Common dental disorders of the degu *(Octodon degus)*

CV Long, BSc, PhD

Summary:

Dental disease is prevalent in the captive degu (Octodon degus), yet little has been documented on the variety of disorders in this species. In this internet-based study, dental cases presented over a 7-year period were collated, analyzed, and grouped. Of the 137 total cases, the most common dental disorder of the degu was found to be molar malocclusion (42.3 %). Other disorders documented included enamel decoloration (13.1 %), molar elodontoma (8.0 %), enamel hypoplasia (6.6 %), incisor tooth fracture (6.6 %), incisor malocclusion (3.6 %), oral abscess (2.2 %), and impacted molar teeth (0.7 %). Details of each condition, pathogenesis, and clinical signs are described. Age was found not to be a significant predictor of dental disease in the degu. ***J Vet Dent 29(3); 158-165, 2012***

Introduction

The degu (*Octodon degus*) is a social, diurnal, caviomorph rodent native to the semi-arid scrublands around central Chile. In recent years the degu has become popular as a household pet throughout the US and Europe due to their inquisitive personalities, relative longevity, and gentle nature. However, captive degus can suffer from a variety of illnesses if not managed correctly. The most common degu complaints presented to veterinarians are dental, with around 60 % of clinical cases demonstrating acquired dental disease.[1] The degu, like their close relative the chinchilla, has a total of 20 elodont (continuously growing) teeth (dental formula 2 x 1/1I 0/0C 1/1P 3/3M), adapted for a high fiber diet typical of the species in the wild.[2-5] This diet consists predominantly of dried vegetation with a high silicate content, and bark.[2,6] The degu belongs to the family *Octodontidae*, the Latin name of which reflects the rather unique dentition of the group with the occlusal surface of the molar teeth forming a distinctive figure-of-eight shape (Fig. 1).[2] Degu molar teeth are hypsodont (high-crowned), showing indentations on the buccal surface and folds on the lingual surface.[2] Similar to the guinea pig and chinchilla, degu molar and incisor teeth are aradicular, having no permanent root structure.[7,8] The maxillary and mandibular incisor teeth extend far back into the skull, consistent with gnawing animals (Fig. 2).[9] The incisor enamel has a multiserial microstructure and is produced continuously by ameloblast cells of the inner enamel epithelium.[2,10,11] In the healthy adult degu, the enamel coloration is a bright orange and is formed only on the superficial rostral edge of the incisor teeth for strengthening and wear-resistance purposes.[2,12,13] This attribute is also helpful during burrow construction when the wild degu uses the incisor teeth to shear away earth.[14] Degu pups are born without orange enamel. The enamel gradually changes at approximately 6-months postpartum to the normal orange color (Fig. 3).

Due to the prevalence of dental disease in the domestic degu, it is important to develop a good understanding of the most common complaints in order to prevent and treat these conditions effectively. This study presents the findings of degu dental disorders compiled over a period of 7-years through veterinarian and owner contact with the Degutopia website (http://www.degutopia.co.uk), the UK's largest degu organization with over 1,400 members, and its world-wide

Figure 1

Diagram of the right maxillary arch in the degu demonstrating the characteristic figure-of-eight pattern of the molar occlusal surface.

