A Need for Natural-food Control Groups in Periodontal Research

Doctors Watson and Rothwell's commentary (Watson & Rothwell, 1995) is welcome because a rigorous examination and debate of the issues will be required while "periodontal disease remains a serious diet-related problem" (Watson, 1994). Unfortunately, prior to any major advance, there is often much initial skirmishing over terminology. This is all the more common where people have the temerity to cross disciplinary borders, importing with them their understanding of terms. Modern authors speak of periodontitis resulting from an interaction of microbial plaque and host factors. Whilst not qualified to specify correct English usage, I believe it is fair to say that periodontal disease is therefore mediated through immune cells and humoral factors. If this were solely a bacterial disease, then administration of corticosteroids (the principal treatment for immune-mediated diseases) should assist the bacteria and worsen the periodontitis. As we all know, this is not the case.

If I seem to suggest an association between the lesions of canine eosinophilic granuloma and the state of an animal's mouth when fed an unnatural diet, then so be it. In 1992, I published the results of treating a case of feline eosinophilic granuloma by thorough dental scaling followed by a short course of corticosteroids to suppress the immune system and then with long-term natural dietary maintenance (Lonsdale, 1992). Some colleagues and I have enjoyed astounding success utilising a similar approach for a range of canine and feline conditions, notably dermatitis, arthritis, feline lower urinary tract disease and plasma cell pododermatitis. Clients frequently remark: ". . . like a puppy/kitten again!" (Bennet & Pollard, 1993; Lonsdale, 1993). This rejuvenation following correction of oral pathology and institution of a natural diet leads us to postulate that much disease is not readily detectable by the modern diagnostic approach. Only in hindsight do we realise that patients were sick.

We hypothesise that there are severe consequences for an immune system exposed to persistent oral disease and the passage of "foreign" food stuffs through the intestinal tract. We believe that the elastases and collagenases either generated by oral bacteria, or by the host in response to the oral bacteria, act initially on the perivascular collagen in the gums (Harvey & Emily, 1993). It is thought that these products circulate to other parts of the body via the lymphatic and vascular systems, and exert a most powerful effect in collagen-rich tissues, e.g., blood vessels, skin, joints and the cardio-pulmonary system (Hamlin, 1992; Lonsdale, 1993).

We are aware that our hypotheses should not be ushered in unhindered. Quite the contrary, we view critical assessment and stringent testing to be essential. We ask our critics to provide criticism based on rigorous analysis and valid research. In order to validate current research, we believe that it will be necessary to establish "normal" natural-food-fed, control groups of cats and dogs. Unless, and until, this is done, conventional veterinary small animal research and analysis, upon which our critics depend, must be viewed as unanchored, anecdotal circumlocution.

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