MARS AND THE INDUSTRIAL PET FOOD EXPERIMENT

Primary Documentary Exhibits 1-6

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5 pages | SHA-256 ba45178447797c7e49c241bd766eb8d716fa0360df27de470a8021b97a367a86

EXHIBIT 2. Brown and Park - Control of Dental Calculus in Experimental Beagles (1968)

9 pages | SHA-256 6b3d23cfdf28c690def265cb061d26889766b6cccc40cc42994cb5788f3b04fd

EXHIBIT 3. R. Borthwick - Complete Waltham Periodontal Report (circa 1986)

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EXHIBIT 4. A. D. J. Watson - Diet and Disease in Companion Animals, AVA commissioned report (January 1994)

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EXHIBIT 5. A. D. J. Watson - Diet and Periodontal Disease in Dogs and Cats (October 1994)

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ORIGINAL COMMUNICATIONS

DENTAL DISEASE IN ANIMALS

BY SIR FRANK COLYER, *K.B.E.*, F.R.C.S.

(Continued from p. 10.)

PARODONTAL DISEASE.

The other common affection of the teeth—parodontal disease—is a chronic infection of the gingiva and adjacent tissues and is characterised by a progressive destruction of the supporting structures of the teeth. In the animal living under natural conditions the disease is rare, but it is common, far commoner than caries, in the captive and the domestic animal. The disease is seen in a wide range of captive wild animals including Marsupials, Rodents, Lemurs, Monkeys, Ungulates, Edentates, Carnivores, and Insectivores.

Localised destruction of the bone due to food packing between the teeth is seen in the wild animal in the natural state but it is uncommon and is almost entirely confined to the Apes and Monkeys. In these " pockets " the surface of the bone is smooth and signs of rarefying osteitis are seldom seen. In the skull (fig. 18), the food is still in position in the pockets.

The process in the captive animal resembles that seen in Man. The septa between the teeth are usually the first portions of the bone to give way, the whole tooth socket becoming gradually involved ; the rarefaction of the bone being generally more marked in the maxillæ than in the mandible.

Of our domestic animals the dog, cat, and horse are most liable to the disease. In the dog and cat

FIG. 18.—*Colobus badius tephrosceles* (Uganda Red Colobus).—Brit. Mus. 30.8.1.3. A skull with " pockets" between the teeth. Food is present in the pocket between the mandibular first and second molars.

FIG. 19.—*Canis familiaris* (Domestic Dog).—Royal College of Surgeons' Museum, Odontological Section. G 145.6. A skull showing the condition of the bone in an advanced stage of parodontal disease.

it starts as a marginal gingivitis due to the lodgment of soft pappy foods around the necks of the teeth ; infection spreads to the bone, which is gradually lost. An idea of the appalling state of some of these animals can be formed from the skull of a dog shown in fig. 19. The disease is mostly seen in pet animals which are often fed on a diet which makes very little, if any, demand on the teeth before it is swallowed. Cats and dogs which lead a freer life and obtain a diet more nearly approaching their natural food, are practically free from the disease.

Lesions of the periodontal membrane are common in the horse. In the examination of the series of skulls of the horse, to which reference has already been made, approximately one-third presented some degree of parodontal disease ranging from a slight destruction of the gum to the most aggravated form of the disease. The first stage of the disease is the loss of the little tag of gum at the interproximal areas. In the triangular cavity thus formed a grain of corn, a little chaffed hay, or other material may be found. The triangular cavity gradually increases in size, the periodontal membrane becomes more and more involved, and with the increase in the size of the space there is a greater accumulation of débris. The muco-periosteum becomes thickened and there is a profuse muco-purulent discharge around the teeth, the breath becoming extremely offensive. As the trouble advances the teeth tend to separate and food wedges between them ; the destruction of the bone continues ; the teeth loosen the infection spreads in the maxilla to the sinus where it sets up suppuration, and in the mandible where it causes an abscess in the bone. These various stages are illustrated in figs. 10, 20, 21, 22, 23, 24, 25.

To what cause or causes can we attribute this almost complete immunity to the disease of the wild animal in the natural state and the frequency of the disease in the captive animal and the domestic animal ?

From the fact that the disease is rare in wild animals living under natural conditions it is evident that their food is of such a character that a healthy condition of the dental tissues is maintained.

If, therefore, wild animals in captivity were

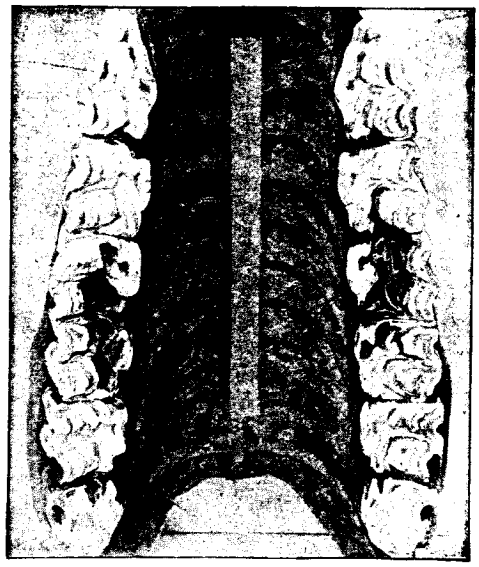

FIG. 20.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 157.01. A specimen with the muco-periosteum of the palate in position. The first stage of parodontal disease is shown in the slight destruction of the muco-periosteum between the last two molars on the right side of the illustration.

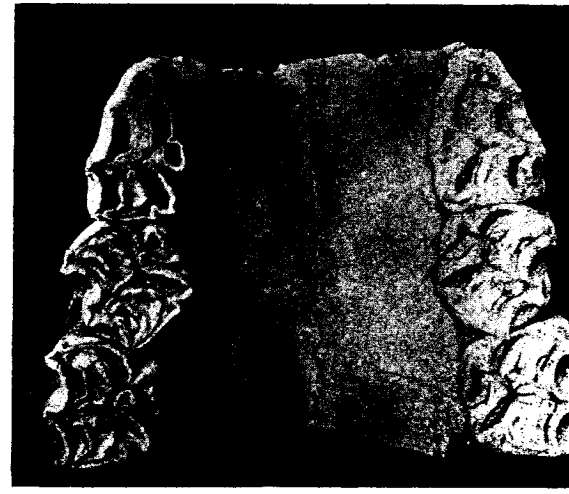

FIG. 21.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 67.41 The condition of the bone at the stage shown in fig. 20

FIG. 22.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 157.3. A specimen showing an early stage of the bone destruction in parodontal disease.

supplied with food which in character and consistency does not differ from their natural food, the healthy condition of the tissues should be maintained. But, as a matter of fact, in captivity the normal and natural food of the animal is not always forthcoming, and the environment of the teeth in these cases is altered and it is in this altered environment of the teeth due to change of diet that we must seek the cause of the disease. It may be of interest, therefore, to contrast the natural food of animals with the diet supplied to them in captivity.

Hedgehogs when kept in captivity frequently develop parodontal disease in a severe form ; in the wild state they are free from the disease. Their natural diet is a varied collection of material including "insects, worms, snails, birds' eggs, rats, mice and other small mammals, while roots and fruit are consumed to a certain extent.[1] In the captive state they are given raw minced meat with milk, as well as bread and milk. Here, although the diet in captivity is partly of a carnivorous type, the mincing has altered its physical character and in the bread we have a fermentable carbohydrate.

Rodents in captivity develop the disease in a severe form ; in the wild state they are immune. These animals in the natural state are vegetable feeders but in captivity in addition to green stuffs they are given crushed oats, potatoes, and such sticky food as biscuits.

The disease is fairly common in captive lemurs, and usually commences around the procumbent incisors. In the wild state where the diet is composed of leaves, fruit, insects, birds and birds' eggs the animals are free from the disease. In captivity they are given vegetables, fruit, dates, and cooked potatoes.

Mongooses and Suricates seem particularly liable to develop the disease. The food of the mongoose in nature is varied. It lives upon rats, mice, snakes and lizards, birds and insects, but at times eats fruit. The Suricates "feed chiefly on succulent bulbs which they scratch up with the long claws on their forefeet." In the captive state Mongooses and Suricates are given minced and small chunks of raw or cooked meat and bananas. Mincing and cooking the meat renders it more liable to cling about the teeth.

The Suricate affords an excellent illustration of the relation between the arrangement of the teeth and the development of the disease. In this animal and its allies the mandible is affected at an earlier stage than the maxilla. The external surfaces of the cheek teeth in the maxilla present an even and regular line, but in the mandible the teeth overlap slightly. Food is, therefore, more likely to lodge around the mandibular teeth than about the maxillary teeth. Suricates in the wild state are immune from the disease. It is clear that in the wild state an irregular arrangement of the teeth is not in itself harmful to the animal, at any rate as long as the diet is of a suitable character and does not cling about the teeth.

FIG. 23.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 157.4. A specimen showing parodontal disease. The bone destruction has advanced sufficiently to loosen the attachment of the first molar.

[1] The data relating to the food of animals in the wild state is taken from the Royal Natural History.

In the case of the Wolf, Fox, Racoon, Coati and other animals developing parodontal disease in captivity it is the same story, a different form of diet in the captive to that obtained in the natural state.

The diet of our domestic animals is often quite different from their natural food. Cats, particularly pet animals, instead of a purely flesh diet obtain food which has a tendency to cling about their teeth and start a marginal gingivitis. Cats which lead a freer life and obtain a diet more nearly approaching their natural food are practically free from the disease. Wild cats with their natural diet of fresh flesh are free from the disease. The dogs affected with parodontal disease are those fed on soft pappy food ; those fed on a diet which necessitates the use of their teeth for the rending of their food are free from the disease. In five skulls of foxhounds between the ages of four and six years from a well-known pack there was definite evidence of parodontal disease. The master of the hunt informed me that the bulk of the pack was only used during the prime of life ; when they were four to six years of age they often became too slow, and by that time their teeth had loosened and in many cases fallen out. The diet of these animals was Scotch oatmeal and molassine hound meal boiled together in a cauldron and, when set and cold, cut up

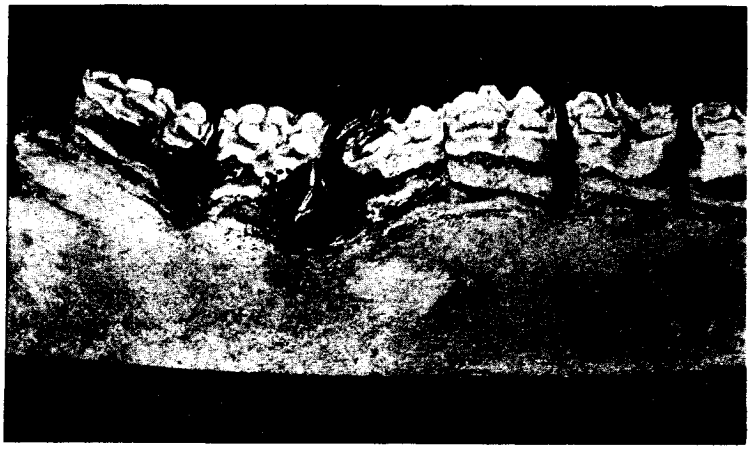

FIG. 24.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 157.53. A specimen of parodontal disease showing spaces between the teeth filled with fodder.

