

Peer-Reviewed Article

A Nutritional Approach to the Prevention of Insulinomas in the Pet Ferret

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Insulinoma is a common endocrinopathy afflicting adult pet ferrets (*Mustela putorius furo*) in the United States. These insulin-secreting nodules within the pancreas are also referred to as *islet cell tumors* and *beta cell tumors*. The hypoglycemia resulting from insulinomas accounts for the various clinical signs: lethargy, episodic weakness, weight loss, nausea (manifested by ptyalism and/or bruxism), seizures, and death. The underlying etiology of this condition is unknown.

Insulinomas may be treated medically or surgically, although the ferret is rarely cured of this malady regardless of the treatment modality. While surgery offers the best long-term survival and disease-free interval,¹ recurrence is common. In one large, retrospective study, approximately half of the ferrets undergoing surgery remained hypoglycemic during the immediate postoperative period and another third developed recurrence within 1 to 23.5 months.²

Insulinomas often lead to the ferret's demise while inflicting an emotional and financial toll on the owner. Veterinarians are often frustrated by the inability to cure this condition. The high prevalence of insulinomas makes ferret ownership less attractive for many pet owners.

As of yet, there is no known method of preventing insulinoma formation. This paper presents a nutritional hypothesis and offers a prevention strategy to minimize the chance of insulinoma formation in pet ferrets.

FERRETS AS OBLIGATE CARNIVORES

Ferrets are obligate carnivores. As such, they would normally consume a diet high in proteins

and fat and low in carbohydrates and fiber. (This author defines high protein as 42% to 55%, low carbohydrate as 8% to 15%, and low fiber as 1% to 3% of the diet. Throughout this paper, all nutrient analyses will be expressed on a dry-matter basis.)

Related mustelids of the domestic ferret include the black-footed ferret (*Mustela nigripes*), the European ferret (*M. putorius furo*), the European polecat (*Mustela putorius putorius*) and the Steppe polecat (*Mustela eversmanni*). All of these species are effective hunters known to consume whole, small prey such as rodents, lagomorphs, and birds. The nutritional content of such prey would qualify as being high in protein and low in carbohydrates. For example, the nutrient analysis of a rat carcass is 55% protein, 38.1% fat, 1.2% carbohydrate, and 0.55% fiber.³ These proportions are considerably different than foodstuffs fed to pet ferrets in the United States. Commercial ferret diets typically fed in this country consist of dry kibble containing the following approximate nutrient ranges: protein 22% to 42%, fat 15% to 28%, and carbohydrate 10% to 45%. At least a moderate amount of carbohydrate is generally required for processing a dry kibble product. In addition, many ferret owners feed treats containing high sugar content to their pets. Clearly, this high carbohydrate load greatly exceeds the level found in the natural diet of ferrets.

It is interesting to note that insulinomas are uncommon in Europe, New Zealand, and Australia.⁴ While this may be explained on the basis of a different genetic pool, it is worth noting that there is a major difference in feeding protocols.

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Rather than being fed strictly commercial cat or ferret food (as in the United States), these ferrets are provided meat scraps, poultry scraps, and fish scraps. Sugary treats are generally not provided. This diet is significantly lower in carbohydrates compared with commercial diets fed in the United States. An Australian veterinarian has speculated that a high-carbohydrate diet may indeed predispose ferrets to insulinoma formation.⁵ From a nutritional standpoint, ferrets are more similar to cats than to dogs. Cats are considered obligate carnivores; dogs are considered omnivores. As such, cats have been described as having unique anatomic, physiologic, metabolic, and behavioral adaptations to survive on a strict carnivorous diet.⁶ The same is likely to be true of ferrets. Being carnivores, the digestive tract of a ferret is designed to process highly digestible, energy-dense food. Like cats, ferrets have a relatively short gastrointestinal (GI) tract. The GI transit time (3.0 to 3.5 hours) of ferrets is even shorter than that of cats (6 to 8 hours).⁷ Ferrets have a lower concentration of some intestinal brush border enzymes compared with other carnivorous mammals.⁸ The ferret colon is short, lacks epithelial folds, and has limited absorptive capacity.⁹ In addition, ferrets lack a cecum and do not have significant intestinal flora to process complex carbohydrates. Thus, it has been suggested that ferrets should not be fed a diet high in carbohydrates.¹⁰

While no rigorous studies have been performed on the carbohydrate requirement of ferrets, it is probable that ferrets (like cats) can be maintained without any carbohydrates. This would require their diet to have an adequate amount of fat (and thus glycerol) or protein (containing glucogenic amino acids).¹¹

A reference to carbohydrate usage by strict carnivores provides interesting information.⁶ Obligate carnivores such as cats (and, presumably, ferrets) require a high-protein diet because they are continuously metabolizing amino acids for energy. They have evolved to process a high-protein, low-carbohydrate diet. Their catabolic amino enzymes cannot be “turned off,” even when deprived of adequate dietary protein. Because the typical prey of wild cats is low in carbohydrates, they have little need for amylase (required for starch and sugar digestion). Cats lack salivary amylase entirely and have only 5% of the pancreatic amylase levels found in dogs. Because cats have adapted to diets low in carbohydrate, gluconeogenesis is maximal during the food absorption phase (immediately after a meal). In omnivores, such as dogs, gluconeogenesis is maximal during the postabsorptive state when the direct contribution of dietary glucose is absent. Adult cats rely primarily on gluconeogenesis from glucogenic amino acids, lactic acid, and glycerol for maintenance of normal blood glucose concentrations. Dietary amino acids are potent stimulators (secretagogues) of insulin release in cats. As a consequence, gluconeogenesis is critically important in maintaining blood glucose levels in the face of high insulin concentrations and low carbohydrate intake.

Even though cats have evolved to live on low-carbohy-

drate diets, they are able to efficiently use simple carbohydrates. With a few exceptions, sugar digestibility in cats is greater than 94%.⁶ It has been reported that ferrets can efficiently utilize the sugars dextrin, maltose, and glucose.¹¹ (This occurs despite the fact that ferrets have relatively low activity of intestinal brush border disaccharide-digesting enzymes.) This ability to digest simple carbohydrates may have untoward repercussions.

DETRIMENTAL EFFECTS OF A HIGH-CARBOHYDRATE DIET

This author hypothesizes that excessive carbohydrate intake stimulates excessive insulin production from the pancreas, leading to hyperplasia and, eventually, neoplasia. A proposed pathogenesis involves the *additive* stimulation of pancreatic beta cells by two potent insulin secretagogues: amino acids (protein) and simple carbohydrates (e.g., simple sugars and rapidly digestible starch). Free fatty acids (FFAs) may play a similar stimulatory role.

A logical rebuttal question to the above hypothesis is, “Why are insulinomas *rarely* seen in *cats*, even when they are fed high-carbohydrate diets?” Furthermore, there is evidence to suggest that cats fed high-carbohydrate diets are prone to the “opposite” endocrinopathy, namely, diabetes mellitus.

Two reasons are offered by this author to explain these differences between cats and ferrets:

1. Cats fed a high-carbohydrate diet initially reach a hyperinsulinemic state, followed by eventual exhaustion atrophy of the islet cells, resulting in diabetes mellitus. The islet cells of the ferret, on the other hand, “rise to the challenge” and become hyperplastic rather than atrophic.
2. Ferrets (at least those in the United States) have a genetic predisposition to insulinoma formation; cats do not.

HYPERINSULINEMIA AS A PRECURSOR STATE

To better understand the hyperinsulinemic state associated with ferret insulinomas, it is worth reviewing recent research on diabetes mellitus in cats. The most common form of feline diabetes is classified as type II diabetes. (Type I diabetes is seen more commonly in dogs and is associated with beta cell destruction by autoantibodies and T cells.) While the exact cause of type II diabetes is unknown, two characteristics are decreased insulin secretion (especially in response to a glucose load) and insulin resistance.