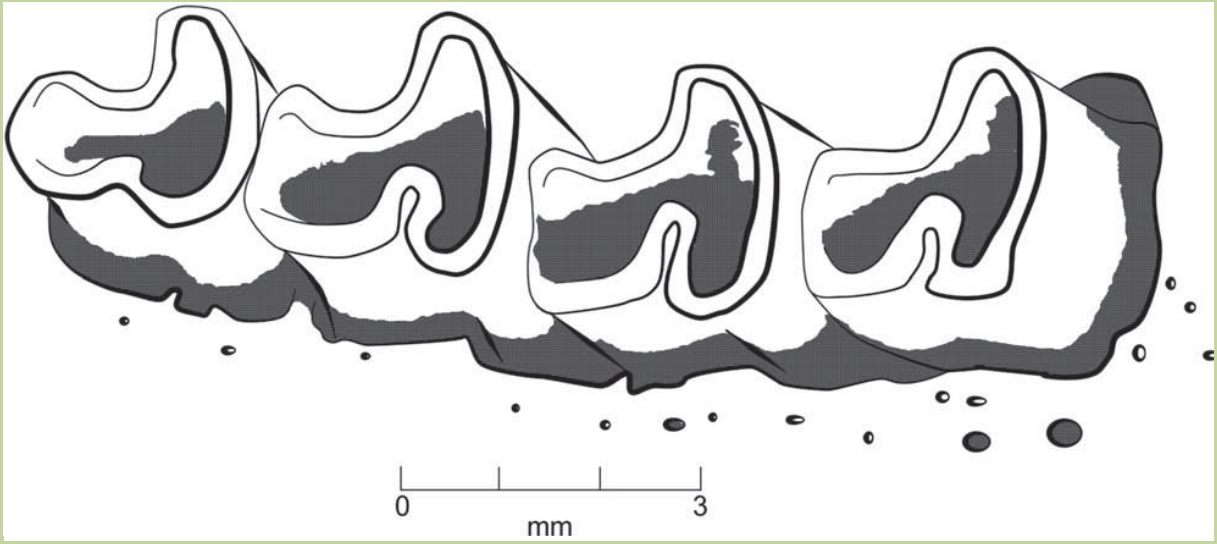


Figure 2

Lateral radiographic view of a healthy degu skull showing molar and incisor tooth alignment.

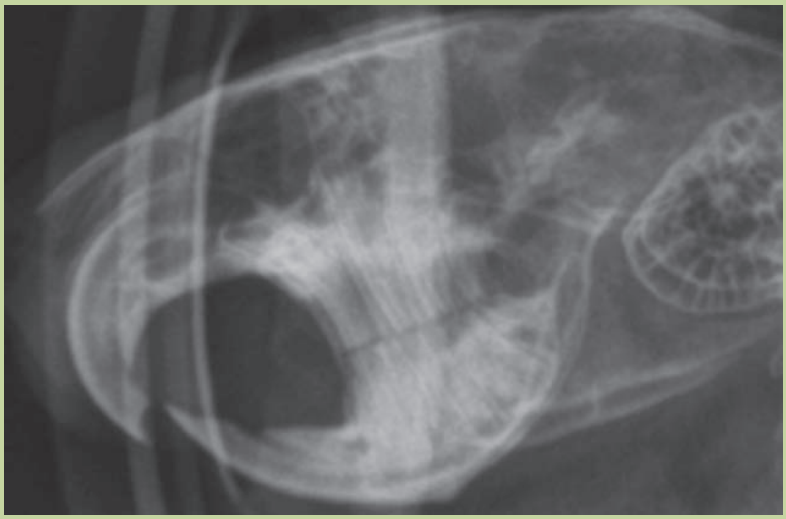
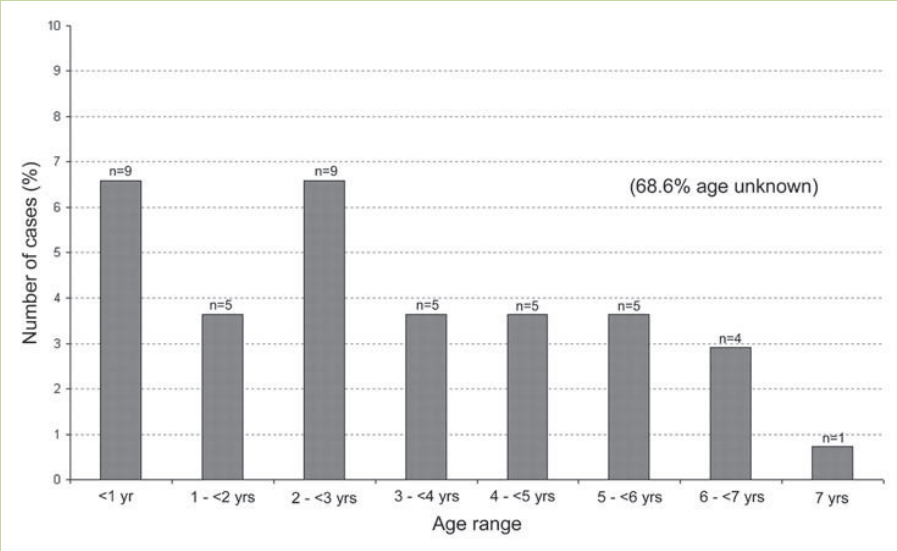


Figure 3

Photographs showing the bright orange enamel coloration of normal adult degu incisor teeth (A). Note the presence of enamel only on the superficial rostral edge of the maxillary and mandibular incisor teeth (B). Photograph showing normal degu pup maxillary incisor teeth at 9-weeks-old. Note the enamel beginning to develop an orange coloration (C).

Figure 4

Histogram of the known age ranges of the degu in the dataset.



sister site and forum, the Degu Information Group (http://pets.groups.yahoo.com/group/degu_information/).

