

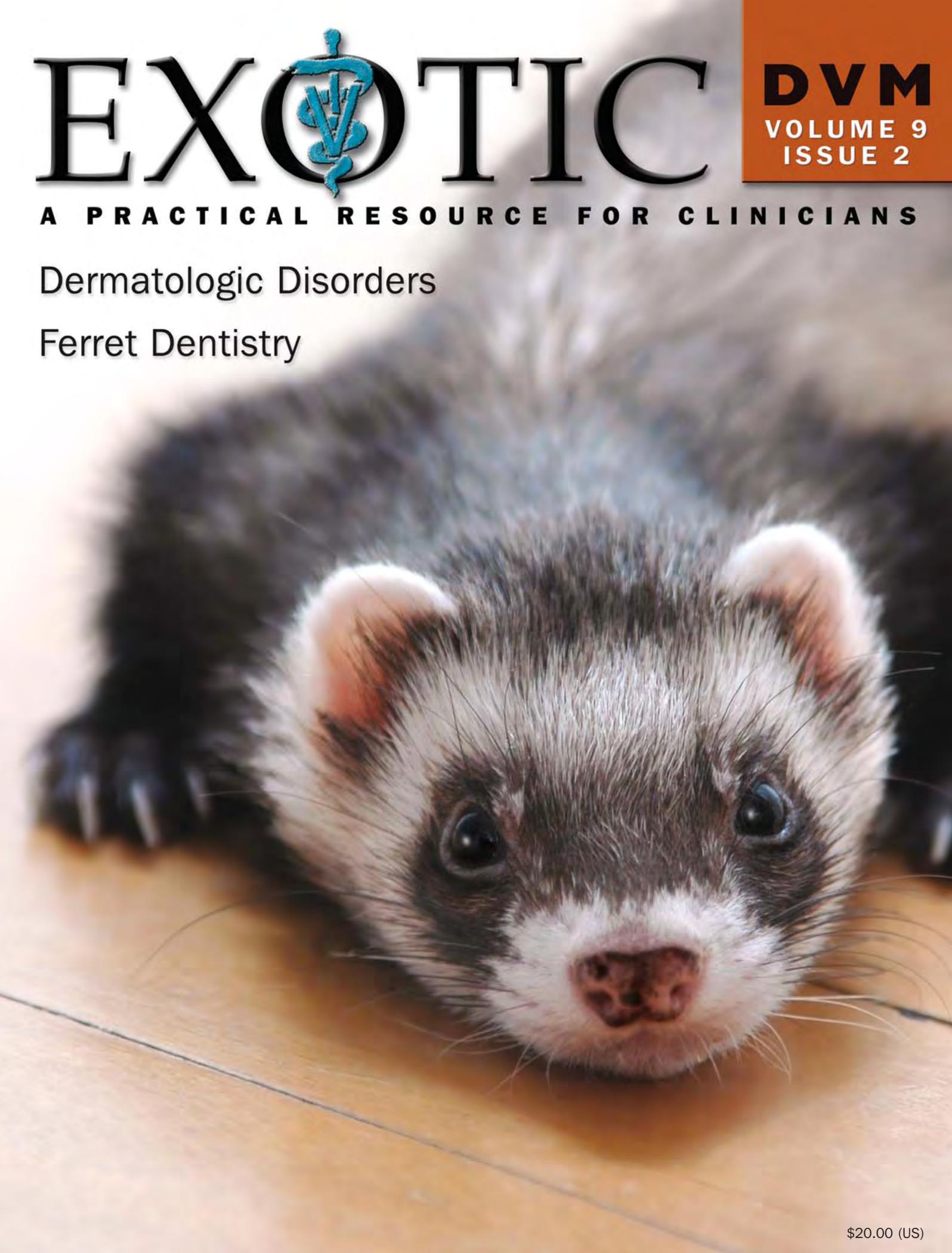
# EXOTIC

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# The Impact of Diet on the Dentition of the Domesticated Ferret

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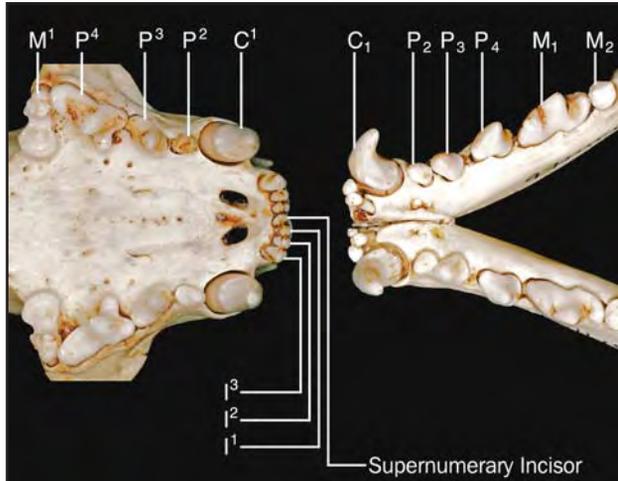
Bob Church worked as a photojournalist for newspapers and wire services before returning to school to earn degrees in biology and anthropology and begin a graduate program at the University of Columbia, Missouri to study zooarchaeology. His current research projects include a study of the odontology and osteology of ferrets and polecats, mammalian behavior and enrichment and ferret nutrition issues. A popular speaker, he has presented at the 2007 North American Veterinary Conference, national and international ferret symposiums, and numerous local events throughout the USA, Canada and Europe.

