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CONGENITAL NEUROPATHY AND DILUTION OF FEATHER MELANIN IN NESTLINGS OF URBAN-BREEDING NORTHERN GOSHAWKS (*ACCIPITER GENTILIS*)

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Abstract: A pair of northern goshawks (*Accipiter gentilis*) breeding in a public park in the city center of Hamburg, Germany, raised 12 healthy and five aberrant offspring between 1996 and 2000. Aberrant nestlings (three males and two females) had pale silver-blue plumage and displayed severe locomotor disorders. Histopathologically, the defects were characterized by adendritic feather melanocytes and mild degeneration of the cerebellar white matter. Epidemiologic results were suggestive of a hereditary autosomal recessive defect, which may have originated from close inbreeding during the foundation of Hamburg's urban population of northern goshawks.

Key words: Northern goshawk, *Accipiter gentilis*, adendritic melanocytes, congenital neuropathy, hereditary disease, inbreeding depression.

INTRODUCTION

The simultaneous occurrence of hereditary disorders of pigmentation and the central nervous system (CNS) or the inner ear has been reported in mammals and birds.[6,22,31] A single developmental disturbance can result in pathologic alterations of both structures because melanocytes and nerve cells have a common developmental origin in the neural crest.[17,27] Congenital syndromes characterized by concurrent manifestation of melanization and CNS defects are particularly well-studied in poultry.[1,11,12,25,29,31]

This report documents a case of an urban-breeding pair of northern goshawks (*Accipiter gentilis*) that produced five aberrant offspring with diluted plumage coloration and severe locomotory disorders, alongside 12 healthy nestlings during a 5-yr observation period. Aberrant pale pigmentation has been described previously in raptors but not in association with a CNS disorder.[7,8,13]

CASE REPORT

Epidemiology

Hamburg (53°34′N, 9°59′E), Germany, is one of the few cities in the world with an urban population of northern goshawks. This population is the subject of an ongoing monitoring program that started in 1996.[18] One pair raised 12 phenotypically normal and five aberrant nestlings in five successive breeding seasons from 1996 to 2000. Nestlings of both sexes were affected (Table 1). In 1995, an immature female and a 2-yr-old male established their breeding territory in a highly frequented public park in the city center. All abnormal nestlings fell out of the nest toward the end of the nestling period (aged 20–29 days) and were subsequently found by members of the public. The affected juveniles were held in captivity for examination.

Clinical findings

All five affected nestlings displayed pale silver-blue feather coloration and severely disordered motor control. The entire plumage of the affected birds was pale silver-gray to silver-blue instead of red-brown as is normal for juveniles of this species (Fig. 1).[8,19] Feather growth, shape, and length were normal, but the feathers were coarse and dull, lacking the normal luster. Primary and secondary feathers and rectrices of all nestlings were complete. The flight feathers of some birds had fault bars, but the number of bars per primary feather was comparable with those observed in other healthy individuals in the population.

Affected nestlings spent the majority of their time resting on their tibiotarsal joints in a manner typical of juvenile raptors, but their entire body swayed continuously. Atactic movements became more pronounced with time. Older individuals attempting to stand would stagger, step on their primaries, and fall over, often rolling forward or backward until exhausted (Fig. 2). Birds would then lie trembling on their backs or sides. The male nestlings in 1997 and

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Table 1. Reproductive output of an urban-breeding pair of northern goshawks (*Accipiter gentilis*) in the city of Hamburg, Germany, during 1996–2000. The pair raised aberrant nestlings alongside healthy offspring in four different years. No mate replacement occurred during the study period.