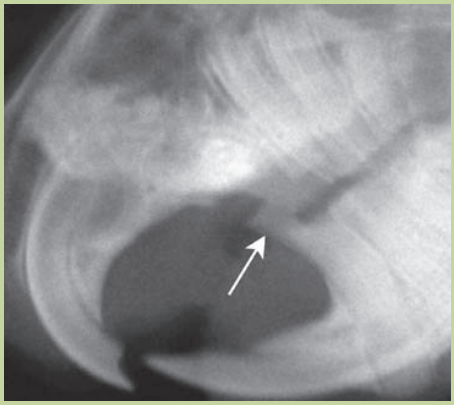
Materials and Methods

In order to investigate dental disease in the degu, a total of 32,677 e-mail and group post archived messages (between August 2003 and December 2010) were searched. Messages were searched by keywords "teeth" OR "tooth", where "OR" was a Boolean operator.[15] Results were grouped into cases for individuals, and from these data were extracted clinical signs, diagnosed disorder, sex, age, and outcome. Cases were listed as 'undiagnosed' when the degu was not seen by a veterinarian prior to message contact by the owner. Cases were then grouped according to condition and clinical signs as described by the owner.

Statistical analysis was performed on the age data using a chi-squared test. To avoid bias due to weighting of data based on the natural age distribution, only individuals less than 6-years-old were included (average life expectancy of the degu in captivity is approximately 6-8 years). Data were compared to a model predicting that dental disease was equally prevalent around the mean of the dataset among all age groups.

Figure 5

Lateral radiographic view of a degu skull showing molar tooth malocclusion (arrow). Also note the apical elongation of the affected molar root below the mandible.



Results

From 32,677 messages to Degutopia, a total of 698 (2.14%) related specifically to degu dental disorders. This equated to a total of 137 individual cases (64 female, 69 male, 4 unknown), aged between 2-months and 7-years-old. The dental disorders of the dataset were collated and age ranges (if known) were noted (Table 1 and Fig. 4). Statistical analysis revealed that age range was not a significant predictor of dental disease in this sample (P = 0.64). The following subsections describe details and pathogenesis of each dental disorder identified.

Molar malocclusion in the degu is due to the misalignment or elongation of one or more of the molar teeth. Due to the elodont nature of these teeth, molar malocclusion typically results in sharp spurs developing on the buccal or lingual edge of the occlusal surface of the tooth. Abrasion of the spurs against the tongue or cheek leads to injury, swelling, laceration, infection, and possible abscess formation.[16] In severe cases, the entire tooth may become maloccluded, resulting in chronic overgrowth (Fig. 5). The clinical signs associated with a diagnosis of molar malocclusion in the dataset included difficulty eating (41.4 %), weight loss (32.8 %), incisor overgrowth (29.3 %), excessive salivation ('slobbers') (17.2 %), pawing at the mouth (10.3 %), cheek laceration (10.3 %), purulent discharge from mouth (8.6 %), loose/missing molars (8.6 %), tongue laceration (6.9 %), change in behaviour/lethargy (5.2 %), swollen cheek (5.2 %), fractured molar teeth (3.4 %), bruxism (3.4 %), swollen tongue (3.4 %), hair loss around mouth (3.4 %), irregular incisor wear (1.7 %), dyspnea (1.7 %), abscess recurrence (1.7%), inability to gnaw (1.7 %), sneezing (1.7 %), epiphora (1.7 %), fur-pulling (1.7 %), and bleeding from the mouth (1.7 %) [Table 2].

The enamel of a healthy degu is typically a bright orange color. Degu enamel can become decolored, resulting in the

Figure 6

Photograph showing enamel decoloration in a 2-year-old degu (A) and partial enamel hypoplasia of the left mandibular incisor tooth in a 5-year-old degu (B).

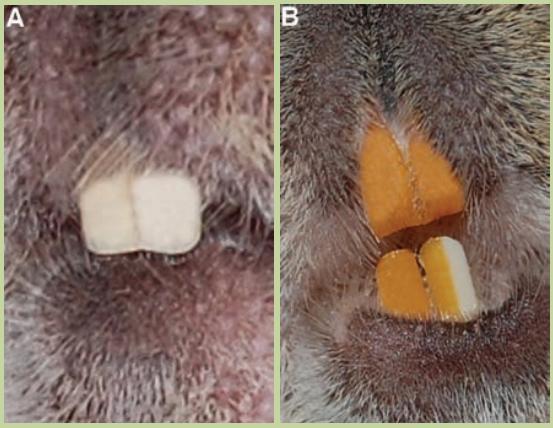


Figure 7

Lateral radiographic view of a degu skull showing mandibular molar elodontoma (arrow) secondary to molar malocclusion.

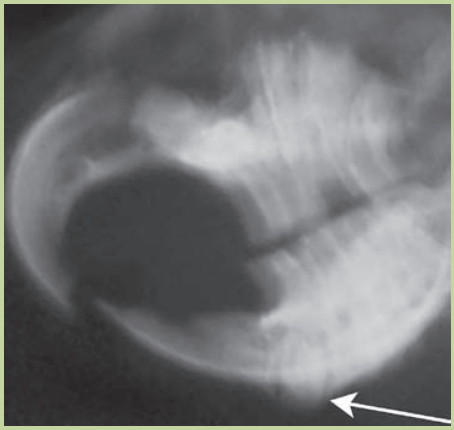


Table 1

Collated dental disorders of 137 degus.