An ongoing long-term investigation is underway to compare the osteology and odontology of domesticated ferrets (*Mustela furo*), European polecats (*M. putorius*), steppe polecats (*M. eversmanni*), black-footed ferrets (*M. nigripes*), and New Zealand feral ferrets (*M. furo*), all members of the polecat group, subgenus *Putorius* (Order Carnivora; Family Mustelidae). A number of pathologies are present in the dental arcades of domesticated ferrets that are missing or less prevalent in animals not kept in a captive environment: accidental injury, chewing injury, and dietary attrition and disease (Table 1). The goal of this research is to document differences between wild and captive animals, both genetic and environmental, in order to improve ferret husbandry.

Standard archaeological procedures were used to identify dental pathology (dental calculus, reactive bone, bone loss) in museum and skeletonized specimens.<sup>1,19</sup> Ambiguous data was discarded. Only the presence or absence of a particular pathological problem was noted, and due to the likelihood of damage, modification, or loss of dental pathologies during skeletalization and subsequent cleaning and handling, no attempt was made to qualify the extent

**Table 1. Generalized Categories of Dental Pathologies Seen in Domesticated Ferrets and Potential Causative Agents**

Accidental injury	Chewing injury	Dietary attrition and abrasion	Disease
<ul style="list-style-type: none"> <li>• Dental fractures</li> <li>• Electrical burns</li> <li>• Foreign bodies</li> <li>• Gingival damage</li> </ul>	<ul style="list-style-type: none"> <li>• Bedding</li> <li>• Cage biting</li> <li>• Natural objects</li> <li>• Toys</li> </ul>	<ul style="list-style-type: none"> <li>• Dietary “grit”</li> <li>• Food abrasiveness</li> <li>• Occlusal strike</li> <li>• Tooth grinding</li> </ul>	<ul style="list-style-type: none"> <li>• Abscesses</li> <li>• Gingivitis</li> <li>• Neoplasia</li> <li>• Periodontitis</li> </ul>



**Fig 1. Ferret Dental Formula.**

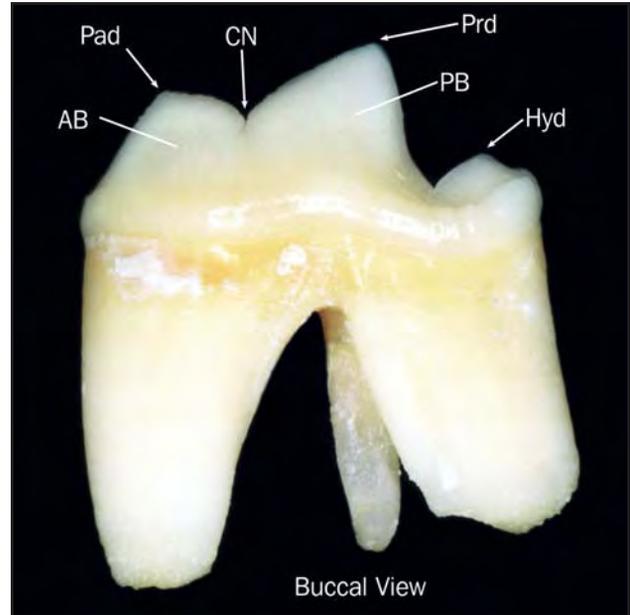
The primitive placental mammalian dental formula is  $I3/3 C1/1 P4/4 M3/3 = 44$ . Most mammals, such as the ferret, have lost one or more teeth during their evolutionary development. Ferrets have lost the first premolar and third molar in their mandibular arcade, and the first premolar and second and third molars in their maxillary arcade. The mandibular carnassial is the first molar ( $M_1$ ) and the maxillary carnassial is the fourth premolar ( $P^4$ ). The dental formula of the ferret is  $I3/3 C1/1 P3/3 M1/2 = 34$ .

Maxillary teeth:

$I^1 - I^2 - I^3 - C - P^2 - P^3 - P^4$  (carnassial) -  $M^1$

Mandibular teeth:

$I_1 - I_2 - I_3 - C - P_2 - P_3 - P_4 - M_1$  (carnassial) -  $M_2$



**Fig 2. Domesticated ferret left mandibular carnassial ( $M_1$ ).**

It is important to have a baseline to establish wear rates. The basic morphology of the mandibular carnassial of a young adult New Zealand feral ferret can serve to show the relationships of the anterior and posterior blades to the carnassial notch.

Patterns of wear on the carnassial blades can demonstrate dietary differences, such as a whole carcass diet compared to a diet of kibble. The trigonid is composed of the anterior and posterior blades and structures, and the talonid is the posterior structure supporting the hypoconid.

AB = anterior blade; CN = carnassial notch; Hyd = hypoconid; Pad = paraconid; PB = posterior blade; Prd = protoconid.

of any condition. Typical morphology of a relatively unworn left mandibular carnassial ( $M_1$ ) of a captive ferret is shown in Fig 2. Mandibular cheek teeth are instrumental in the modification of food for consumption, and their attrition can be interpreted as evidence of dietary abrasiveness. In most cases, if the cheek teeth are worn, at least some wear will be apparent on the carnassial.

