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The Dental Pathology of Feral Cats on Marion Island, Part II: Periodontitis, External Odontoclastic Resorption Lesions and Mandibular Thickening

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Summary

Skulls ($n=301$) of adult feral cats from Marion Island were examined macroscopically. Dental calculus was found infrequently (9·0% of cats, 0·76% of teeth), unlike the hard tissue lesions of moderate and advanced periodontitis and tooth loss (presumably due to periodontitis), which were commonly seen (61·8% of cats, 14·8% of teeth). Relatively few of these abnormalities were associated with external odontoclastic resorption lesions, which affected 14·3% of cats and 1·2% of teeth—less than in most recent surveys in domestic cats. Abnormal thickening of the mandibula, found in 39·5% of specimens, was most commonly bilateral (83·3%). The lesions ranged from a focal periosteal reaction, to localized exostosis, to generalized swelling and loss of density, to grossly enlarged mandibles with increased bone density. Mandibular swelling was significantly associated with other abnormalities (periodontitis, dental fractures, external odontoclastic resorption lesions and periapical lesions), but many cases of mandibular swelling were accompanied by only minor dental defects.
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Introduction

Periodontitis is generally considered the most common dental disease of cats (Frost and Williams, 1986). In a study of 200 feline patients, Schlup (1982) found that 57·5% had periodontitis and 77·0% dental calculus; slightly higher figures were obtained for purebred cats. In a similar survey, Crossley (1994) found corresponding figures of 32% and 90%. In peri-urban feral cats, Lüps (1977) found a prevalence of 25·4% for severe periodontitis and of 62·5% for calculus, based on examination of the maxillary fourth premolar. Reichart *et al.* (1984) showed that periodontitis in the cat was associated with the occurrence of external odontoclastic resorption lesions (EORLs). In the cat, oral inflammatory disease appears to play a particularly important role in the pathogenesis of periodontal disease (Pedersen, 1992). Recent surveys, summarized by van Wessum *et al.* (1992), have shown EORLs in 20·0–67·0% of domestic cats; these studies predominantly included cats presented for medical care or dental treatment but also included randomly selected cats, which

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makes comparison difficult. On average, 2.3–4.1 lesions per affected cat were found, the most commonly affected teeth being the maxillary fourth premolar and the mandibular premolars and molars. The prevalence of EORLs appears to have increased from the 1960s onwards. There are few data on the occurrence of EORLs in wild or feral cats, except those derived from two Swiss studies. In one of these studies (Lüps, 1977), no lesions were found in 257 feral cats shot in 1971–1974, although reference was made in passing to "cavities". The other study, which is not fully documented, apparently identified EORLs in the skulls of one of 14 wild cats and 58 of 244 feral cats (Schlup and Stich, 1982).

Mandibular thickening in the cat is well recognized; in a prospective study of 24 cats with mandibular thickening, 12 cases were caused by malignant oral neoplasia, 11 by osteomyelitis and one by a benign bone tumour (Kapatkin *et al.*, 1991). The clinical signs were similar in these cases. Osteomyelitis was associated with retained tooth roots, EORLs, periapical lesions and deep periodontal pocket formation. The malignant tumours were predominantly squamous cell carcinomas and malignant lymphomas. In a large survey of feline oral neoplasia, squamous cell carcinoma accounted for 61.2% of cases, fibrosarcoma 12.9%, malignant lymphoma 2.9% and osteosarcoma 2.4% (Stebbins *et al.*, 1989), the average age of the affected cats being 12·5, 10·3, 10·7 and 12·5 years, respectively. A few isolated cases of odontogenic tumours were diagnosed in relatively young patients. Tumours and tumour-like lesions of the jaw bones described in the cat include osteoma, ossifying fibroma, fibrous dysplasia, feline osteochondromatosis and primary bone sarcoma (Pool, 1990). Osteitis fibrosa associated with secondary hyperparathyroidism of renal origin ("rubber jaw") occurs in the cat, although rarely (Cavanagh and Kosovsky, 1993). Causes of mandibular thickening in other species include craniomandibular osteopathy in the dog (Alexander, 1983), actinomycosis in ruminants, pigs and macropods (Harvey, 1985), and chronic fluorosis in cattle (Harvey, 1985). Fluorosis has been induced experimentally in the cat, but natural cases are apparently unknown (Chavassieux, 1990). Hypertrophy of the facial bones is a prominent feature of acromegaly in the cat (Peterson *et al.*, 1990), as in man (Crespi, 1993). Experimental hypervitaminosis A in young cats resulted in complete tooth loss by the age of 12 months and a low-grade chronic osteomyelitis of the mandibula (Seawright *et al.*, 1970).

The subjects of the present study are described in an accompanying paper (Verstraete *et al.*, 1996). A serological survey in 1974 had failed to reveal evidence of feline leukaemia virus, feline infectious peritonitis virus, calicivirus or parvovirus, but a few feline herpesvirus-positive cats were found (P. G. Howell, personal communication). The objective of the present study was to document the prevalence of dental calculus, periodontitis, EORLs and mandibular thickening.

Materials and Methods

Skulls ($n=301$) of cats with a permanent dentition were examined macroscopically as previously described (Verstraete *et al.*, 1996). Radiographs were not taken. Thickening of the mandible was evaluated by a single investigator familiar with the normal

Table 1
Diagnostic criteria for periodontal conditions, external odontoclastic resorption lesions and mandibular thickening

Observation	*Criteria*
Calculus	Presence of dental calculus on the dental crowns.
Periodontitis stage 2	Evidence of increased vascularity at the alveolar crest (more prominent vascular foramina in, and slightly rougher texture of, the bone of the alveolar crest).
Periodontitis stage 3	Rounding of the alveolar crest; moderate horizontal or vertical bone loss.
Periodontitis stage 4	Widening of the periodontal space; severe horizontal or vertical bone loss; tooth unstable in the alveolus.
EORLs stage 1–2	Lesions confined to cementum and enamel, or progressing into the dentine through enamel or cementum.
EORLs stage 3–4	Lesions affecting cementum or enamel, and dentine, and extending into the pulp chamber with varying amounts of tooth structure lost.
Tooth lost—presumably acquired (i.e. due to disease)	Tooth absent; alveolus or remnant of alveolus visible; alveolar bone shows evidence of dental disease (rounding of the alveolar crest, shallow alveolus, periosteal reaction on alveolar bone, increased vascular foramina).
Thickening of mandible—moderate	Periosteal reaction on a large area of the mandible; or thickening of the mandible with the normal contour still recognizable.
Thickening of mandible—severe	Gross thickening of the mandible with loss of normal contour.
Localized exostosis on mandible	Presence of a well-circumscribed hard, smooth bony lesion, macroscopically confluent with the surrounding cortical bone.
Suspected oral tumour	Pattern of bone resorption and new bone formation suggestive of neoplasia, e.g. "hair-brush" periosteal reaction.

feline oral anatomy; no attempt was made to measure any bone dimensions. The periodontal and related conditions noted and a description of the applicable criteria used for inclusion are listed in Table 1.