Type II diabetes is characterized by impaired insulin action, referred to as *insulin resistance*. With insulin resistance, more insulin is required for the same glucose-lowering effect compared with the normal state. Hyperinsulinemia is an early feature of type II diabetes in humans and occurs in cats with impaired glucose tolerance,¹² suggesting insulin resistance may be the initial defect in the pathogenesis of type II diabetes.

Feline type II diabetes is associated with pancreatic islet amyloid deposition.¹³ These amyloid fibrils are synthesized

from islet amyloid polypeptide (IAPP), which is produced in pancreatic beta cells, stored in secretory granules along with insulin, and cosecreted with insulin.¹⁴ The role of IAPP in type II diabetes is speculated to be multifactorial through its direct effect on amyloid deposition, inhibition of insulin secretion, and induction of insulin resistance. Cats having diabetes develop these amyloid deposits; diabetic dogs do not. However, dogs with insulinomas do develop these amyloid deposits, presumably due to overproduction of IAPP, which parallels overproduction of insulin.¹⁴ According to one veterinary pathologist with extensive ferret experience, ferret insulinomas lack amyloid deposits.^a

Abnormal beta cell function is another proposed mechanism of feline diabetes development. Under this model, increased insulin levels indicate beta cell dysfunction, with hypersecretion of insulin being the primary abnormality, inducing insulin resistance and eventual beta cell exhaustion.¹⁵ Feeding cats a diet rich in highly digestible carbohydrates may induce a lifelong high insulin demand, contributing to beta cell failure. Perhaps the beta cells of ferrets, following chronic stimulation by a high-carbohydrate diet, undergo compensatory hyperplasia rather than becoming exhausted as occurs in cats.

This author hypothesizes that ferrets develop a hyperinsulinemic (“preinsulinoma”) state due to the additive effect of various insulin secretagogues in their diet. This theory presumes that insulin secretion in ferrets is similar to that in cats, dogs, humans, and rodents. Glucose and other simple carbohydrates are strong stimuli and elicit a rapid and marked rise in insulin levels. Insulin release in cats is biphasic, just like in other species.¹⁵ In cats, glucose increases first-phase secretion more than amino acids do, but amino acids are equipotent in stimulating second-phase release of insulin. Glucose and amino acids are additive stimuli in cats.¹⁶ In addition, free fatty acids (FFAs) can induce insulin secretion. FFAs cause increased hepatic glucose production and decreased peripheral glucose uptake, at least in obese cats (and possibly ferrets). The additive effects of these three secretagogues (simple sugars, amino acids, and FFAs) may indeed cause a chronic hyperinsulinemic state in ferrets, triggering beta cell hyperplasia in response to the increased demand for insulin.

It has been speculated that a high glucose load is more detrimental to cats (and ferrets?) than dogs or humans because cats are less efficient in lowering blood glucose levels. This is primarily due to the fact that cats have minimal glucokinase activity compared with dogs and humans. Cats rely on the enzyme hexokinase to help clear glucose from the blood, and hexokinase acts more slowly than glucokinase. Intravenous glucose tolerance tests are similarly different between dogs and cats: Glucose concentrations in dogs return to baseline in 60 minutes, while cats require 2 hours.¹⁵ It is interesting that the consumption of protein (a cat’s nat-

^aWilliams BH: Personal communication, [please provide affiliation] 2003.

ural diet) generates a smaller and more delayed insulin response compared with carbohydrate consumption. If ferrets are like cats, a high-carbohydrate diet would invoke a prolonged stimulatory effect for insulin secretion. The author is unaware of any published data on glucose tolerance curves, glucokinase levels, or hexokinase levels in ferrets.

As obligate carnivores, cats (and presumably, ferrets) have evolved to consume a high-protein diet.¹⁷ Proteins do not stimulate as much insulin secretion as carbohydrates. In short, feeding ferrets a high-carbohydrate diet triggers an unnatural level of insulin secretion. This author hypothesizes that it is this lifelong stimulation that leads to hyperplasia of pancreatic beta cells with subsequent insulinoma formation.

DIETARY RECOMMENDATIONS

This author proposes the following dietary recommendations for adult, nonbreeding ferrets:

1. Feed a diet high in protein (42% to 55%), high in fat (18% to 30%), low in carbohydrates (8% to 15%), and low in fiber (1% to 3%).
2. Avoid snacks and treats that are high in simple sugars (e.g., raisins, corn-syrup-based treats).

Many of the ferret-labeled diets reveal the following nutrient analyses ranges: protein: 36% to 40%; fat: 20% to 23%; carbohydrate: 25% to 26%. This author suggests that the carbohydrate level of these commercial diets may be excessive for domestic ferrets. The “carbohydrate loading” is exacerbated by supplementation of the diet with sugar-rich ferret treats (which, unfortunately, are commonly sold in pet stores). Indeed, there is anecdotal evidence (from long-standing, small-scale ferret breeders) that such commercial diets are acceptable provided sugary snacks are avoided. These breeders claim a low incidence of insulinomas in their ferret lines when commercial ferret diets are fed along with occasional meat scraps as treats.

From a practical standpoint, feeding a high-protein/low-carbohydrate diet can be achieved in a variety of ways. Home-cooked diets may be prepared; however, a nutritionist should be consulted to ensure the diet is completely balanced with regard to all nutrients and of adequate digestibility and caloric density. Feeding a natural diet (e.g., mice, juvenile rats) is possible but would be unappealing to most ferret owners and would require a large supply of “prey.” (The average mouse contains 30 kcal¹⁸; the average adult ferret requires 200 to 300 kcal/kg per day¹¹; thus a 1-kg ferret would need to consume seven to 10 mice per day.)

There are various *feline* diets that are considered high protein/low carbohydrate. Many canned kitten foods fall into this category. Most ferret owners, however, prefer to feed a dry kibble to their pets. Two prescription feline diets are touted as being high in protein and low in carbohydrate and are available in a dry kibble. Purina DM Feline Formula was developed to aid in the medical management of cats with diabetes. The nutrient analysis differs between its dry

formula (protein: 58%; fat: 18%; carbohydrate: 15%; fiber: 1.3%) and its canned formula (protein: 57%; fat: 24%; carbohydrate: 8%; fiber: 3.6%). Hill's Prescription Diet Feline m/d was developed to aid in obesity management of cats. It is available in a dry formula (protein: 52%; fat: 22%; carbohydrate: 15.5%; it is a bit high in fiber at 5.5%). It is important to note that these feline diets were neither developed nor tested for use in ferrets; they are labeled for cats only. It is not known if they contain sufficient vitamin and mineral levels for optimum long-term ferret health.

At least one ferret nutritionist has raised concerns about feeding feline diets to ferrets.^b The main issue is the relative amount of plant (versus animal) protein in the diet. Many feline diets obtain more than 50% of their protein from soy and corn gluten meal rather than from animal sources. Whenever plant protein is utilized, the amino acid balance and digestibility may be less than ideal for ferrets. With their somewhat primitive digestive tract, it is critical that ferrets receive a diet containing proteins of high biologic value and in the optimum amino acid balance.

There is a recently developed ferret diet that is being touted as high-protein and low in carbohydrate (Natural Gold for Ferrets, Pretty Bird, Stacy, MN). This dry kibble has a minimum crude protein level of 55.6% with less than 10% carbohydrate. The primary protein source is chicken meal, and the main carbohydrate source is potato starch. According to the manufacturer, their goal was to develop a diet that is closely based on the nutritional analysis of a mouse diet. Because it is a new food, long-term feeding trials should be evaluated before an endorsement is considered.

Another means of achieving a high-protein/low-carbohydrate diet is to add animal protein to commercial ferret food. Various home recipes have been posted on the Internet. Before this can be recommended, a complete nutrient analysis needs to be performed. Simply increasing the protein content can adversely affect the balance of other nutrients.

