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Dear Colin,

Thank you for your July 20 letter and accompanying documentation. That we had opportunity for discussion will indeed prove to be fortuitous.

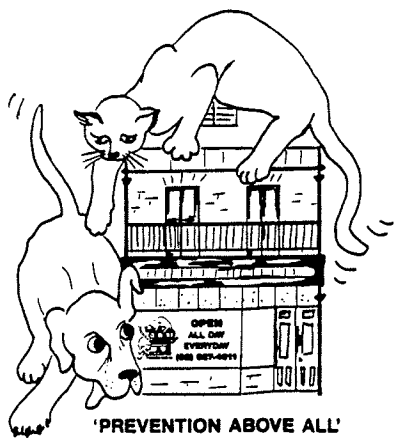
Having access to the early draft form on the proposal and accompanying PEN Periodontal Fund Info is an honour. You will note 'my quick response' early input in numbered sequence. Naturally I expect your proposal to metamorphose over time. The periodontal disease debate rages here. The living legend, Tom Hungerford, was kind enough to nominate me for election to the AVA Executive Committee. The elections will be held in a couple of months time. The fact that you and I shared broadly similar views has been used to bolster my campaign. Is there anything that you would specifically request I do not use or say? I have kept private your reference to the Borthwick report.

Hoping that this finds you well and meeting with much success. With best wishes.

Yours sincerely,

Tom Lonsdale

PS: Thanks for the update of July 29.
Addition to letter
+ JPs Glucose/Parvo.



Effect of the Form of the Diet on the Development of Periodontal Disease in the Dog - A Long-Term Clinical Trial

Introduction

The natural diet of the wild carnivore has a plaque-retarding effect. In rigidly controlling and optimizing the nutritional content, palatability to the pet, and acceptance of commercially available foods by the pet-owning public, pet food manufacturers have created nutrition materials that do not resemble the natural diet of wild carnivores in gross form. In addition, by selective breeding for specific body size, head shape and occlusive pattern, dog owners have created dogs that would not manage well even with a diet that closely resembles that of wild carnivores. By either route, plaque formation is enhanced, inflammatory periodontal disease is more common, and long-term health hazards secondary to intermittent or on-going bacteremia are more likely in an aging pet population. Periodontal disease thus can be considered as a disease of domestication.

Several studies have shown that the form of the diet is much more important in controlling plaque build up and gingival inflammation than is the nutritional content of the diet in dogs. At least over the short-medium term, gross changes in carbohydrate and protein content have no effect on rate of plaque build-up. A diet grossly deficient in calcium leads to secondary nutritional hyperparathyroidism and demineralization of periodontal bone, but does not cause more rapid periodontal tissue break-down. The 'rubber jaw' syndrome of secondary (nutritional or renal) hyperparathyroidism is a periodontosis that does not affect connective and epithelial tissues unless the soft bone permits mobility of teeth that is mechanically harmful.

The general conclusion from reported studies is that a fibrous or dry food diet is beneficial compared to a soft food diet, though canned food and dry food diets have not been compared directly in reported controlled studies in dogs. Even if dry food is somewhat better at retarding plaque formation than is canned food, it is far from optimal: calculus accumulation still occurs, and few studies extend over a long enough period to permit documentation of any disease producing effect of a 'well balanced diet' that is inadequate as a dietary abrasive.

From published studies to date, the optimal oral health diet for dogs contains large pieces, each of which contain calcified material and softer but fibrous material (eg. whole ox-tail or whole trachea-esophagus). These materials may not be attractive to many owners of companion animals, particularly when the material has been partially chewed, and then left for some time as dogs are wont to do. Where esthetics are not a problem, such as in laboratory housed dogs, these materials are effective: for Beagle dogs, half of a raw oxtail given in addition to dry food every two weeks results in accumulation of plaque and calculus per year at a rate that approximates the rate per week in Beagles not given access to the ox-tails. The rapidity of the dietary abrasion effect has been demonstrated with whole versus minced trachea-esophagus fed to dogs - there is a significant increase in gingival fluid flow (a measure correlated with gingival inflammation) within 24 hours when the diet is switched from whole to minced, and vice-versa. Many owners are reluctant to feed 'meat' pieces or bones to their dog or cat because of the purported risk of intestinal irritation, obstruction or perforation. Chicken limb bones that can develop sharp ends during eating, and pork chop or steak bones that have sawn edges that form spikes, probably should not be fed. Large knuckle bones are

acceptable to some owners because they are 'clean', however this means there is little 'flossing' activity during chewing by dogs. The 'ideal' self-flossing material for dogs and cats is a whole prey animal or large part of a carcass that requires much oral work to separate into swallowable sections. Most zoological gardens have reverted to a 'large pieces' menu for carnivores because periodontal disease was rampant during the "mince and mix for balance" era of zoo animal feeding regimes. An often-overlooked source of chewing activity for dogs is the addition of raw vegetables to the diet, particularly items such as broccoli and cauliflower, which are attractive to some animals.