Captive domesticated ferrets displayed 90% of the apparent dental pathologies in a group of 1053 animals showing 1717 pathologies. Comparison of the dentition and associated boney structures of ferrets and polecats indicated that the majority of dietary-mediated dental pathologies in domesticated ferrets could be attributed to one or more of the following factors: wear of tooth surfaces from mastication and occlusion (dental attrition), wear from abrasive substances in the environment that are accidentally ingested with food (dental abrasion), gingivitis and periodontal disease, and abscesses (Table 2).

## Dental Calculus

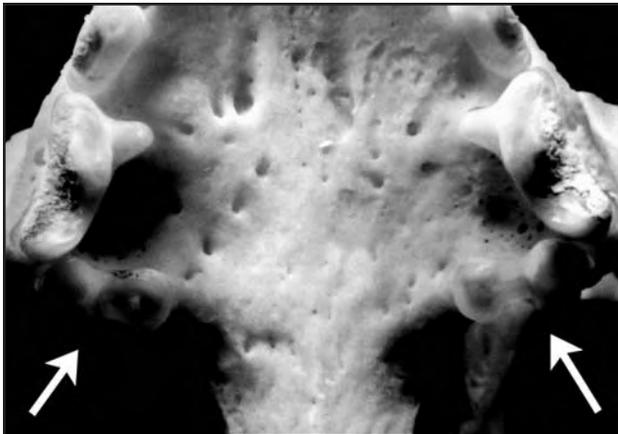
Dental calculus, or tartar, is a hard deposit on the teeth resulting from the mineralization of plaque. Dental calculus has been reported to be insignificant in polecats and ferrets consuming whole-bodied prey, but significant in those eating a pelleted diet.<sup>3,15</sup> These findings are confirmed by this study, where 93.3% of all dental calculus was seen in domesticated ferrets (Table 2; Fig 3). This seems to contradict reports in other species that dental calculus forms on carnivore teeth regardless of diet.<sup>5,7,13</sup>

Still, one of the most profound differences between domesticated ferrets and their wild counterparts in the polecat group is diet. In the United States, most domesticated ferrets are fed a dry kibble diet, while wild polecats and feral ferrets consume whole prey. The biomechanics of consuming a whole carcass are quite different than those of breaking a piece of kibble into small fragments that can be safely swallowed (Fig

**Table 2. Polecat and Ferret Dental Pathologies**

	Ferret	European polecat	Steppe polecat	Black-footed ferret	New Zealand feral ferret	Non-captive polecat group	Total number
<b>Number of individuals</b>	418 3.7 PPA	235 0.2 PPA	211 0.5 PPA	101 0.1 PPA	88 0.1 PPA	635 0.3 PPA	<b>1053</b> 1.6 PPA
<b>Dental calculus</b>	391 93.3% TP 93.5% SP	8 1.9% TP 3.4% SP	15 3.6% TP 7.1% SP	3 0.7% TP 3.0% SP	2 0.5% TP 2.3% SP	28 6.7% TP 4.4% SP	<b>419</b> 24.4% PP
<b>Reactive bone (gum line)</b>	297 88.7% TP 71.1% SP	14 4.2% TP 5.6% SP	21 6.3% TP 10.0% SP	2 0.6% TP 2.0% SP	1 0.3% TP 1.1% SP	38 11.3% TP 6.0% SP	<b>335</b> 19.5% PP
<b>Bone loss (gum line)</b>	259 83.8% TP 62.0% SP	19 6.2% TP 8.1% SP	30 9.7% TP 14.2% SP	1 0.3% TP 1.0% SP	0 0.0% TP 0.0% SP	50 16.2% TP 7.9% SP	<b>309</b> 18.0% PP
<b>Dental abscess</b>	56 74.7% TP 13.4% SP	4 5.3% TP 1.7% SP	14 18.7% TP 6.6% SP	1 1.3% TP 1.0% SP	0 0.0% TP 0.0% SP	19 25.3% TP 3.0% SP	<b>75</b> 4.4% PP
<b>Dental abrasion (cage)</b>	103 98.1% TP 24.6% SP	0 0.0% TP 0.0% SP	1 1.0% TP 0.5% SP	1 1.0% TP 1.0% SP	0 0.0% TP 0.0% SP	2 1.9% TP 0.3% SP	<b>105</b> 6.1% PP
<b>Dental abrasion (object)</b>	69 100.0% TP 16.5% SP	0 0.0% TP 0.0% SP	0 0.0% TP 0.0% SP	0 0.0% TP 0.0% SP	0 0.0% TP 0.0% SP	0 0.0% TP 0.0% SP	<b>69</b> 4.0% PP
<b>Dietary attrition (dental attrition)</b>	369 91.1% TP 88.3% SP	6 1.5% TP 2.6% SP	23 5.7% TP 10.9% SP	4 1.0% TP 3.7% SP	3 0.7% TP 3.4% SP	36 8.9% TP 5.7% SP	<b>405</b> 23.6% PP
<b>Total pathologies</b>	1544 90.0% TP	51 3.0% TP	104 6.1% TP	12 0.7% TP	6 0.4% TP	173 10.1% TP	<b>1717</b>

PP = Percent total pathology PPA = Pathologies per animal SP = Percent species (conspecific) population TP = Percent total (contraspecific) population



**Fig 3. Ferret maxillary dentition showing dental calculus and dental attrition.** There is significant wear to the maxillary molars ( $M^1$ ) that has abraded the tooth material to the level of the pulp cavity. The two arrows point out where the maxillary molars have lost a significant amount of tooth material due to attrition and are in eminent danger of fracture. The right maxillary molar has open pulp cavities. Dental calculus is clearly present on the buccal aspect of the right and left maxillary carnassials ( $P^4$ ), as well as on the third premolars ( $P^3$ ). Not shown in the figure are the reactive bone, bone loss, and exposed roots consistent with severe periodontal disease.