The stages of periodontitis were based on the classification described by Holmstrom *et al.* (1992). For obvious reasons, stage 1, which is entirely a soft tissue lesion (gingivitis), was not diagnosed. After a pilot examination of the first 40 skulls, EORLs were classified by a modification of the system described by Lyon (1992). Stages 1 and 2 were combined, as it was virtually impossible to distinguish between them without microscopical examination. Stages 3 and 4 were also combined, as the extent of the loss of tooth structure required to distinguish between them was unclear; root resorption (with the exception of periapical resorption associated with periapical lesions) and root ankylosis, both characteristic of stage 4 EORLs, were not noted macroscopically. Root fractures with loss of the coronal fragments and retention of the root tips were noted as such—not as stage 4 EORLs, because this would have implied an aetiology not obvious on examination. Teeth lost, presumably due to dental disease (especially periodontitis), were distinguished from teeth unavailable for examination for various reasons including congenital absence (see Verstraete *et al.*, 1996).

Logistic regression (Selvin, 1991) was used to evaluate the magnitude and significance of univariate and multivariate associations between (1) periodontitis and dental calculus, enamel hypoplasia, dental fractures, EORLs or periapical lesions, and (2) mandibular thickening and periodontitis, dental fractures, EORLs or periapical lesions.

Results

Dental calculus was found in 9·0% of cats but only on 0·76% of teeth. It occurred only on the carnassial teeth, P4max in particular (Fig. 1). On no

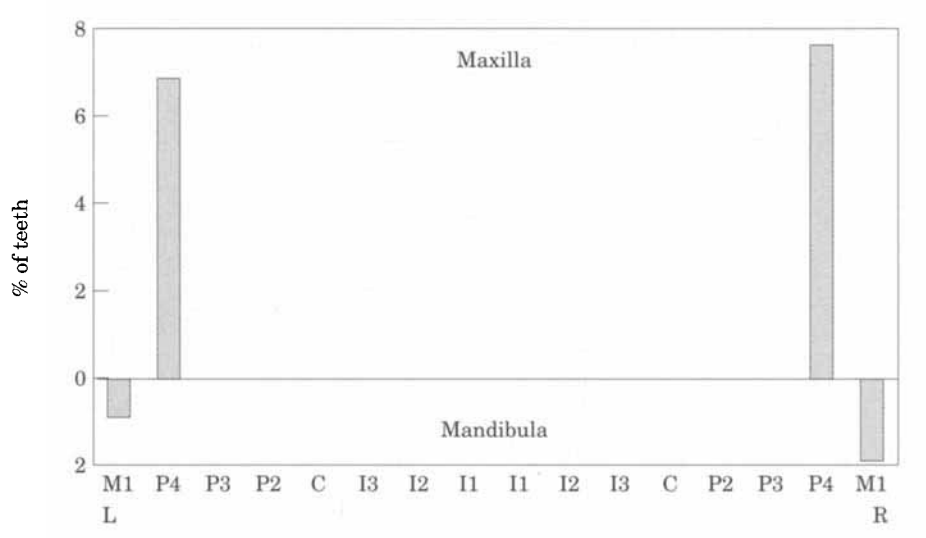

Fig. 1. Distribution of dental calculus.

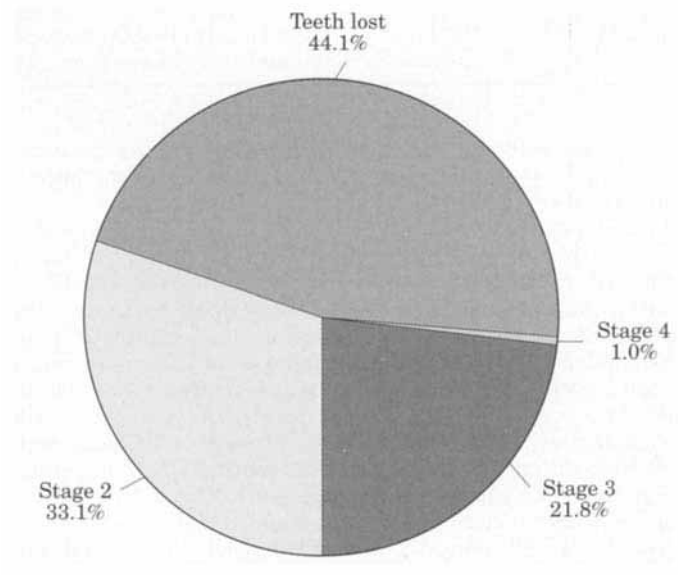

Fig. 2. Prevalence of stages of periodontitis ($n = 862$ teeth) and teeth lost presumably due to periodontitis.

tooth did the calculus cover more than a quarter of the buccal crown surface (equivalent to a calculus score of 1; Logan and Boyce, 1994).

Stage 2–4 periodontitis was found in 48·0% of cats and 8·3% of teeth. If the number of cases with teeth lost, presumably due to periodontitis, is taken into account, the prevalences increase to 61·8% of cats and 14·8% of teeth. The prevalence of stage 2–4 periodontitis and teeth lost, presumably due to periodontitis, is shown in Fig. 2, and the distribution of stage 3 periodontitis