Dental Disorder	Number of Cases (n=137)	%	Gender % Male	% Female	% Unknown
Molar malocclusion	58	42.3	51.7	48.3	0
Undiagnosed	23	16.8	52.2	47.8	0
Enamel decoloration	18	13.1	50.0	38.9	11.1
Molar elodontoma	11	8.0	45.5	54.5	0
Enamel hypoplasia	9	6.6	33.3	44.4	22.2
Incisor fracture	9	6.6	44.4	44.4	11.1
Incisor malocclusion	5	3.6	60.0	40.0	0
Dental abscess	2	1.5	100	0	0
Cheek abscess	1	0.7	0	100	0
Impacted molars	1	0.7	0	100	0

Table 2

Outcomes of the cases of molar malocclusion in the degu.

Outcome	Number of cases (n=58)	%	Age range	Number of cases (n=58)	%
Molar work under general anesthesia	15	25.9	<1 yr	3	5.2
Regular molar work under general anesthesia	14	24.1	1 - <2 yrs	3	5.2
Outcome unknown	10	17.2	2 - <3 yrs	3	5.2
Degu euthanized	9	15.5	3 - <4 yrs	1	1.7
Degu died	7	12.1	4 - <5 yrs	3	5.2
Incisor teeth trimmed	6	10.3	5 - <6 yrs	3	5.2
Molar(s) extracted	4	6.9	6 - <7 yrs	2	3.4
Condition improved with dietary change	2	3.4	7 yrs	1	1.7
			Age unknown	39	67.2

An outcome of 'molar work under general anesthesia' indicates the degu received veterinary dental treatment on one occasion only, while 'regular molar work' indicates the degu received veterinary dental treatment on more than one occasion.

Table 3

Outcomes of the cases of enamel discoloration in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Outcome unknown	12	66.7	<1 yr	1	5.6
Enamel color restored after dietary improvement	4	22.2	1 - <2 yrs	0	0
Degu died	1	5.6	2 - <3 yrs	0	0
Enamel color restored after removal of stress source	1	5.6	3 - <4 yrs	0	0
			4 - <5 yrs	0	0
			5 - <6 yrs	0	0
			6 - <7 yrs	2	11.1
			7 yrs	0	0
			Age unknown	14	77.8

superficial rostral edge of all four incisor teeth becoming a pale creamy-white color (Fig. 6). In the degu, there appears to be a strong link between diet, general health, and enamel coloration, although the exact process underlying this is not yet well understood in this species.[17] Disruption to mineral metabolism, especially iron, in the degu has been found to result in enamel decoloration.[18] The clinical signs associated with enamel decoloration in the dataset included pale incisors (100 %), behavioural stress (5.6 %), lethargy (5.6 %), fractured incisor teeth (5.6 %), incisor tooth overgrowth (5.6 %), sneezing (5.6 %), and respiratory infection (5.6 %) [Table 3].

Molar elodontoma occurs due to chronic overgrowth of the molar tooth roots in elodont species.[19] Both maxillary and mandibular molar tooth roots may be affected in the degu. Maxillary molar tooth elodontoma can be severely disruptive to the sinuses and nasal passages, while mandibular molar tooth elodontoma is generally less symptomatic, causing "lumps" along the ventral mandible (Fig. 7).[20] Molar elodontoma in the degu frequently occurs secondary to molar malocclusion and may be triggered by the subsequent effects

on the germinal cells of the root.[21] The clinical signs associated with molar elodontoma in the dataset included dyspnea (54.5 %), sneezing (27.3 %), excessive intestinal bloat (18.2 %), discharge/bleeding from nose (18.2 %), weight loss (18.2 %), sinus/lacrymal problems (18.2 %), difficulty eating (18.2 %), "growth" on mandible (9.1%), incisor tooth overgrowth (9.1 %), purulent discharge from mouth (9.1 %), bruxism (9.1 %), excessive salivation ['slobbers'] (9.1 %), and prolapsed eye (9.1 %) [Table 4].