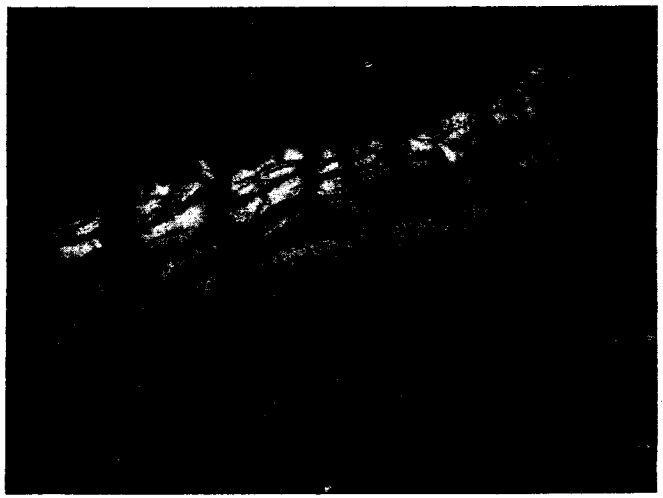

FIG. 25.—*Equus caballus* (Horse).—Royal College of Surgeons' Museum, Odontological Section. G 157.6. A specimen showing suppuration in the body of the mandible from a case of parodontal disease.

with a spade. He stated that care was taken that no bones were served, as the hounds will fight to death over a bone. Here we have a diet entirely different from the natural food of dogs and one likely to cling about the teeth.

That the physical character of the food is an important factor in the production of the disease is well exemplified in the case of the horse. In the wild state the horse lives on grass which it breaks off in long strands which lie across the cheek teeth; and in the process of mastication no damage ensues to the soft tissues. To obtain more work out of the horse it is often fed intensively. The hay, straw, etc., are cut into short lengths (chaff). The short lengths of chaff are apt to cause injury to the muco-periosteum by being pressed up into the interproximal spaces during mastication. When a cavity, however small, has once been formed, food tends to clog it, with the inevitable result that the cavity increases in size. The cavities become filled with food débris, and to this condition infection is added. The disease in the horse is thus to be traced to an alteration in the physical character of the food rather than to an alteration in the composition of the food. This view of the causation of the disease in horses is supported by the fact that the disease is prevalent wherever the conditions are such that fragments of thorns, etc., become mixed with the food. Major Kirby[1] informed me that he had seen many cases of "alveolar periodontitis" in England and other countries. The disease he found was caused "largely by the awns of worthless grasses in hay of inferior quality, e.g. brome grass, false brome, barley grass, couch grass, etc." In India he had seen it from the use of spear grass, and in South Africa from stik grass. In America needle grass or porcupine grass causes the same condition.

Horses running almost wild in countries where there are prolonged droughts, according to G. Thomson,[2] die more from parodontal disease than actual starvation. The horses dig up roots, and when chewing these, grit and sand get between the gum and teeth and set up disease.

Conclusion.

From the facts given above it is evident that caries and parodontal disease are always associated with an alteration in the physical or chemical character of the diet of the animal—in other words with a departure from natural diet and conditions. If this is the case with the lower animals there is no reason why the same should not be true of the animal Man. Expressed in biological terms caries and parodontal disease are due to an alteration in the environment of the teeth.

Correction.—Figs. 14 and 16 in the first part of Sir Frank Colyer's Lecture, pp. 9 and 10, *B.D.J.*, Jan. 3, 1947, were unfortunately transposed.

[1] Private communication.

[2] *Transactions Odontological Society*, 1905-06, xxxviii, 78.

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CONTROL OF DENTAL CALCULUS IN EXPERIMENTAL BEAGLES[1]

M. GILBERT BROWN AND JAMES F. PARK[2]

SUMMARY • *Dental calculus was a major cause of secondary tooth loss in beagle dogs housed in runs and cages and fed soft diets. Dental calculus was reduced when the diet was supplemented with one-half oxtail weekly. Four groups of 6–8 dogs were fed oxtails at one- and two-week intervals to determine the amount and frequency of feeding oxtails necessary for the control of calculus formation. Approximately 85% of the accumulated dental calculus was removed from the surface of the teeth after four-oxtail feedings. Approximately 35% of the individual teeth of the dogs showed calculus deposits after the test period, compared to 85% before oxtails were fed. The test confirmed the feasibility of preventing the accumulation of dental calculus in experimental beagle dogs by regular weekly feedings of oxtails.*

Experimental beagle dogs, in controlled housing, fed soft diets tend to accumulate heavy deposits of dental calculus. Dental calculus (tartar) is the major cause for loss of secondary teeth in dogs. Gingival ulcers form and cause the gum to recede from the teeth which can cause alveolar periostitis and loosening and loss of the teeth (*3*). The calculus is composed of calcium salts, epithelial cells, bacteria, and food debris. It collects on the teeth in a soft, light-grey film. As calculus accumulates, the under layers change to a hard, brown, brittle compound. Calculus is either supragingival or subgingival, the latter being harder and darker in color, and held to the teeth by mucin (*2*).

Manual removal of calculus is a laborious process, usually requiring anesthesia, which may interfere with the experimental protocol. Whitney (*4*) stated that hard biscuits fed exclusively as the dogs only diet for 3 weeks removed the heavy tartar accumulation on the molars and most of the staining on the canine teeth. Andersen and Hart (*1*) reported that regular feeding of oxtails to dogs decreased calculus accumulation. Oxtail feeding in our colony confirmed its therapeutic and prophylactic value. However, it was necessary to determine the amount of oxtails and frequency of feeding required to obtain optimum effectiveness at minimal cost.

METHODS

Twenty-six, 4–5-year old female beagles were randomly separated into 4 groups of 6–8 dogs. The dogs were not given the regular oxtail diet for 5 weeks prior to the test. Group I was fed one-half oxtail and Group III, 1 oxtail, at 7-day intervals. Group II was fed one-half oxtail and Group IV, 1 oxtail at 14-day intervals. All groups were examined for calculus deposits before and after each oxtail feeding. Groups I and III were examined 7 days after the final oxtail feedings and Groups II and IV were examined 14 days after the final oxtail feedings. Tooth charts were scored from visual observations before and after chewing oxtails. Each dog's teeth were observed for the percent of total teeth surface covered by dental calculus (Fig. 1) and the percent of teeth with calculus (Fig. 2). All calculations were based on the tooth charts (Figs. 3–7).

The standard diet was a moist kibble diet fed once daily. No kibble diet was fed on the day oxtails were fed. Frozen, U. S. Department of Agriculture inspected oxtails, weighing approximately 2 pounds each were

[1] This paper is based on work performed under United States Atomic Energy Commission Contract AT(45-1)-1830.
[2] Biology Department, Battelle Memorial Institute, Pacific Northwest Laboratory, Richland, Washington 99352.

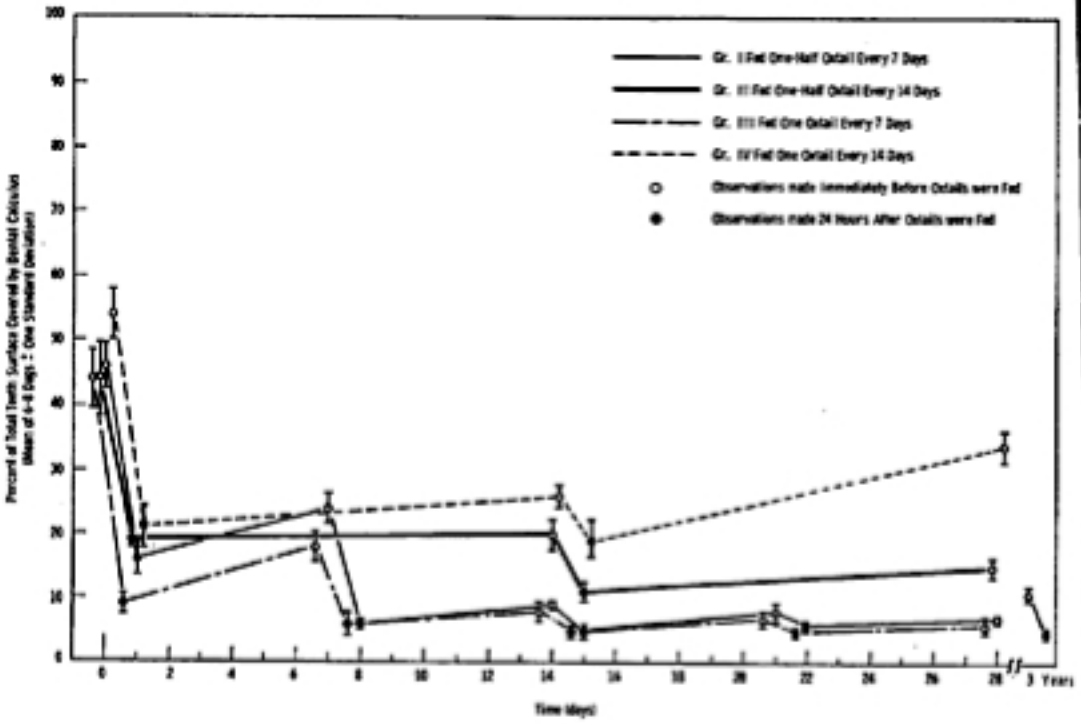


Fig. 1. *Fraction of dental calculus removed by oxtail feeding.*

fed raw. The dogs had access to the oxtails for 24 hours.

Results

Approximately 45–55% of all dogs' teeth surfaces were covered with dental calculus at the beginning of the test period, before oxtails were fed. Approximately two-thirds of the accumulated calculus was removed 24 hours after the first oxtail feeding (Fig. 1). Figure 3 shows the calculus accumulated and the amount remaining on the teeth of 1 of the dogs in Group II 24 hours after the first one-half oxtail was fed. The premolars and molars were almost completely covered with calculus before the oxtail feeding. Twenty-four hours after one-half oxtail feeding, the canines, incisors, and premolars showed some calculus deposition remaining at the gingival line. The chart indicates, however, that the molars were almost completely clean and the calculus deposits on the incisors, canines, and premolars were greatly reduced.

In Groups I and III, calculus accumulation dropped to approximately 5% of the total teeth surfaces after the second oxtail feeding and remained about the same throughout the test period. Figures 4 and 5 show the calculus deposits on the teeth of representative dogs from Groups I and III before oxtails were fed and at the end of the test period. In both groups, calculus was removed effectively when dogs were fed one-half or more oxtails once a week. The molars and premolars were almost completely free from calculus deposits, and the amount on the canines and incisors was greatly reduced at the end of the test period. In a dog from Group I, the tooth chart indicated the loss of a loose first molar, covered with calculus, in the left maxilla after the second feeding of oxtails.

