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RESEARCH ARTICLE

Focal Palatine Erosion in Captive and Free-Living Cheetahs (*Acinonyx jubatus*) and Other Felid Species

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We examined 1,092 skulls of captive and free-living individuals, representing 33 felid species, to determine the prevalence of focal palatine erosion (FPE). FPE was detected in 3.2% of cats evaluated, including cheetah (*Acinonyx jubatus*) and 14 other felid species. The prevalence of FPE between cheetah (9.4%; $n = 64$) and non-cheetah species (2.8%; $n = 1{,}028$) (χ^2 test; $P = 0.004$) and between captive (5.7%; $n = 246$) and free-living (2.4%; $n = 824$) individuals (χ^2 test; $P = 0.010$) were significantly different, with prevalence between captive (19%; $n = 21$) and free-living (2.9%; $n = 34$) cheetahs approaching significance (Fisher's exact test; $P = 0.064$). FPE was diagnosed with equal prevalence in skulls from individuals in which the lower molars did not meet the palatine bone (60.6%) and individuals in which it did (39.4%; $n = 33$) (χ^2 test; $P = 0.139$). In cheetahs with FPE, one was a captive animal in Germany, one a free-living cheetah from Mali, one captive cheetah from Kenya, and three captive cheetahs of unknown origin. Additionally, we evaluated the medical records of 49 captive cheetahs in Namibia. Of these cheetahs, 48 (98.0%) had clinical signs consistent with FPE, although only 16 of these 48 (39.6%) had perforation of the palatine bone. Based on physical examinations, FPE was diagnosed in two caracals (*Caracal caracal*) and one fishing cat (*Prionailurus viverrinus*) from a North American Zoo. Results from this study confirm FPE in cheetahs outside of Namibia, in a minimum of 15 felid species, and a higher FPE prevalence in captive individuals than free-living ones. Clinical implications of these findings and recommendations for future studies are provided. Zoo Biol 30:1–8, 2011.

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INTRODUCTION

In the early 1980's, a severe palatine disorder termed focal palatine erosion (FPE) was described in a group of captive cheetahs (*Acinonyx jubatus*) from Namibia [Fitch and Fagan, 1982]. FPE, as the name implies, is characterized by oral erosion of the gums with subsequent perforation of the palatine bone in advanced stages (Fig. 1A). This disease was previously thought to be associated with mechanical pressure exerted by the lower first molar on the surface of the dorsal oral cavity, medial to the carnassial (upper PM4) tooth. In the first stage of the disease, clinical signs may include bloody-mucus nasal discharge, halitosis, sneezing, and coughing [Fitch and Fagan, 1982]. As the disease progresses, severe FPE often leads to a localized osteomyelitis, and may result in intermittent chronic septicemia with associated systemic consequences and ultimately death in untreated individuals. FPE has been reported in captive cheetahs as young as 7 months old with complete perforation of the hard palate [Phillips et al., 1993]. Reported treatments include trimming the tip of the lower molars, systemic antibiotics, and possibly surgical repair [Fitch and Fagan, 1982].

It was previously thought that FPE only occurs in captive cheetahs originating from Namibia [Fitch and Fagan, 1982; Phillips et al., 1993]. More recently, it was demonstrated to occur in free-living cheetahs in Namibia [Marker and Dickman, 2004]. A genetic component has been suggested based on the first description of FPE, in which 86% of the FPE-positive cheetahs were from Namibia [Fitch and Fagan, 1982]. Dietary factors have also been hypothesized to play a role in the development of clinical disease, because all animals that exhibited FPE in the study by Fitch and Fagan [1982] were raised on a soft commercially prepared diet. These authors proposed that the lack of biting, tearing, and pulling action, necessary in the capture and consumption of prey, resulted in atrophy of the muscles of mastication causing malocclusion and subsequent FPE [Fitch and Fagan, 1982].