Enamel hypoplasia is characterised by a reduction in enamel thickness, resulting in part of the incisor tooth enamel becoming pale, or one or more of the incisor teeth becoming pale (Fig. 6). Note that this condition is distinct from enamel decoloration that may affect otherwise normally formed enamel. The condition can arise due to damage to the enamel-forming ameloblast cells near the germinal root of one or more incisor teeth secondary to direct trauma to the teeth, infection, or tumor formation which affects normal enamel production.[22-24] A diet high in phosphorous, or with a poor calcium:phosophorous ratio, can also cause enamel hypoplasia in the degu.[18] The condition is distinct from enamel aplasia or dysplasia, since ameloblast cells are not permanently defective. The clinical signs associated with enamel hypoplasia in the dataset included a single pale incisor tooth (77.8 %) and double pale incisor teeth (22.2 %) [Table 5]. Of these cases, 22.2 % had maxillary incisor tooth enamel hypoplasia and 22.2 % had mandibular incisor tooth enamel hypoplasia (55.6 % did not specify hypoplasia location).

Incisor tooth fracture was categorized with one or more broken or missing incisor teeth, or a loose/mobile incisor tooth if a root fracture occurred. Tooth fracture most commonly occurs due to direct trauma to incisor teeth, such as after a fall. Degu incisors grow at a rate of approximately 0.5 to 1.0 mm day, resulting in re-growth of the affected tooth after several days, depending on the fracture location.[1] The clinical signs associated with incisor tooth fracture in the dataset included loss of a single incisor tooth (33.3 %), loss of two or more incisor teeth (22.2 %), loose/mobile incisor(s) (22.2 %), difficulty eating (11.1 %), opposing incisor tooth overgrowth (11.1 %), uneven incisor teeth (11.1 %), and bleeding around the gingiva (11.1 %) [Table 6].

Malocclusion of the incisor teeth is characterized by misalignment of one or more teeth, frequently resulting in overgrowth of the incisor teeth (Fig. 8). Incisor malocclusion can be caused by congenital abnormalities in incisor development or skull deformities, developmental defects, or as a result of trauma to the jaw.[25] Care should be taken to isolate this condition from secondary incisor problems caused by molar malocclusion. The clinical signs associated with incisor malocclusion in the dataset included incisor overgrowth (100 %), difficulty eating (80 %), and weight loss (40 %) [Table 7].

Periapical abscess formation in caviomorph species is reportedly less common than in other rodents and lagomorphs.[25] Abscessation may form after molar malocclusion due to food impaction, tooth fracture, or root damage.[26,27] Only two degu (ages unknown) in this sample had a dental abscess. Associated clinical signs included mobile molar teeth (50

Table 4

Outcomes of the cases of molar elodontoma in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Outcome unknown	4	36.4	<1 yr	0	0
Degu euthanized	4	36.4	1 - <2 yrs	0	0
Degu died	3	27.3	2 - <3 yrs	1	9.1
			3 - <4 yrs	2	18.2
			4 - <5 yrs	2	18.2
			5 - <6 yrs	0	0
			6 - <7 yrs	0	0
			7 yrs	0	0
			Age unknown	6	54.5

Table 5

Outcomes of the cases of enamel hypoplasia in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Outcome unknown	8	88.9	<1 yr	1	11.1
Enamel color restored after several weeks	1	11.1	1 - <2 yrs	1	11.1
			2 - <3 yrs	0	0
			3 - <4 yrs	1	11.1
			4 - <5 yrs	0	0
			5 - <6 yrs	1	11.1
			6 - <7 yrs	0	0
			7 yrs	0	0
			Age unknown	5	55.6

%), difficulty eating (50 %), purulent discharge from mouth (50 %), and weight loss (50 %). The outcome of case 1 was extraction of the affected molar tooth, and in case 2 the degu died.

Abscess formation in the soft tissue of the mouth is typically secondary to molar malocclusion-related laceration.[16,28] Only one degu (age unknown) in the sample presented with this diagnosis. Clinical signs included purulent discharge from the mouth and difficulty eating. The outcome of this case was that the abscess was drained under general anesthesia, and the molar teeth were also examined.

Impacted molar teeth are not commonly reported in rodents. This condition may be associated with unerupted molar teeth. In rats, it has been associated with impaired molar tooth root formation and ankylosis.[29] Only one degu in the sample was diagnosed with impacted molars (age unknown). Clinical signs included loose molars, difficulty eating, purulent discharge from mouth, and weight loss. The degu was euthanized in the outcome of this case.

Individual degus without an official diagnosis exhibited a variety of clinical signs including incisor tooth overgrowth (78.3 %), difficulty eating (26.1 %), inability to gnaw (13 %), bruxism (8.7 %), weight loss (8.7 %), uneven incisor tooth wear (4.3 %), mobile molar teeth (4.3 %), pawing at

Figure 8

Lateral photographic view showing incisor malocclusion in a 6-year-old degu. The maxillary incisor teeth are at an obtuse angle, resulting in chronic overgrowth of the mandibular incisor teeth.