16, see page 37). In the predation and consumption of whole prey, the canines and cheek teeth are repeatedly thrust into yielding materials, which include muscle tissue, tendons and ligaments, tough connective tissue, skin, and fur. These tissues envelope the tooth to the level of the gingiva, rub against them, and act as a combination of a toothbrush and dental floss. In contrast, the shape and size of the ferret cheek teeth force them to act like chisels to fracture the kibble biscuit with minimal crown contact.

## Reactive Bone

Reactive bone is new bone deposited over older bone due to some stimulative action on the periosteum, such as inflammation, infection, or mechanical injury.<sup>18</sup> When present with bone loss and dental calculus, reactive bone likely indicates gingivitis, periodontitis, or osteomyelitis.

Reactive bone was found in 71.1% of domesticated ferrets compared to 6.0% within the polecat group (Table 2; Figs 4-8). This supports the hypothesis that gingivitis and periodontal disease are widespread among domesticated ferrets.



**Fig 4. Multiple mandibular dental pathologies.** This ferret mandible shows multiple dental pathologies, including severe dental attrition in the carnassial ( $M_1$ ), moderate to severe dental attrition of the second and third premolars ( $P_2$  and  $P_3$ ), avulsion of the fourth premolar ( $P_4$ ) and second molar ( $M_2$ ), gingival bone loss, some reactive bone deposits, and the presence of fistulas that could be indicative of a periapical abscess. Not evident in the photograph is an inflated morphology of the bone surrounding the fistulas.



**Fig 5. Bone destruction associated with the presence of a dental abscess.** Prior to euthanasia and eventual skeletalization, a draining fistula was noted on the lingual side of this ferret's left maxillary carnassial ( $P_4$ ). The support for the left maxillary molar ( $M_1$ ) was eroded and eventual avulsion was likely. Other pathologies include dental attrition, reactive bone, and bone loss.



**Fig 6. Multiple dental pathologies in a ferret mandible.** There are extensive dental pathologies present in this ferret mandible, including bone loss consistent with periodontal disease, worn canines due to long-term chewing of bedding, avulsion of the second and third premolars ( $P_2$ ,  $P_3$ ) and second molar ( $M_2$ ), moderate dental attrition of the fourth premolar ( $P_4$ ), and extreme dental attrition of the carnassial ( $M_1$ ). The presence of multiple fistulas and cloacae, reactive bone, a "bumpy-lumpy" surface morphology, and bone loss are consistent with the presence of abscesses.



**Fig 7. Probable periapical abscess in a ferret mandible.** Periapical lesions and abscesses, especially at the fourth premolar ( $P_4$ ), appear to be a common problem in ferrets. In many cases, the second molar ( $M_2$ ) has either been lost or is heavily worn as well (although not seen in this example), and hypothetically might have been the original site of infection. Some dental attrition to the carnassial's anterior blade is evident.



**Fig 8. Severe periodontal disease with probable osteomyelitis.** This ferret jaw shows multiple dental pathologies: dental calculus, reactive bone with involucrum, bone loss, abscesses, extreme dental attrition, and avulsed mandibular molar ( $M_2$ ). This photo represents an uncommon “worst-case scenario;” nonetheless, in the 418 ferret specimens investigated, this type of pathology was seen in 4 individuals, suggesting periodontal disease has significant impact in domesticated ferrets.



**Fig 9. Bone loss and dental attrition.** Although this photo is of a ferret the multiple dental pathologies reflect similar damage in polecats: the carnassial ( $M_1$ ) shows extensive dental attrition, the fourth premolar ( $P_4$ ) and the second molar ( $M_2$ ) have been avulsed, reactive bone is present, isolated bone loss is evident, and there is a probable abscess at  $P_4$ . Note that the bone loss is isolated to the area around the probable abscess site, suggesting that while the ferret suffered from an infection, it did not have periodontal disease. Also note the anterior blade of the carnassial is worn down to the level of the carnassial notch, which is a significant amount of wear. This type of wear is consistent with consuming kibble.

## Bone Loss

Bone loss, when grouped with reactive bone and the presence of dental calculus to form a pattern of pathology, becomes a powerful tool in diagnosing past periodontal disease.<sup>1,5,7,11-13</sup> When seen in a skeleton, bone loss does not mean the animal had periodontal disease at the time of death, but that the condition was present at some point during the lifespan.

The prevalence of bone loss in domesticated ferrets was found to be 62%, while bone loss in the polecat group was only 7.9% (see Table 2; Figs 4-9). Importantly, bone loss in ferrets tended to extend along several teeth in the arcade, while in polecats, it tended to remain isolated to a small area (Fig 9). It is likely that the involvement of multiple teeth indicates periodontal disease, while a small, isolated area of bone loss suggests an infection or injury was present, but not specifically periodontal disease.