In Groups II and IV, calculus accumulation dropped to about 15% after the sec-

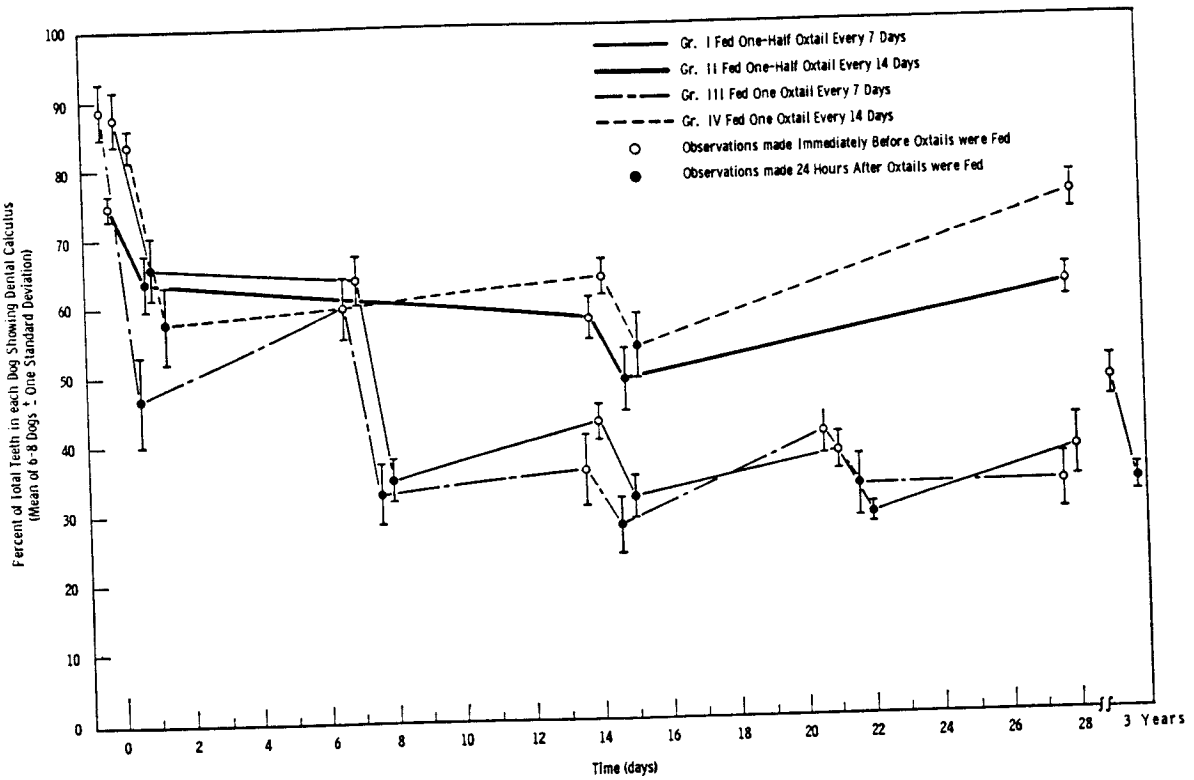


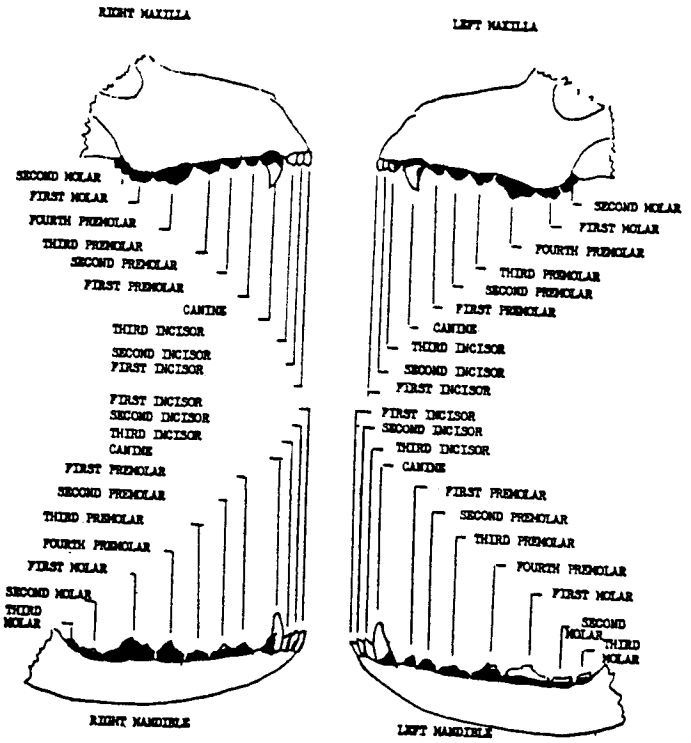
Fig. 2. *Effect of oxtail feeding on number of teeth showing dental calculus.*

ond feeding, followed by a slight increase at the end of the test period. Tooth charts (Figs. 6 and 7) of representative dogs from Groups II and IV show the calculus deposits before and after oxtail tests. No significant differences were seen between the 2 groups fed oxtails every 2 weeks. Almost all teeth showed small amounts of calculus deposits along the gingival line.

Approximately 75–90% of the individual teeth in each dog in all groups had calculus deposits at the beginning of the test period (Fig. 2). The dogs in Groups I and III had about 30% fewer teeth with calculus deposits after the first oxtail feeding and approximately 50% fewer after the second and subsequent feedings. Tooth charts (Figs. 4 and 5) show that 75% of the dogs' molars, premolars, and incisors were completely cleaned of dental calculus. Groups II and IV dogs had about 20% fewer teeth with calculus deposits after the first oxtail feeding and about 30% fewer after the second feeding. Tooth charts (Figs. 6 and 7) of representative dogs in these 2 groups show that 70% of their teeth had accumulated calculus deposits 14 days after the final feeding. Only 10% of the canine and the first and second premolar teeth were completely clean at the end of the test period. Fifty percent of the third premolars and molars were cleaned.

Only 1 dog in Group IV refused to chew oxtails initially, but was soon encouraged to do so. Fights occurred between some dogs housed together when oxtails were fed, but these were minimized if the dogs were separated the first 2 hours after they were given oxtails. Ten dogs used in the above test are still in the colony. Tooth charts were prepared after they received one-half oxtail every week for the last 3 years. Calculus accumulation was observed to be approximately the same as was observed in Group I dogs (Figs. 1 and 2) fed oxtails.

The cost of feeding oxtails once weekly



Dental Calculus Records

Before half oxtail feeding.

Black Mark Out = Dental Calculus

24 hours after half oxtail feeding.

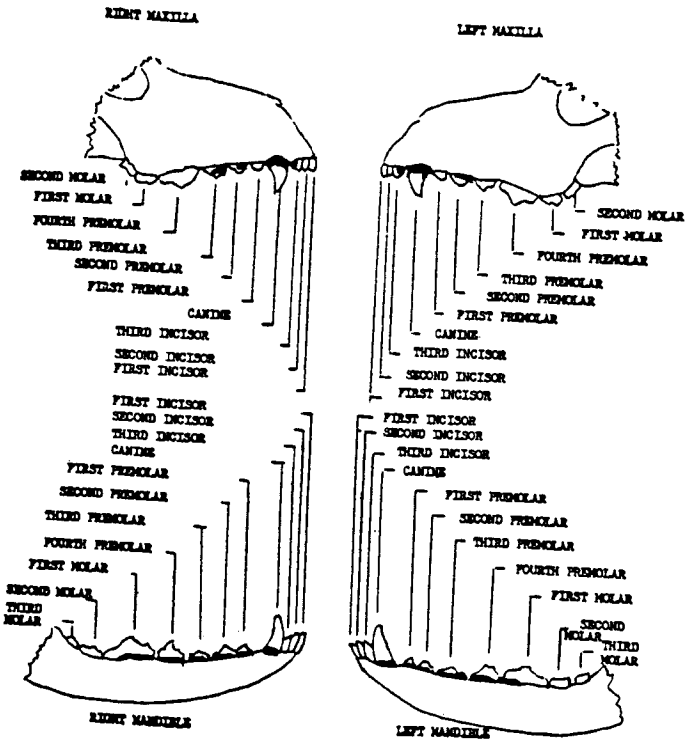
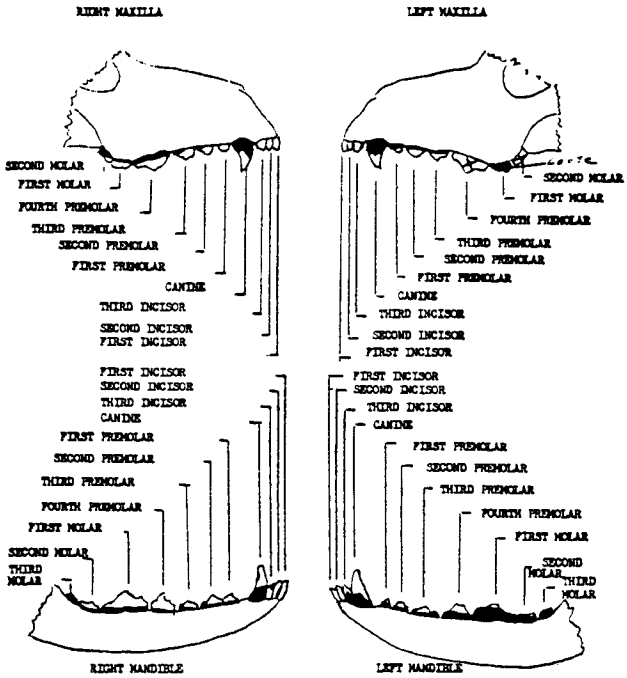


Fig. 3. *Calculus deposits on teeth before and 24 hours after half oxtail feeding.*



Dental Calculus Records

Before oxtail test.

Black Mark Out = Dental Calculus
Black X Mark = Teeth Missing

After oxtail test.

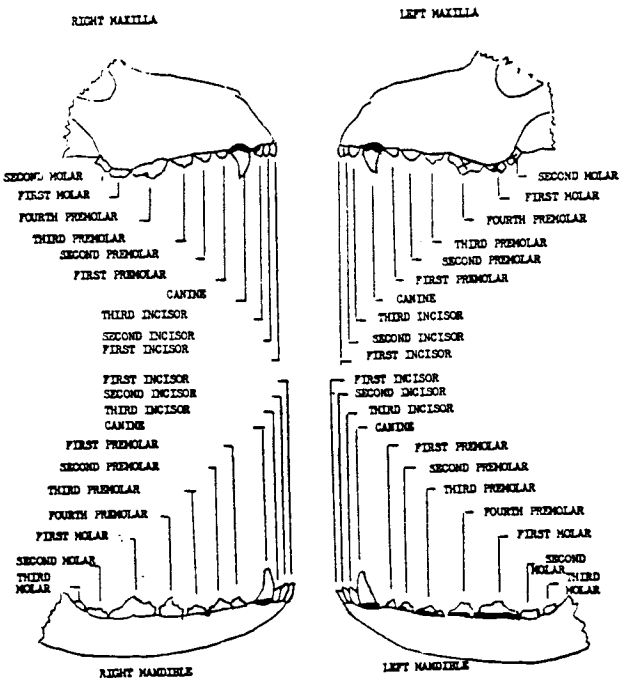
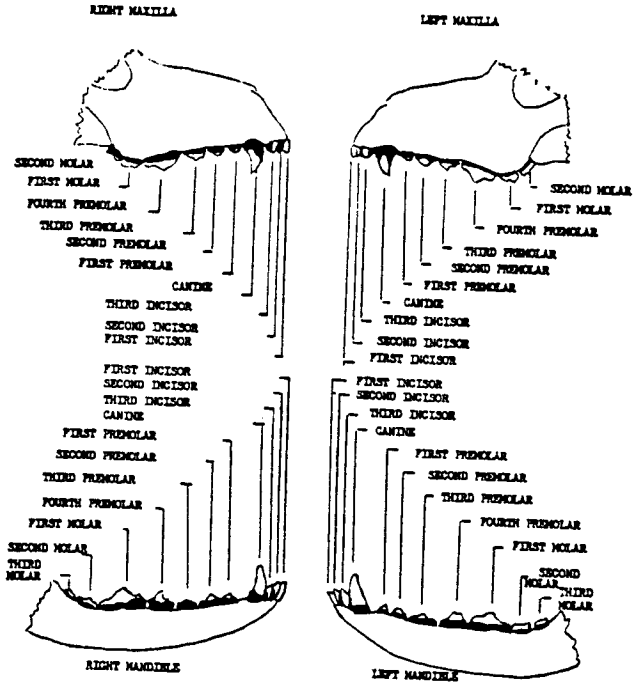


Fig. 4. *Group I: Fed half oxtail at 7-day intervals. Calculus deposits on teeth before and after test.*



Dental Calculus Records

Before oxtail test.

Black Mark Out = Dental Calculus

After oxtail test.