Because of their GI anatomy and physiology, it is important that a ferret's diet be of high caloric density, typically measured as metabolizable energy (ME). The approximate ME levels (in kcal/100 g of food) for various diets are: Totally Ferret for Active Show and Pet Ferrets (Performance Foods, Broomfield, Colorado) [435]; Marshall Premium Ferret Diet (Marshall Pet Products,

^aWillard TR: Personal communication, Performance Foods, June 2003.

North Rose, New York) [389]; Natural Gold (Pretty Pets, Stacy, Minnesota) [444]; Purina DM Feline Formula [452, dry; 454, can]; and Hill's Prescription Diet Feline m/d [421].


It must be stressed that the benefit of feeding a high-protein and low-carbohydrate diet can be negated by feeding high-sugar snacks and other simple carbohydrates. Some commercially available ferret treats are high in corn syrup and other undesirable carbohydrates. Furthermore, many ferrets develop an acquired taste for sugary treats such as raisins. It is better to substitute commercially available fatty acid supplements and high-protein snacks or meat scraps for sugary treats. Many ferret owners complain that their pets will not consume meat scraps. One method of fostering such dietary preferences is to "imprint" juvenile ferrets on meat scraps, while avoiding the sugary treats.

CONCLUSION

With insulinomas reaching epidemic proportions in adult pet ferrets in the United States, prevention strategies must be explored. This article promotes a nutritional approach by advocating the feeding of a diet low in carbohydrates (with complete avoidance of simple sugars) and high in protein and fat. It is postulated that the lifelong feeding of an excessive amount of carbohydrate leads to a hyperinsu-

(continues on p. 15)

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Diagnosis and Treatment of Dental Disease in Pet Rabbits and Rodents: A Review

Vittorio Capello

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Dental disease is common in pet rabbits and rodents and can produce a wide variety of clinical presentations, many related to the significant anatomic and physiologic differences among these species. While some debate lingers on the exact etiology of dental disease, treatment options rely on the return of dental anatomy and function to as near normal as possible and control of associated infection and inflammation.

ANATOMY AND PHYSIOLOGY¹⁻⁹

The teeth of lagomorphs and rodents are similar to those of other orders of mammals in that the evolutionary process has adapted them to accommodate their unique diets. However, the dental formulas, anatomy, and physiology of lagomorphs are rather different from those of carnivorous, insectivorous, or omnivorous mammals (Table 1).

All lagomorphs and rodent species lack canine teeth and have hypsodont (open-rooted) incisor teeth, which grow throughout life. All lagomorph species have two pairs of upper incisors (a third pair is present before or immediately after birth, then shed); by contrast, all rodent species have just one pair of upper incisors. Another difference, albeit less important to practitioners, is that lagomorphs are diphyodont, with two sets of teeth (the first set is shed immediately before or after birth), while rodent species are monophyodont. The appearance of incisors is different between lagomorphs and rodents. Rabbits, hares, and pikas have a vertical ridge on the labial surface of upper incisors. However, the labial surface in many species of rodents is covered by a thick layer of enamel, which makes these teeth stronger. The size and development of incisors vary greatly among rodent species, but generally they are relatively bigger and stronger than the incisor teeth of the lagomorph species.

Premolar and molar teeth are called *cheek teeth*. They are open-rooted in lagomorphs and in most herbivorous rodent species. The base of the root (the apical end) is called the *apex*. From a zoologic standpoint, rodent species are classified into three main groups: miomorph or “rat-like” rodents (golden and Russian hamster, gerbil, duprasi-fat-tailed gerbil-rat); sciurumorph or “squirrel-like” rodents (prairie dog, chipmunk); and hystrychomorph or “porcupine-like” rodents (guinea pig, chinchilla, degu). Rat-like and squirrel-like rodents have hypsodont incisors and brachyodont (short crowns and anatomically formed roots) cheek teeth; porcupine-like rodents have all open-rooted teeth (both incisor and cheek teeth), similar to lagomorphs.

The cheek teeth of herbivorous species are flat but not smooth, with crests of enamel and grooves in dentine for the

proper crushing of food. The anatomy of the cheek teeth of rabbits and porcupine-like rodents is very similar, but guinea pigs show an important structural peculiarity. The lower cheek teeth of guinea pigs are curved medially, while their roots are curved laterally; their upper cheek teeth are curved laterally, while their roots are curved medially. This results in an occlusal plane that slopes down about 30 degrees from lateral to medial. Deviation from and maintenance of proper occlusal surfaces have important significance in the diagnosis and treatment of dental disease in this species.

Despite being herbivorous, prairie dogs are brachyodont and do not have open-rooted cheek teeth as is sometimes reported in the literature. The significance of this is that malocclusion of premolar and molar teeth is not as much of a concern in this species as it is in porcupine-like rodents because the cheek teeth of prairie dogs do not continue to grow throughout life.

CLASSIFICATION AND DEFINITION OF DENTAL DISEASE

Dental disease has been extensively studied in laboratory animals and also well described in pet rabbits and some rodent species.^{1,2,5,6,8,10-18} Different etiologies have been demonstrated, and different classification schemes have been proposed.^{5,7,19,20} Classification of dental disease based on etiology must take into consideration the fact that there may be more than one underlying cause. In addition, diseases of incisors and cheek teeth may occur independently, although they are frequently linked. Therefore, pet rabbits and rodents may present with incisor disease alone, cheek teeth disease alone, cheek teeth disease following incisor malocclusion, and vice versa.⁵

Many early texts suggest the terms “lumps” or “slobbers” when referring to dental disease. These terms related to clinical signs frequently associated with dental disease, specifically abscesses and hypersalivation due to intraoral lesions. As more work on pathophysiology led to a better understanding of this disease, the term “acquired dental disease” was proposed.⁷ This definition, however, necessarily excluded congenital abnormalities.

Simple “dental disease” (abnormalities of the teeth, including molars, incisors, cheek teeth, and gums) appears to be the most comprehensive definition. Definition aside, however, the most important thing to consider is that this disease is a syndrome, with a complex combination of clinical signs.^{5,8}

PATHOPHYSIOLOGY OF DENTAL DISEASES^{1-8,11,13-18,21-26}

The pathophysiology of dental disease in pet rabbits

TABLE 1. Dental Formula and Physiology of Lagomorphs and Rodents

| <i>Species</i> | <i>Cheek Teeth</i> | | | <i>Number of Cheek Teeth (Upper/Lower)</i> | <i>Total Teeth</i> |
|--|-----------------------------|------------------------------|---------------------------|--|--------------------|
| | <i>Incisors Upper/Lower</i> | <i>Premolars Upper/Lower</i> | <i>Molars Upper/Lower</i> | | |
| Lagomorphs (rabbits, hares, pikas) | 2/1 Open-rooted | 3/2 Open-rooted | 3/3 Open-rooted | 22 (6/5) Open-rooted | 28 |
| Porcupine-like rodents (guinea pigs, chinchillas, degus) | 1/1 Open-rooted | 1/1 Open-rooted | 3/3 Open-rooted | 16 (4/4) Open-rooted | 20 |
| Rat-like rodents (e.g., rats, mice, hamsters, gerbils) | 1/1 Open-rooted | 0/0 | 3/3 Rooted | 12 (3/3) Rooted | 16 |
| Squirrel-like rodents | 1/1 | 1–2/1 | 3/3 | 16–18 (4–5/4) | 20–22 |
| Prairie dogs | 1/1 Open-rooted | 2/1 Rooted | 3/3 Rooted | 18 (5/4) Rooted | 22 |

and rodents is mostly related to the continuous growth of incisors (in all species) and cheek teeth (in species with open-rooted premolars and molars). Four different primary etiologies have been reported: congenital and developmental abnormalities, traumatic injuries, abnormal wear (sometimes due to improper nutrition) and metabolic bone disease.