			At time of banding			Age at fall from nest	
Year	Sex	Defect	Age (days)[a]	Weight (g)	Note	(days)[a]	Comments
1996	♀	+	(23)	—	vocalizing[b]	24	put back into nest after first fall; found dead on forest floor the next day
	♂	−	(23)	—	—	—	—
1997	♂	+	21	620	vocalizing[b]	28	euthanatized (about 33 days old)
	♂	−	23	670	vocalizing[b]	—	radiotracked in 1997;[c] found with broken wing on 15 Dec 1997; radiographed
	♀	−	24	960	vocalizing[b]	—	radiotracked in 1997–1998;[c] contact lost end of Mar 1998
1998	♂	+	22	590	defect visible[d]	29	euthanatized (aged 31 days); radiographed
	♀	+	22	650	defect visible[d]	24	movements recorded on videotape; euthanatized (aged 25 days)
	♀	−	25	940	—	—	bird raised clinically normal offspring in 1999–2001; radiotracked in 2001
	♀	−	25	970	—	—	—
1999	♂	−	25	680	—	—	recovered on 30 Oct 1999; exhausted but healthy; released on 04 Nov 1999
	♂	−	26	700	—	—	—
	♀	−	25	860	—	—	—
2000	♂	+	18	520	defect visible[d]; vocalizing[b]	20	dissected (aged 33 days); histologic examination of feathers and brain (see text)
	♂	−	18	500	—	—	—
	♂	−	16	480	—	—	—
	♀	−	18	740	—	—	—
	♀	−	15	560	vocalizing[b]	—	—

[a] Ages were estimated, using wing lengths of nestlings taken at the time of banding (values in brackets are not based on morphometric measurements).

[b] Birds showed aggressive behavior and vocalized during the banding procedure.

[c] For details, see Rutz.[20]

[d] Some nestlings showed first signs of locomotory disorders at the time of banding (i.e., before falling out of the nest).

2000 were unable to stand upright even at 33 days of age. Some nestlings showed the first signs of locomotory disorders at the time of banding, i.e., before falling out of the nest and hitting the forest floor (Table 1). Three affected birds vocalized during the banding procedure and appeared highly distressed. Similar behavior has not been observed in about 230 nestlings of the study population during 5 yr of systematic banding.

Total body weights of the affected birds (Table 1) were within reference ranges.[2,8] No evidence of trauma or juvenile metabolic bone disease was present. Other than their plumage, the birds exhibited normal morphology and development for their age. They did not appear to be visually impaired, and there were no macroscopic abnormalities in the pigmentation of their irises. In clinical and radiographic examinations of the 2000 male nestling, no additional aberrations were found. Because of severe neurologic dysfunction, all five nestlings were euthanatized after examination (pentobarbital, Narcoren®, Merial GmbH, 85399 Hallbergmoos, Germany; 400 mg/kg, i.v.).

Histopathology

The affected male nestling hatched in 2000 was necropsied, and tissues were examined histologically.

Figure 1. Dorsal view of a male northern goshawk nestling (approximately 30 days old) with diluted plumage coloration and central nervous clinical signs. The bird hatched in 2000 in an urban public park in the city of Hamburg, Germany (photograph: P. Grell).

Feathers: Growing primary feathers were cut into pieces of 10-mm length and immersed in a formaldehyde–glutaraldehyde fixative solution containing trinitro compounds. Fixed specimens were dehydrated through a graded ethanol series, embedded in methacrylate, and sectioned (10 μm) before staining with toluidine blue (all chemicals: Merck [Switzerland] AG, 8953 Dietikon, Switzerland). The feathers had altered melanocyte morphology. The melanocytes had an adendritic, round to ovoid shape and showed dense melanin deposition, resulting in a dark brown color. Melanocytes were located within the barb ridges as well as in the columns of barbule cells (Fig. 3). Similar cells occurred within the developing rachis but could not be found within the feather pulp or the non–feather-formative regions of the developing feather. The density of the melanocytes decreased distally. Delicately granulated melanocytes were observed in these loci and were probably a consequence of a diminution in the number of melanosomes. The cortical portions of the radii and rami contained numerous delicately dispersed melanosomes. The lighter areas of the feathers were characterized by sporadic adendritic melanocytes of yellow-brown pigmentation.

Brain: Examination of the skull revealed a conspicuous adhesion of calvaria, meninges, and brain within the area of the vertex. The brain was fixed for examination in 4% formalin, and coronary sections at different levels were cut and embedded in paraffin wax. Tissue sections were stained with hematoxylin and eosin, as well as Luxol fast blue, a specific stain for myelin. The histologic architecture of the brain appeared normally developed. Moderate vacuolation of the white matter was present bilaterally in the tectocerebellar tracts and more mildly in the cerebral medulla. No evidence of neuronal damage or inflammatory lesions was present in any of the examined sections.