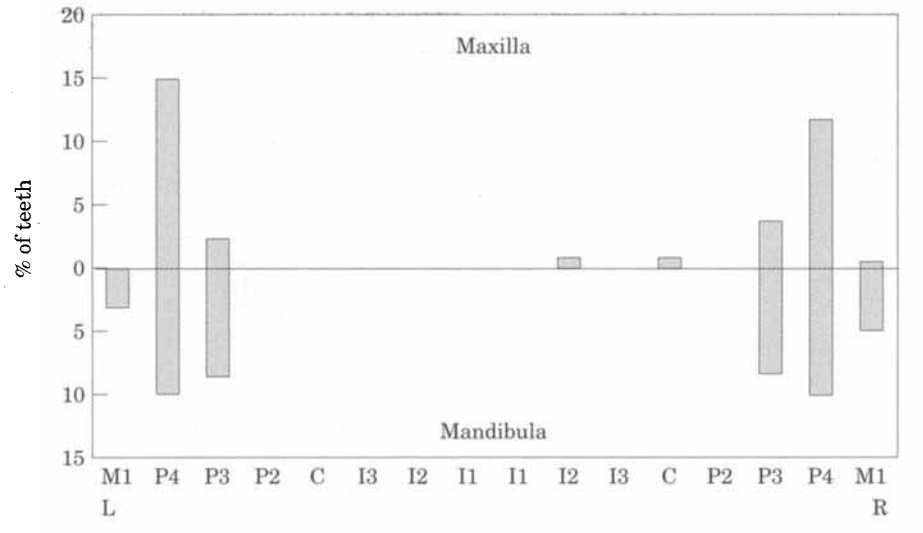

Fig. 3. Distribution of stage 3 periodontitis lesions.

Table 2
Associations between periodontitis (stages 2–4) and presence of dental lesions

Lesions	*Periodontitis*		*Unadjusted*			*Adjusted*		
	Present (n = 143)	*Absent* (n = 158)	*POR*	*95% CI*	P *Value*	*POR*	*95% CI*	P *Value*
Calculus	19	8	2·87	1·22–6·79	0·016	2·15	0·83–5·59	0·11
Enamel hypoplasia	30	44	0·69	0·40–1·17	0·17	0·90	0·49–1·67	0·74
Dental fractures	107	58	5·13	3·12–8·43	<0·001	3·36	1·93–5·85	<0·001
EORLs	41	2	31·35	7·42–132·50	<0·001	18·09	4·11–79·70	<0·001
Periapical lesions	38	9	5·99	2·78–12.92	<0·001	2·5	0·84–5·01	0·12

POR = prevalence odds ratio; CI = confidence interval. Unadjusted results are for univariate analyses; adjusted model controls for the presence of all other explanatory variables in the regression equation.

in the oral cavity in Fig 3. In cats with calculus ($n = 27$), the prevalence of stage 2–4 periodontitis was 70·4%; although this was a significant association ($P = 0·016$), teeth affected by both periodontitis and calculus accounted for only 4·4% of the total number of teeth affected by periodontitis (Table 2). Furthermore, after controlling for the presence of enamel hypoplasia, dental fractures, EORLs and periapical pathology, the association decreased and became non-significant ($P = 0·11$). A significant association ($P < 0·001$) was found between the prevalence of fractures, EORLs and periapical lesions. However, these also accounted for only a small percentage of teeth affected by periodontitis. After simultaneously adjusting for the presence of all other variables, only two factors retained a significant association with periodontitis, namely, dental fractures ($P < 0·001$) and EORLs ($P < 0·001$). The prevalence of periodontitis in cats with enamel hypoplasia was somewhat lower, and there was no significant association between the two defects.

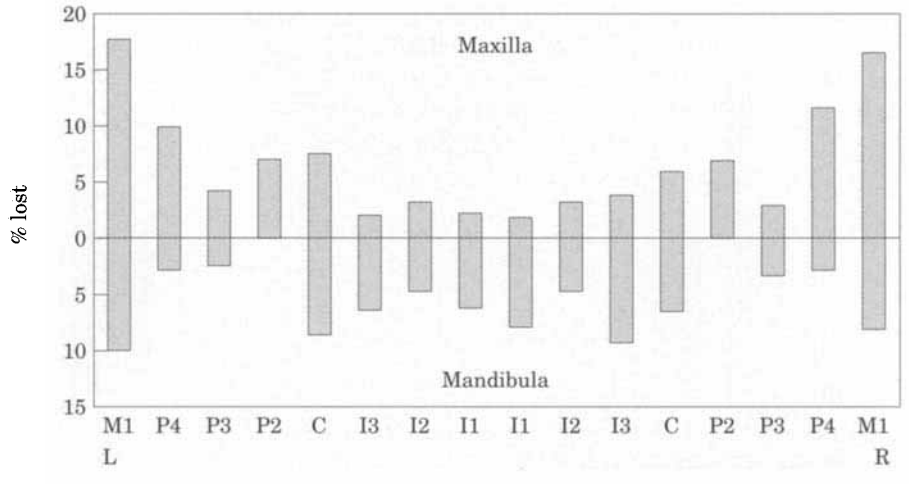

Fig. 4. Distribution of teeth lost presumably due to periodontitis.

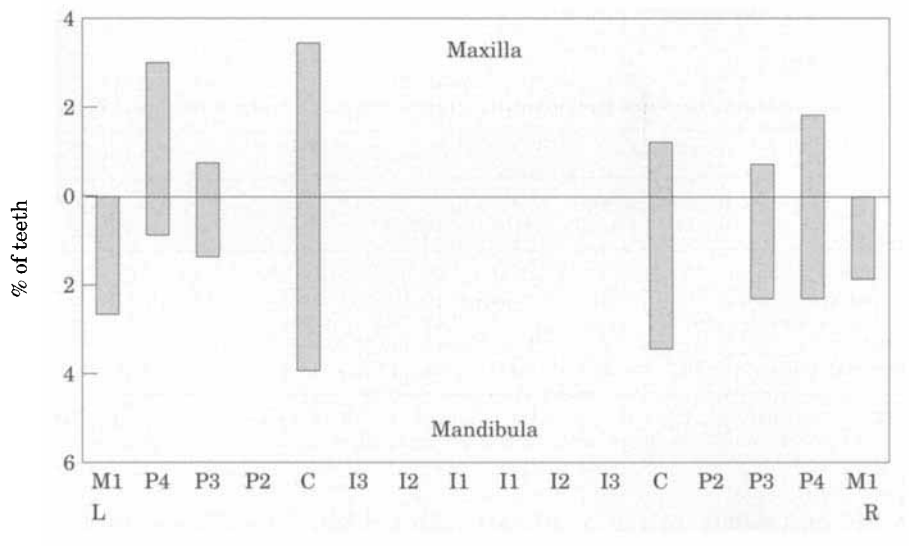

Fig. 5. Distribution of external odontoclastic resorption lesions (all stages combined).

Teeth lost, presumably due to periodontitis (possibly including periodontic-endodontic lesions) numbered 380 (6·5%) (Fig. 4). Incisors, canines, P2max and M1max were frequently lost; this differed from the distribution of stage 3 periodontitis lesions (Fig. 3).