To the authors' knowledge, there is only one published case of FPE in a non-cheetah felid species, a clouded leopard (*Neofelis nebulosa*) [Fitch and Fagan, 1982], and no publications of FPE in cheetah originating outside Namibia. Therefore, the objective of this study was to determine FPE prevalence in cheetahs from different geographic locations and in other felid species.

Fig. 1. (**A**) Cheetah (*Acinonyx jubatus*) skull and mandible with severe focal palatine erosion, notice two perforations in the palatine bone caused by the left lower molar. (**B**) Ventral view of a Sand Cat (*Felis margarita*) skull, showing a perforation of the palatine bone that creates an oral–ocular fistula.

MATERIALS AND METHODS

Skulls ($n = 1{,}077$) from six museums, including the Natural History Museum of Chile ($n = 3$), Natural Sciences Museum of Madrid ($n = 19$), Natural History Museum of Vienna ($n = 35$), Natural History Museum of Tel Aviv ($n = 115$), the Chicago Field Museum ($n = 879$), and the American Museum of Natural History ($n = 26$), were inspected visually. Additionally, 15 skulls from the University of Texas database were evaluated using high-resolution radiography and computed tomography [DigitalMorphology, 2010]. All skulls ($n = 1{,}092$) were evaluated by one of the authors (MZ). Details on the species and numbers of specimens sampled are provided in Table 1. The criterion used to determine FPE in skull specimens was perforation of the palatine bone medial to the upper carnassial tooth (PM4). If a perforation was noted, the skull and mandible were assembled to determine if the perforation was caused by the insertion of one or both lower first molars. Data were collected on each specimen's age, free-living or captive status, and region of origin for cheetahs.

Additionally, the medical records from 49 captive cheetahs in Namibia and 2 caracals (*Caracal caracal*) and 1 fishing cat (*Prionailurus viverrinus*) at a zoological facility in North America were reviewed. Evidence of FPE, including soft tissue inflammation, mucosal depression, bleeding, and bone perforation, was recorded for each individual. All physical examinations were performed and clinical notes recorded by one of the authors (CRS), using a grading score for FPE modified from Marker and Dickman [2004]. The modifications included adding an extra score 0 meaning "no sign of erosion" and modification of score 1 to "little erosion."

Prevalence was defined as the proportion of individuals with FPE, and 95% confidence intervals are provided [Thrusfield, 2007]. χ^2 tests were used to compare prevalence of FPE in captive vs. free-living individuals, cheetahs with other felid species, and between FPE positive cats with and without molar contact. Fisher's exact test was used to compare captive with free-living cheetahs. Results were analyzed using a commercial statistical software package (NCSS, Kaysville, Utah; SPSS, version 13.0, Chicago, IL).

RESULTS

Details of the species sampled, captive vs. free-living status, and FPE prevalence are presented in Table 1, and more detailed information for cheetahs in Table 2. All skull specimens evaluated were from adults and included at least one individual for 33 felid species. All but one live animal in which medical records were evaluated were also adults; the one nonadult was an 8-month-old cheetah with advanced FPE (e.g., bone perforation).

Total FPE prevalence in all felid species evaluated was 3.2% (95.0% confidence intervals; 2.2–4.4%). There were significant differences in FPE prevalence between captive (5.7%; 3.1–9.4%; $n = 246$) and free-living (2.4%; 1.5–3.7%; $n = 824$) individuals (χ^2 test; $P = 0.010$), and between cheetahs (9.4%; 3.5–19.3%; $n = 64$) and other species (2.8%; 2.0–4.0%; $n = 1{,}028$) (Fisher's exact test; $P = 0.004$). The difference in FPE prevalence between captive (19%; 5.4–41.9%; $n = 21$) and free-living (2.9%; 0.07–15.3; $n = 34$) cheetahs approached significance (Fisher's exact test; $P = 0.064$). In the live cheetahs evaluated, 48 of 49 (98.0%; 89.3–99.6%) were

TABLE 1. Number of Skulls with Focal Palatine Erosion/Number of Evaluated Felid Skulls. (Prevalence Estimated with 95% Confidence Intervals)