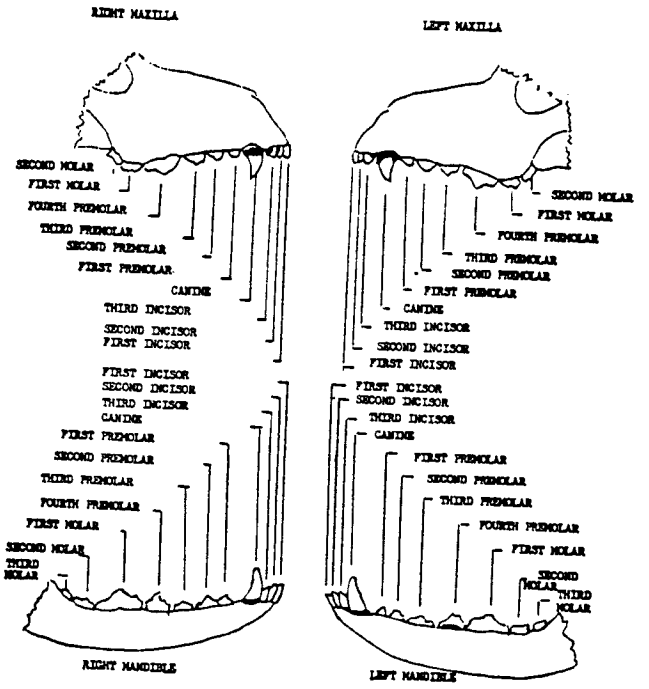
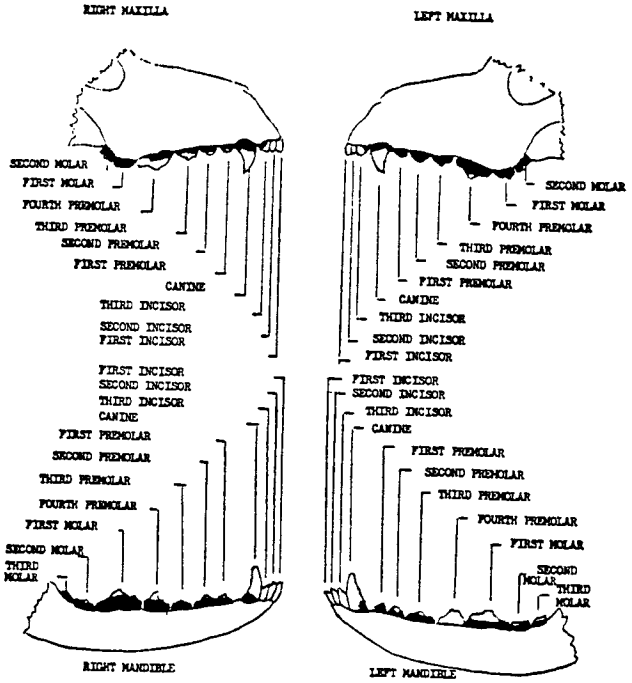


Fig. 5. *Group III: Fed 1 oxtail at 7-day intervals. Calculus deposits on teeth before and after test.*



Dental Calculus Records

Before oxtail test.

Black Mark Out = Dental Calculus

After oxtail test.

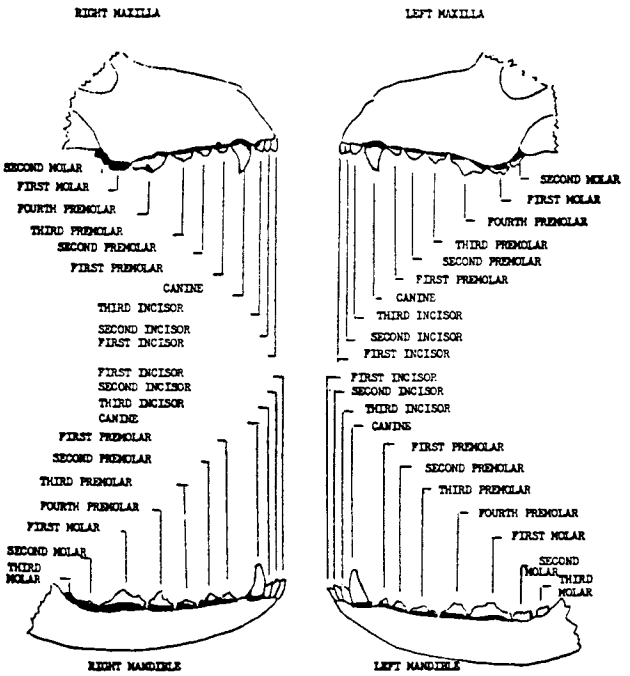
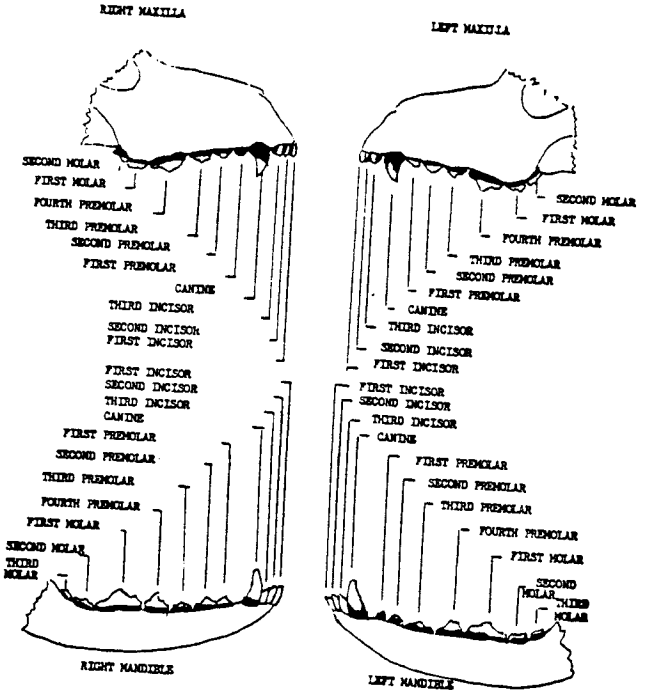


Fig. 6. *Group II: Fed half oxtail at 14-day intervals. Calculus deposits on teeth before and after test.*



Dental Calculus Records

Before oxtail test.

Black Mark Out = Dental Calculus

After oxtail test.

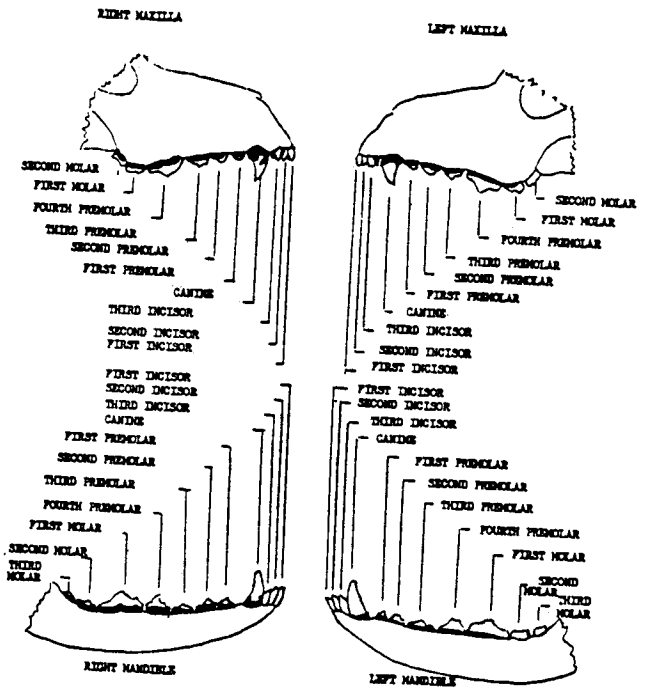


Fig. 7. *Group IV: Fed 1 oxtail at 14-day intervals. Calculus deposits on teeth before and after test.*

was approximately twice as much per feeding as dry kibbled dog food. No harmful effects of feeding oxtails have been observed in the colony of 200 dogs after more than 6 years.

Conclusions

This test confirmed the feasibility of preventing the accumulation of dental calculus in experimental beagle dogs by regular feeding of oxtails. Manual removal of calculus was not required when dogs were fed one-half or one whole oxtail per week. Biweekly feeding of one-half to one oxtail was 20–30% less effective.

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"The Distribution, Prevalence and Some Factors Associated with Periodontal Disease in Dogs and Cats"

Report to
The Animal Studies Centre, Freeby Lane, Waltham-on-the-Wolds,
Melton Mowbray, Leicestershire

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INTRODUCTION

Periodontal disease, (P.D.), a chronic apparently progressive inflammatory condition of the gingivae and other tissues surrounding teeth is seen in many species of animals and has been reviewed extensively by Page & Schroeder (1982). These authors referred to the disease in the dog but not in cats.

In man, there is some controversy as to whether gingivitis and periodontitis are evidence of a single disease or whether they occur independently. Page & Schroeder (1982) stated that in the dog periodontitis began on the basis of pre-existing gingivitis, and increased in prevalence and severity with age. There is no published evidence regarding the situation in cats, but it is generally assumed that it is similar to the dog.

The aetiology of periodontal disease in man and the domestic species is controversial also; in man evidence supports a bacterial origin (Van Pallenstein Helderman, 1981) leading to and probably exacerbated by hypersensitive and immunological phenomena (McPhee, 1972). The bacteria associated with gingivitis are those found in supra- and sub-gingival dental plaque which in man, and presumably in the dog and cat, accumulates due to inadequate oral hygiene. Page & Schroeder (1982) regarded periodontitis in the dog as a bacterial disease.

In the dog, veterinarians and others, have colloquially and anecdotally implicated variously, diet, the presence of dental calculus and the anatomy of skull and jaws in the development of periodontal disease. Similar observations

were also made for the cat. Mellanby (1930) purported to demonstrate that dietary deficiences, particularly vitamins A and D,in pups led to periodontal disease in later life. Subsequent authorities, by omission, have not seemed to consider either the nutritional or other biochemical aspects of diet as contributory factors in periodontal disease (Edney, 1982). The more recent research in man (Van Pallenstein Helderman, 1981) and in the dog (Page & Schroeder, 1982) implicating bacterial influences is more convincing.

None-the-less, diet consistency may be important in the progress of periodontal disease in the dog. This has been postulated by several authors including Gray (1923, and 1926); Wright (1939); Colyer (1947); Fiennes & Fiennes, (1968).

Colyer (1947) described the occurrence of periodontal disease in a variety of animals including dogs and domestic cats and noticed that 'in animals living under natural conditions, the disease is rare, but is common ..... in captive and the domestic animal'. He pointed out that the associated rarification of bone is generally more marked in the maxillae than the mandibles. Colyer (1947) stated farther that dogs and cats which lead a freer life and obtain a diet more nearly approaching their natural food were practically free from P.D., concluding that an alteration in the physical or chemical character of the diet must induce periodontal disease. Brown & Park (1968) showed that the inclusion of raw ox-tail in the diet of colonies of Beagles reduced the accumulation of dental calculus and presumably by inference the prevalence of periodontal disease.

Research into periodontal disease in the dog has been undertaken principally in colonies of Beagles, but no scientific data has been reported for similar studies in domestic dogs nor cats. Bell (1965) and Borthwick (unpublished) showed the prevalence of periodontal disease in dogs in proportion to other dental and gingival pathology to be high, approximately 70 per cent and similarly in cats (Borthwick unpublished). The previous studies by the latter indicated that the prevalence of clinical dental conditions in the dog was 2.8 per cent in the population at risk and 1.5 per cent in cats; while dental treatment accounted for 13 per cent of all surgical therapy in dogs and six per cent in cats in the Department of Veterinary Surgery, R.(D).S.V.S., Edinburgh. There also appeared to be a wide range in prevalence and severity of periodontal disease between the breeds of dogs examined.

Periodontal disease is known to be a major cause of tooth loss in man (Prevent 1: 1972) and in the dog (Bell, 1965; Borthwick, unpublished; Brown & Park 1969; Gray 1923, 1926; Page & Schroeder 1982 and Wright 1939) and in the cat (Borthwick, unpublished). Great concern has been expressed regarding the general lack of endeavour to prevent P.D. in man and world wide international efforts have been encouraged in research and prophylaxis (W.H.O. Technical Report, 1978).

This present study was undertaken to establish the periodontal status of dogs kept as companion, sporting or working animals and in household cats, to promote a better understanding of periodontal disease with a view to developing programmes of prophylaxis.

MATERIALS & METHODS

The population for study was pure bred canine and all feline patients attending the Department of Veterinary Surgery, Royal (Dick) School of Veterinary Studies, University of Edinburgh over a period of two years. The sub-populations of the animals examined were those over six months old with complete permanent dentition which had general anaesthesia for therapy or investigation. Dogs and cats admitted to the survey were those which were available when the author was available to examine them, thus no special methods of selection were applied. Patients which had dental therapy in the previous 12 months were excluded.

