

# Prevalence of deafness and association with coat variations in client-owned ferrets

Stéphanie Piazza, DMV; Marie Abitbol, DMV, PhD; Kirsten Gnirs, DMV;  
Minh Huynh, DMV; Laurent Cauzinille, DMV

**Objective**—To evaluate the prevalence of congenital sensorineural deafness (CSD) and its association with phenotypic markers in client-owned ferrets.

**Design**—Epidemiological study.

**Animals**—152 healthy European pet ferrets.

**Procedures**—Brainstem auditory evoked response tests were recorded in ferrets during general anesthesia. Phenotypic markers such as sex, coat color and pattern, coat length (Angora or not), and premature graying trait were assessed.

**Results**—Overall, 44 of the 152 (29%) ferrets were affected by CSD; 10 (7%) were unilaterally deaf, and 34 (22%) were bilaterally deaf. There was no association between CSD and sex or Angora trait, but a strong association between CSD and white patterned coat or premature graying was identified. All panda, American panda, and blaze ferrets were deaf.

**Conclusions and Clinical Relevance**—The ferrets in this study had a high prevalence of CSD that was strictly associated with coat color patterns, specifically white markings and premature graying. This seemed to be an emerging congenital defect in pet ferrets because white-marked coats are a popular new coat color. Breeders should have a greater awareness and understanding of this defect to reduce its prevalence for the overall benefit of the species. (*J Am Vet Med Assoc* 2014;244:1047–1052)

Congenital sensorineural deafness in pet ferrets is a controversial subject among breeders and owners. It seems to be associated with white or white-marked coats as it is in other species.<sup>1,2,a</sup> Coat color-related sensorineural deafness has been described in numerous species, including cats, dogs, horses, mink, mice, guinea pigs, llamas, and alpacas.<sup>3–5</sup> A common phenotype for these animals, regardless of the species, is that they have substantial white or merle- or dappled-colored coats.<sup>5,6</sup> Deafness is linked to failure of migration or maturation, premature death, or dysfunction of neural crest melanocytes in the inner ear, in a region of the cochlea known as the stria vascularis.<sup>5</sup> There are many theories and misconceptions about this subject in ferrets, including the notion that other neural crest defects are associated with white markings and deafness. However, it appears that they are not confirmed by scientific studies and to the authors' knowledge, the prevalence and inheritance of CSD in ferrets are unknown.

From the Department of Neurology (Piazza, Cauzinille) and Exotics (Huynh), Veterinary Hospital Center FREGIS, 43 ave Aristide Briand, 94110 Arcueil, France; INRA, UMR955 Functional and Medical Genetics Unit; Université Paris-Est Créteil, Ecole nationale vétérinaire d'Alfort, 7 ave du Général de Gaulle, 94704 Maisons-Alfort Cedex, France (Abitbol); and the Department of Neurology, Veterinary Clinic ADVETIA, 5 rue Dubrunfaut, 75012 Paris, France (Gnirs). Dr. Piazza's present address is Willows Veterinary Centre and Referral Service, Highlands Rd, Solihull, West Midlands, B90 4NH, England.

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Address correspondence to Dr. Piazza (spiazzavet@yahoo.fr).

## ABBREVIATIONS

BAER	Brainstem auditory evoked response
CSD	Congenital sensorineural deafness
DEW	Dark-eyed white
MITF	Microphthalmia-associated transcription factor

Traditional clinical tests to assess hearing are difficult to perform in active animals such as ferrets and are not appropriate for detection of unilateral deafness. The BAER test has been used successfully to identify deafness in ferrets that were thought to have normal hearing. This electrodiagnostic test provides information about the functional state of the peripheral and brainstem component of the auditory portion of the nervous system and can be defined as the electrical response of the auditory pathway to a series of auditory stimuli.<sup>7</sup> This procedure can be easily performed in a clinical setting in ferrets as a screening test before adoption.<sup>b</sup> Brainstem auditory evoked response testing is an objective method for the diagnosis of CSD and can potentially be used in a program for screening and selecting breeding stock.

