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Antibodies to Adenoviruses in Free-Living Common Buzzards from Germany

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ABSTRACT: One hundred sixty-seven plasma samples of free-living birds of prey from Berlin and Brandenburg State (Germany) were tested for antibodies against avian adenovirus (FAV, group I) using agar gel precipitation test. Antibodies to FAV were detected in seven (4%) of 167 total samples. The positive samples originated only from common buzzards (*Buteo buteo*; seven [12%] of 59). This serologic survey provides evidence of natural exposure of free-living common buzzards from eastern Germany to adenoviruses.

Key words: Avian adenoviruses, *Buteo buteo*, common buzzards, eastern Germany, free-living birds of prey, serologic survey.

Adenoviruses are nonenveloped and 70–100 nm in diameter (MacPherson et al., 1961; Horne, 1962). The *Adenoviridae* family consists of two genera: *Mastadenovirus* that contains mammalian strains and *Aviadenovirus*. The two genera have a distinct group antigen (Sharpless, 1962). Avian adenoviruses are currently divided into three groups (I–III) based on shared characteristics. In contrast to group II (hemorrhagic enteritis) and group III (egg drop syndrome) the role of group I (fowl adenoviruses, FAV) in disease is not well defined (Russell et al., 1995; McFerran, 1997). Fowl adenoviruses are common in healthy birds and marked differences in pathogenicity among isolates of same serotypes have been demonstrated (Cook, 1972; Bülow et al., 1986). In general avian adenoviruses are more virulent in non-host adapted species than in their typical host. In several outbreaks of adenovirus infections involving psittacine birds, non-psittacine species in the same facility were unaffected (Bryant and Montali, 1987; Gerlach, 1994). Some adenoviruses recovered from pigeons, ducks, and budgerigars (*Melopsittacus undulatus*) were serologi-

cally similar to those found in gallinaceous birds (McFerran et al., 1976; McFerran and Adair, 1977; Takase et al., 1990). Only limited information exists about the occurrence of adenoviruses in raptors. Adenovirus was recovered from the brain of a free-living goshawk (*Accipiter gentilis*) with neurologic signs (Gerlach, 1994). Adenovirus-like lesions were reported in a group of nine captive juvenile and adult American kestrels (*Falco sparverius*) with hemorrhagic enteritis (Sileo et al., 1983). Additionally, adenovirus was described in association with hemorrhagic enteritis in a free-living tawny frogmouth (*Podargus strigoides*) in Australia (Reece and Pass, 1985), in a merlin (*Falco columbarius*) from the USA with hepatitis (Schelling et al., 1989), and in captive Mauritius kestrels (*Falco punctatus*) from the UK showing various clinical signs such as hemorrhagic diarrhea, hepatitis, and acute death (Forbes et al., 1997). Our objective in the present study was to determine the antibody prevalence against FAV in free-living birds of prey in parts of eastern Germany.

In collaboration with various raptor rehabilitation centers (Fig. 1), 167 plasma samples were collected from free-living raptors between 1994 and 1997 (Table 1) and tested for antibodies against FAV (group I) using an agar gel precipitation test (AGP). The samples originated from Berlin (52°30'N, 13°20'E) and Brandenburg State (51°35'–53°45'N, 11°60'–14°70'E) in eastern Germany. In general, plasma samples were either obtained from apparently healthy birds during ringing procedures ($n=40$) or from injured or sick birds during clinical examinations at the raptor center ($n=127$). During their re-

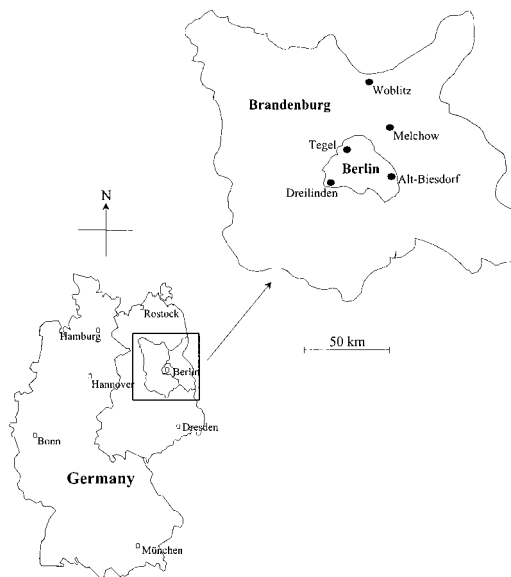


FIGURE 1. Distribution of the collaborating raptor rehabilitation centers in the Berlin and Brandenburg area in eastern Germany.

habilitation period all buzzards were fed mice and 1 day old chickens. Blood was usually taken within 48 hr after arrival at the raptor centers. Samples were stored at -20°C until examined.