Follow-up epidemiology

The origin of the breeding pair and their degree of relatedness were unknown. In *Accipiter* species, the basic characters of primary feathers (length, shape, coloration and patterning) remain fairly con-

Figure 2. Male northern goshawk nestling (approximately 30 days old) displaying a severe locomotory disorder and aberrant plumage coloration (full sibling of the individual shown in Fig. 1). When attempting to stand up, the bird stepped on the flight feathers of its left wing, rolled forward, and clenched its talons into the grass. The bird hatched in 1998 in an urban public park in the city of Hamburg, Germany (photograph: C. Saar).

sistent throughout life. This allows individual identification of birds by comparing molted feathers of equivalent position, which were found in subsequent years at the same nest site.[2] Using this simple but reliable field method, the identity of the adult goshawks could be established for the whole observation period. According to these investigations, no mate replacement occurred in 1996–2000. In 1997, both adult goshawks were trapped to check for signs of phenotypic aberrations similar to those found in their offspring. Both hawks were in a healthy condition and had normal adult plumage coloration. The male was equipped with a tail-mounted radiotransmitter (TW-3, Biotrack Ltd., Dorset BH20 5AX, U.K.) and systematically monitored for 5 mo during the breeding season in 1997.[18] It exhibited normal behavior during 370 hr of continuous tracking.

In 1997, prey choice of this breeding pair was assessed by recording kills of the radiotagged male during monitoring sessions ($n = 53$ prey items) and by scanning the nesting territory for prey remains ($n = 85$). The principal prey species were feral pigeon (*Columba livia* f. dom.), magpie (*Pica pica*), and blackbird (*Turdus merula*), which comprised 40%, 22%, and 10% of the diet, respectively (detailed description in Rutz[21]). Comparison of this sample with prey lists compiled for 23 different northern goshawk pairs during five successive breeding seasons (1996–2000) showed that this diet composition was typical for urban-breeding northern goshawks in the city of Hamburg (Rutz, unpubl. data).

The male did not appear to visit foraging sites with access to potentially contaminated prey animals (e.g., rats on rubbish dumps, see Tornberg and Colpaert[28]). Heavy-metal contamination of a primary feather molted from the breeding female was analyzed with the MeV-Hamburg-Proton-Microprobe.[15] Concentrations of all examined elements (Ca, Ti, Cr, Mn, Fe, Ni, Cu, Zn, Se, Br, Sr, Hg, and Pb) were not elevated in comparison with those found in control samples of nine other adult breeding females of the same population (Niecke and Rutz, unpubl. data).