The objectives of the study reported here were to evaluate the prevalence of CSD and its association with phenotypic markers in client-owned ferrets by use of the BAER test. The hypothesis was that CSD is associated with certain coat colors or patterns in ferrets.

## Materials and Methods

**Animals**—Data were compiled from ferrets evaluated between 2008 and 2012 at Veterinary Hospital Center FREGIS, Arcueil, France (n = 118) and the Veterinary

Clinic ADVETIA, Paris, France (34). Data were obtained prospectively from 110 ferrets and retrospectively from the medical records of 42 ferrets. All ferrets were healthy. Breeders provided most of the ferrets evaluated and included those suspected of having hearing abnormalities as well as those with clinically normal hearing. All breeding and nonbreeding ferrets (from various bloodlines) of each breeder were tested. Breeders also routinely requested testing of entire litters prior to adoption. The remaining ferrets were client-owned pets, evaluated for deafness or with clinically normal hearing and evaluated because their owners were interested in having their ferrets included in the study. For all prospective cases, informed owner consent was obtained prior to study enrollment. The protocol was approved by the French animal welfare regulatory authority (Direction Départementale de la Protection des Populations). The study was also conducted in accordance with the Directive 2010/63/EU of the European Parliament and of the Council on the protection of animals used for scientific purposes.

**Phenotypic data**—For each ferret, sex, coat color and pattern, coat length, and premature graying trait



Figure 1—Representative photograph of an albino ferret.

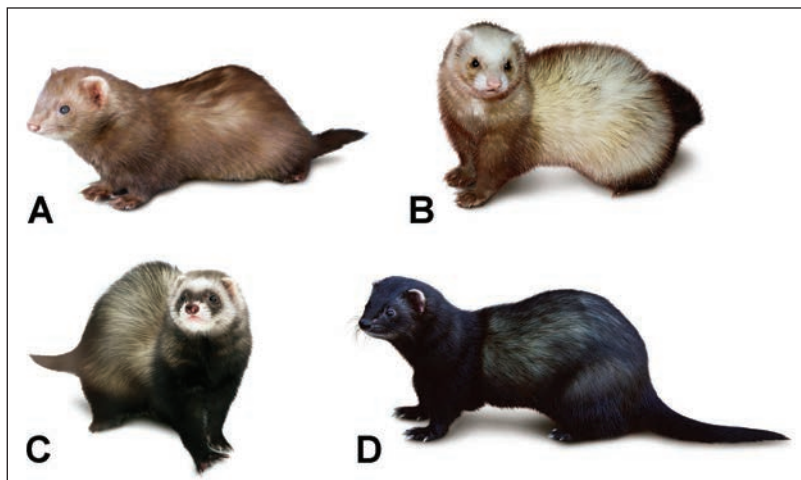


Figure 2—Representative photographs of ferrets with the 4 basic coat colors without white markings: chocolate (A), pastel (B), sable (C), and black (D).

were recorded, although there is currently no recognized worldwide standard for coat color in ferrets and terminology can vary depending on country and breeder. The classification used here was based primarily on color and in accordance with the Coat Color Standard and Pattern of the American Ferret Association, and was modified to include colors and patterns that did not exist in this standard. Coat color was divided into 3 classes: coat without white markings, coat with white markings, and albino. Albino ferrets were solid white with red eyes because of deficient pigmentation in the skin, eyes, and hair (Figure 1). Coat colors without white markings included sable, pastel, black, and chocolate (Figure 2). Coats with white markings were classified according to the white pattern (Figure 3); all ferrets of this class have white feet. Panda ferrets have an entirely white head, whereas American panda ferrets have large white areas anywhere on the trunk, head, and feet. Blaze ferrets have a white stripe on the head that begins on the muzzle and extends to at least the level of the ears. Dark-eyed white ferrets are solid white, but unlike albino ferrets, they have dark-colored rather than pink eyes. Juvenile DEW ferrets also have a darkly pigmented line along their back. Silver ferrets have a variable proportion of white hairs disseminated throughout the coat. Ferrets with a coat pattern including white feet and a white bib and sometimes other small patches of white on the ventral portion of the abdomen, stifle areas, and head were classified as having a mitt pattern. Some coats with white markings change over time and turn light gray or completely white. This trait is called premature graying and is seen in ferrets by 1 to 3 years of age (Figure 4); therefore, after testing, a follow-up check of coat color was done when possible. Angora ferrets are characterized by a long coat, lack of an undercoat, bifid nostrils (extra fold of skin on the nostrils), and small tufts of hair on or inside the nose.