Congenital and developmental abnormalities (agenesis of the dentition, cleft palate, exposure to teratogens, and maternal malnutrition) have been demonstrated but are undoubtedly rare and, for this reason, do not represent a frequent concern for practitioners. Congenital or hereditary jaw length mismatch may be due to true prognathism of the mandible, or secondary to brachygnathism of the maxilla. This anatomic condition is more frequently recognized in purebred dwarf rabbits weighing less than 1 kg. A rounded brachymorph skull leads to malocclusion of incisors. In addition, functional mandibular prognathism may occur and is described later.^{5,6}

Traumatic injuries due to falls or improper caging are a common presentation in pet rabbits and rodents. Injuries can range from fractures of the incisors to much less common fractures of the mandible or maxilla, which often carry a poor prognosis. Trauma can damage the apical germinal tissue, which impacts the growth of incisor teeth, frequently leading to malocclusion. Incisor fractures that expose the pulp can result in pulpitis and abscesses. It should be noted that these types of injuries occur iatrogenically secondary to improper or unnecessary trimming of incisors, particularly in rodents and guinea pigs.^{5,24}

The largest and most important etiologic group relates to improper nutrition and abnormal wear.^{5,6} Vitamin A deficiency has been reported to cause malocclusion of incisors in rats, but this condition rarely occurs in pets. More frequently, vitamin C deficiency is a predisposing factor for malocclusion in pet guinea pigs. Nevertheless, the most significant etiology is abnormal wear due to the consumption of improper food. Lagomorphs and herbivorous rodents have

very specific nutritional requirements, and their natural food is rich in mildly abrasive silicates. With the exception of those kept free in the garden, pet rabbits and rodents fed with hay and vegetables do not receive the same types and variety of food as free-ranging animals. This is critical in understanding why all pets are potentially susceptible to the development of at least some degree of acquired dental disease during their lifetime. Despite the popularity of rabbits and rodents as pets as well as many advances in the understanding of proper nutritional requirements, many are still fed substandard diets that are deficient in hay, fruit, seeds, breads, and other treat items.

Since open-rooted teeth grow continuously, inadequate wear leads to overgrowth. Incisors tend to deviate from their normal resting position, where the end of the lower incisors contacts the space between the main upper and peg incisors. The earliest change is the loss of the chisel-like shape of the end of the incisors. Elongation and deformation of crowns and roots lead to further severity in degrees of malocclusion, predisposing to fracture, pulpitis, and periapical infections. Severely deviated incisors can produce soft tissue lesions to the lips, tongue, hard palate, and nose. These types of lesions are more typical in rat-like rodents.

The development of cheek teeth malocclusion often follows a typical pattern, with few exceptions. Improper wear leads to excessive elongation of crowns and roots. This may not be apparent during inspection of the oral cavity because the gingiva often elongates with the crown, masking the elongation.^{5,6} Elongation of teeth is partially counteracted by the strength of masticatory muscles. The apposing forces result in curving of cheek teeth as they continue to grow. Lower cheek teeth typically begin to bend medially with lateral convexity while upper cheek teeth begin to bend laterally with medial convexity. This change is similar to the normal anatomy of the cheek teeth in guinea pigs but is abnormal in rabbits and chinchillas, which normally have a

horizontal occlusal plane. Therefore, these conditions are more difficult to recognize in guinea pigs and other porcupine-like rodents.^{1,4,8} The result is that the occlusal plane begins to malocclude, and lateral masticatory movements are not enough to fill the gap. Other forms of abnormal occlusal planes are described as “step mouth,” when individual molars elongate at different rates, or as “wave mouth,” when the entire occlusal plane is curved rather than flat.^{6,22} Worsening malocclusion results in improper wear, which again contributes to malocclusion. Typical molar malocclusion results in lack of wear between the lingual aspect of the lower cheek teeth and the buccal aspect of the upper cheek teeth. The result is an advanced stage of acquired dental disease leading to growth of spurs and spikes. These can be very sharp, creating lesions and ulcerations to the tongue (usually from lower cheek teeth) and the cheek mucosal surface (usually from upper premolars and molars).^{6,22}

Besides elongation of crowns, excessive elongation also occurs at the apex of tooth roots. The result is the stretching of cortical bone of the alveolus and possible perforation. Abnormal tooth roots cause a widening of the alveolar bone, which is an important predisposing factor for periapical abscessation.

Excessive elongation of crowns of the cheek teeth, particularly the premolars, may also lead to functional mandible prognathism. The jaw is forced open wider and more rostrally, which initially results in the malocclusion of incisor teeth. This condition is often improperly diagnosed as congenital and occurs most frequently in porcupine-like rodents. In these species, primary malocclusion of the incisors does not occur as frequently as in pet rabbits and is mostly related to acquired dental disease of cheek teeth.

Guinea pigs and chinchillas usually show less evidence of elongation malocclusion of crowns compared with pet rabbits.^{1,5,6,8,22} Spurs are rarely present, are not so long and sharp, and rarely cause lesions to the tongue. A typical lesion in guinea pigs is mesial elongation of either one or both lower premolars, creating a “bridge” over the tongue.¹ In chinchillas, obvious cheek teeth elongation occurs in the late stage of acquired dental disease.^{13,26}

The most common pathologic changes following acquired dental disease are periapical abscessation and osteomyelitis but can also include longitudinal fractures, the loosening of teeth, food impaction, and abscess fistulas. In cases of osteomyelitis, bone infection is usually focused at the apex of one tooth, but other teeth can be involved as well as maxillary or mandibular bone.^{1,6,8,10,21,23} Because of rapid tooth wear, plaque accumulation, periodontal disease, and caries are very rare in herbivorous species with open-rooted cheek teeth.⁵

Harcourt-Brown proposed metabolic bone disease as a cause of acquired dental disease in pet rabbits.^{7,19,20} Patients affected by severe malocclusion show poor calcification of the alveolar bone and, in general, all of the bones of the skull. This theory has been supported by a study performed on 81 pet rabbits that demonstrated abnormally high

parathyroid hormone concentrations and low calcium concentrations, suggesting that acquired dental disease in pet rabbits is associated with alterations in calcium metabolism.²⁰ Loss and weakness of alveolar bone lead to loosening of the teeth, distortions, change of positions, deformation, and eventually perforation of the periosteum. Malocclusion of crowns and the growth of spurs occur secondarily to improper wear as a result of abnormal position of cheek teeth. This condition has not been investigated in guinea pigs, chinchillas, or other rodent species.

The pathophysiology of odontoma of the apex of incisors in prairie dogs is still unknown but may be related to frequent incisor trauma.^{5,11,27} Incisor fractures are common in this species because they typically habitually chew the metal bars of their enclosures.

In rat-like and squirrel-like rodents, the pathophysiology of dental diseases of cheek teeth is mostly due to infections and abscessation following cavities and fractures of cheek teeth.

CLINICAL PRESENTATION^{1,2,5-8,12,13,15-18,24,26}

Presenting clinical signs and related pathologies have been described in the literature both for pet rabbits and rodents affected by dental disease. The most common are anorexia, dysorexia, dysphagia, excessive salivation, weight loss, poor general condition, poor coat conditions, digestive problems, changes of fecal droppings, ocular discharge, skin disease, and facial lumps.

Practitioners who are presented with a patient having suspected dental disease must consider a wide range of possible clinical signs. Among potential signs, one group can be considered specifically for dental disease (anorexia, dysorexia, dysphagia), another group is indicative of lesions closely related to dental disease (excessive salivation, ocular discharge, changes of fecal droppings, facial abscesses), and yet a third group indicative of systemic diseases (weight loss, digestive problems, poor general condition, skin diseases, death).