Species	Captive	Free living	Unknown	Total
All species evaluated	14/246 5.7%; 3.1–9.4%	20/824 2.4%; 1.5–3.7%	1/22 4.5%; 0.1–22.8%	35/1,092 3.2; 2.2–4.4%
All non-cheetah species	10/225 4.4%; 2.4–8.0%	19/790 2.4%; 1.5–3.7%	0/13 0%; 0–2.3%	29/1,028 2.8%; 2–4%
Acinonyx jubatus	4/21 19%; 5.4–41.9%	1/34 2.9%; 0.07–15.3%	1/9 11.1%; 0.3–48.2	6/64 9.4%; 3.5–19.3%
Caracal aurata	1/1	0	0	1/1
Caracal caracal	0/11	0/11	0	0/22
Felis chaus	0/10	0/61	0	0/71
Felis margarita	2/28	0/4	0	2/32
Felis silvestris	0/1	6/94	0	6/95
Leopardus colocolo	0/1	0/11	0	0/12
Leopardus geoffroyi	1/4	1/4	0	2/8
Leopardus guigna	0	0/3	0	0/3
Leopardus pardalis	0/3	0/39	0	0/42
Leopardus tigrinus	0	0/9	0	0/9
Leopardus wiedii	2/5	1/17	0	3/22
Leptailurus serval	0/9	0/23	0	0/32
Lynx canadensis	0/4	0/63	0	0/67
Lynx lynx	0/1	0/1	0	0/2
Lynx pardinus	0	2/8	0	2/8
Lynx rufus	0/2	3/165	0/1	3/168
Neofelis nebulosa	1/9	0/4	0/1	1/14
Otocolobus manul	0/13	0	0	0/13
Panthera leo	0/19	1/33	0/6	1/58
Panthera onca	0/8	0/37	0/1	0/46
Panthera pardus	2/13	0/44	0/3	2/60
Panthera tigris	0/35	0/6	0	0/41
Panthera uncia	0/12	0/1	0	0/13
Pardofelis badia	0	0	0/1	0/1
Pardofelis marmorata	0/3	0/3	0	0/6
Pardofelis temminckii	0/5	0/4	0	0/9
Prionailurus bengalensis	0/3	4/48	0	4/51
Prionailurus planiceps	0/7	0	0	0/7
Prionailurus rubiginosa	0	0/5	0	0/5
Prionailurus viverrinus	0/5	0/6	0	0/11
Puma concolor	0/9	1/69	0	1/78
Puma yagouaroundi	1/4	0/17	0	1/21

diagnosed as FPE positive based on clinical signs of FPE, including soft tissue swelling, mucosal depression, and/or bleeding in the area medial to PM4. Only 16 of these 48 (33.3%; 20.4–48.4%) cheetahs had perforation of the palatine bone, and there was a significant difference between those cheetahs with skeletal modifications (33.3%) and those that had soft tissue lesions only (66.7%; 51.6–79.6%) (χ^2 test; $P = 0.04$).

Six of the sixty-four cheetah skulls inspected had FPE (Table 1). One of the affected skulls was a free-living cheetah from Mali, the other five were captive cheetahs, one wild caught in Kenya, another captive born in Germany, and the

TABLE 2. Prevalence of Focal Palatine Erosion in Cheetah (*Acinonyx jubatus*) Skulls

Location	Status	Positive	Total	Percent; 95% CI
Zoos	Captive	4	21	19%; 5.4–41.9%
Unknown	Captive/free living	1[b]	7	14.3%; 0.4–57.9%
North Africa[a]	Free living	1	4	25%; 4.6–69.9%
East Africa[a]	Free living	0	25	0%; 0–13.7%
Namibia	Free living	0	3	0%; 0–56.1%
Angola	Free living	0	2	0%; 0–84.2%

[a]North Africa = Libya, Mali; East Africa = Ethiopia, Somalia, Kenya, Tanzania, Mozambique, Zimbabwe.
[b]Specimen caught in Kenya and kept in captivity for an unknown period of time