Details of the breed; age; sex; weight; diet; history of previous periodontal treatment and whether they were to be treated for periodontal disease or for other diseases, were recorded.

Several elaborate scoring protocols for gauging periodontal status, dental plaque and dental calculus levels have been used in man; however as the examination of the animals in this study was intrusive simpler but adequate systems were employed. The method described by Ramfjord (1967) for comparing periodontal status in different races of people was employed in dogs and cats, and gave a Periodontal Index (P.D.I.) for each patient. The simplified scoring system for measuring dental plaque and calculus used by Rosenberg & Ash (1974) slightly modified, was employed to give an Irritation Factor (I.F.) i.e. combined plaque and calculus and also for calculus independant of plaque, the Calculus Index (C.I.). The following instruments were used, Ash De Trey No. 4

mouth mirror; Williams 14W and Ash No. 2 pocket measuring probes; Ash upper molar type dental forceps and Gray's pattern mouth gags. The recorded data were used to create data files in the Edinburgh Regional Computing Centre; programmes from "Statistical Packages for Social Sciences" were used for retrieval and statistical analysis.

RESULTS - DOG

Three hundred and thirty two pure bred dogs were examined during the two year period, 83 had clinical periodontal disease (dental cases), 249 were admitted for other surgical therapy or examinations (non-dental cases). Thirty six of the patients had had previous treatment for periodontal disease, Table 1.

The ages of the animals ranged from six months to 16 years, Table 2, and the sex ratios are given in Table 3.

Fifty one breeds of dogs were represented and are listed in Table 4 with the respective numbers. The dogs ranged in weight from less than 2.5 kilogrammes to over 50 kilogrammes, the weights were divided into twelve groups for convenience of analysis. Tables 4 and 5.

The mean age of non-dental cases 6.1365 years, S.D. 4.1974, differed significantly from those with P.D. 9.3253 years, S.D. 2.9138, P = 0.0000, Table 6. A similar significant statistical difference was noted between the ages of dogs with no previous P.D. therapy and those which had, 6.50 years, S.D.. 4.09 and 10.47 years, S.D. 2.70, P = 0.0000, Table 7.

Periodontal Disease Index

The Periodontal Disease Index (P.D.I.) for the population ranged from 0.00 to 5.17 (possible maximum 6), with mean 1.599(S.D. 1.169). P.D.I. by age is shown in Table 8 which also gives the statistical analysis of variance, indicating a highly significant difference between the age groups, with good linearity i.e. severity increased with increase in age.

Table 5 gives P.D.I. data for dogs in the twelve weight groups, statistical analysis showed a highly significant difference between the groups and good negative linear correlation suggesting decrease in P.D.I. with increase in body size. There was no significant difference in P.D.I. between the four sex divisions, P = 0.0981, (Table 9).

P.D.I. data from the non-dental patients was very significantly different from data from dental patients, P = 0.0000, as were the data from dogs with no dental history and those with, P = 0.0000. (Tables 6 and 7). The P.D.I. data and statistical analysis for each breed of dog, Table 10, showed a distinct breed difference which was highly significant, P = 0.0000.

Irritation Factor (Plaque & Calculus) (I.F.)

The dental deposit or Irritation Factor (I.F.) for the population ranged from 0.00 to the possible maximum 3.00, mean 1.923, S.D. 0.728.

I.F. data by age (Table 8) and by weight (Table 5) were significantly different between the respective groups, P = 0.0000 for each, with good positive linearity for the age data and good negative linearity for the weight figures.

A highly significant difference was noted for the breed data, P = 0.0000, but no significant difference between sexes, P = 0.0693. (Tables 10 and 9).

Again I.F. differed significantly between the non-dental and dental cases, P = 0.0000 and between dogs without and with previous periodontal therapy, P = 0.000. (Tables 6 and 7).

Calculus Index (C.I.)

Deposits of dental calculus measured separately from the total deposits for the population scored from 0.000 to 3.000 the maximum possible, with mean 0.776, S.D. 0.815. C.I. for the separate breeds is given in Table 10.

The C.I. by age (Table 8) and for the weight groups (Table 5) were statistically significantly different, P = 0.0000 for each criterion, with good positive linearity for the age data, and negative linearity in the weight data. No significant difference was observed in the sex data, P = 0.1067, (Table 9).

C.I. for non-dental and dental patients differed significantly, P = 0.000, as did that for those which had no previous dental therapy and those which had, P = 0.0000. (Tables 6 and 7).

Comparison Between P.D.I., I.F., and C.I.

Pearson correlation co-efficients showed a high correlation between the three factors (Table II). Higher mean indices particularly P.D.I. were observed at the canine, premolar and molar areas.

Table 12 gives the mean indices for the dental arcade segments for upper and lower jaws and demonstrates the significant difference between the data for maxillary and mandibular areas and between the different segments of the arcades.

Diet: P.D.I., I.F. and C.I.

Accurate details of diet were ascertained for 311 of the dogs examined.

Diets seldom comprised of one component only; the majority of the dogs were given a meat staple with biscuit mixed or fed separate from the meat, 190 received canned meat while most of the remaining 121 dogs had cooked or raw fresh meat or a mixture of canned and fresh, with very few having commercially prepared whole diet mixtures, fish or household food.

Approximately half of the 311 dogs - 157 - also had "chews" on a regular basis i.e. they were available always and replaced when required. The 'chews' were bones, rawhide or super-hard baked biscuit.

The statistical significance of the data relating to the main diets and P.D.I., I.F. and C.I. are listed in Table 13. The type of meat did not affect the degree of periodontitis (P.D.I.), volume of dental deposit (I.F.) nor the accumulation of calculus (C.I.); addition to the diet of biscuit "mixers" did not alter this. On the other hand "chews" did have an important influence in that dogs which had regular access to them had highly significant lower P.D.I., I.F. and C.I. values, Table 14.

L.S.D. and Scheffe statistical tests at 95 per cent confidence levels did not indicate that any one "chew" was much superior to any of the others: however, super-hard biscuit was associated with lower mean P.D.I.; bone with lower mean I.F. and C.I. while super-hard biscuit also influenced I.F. and C.I. though to a lesser degree than bone.

RESULTS - CATS

Fifty two cats were examined during the period of study, 40 were non-dental patients, 12 were treated for periodontal disease. Only one cat had had previous dental therapy. Table 15.

The cats' ages ranged from six months to 15 years, the numbers in each age group are given in Table 16. The sex distribution is shown in Table 18. Body weights ranged from 2 to 6.5 kilogrammes, the numbers in three weight groups are listed in Table 17.

Cats were not defined by breed nor type.

Periodontal Disease Index (P.D.I.)

P.D.I. for the population ranged from 0.00 to 3.00; mean 1.78 S.D. 0.767. The data for age (Table 16) was significantly different statistically between the groups, P 0.0316 with significant positive linearity. A similar pattern was seen between the weight groups P = 0.0122 and P = 0.0092.

Analysis of P.D.I. data for the four sex groups given in Table 18 showed a very significant difference between the groups, P = 0.0000.

Mean P.D.I. for patients with non-dental diseases and for those with clinical periodontal disease are listed in Table 19, analysis gave a highly significant difference between the groups P = 0.0000.

Irritation Factor (I.F.) (Plaque & Calculus)

The range for the population was 0.00 to 3.00 mean 1.788, S.D. 0.746. I.F. indices for cats in age groups (Table 16) when analysed did not show a clearly significant difference between them, P = 0.0651, although the linearity was good. Analysis of data between the weight groups (Table 17) showed a highly significant difference with good positive linearity.

A highly significant difference, P=0.0000 was found between the sex groups (Table 18). The I.F. score data for non-dental and dental cases differed significantly, P = 0.0062. (Table 19).

Calculus Index (C.I.)

The C.I. for the population ranged from 0.00 to 3.00, mean 0.585, S.D. 0.539. Indices for the age group sub-populations differed highly significantly, P = 0.0000, with excellent positive linearity, P = 0.0000, Table 16. Data from the non-dental cats differed very significantly from the dental cats, P = 0.0000. Table 19.

Correlation of P.D.I., I.F. and C.I.

As in the dog a strong correlation was noted between P.D.I., I.F. and C.I.

The apparent anomally regarding the analysis of P.D.I., I.F. & C.I. in the sex and weight groups, compared with the dog led to farther examination of these data.

The average age of entire male and entire female cats was 1.17 and 3.08 years respectively, while the average ages for neutered animals were 7.89 and 8.86 years, which suggested that age was the factor influencing the lower mean P.D.I., I.F. and C.I. in entire animals. (Table 18a).

Heavier i.e. larger cats should have had lower mean indices if compared to dogs. Analysis of the ages of cats in the three weight groups gave respective mean ages of 3.25, 6.4286 and 8.80 years, P = 0.0017,(Table 17a) and again would relate higher P.D.I., I.F. and C.I. to increase in age rather an association with body size when the three indices would have been lower in the heavier groups.

Diet: P.D.I., I.F. and C.I.

Cats were expected to be more conservative in their eating habits than dogs, but although the majority received canned meat all the cats examined also had high and varying proportions of raw or cooked meat and fish. That pattern of feeding precluded grouping of diets according to a main or sole constituent.

Cats in this study did not have biscuit supplement or "mixers" although a few were given "tit-bits" of proprietory prepared whole dry food, but not in the quantity nor frequency expected to have influenced their periodontal status.

None of the diet groupings was associated with statistical differences in P.D.I., I.F. nor C.I. data within 95 per cent confidence limits.

None of the cats were provided with "chews" and very few owners were sure if nor how often their pets caught or ate small prey which may be the feline equivalent.

DISCUSSION

Gingivitis and periodontal disease in dogs and cats appeared to be ubiquitous in the population studied, (Tables 8 & 10). A very small number had zero or miminal P.D. Indices and these were mainly young animals whose permanent dentition had been completed recent to examination or occasional less young animals of large breeds of dog. Those dogs and cats also had minimal dental

deposit scores. The disease was prevalent albeit at sub-clinical level in patients which were seen for non-dental problems (Table 6). No specific criteria were established for defining the difference between non-clinical and clinical periodontal disease, apart from owners noticing "halitosis", oral bleeding or difficulty in mastication. It was evident that many of the non-dental patients were in need of oral therapy. Close correlation between P.D.I., I.F. and C.I. (Table 11) demonstrated a strong association between the amount of dental deposits and the degree of periodontal disease, consequently it was noted that P.D.I. increased linearly with age (Tables 8 and 16). Presumably the more deposit and the longer it was present there was a greater likelihood of periodontal disease to develop and increase in severity, supporting the contentions of Gray (1923, 1926), Wright (1939), Fiennes and Fiennes (1969), Brown & Park (1969) and many others. In man it was suggested that the strong correlation of periodontal disease with age probably reflected the cummulative effects rather than diminishing resistance in older people and that the length of time the tissues were exposed to dental plaque was important in the development of periodontal destruction (W.H.O. Technical Report, 1978).

It has been thought that female dogs and cats were less prone to periodontal disease than males, this again may have been an extrapolation from the human situation where the W.H.O. Technical Report (1978) stated that in most surveys women were found to have a lower severity of P.D. than men. This was not so in dogs where no statistical difference was seen between the four sex groups, nor in the age corrected data from cats (Tables 9 and 19, 19a). Possibly women are more fastidious than men with regard to oral hygiene, but domesticated animals are complete populations which are managed similarly.

The results obtained in this study for dogs regarding the reducing severity of periodontal disease with increasing body weight (Table 5) i.e. size of the animal, were reflected in the breed prevalence, (Table 10), so that the smaller breeds, particularly those of the Toy Group, were at greater risk of developing P.D. early in life, of showing increasing severity sooner and were more prone to tooth loss. The shape and relative size of skull and jaws must obviously have played some part in the predisposition to clinical P.D., brachycephalic breeds and those with "squarer" muzzles were less favoured than those with more normal, longer skulls and jaws: however there were some exceptions and farther studies in this area would be desirable. The results from cats, whose body size and skull anatomy did not show such great variation as dogs, were less easy to reconcile regarding P.D. severity to body weight, the main influence appeared to be the length of time that their gingivae were exposed to dental deposits (Tables 17, 17a & 16).