**BAER testing protocol**—Brainstem auditory evoked response tests were performed following established methods in dogs that are practicable in ferrets,<sup>b</sup> with 2 standard electrodiagnostic machines (1 at each study location).<sup>c,d</sup> Ferrets were tested during general anesthesia induced by administration of isoflurane<sup>e</sup> by mask. Ferrets were placed on a heated circulating-water pad to maintain body temperature, and rectal temperature was monitored. Duration of anesthesia was approximately 10 minutes for each ferret. Ooscopic examination of both ear canals was performed in all ferrets prior to BAER measurements. Ferrets with a clinical abnormality (eg, cerumen accumulation and infection), with a history of either ear disease or administration of drugs with potential ototoxicity or with BAER waveforms consistent with conductive deafness, were excluded from the study. Potentials were recorded from subcutaneously placed needle electrodes; the reference electrode was placed at the vertex of the head, and the ground electrode was inserted along the dorsal mid-

line. Recording electrodes were placed at the base of each ear. Alternating click sequences were generated at 90-dB sound pressure level for the first machine and 90-dB normal hearing level for the second machine (each machine at a different veterinary clinic) by use of earphones placed in the external ear canal. Sensorineural deafness is typically characterized by an absence of expected waves for the affected ear.

**Statistical analysis**—Each ferret was classified as bilaterally hearing, unilaterally deaf, or bilaterally deaf on the basis of BAER test results. Deafness prevalences were calculated from the BAER data. Association between hearing status and phenotypic markers was tested by means of  $\chi^2$  tests with Yates correction when an expected value was  $> 3$  but  $< 5$ .<sup>18</sup> The level of significance was set at  $P < 0.05$ .

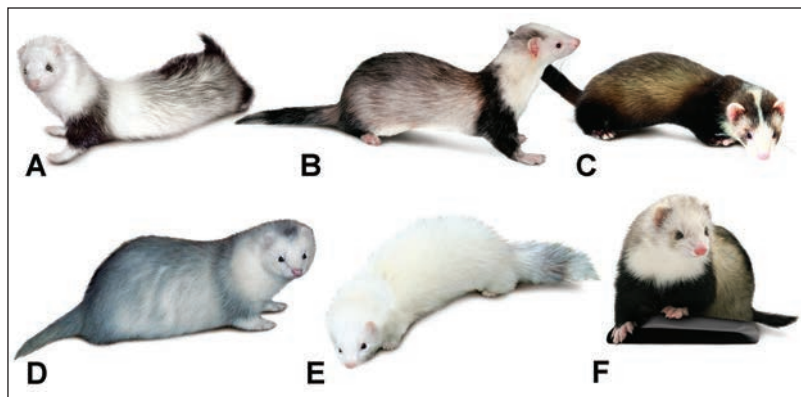


Figure 3—Representative photographs of ferrets with 6 white patterns: panda (A), American panda (B), blaze (C), silver (D), DEW (E), and mitt (F).