The AGP test was performed according to Woernle and Brunner (1963). Briefly,

specific pathogen-free embryonated chicken eggs were inoculated by allantoic route with chicken embryo lethal orphan (CELO), Phelps strain of group I adenovirus. The chorioallantoic membranes were collected 72 hr postinoculation, homogenized, and used in the AGP with positive control serum.

Antibodies to FAV antigen were found in seven (4%) of 167 tested blood samples. These positive samples originated only from common buzzards (seven [12%] of 59; Table 1). Due to the small sample size of all other tested species the 95% confidence interval of the common buzzard's prevalence overlaps the corresponding interval for each of the other species (Table 1). Therefore, a difference between prevalences cannot be statistically confirmed.

All seven positive samples were obtained from sick or injured birds which were kept at the Wobnitz ($n=5$) and Melchow ($n=2$) raptor rehabilitation centers (Fig. 1). Of five birds with known history, four had several traumatic bone fractures and one had hepatitis and air sacculitis. However, most of the common buzzards sampled in this study were sick or injured

TABLE 1. Antibodies to avian adenovirus in common buzzards in Germany.

Species	Number of positive reactors/ number of samples tested	95% CI ^a
Common buzzard (<i>Buteo buteo</i>)	7/59	5–23
Eurasian kestrel (<i>Falco tinnunculus</i>)	0/27	0–13
Goshawk (<i>Accipiter gentilis</i>)	0/20	0–17
European hobby (<i>Falco subbuteo</i>)	0/9	0–34
Tawny owl (<i>Strix aluco</i>)	0/19	0–18
Osprey (<i>Pandion haliaetus</i>)	0/2	0–84
Long-eared owl (<i>Asio otus</i>)	0/6	0–46
Red kite (<i>Milvus milvus</i>)	0/4	0–60
Barn owl (<i>Tyto alba</i>)	0/2	0–84
Black kite (<i>Milvus migrans</i>)	0/1	0–97
White-tailed sea eagle (<i>Haliaeetus albicilla</i>)	0/6	0–46
Northern rough legged buzzard (<i>Buteo lagopus</i>)	0/1	0–97
Peregrine falcon (<i>Falco peregrinus</i>)	0/4	0–60
Honey buzzard (<i>Pernis apivorus</i>)	0/4	0–60
Marsh harrier (<i>Circus aeruginosus</i>)	0/3	0–71
Total	7/167	

^a CI = confidence interval.

birds ($n=53$). Only six samples were collected during ringing procedures.

Several investigations demonstrated that free-living birds can be infected with avian adenovirus (Gerlach, 1994; Forbes et al., 1997). Most of these studies were conducted using serologic tests to detect avian adenovirus group antibody such as agar gel immunodiffusion (AGID), immunofluorescence (IFT), or enzyme-linked immunosorbent assay (ELISA) test, and therefore it was not possible to conclude that these birds were infected with avian serotypes (McFerran and Adair, 1977). Avian adenoviruses were detected in several raptors that died shortly after being found (Ritchie and Carter, 1995). Currently, no information is available on virus shedding and the persistence of antibodies to avian adenoviruses in raptors.

The AGP test used in this survey may not have high sensitivity. This insensitivity, however, may not be important in field situations (McCracken and Adair, 1993), where repeated infections with different adenovirus serotypes take place early in life and each successive infection stimulates the group specific antibody response. In the present investigation, a serologic test with higher sensitivity like ELISA could not be used due to lack of species-specific conjugates.

Based on several studies on the pathogenesis of avian adenovirus infections, the excretion of the virus in feces and to lesser extent in the naso-oral secretions may be a major factor in spread of infections (McFerran, 1997). Transmission primarily occurs by the fecal-oral route. In addition, virus has been isolated from fertile and non-fertile eggs (McFerran and Adair, 1977). Forbes et al. (1997) reported adenovirus infection in Mauritius kestrels fed on 1 day old chicks, quail, and turkey poults from which the same adenovirus was isolated. In the present survey, buzzards were fed mice and 1 day old chicks during their rehabilitation period. Adenovirus infection history of the chicks is not known, but infections are widespread in

domestic poultry (McFerran and Adair, 1977). However, in this study blood was usually collected within 48 hr after the birds were taken to the raptor center. Therefore, transmission of adenoviruses to these raptors from chicks is unlikely.

In conclusion, this serologic survey reveals evidence of natural exposure of free-living common buzzards to group I adenoviruses in Berlin and Brandenburg State in Germany. Adenoviruses are frequently demonstrated in apparently healthy birds and detection of antibodies may not indicate that the virus is causing disease (Ritchie and Carter, 1995). However, apparently apathogenic strains might induce disease if the host become immunocompromised.