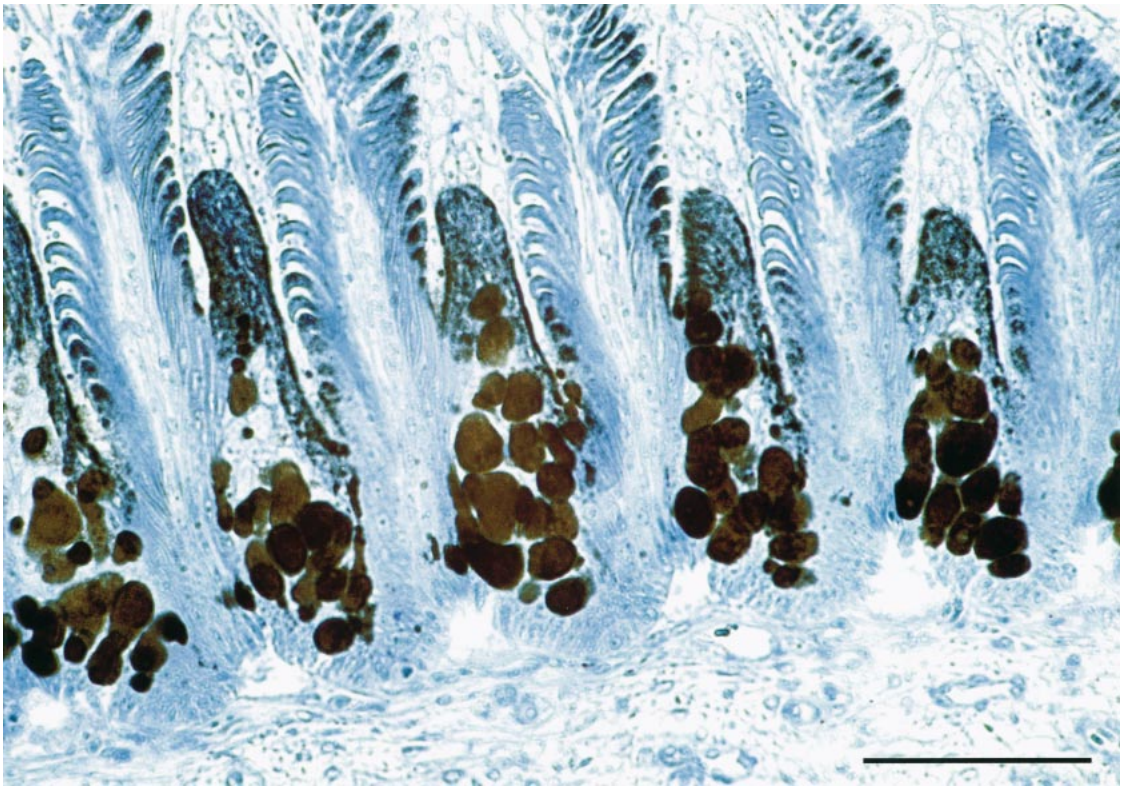

Figure 3. Photomicrograph of a transverse section of a growing primary feather from a northern goshawk nestling with aberrant plumage coloration (same individual as in Fig. 1). Note the altered melanocytes with adendritic shape and dense melanin deposition (toluidine blue). Bar = 100 μm.

Healthy nestlings from all 5 yr developed normally and left the nest at about 40 days of age. Further observations during regular control visits to the nest site revealed normal postfledging behavior.[8,18,20] In 1997, the two unaffected young (one male and one female) were trapped in the late postfledging period, fitted with radiotransmitters, and subsequently tracked on their natal dispersal.[20] Neither bird showed abnormal behavior. One of the unaffected females of the 1998 brood replaced an adult female at an established breeding territory 10.5 km away from the natal nest site. Together with its mate, it raised three nestlings in 1999, four in 2000, and at least two in 2001. No abnormalities were noted in any of these offspring. In 2001, this adult female was radiomonitored during the breeding season as part of a quantitative radiotracking study (Rutz, unpubl. data), and no abnormal behavior was observed.

DISCUSSION

An urban-breeding pair of northern goshawks raised offspring with similar diluted plumage coloration and central nervous signs in four of five observed breeding seasons. Affected nestlings did not appear to be malnourished or suffering from metabolic bone disease. Histologic examination of the brain of one affected nestling indicated a degenerative or dysplastic neuropathy or myelinopathy. The histologic lesions were not of a particular etiology, but a toxic or hereditary disorder cannot be excluded.[6,22,31]

Ingestion of pollutants as a cause of these abnormalities is unlikely because the pair showed normal prey choice, feather heavy-metal levels of the breeding female were within the range of values from control birds, and the parents and clinically normal nestlings neither exhibited any permanent or transient signs of the disease nor developed any delayed clinical signs. Epidemiologic and pathomorphologic results of the presented case suggest an autosomal recessive hereditary defect.