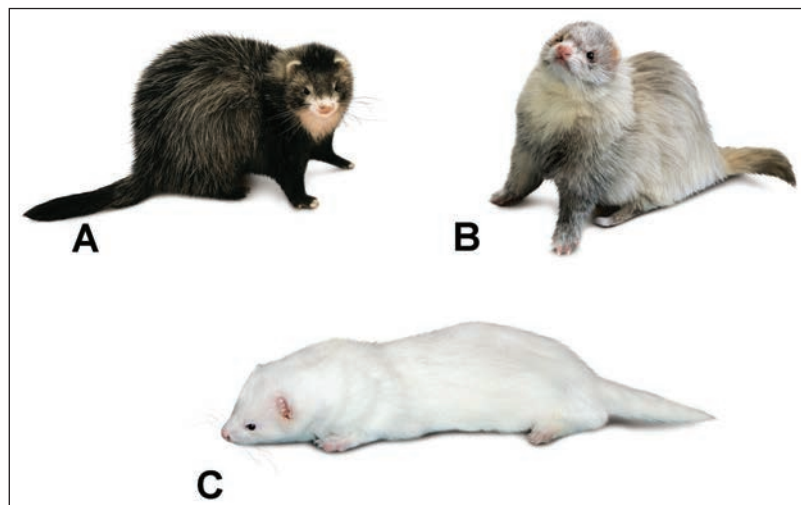


Figure 4—Representative photographs of a mitt ferret with premature graying at 2 months of age (A), 1 year later (B), and 2 years later (C).

## Results

**Animals**—Inclusion criteria were met for 152 ferrets, including 67 males and 85 females (27 Angoras and 125 shorthairs). All colors and patterns were represented. Age ranged from 8 weeks to 6 years. Ferrets were from various European lineages (French, Belgian, German, Dutch, and Danish).

**Prevalence**—Of the 152 ferrets tested, 108 (71%) had normal hearing and 44 (29%) were deaf. Of the 44 deaf ferrets tested, 10 (7%) had unilateral deafness and 34 (22%) had bilateral deafness (Table 1).

**Sex**—There was no significant difference between the number of males ( $n = 67$ ) and females (85) in the cohort ( $P = 0.14$ ) or between the number of males and females with normal hearing versus CSD ( $P = 0.14$ ). Additionally, there were no differences between numbers of unilaterally deaf males and females ( $P = 0.53$ ) and numbers of bilaterally deaf males and females ( $P = 0.73$ ); therefore, no association between deafness and sex was identified (Table 1).

**Angora trait**—Prevalence of deafness between Angora and non-Angora ferrets was not significantly ( $P = 0.19$ ) different. Therefore, there was no association between hearing status and the Angora trait (Table 1).

**Coat color and pattern**—A significant ( $P < 0.001$ ) difference was observed

Table 1—Prevalence (number [%]) of deafness in 152 ferrets according to various phenotypic markers.

Phenotypic marker	Bilaterally hearing	Unilaterally deaf	Bilaterally deaf	Total deaf	P value*
Total cohort ( $n = 152$ )	108 (71)	10 (7)	34 (22)	44 (29)	—
Males ( $n = 67$ )	45 (67)	6 (9)	16 (24)	22 (33)	0.14
Females ( $n = 85$ )	63 (74)	4 (5)	18 (21)	22 (26)	
Nonalbino ( $n = 142$ )	99 (70)	10 (7)	33 (23)	43 (30)	0.31†
Albino ( $n = 10$ )	9 (90)	0 (0)	1 (10)	1 (10)	
No white markings ( $n = 63$ )	63 (100)	0 (0)	0 (0)	0 (0)	$< 0.001$
White markings ( $n = 79$ )	36 (46)	10 (13)	33 (41)	43 (54)	
No premature graying ( $n = 83$ )	75 (90)	4 (5)	4 (5)	8 (10)	$< 0.001$
Premature graying ( $n = 23$ )	3 (13)	2 (9)	18 (78)	20 (87)	
Non-Angora ( $n = 125$ )	86 (69)	9 (7)	30 (24)	39 (31)	0.19
Angora ( $n = 27$ )	22 (81)	1 (4)	4 (15)	5 (19)	

\* $\chi^2$  Tests were performed with total numbers of deaf ferrets (including unilaterally and bilaterally deaf animals). †Yates correction was applied.