Table 12 shows the validity of Colyer's (1947) observation regarding greater severity of P.D. in upper than in lower jaws in dogs. This applied also in cats.

The highly significant difference between P.D.I., I.F. and C.I. of non-dental and dental patients was expected and would seem to have been associated with the age factor in dogs and cats and possibly with body size in dogs, (Tables 6, 19 & 5). The question of when sub-clinical P.D. became a clinical problem remained unanswered, with the apparent need for therapy in many of the non-dental cases one must assume that dogs and cats reach a state or age at which they manifest clinical signs or behaviour which is recognised by owners or veterinary surgeons. On the other hand owners may not be aware of the importance of the signs till the disease is at an advanced stage. Again, the highly significant difference between the age, P.D.I., I.F. and C.I. of those dogs which had not or had previous periodontal therapy (Table 7) may have been purely age, breed or size dependant. The age distribution and linear increase in severity of P.D. in dogs and cats and the recurrent nature in dogs suggested continuing susceptibility and aetiological factors and that the disease was progressive.

The WHO Technical Report (1978) stated that there was little evidence as yet to implicate nutritional excesses or deficiencies in chronic periodontal disease in man, even as modifying factors, and that moderate changes in diet consistency did not seem to affect plaque accumulation. Table 13 indicated that the diets of dogs in this survey did not influence P.D.I., I.F. nor C.I. significantly one from another. The fact that there was an over-all continuing and increasing severity of P.D. suggested that although the nutritional value of the food might have been adequate it certainly did nothing to reduce the hazzards of P.D. These findings along with modern concepts of P.D. aetiology (Page & Schroeder, 1982; Van Pallenstein Helderman 1981) tended to invalidate Mellanby's (1932) conclusions for spontaneous clinical P.D. and lend support to Gray, (1923, 1926); Wright (1939); Colyer (1947); Edney (1982), and many others. Likewise none of the foods fed to cats influenced the prevalence of P.D.I. & I.F. nor the increasing severity of P.D. It is interesting to note in Table 12 that the higher indices were at the canines, which have become largely obsolete anyway in domestic dogs, and at the sectorial divisions - the premolar and molar areas. The upper third and fourth premolars with the upper first molars (and to some extent the upper second molars) apposed to the lower fourth premolars and the lower first and second molars form composite shearing,

masticatory units in most breeds of dogs. High P.D.I., I.F. and C.I. in the premolar and molar segments might again indicate a degree of obsolescence because of the types of food which dogs are given, exacerbated by gnathic bone abnormalities in some other breeds

Table 14 shows the strong beneficial effect of "chews" on P.D.I., I.F. and C.I. in the dogs studied. Brown and Park (1969) demonstrated a similar pattern for dental calculus, while Gray (1923, 1926) wrote that food which required to be masticated as opposed to just swallowed would also reduce the incidence of periodontal problems in dogs and cats. Colyer's (1947) views have been exposed already. There are problems associated with including "chews" in the diet of dogs, Fiennes and Fiennes (1969) and Gray (1923; 1926) observed that smaller dogs with finer or weaker jaws were reluctant to eat hard materials and consequently suffered more severe periodontal disease. It has also been observed clinically in man, that reduced masticatory function usually led to increased plaque and dental calculus formation, (W.H.O. Technical Report 1978). Gray contended, however that if dogs were introduced to firmer food early in life even the smaller breeds could cope and benefit. It is known clinically that continual bone chewing is one of the major causes of dental attrition in dogs, where even in early middle age some dogs will have worn their tooth crowns to gum level. Many dogs also which have irregular access to bone develop very severe blockage of the rectum and colon requiring surgical intervention for relief. Brown and Park (1969) did not mention these problems in their Beagles, so if bone is the "chew" of choice it probably should be included in a controlled pattern. Neither of these features of dental attrition or rectal blockade seemed to be associated with super hard biscuit which scored well in this study, therefore it might be preferred to bone, or alternated with bone and/or rawhide.

There is no scientific evidence from this survey that firmer diets or the inclusion of hard commercially prepared whole foods would be equally beneficial in reducing P.D. in cats, but one would think, intuitively, that the opinions of Gray (1923 & 26) and Colyer (1947) may be justified in this respect.

Summary and Conclusions

Periodontal disease was common to all ages of dogs and cats apart from the very young, and was more prevalent and severe in small dogs. The severity increased with increasing age and eventually surgical therapy was required by most patients. If the aetiology is understood correctly, dental deposits - plaque and calculus, - which were strongly correlated statistically with P.D. were the principal cause despite conjecture as to which of the plaque bacteria or their products were responsible ultimately for periodontal pathology. Dental calculus alone was unlikely to have been the initiating factor.

It was evident, as in man (W.H.O. Technical Report 1978), that the amount of dental deposit which can be accumulated or tolerated varied from individual to individual. This was remarked in breeds with high P.D.I. and in those with low indices; this factor also would be an interesting field for farther study.

Modern methods of feeding dogs and cats did not appear to be an improvement as far as dental health was concerned, observers may argue that P.D. was less severe than when described by Gray in 1923 and 1926, but currently the effects of P.D. and the clinical signs are allayed or masked by the administration, for convenience or lack of understanding, of systemic antibacterial therapy. Increased masticatory function might be improved, by changing the consistence of diets or by offering chewing material, to great advantage in controlling periodontal problems. There does not seem to be any method, at present, to simulate in dogs or cats, the protocols of oral hygiene undertaken in man. The marketing of a canine dentifice some decades ago was a failure, the patients did not permit having their teeth brushed regularly and owners could not or would not attempt to do so for very long.

It would appear that the veterinary surgeon - patient approach to reducing periodontal disease in dogs and cats is largely ineffective and that people have come to accept that P.D. is inevitable. A significant part of canine and feline veterinary surgery is taken up with dental therapy, removing dental calculus and/or extracting loose teeth or those showing excessive root exposure, i.e. dealing with a chronic longstanding situation.

Accumulated knowledge has provided a rational explanation of the aetiology and progress of periodontal disease in man which seem to be paralleled in the dog and cat, so it should not be too difficult to devise schemes of prevention. Serious consideration might be given to modifying diets by including elements to increase masticatory function and also to re-orientating the provision of veterinary dental care towards early preventative treatment rather than palliation when periodontal disease has reached a terminal stage.

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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

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).

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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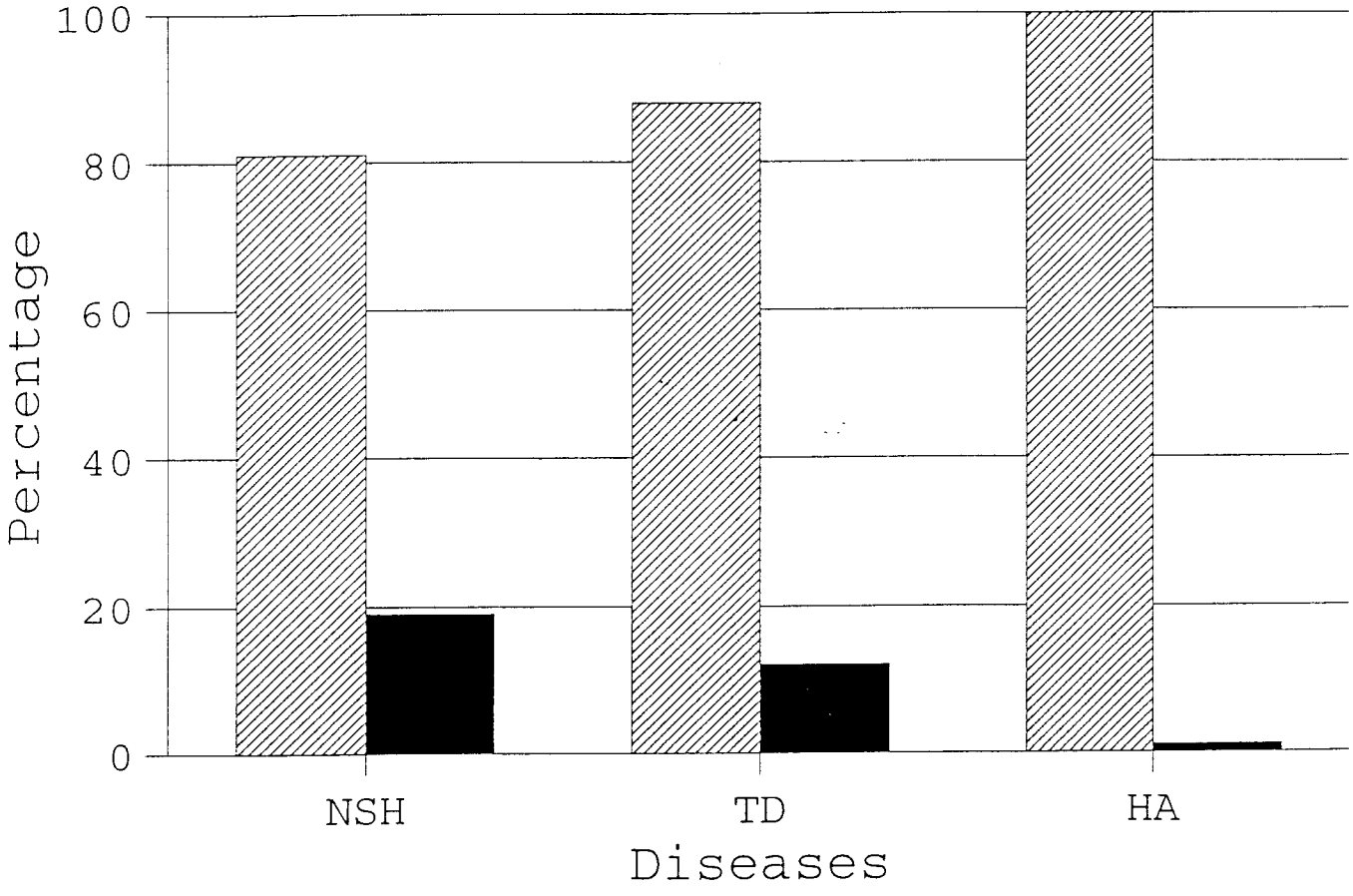
CAPTION

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)


100
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Diet and periodontal disease in dogs and cats

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SUMMARY: A review of relevant literature was undertaken because of concerns about a possible relationship between pet foods, development of periodontal disease, and secondary adverse effects on general health. It was concluded that, while changes in feeding methods in recent decades have arguably improved pet health by reducing or preventing diseases associated with nutritional deficiencies and excesses, periodontal disease remains a serious, diet-related problem. There is reasonable evidence that soft diets are associated with increased frequency and severity of periodontal disease, and that harder foods requiring vigorous prehension and mastication are preferable for dogs and cats. While it is plausible that periodontal disease could cause diseases in other organs and tissues, the evidence for this is limited at present. Further research is needed to better define the relationship between diet types and oral health. In the meantime, veterinarians and pet owners should pay attention to the physical qualities (textures, abrasiveness, 'chewiness') of foods they provide for dogs and cats, as well as to their nutrient content, and should consider additional methods to control plaque and prevent periodontal disease where necessary.

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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 substantially (Anon 1985a). An obvious change in application has been the trend away from home-prepared rations towards commercial pet foods, 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. For example, review of three nutrition-related diseases (nutritional secondary hyperparathyroidism in dogs and cats, hypervitaminosis A and thiamine deficiency in cats) at the Sydney University Veterinary Teaching Hospital showed a 75% decline in these diagnoses between 1975 and 1993 (Watson 1994). However, comparable data for other diseases possibly related to diet, such as periodontal disease, are lacking.

The present review was prompted by the recent discussions in Australia and elsewhere about the relationship between diet and the development and sequelae of periodontal disease in dogs and cats.

Materials, Methods and Limitations

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.