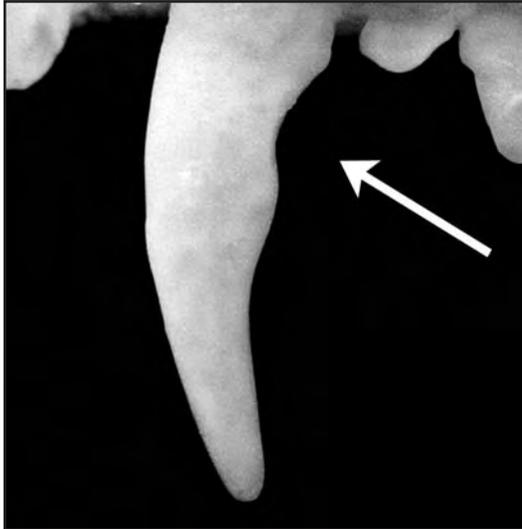
## Abscesses within the Dental Arcade

Abscesses are almost always undercounted when assessed in skeletal remains. An abscess in soft tissue might not leave an identifiable change in bone morphology, and serious infection without an abscess could have similar bone pathology to an active abscess

site. The distinguishing characteristics used here were the presence of a fistulous tract or cloacae (see Fig 4), bone destruction (see Fig 5), and/or morphological changes, such as inflation or bubbling on the cortical surface of the bone (see Fig 6). Periapical lesions and abscesses are also included in this category (see Fig 7). By this definition, some dental abscesses qualify as osteomyelitis (Fig 8) and are a potentially lethal problem due to the dangers of bacteremia.<sup>1,14</sup>

Abscesses within the dental arcade most commonly result from periodontal disease, but other gingival injury or infection can result in their formation.<sup>1,7,11,14</sup> In this study, the high rate of dental pathologies seen in steppe polecats (some individuals showed 5 or more pathologic conditions) could be traced to the presence of facial fractures, most of which had evidence of abscesses (see Table 2). This likely skewed the incidence of abscesses in the polecat group (3% versus 1.2% when steppe polecats are excluded). Domesticated ferrets show an abscess rate of 13.4%, well above the rates seen for the wild polecat group as a whole, even if skewed.

In the wild polecat group, abscesses tend to be small, localized, and associated with mandibular or tooth fractures. This is likely to be because affected animals die from malnutrition or are preyed upon



**Fig 10. Cage biting abrasion in a ferret canine.**

Ferrets, especially those confined to cages for long periods of time, are prone to biting cage wire in an attempt to escape. Over time, distinct wear patterns emerge that are distinguishable from dietary attrition. The notching on the canine indicated by the arrow significantly reduces the biomechanical strength of the tooth, increasing its likelihood of fracture. The rate of identifiable cage biting abrasion seen on ferret teeth is 24.6%, indicating a great potential for fractures of the canines, as well as an important clue to the effectiveness of current ferret husbandry.



**Fig 11. Dental attrition in ferret mandibles.**

Dental attrition is clearly visible in both the right and left carnassials ( $M_1$ ), while most of the remaining dentition is relatively unchanged. In both carnassials, the anterior and posterior blades are worn nearly to the carnassial notch. This “flattening” of the carnassial is a diagnostic feature of kibble-mediated dental attrition in ferrets. Other pathologic conditions include the presence of reactive bone, some areas of bone loss, and the avulsion of both second molars ( $M_2$ ).

before the abscesses become more extensive. Domesticated ferrets, on the other hand, have food and water readily available and can survive much longer, allowing abscesses to become extensive and more potentially life-threatening from a medical point of view.

## Dental Attrition and Abrasion

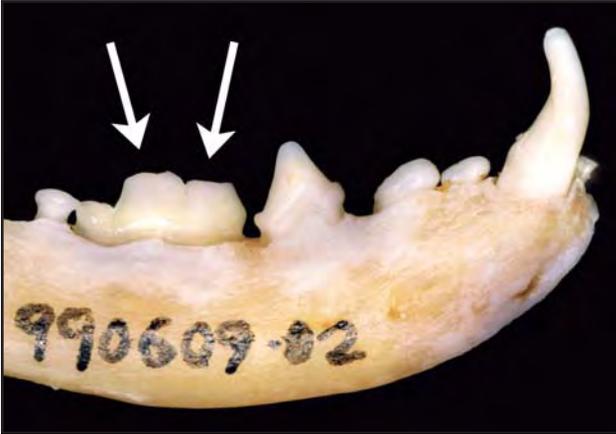
Dental attrition, or tooth wear, is the loss of tooth material due to tooth-to-tooth occlusal contact. It is a lifelong process that starts with the eruption of the crown through the gingiva.<sup>1,14,19</sup> Dental abrasion is the loss of tooth material that is not caused by normal occlusal wear and it can occur on any tooth surface. In ferrets, abrasion is commonly caused by cage biting (Fig 10), inappropriate chewing of fabrics (toys, bedding, towels), grit in the diet, or some plant materials (see Table 2). Wear from food mastication is commonly considered as dental attrition. However, if attrition significantly exceeds the rate found in a random wild population consuming an evolutionary diet, it should be considered pathologic.