Table 6

Outcomes of the cases of incisor tooth fracture in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Outcome unknown	3	33.3	<1 yr	2	22.2
Re-growth of incisor(s) after several days	2	22.2	1 - <2 yrs	0	0
Incisors clipped	2	22.2	2 - <3 yrs	0	0
Re-growth of incisors after soft diet	1	11.1	3 - <4 yrs	1	11.1
Degu died due to other trauma	1	11.1	4 - <5 yrs	0	0
			5 - <6 yrs	0	0
			6 - <7 yrs	0	0
			7 yrs	0	0
			Age unknown	6	66.7

Table 7

Outcomes of the cases of incisor tooth malocclusion in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Regular incisor clipping	5	100	<1 yr	1	20
Examination of molars under general anaesthesia	1	20	1 - <2 yrs	0	0
			2 - <3 yrs	0	0
			3 - <4 yrs	0	0
			4 - <5 yrs	0	0
			5 - <6 yrs	0	0
			6 - <7 yrs	0	0
			7 yrs	0	0
			Age unknown	4	80

Table 8

Outcomes of the undiagnosed cases of dental abnormalities in the degu.

Outcome	Number of cases (n=18)	%	Age range	Number of cases (n=18)	%
Outcome unknown	10	43.5	<1 yr	1	4.3
Regular incisor clipping	5	21.7	1 - <2 yrs	1	4.3
Degu died	3	13	2 - <3 yrs	4	17.4
Incisors clipped	2	8.7	3 - <4 yrs	0	0
Degu euthanized	1	4.3	4 - <5 yrs	0	0
Molar extraction	1	4.3	5 - <6 yrs	1	4.3
Regular molar work under general anaesthesia	1	4.3	6 - <7 yrs	0	0
			7 yrs	0	0
			Age unknown	16	69.6

the mouth (4.3 %), swollen cheeks (4.3 %), incisor teeth growing apart (4.3 %), and hair loss around the mouth (4.3 %) [Table 8].

Discussion

Analysis of this dataset revealed that the most common dental disorder of the degu was molar malocclusion. Other studies also report the prevalence of molar complaints in the degu and chinchilla.[1,26] In some cases, there may be a congenital component of the condition, however more natural environmental conditions and dietary management are likely to be key factors in the prevention of caviomorph molar malocclusion.[26,30] The wild degu is adapted to a high-fiber diet and this should not be replaced solely by hard feed mixes or pellets that frequently do not contain the dietary fiber necessary for the required molar tooth wear.[26] Owners should be encouraged to provide a more natural diet consisting predominantly of good quality hay *ad lib* with access for browsing, and to limit the quantity of energy-rich hard feeds to prevent selective feeding. Routine body weight monitoring (monthly) of any degu with a history of molar malocclusion should help to provide an early indication of molar tooth problems, as difficulty eating and weight loss are the most common clinical signs of the disorder. This is particularly important due to the chronic nature of the condition, relatively high mortality rate (28 % of known follow-up cases) [Table 2], and tendency to lead to other conditions such as molar elodontoma and oral abscess formation.

Enamel decoloration was also found to be a prevalent dental disorder of the degu. While there is known to be a link between diet, general health, and enamel color in the degu, little is currently known about the underlying process.[17] Evidence for a link between diet and enamel color was supported by the dataset, with 67 % of known follow-up cases showing color restoration with dietary improvement (switching to a diet with increased vitamin and mineral content and occasional fresh vegetable matter) [Table 3]. In murine rodents, enamel coloration is linked directly to the availablity of iron to ameloblast cells during enamel production.[31,32] In addition, iron content is thought to provide acid protection to the tooth.[33] There may also be a link between dietary carotenoid intake, enamel coloration, and immune system health.[34] Carotenoids, such as vitamin A, are known to be important for normal germinal tissue and ameloblast function in other rodents.[35] Offering supplemental foods rich in iron, vitamin C (assisting non-heme iron uptake by the body[36]) and carotenoids on a routine basis may help to prevent or improve enamel decoloration in the degu. It should be noted that enamel hypoplasia can be a cause of enamel decoloration, although in degus hypoplasia rarely affects all 4 incisor teeth simultaneously, unlike decoloration.

Molar elodontoma in the degu was only reported in the literature relatively recently,[20] although it appears to be a relatively common dental disorder according to this dataset. This is most likely due to the high occurrence of molar tooth problems that can lead to this condition.[21] Given that the mortality rate of all known follow-up cases was extremely high (100 %) suggesting that formation of elodontoma represents advanced, end-stage dental disease in the degu.