EORLs found in 14·3 of cats, affected only 1·2% of teeth (mean, 1·4 lesions per affected cat). Because the proportions (32:30) and distribution of stage 1–2 and stage 3–4 lesions were virtually identical, the data are pooled in Fig. 5. No EORLs were noted on the incisor, P2max or M1max teeth. The teeth most commonly affected were the left canines, closely followed by the right canines, and upper and lower carnassials.

It should be noted, however, that root fractures, with loss of the coronal

fragment, were recorded as root fractures, even though EORLs may have been the underlying cause. Root fractures were found in 27·9% of cats, affecting 2·4% of teeth (see Verstraete *et al.*, 1996).

Both mandibles were missing in 73 cats. Of the remaining 228 animals, 90 (39·5%) showed mandibular thickening or exostosis. This was bilateral in 75 (83·3%) of these cases; unilateral thickening was found in 10 (11·1%) left and 5 (5·6%) right mandibles. The sexes were equally represented (33 male, 35 female) in the animals whose sex was known. Moderate thickening was found most commonly (80·6%), followed by severe thickening (13·3%), and exostosis (11·5%). The latter was occasionally found in combination with thickening.

The appearance of the thickening or periosteal reaction was variable. Some specimens showed a distinct periosteal reaction on the buccal, ventral and particularly the lingual aspect of the body of the mandible (Fig. 6). In most cases, however, the distinction between periosteal new bone formation and the underlying cortical bone was lost and the thickened mandible appeared to consist of smooth, dense cortical bone (Figs 7 and 8). In a few cases the periosteal new bone formation was irregular and consistent with osteomyelitis (Fig. 9).

The thickening of the mandibles sometimes resulted in occlusal contact between P3max and P4mand, with consequent attrition. A traumatic occlusion was occasionally seen, sometimes causing the P4max to impinge on the hypertrophied mandibular bone buccally to the mesial root of M1mand (Fig. 7). This may have led to the relatively frequent occurrence of an exostosis on the buccal aspect of the mandible at P4mand-M1mand. However, this abnormality also occurred without concurrent malocclusion or dental lesions (Fig. 10A).

In a few cases periosteal new bone formation was also noted on the skull, hard palate and tympanic bullae (Fig 10B and C). Apart from a localized periosteal reaction associated with periapical lesions and dental sinus tract formation, no maxillary thickening was found.

One lesion was suspected to be neoplastic (possibly osteosarcomatous) in origin, from the nature and extent of the periosteal reaction and new bone formation.

Table 3 shows that, on univariate analysis, periodontitis, dental fractures, EORLs and periapical lesions were all associated with mandibular thickening ($P \leq 0{\cdot}001$). After simultaneously controlling for the presence of all four measures, only periodontitis and dental fractures maintained this association ($P < 0{\cdot}05$).

Discussion

The diagnosis of periodontal lesions from dry feline skulls is inherently flawed, as soft tissue lesions, so important in pathogenesis, cannot be detected. However, the hyperaemia associated with inflammation is reflected in the increased vascular foramina and rough texture of the bone of the alveolar process, and bone loss is easily discernible. No references to periodontal indices designed for use on dry skulls could be found, with the exception of a 3-stage

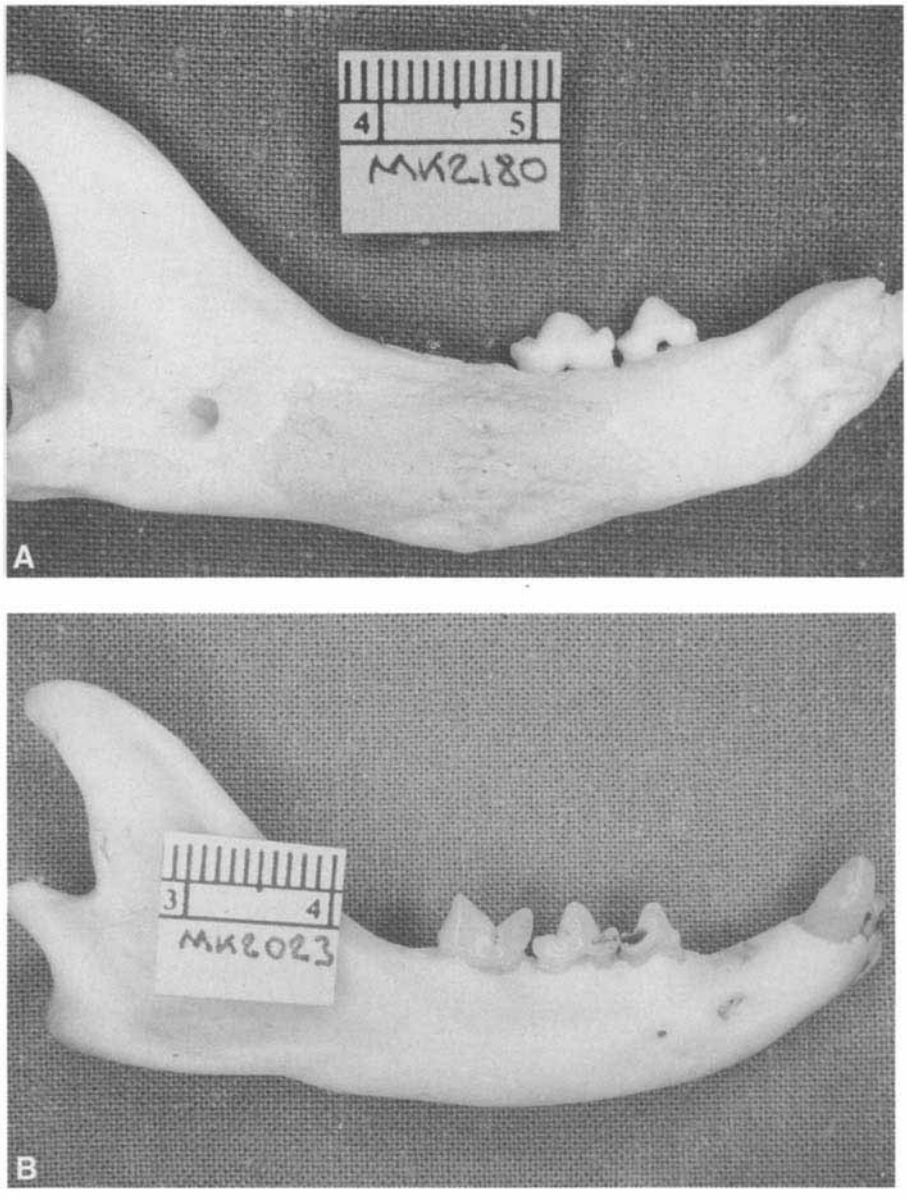

Fig. 6. Periosteal reaction on (A) the lingual aspect of the mandibular body, and (B) the ventral aspect of the mandibular body.

system, used in archaeology, which mainly quantifies horizontal bone loss (Brothwell, 1981). In the present study, a veterinary method of classifying periodontitis (Holmstrom *et al.*, 1992) was adapted for use on skulls.