Among this wide range of presentations, some are more typical for selected species. In the author's experience, pet rabbits typically present for overgrowth of incisors, reduced food intake, dysphagia, and/or the presence of facial abscesses. Usually, infection of soft tissues presents as a large, firm, nonpainful facial mass and is more frequently located ventral or lateral to the mandible. However, infection may also involve the skull or the eye and present as a retrobulbar abscess. Abscesses are delimited by a thick capsule and contain white, very dense creamy purulent exudate.

Guinea pigs typically present with anorexia, and it is not uncommon for owners to report this as sudden onset. Chinchillas commonly present for excessive salivation, poor coat condition, and digestive problems but often are not anorexic.

Stretching and deformation of periosteum can produce subtle but important differences among species. This is usually most evident in pet rabbits, especially on the ventral

TABLE 2. Diagnostic Options in Suspected Dental Disease of Rabbits and Rodents and Related Significance

| <i>Diagnostic Test</i> | <i>Species</i> | | | |
|---------------------------------------|--|--|--|--|
| | <i>Rabbits</i> | <i>Porcupine-Like Rodents</i> | <i>Rat-Like Rodents</i> | <i>Squirrel-Like Rodents</i> |
| Dental examination without anesthesia | Always | Always; sometimes difficult in chinchillas; often not feasible in degus | Difficult with exception of selected individuals | Mostly not feasible |
| Dental examination with anesthesia | Always | Always | Always | Always |
| Skull radiography | Always | Always | Always | Always |
| Oral endoscopy | Very useful | Very useful or mandatory; mandatory in degus | Mandatory | Mandatory for most species; very useful in prairie dogs |
| Blood work and other examinations | In case of other diseases and/or complications related to dental disease | In case of other diseases and/or complications related to dental disease | In case of other diseases and/or complications related to dental disease | In case of other diseases and/or complications related to dental disease |
| Culture and sensitivity testing | In case of periapical infections or abscessations | In case of periapical infections or abscessations | In case of periapical infections or abscessations; less useful due to poor prognosis | In case of periapical infections or abscessations |
| Hystopathologic examination | Useful in selected cases | Useful in selected cases | Useful in selected cases | Useful in selected cases; useful in case of dental disease of incisors in prairie dogs |
| Computed tomography | Feasible, very useful, but not yet practical | Feasible, very useful, but not yet practical | A future perspective; no reported experiences | A future perspective; no reported experiences |

edge of the mandible. Periosteal and apex deformation of the upper cheek teeth predisposes to occlusion of the nasolacrimal duct, exophthalmos, and retrobulbar abscessation. Guinea pigs typically have much more severe deformation without as much clinical evidence. However, this condition appears to be much more painful, which is why the simple restoration of a proper occlusal plane can be unrewarding in this species.^{1,5,8} Chinchillas usually develop very severe root deformation and perforation of periosteum.^{5,12,13,26} Some show no clinical signs and seem to be able to tolerate severe disease much better and for a longer duration when compared with guinea pigs. Because disease tends to be severe by the time signs appear, early diagnosis is even more critical in this species, and for porcupine-like rodents in general, than for pet rabbits.

The most common presentation of hamsters and other rat-like rodents is malocclusion of incisors and facial abscesses.¹ Prairie dogs present with fractured incisors and, in some cases, dyspnea related to development of odontoma.^{1,5,27}

DIAGNOSTIC WORKUP

The diagnostic workup in cases of suspected dental disease must be thorough (Table 2). During physical examination of pet rabbits, it is mandatory to perform a complete examination of both incisor and cheek teeth. The practitioner must never neglect inspection of the oral cavity and cheek teeth because this can lead to misdiagnosis or omissions. Initial intraoral inspection can be performed with an otoscope if proper restraint is used. It can also be useful to introduce a finger into the mouth to check for the presence of sharp spurs, but this technique cannot replace intraoral inspection. If dental disease is suspected, a complete dental examination under anesthesia is critical. Inspection of the cheek teeth and oral cavity is even more difficult in porcupine-like rodent species, particularly in degus; therefore, examination under anesthesia is even more important. Occasionally, very calm rats or golden hamsters may allow intraoral examination without anesthesia, but this is an exception.

Anesthetic protocols for rabbits and rodents are

described in the literature. Endotracheal intubation can be performed in rabbits and in selected species of rodents. However, in the author's experience, the endotracheal tube impedes oral examination.^{1,22}

Specialized instruments have been designed to facilitate small, exotic mammal dentistry. Several companies manufacture rabbit and rodent mouth gags, cheek dilators, rasps, and even table-top mouth gags, which optimize the operator's view and reduce the need for assistants.

A thorough dental exam must include a radiographic examination of dental roots and apices.^{1,5-8,23} High-resolution radiography film is recommended. At least five radiographic projections of the skull are useful for each patient: lateral, right oblique, left oblique, ventrodorsal or dorsoventral, and rostrocaudal (skyline). Smaller films for dog intra-oral radiographs can also be used to obtain smaller views of the incisors and cheek teeth of pet rabbits.

It is also mandatory to know the normal radiographic anatomy of each species for a proper evaluation of pathologic changes. The most important peculiarity is related to guinea pigs. Due to the normal bending of cheek teeth in this species, the skyline projection is the only one that allows evaluation of the occlusal planes.^{1,4,5,8}

Optimal visualization of the oral cavity of rabbits and rodents is greatly facilitated by endoscopy, especially in smaller patients. Many avian and exotic practitioners are already familiar with the 2.7-mm endoscope and accessories and find it ideal for this purpose. Normal and pathologic patterns are described in the literature.^{1,22,24,28,29}

The use of computed tomography (CT) has been described in chinchillas.²⁶ Although it is currently not practical, CT represents a viable future diagnostic option.

Culture and sensitivity are important in cases of dental disease-related infection and abscessation. Rabbits and guinea pigs have long been used as laboratory models for human gingivitis and periodontal disease, and studies indicate anaerobic bacteria play a large role in infections and abscesses. Therefore requests for culture should specify screening for both aerobic and anaerobic organisms.^{10,30} Purulent material from the core of an abscess is usually sterile, which necessitates collection of samples from the abscess capsule wall.²³

Histopathology can also be useful in selected cases, particularly when bone neoplasia or dental dysplasia is suspected.

DIAGNOSIS AND PROGNOSIS

Accurate diagnosis and especially prognosis of dental disease in pet rabbits and rodents must be based on the results of a thorough diagnostic workup. Prognosis must be expressed in terms of severity of disease, age and condition of the patient, estimated level of continued care required, likelihood of owner compliance, and expected cost. Resolution of dental disease is possible, but a more common scenario is lifetime management. Dental abscesses carry the possibility of recurrence, even with aggressive therapy. The presence of osteomyelitis carries a guarded prognosis.

In general, the prognosis for pet rodents with dental disease is worse than that in rabbits due to anatomic and pathophysiologic peculiarities as well as the relative difficulty of surgical intervention.^{1,5,8} In cases of cheek teeth malocclusion in guinea pigs and chinchillas, the apices are usually overgrown and deformed with severe stretching of cortical bone.^{1,5,8,13} For this reason, reduction of dental arcades back to a normal occlusal plane may be partially or completely unrewarding due to the lack of pain relief.^{1,5,8} Guinea pigs seem also to be more severely affected by the stretching of masticatory muscles after prolonged elongation of dental arcades and can show delayed willingness to eat after treatment.⁸

Prognosis for rat-like rodents with abscesses is guarded to poor due to the difficulties of early diagnosis, surgical intervention, and extraction. Odontoma in prairie dogs carries a poor prognosis because of the presence of deformation of the apices and ankylosis within the alveolus.^{11,27,31}

MEDICAL TREATMENT

Most authors agree that medical therapy alone is generally inadequate for the treatment of dental disease, but it is an important adjunct to surgical therapy.^{10,21,23,32} Medical therapy should address several concerns, the first of which is control and management of systemic and soft tissue infections and osteomyelitis. As mentioned previously, antibiotic selection should be based on aerobic and anaerobic culture and sensitivity. Antibiotic selection must also take into consideration species-specific contraindications, such as oral penicillins in rabbits.³³ It must be emphasized that appropriate antibiotic selection for one species may be completely contraindicated in another species. Analgesia is critical for successful management of dental disease and to help prevent common, pain-related anorexia. Anorectic animals must be encouraged to eat as soon as possible. Commercial hand-feeding products for convalescing small exotic mammals are excellent for this purpose. Adjunct fluid therapy may be necessary in some patients.