Several difficulties became apparent during the review. Firstly, terms such as periodontal disease, periodontitis, gingivitis, plaque, and so on, were often used without prior definition, and one cannot be certain they were used appropriately. The terms had to be accepted at face value, despite possible inaccuracies. Secondly, much of the material was anecdotal, and some of the experimental work was flawed by small sample sizes, poor statistical methods or incomplete reporting of data. Accordingly, rigorous scientific criticism of the material was not undertaken – the reviewer attempted to discern the underlying pattern rather than to insist on strict scientific proof. Thirdly, there was a problem with feline dental resorptive lesions (also known as neck lesions or cervical line lesions). These appear to be a recent phenomenon, occur with increasing frequency in older cats, and may be associated with gingival inflammation (Okuda and Harvey 1992). The cause and pathogenesis of these lesions are incompletely understood. They may be a unique pathological consequence of feline periodontitis, or gingival inflammation may be a response to initial dental resorptive changes (Lyon 1992; Okuda and Harvey 1992; van Wessum *et al* 1992). As there is also little information about dietary influences on feline dental resorptive lesions, they were not included in this review.

Results

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 to 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 necropsied dogs between four and eight years of age. Among 125 colony Beagles, about 95% had heavy calculus deposits after 26 months of age, and gingival inflammation developed concomitantly (Rosenberg *et al* 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 *et al* 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 *et al* 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 *et al* 1989).

Similar population survey data are lacking in Australia, but 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 of 80 to 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 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 *et al* 1992; Harvey 1993a, b; Sarkiala *et al* 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 that 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. However, accumulation of plaque can be prevented by some chemicals, such as chlorhexidine (Reed 1988)
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 *et al* 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. Burwasser 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 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 *et al* 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), with the same food 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 over an 18-month period. Dogs fed the dry form acquired less "soft debris" on their teeth, but there were no differences in gingivitis, calculus or periodontal measurements (Saxe *et al* 1967). While this is at variance with most other studies quoted, the contrast in food consistencies might have been insufficient to produce other differences between groups.

That the effect of diet was related to soft consistency rather than mineral imbalance was shown by Svanberg *et al* (1973). For 18 months, four Beagles were fed a soft diet with low calcium and high phosphate contents and two controls received soft food with adequate mineral content. Teeth on one side of the mouth were cleaned regularly. While the test dogs lost alveolar bone, the osteopenia did not promote loosening of teeth, gingivitis or accumulation of dental deposits. Furthermore, equivalent degrees of periodontitis developed in test and control dogs in areas where dental deposits were allowed to accumulate.

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 *et al* 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 that 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 *et al* 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).

The feeding of large pieces of raw vegetables (cauliflower, broccoli, cabbage, swede), flavoured with gravy, has been suggested as an anti-plaque measure (Emily and Penman 1990) but has not been evaluated clinically. However, its value might be limited, as eating three raw carrots thrice daily did not prevent plaque accumulation in Swedish dental students (Lindhe and Wicen 1969).

Population surveys – Several surveys have provided data on the relation between oral health and diet. Among 63 dogs surveyed in the USA (Golden *et al* 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 for the Waltham group (Markwell 1986), indices of oral health were worse in cats fed 30 to 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 might have affected the outcome.

Harvey (1992, 1993c) reported preliminary analyses of survey data for 1350 dogs and > 700 cats anaesthetised at veterinary hospitals in the USA. More recent and rigorous analyses of the data (CE 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.

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 used 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). About half of the teeth surfaces of all dogs were covered with calculus initially and about 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 to 30% less effective. No harmful effects were observed from feeding oxtails to > 200 dogs for > 6 years.

Lage *et al* (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 involving various organs or tissues. 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), and Lonsdale (1993a, b) has mentioned numerous canine and feline illnesses as possible sequelae. DeBowes (1993) listed bacterial endocarditis, glomerulonephritis, polyarthritis, polyvasculitis, autoimmune disorders, discospondylitis, endotoxaemia, and pulmonary disorders as complications proposed by others, but the references given provide circumstantial evidence rather than proof of a causal relationship.

Most recently, a preliminary report on histopathological changes in dogs with periodontal disease indicated that those with the most severe periodontal disease had the most inflammatory changes in their kidneys (DeBowes 1994). In this group of dogs there were no detectable changes in hepatic enzymes related to the periodontal disease.

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, lungs and kidneys. This combination could permit interaction between sepsis originating in periodontal pockets and target organs (Hamlin 1990, 1991). 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 *et al* 1980). On the other hand, Harari *et al* (1993) found a similar prevalence of bacteraemia in 30 dogs with periodontal disease as in 30 control dogs not subjected to dental procedures. None of the bacteraemic dogs had illness attributable to bacteraemia in the month after testing (Harari and Besser 1993). In this study, only 30% of patients were bacteraemic after dental manipulation, but the work was criticised for several reasons including the elimination of animals with very severe disease (Harvey and Hammond 1993). As usual, data for cats are limited, but Harari *et al* (1991) found four of 11 cats (36%) with periodontal disease had one or more positive blood bacterial cultures after dentistry procedures whereas all predentistry samples yielded no growth.

Evidence for a causal association between periodontal disease and other disorders is limited at present. However, as periodontal disease may be the most common disease seen in small animal practice (Harvey and Emily 1993), and its effects on gums and teeth can significantly affect a pet's health and well-being, there is *already* reason for concern. Proof of additional systemic effects of periodontal disease may increase the level of concern, but is not needed to justify action.

Conclusions

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 or not 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.

The evidence implicating soft food diets in the aetiology of periodontal disease is suggestive rather than conclusive, but the author believes a *prima facie* case exists. 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 that such foods will prevent periodontal disease in an individual pet, but they should help.

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

Recommendation and Suggestions

Further research should be undertaken to better define the relationship between diet types and oral health in dogs and cats. In particular, there is a need to explore more thoroughly the possible effects of softer diets on the occurrence of periodontal disease in both species, and of dental resorptive lesions in cats. In the meantime, it would be prudent for veterinarians to consider the following points in relation to diets. These suggestions should be regarded as provisional 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 and favour 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, fibrous vegetables to encourage chewing
   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, dentifrices
   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 oral health 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 may 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 carcases, whole rabbits, kangaroo tails, lamb flaps, ox-tails, and chicken necks (Lonsdale 1993a). These authors generally suggest that bones are safer if given raw rather than cooked.

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Periodontal disease and leucopenia

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ABSTRACT

Periodontal disease is rare in nature but widespread in domestic dog and cat populations. Unnatural diets are known to facilitate the build-up of oral microbial communities which then interact with host-immune defences giving rise to periodontitis. Eight of 14 animals undergoing dental treatment and dietary change at a suburban veterinary practice were investigated and found to have low leucocyte counts. Follow-up testing revealed changes averaging a 77·7 per cent increase with concomitant 'subjective good health'. These findings serve to cast doubt on the commonly used haematological reference ranges where the subject animals may have suffered from periodontal disease. The demonstrated reversibility of white cell depression associated with periodontal disease should provide a focus for further research.

INTRODUCTION

Periodontal disease is an inflammatory process involving the supporting structures of the teeth. Whilst plaque organisms are known to be closely involved in the initiation of disease, emphasis is now shifting to investigations of the factors that influence host/plaque interaction (Harvey 1993b).

Removal of plaque, either by physical or chemical means (Sanz and Newman 1994) or by the abrasive forces of feeding, exerts control over the development of periodontal disease. Various authors have commented that the natural diet of wild carnivora has a plaque-retarding effect (Page and Schroeder 1982, Higgins 1987, Harvey 1993a, Watson 1994). The human dentist and comparative periodontist, Sir Frank Colyer (1947) concluded: 'Paradontal disease is always associated with an alteration in the physical or chemical character of the diet of the animal – in other words with a departure from natural diet and conditions.'

There is accumulating evidence of the systemic consequences of periodontal disease – in its simplest view, pain, local to the lesion, is registered in the brain with concomitant lack of appetite and evidence of malaise. Various authors (Beard 1991, Hamlin 1991, Lonsdale 1993a, DeBowes 1994) cite a range of systemic conditions, eg, bacterial endocarditis, glomerulonephritis, polyarthritis and polyvasculitis, believed to be consequences of chronic oral inflammation.

It is thought that systemic conditions may actively exacerbate periodontal disease (Harvey and Emily 1993, Liebana and Castillo 1994). One theory postulates that there is an interactive cascade of events involving a progressive, two-way worsening of periodontal disease and body sytems giving rise to a range of degenerative diseases (Lonsdale 1993a). Due to the reserve capacity of the various organ systems (eg, liver, cardiovascular system and kidneys) it is believed that the interactive spiral decline may remain clinically undetectable until end-stage developments. The same could be said for circulating leucocytes which may exhibit minimal effects on circulating numbers but a profound alteration in the function of the cells (Latimer and Meyer 1989b). Due to a wide reference range, leucocyte numbers can increase or decrease markedly whilst still disguising underlying disease. In the event of reduced circulating numbers this can be explained by a decreased production of the leucocytes, redistribution between the circulating and marginal pool and emigration into the tissues (Latimer and Meyer 1989a).

This paper details the outcome of eight cases presented to a suburban clinic showing advanced periodontal disease and reduced leucocyte and

erythrocyte numbers. Surgical treatment of the presenting condition and change of diet resulted in a marked increase in circulating leucocytes with a return to 'subjective good health'.

MATERIALS AND METHODS

Eight animals presented at a suburban practice for a number of reasons were selected. These comprised six dogs and two cats. In each instance clinical examination revealed advanced stage periodontal disease. Treatment consisted of radical dentistry including removal of diseased teeth, and the institution of a five-day course of amoxycillin (Amoxil; SmithKline Beecham Animal Health) or clindamycin (Antirobe; Upjohn).

From day 1 after surgery the animals were fed a raw meaty bone diet with occasional supplemental table scraps. The small dogs and cats received chicken wings, rabbit legs and whole raw fish whilst the larger dogs received lamb flaps, ox brisket, kangaroo tails, etc. The size of pieces was important as a regulator of chewing function. Pieces too small would permit swallowing whole with the risk of obstruction. Large bones without meat have lesser nutrient value whilst risking the wear or breakage of teeth. At the time of dental treatment, five of the cases had full blood counts performed; of the remaining three cases a white cell count plus differential cell count were performed. Blood tests were repeated at the time of the animals' check up.

RESULTS

The results of the present study are summarised in Table 1.

Cases 1 and 2, a seven-year-old male and nine-year-old female silky terrier, were assessed first. During the previous six years the dogs had been presented to the surgery on numerous occasions with non-specific illness, lethargy or dermatitis.