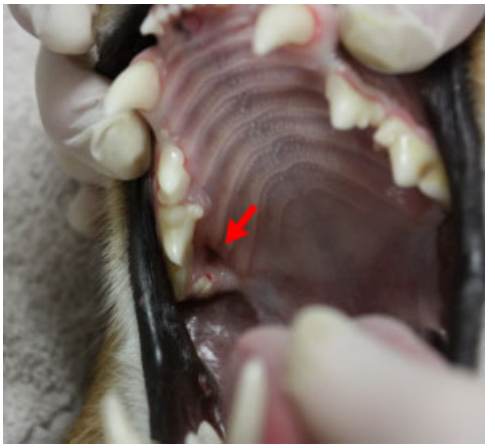

Fig. 2. Mucosal depression caused by lower right molar in the palate of a captive Caracal (*Caracal caracal*) detected during physical examination.

remaining three without additional information available. There was no evidence of FPE in free-living cheetahs from East Africa and none of the three cheetah skulls from Namibia was positive for FPE (Table 2).

FPE was also observed among other species of felids (Table 1). Two lives *C. caracal* and one live *P. viverrinus* evaluated were FPE positive (Fig. 2). In 23/24 (95.8%; 79.8–99.3%) of the cases of FPE (data not shown) in small- and medium-sized species (excluding *Panthera* spp., *N. nebulosa*, *A. jubatus*, and *Puma concolor*), FPE resulted in perforation of the palatine bone, and communication with the ocular orbit and not with the nasal cavity as has been described for *A. jubatus* [Fitch and Fagan, 1982]. In these 23 individuals, an oral–orbital fistula was observed (Fig. 1B). These specimens were from the following species; *Felis silvestris*, *Felis margarita*, *Leopardus wiedii*, *Lynx rufus*, *Lynx pardinus*, *Caracal aurata*, and *Prionailurus bengalensis*. Among the small- and medium-sized cats, only *Puma yagouaroundi* had a perforation that connected with the nasal cavity.

Last, in 20/33 skulls (60.6%; 42.1–77.1%) with FPE, regardless of species, the lower molars did not make contact with the palatine bone. Two individuals were excluded from this analysis, because the molar made contact on one side but not on

the other side. There was a similar prevalence of FPE in cats without molar contact and those with confirmed contact (39.4%; 22.9–57.9%) (χ^2 test; $P = 0.139$). There was no difference in molar contact based on sex or captive and free-living status (data not shown). For a number of species, including *Panthera tigris* ($n = 41$), *Panthera onca* ($n = 46$), *Leopardus pardalis* ($n = 42$), *Lynx canadensis* ($n = 67$), and *Felis chaus* ($n = 71$) there was no evidence of FPE.

DISCUSSION

When first described, FPE was thought to be a pathological condition of captive cheetahs imported to the United States from Namibia [Fitch and Fagan, 1982]. The findings in this study indicate that the disease is also present in free-living cheetahs, which is consistent with the findings of Marker and Dickman [2004] in which FPE was detected in free-living Namibian cheetah. Additionally, FPE was present in cheetahs from populations other than Namibia, including a free-living female cheetah from Mali collected in 1930. This specimen was characterized by a different mitochondrial haplotype than Namibian cheetahs [Burger, personal communication]. Also positive for FPE was a free-living cheetah captured in Kenya and brought into captivity. Unfortunately, no information was available about the age of the cheetah at the time of capture or how long it had spent in captivity. However, this skull provides evidence that cheetahs from Kenya may develop FPE, but the lack of information means that its development of FPE may have been induced by captivity.

When using the perforation of one or both sides of the palate as the criterion for diagnosis, the prevalence of FPE detected in captive live Namibian cheetahs was higher (32.7%; 16/49) than those described in three previous studies. Prevalence has been found to be 11.9% (7/59) for captive Namibian animals in North America [Fitch and Fagan, 1982], 6.3% (13/208) [Marker and Dickman, 2004] and 9.1% (4/44) [Phillips et al., 1993] for free-living specimens in Namibia.