Several strategies have been described for local therapy of abscesses, including frequent flushing, antibiotic-impregnated beads, honey and sugar therapy, and others.^{10,21,23,34-38}

SURGICAL TREATMENT AND OUTCOMES^{1,2,5-8,10,11,18,21-24,31,34-41}

Treatments of dental disease of incisors include coronal reduction, coronal amputation and partial pulpectomy, and extraction (Table 3). The first two options are limited to selected cases in which the malocclusion is not severe and has been detected early and proper restoration of occlusion of incisors and cheek teeth is possible. Coronal reduction of both incisors and cheek teeth must be performed using proper dental low-speed power equipment and burs.^{5,6,22} Alternatively, many authors use rotating hobby tools for this purpose.²²

Trimmers, clippers, and rasps must not be used to reduce the length of elongated incisors, especially when repeated treatment is necessary.^{5,24} Repeated use can result in

TABLE 3. Surgical Treatment Options for Dental Diseases in Rabbits and Rodents

| <i>Tooth Test</i> | <i>Species</i> | | | |
|-------------------------|---|---|--|---|
| | <i>Rabbits</i> | <i>Porcupine-Like Rodents</i> | <i>Rat-Like Rodents</i> | <i>Squirrel-Like Rodents</i> |
| Incisor teeth | Coronal reduction Crown amputation and partial pulpectomy Extraction | Coronal reduction Extraction | Coronal reduction Extraction | Coronal reduction Extraction in prairie dogs; check for indications in other species |
| Open-rooted cheek teeth | Coronal reduction and burring of spurs Extraction (intraoral or extraoral technique) Debridement of abscessations | Coronal reduction and burring of spurs Extraction (intraoral or extraoral technique) Debridement of abscessations | | |
| Rooted cheek teeth | | | Extraction (intraoral technique) Debridement of abscessations | Extraction (intraoral technique) Debridement of abscessations |

tooth fracture, exposure of pulp, and eventual infection. Moreover, this rough technique is painful when performed without anesthesia. Severe elongation or deformation of roots is also a common sequela, leading to other complications such as obstruction of nasolacrimal ducts, dacriocystitis, chronic ocular discharge, and ocular diseases such as conjunctivitis and retrobulbar abscess.

The only definitive and completely effective treatment for severe malocclusion of incisors is extraction. The technique and special dental instruments for extraction of incisors have been extensively described in literature.^{5-8,24,39,42} With rare exception, extraction of the entire set of incisors is necessary. Experience has shown that rabbits and herbivorous rodents adapt easily to the lack of incisors and readily learn to use lips for prehension of food.

Extraction can be extremely difficult in rat-like and squirrel-like rodents, particularly in smaller hamsters and mice.¹ In these cases, frequent trimming may become necessary. Some mammals like squirrels may not completely adapt to complete excision of incisors.

Another challenging extraction is the incisors of prairie dogs affected by odontoma or pseudo-odontoma. Severe deformation, adhesions, and ankylosis within the alveolar bone increase the difficulty of this procedure.¹¹ Some authors have reported use of techniques such as rhinotomy and access to teeth via the hard palate, again illustrating the difficulty of extraction in these cases and the overall poor prognosis.^{27,31}

Different options for treatment of dental disease of cheek teeth have been described, depending on the severity of pathologic changes and the anatomic type of the affected teeth.^{1,5-8,22-24} The most common indication is removal of

sharp spikes and spurs from the lingual and buccal aspects of the teeth and reduction of overall length of open-rooted cheek teeth of herbivorous species. In some cases, extraction can also become necessary. All these treatments can sometimes be combined in a single patient, especially in rabbits.

Radiographs may give the best indication for the amount of coronal reduction that must be done in specific patients because in many cases overgrowth of gingival tissue may give a false impression of the overall length of cheek teeth when they are viewed directly. In some cases, it is necessary to remove excessive gingival tissue as well.^{5,6}

Early and repeated treatments are critical to prevent bending of crowns and apical changes because normalcy cannot be restored once they occur. These changes are much more frequent in guinea pigs and chinchillas than in rabbits, and this can make a great prognostic difference in cases of advanced, acquired dental disease.

In cases of fracture, loose teeth, and/or periapical abscessations, it may be advisable to extract one or more cheek teeth. Small mammal dental luxators have been specially designed for this purpose.

Extraction of cheek teeth may be very challenging, depending on the species, position of the tooth to be extracted, and conditions of the tooth. Even after proper luxation, it is easy to fracture the tooth during extraction. Sometimes it is necessary to cut the crown during extraction because the narrow opening of the mouth won't allow one extraction of the entire cheek tooth. In most cases, where an advanced stage of acquired dental disease is present, complete intraoral extraction of cheek teeth is impossible due to root fracture or ankylosis into the alveolar bone.

When the intraoral approach to extraction is not feasible or not effective, the extraoral approach for extraction of lower cheek teeth has been described.^{5,23} Surgical access to the apex is performed on the ventrolateral edge of the mandible. The cortical bone is burred to create a fenestration under the apex. The entire tooth or some remaining fragments can be extracted from this site or pushed into the oral cavity and removed from there. After an extraoral approach, soft tissues may be sutured or not, depending on the aseptic level maintained during the procedure.

After intraoral extraction, the socket must be protected to prevent food impaction and infection. After cleansing with chlorhexidine, the gingiva can be sutured to cover it or the socket can be filled with Doxirobe gel.^{6,8} When a single tooth is extracted in open-rooted species, the opposite tooth continues to grow, necessitating extraction or frequent trimming.^{5,6} Alternative techniques to arrest the growth of the opposing tooth have been described. These include apical cryotherapy and surgical apicoectomy.⁵

Surgical treatment of periapical infections is designed to remove the entire abscessation with the whole capsule (the soft tissues will eventually be affected by infection outside the capsule) and to debride the bone site of osteomyelitis, including the extraction of the tooth or teeth involved or their fragments.^{10,23,24} Local and systemic antibiotic therapy is also mandatory to resolve infections after surgical debridement.

In the majority of cases, simple incision of the abscess and flushing of purulent exudates is not effective to prevent recurrences. In addition, the thick pus is often sterile and the bacteria involved, both aerobic (*Streptococcus*, *Staphylococcus*, *Pasteurella*) and anaerobic (*Fusobacterium*, *Actinomyces*),^{10,21,30} are present on the inner surface of the capsule. For this reason, the capsule has to be surgically removed and a sample submitted for culture and sensitivity testing.

Surgical guidelines for excision of abscessations and debridement of the osteomyelitic site have been described.^{10,23} The capsule of the abscess should ideally not be perforated until it is completely dissected from surrounding soft tissues. Then, the capsule attachment to the cortical bone is dissected and the abscess removed. The osteomyelitic site is flushed with sterile saline, periosteal necrotic tissue is removed, and bone is debrided down to bleeding tissue. Usually, the tooth or teeth (or their fragments) from which the periapical infection originated appear in the bone cavity and are removed. In the author's experience, removal of tooth fragments is mandatory to prevent recurrence, even if other anecdotal reports show complete healing without this step.