Melanocytes and nerve cells have a common ontogenetic origin in the neural crest.[17] This explains why disorders in pigmentation are often associated with pathologic conditions of the CNS, as

exemplified by the case reported here.[1,31] Similar syndromes have been documented in various other species. In the domestic house mouse (*Mus musculus* f. dom.), hereditary color abnormalities including many spotting and coloration genes are often associated with defects of the CNS.[23] Several congenital syndromes have been reported in poultry. The ''faded shaker'' condition in the domestic chicken (*Gallus gallus* f. dom.) is an autosomal recessive hereditary defect that results in a congenital tremor and dilution of down- and flight-feather melanin.[25] Autosomal recessively inherited cerebellar functional disorder associated with an abnormal dark and frayed plumage (''dark feather nervous disorder'') has been observed in Japanese quails (*Coturnix japonica* f. dom.).[11,29] The ''faded bronze plumage'' syndrome in the domestic turkey (*Meleagris gallopavo* f. dom.) is characterized by color dilution and neuropathy. Breeding tests suggested an autosomal recessive mode of inheritance.[12]

Feather coloration is determined by pigments and structural features. Melanins are pigments responsible for various shades of black, gray, brown, and tan. Melanocytes are derived from the neural crest, migrate to the dermis, and form dendritic processes that transport melanosomes into the growing feather.[26] Pigment dilution can be caused by several genetically distinct alterations of granule synthesis or dispersal.[4] In goshawks we studied, the development of adendritic melanocytes produced diluted plumage coloration. The loss of dendrites results in inefficient transport of melanosomes.[14] Similar melanocyte alterations unassociated with CNS disorders have been reported for domestic chicken with a plumage color mutation called ''lavender [*lav*]'' or ''true breeding blue''.[4] Diluted pigmentation can also be due to melanocytes that are morphologically normal but show a partial deficiency and an incomplete pigmentation of melanosomes. Examples of this condition include the ''faded shaker'' syndrome, ''sex-linked imperfect albinism,'' and thyroxin deficiency in domestic chickens.[9,24,25] A wide variety of plumage disorders are documented in wild bird species.[10]

Neurologic changes were characterized by vacuolation restricted to the white matter of the cerebrum and the brain stem. These alterations were nonspecific, but they may have been responsible for the observed neurologic signs. In contrast to the ''congenital quiver'' or ''faded shaker'' syndrome of poultry, neither neuronal nor cerebellar myelin loss were found in the present case.[6,25]

Adult northern goshawks with color dilution but no neurologic disorder had been trapped in the 1950s in the Lausitz area, Germany.[8] Two cases of suspected congenital anomalies in northern goshawk nestlings have been documented in a Dutch population.[3,16] Both broods contained a single aberrant nestling as well as healthy offspring. Affected birds had missing or malformed main feathers but presented neither of the characteristic aberrations observed in the birds of this study (note that it has recently been suggested that the symptoms presented by the Dutch nestlings were caused by an infection with avian polyoma virus; Vedder[30]).

Developmental disorders similar to those described here have not been reported previously in birds of prey. Sick birds in rural and wild habitats are rarely discovered. If recovery rates in nonurban areas, where the majority of the raptor-monitoring projects are carried out, are indeed lower than those in urban environments, comparable cases of aberrant nestlings may have passed unnoticed. The nesting tree of the Hamburg pair was situated only 5 m from a well-traveled pathway, and nestlings were discovered soon after falling from the nest, before predation or scavenging occurred.

The urban population of northern goshawks in Hamburg was recently established. Breeding pairs first appeared in woods at the periphery of the city in the mid-1980s (Wirth and Risch, pers. comm.). After a phase of continuous expansion, the population reached carrying capacity in the mid-1990s with about 17 breeding pairs in a 320-km^2 study area (Rutz, unpubl. data). Band recovery data from 1996 to 2000 showed that a considerable proportion of urban-fledged immature hawks disperse only short distances and attempt to find a free nest site within the city (Rutz, unpubl. data). Negligible resource competition in the city and restricted gene flow between urban and rural habitats could have resulted in increased homozygosity during the early stages of the colonization process. Homozygosity in a high proportion of genes facilitates the accumulation of deleterious recessive mutations, which can lead to a decline in fecundity coupled with increased nestling or fledgling mortality, a phenomenon known as inbreeding depression.[5] In the concerned pair, mean brood size of 3.4 juveniles/brood was well above the average for this species.[8] However, reproductive output of the pair was reduced by about 30% because of the nestling mortality. In poultry, partial or complete loss of plumage coloration similar to the described aberrations is a well-known effect of increased homozygosity.[6] The observed defects may have originated from an increase in homozygosity due to close inbreeding or from coincidental spontaneous mutations.

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