All previous studies on FPE have focused on Namibian cheetahs. This may simply reflect the more extensive health data available for this population and the fact that most of the captive cheetah in the United States at the time of Fitch and Fagan [1982] study were imported from this region [Marker, 1998; Marker-Kraus, 1997]. Because FPE was first identified in this population, it is also possible that more attention has been given to oral examinations in this species and population. When considering our findings for 12 cheetahs in Kenya that had never been in captivity, we did not find evidence of the occurrence of FPE in contrast with the substantial amount of data gathered for the free-ranging Namibian population. This could suggest that FPE is more frequent in the Namibian population, but more research is needed. An effort must be made to study the occurrence of FPE in other populations.

In this study, FPE was detected in cheetahs as well as 14 other species of felids, with higher prevalence in cheetah (9.4%) than in non-cheetah species (2.8%). Therefore, FPE cannot be considered a disease unique to cheetahs. We did find a significantly higher prevalence of FPE in captive than in free-living animals, which concurs with one of our original hypotheses.

A significantly higher FPE prevalence was detected in live cheetahs when categorized based on soft tissue damage (e.g., inflammation, mucosal depression, bleeding) vs. those that only had palatine bone perforation. Therefore, it is possible

that the prevalence data based on skull evaluations is an underestimation, because we would only be able to detect the most advanced cases with bone pathology.

The high number of felid skulls with FPE for which the lower molar was not in contact with the upper palate was an unexpected finding, because it has previously been hypothesized that FPE is a result of molar pressure medial to the carnassial tooth [Fitch and Fagan, 1982].

For 96% of the small- and medium-sized felids with FPE, the perforation of the palatine bone did not create a fistula between the oral cavity and the nasal cavity. In these individuals, there was an oral–orbital fistula, and therefore it is possible that clinical signs of FPE would present in these species associated with ocular pathology.

The etiopathogeny of FPE is not fully understood. It was originally proposed that a soft diet in captivity may affect the masticatory muscles, causing a rotation of the lower molar with subsequent pressure on the dorsal palate [Fitch and Fagan, 1982]. In this study, a high prevalence of FPE (98.0%) was found in Namibian cheetahs fed a whole prey diet (e.g., donkey and horse carcasses, including bones). Therefore, our findings do not support this theory. Several studies have demonstrated that captive animals may have an abnormal anatomy compared with free-living individuals [O'Regan and Kitchener, 2005]. Results from our study do support a link between captivity and the prevalence of FPE, although the factors related to captivity that may influence FPE have yet to be recognized.

Physical examinations of all species of nondomestic cats should include an oral examination to determine the presence and severity of FPE. These examinations should be standardized (e.g., by the AZA felid TAG) and made available for all institutions holding nondomestic felids. We suggest that the grading system defined by Phillips et al. [1993] should be used as a starting point for establishing a standard system.

Our data demonstrate that FPE is a condition affecting a wide range of felid species, including captive and free-living individuals. The condition seems to be more frequent in cheetahs than in other species, particularly when in captivity. We encourage future studies of captive and free-living felid species to include examination of the oral cavity, and a meta-analysis of available data is warranted in order to gain a better understanding of the prevalence and clinical significance of FPE in felid species is gathered.

CONCLUSIONS

This study highlights several factors in the epidemiology of FPE in nondomestic felid species.

1. Captive and free-living cheetahs from different geographical populations may present with FPE.
2. FPE was documented in 15/33 felid species with a prevalence of 3.2%.
3. Prevalence of FPE is higher in captive than in free-living felids.
4. Prevalence is higher in cheetahs than in non-cheetah species.
5. In cheetahs and the majority of other affected large felid species, perforations seemed to create oral–nasal fistulas. In most of the medium- and small-sized felid species, the perforation resulted in oral–orbital fistula. It is necessary to assess if there are differences in clinical signs.

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