Different options have also been reported at the end of this surgical procedure.^{10,21,23,29,34-37} Antibiotic-impregnated polymethylmethacrylate beads can be placed into the bone defect after debridement, and the surgical site is sutured.^{10,21,34,43} This will prevent additional flushing after surgery, while the beads release antibiotics locally, maintaining therapeutic levels. Beads can be removed or left in place if removal is not practical. In addition to beads, wounds have

been packed with antibiotic-moistened gauze strips. Antibiotic selection was based on culture and sensitivity but included drugs such as penicillins, cefazolin, and metronidazole.³⁸

Calcium hydroxide has been used to fill the osteomyelitic site. The basic pH (12.0) of this product is very effective as an antimicrobial.³⁷ Nevertheless, this material can severely damage surrounding soft tissues, and unsuccessful outcomes have been reported.

The bactericidal effects of natural substances such as honey and sugar have been reported in other species for different infections and may be useful in the management of infections in pet rabbits.^{35,36} Anecdotal reports of the successful use in the abscesses of pet rabbits have been reported.

Bioactive ceramics are used to fill bone defects after dental surgery. These products can attach to both soft tissues and bones and are osteoconductive.¹⁰

The author's treatment of choice for dental abscesses is marsupialization of soft tissues around the osteomyelitic socket.²³ This allows continuous flushing and debridement of both hard and soft tissues. Surgery is followed by the introduction of antibiotic ointment for 3 to 4 weeks until second-intention healing occurs. This requires intensive postsurgical care and initially results in an unaesthetic appearance, which must be discussed with the owners in advance.

Systemic antibiotic therapy that is effective for both aerobic and anaerobic bacteria is administered after surgery and maintained or modified on the basis of results of culture and sensitivity tests for at least 3 to 4 weeks.^{10,32}

Patients surgically treated for periapical abscessations should be monitored closely due to the high incidence of recurrence. Follow-up should be continued until radiographs demonstrate healing of bone tissue at the initial site of osteomyelitis.

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CLASSIFIEDS

Exotic Animal, Wildlife, & Zoological Medicine Internship at University of Georgia

The exotic animal, wildlife, and zoological medicine 1-year internship emphasizes clinical training in basic and advanced principles of exotic animal medicine and surgery. The clinical work is approximately distributed as follows: 45% avian, 35% mammals (including primates), 15% reptiles (including venomous), and 5% fish/amphibians/invertebrates. There will also be opportunities to participate in clinical research. Publication in peer-reviewed scientific journals or other veterinary periodicals is encouraged. Past interns of this program have been very successful at achieving residency/zoo placements. This program is also accredited by the Royal College of Veterinary Surgeons as an approved center for specialist training in zoological medicine. Application must be made through the Veterinary Internship and Residency Matching Program. Details are available from www.virmp.org/virmp/

For questions about this program, please contact either of the following individuals: Stephen Hernandez-Divers, BvetMed, DzooMed, MRCVS, RCVS Specialist in Zoo & Wildlife Medicine, shdivers@vet.uga.edu, phone 706-542-6378, or Heather Wilson, DVM, ABVP (Avian), hwilson@vet.uga.edu, phone 706-542-6328.

Selected Abstracts on Exotic Mammal Medicine and Surgery

Immunization of Ferrets Against *Dirofilaria immitis* by Means of Chemically Abbreviated Infections

Flair LS, Campbell WC: *Parasite Immunol. Summer*:3(2):143–147, 1981.

ABSTRACT

Ferrets were exposed to two successive inoculations with 30 third-stage larvae of the canine heartworm *Dirofilaria immitis*, with inoculations given approximately 5 months apart. Each infection was terminated by ivermectin therapy approximately 2 months after inoculation. The ferrets were challenged with 30 larvae 3 weeks after the termination of the second infection and were necropsied approximately 6 months after the challenge. Of the four ferrets that survived this protracted experimentation, two were entirely free of heartworms and two had only a single female worm each. By contrast, 14 control ferrets that had not been immunized (four of which had been given ivermectin doses) were all infected at necropsy, yielding a mean of 6.6 worms per ferret.

Dirofilariasis in the Domestic Ferret.

McCall JW: *Clin Tech Small Anim Pract. May*:13(2):109–112, 1998.

ABSTRACT

The popularity of pet ferrets in heartworm-endemic and nonendemic areas is growing, with ferret ownership in the United States currently exceeding 10 million. The domestic ferret (*Mustela putorius furo*) has been reported to be susceptible to naturally acquired and experimentally induced infections of *Dirofilaria immitis*. Host–parasite

relationships between *D. immitis* and domestic dogs and cats have been well studied, but there have been relatively few reports on infections in ferrets. Laboratory studies have shown the ferret to be highly susceptible, with infection and recovery rates similar to those achieved in dogs and higher than those seen in cats. Microfilaremia is characteristically of low concentration and transient in nature, similar to that seen in heartworm-infected cats. A definitive diagnosis can be made from ELISA-based antigen tests, echocardiography, and angiography, but suggestive radiographic findings require additional supportive information to confirm a tentative diagnosis. Prevention has been shown to be effective with currently used canine prophylactic pharmaceuticals, but effective treatment of adult heartworms in ferrets has not yet been confirmed by controlled studies. There is currently no approved drug for prevention or treatment of *D. immitis* in ferrets.

Efficacy of Ivermectin Against Third-Stage *Dirofilaria immitis* Larvae in Ferrets and Dogs

Blair LS, Williams E, Ewanciw DV: *Res Vet Sci. Nov*:33(3):386–387, 1982.

ABSTRACT

Ivermectin prevented maturation of *Dirofilaria immitis* when given orally to ferrets at a dosage of 0×1 mg ivermectin per kg body weight 2 days after inoculation with third-stage heartworm larvae. Similar results were found in dogs treated 1 day after infection at a dosage of 0×05 mg ivermectin per kg. No heartworms were found in the hearts and lungs of five treated dogs compared with an average of 11 worms per dog in five control animals.

COMMENTARY

*These three abstracts on this page illustrate the potential significance of heartworm disease in ferrets. It is interesting to note that ferrets are considered highly susceptible, with infection and recovery rates more similar to dogs than cats. However, microfilaremia is brief, which is more similar to the disease progression in cats. Studies have shown that common canine prophylactic regimens are effective in ferrets. An additional study showed that ivermectin is effective in preventing maturation of *Dirofilaria* in ferrets and has been used to successfully terminate heartworm infections in research animals. Also of interest is a challenge study demonstrating that ferrets that have been successfully treated for dirofilariasis mount an immune response that effectively prevents or reduces future infections. This information can help practitioners make decisions regarding their patient's relative risk for heartworm infection and help to design reasonable prevention strategies.*

Efficacy and Safety of Topical Administration of Selamectin for Treatment of Ear Mite Infestation in Rabbits

McTier T, Hair JA, Walstrom D, Thompson L: *JAVMA* 223(3): 322–324, 2003.

ABSTRACT

Psoroptes cuniculi is a common parasite that inhabits the body and ears of rabbits. As many as 10,000 mites may inhabit a single pinna, resulting in severe otitis externa. Removing the crusts and scabs associated with the mite infestation and treating the ears topically is often painful.

Psoroptes mites are generally sensitive to macrocyclic lactones, including ivermectin. However, multiple subcutaneous injections of ivermectin at varying intervals are generally needed to kill all the mites. Another macrocyclic lactone of the avermectin subclass, selamectin, is absorbed percutaneously after topical administration in dogs and cats. It reaches rapid plasma and tissue concentrations that are sustained for several weeks; therefore, a single application may result in complete resolution of topical mite infestations.

Forty-eight male and female mixed-breed domestic rabbits naturally infected with *Psoroptes* mites were used to determine the efficacy and safety of topical selamectin therapy. Rabbits were divided into six groups according to lesion severity and treated topically with either (isopropyl alcohol and glycol methyl ether) or variable doses of selamectin, according to body size. Selamectin was dosed at 6 and 18 mg/kg. Either selamectin or vehicle was applied directly to the skin at the base of the neck. Half of the groups received a second dose of vehicle or selamectin on day 28. Otoscopic examination for viable mites and determination of ear lesion were performed at weekly intervals throughout the 57-day study.

