogs' teeth clean.

At the Carnation Feline Research Centre, dry and canned foods were compared in two groups of kittens. They were eight weeks old when the experiment started, but the length of study was not stated. On dry food, gums remained healthy "with little, if any, inflammation of the gums and accumulation of tartar" (sic). Cats fed the canned product developed halitosis, gingivitis, tartar, calculus and gum recession (Studer and Stapley 1973).

In captive Amur tigers, the supplementation of a frozen meat-based diet with beef bones twice a week reduced dental plaque and calculus accumulation and improved gingival health, but bones once weekly appeared less beneficial (Haberstroh and others 1984).

A short term (two weeks) cross-over study with domestic cats showed plaque accumulation was more extensive with a soft canned diet that when hard dry food was offered (Boyce 1992).

Population surveys. Several surveys have provided data on the relation between oral health and diet. Amongst 63 dogs surveyed in the USA (Golden and others 1982), gingivitis and calculus were less common in those fed dry food, although plaque, tooth mobility, tooth loss, and periodontal disease did not differ with diet. In a health survey of 2649 dogs by Japanese veterinarians (Anon 1985b), dental calculus was significantly less common in those for which the major dietary component was dried food (34% prevalence) than in those where the major food component was home-cooked, canned or leftovers (42%). In the same survey, 745 cats were examined and calculus was significantly less common when dry food predominated (25% prevalence) than when home-cooked food did (41%). Another method of evaluating the results suggested that "dried pet food" and "leftovers" were less likely to be associated with calculus formation in dogs and cats, while "home-cooked food" and "canned dog food" were more likely associated with calculus.

An unpublished survey for the Waltham Centre for Pet Nutrition, found no differences in plaque, calculus or periodontal disease between dogs fed meat, canned food, dry food, or mixtures of all three (Borthwick 1986). However, bones reduced plaque and calculus, as did, to a lesser degree, rawhide chews and very hard biscuits (Higgins 1987). In another unpublished report from the same group (Markwell 1986), indices of oral health were worse in cats fed 30-40% dry food than in cats fed only canned food. However, the cats on dry food were older (means 5.2 and 3.3 years), which may have affected the outcome.

Harvey (1992, 1993c) reported preliminary analyses of survey data for 1350 dogs and > 700 cats anaesthetized at veterinary hospitals in the USA. More recent and rigorous analyses of the data (Harvey, personal communication) suggested that use of rawhide chews had some protective effect against aspects of periodontal disease in some areas of the mouth of dogs. Effects of bones, dry food, and fibre or nylon chew toys were less apparent and more variably distributed in the mouth. For cats, dry food tended to be associated with fewer indications of periodontal disease, while bones, rawhide chews and synthetic chew toys were without protective effect. In both species, the occurrence of periodontal disease showed strong direct association with age, and in dogs it was related inversely to body weight < 10 kg.

Hard foods can reduce established calculus accumulation

Several experiments have examined the effects of increasing the hard component of the diet in dogs and cats with oral inflammation and, or, calculus. Whitney (1960) took six Beagles with heavy calculus accumulation and subsequently fed three controls with their usual soft food and the other three with large hard dog biscuits only. After three weeks, the teeth of controls were unchanged, but most of the calculus had disappeared from one dog fed biscuits, and variable calculus loss occurred in the other two, each of which had a damaged tooth limiting mastication. When the same biscuits were fed to a group of hounds with "excellent teeth" and various degrees of calculus accumulation, all calculus was removed within two weeks.

Another study utilised Beagles in a colony where soft food was being fed and dental calculus and tooth loss were common. Four groups of dogs (six to eight dogs in each) had their daily moist kibble ration replaced at intervals by oxtail: either a whole

(900 g) or one-half an oxtail was fed once every seven or 14 days (Brown and Park 1968). Approximately 45-55% of all dogs' teeth surfaces were covered with calculus initially and approximately two-thirds of this was removed by 24 hours after the first oxtail feeding. With the once-weekly regimen, calculus accumulation dropped to about 5% of the total dental surfaces after the second week, and remained near that level subsequently. The one-half oxtail was as effective as a whole tail. Oxtail every two weeks was about 20-30% less effective. No harmful effects were observed from feeding oxtails to > 200 dogs for > 6 years.