In a Japanese study of 2,649 companion animal dogs, the prevalence of calculus ('present' or 'abundant') was significantly lower in dogs fed dry food or 'left overs' compared to canned, soft moist and home-cooked food. In a study of 1500 dogs in the USA that collected more specific data, regression analysis identified body weight and age as significantly correlated, inversely and directly respectively, with increasing calculus deposition and gingival inflammation; correlation with a dry food diet was less significant. Recently, there has been a strong resurgence in interest in feeding 'natural' diets to dogs and cats to prevent periodontal disease.

Purpose

To compare the effects in dogs of food presented in three forms over a long period. Specific questions to be addressed:

1. Can a 'natural diet' keep the mouth healthy (absence of periodontal inflammation)?
2. Is dry food really more effective than canned food in preventing accumulation of plaque and calculus?
3. Is there a difference between processed foods (dry or canned materials) and the 'natural' diet?

Materials and Methods

Young mature dogs (9-12 months at start of trial) will be studied. Following prophylaxis (thorough dental scaling and polishing, followed by daily toothbrushing so that the calculus, plaque and gingival indices for target teeth is 0), dogs will be randomly assigned to one of three groups of 50 dogs, blocked for sex. Following the initial randomization, the means and standard deviations for age (in months) and body weight will be compared by t-test; if there are significant differences, the randomization process will be repeated to achieve even distribution of age and weight.

A time 0, each dog will be sedated to permit accurate measurement and recording of the oral criteria (see below). This sedation and observation process will be repeated at 6 month intervals for the 3 year (and potentially 5 year) period of the trial.

Criteria to be examined: For both sides of the mouth, the following tooth surfaces will be observed - buccal and palatal/lingual surface of the upper and lower second incisors and upper and lower canine teeth, buccal surface of the upper third and fourth premolar and first molar teeth and the lingual and buccal surfaces of the fourth premolar and lower first molar teeth (total of 18 teeth, 30 surfaces).

Information to be recorded for each tooth surface (for multi-rooted teeth, the most severely affected area on that surface):

Plaque index
Calculus index
Gingival index
Pocket depth
Loss of attachment
Mandibular bone density - parallel position film taken at same location, exposure factors, for densitometric data.
Mobility
Furcation

Diets to be fed:

Diet 1	Commercially available dry dog food, fed dry, ad-lib; water ad-lib.
Diet 2	Commercially available canned dog food, fed once daily; water ad-lib.
Diet 3	'Natural diet' consisting of chicken (large sections, including skin and bone) or beef meat pieces with bone attached, fed once daily, plus mineral-vitamin supplement feed weekly(?); water ad-lib.

(11)

Housing

All dogs to be in individual identical housing, same husbandry practices. (12)

Other observations

Body weight - recorded monthly
Daily - food consumed - YES/NO (report by attending animal husbandry personnel)
Daily physical observation by technician with report to attending veterinarian if abnormalities noted - e.g., gagging, vomiting.
If there are any diet-related problems (e.g., constipation, obstruction, perforation), note type of material, location, seriousness of problem (13)

Justification of number of dogs to be used

Group n of 50 is proposed because of the surmised low risk of intestinal problems - the group will need to be far larger than the 6-10 per group required for identifying statistical differences in periodontal indices.

Costs

Assuming 150 dogs, housed individually for 3 year, at current Penn rates for purchase of dogs and per diem, the animal cost alone would be about $800,000! This does not include scaling and on-going charting costs. This in unlikely to be financially supportable. It may be possible to arrange to 'rent' breeding dogs from a commercial laboratory (eg., Marshall Farms).

Funding Sources

The most likely funding source is a grant from grouped pet food manufacturers, either in a consortium put together for this purpose, or through an industry-wide existing arrangement (eg., Pet Food Institute). No commercial organization will want to see its

product singled out for study with potentially negative results for that product; perhaps the food for diets 1 and 2 could be donated by 3-5 organizations and used for one to several week periods in rotation.

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CANINE-FELINE PERIODONTAL FUND
of the
UNIVERSITY OF PENNSYLVANIA

Introduction

Periodontal disease is the most common clinically evident disease in companion animals. As cats and dogs live longer and healthier lives (thanks to substantial progress in prevention or treatment of other conditions), chronic and progressive periodontal disease becomes more severe.