in prevalence of deafness between white-marked ferrets and non-white-marked ferrets (Table 1). All panda ( $n = 11$ ), American panda (7), and blaze (9) ferrets were deaf. On the other hand, all ferrets without white markings ( $n = 63$ ), including 8 black, 3 chocolate, 15 pastel, and 37 sable ferrets, had intact hearing. Only 1 of the 10 albino ferrets tested was deaf. Eight of 26 (31%) mitt ferrets without other white markings and 3 of 14 mitt ferrets with other white markings were deaf. Two of 5 DEW ferrets and 3 of 7 silver ferrets were deaf.

**Premature graying**—The percentage of deaf ferrets was significantly ( $P < 0.001$ ) higher among ferrets with premature graying (87%) than in ferrets without premature graying (9.6%; Table 1). Because this trait changes over time, data were available and analyzed for 106 of 152 ferrets.

## Discussion

This study identified a high prevalence of CSD in ferrets. In dogs, > 80 breeds have been identified with CSD and deafness is routinely and objectively assessed by means of BAER testing in breeds at risk.<sup>2,6</sup> The highest prevalence (30%) of pigment-associated CSD, with 8% bilateral and 22% unilateral involvement, has been reported in Dalmatians from the United States.<sup>6,8-11</sup> In the present study, the observed prevalence of CSD in ferrets (29%) was similar to that observed in US Dalmatians. However, in ferrets, bilateral CSD was more common than unilateral CSD (22% vs 7%).

A large number of ferrets were recruited for this study and represented the full spectrum of colors and color patterns, ages, and coat length. Some ferrets were suspected of having deafness, but most were clinically normal. The cohort included entire litters of ferrets, ferrets used as breeding stock, and neutered pet ferrets from various lineages. Nevertheless, only European ferrets were represented, and comparison of these data with data obtained in American ferrets will be necessary in the future.

In this study, we observed no significant association of CSD with sex or with Angora trait, but a strong association with coat color was identified. Ferrets with no white markings were not affected by CSD, whereas panda, American panda, and blaze were all deaf. Only 1 albino ferret had CSD. It is known that white coat color can be the result of expression of 2 phenotypic traits: white markings and albinism. Occulocutaneous albinism is caused by lack of pigmentation in the hair, skin, and eyes and occurs in several animal species, including ferrets.<sup>12</sup> Albino ferrets are homozygous for a mutation identified in the *TYR* (tyrosinase) gene.<sup>12</sup> In albino animals, melanocytes are present but tyrosinase, an enzyme that catalyses 3 steps in melanin synthesis, is inactive.<sup>12,13</sup> During embryological development, melanoblasts are distributed normally but persist as clear cells.<sup>14</sup> Albinism is thus the result of impaired expression or function of a pigment-producing gene.<sup>15</sup> By contrast, white spotting results from defects in pigment cell differentiation, proliferation, or migration from the neural crest to skin or hair during development.<sup>3,16,17</sup> This results in lack of melanocytes in the entire skin or

in patches. White spotting is therefore the result of impaired expression or function of pigment-distribution genes that control whether melanoblasts migrate to the skin and how many melanocytes are contained in the skin.<sup>4,5</sup> In mammals, several loci are responsible for white markings or a completely white coat. Dominant white (*W*) locus in cats, piebald or white spotting (*S*) locus and merle (*M*) locus in dogs, and Waardenburg syndrome in humans all are associated with CSD.<sup>5-7,9,18-20</sup> In all, deafness is caused by degeneration of the stria vascularis of the cochlea in the inner ear.<sup>4,16,21</sup> Intermediate cells of the stria vascularis are actually melanocytes that are derived from the neural crest.<sup>3,13</sup> In several mammals with primary neural crest defects such as white spotting, deafness is associated with the absence of melanocytes in the stria vascularis.<sup>3,4,16</sup> Although the exact pathophysiology is unclear, melanocytes, but not melanin, appear to be vital for normal stria vascularis development and function.<sup>4,16,21</sup> On the other hand, albino animals are not usually afflicted with CSD because they have a normal distribution of melanocytes in the skin and inner ear.<sup>4</sup> Furthermore, it is interesting that the albino phenotype conceals other coat colors and patterns carried (the albino genotype is epistatic over all other coat color mutations). In this study, in which 1 of 10 albino ferrets was deaf, a possible explanation for the deafness in the albino ferret was that a genotypic white marking trait was otherwise concealed by the albino phenotype. Acquired deafness (drug ototoxicosis or otitis media) was also possible, but considering the history and young age of this ferret, it seemed less probable. Except for the study's exclusion criteria, no attempt was made to distinguish between hereditary and acquired origins when deafness was diagnosed, but the percentage of ferrets with acquired deafness was assumed to be small and probably the same for each coat color pattern.