Table 1. Clinical details and haematology profiles for eight animals undergoing radical dental treatment and diet change

Case	1	2	3	4	5	6	7	8
Breed	Silky terrier	Silky terrier	Terrier cross	Chihuahua	DLH	Golden retriever cross	DSH	Maltese terrier
Age (years)	10	12	11	8	7	13	12	12
Sex	M(E)	F(S)	F(S)	M(E)	F(S)	F(S)	F(S)	F(E)
Presenting complaint/signs	Routine vaccination	Routine vaccination	Routine vaccination	Routine vaccination	Prolapsed third eyelids, temperature 39°C	Halitosis, lipoma, arthritis, fleas	Inappetence, lethargy, dehydration	Lame right hind leg, severe heart murmur, mammary lump, dermatitis, otitis, fleas
Treatment	Radical dentistry, flea treatment, dietary change	Radical dentistry, flea treatment, dietary change	GA, multiple extractions, dietary change	Dentistry, blood tests, dietary change	Liquid paraffin and dentistry, dietary change	Dentistry, lipoma removal, dietary change	Dentistry, dietary change	Dentistry, ear and flea treatment, dietary change
Blood results at presentation	12.8.91	12.8.91	12.1.93	5.2.93	27.12.92	19.12.92	27.11.92	28.10.92
RCC ($\times 10^{12}$/litre)	6·0	5·6	5·27	6·03	Not done	6·34	7·16	7·21
WBC ($\times 10^{9}$/litre)	5·1	5·2	4·9	5·7	2·6	7·0	6·8	6·8
Neutrophils ($\times 10^{9}$/litre)	3·4	4·0	3·8	4·0	1·0	4·6	4·8	4·9
Lymphocytes ($\times 10^{9}$/litre)	1·5	0·8	1·0	1·3	1·4	1·4	1·2	1·0
Monocytes ($\times 10^{9}$/litre)	0·1	0·2	0·1	0·3	0·1	0·4	0·1	0·5
Eosinophils ($\times 10^{9}$/litre)	0·2	0·3	0·0	0·1	0·2	0·5	0·7	0·3
Repeat blood results	17.6.92	17.6.92	6.3.93	11.3.93	17.3.93	13.1.93	12.1.93	17.12.92
RCC ($\times 10^{12}$/litre)	7·1	7·4	7·68	8·83	9·2	Not done	7·29	6·98
WBC ($\times 10^{9}$/litre)	8·5	8·2	7·8	8·0	6·5	14·4	9·3	14·0
Neutrophils ($\times 10^{9}$/litre)	4·8	5·9	5·0	4·1	2·7	11·7	7·4	11·1
Lymphocytes ($\times 10^{9}$/litre)	2·7	1·7	2·2	2·2	3·3	2·0	1·3	1·3
Monocytes ($\times 10^{9}$/litre)	0·4	0·4	0·6	0·6	0·1	0·3	0·2	0·4
Eosinophils ($\times 10^{9}$/litre)	0·5	0·2	0·0	1·1	0·4	0·4	0·4	1·3
% change								
RCC ($\times 10^{12}$/litre)	18	32	46	46	NA	NA	2	-3
WBC ($\times 10^{9}$/litre)	67	58	59	40	150	106	37	105
Neutrophils ($\times 10^{9}$/litre)	40	47	31	2	170	154	54	126
Lymphocytes ($\times 10^{9}$/litre)	80	112	120	69	136	42	8	30

DLH Domestic longhaired cat, DSH Domestic shorhaired cat, M(E) Entire male, F(S) Neutered female, F(E) Entire female, GA General anaesthesia, RCC Red cell count, WBC White blood cell count, NA Not applicable

A recurrent complaint was that the male had 'anxiety attacks'. Several theories and treatments had been propounded by different clinicians in the practice but all to no avail. At the time of the animals' annual vaccinations the owners were not aware of any unusual features concerning their pets, but a clinical examination revealed a sparse hair coat and marked generalised periodontitis in both animals. Up until that time dietary advice had been to feed dry dog food, table scraps and the occasional raw bone. The raw bone component had been progressively overlooked by the owners and their pets' halitosis had been accepted as normal.

The owners were persuaded that radical dentistry involving tooth extractions followed by a change of diet to raw meaty bones would benefit their pets. In an attempt to obtain objective information about the animals, a haemogram was performed. The dogs were discharged postoperatively with a five-day course of amoxycillin and instructions for the feeding of raw meaty bones.

After 10 months the owners reported how delighted they were with the change in health and wellbeing of their animals. The initial change of diet had created some difficulties but a regimen had become well established. Clinical assessment revealed a much-reduced halitosis, although there was evidence of calculus on those teeth which lacked an opposing member in the opposite jaw. The hair coat of both animals was generally more lustrous and the non-specific dermatitis of past years was absent. Comparison blood tests were commissioned and are recorded in Table 1.

The percentage change in cell counts in these two cases prompted further investigation in other cases. Blood tests were undertaken in the next 12 cases presenting at the clinic for radical dentistry. In six cases the cell counts were considered low or at the low end of 'normal' and in these cases the owners were asked to bring their pets in for follow-up screening at a later date. In each instance treatment involved radical dentistry with teeth scaling and, or, extractions together with a postoperative course of antibiotics and a change in diet.

Owner assessment of all the test animals was of a markedly increased vitality and wellbeing; four of the cases (1,2,3 and 4) were only considered to be healthier in hindsight. These cases had been presented for routine vaccination by their owners who were unaware that their animals were unwell.

Fuller clinical findings were recorded in case 8, a 12-year-old female Maltese terrier. The owner's presenting complaint was of a sudden onset lameness of the right hind leg. Clinical examination revealed a generalised dermatitis, bilateral otitis, flea infestation, pansystolic murmur, mammary lump, marked dehydration and severe oral disease. The weight was recorded as 3·8 kg. Postoperative clindamycin was prescribed together with eardrops and a splint applied to the presumed sprained hock. The owners were asked to institute a raw chicken wing diet immediately with the addition of a few table scraps from time to time commencing one week later. Although the majority of teeth had been removed from the maxilla and pre-maxilla, case 8's bodyweight had increased to 4·55 kg within 50 days.

Table 2. Normal values utilised by Macquarie Vetnostics Services (MVS), Veterinary Pathology Services (VPS) and Jacobs and others 1992 (Guelph)

	MVS		VPS		Guelph	
	Dogs	Cats	Dogs	Cats	Dogs	Cats
RCC ($\times 10^{12}$/litre)	5·0-8·0	5·5-10·0	5·5-8·5	5·5-10·0	5·6-8·5	5·0-10·0
WBC ($\times 10^{9}$/litre)	6·0-14·0	6·0-16·0	6·0-17·0	5·5-19·0	6·1-17·4	5·5-15·4
Neutrophils ($\times 10^{9}$/litre)	4·1-9·4	3·8-10·1	4·0-12·0	2·0-13·0	3·9-12·0	2·5-12·5
Lymphocytes ($\times 10^{9}$/litre)	0·9-3·6	1·6-7·0	0·9-5·0	0·9-7·0	0·8-3·6	1·5-7·0
Monocytes ($\times 10^{9}$/litre)	0·2-1·0	0·1-0·6	0·1-0·6	0·1-0·4	0·1-1·8	0·0-0·85
Eosinophils ($\times 10^{9}$/litre)	0·1-1·2	0·2-1·4	0·1-0·5	0·1-0·8	0·0-1·9	0·0-0·75

RCC Red cell count, WBC White blood cell count

Case 8's red cell count was found to have decreased by 3 per cent. However, it should be borne in mind that the initial reading was taken from a markedly underweight, dehydrated animal. By the second reading the animal's bodyweight had increased by 20 per cent and as such it can be assumed that the total circulating erythrocytes would have increased significantly.

DISCUSSION

The eight cases studied provided a strong subjective correlation between increase in health and increase in blood cell counts – this result being demonstrated by a simple percentage change. Whilst correlation does not imply causation, the fact that all blood cell counts moved in the same direction (with one small exception) provides persuasive evidence that either the periodontal disease or diet or both were continuing to influence the overall health of the patients.

Dental treatment and dietary change represent generic concepts that incorporate numerous elements in numerous interrelationships. In the light of the known presence of endotoxins and exotoxins and the immune interaction at the gingival sulcus (Liebana and Castillo 1994), these factors might be considered in the lowering of white cell counts. Cooked, macerated processed food has no cleansing effect on teeth and gingiva,

so can be expected to potentiate periodontal disease. Page and Schroeder (1982) state that in order to produce periodontal disease 'somewhat extreme measures such as placement of ligatures in a subgingival position, mono-infection of the oral cavity, or feeding of grossly abnormal diets are required'. Adverse reactions to ingesta are well known and can affect many systems (Wills and Harvey 1994) which, in turn, can be expected to directly or indirectly affect white cell counts.

This study made no attempt to provide a qualitative assessment of the circulating red and white blood cells. It should not be overlooked that the health of an immune system involves a balance of number and function of immune components. Valid concerns arise that dogs and cats suffering from severe periodontal disease can show cell counts within the standard reference range. In our study, four animals were alleged to be normal by their owners but then showed average red blood cell increases of 35 per cent and white blood cell increases of 55 per cent following dentistry. Since these increases coincided with an increase in the apparent wellbeing of the animals then it would be reasonable to assume that the higher values were closer to the true 'normals' for the subject animals.

It is recognised that the use of reference values is in itself a controversial topic (Lumsden and Mullen 1978). Previously, debate has centred on the statistical treatment of results and the method of population selection for the gathering of data. To the author's knowledge consideration as to periodontal health and diet of subject groups has not been well considered. Details were obtained of three published reference ranges from two large commercial pathology laboratories operating in NSW, Australia. Their data was obtained from animals presented to suburban veterinary clinics and variously assumed to be healthy by their owners and or veterinarians (Macquarie Vetnostics Services 1986, Veterinary Pathology Services 1989 to 1991). Questions regarding unnatural artificial diets were seldom raised and the appreciation of periodontal disease problems of domestic pets was at that time in its infancy. The reference ranges published by Jacobs and others were derived from the University of Guelph between 1989 and 1991. These were colony dogs and cats fed a named commercial diet, it is widely reported that colony dogs and cats suffer from periodontal disease (Brown and Park 1968). It is for these reasons that the reference ranges of laboratories may be suffering distortion.

In a broader context, reference ranges represent an establishment of values from a 'normal control group'. If these values can be so sharply affected, then one must have concerns for the validity of any control group where the subject animals can suffer periodontal disease and at the same time be fed processed food (AAFCO 1993). This sentiment was expressed in another way by the authors of the diet and periodontal disease literature search in the Australian Veterinary Association News: 'Those investigating small animal health problems should also take diet and diet consistency into account when researching systemic diseases – possible confounding effects of diet and poor oral health must be considered in such studies.' (Australian Veterinary Association News 1994).

In our general practice, no further comparative blood testing is performed due to the inconvenience suffered by clients and the costs involved. Standard care does involve, regardless of presenting conditions, the provision of dental care and advice to return to a natural diet (Lonsdale 1993b). The results more than justify our confidence with the rapid resolution and reduction in recurrence of medical complaints.

The research potential of this area remains enormous. Extensive trials can be performed to examine the importance of periodontal disease and diet in both the initiation and maintenance of ill health. The standard range of laboratory parameters can be re-examined as can other less familiar haematological and biochemical systems. The apparent reversibility of immune cell depression in carnivores by correcting periodontal disease and diet suggests a potential model in which to explore immune deficiency syndromes.

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BOOK REVIEW

Manual of Small Animal Diagnostic Imaging. Edited by R. Lee. Published by the British Small Animal Veterinary Association, Cheltenham. Paperback. Price £29.00 (members), £39.50 (non-members), £3.75 postage and packing. 200 pages. 1995.

THE renaming of this manual reflects the rapid evolution of veterinary imaging. These days, as well as conventional radiography many practitioners will be utilising ultrasound; they will also be familiar with what computed tomography (CT) and magnetic resonance imaging (MRI) can offer their patients. It is therefore somewhat disappointing that the changes from the 1989 edition of 'Radiography and Radiology in Small Animal Practice' are more of format than content.

While the introduction does indeed include a brief description of CT and MRI, these imaging techniques are not mentioned in any of the subsequent chapters even when relevant to the structures or conditions being described. Indeed these chapters have had little or no revision from the previous edition. The main change is that the layout of the text has been altered to a two-column format. This is justified as when it is combined with the line diagrams being limited to the width of a column, it does seem to improve the assimilation of the subject matter.

The addition of a chapter on diagnostic ultrasound is welcome but suffers from having only limited accompanying illustrations.

Probably the most significant observation that can be made about this manual of diagnostic imaging is that it contains no radiographs or ultrasonographs. Other BSAVA titles now routinely include numerous radiographs and their absence from this manual does seem rather incongruous. The accompanying line diagrams are of high quality and certainly are more than adequate for the positioning diagrams. However I feel their usual role in this sort of publication should be to aid the readers interpretation of the accompanying radiographs rather than a stylised representation of a lesion or condition.

My worry is that a neophyte in veterinary imaging will sometimes struggle to relate the written descriptions and line diagrams to the images obtained by themselves without supporting high quality radiographic and ultrasonographic illustrations.

This manual does contain a wealth of information on imaging from knowledgeable authors in the field. However, I feel that a more comprehensive revision of the text and illustrations is required before one could justify purchasing it to replace the previous edition.

GRAHAM PECK