At the conclusion of the study, all animals were euthanized, populations of mites counted, and lesions measured. All rabbits in the control groups were positive for mites for the duration of the study, while all four selamectin-treated groups were free of mites from days 7 through 56. No adverse clinical signs were observed for the duration of this study. This report suggests a single application of selamectin, at a dose of 6 to 18 mg/kg, is a safe treatment option for complete elimination of mites from rabbits.

COMMENTARY

The apparent efficacy of selamectin for the treatment of Psoroptes mite infestations offers another treatment option to add to our therapeutic tool chest. Ivermectin has been shown to be effective in the treatment of ear mites in rabbits but necessitates the additional injection of drug within 7 to 14 days for complete resolution of infestation. The cost, efficacy, and convenience of treating rabbits with a single dose of selamectin must be taken into consideration. In this study, animals were housed singly, minimizing the possibility of ingestion of this

product. While no adverse reactions were seen with topical selamectin, detailed toxicity and oral ingestion studies have not been performed. Selamectin is not approved for use in rabbits.

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Ascorbic Acid Increases the Severity of Spontaneous Knee Osteoarthritis in a Guinea Pig Model

Kraus VB, Hueber JL, Stabler T: *Arthritis Rheum* 50(6):1822–1831, 2004.

ABSTRACT

Ascorbic acid (vitamin C) has been shown to play a role in various factors associated with joint health, including serving as a cofactor in extracellular matrix production, which is important for collagen synthesis; acting as a stimulant of messenger RNA synthesis of several cartilage matrix components; acting as a stimulus to protein expression of type II collagen and proteoglycan in articular cartilage; and possibly serving as an antioxidant that protects cartilage degradation. This study aimed to determine whether supplemental ascorbic acid might be of benefit for the treatment of spontaneous osteoarthritis when administered over a prolonged period of time. (Recall that guinea pigs, like humans, possess a nonfunctional gene for L-gulonolactone oxidase that renders them unable to synthesize ascorbic acid.) The study, using 46 male Harley guinea pigs, was designed to investigate the effects of 8-month exposure to low (2.5–3 mg/day), medium (30 mg/day), and high (150 mg/day) doses of ascorbic acid on the development of histologic knee osteoarthritis.

After 8 months, the guinea pigs were humanely euthanized and stifle joints were examined histologically. Results showed that ascorbic acid increased osteoarthritis in a dose-dependant manner, with increases in cartilage damage, proteoglycan loss and small- to medium-sized osteophytes at the joint margins being most severe in the high-dose (150 mg ascorbic acid daily) group.

Ascorbic acid has been shown to activate latent transforming growth factor-β (TGF-β). Prolonged exposure to TGF-β has been shown to cause osteoarthritis-like changes. The study found increased TGF-β levels in the osteophytes of diseased joints and that osteophyte size and number climbed higher with increased ascorbic acid intake. It was therefore theorized that prolonged ascorbic acid exposure may be mediated in part by TGF-β.

COMMENTARY

This study suggests that guinea pigs should not be supplemented with more than the currently recommended dietary dosage of ascorbic acid: 30 to 50 mg/day in healthy guinea pigs

Diagnosis and Treatment of Dental Disease

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linemic state, which promotes beta cell hyperplasia and, eventually, neoplasia.

While the probability of a genetic basis for insulinoma formation should not be discounted, it is suggested that a nutritional strategy offers a more expedient approach to prevention of this common endocrinopathy.

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Slide Shows for Clients

If you have a computer in your exam room, you can turn its monitor into a slide show for your clients while they wait. A software program called *Show Time* (www.alienszone.com) can help convert presentations you have created in programs such as PowerPoint into screen savers. After creating the screen saver, you can install it as the default screen saver on your exam room computer. When you leave the room, simply activate the screen saver.

Exotic DVM of the Year Named

Dr. Peter Fisher was awarded the Exotic DVM of the Year by *Exotic DVM* magazine. Congratulations, Peter!

AEMV News

2004 and 2005 "First Step" Meetings: AEMV's 2004 First Step meeting, held May 4 in conjunction with the International Conference on Exotics, was a resounding success. Drs. Susan Kelleher, Heidi Hoefler, and Peter Fisher spoke on rabbit medicine, rabbit and rodent dentistry, introduction to hedgehogs and chinchillas, ferret medicine, and small mammal surgery. The 60 attendees included veterinary students, veterinary technicians, and international veterinarians. Our meeting continues to receive support by the Zoological Education Network and *Exotic DVM* magazine. Planning is now under way for the 2005 First Step meeting, which will be expanded from 4 to 8 hours of CE and will focus on dental disease in rabbits and rodents, including a wet laboratory. For more information, visit www.aemv.org.

2005 Membership Dues Update: Requests for dues for 2005 will be sent out in January 2005. Email will be used extensively, so please make sure your email address is updated on the AEMV Web site (www.aemv.org). If you either renewed your AEMV dues or joined AEMV via PayPal, you will need to go into your account and cancel the subscription. Dues were set up as an annual subscription and therefore will automatically renew 1 year from the date of renewal or activation. Canceling your subscription will not cancel payment, only the automatic renewal.

ABVP Board Certification Process Moves to Next Stage: We have completed stage 1 of the board certification process for exotic mammals by the American Board of Veterinary Practitioners. We are working hard on stage 2. If you are interested in helping with this endeavor, please contact Dr. Mike Dutton at mdutton@weareanimalhospital.com.

2004 Annual Business Meeting Minutes: Minutes of the 2004 Annual Meeting can be found online (www.aemv.org).

Veterinary Student Case Report Contest: The AEMV is sponsoring a Veterinary Student Case Report contest. The subject of the case report must be a pet exotic mammal that is commonly seen by companion animal/exotics veterinarians. Complete author guidelines are available at www.aemv.org. The winner will receive free admission to the AEMV-sponsored First Step 2005 Lecture and Wet Laboratory. Also included is a 2-night hotel stay at the Harbor Beach Marriott, conference hotel for ICE 2005. The deadline for entry is January 15, 2005.



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and 100 mg/day in sick or nutritionally stressed guinea pigs. Ascorbic acid is relatively unstable, and it is felt that most commercial guinea pig pellets are a poor source of this essential nutrient. As a result, additional ascorbic acid in the form of fresh vegetables or commercial supplements with known ascorbic acid concentration must be considered. At the same time, indiscriminant supplementation is discouraged.

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Efficacy of Six Anthelmintics Against Luminal Stages of *Baylisascaris procyonis* in Naturally Infected Raccoons (*Procyon lotor*)

Bauer C, Gey A: *Vet Parasitol.* Nov:60(1-2):155-159.

ABSTRACT

The efficacy of six anthelmintics against natural infections of *Baylisascaris procyonis* in raccoons (n = 7 per drug) was determined in a series of critical tests. The drugs were given via moist cat food as a single dose or once daily for 3 consecutive days. Raccoons treated with pyrantel embonate (1 × 20 mg/kg body weight, ivermectin (1 × 1 mg/kg), and moxidectin (1 × 1 mg/kg) expelled 1-198, 2-24, 2-14, 3-80, 2-70, or 2-35 *B. procyonis* stages, respectively, within the feces. No roundworm was detected in any raccoon at post-mortem examinations 7 days after the end of treatment. These results suggest that any of the six anthelmintics can be used at the dose rates tested in a deworming program for captive raccoons.

COMMENTARY

Although raccoons are usually considered undesirable as pets, exotic animal practitioners are occasionally asked to treat them. Baylisascaris is a common parasite of raccoons and carries a significant zoonotic risk to humans. Although it is assumed that a number of anthelmintics would be effective in the treatment of Baylisascaris, this study demonstrates the efficacy of five different drugs to clear infections in naturally infected raccoons. This information is valuable for clinicians planning effective treatment and prevention strategies for pet raccoons.