In this study, white pattern-associated deafness was identified in ferrets. Except for albinism, the genetics of coat color in ferrets is unknown. In dogs with white-spotted or piebald phenotype, the *S* locus has been mapped on chromosome 20 and complex polymorphisms were identified in the vicinity of the *MITF* gene.<sup>22</sup> Additionally, mutations in the *EDN3* (endothelin 3), *EDNRB* (endothelin receptor type B), *PAX3* (paired box 3), *MITF*, and *SOX10* (SRY-box 10) genes that are involved in cochlear melanocyte development have been associated with white marking phenotypes and deafness in various species, including dogs, horses, rats, and mice.<sup>23,24</sup> In humans, mutations in the same genes and in *SNAI2* (snail homolog 2) cause several types of Waardenburg syndrome, resulting in deafness, blue irises, and a stripe of white in the hair and beard with premature graying and minor structural facial deformities.<sup>5,23,25</sup> Waardenburg syndrome is a consequence of abnormal proliferation, survival, migration, or differentiation of neural crest-derived melanocytes.<sup>25</sup> This syndrome in humans shares similarities with deafness in ferrets with a white stripe on the head and premature graying; however, abnormalities like facial deformities and other defects of tissues derived from the neural crest known to happen with disorders of melanocyte development have not been described in ferrets. Aside

from deafness, other health-related problems such as short life expectancy or reproductive disorders could be associated with white markings, according to some owners and breeders; there are no scientific data to prove such correlation, and a long-term study would be necessary.

Premature graying was strongly associated with CSD (prevalence, 87%) in this study. This trait has been described in other mammals. The graying trait in horses associated with non-UV-induced melanoma and premature graying in mice are related to the expression of the *MITF* gene,<sup>26,27</sup> which is crucial for melanocyte development and survival.<sup>17</sup> A series of recent observations has provided alternative evidence that hair graying is caused by loss of melanocyte stem cells rather than cytotoxic damage of mature melanocytes in the hair matrix.<sup>28</sup> In ferrets, the American panda coat is consistently associated with premature graying. Unfortunately, this trait may not become evident before 2 to 3 years of age, during which time the ferret becomes completely white. Without prior knowledge of the color at birth, it may be impossible to categorize prematurely gray ferrets as having DEW, panda, American panda, or other white-marked coats. Moreover, it is also impossible to know whether a juvenile white-marked ferret will be affected by premature graying. This is the reason why this trait was only known for 106 of the 152 ferrets. Some ferrets with premature graying were tested as deaf as young as 4 months old, before the graying process began and when they were still pigmented. Consequently, deafness in these ferrets did not appear later in life with graying.

Another interesting finding was that all ferrets with large amounts of white on the head were deaf; some ferrets with just small spots on the head (such as mitt ferrets with other white markings) had hearing or were deaf. This observation has already been made in deaf American Paint Horse<sup>29</sup> and deaf Border Collies with a greater amount of excess white on the head than those with hearing.<sup>7</sup> On the other hand, some ferrets with no white markings on the head but some on the body (mitt ferrets) or with white hair disseminated in the coat (silver) were deaf.