Enamel hypoplasia is occasionally reported in rats,[37] guinea pigs after ameloblast trauma,[38] or with genetic amelogenesis defects in mice.[39] Cases in this dataset indicate the condition may be self-correcting after the recovery of affected ameloblasts (Table 5) and is not typically apparent or problematic to the degu (Table 5). Because there may also be a link between dietary phosphorous and calcium levels, the diet should be checked and modified accordingly.[18] 'Normal' ratios are approximately 2:1 (calcium:phosphorous), provided at about 1.2 % calcium to 0.6 % phosphorous.[18]

Fractured incisor teeth appear to require little direct treatment other than maintenance of the opposing incisor to prevent overgrowth while the affected incisor re-grows (Table 6). In cases where the incisors are fractured after direct trauma to the teeth, the general prognosis is good provided no other significant bodily trauma is sustained. Similarly, for cases where true incisor malocclusion is diagnosed, only regular monitoring of the incisor length and trimming may be needed (Table 7). Note that incisor tooth trimming should preferentially be done using diamond burring since clipping can cause the tooth to splinter.[40]

Given that the most common clinical signs in the undiagnosed cases were incisor tooth overgrowth and

difficulty eating, it seems likely that these degus had molar malocclusion and/or incisor malocclusion. Because incisor overgrowth is much easier to see on visual examination than molar conditions, and due to the relatively uncommon nature of true incisor malocclusion, molar tooth examination under general anesthesia in cases presenting with these clinical signs is recommended. Radiography of the animal may also be beneficial in diagnosing molar malocclusion and elodontoma.

It is important to emphasize the fact that all data gathered in this study were from an internet source, provided in large part by degu owners. While this can have some drawbacks such as misinterpretation of veterinary consultation, there are also benefits such as being able to draw on a large network of information not localized to one specific region. The dataset provides a valuable reference of examples of dental disease in the degu.

In conclusion, the captive degu can present a variety of dental disorders according to feedback predominantly from owners. The most common of these is molar malocclusion, which may lead to secondary conditions such as molar elodontoma and abscess formation. Proper dietary management and routine weight monitoring are likely to be key factors in preventing and managing these conditions. In addition, age may not be a reliable indicator of dental disease in the degu.

Author Information

The author's address is Degutopia,
UK. Email: Chloe@degutopia.co.uk

Acknowledgements

The author would like to thank the members of Degutopia and the Degu Information Group for their assistance, and staff at the King Richard Veterinary Centre, Leicester for their support. Particular thanks to M. Tiley-Waters, E. Bown and Dr. J. Hague for support and helpful discussion. This article is dedicated to N. Natalie.