The nature of the specimens and the method of preparation may have accounted for the relatively low prevalence of stage 4 periodontitis (Fig. 2). Some stage 4 periodontitis teeth may have been lost during the preparation of the specimens and counted as teeth missing due to dental disease. Inclusion

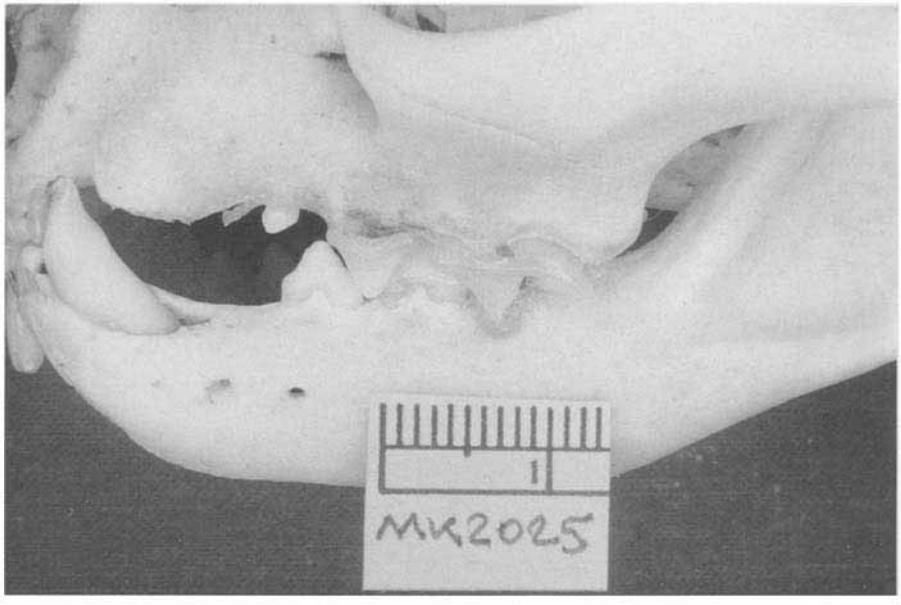

Fig. 7. Severe thickening of the left mandible. A complicated crown fracture of CmandL and associated endodontic-periodontic lesion were present. Note the traumatic occlusion of P4maxL engaging the overlying mandibular bone.

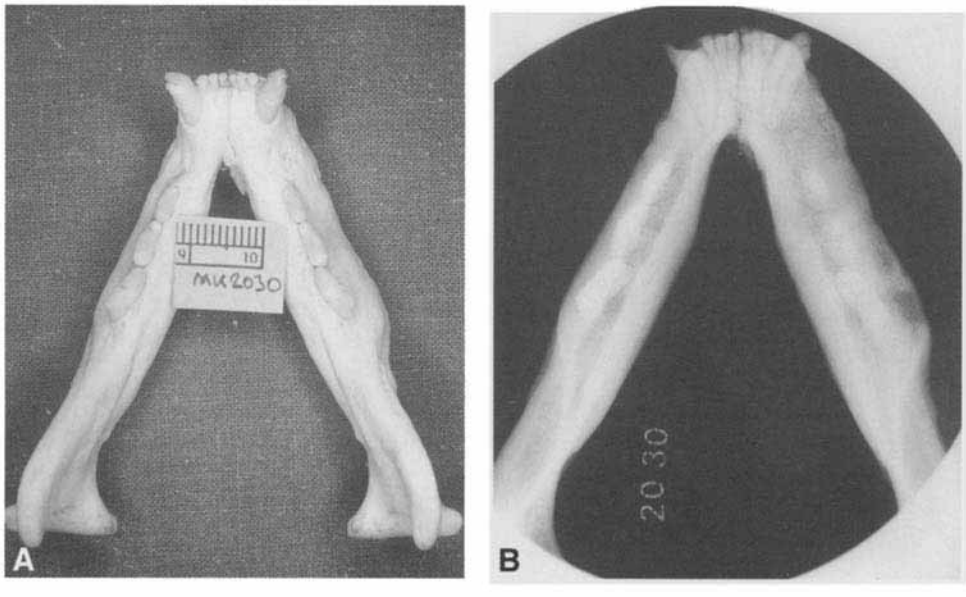

Fig. 8. (A) Bilateral severe mandibular thickening with complicated crown-root fractures of both Cmand, but no evidence of periapical lesions. (B) Occlusal radiograph of the same case; note the apparent density of the mandibular bone.

of such teeth in the assessment of periodontitis seems justifiable, especially as teeth missing as a result of artefact, dental disease or true hypodontia were clearly distinguished.