Lage and others (1990) investigated the effects of chewing rawhide or cereal biscuits on dental calculus in 67 dogs. The dogs were maintained on dry kibbled food and given the other items as supplements. The observation period was three weeks. Chewing rawhide led to removal of supragingival calculus from teeth, although the effect was better for some teeth than others. Processed biscuits were also sometimes effective, but less so than rawhide.

Periodontal disease may lead to other illnesses

The possibility of damage to other organs and tissues as a consequence of periodontal disease has long been mooted. Gray (1923) suggested a variety of secondary complications, namely: septicaemia, gastritis, gastroenteritis, wound infections, keratitis, iritis, pharyngitis, bronchitis, pneumonia, persistent dermatoses and spinal and joint disorders.

Subsequent anecdotal reports have raised the same issue focusing mainly on possible renal, hepatic and cardiorespiratory disorders (Penman and Harvey 1990, Bennet and Pollard 1993); bacteraemia, viraemia, immune-mediated disease, immunoparesis and

multi-organ disease (Lonsdale 1993a); and miliary dermatitis, inflammatory bowel disease, feline urologic syndrome and plasma cell pododermatitis (Lonsdale 1993b).

In a recent review of potential systemic effects of periodontal disease, DeBowes (1993) listed these possibilities: bacterial endocarditis, glomerulonephritis, polyarthritis, polyvasculitis, autoimmune disorders, discospondylitis, endotoxaemia, and pulmonary disorders; the references given generally provide circumstantial evidence rather than proof of a causal relationship. However, both Harvey (1993a) and DeBowes (1993) referred to unpublished data which showed correlation between an increasing extent of periodontal disease and severity of microscopic kidney damage. Hamlin (1991) investigated 14 dogs with mitral insufficiency and chronic cough, and recovered Gram-negative bacteria from periodontal spaces and bronchial washes from three dogs, and from periodontal spaces and mitral valves of five dogs. He suggested that periodontal bacteria had seeded the airways and, or, valves, but the evidence presented is not compelling.

An association between periodontal disease and other diseases is plausible. Periodontal disease is common in older animals, which may have compromised immune systems and primary diseases of heart, lung and kidneys. Bacteraemia is known to occur in some dogs with periodontal disease: 15% of 39 dogs with periodontal disease had bacteraemia, increasing to 67% after dental manipulation (Black and others 1980). This combination could permit interaction between sepsis originating in periodontal pockets and target organs (Hamlin 1990, 1991). On the other hand, Harari and others (1991) found a similar prevalence of bacteraemia in 30 dogs with periodontal disease as in 15 healthy control dogs. In this study, only 27% of patients were bacteraemic after dental manipulation.

Evidence for a causal association between periodontal disease and other disorders is weak at present, but this may not matter in the present context. As periodontal disease is "arguably the most common disease condition seen in small animal practice" (Harvey 1989), and its effects on gums and teeth can significantly affect the health and well-being of affected individuals, there is *already* sufficient reason for concern. Proof of additional systemic effects of periodontal disease might increase the pressure to find solutions, but is not needed to justify action.

CONCLUSIONS

Changes in feeding methods for dogs and cats over recent decades have arguably improved many aspects of pet health, especially by reducing or preventing diseases associated with nutritional deficiencies and excesses. However, detailed documentation of this is difficult to achieve with the present data. The experiences of older practitioners may provide the best available guide to changes in disease prevalence.

The evolution of the commercial pet food industry has been an important development. It has helped improve dog and cat nutrition and provided convenience for their owners. Along with these benefits, however, there have been some specific problems which were quickly recognised and rectified, such as taurine deficiency in cats.

Periodontal disease is a common and serious diet-associated problem, which is related to food consistency rather than to nutritional deficiencies or excesses. While it is not known with certainty whether periodontal disease is becoming more common, it may be if pet owners are feeding more foods of softer types without adopting additional methods to maintain dental hygiene.