Dark-eyed white ferrets were not systematically deaf. The popular theory that the DEW color is a single large white spot covering the entire body of the ferret is probably wrong. Deafness prevalence associated with DEW in this study (2/5) must be interpreted with caution because the number of ferrets tested was low.

The behavior of deaf dogs and cats and how to manage them has been the subject of numerous publications.<sup>2</sup> As in dogs, unilateral deafness does not seem to pose problems except difficulty in localizing the sound.<sup>2,10</sup> Bilateral deafness in dogs and cats is often associated with behavioral problems such as anxiety, aggressiveness, and training difficulty.<sup>10,20</sup> Although most breeders and owners did not recognize deafness in their ferrets, it was occasionally associated with behavioral and training problems. Owners reported abnormal social interactions with other ferrets, biting tendencies, and louder than normal vocalizations.<sup>3</sup>

Because CSD often goes unrecognized by owners and breeders, this defect is probably underdiagnosed

despite its high prevalence. Moreover, other neural crest defects that could be associated with CSD are not yet identified in ferrets. In an effort to promote breeding of healthy ferrets and to reduce deafness, breeders should be educated about CSD and its association with coat color pattern. To reduce the prevalence of deafness in this species, panda, blaze, American panda, and other deaf ferrets should not be bred, especially not together. Breeding only bloodlines without a white pattern would seem to guarantee the absence of CSD; however, white patterns are popular with ferret breeders and pet owners alike. One possible strategy to minimize deafness while maintaining desirable white coat markings would be to breed hearing mitt ferrets. Simple guidelines such as breeding only bilaterally hearing BAER-tested ferrets, avoiding repeated matings that result in high rates of deaf ferrets, and avoiding bloodlines with high rates of deafness are recommended. Unfortunately, some breeders do not view CSD as unacceptable. Changing this perspective should be a goal of breeders and ferret owners and would benefit the species overall.

Congenital sensorineural deafness seems to be common in pet ferrets. It appears to be associated with white markings and premature graying; panda, American panda, and blaze ferrets are highly predisposed. Further investigations about the inheritance pattern, the underlying genes, and other phenotypic marker associations (eg, with eye color) are needed in the future. A greater awareness of this congenital defect is necessary to protect the species. Breeders and owners of ferrets at risk must be informed, and BAER testing should be routinely used to permit owners to identify CSD in their ferrets and to allow breeders to select unaffected breeding stock to reduce the overall prevalence of this condition.

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## From this month's AJVR

### Effects of tylosin administration on C-reactive protein concentration and carriage of *Salmonella enterica* in pigs

Hyeun Bum Kim et al

**Objective**—To evaluate the effects of tylosin on C-reactive protein concentration, carriage of *Salmonella enterica*, and antimicrobial resistance genes in commercial pigs.

**Animals**—120 pigs on 2 commercial farms.

**Procedures**—A cohort of sixty 10-week-old pigs in 4 pens/farm (15 pigs/pen) was randomly selected. Equal numbers of pigs were given feed containing tylosin (40 µg/g of feed) for 0, 6, or 12 weeks. C-reactive protein concentrations were measured, microbial culture for *S enterica* in feces was performed, and antimicrobial resistance genes in feces were quantified.

**Results**—No significant associations were detected between C-reactive protein concentration or *S enterica* status and tylosin treatment. During the 12 weeks of tylosin administration, increased levels of 6 antimicrobial resistance genes did not occur.

**Conclusions and Clinical Relevance**—Treatment of pigs with tylosin did not affect C-reactive protein concentration or reduce carriage or load of *S enterica*. There was no evidence that pigs receiving tylosin had increased carriage of the 6 antimicrobial resistance genes measured.

**Impact for Human Medicine**—*S enterica* is a public health concern. Use of the antimicrobial growth promoter tylosin did not pose a public health risk by means of increased carriage of *S enterica*. (*Am J Vet Res* 2014;75:460–467)



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