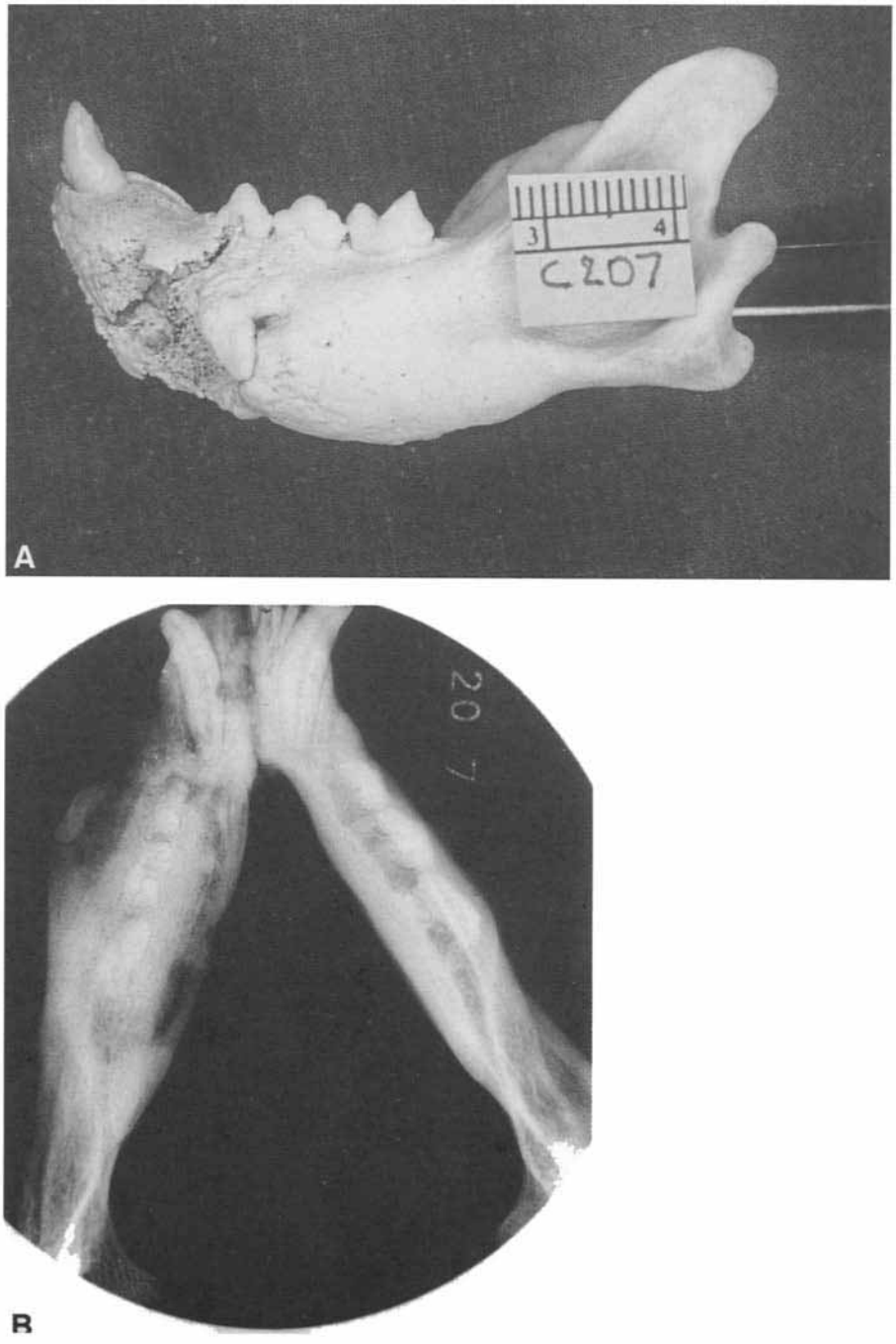

Fig. 9. (A) Severe mandibular thickening with irregular new bone formation, suggestive of osteomyelitis; a complicated crown fracture with periapical lesions of CmandL, stage 3 periodontitis of P3mandL and stage 2 periodontitis of P4mandL were present. (B) Occlusal radiograph of the same case; the right mandible is also thickened and a complicated crown fracture (without periapical lesions) of CmandR is present.

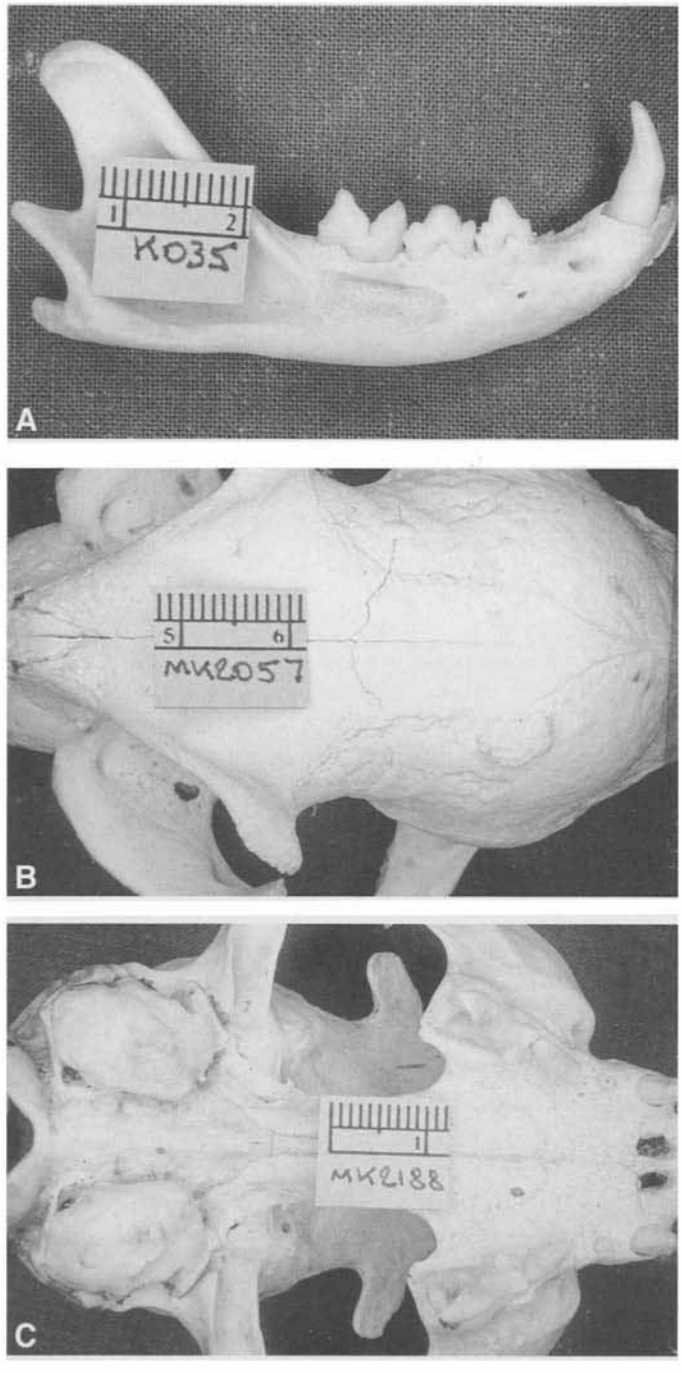

Fig. 10. (A) Localized exostosis; no dental lesions present and premolar-molar occlusion normal. (B) New bone formation on the left parietal bone. (C) Periosteal reaction on the hard palate and new bone formation on the tympanic bullae.