Significant attritional changes have been documented in ferrets fed a dry pelleted diet, with unique

flattening of the cheek teeth, and wear facets in different locations compared to polecats consuming a wild diet (Figs 11, 12).<sup>3</sup> In this study, demonstrable dental attrition was found in 88.3% of domesticated ferrets, but only 5.7% of polecats (see Table 2; Figs 3-9).<sup>14,18</sup> This clearly meets the condition set above as pathologic, and the simplest explanation for this is diet.

## The Role of Kibble in Dental Attrition: Hypercarnivory

Polecats and ferrets are primary, obligate carnivores that have evolved a dietary specialization towards hypercarnivory.<sup>6,10</sup> This is reflected in their dentition by the exaggeration of the carnassial (see Fig 2) and the diminution and shift in function of the molariform teeth (Fig 13). There is a loss of grinding function in the molars, and it is not unusual to find the mandibular second molar ( $M_2$ ) has only partial or no occlusion with the maxillary dentition, and in many cases, does not come into actual contact the maxillary first molar ( $M^1$ ). These teeth, located in the back of the jaw where leverage and muscular force are the greatest, probably serve to crack bones and insect carapaces, rather than



**Fig 12. Dental attrition in ferret mandible.** Dental attrition is clearly visible in the right carnassial ( $M_1$ ), while most of the remaining dentition is relatively unchanged. Both the anterior and posterior carnassial blades are worn nearly to the carnassial notch. This “flattening” of the carnassial is a diagnostic feature of kibble-mediated dental attrition in ferrets. Other pathologic conditions include the presence of reactive bone and some areas of bone loss.



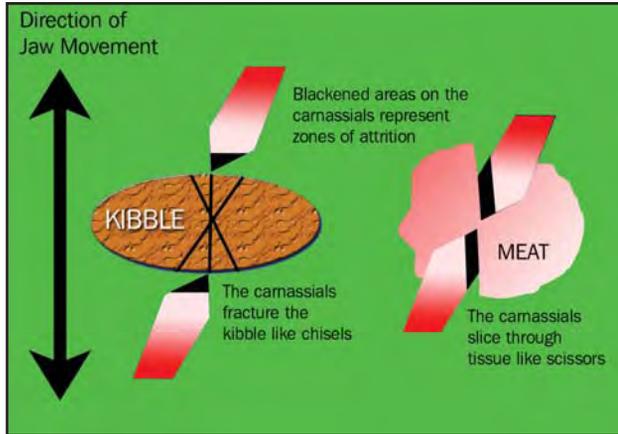
**Fig 13. Normal occlusion of ferret molars.** Polecats, and ferrets by extension, are hypercarnivores that have specific dental adaptations to a non-vegetable diet. One such adaptation is the loss of grinding function in the molars. It is not unusual to find the mandibular second molar ( $M_2$ ) has only partial or no actual occlusion with the maxillary dentition. These teeth, located in the back of the jaw where leverage and muscular force are the greatest, probably serve to crack bones and insect carapaces, rather than for grinding food.



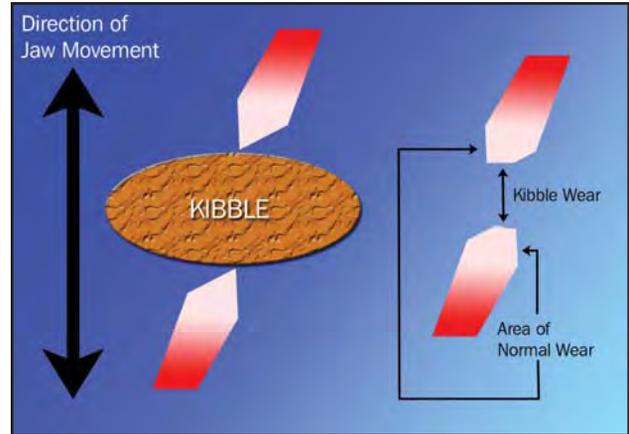
**Fig 14. Mandibular articulation to the ferret skull.** In reducing the loss of bite force and possibility of disarticulation during predation and consumption, the ferret’s mandibles are locked into place on the skull with an exaggerated TMJ that prevents dislocation. This adaptation effectively prevents grinding motions, limiting the jaw motion to a scissor-like up-and-down movement. Likewise, the close interdigitation of the ferret mandibular dentition within the maxillary arcade prevents side-to-side motion. The ferret jaw is capable of cutting, but not of grinding, which is a major adaptation to a hypercarnivore specialization.



**Fig 15. Canines designed for a killing bite.** Ferret canines are designed to safely puncture bone and tissue to render a killing bite. The angles of the canines and cheek teeth align the lines of bite force into classical arch shapes, allowing for great force to be safely exerted on the skull.



**Fig 16. Dietary impact on mastication in ferrets.** The consumption of kibble results in differences in dental attrition compared to those resulting from consuming a meat-based diet, such as whole prey. When consuming meat, the carnassials slice through the tissue like scissors, creating a wear facet that reflects carnassial-carnassial contact. This wear facet is markedly different than the one created when consuming kibble, where the carnassials act like chisels to break the kibble into small fragments suitable for swallowing. This creates a wear facet that reflects carnassial-kibble contact, and explains the “flattened” appearance of the cheek teeth of kibble-fed ferrets.