Periodontal disease results from the accumulation and maturation of bacterial plaque on the surface of teeth. Calculus (dental tartar), the mineral-rich hard substance deposited on the teeth from saliva, provides a rough surface that permits further accumulation of plaque. Plaque-induced inflammation results in disruption of the attachment of the gingival epithelium to the tooth, followed by tissue resorption, bone loss, pocketing or recession of soft tissues, and eventually loss of the tooth.

Periodontal disease causes local pain, and bone and soft tissue infection. In addition, it has recently been documented that increasing severity of periodontal disease is associated with chronic kidney and possibly heart, lung and liver diseases in dogs.

Processed pet foods are nutritionally wholesome, but may not be as effective as a natural diet at keeping the teeth clean. Selective breeding has produced malocclusions. Thus periodontal disease in dogs and cats can be thought of largely as resulting from domestication. Once established, treatment of periodontal disease requires general anesthesia, often in aging or sick animals. With more effective prevention, we can avoid the bad breath, the medical problems and need for anesthesia.

Purpose of the Fund

The overall purpose of the Canine-Feline Periodontal Fund is to provide a means of advancing research, disseminating knowledge and improving prevention and control of periodontal disease in companion animals.

Specific Objectives

1. Examine currently-available epidemiological information to determine the factors that increase the risk of development of severe periodontal disease in dogs and cats.
2. Develop a list of research objectives of prime importance in improving the prevention and control of periodontal disease. Determine the resources necessary to conduct such studies, and develop a plan for identifying funding to meet these costs. Such research objectives could include:

A. Understanding the interaction between nutrition, salivary physiology and calculus deposition.

B. Defining more clearly the effects of the form of the food on development of periodontal disease.

C. Testing of risk factors determined in item 1, above. This may include more specific epidemiological field studies, or controlled studies of laboratory-housed dogs and cats.

D. Conducting microbiological, immunological and pathological studies of the etiopathogenesis of periodontal disease, and the reason for the documented differences associated with various risk factors.

E. Developing a protocol for recording the location, type, extent and severity of oral lesions in cats for use in feline research projects that require measurement of gingivitis, stomatitis or both.

3. Enlarge the existing University of Pennsylvania veterinary dental bibliographic database to include all known data and literature on canine and feline periodontal disease, including data not currently listed in computer accessible sources. This will include all published or unpublished public-domain studies of treatment or prevention of periodontal disease in dogs and cats, including testing of specific products.

4. Develop criteria for testing the effectiveness of products designed to treat or prevent periodontal disease. The Center for Veterinary Medicine of the US Food and Drug Administration has indicated an interest in this objective, and the American Veterinary Medical Association wishes to be kept informed. Information obtained from item 3, above will be a major source for this project.

5. Investigate ways in which products that meet or exceed agreed criteria (from item 4, above) can be recognized as such. This could include ground work for establishment of an independent authoritative agency that would review clinical trial data, and grant use of a 'seal of acceptance' on packaging and advertising.

6. Improve the quality of treatment and prevention of periodontal disease available to the pet-owning public.

<u>Mechanisms to Achieve the Objectives</u>

1. Obtain commitments for funding the development stage of the proposed activities in 1993-94.
2. Identify consultants or an advisory committee and invite their participation.
3. Continue contacts with CVM-FDA to coordinate activities regarding claim testing protocols relevant for veterinary consideration.
4. Invite comment on activities of the Fund by sending letters to potentially interested organizations and by submitting news releases to the Journal of Veterinary Dentistry and the veterinary news magazines.
5. Develop position papers for submission to the Journal of Veterinary Dentistry, and organize symposia on the cause and prevention of periodontal disease.

6. Conduct continuing professional education materials and courses for veterinarians and veterinary technicians to improve the quality of periodontal care made available to the pet-owning public. This could include: intensive, hands-on courses conducted at Penn; coordination with national or regional continuing education programs (eg., LifeLearn V); participate in computer interactive modem-based question-answer forums (e.g., VIN on America-on-line); developing hypertext versions of review papers or textbook material.

STAFF

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Policy on Openness and Conflict of Interest

The University of Pennsylvania has a policy requiring the right to publish material generated by any project conducted under its auspices. Testing protocols generated by this group would be in the public domain. Development of specific marketable products will not be a function of this program. Should a claim-testing review system evolve from this effort, administration of the system would be devolved to a separate organization so that data from specific product claim tests could be kept confidential.