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LITERATURE CITED

- BRYANT, W. M., AND R. J. MONTALI. 1987. An outbreak of a fatal inclusion body hepatitis in zoo psittacines. *Proceedings International Conference on Avian Medicine* 1: 473.
- BÜLOW, V. V., R. RUDOLPH, AND B. FUCHS. 1986. Folgen der Doppelinfektion von Küken mit Adenovirus oder Reovirus und dem Erreger der aviären infektiösen Anämie (CAA). *Journal of Veterinary Medicine (B)* 33: 717–726.
- COOK, J. K. A. 1972. Avian adenovirus alone or followed by infectious bronchitis virus in laying hens. *Journal of Comparative Pathology* 82: 119–128.
- FORBES, N. A., G. N. SIMPSON, R. J. HIGGINS, AND R. E. GOUGH. 1997. Adenovirus infection in Mauritius kestrels (*Falco punctatus*). *Journal of Avian Medicine and Surgery* 11: 31–33.
- GERLACH, H. 1994. Viruses. In *Avian medicine: Principles and application*, B. W. Ritchie, G. J. Harrison and L. R. Harrison (eds.). Wingers Publishing, Lake Worth, Florida, pp. 862–948.
- HORNE, R. W. 1962. The comparative structure of avian adenoviruses. *Annals of the New York Academy of Sciences* 101: 475–484.
- MCCRACKEN, R. M., AND B. M. ADAIR. 1993. Avian adenoviruses. In *Virus infections of birds*, J. B. McFerran and M. S. McNulty (eds.). Elsevier Science Publishers B.V., Amsterdam, Holland, pp. 123–144.
- McFERRAN, J. B. 1997. Group I adenovirus infec-

- tions. In Diseases of poultry, B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDonald and Y. M. Saif (eds.). 10th Edition, Iowa State University Press, Ames, Iowa, pp. 608–620.
- , AND B. M. ADAIR. 1977. Avian adenoviruses—A review. *Avian Pathology* 6: 189–217.
- , T. J. CONNER, AND R. M. MCCracken. 1976. Isolation of adenoviruses and reoviruses from avian species other than domestic fowl. *Avian Diseases* 20: 519–524.
- MACPHERSON, I. A., P. WILDY, M. G. P. STOKER, AND R. W. HORNE. 1961. The fine structure of GAL—an avian orphan virus. *Virology* 13: 146–149.
- REECE, R. L., AND D. A. PASS. 1985. Inclusion body hepatitis in a tawny frogmouth (*Podargus strigoides*: Caprimulgiformes). *Australian Veterinary Journal* 62: 426.
- RITCHIE, B. W., AND K. CARTER. 1995. Adenoviridae. In *Avian viruses: Function and control*, B. W. Ritchie and K. Carter (eds.). Wingers Publishing, Lake Worth, Florida, pp. 313–333.
- RUSSELL, W. C., T. ADRIAN, A. BARTHA, K. FUJINAGA, H. S. GINSBERG, J. C. HIERHOLZER, J. C. DEJONG, Q. G. LI, V. MAUTNER, I. NÁSZ, AND G. WADELL. 1995. Family *Adenoviridae*. In *Virus taxonomy. Classification and nomenclature of viruses*, Sixth report of the International Committee on taxonomy of viruses, T. A. Murphy, C. M. Fauquet, D. H. L. Bishop, S. A. Ghabrial, A. W. Jarvis, G. P. Martelli, M. A. Mayo and M. D. Summers (eds.). Springer Verlag, New York, New York, pp. 128–133.
- SCHELLING, S. H., D. S. GARLICK, AND J. ALROY. 1989. Adenoviral hepatitis in a merlin (*Falco columbarius*). *Veterinary Pathology* 26: 529–530.
- SHARPLESS, G. R. 1962. GAL virus. *Annals of the New York Academy of Sciences* 101: 515–519.
- SILEO, L., C. FRANSON, D. L. GRAHAM, C. H. DOMERMUTH, B. A. RATTNER, AND O. H. PATTEE. 1983. Hemorrhagic enteritis in captive American kestrels (*Falco sparverius*). *Journal of Wildlife Diseases* 19: 244–247.
- TAKASE, K., N. YOSHINAGA, T. EGASHIRA, T. UCHIMURA, AND M. YAMAMOTO. 1990. Avian adenovirus isolated from pigeons affected with inclusion body hepatitis. *Japanese Journal of Veterinary Science* 52: 207–215.
- WOERNLE, H., AND A. BRUNNER. 1963. Über das Vorkommen von CELO-Virus-Infektionen des Huhnes und ihre Diagnose mit Hilfe des Agar-Gel-Präzipitationstestes. *Monateshefte für Tierheilkunde* 15: 262–270.

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