**Fig 17. Dietary impact of ferrets consuming kibble.** The shift from a tissue-based diet to one of a dry kibble has greatly impacted the ferret’s dietary adaptations. One important change is the shift of the carnassial wear facet from a normal position on the edge of the carnassial to the top surface. This changes the biomechanical stress on a tooth adapted to a different set of bite forces.

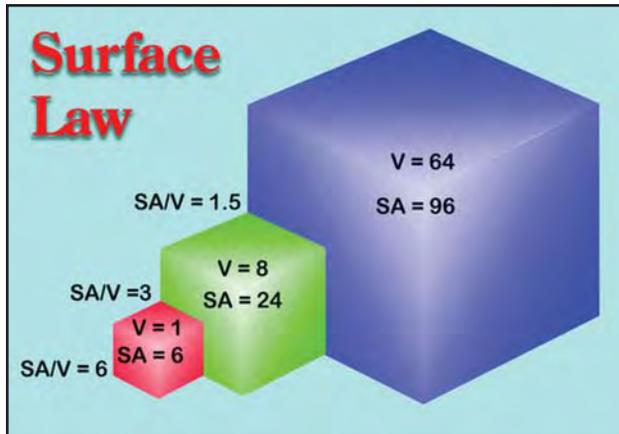
grinding food.<sup>4,8,17</sup> Another adaptation, the overlapping and interdigitation of the mandibular arcade by the maxillary arcade (Fig 14), prevents lateral movement of the mandibles, and thus the grinding necessary to process plant materials and abrasive foods, but allows the dorsoventral movement necessary for cheek teeth and carnassials to shear tissue-based foods. An exaggerated temporomandibular joint (TMJ) effectively locks the mandible into the skull, preventing the loss of bite force during predation and subsequent rendering of the carcass into fragments the ferret can swallow (Fig 14). Further, the maxillary canines and cheek teeth are aligned into what effectively become arches (Fig 15), a common adaptation of carnivores that strengthens the skull without adding bone mass.<sup>9,16</sup>

The biomechanics of these adaptations are markedly different from those of herbivores.<sup>4,8,9,16,17</sup> It is likely that the shift from a whole prey diet to one composed of dry kibble has a deleterious impact on the function of the ferret’s specific dental adaptations (Figs 16, 17), although this has yet to be the focus of a published research study. Kibbles are crunchy and abrasive, a selling point in efforts to reduce dental calculus, but they may cause structural changes to the tooth and underlying boney support and ultimately fractures and loss of teeth.

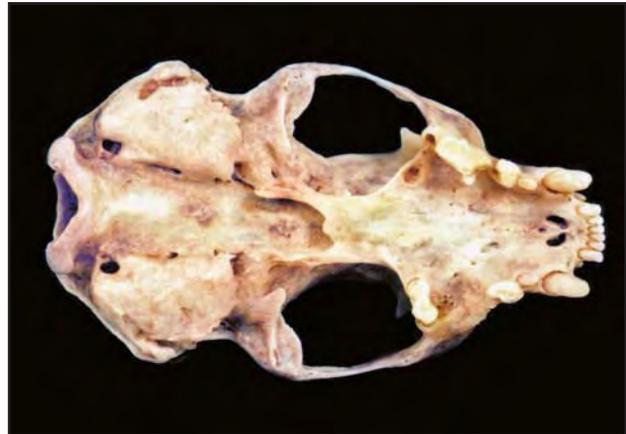
## The Role of Kibble in Dental Attrition: Surface Law

If kibble is wearing down the teeth of ferrets, why isn’t it as notable in cats or dogs, both of which have consumed kibble for decades? Surface Law governs the relationship between surface area and volume<sup>2,20</sup> and may be an important factor in understanding differences in dental attrition between animals of dissimilar sizes.

When comparing carnassials between carnivores, such as the dog, cat, and ferret, the most obvious difference is size. As a tooth increases in size, the interior volume increases proportionately, driving down the surface area/volume ratio (Fig 18). Simply put, smaller teeth also have less dental material that makes up the tooth. The application of Surface Law to dental attrition allows some predictability of wear rates. If the wear rate of dental material against a particular kibble is a centimeter within a specific period of time, then both a cat’s and a ferret’s carnassial should lose a centimeter in height during that time period, assuming the same kibble is consumed. However, because of the difference in interior volume, the small, blade-like ferret teeth wear down proportionately faster than larger, more voluminous teeth of a cat.



**Fig 18. Surface Law.** Surface Law governs the ratio of surface area to interior volume of an object, such as a tooth. As a tooth gets smaller, the interior volume proportionately decreases as well. Simply put, this means that not only is the tooth smaller, but it also has proportionately less interior volume, so there is simply less tooth material to wear away in similar periods of time. The end result is that smaller teeth have accelerated wear rates in comparison to larger teeth of a similar morphology.