Table 3
Associations between mandibular thickening and presence of dental lesions

Lesions	*Periodontitis*		*Unadjusted*			*Adjusted*		
	Present (n = 143)	*Absent* (n = 158)	*POR*	*95% CI*	P *Value*	*POR*	*95% CI*	P *Value*
Periodontitis	66	24	3·91	2·19–6.96	<0·001	2·10	1·07–4·14	0·032
Dental fractures	80	10	10·71	5·12–22·43	<0·001	7·53	3·47–16·35	<0·001
EORLs	25	65	3.15	1.56–6.40	0·001	1·20	0·51–2·86	0·68
Periapical lesions	26	64	4·27	2·02–9·01	<0·001	1·57	0·66–3·73	0·31

POR = prevalence odds ratio; CI = confidence interval.
Unadjusted results are for univariate analyses; adjusted model controls for the presence of all other explanatory variables in the regression equation.

Dental calculus was found on few teeth, mainly the carnassial teeth. The low prevalence can be attributed to the abrasive nature of the cats' diet. Reichart *et al.* (1984), in a study of 15 domestic cats, found dental calculus on the buccal surface of 32% of premolars and molars. In Switzerland, 77·0% of a hospital population of cats and 62·5% of feral cats whose diet consisted mainly of household food were affected (Lüps, 1977; Schlup, 1982).

The high prevalence of periodontitis in the present population of feral cats resembled that in domestic cats presented for veterinary care (Schlup, 1982; Crossley, 1994). In peri-urban feral cats, a prevalence of 25·4% was noted (Lüps, 1977). The cause of the frequent occurrence of periodontitis in Marion Island cats is unclear. Periodontitis was associated (Table 2) with calculus, fractures, EORLs or periapical lesions; however, such associations accounted for only a small percentage of the teeth affected by periodontitis. Three other explanations come to mind. First, there may have been a genetic predisposition, although van Aarde and Robinson (1980) concluded that a considerable degree of genetic diversity was present in the founder group. Second, there may have been an underlying systemic disease. Metabolic diseases, such as hypervitaminosis A caused by an all-liver diet, may result in oral lesions in the cat (Seawright *et al.*, 1970). However, it was evident from the gastric contents that the cats ate whole avian carcasses (van Aarde, 1980). Many immune-mediated and infectious diseases (e.g. calicivirus infection) also cause oral lesions (Pedersen, 1992). Third, the sharp, splintering bones of large marine birds, which constituted 95·6% of the cats' diet may have resulted in gingival trauma, leading to periodontitis.

The prevalence of EORLs was lower in this study than in any other recent survey (van Wessum *et al.*, 1992). The pathogenesis of EORLs is unclear (Okuda and Harvey, 1992), but the occurrence of such lesions in the present study demonstrates that they are not confined to domestic cats or induced by commercial cat food. The findings do not support the suggestion that a calcium-poor diet and poor mineralization of the jaw bones are associated with EORLs (Harvey and Emily, 1993); the diet of the feral cats was rich in minerals and no evidence of hypomineralization of the jaw bones was evident. Possibly EORLs are associated with the inflammatory response of oral tissues in the cat. Resorption, therefore, mainly occurs with periodontitis. Root

resorption was occasionally seen in association with complicated dental fractures and consequent periapical lesions. Periodontal disease in the feral cats may have differed from that in domestic cats in terms of aetiopathogenesis; as a result, the prevalence of concurrent EORLs may also have been somewhat different.

The high prevalence of periodontitis and, to a lesser extent, of EORLs, may have contributed to short life-expectancy, but it is difficult to reconcile this notion with the cats' successful colonization of the island.

Enlarged mandible was an unexpectedly common finding. Such thickening, associated with dental lesions or neoplasia, is recognized in the cat (Kapatkin *et al.*, 1991), but its incidence is low.

One specimen in the current study was suggestive of oral neoplasia. The diagnosis of neoplasia in dry skulls is inherently flawed as soft tissue lesions cannot be detected, subtle bone infiltration is indistinguishable from the bone changes associated with hyperaemia and inflammation, and histopathological typing is not possible. However, bone tumours occur in only 3·1–4·9 per 10^5 cats (Pool, 1990) and inductive odontogenic tumours are probably even rarer. Moreover, oral neoplasia is usually seen in cats of advanced age (Stebbins *et al.*, 1989), whereas the feral cats were relatively young (van Aarde, 1983). It is therefore most unlikely that neoplasia could have accounted for a substantial number of the cases of mandibular thickening.

Periodontitis and dental fractures were significantly associated ($P=0·032$ and $P<0·001$, respectively) with mandibular thickening. The concomitant presence of mandibular thickening and dental lesions may sometimes have been a manifestation of a causative relationship, especially in the instance of extensive periapical lesions. The latter, as well as the avian bone splinters which probably resulted in gingival trauma, may explain the cases in which the new bone formation was irregular and suggestive of osteomyelitis.

Libke and Walton (1974) described a case of actinomycosis of the mandible in a cat. Other published causes of feline mandibular enlargement include acromegaly (Peterson *et al.*, 1990) and osteitis fibrosa (Cavanagh and Kosovsky, 1993), both of which are rare.

Hypervitaminosis A, which can cause dental lesions, would, had it occurred, probably have resulted in loss of male fertility (Seawright *et al.*, 1970); this, however, would have precluded the population increase at the reported rate of 17–23% (van Aarde, 1984).

No reference could be found to spontaneous fluorosis in the cat, but this species, which has been used in experimental studies of fluorosis (Chavassieux, 1990), has a high fluoride plasma clearance, extra-renal clearance and renal clearance (Whitford *et al.*, 1991). An increase in bone density and cortical thickness was noted in kittens receiving fluoride (Burkhart and Jowsey, 1968). Enamel mottling due to hypomineralization is a prominent feature of fluorosis in man and animals (Richards, 1990). Enamel hypoplasia as a result of fluorosis occurs only at high doses. The prevalence of enamel hypoplasia in this population of feral cats was high (Verstraete *et al.*, 1996), but whether this was true hypoplasia or hypomineralization resulting in weaker enamel, prone to chipping off, was not established. The cats were probably often subject to

maxillofacial trauma, but the presence of exostoses on other parts of the skull suggests an underlying metabolic disease. However, no information is available on fluoride intake.

References

Alexander, J. W. (1983). Selected skeletal dysplasias: craniomandibular osteopathy, multiple cartilaginous exostoses, and hypertrophic osteodystrophy. *Veterinary Clinics of North America: Small Animal Practice*, **13**, 55–70.