Because the focus of this review was diet and disease, other factors contributing to periodontal disease were not considered in detail. Involvement of other factors could explain, for example, discrepancies between individuals fed identically, and the higher prevalence of periodontal disease in small dogs.

There is sufficient evidence to implicate soft food diets in the aetiology of periodontal disease. The indications are that softer foods are inefficient in abrading plaque from the teeth, and that harder foods requiring prehension and mastication are preferable for dogs and cats, all other things being equal. There is no guarantee this will prevent periodontal disease in an individual pet, but it should help.

SUGGESTIONS AND RECOMMENDATIONS

The following points are based on indications from the available literature and may change as more information becomes available.

1. A suitable ration for dogs and cats should be nutritionally adequate and have physical qualities (texture, abrasiveness, chewiness) that will help control plaque and maintain oral health.

2. Diets consisting largely of soft foods, even if nutritionally complete, may be physically inadequate, thereby favouring development of periodontal disease.

3. Soft foods of home-prepared or commercial origin may not differ in this regard.

4. When *soft foods* form the basis of a pet's ration, additional methods are advisable to remove plaque. These could include combinations of the following:

    a. supplementing the diet with raw bones with attached meat and connective tissue - the type, size and frequency to suit the circumstances

b. replacing part of the diet by large biscuits of appropriate size, shape and texture to encourage use of teeth during ingestion

c. adding large pieces of palatable raw vegetables to encourage chewing: Emily and Penman (1990) suggested cauliflower, broccoli, cabbage or swede, flavoured with gravy. However, the value of this might be limited: eating three raw carrots thrice daily did not prevent plaque accumulation in Swedish dental students (Lindhe and Wicen 1969).

d. providing rawhide chew toys, or similar items, to promote dental, gingival and periodontal exercise

e. additional home dental care if needed, such as daily rubbing or brushing of teeth and gums, antibacterial sprays, dentrifices

f. regular oral examinations and dental procedures as necessary.

5. *Dry foods* made by pet food companies are, on balance, likely to be more effective than soft foods in removing plaque. However they are far from ideal in this regard at present and are likely to perform variably depending on the size, shape and consistency of individual pieces. Until data become available on the optimum characteristics for these products, veterinarians should make their own assessments from the animals they see.

6. *Raw meaty bones* have good physical characteristics to promote oral health, but they do not provide by themselves complete and balanced nutrition. Other food items are needed as well to provide essential nutrients.

* The frequency with which bones need to be fed to maintain clean teeth is likely to vary with the dog, the basic diet, the type of bone, and other factors. Once weekly feeding of 450 g of oxtail was adequate for

prophylaxis and therapy in Beagles on a plaque-producing diet. Once a fortnight was less satisfactory. Therefore, at least once a week is suggested, but two, three or more times per week might be better, depending on circumstances.

* The type of bone-with-meat selected may depend on the animal's capacity to handle it. A wide variety of bones may be suitable (Billinghurst 1993). Specific suggestions for cats include whole necks of chicken or turkey (Harvey 1993c) or chicken necks and wings, quail, rabbit, and whole raw fish (Lonsdale 1993a,b). Some suggestions for dogs are whole oxtail or whole trachea-oesophagus (Harvey 1993c), lamb breast or lamb vertebrae (Hungerford 1992), or chicken carcasses, whole rabbits, kangaroo tails, lamb flaps, oxtails, and chicken necks (Lonsdale 1993a).

* There is a consensus of opinion that bones are safer if given raw, rather than cooked.

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Figure 1. Proportion of total cases of three nutrition-related diseases diagnosed in the first and second halves of the period between 1975 and 1993: ▨ first half ▉ second half. The number of new canine and feline patients seen per year averaged 14% higher in the second half.

NSH = nutritional secondary hyperparathyroidism in dogs and cats (total 108 cases)

TD = thiamine deficiency in cats (total 17 cases)

HA = hypervitaminosis A in cats (total 3 cases)