**Fig 19. Loss of maxillary dentition due to attrition and periodontal disease.** Pathologies present include moderate to severe dental attrition, periodontal disease, reactive bone, bone loss, and avulsion of the left maxillary carnassial ( $P^4$ ) and right maxillary molar ( $M^1$ ). Dental attrition from a kibble diet wears down the molar dental tissues until the tooth becomes structurally unsound, fractures in the middle, and is lost. A careful examination of the right maxillary molar ( $M^2$ ) shows the buccal edge remains partially rooted, while the lingual portions have been lost. Not apparent in the photo is that the left maxillary molar ( $M^2$ ) is worn down to the pulp cavities and is in danger of fracturing.

Interestingly, wear rates can be remarkably different between the maxillary and mandibular dental arcades. Maxillary dentition is larger than mandibular dentition, so attrition rates will differ according to Surface Law. Also, in chewing, the maxillary arcade is held stationary while the mandibular arcade moves, increasing the abrasion rate to the lower dentition (Fig 19). Further, some ferrets favor chewing in specific areas of the mouth, wearing down one tooth more than the other.

## Summary and Conclusions

Switching from a whole prey diet to a modern convenience diet (dry kibble) has had a profound impact on the dental health of domesticated ferrets. Problems can be divided into two categories: the formation of dental calculus and subsequent related diseases; and accelerated dental attrition and its consequences. These problems are not mutually exclusive, but they require different solutions.

Veterinarians should be aware of the prevalence of dental disease in ferrets and adjust their examinations accordingly. Ferrets with bad breath, facial swellings, loose teeth, and bleeding or puffy gums should have dental radiographs taken to check for abscesses, bad teeth, and bone loss. Veterinarians should also guide

and educate ferret owners in proper oral health practices.

To remove and minimize formation of dental calculus in ferrets that are not consuming a whole prey diet, regular tooth brushing with a non-fluoridated dentifrice, periodic dental inspections, gingival probing, and tooth cleaning and polishing are desirable.<sup>6,9</sup> Commercially available chewing treats, such as gelatin chews or edible sticks, could help reduce dental calculus, stimulate the gums and satisfy the urge to chew.

The captive environment can be modified to prevent dental damage in ferrets that are cage biters or fabric chewers. Cage wire can be replaced or covered and cloth materials removed and substituted with shredded paper bedding.

Modifications to kibble to soften its impact on teeth must be achieved at the manufacturing level. Until then, kibble can be softened with chicken broth or water to lessen its abrasiveness. As a softer diet may increase the rate of deposit of dental calculus, an emphasis on tooth brushing should be concurrent with this recommendation.

Most importantly, more research is needed on the impact of periodontal disease on ferret health, including its involvement in other organ diseases.

## Acknowledgements

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## Comments from a Ferret Practitioner

**Cathy A. Johnson-Delaney, DVM, Dipl ABVP-Avian Practice**

This article raises a number of questions, but the conclusions are still theory. There are several important missing pieces of analysis before one can truly conclude that kibble causes more dental disease than the wild-type diet.

What were the ages of the jaws and teeth examined? This could greatly influence how much dental wear is being seen. Even in very old pet ferrets, I don't see the extent of dental problems and wear illustrated in these skulls.

Dental disease is greatly influenced by dental and gingival flora and pH of saliva. These factors also influence the wear of the teeth. The influence of the diet on the flora and pH of the saliva is relevant to any discussion of abscesses, caries, fractures, and enamel wear. In monkey and human periodontal research, AST (enzyme) levels in the gingiva were found to highly correlate with enamel erosion, gingivitis, and periodontal disease. I have not yet seen this measured in pet ferrets vs wild mustelids, and it will differ depending on sex, age and diet.

Other systemic diseases affect dental disease. How many of the animals studied had major systemic problems, such as

neoplasia, cardiomyopathy, and especially, immune suppression?

The domestic ferret is extremely inbred. As a result, there are alterations in the immune system and probably many other changes. Inbreeding may influence dental health and even formation of the teeth. At least one aberrant amino acid pathway has been identified in the domestic ferret. There may be more, and these can alter dental structure, wear, and repair. Changes in face shape from inbreeding is seen in dogs and cats, and it is notable that the pet ferret does have a different face shape than its wild polecat ancestor.

Neutering/spaying alters muscle mass and face/skull shape. The adult male mustelid has a broad skull with large temporal muscles. In contrast, early neutered male ferrets have a pointed face similar to females, with no large muscle masses.

General nutrition is important. Does processed food have more carbohydrates, influencing pH and flora? I have seen the same dental disease issues in ferrets eating a soft mink diet, canned food or soup as those strictly eating kibble. If whole prey (killed domestic mice or rats) were fed to

captive ferrets instead of kibble, which some ferret owners do, nutritional value may be compromised.

I don't think we can place all blame of dental disease in ferrets on a kibble diet. I see a large number of well cared for pet ferrets that live to 5-7 years of age, and with regular dental care have very little overall wear and have eaten kibble their entire lives. I see more dental problems with the maxillary teeth including general wear, but I have never seen pet ferret teeth worn down to the gum line. While plaque first builds on the maxillary carnassial tooth, that tooth is rarely the one with severe wear or disease of the gums surrounding it. The most common dental problem I see from cage biting and toy chewing is fracture of the tip of the maxillary canine teeth (with or without pulp exposure).

It is important to recognize that dental disease issues are more complex than outlined in this paper. A longitudinal study is needed to compare the hardness and size of pelleted food on the wear of the teeth, while also measuring flora, pH and related factors.



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