Brothwell, D. R. (1981). *Digging up Bones. The Excavation, Treatment and Study of Human Skeletal Remains*, 3rd Edit., Cornell University Press, Ithaca, pp. 154–155.

Burkhart, J. M. and Jowsey, J. (1968). Effect of variations in calcium intake on the skeleton of fluoride-fed kittens. *Journal of Laboratory Clinical Medicine*, **72**, 943–950.

Cavanagh, P. G. and Kosovsky, J. E. (1993). Hyperparathyroidism and metabolic bone disease. In: *Disease Mechanisms in Small Animal Surgery*, 2nd Edit., M. J. Bojrab, Ed., Lea & Febiger, Philadelphia, pp. 865–875.

Chavassieux, P. (1990). Bone effects of fluoride in animal models *in vivo*. A review and a recent study. *Journal of Bone and Mineral Research*, **5** (Suppl. 1), S95–99.

Crespi, P. V. (1993). Metabolic and genetic jaw diseases. In: *Oral Pathology: Clinical-Pathologic Correlations*, J. A. Regezi and J. J. Sciubba, Eds, W.B. Saunders, Philadelphia, pp. 458–493.

Crossley, D. A. (1994). Survey of feline dental problems encountered in a small animal practice in NW England. *European Veterinary Dental Society Forum*, **2**, (unpaginated).

Frost, P. and Williams, C. A. (1986). Feline dental disease. *Veterinary Clinics of North America: Small Animal Practice*, **16**, 851–873.

Harvey, C. E. (1985). *Veterinary Dentistry*, W.B. Saunders, Philadelphia, pp. 256–311.

Harvey, C. E. and Emily, P. P. (1993). *Small Animal Dentistry*, Mosby Year-Book, St Louis, p. 223.

Holmstrom, S. E., Frost, P. and Gammon, R. L. (1992). *Veterinary Dental Techniques*, W.B. Saunders, Philadelphia, pp. 107–108.

Kapatkin, A. S., Manfra Marretta, S., Patnaik, A. K., Burk, K. L. and Matus, R. E. (1991). Mandibular swellings in cats: prospective study of 24 cats. *Journal of the American Animal Hospital Association*, **27**, 575–580.

Libke, K. G. and Walton, A. M. (1974). Actinomycosis-like infection in the mandible of a cat. *Modern Veterinary Practice*, **55**, 201–202.

Logan, E. I. and Boyce, E. N. (1994). Oral health assessment in dogs: parameters and methods. *Journal of Veterinary Dentistry*, **11**, 58–63.

Lüps, P. (1977). Gebiß- und Zahnvariationen an einer Serie von 257 Hauskatzen (*Felis silvestris f. catus* L., 1758). *Zoologische Abhandlungen Staatliches Museum für Tierkunde in Dresden*, **34**, 155–165.

Lyon, K. F. (1992). Subgingival odontoclastic resorptive lesions—classification, treatment, and results in 58 cats. *Veterinary Clinics of North America: Small Animal Practice*, **22**, 1417–1432.

Okuda, A. and Harvey, C. E. (1992). Etiopathogenesis of feline dental resorptive lesions. *Veterinary Clinics of North America: Small Animal Practice*, **22**, 1385–1404.

Pedersen, N. C. (1992). Inflammatory oral cavity diseases of the cat. *Veterinary Clinics of North America: Small Animal Practice*, **22**, 1323–1345.

Peterson, M. E., Taylor, R. S., Greco, D. S., Nelson, R. W., Randolph, J. F., Foodman, M. S., Moroff, S. D., Morrison, S. A. and Lothrop, C. D. (1990). Acromegaly in 14 cats. *Journal of Veterinary Internal Medicine*, **4**, 192–201.

Pool, R. R. (1990). Tumors of bone and cartilage. In: *Tumors in Domestic Animals*, 3rd Edit, J. E. Moulton, Ed., University of California Press, Berkeley, pp. 157–230.

Reichart, P. A., Dürr, U.-M., Triadan, H. and Vickendey, G. (1984). Periodontal disease in the cat—a histopathological study. *Journal of Periodontal Research*, **19**, 67–75.

Richards, A. (1990). Nature and mechanisms of dental fluorosis in animals. *Journal of Dental Research*, **69**, (Special Number), 701–705.

Schlup, D. (1982). Epidemiologische und morphologische Untersuchungen am Katzengebiß—I. Mitteilung: Epidemiologische Untersuchungen. *Kleintierpraxis*, **27**, 87–94.

Schlup, D. and Stich, H. (1982). Epidemiologische und morphologische Untersuchungen am Katzengebiß—II. Mitteilung: Morphologische Untersuchungen der "neck lesions". *Kleintierpraxis*, **27**, 179–188.

Seawright, A. A., English, P. B. and Gartner, R. J. W. (1970). Hypervitaminosis A of the cat. *Advances in Veterinary Science and Comparative Medicine*, **14**, 1–27.

Selvin, S. (1991). *Statistical Analysis of Epidemiologic Data*, Oxford University Press, New York.

Stebbins, K. E., Morse, C. C. and Goldschmidt, M. H. (1989). Feline oral neoplasia: a ten-year survey. *Veterinary Pathology*, **26**, 121–128.

van Aarde, R. J. (1980). The diet and feeding behaviour of feral cats, *Felis catus* at Marion Island. *South African Journal of Wildlife Research*, **10**, 123–128.

van Aarde, R. J. (1983). Demographic parameters of the feral cat *Felis catus* population at Marion Island. *South African Journal of Wildlife Research*, **13**, 12–16.

van Aarde, R. J. (1984). Population biology and the control of feral cats on Marion Island. *Acta Zoologica Fennica*, **172**, 107–110.

van Aarde, R. J. and Robinson, T. J. (1980). Gene frequencies in feral cats on Marion Island. *Journal of Heredity*, **71**, 366–368.

van Wessum, R., Harvey, C. E. and Hennet, P. (1992). Feline dental resorptive lesions—prevalence patterns. *Veterinary Clinics of North America: Small Animal Practice*, **22**, 1405–1416.

Verstraete, F. J. M., van Aarde, R. J., Nieuwoudt, B. A., Mauer, E. and Kass, P. H. (1996). The dental pathology of feral cats on Marion Island, Part I: congenital, developmental and traumatic abnormalities. *Journal of Comparative Pathology*, **115**, 265–282.

Whitford, G. M., Biles, E. D. and Birdsong-Whitford, N. L. (1991). A comparative study of fluoride pharmacokinetics in five species. *Journal of Dental Research*, **70**, 948–951.

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