References

1. Jekl V, Hauptman K, et al. Diseases in pet degus: A retrospective study in 300 animals. *J Small Anim Pract* 2011; 52: 107-112.
2. Woods C, Boraker D. Octodon degus. *Mamm Species* 1975; 67: 1-5.
3. Bozinovic F. Nutritional energetics and digestive responses of an herbivorous rodent (Octodon degus) to different levels of dietary fiber. *J Mammal* 1995; 76: 627-637.
4. Kenagy G, Nespolo R, et al. Daily and seasonal limits of time and temperature to activity of degus. *Rev Chil Hist Nat* 2002; 75: 567-581.
5. Bozinovic F, Gallardo PA, et al. Seasonal acclimatization in water flux rate, urine osmolality and kidney water channels in free-living degus: Molecular mechanisms, physiological processes and ecological implications. *J Exp Biol* 2003; 206: 2959-2966.
6. Gutiérrez J, Bozinovic F. Diet selection in captivity by a generalist herbivorous rodent (Octodon degus) from the Chilean coastal desert. *J Arid Environ* 1998; 39: 601-607.
7. Crossley DA, Jackson A, et al. Use of computed tomography to investigate cheek tooth abnormalities in chinchillas (Chinchilla laniger). *J Small Anim Pract* 1998; 39: 385-389.
8. Reiter AM. Pathophysiology of dental disease in the rabbit, guinea pig, and chinchilla. *J Exot Pet Med* 2008; 17: 70-77.
9. Frahnert S. Morphology of the Glires ancestral rostral cranium and its implications for the monophyly of this clade. *Mit Zool Mus Berl* 1999; 75: 229-246.
10. Wood AE. The evolution of the old world and new world hystricomorphs. *Symp Zool Soc* Lond 1974; 34: 21-60. In: Rowlands, I.W. and Weir, B.J., eds. The Biology of Hystricomorph Rodents. London: Academic Press, 1974.
11. Fincham AG, Simmer JP. Amelogenin proteins of developing dental enamel. In: Chadwick, D.J. and Cardew, G., eds. Ciba Foundation Symposium 205- Dental Enamel. Chichester: John Wiley & Sons, 2007; doi: 10.1002/9780470515303.ch9.
12. Pfretzschner HU. Structural reinforcement and crack propagation in enamel. *Mém Mus Natl Hist Nat Paris C* 1988; 53: 133-143.
13. Martin T. Early rodent incisor enamel evolution: Phylogentic implications. *J Mamm Evol* 1993; 1: 227-254.
14. Ebensperger L, Bozinovic, F. Energetics and burrowing behaviour in the semifossorial degu Octodon degus (Rodentia: Octodontidae). *J Zool* 2000; 252: 179-186.
15. Boole G. An investigation of the laws of thought, on which are founded the mathematical theories of logic and probabilities. London: Walton and Maberly, 1854.
16. Harvey SB, Alworth LC, et al. Molar malocclusions in the pine vole (Microtus pinetorum). *J Am Assoc Lab Anim Sci* 2009; 48: 412-415.
17. Richardson VCG. ***Diseases of small domestic rodents*** (2nd ed.). Oxford: Blackwell Publishing Ltd, 2003; 75-90.
18. Jekl V, Krejcirova L, et al. Effect of high phosphorous diet on tooth microstructure of rodent incisors. *Bone* 2011 49:479-84.
19. Boy SC, Steenkamp G. Odontoma-like tumours of squirrel elodont inscisors- elodontomas. *J Comp Path* 2006; 135: 56-61.
20. Jekl V, Hauptman K, et al. Elodontoma in a degu (Octodon degus). ***J Exot Pet Med*** 2008; 17: 216-220.
21. Slootweg PJ, Kuijpers MHM, et al. Rat odontogenic tumors associated with disturbed tooth eruption. *J Oral Pathol Med* 1996; 25: 481-483.
22. Sarnat BG, Schour I. Enamel hypoplasia (chronological enamel aplasia) in relation to systemic disease: A chronologic, morphologic and etiologic classification. *J Am Dent Assoc* 1940; 29: 67-75.
23. Watts A, Addy M. Tooth discolouration and staining; A review of the literature. *Br Dent J* 2001; 190: 309-316.
24. Wolff J, Edwards M, et al. Mystery of epizootic oral tumors in spring chinook salmon. *Proceedings of the 29th Annual Eastern Fish Health Workshop* 2002; 1-7.
25. Capello V. Diagnosis and treatment of dental disease in pet rodents. *J Exot Pet Med* 2008; 17: 114-123.
26. Derbaudrenghien V, van Caelenberg A, et al. Dental pathology in chinchillas. Vlaams Diergen Tijds 2010; 79: 345-358.
27. Legendre LFJ. Oral disorders of exotic rodents. *Vet Clin North Am Exot Anim Pract* 2003; 6: 601-628.
28. Verstraete FJM. Advances in diagnosis and treatment of small exotic mammal dental disease. *Semin Avian Exotic Pet Med* 2003; 12: 37-48.
29. Hiraga T, Ninomiya T, et al. Administration of the bisphosphonate zoledronic acid during tooth development inhibits tooth eruption and formation and induces dental abnormalities in rats. *Calcif Tissue Int.* 2010; 86: 502-510.
30. Wiggs RB, Lobprise HB. Oral Anatomy and Physiology. In: Wiggs, RB Lobprise, HB, eds. *Veterinary Dentistry Principles and Practice.* Philadelphia: Lippincott-Raven, 1997.
31. Halse A. Electron microprobe analysis of iron content of incisor enamel in some species of rodentia. *Arch Oral Biol* 1974; 19: 7-11.
32. Yanagawa T, Itoh K., et al. Nrf2 deficiency causes tooth decolorization due to iron transport disorder in enamel organ. *Genes Cells* 2004; 9: 641-651.
33. Selvig KA, Halse A. The ultrastructural localization of iron in rat incisor enamel. *Scand J Dent Res* 1975; 83: 88-95.
34. Blount JD, Metcalfe NB, et al. Carotenoid modulation of immune function and sexual attractiveness in zebra finches. *Science* 2003; 300: 125-127.
35. Schour I, Hoffman MM, et al. Changes in the incisor teeth of albino rats with vitamin A deficiency and the effects of replacement therapy. *Am J Pathol* 1941; 17: 529-562.
36. Bates CJ, Cowen TD, et al. Growth, ascorbic acid and iron contents of tissues of young guinea pigs whose dams received high or low levels of dietary ascorbic acid or Fe during pregnancy or suckling. *Br J Nutr* 1988; 60: 487-497.
37. Levy BA. Effects of experimental trauma on developing first molar teeth in rats. *J Dent Res* 1968; 47: 323-327.
38. Boyle PE. The effect of ascorbic acid deficiency on enamel formation in the teeth of guinea pigs. *Am J Pathol* 1938; 14: 843-848.
39. Gibson CW, Yuan Z, et al. Amelogenin-deficient mice display an Amelogenesis Imperfecta phenotype. *J Biol Chem* 2001; 276: 31871-31875.
40. Legendre LFJ. Malocclusions in guinea pigs, chinchillas and rabbits. *Can Vet J* 2002; 43: 385-390.