Over the last 10 years, Colin E. Harvey has engaged in consultation activities, product testing or correspondence with ALPO Pet Foods, Booda Products, Fort Dodge Laboratories, Friskies-Nestle, Hill's Pet Nutrition, Nabisco Brands, Nylabone Corp., Ralston-Purina, Rhone-Merieux Inc, Quaker Oats, ST. JON Laboratories, SmithKline Beecham, Henry Schein Inc, Upjohn Co, and Waltham Pet Nutrition, among others. These and other interested organizations will be invited to participate in supporting and setting the agenda for this project. Should the program activities lead to establishment of a claim review and recognition system, C. Harvey will propose inclusion of a conflict of interest policy for the review organization that requires that anyone involved in the development or pre-review testing of a product declare their involvement and remove themselves from the review process for that product.

EFFECT OF THE FORM OF THE DIET ON THE DEVELOPMENT OF PERIODONTAL DISEASE IN THE DOG - A LONG TERM CLINICAL TRIAL

Early and Hopefully Constructive Comment

1. Domestic dogs when protected from predation and other environmental ills gain the extended time necessary for tackling a diet that closely resembles that of wild carnivores.

2. I believe the emphasis should be on toxaemia with bacteria being secondary. Whilst problems show up in the aging pet I believe the genesis is frequently in the immature animal.

3. Probably much of the work you now propose to undertake has already been completed in in-house studies. Specific enquiries directed to the pet food manufacturers could elicit interesting responses.

4. This is conjecture. Natural material provided as food gets eaten.

5. All food can be presented in unnatural ways. Large pig or ox would almost certainly never form part of the natural diet of the small carnivore. Large (dry) bones are hazardous. Animals are more likely to break their teeth on these and if persuaded to chew for too long then wear their teeth prematurely. (Teeth are in the main provided for an expected life-span consuming an expected diet)

6. Large knuckle bones should be discouraged.

7. 'Left overs' appear to be realistic, practical and beneficial when compared with the alternatives. Consequently I believe they should be part of the so-called natural diet.

8. I would be inclined to include a further category here, 'Other Health Consequences'. For good reason I am sure you have chosen nine to twelve month old animals for the trial. Nevertheless I feel opportunities will be missed and bias will be factored in if younger animals are not selected. It is my confirmed view that the genesis of many problems arise around teething. (c.f. Cybernetic Hypothesis) This is especially the case in the four to six month age period. The changes should be plotted during this period.

9. As to bias I believe that anatomical and physiological/pathological changes can occur at an early stage. If puppies start on a natural food from the outset they seem to learn how to use all of their mouth for the apprehending and chewing functions. Naturally they would guard their canines against over use. Animals introduced to natural food late in the piece almost invariably continue to accumulate calculus on the labial surface of the canines.

10. I suggest that time zero should be three to four weeks of age. This being the time the animals commence to eat solids and is relevant to the needs of

puppies being raised in the real world.

11. I believe the selected 'natural diet' should be a real and viable alternative under present commercial conditions. The raw meaty bones component should feature chicken, lamb, rabbit and fish. An agreed table scrap component should be included. I would suggest at a minimum chopped cabbage, apple, carrot both cooked and raw and some cooked pasta, rice and potato.

12. Contrary to orthodox opinion, the relevance of the study would be markedly increased if all the dogs have access to a large long grassed area. In this way they would be able to consume/lick herbage, soil and faecal material just as in the real environment.

13. I am confident that huge difference will emerge on a subjective basis. Charting objective findings are likely to be difficult given our inadequate clinical protocols.

14. You are right that organisations will not wish to have their products exposed to adverse findings. However, chopping and changing the brand-name materials is likely to produce variable findings. eg. Bouts of diarrhoea or refusal to eat the new ration. It can be argued that this would reflect the real world. However many owners tend to be 'brand loyal'.

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Comments

1. I tend to dispute this statement. The concept of cause is semantically unsound and I feel should be relegated to an inferior role.

2 Cause and effect problems again.

3. Processed pet foods are likely to be found chemically unsuitable besides their physical imperfections.

4. Indeed cats do appear to differ markedly from dogs. The swollen jugar, super eruption and neck lesions all in the absence of the conventional 'pockets' makes for quite a different clinical picture. On the subject of cats they would make a much cheaper experimental animal for a major study either replacing or in parallel with the dog study.

5. This data base should be extremely valuable for the AVA Periodontal Disease Sub-committee. Should it be possible I would much appreciate an up-to-date printout for my own study purposes.

6. 'Cause' raises its head yet again. This may have been OK in Koch's day when he was identifying single species bacteria as the significant variable. Where a multiplicity of interrelated entities contribute to the overall outcome we should shun the term.

7. At the end of the survey animals should be destroyed and histopathology be performed. Any survey work should be designed to provide maximum data. Sabotage should be guarded against at every stage.