Krone O, Langgemach T, Sömmer P, Kenntner N (2003) Causes of mortality in white-tailed sea eagles from Germany. In: Sea Eagle 2000. Proc. Swedish Soc. For Nat. Conserv./SNF, Helander B, Marquiss M, Bowerman W (eds), Stockholm, Sweden, 211-218.

CAUSES OF MORTALITY IN WHITE-TAILED SEA EAGLES FROM GERMANY

Oliver Krone[1], Torsten Langgemach[2], Paul Sömmer[3] and Norbert Kenntner[1]

[1] *Institute for Zoo and Wildlife Research, P.O. 601103, D-10252 Berlin, Germany*
[2] *Brandenburg State Environmental Agency, Bird Conservation Centre, D-14715 Buckow, Germany*
[3] *Brandenburg State Institution for large protected Areas, Nature Centre Woblitz, D-16798 Himmelpfort, Germany*

ABSTRACT

White-tailed Sea Eagles *Haliaeetus albicilla* found moribund or dead in the field were submitted for necropsy to the Institute for Zoo and Wildlife Research (IZW) and to the Institute for Food, Drugs and Animal Diseases (ILAT), Berlin, Germany. The moribund eagles had died in rehabilitation stations or were euthanized. Onehundred-twenty White-tailed Sea Eagles were examined between 1990 and 2000, comprising 47% females, 38% males, and 15% undetermined. Nearly half (47.5%) of the birds were adult, 12.5% were subadult, 34% immature, 3% nestlings and in 3% no age class could be determined. The main causes of death in White-tailed Sea Eagles were collisions with trains (14%), lead intoxication (12%), infectious diseases (11%), trauma (10%), electrocution (9%), collision with wires (7%), injuries sustained during intra-specific conflict (5%), poisoning (3%), malformation (2%), and starvation (1%). Because of their decomposed condition no diagnosis could be made in 29 cases. The White-tailed Sea Eagle was documented as a new host for four nematode and one trematode species. For the first time trematode *Metorchis* was shown to be pathogenic to White-tailed Sea Eagles resulting in the death of six birds. Gunshot pellets were found in five out of 58 birds radiographed.

INTRODUCTION

The persecution of White-tailed Sea Eagles by shooting, trapping and robbery of nestlings led to the extinction of populations in many parts of Europe until the beginning of the 19th century (Fischer 1984). Following legal ban in 1935 the population in Germany increased slowly until the middle of the 20th century (Oehme 1961, 1987a). The stagnation in the number of breeding pairs until the 1980s can be explained by the effects of the pesticide DDT (Oehme 1987b). The prohibition of the use of DDT in Germany in the early 1970s resulted ten years later in a new increase of White-tailed Sea Eagles in Germany that still continues. Anthropogenic factors such as collisions with trains and cars, collisions with power lines, electrocution and poisoning are at present the main causes of death (Hauff 1998, Struwe-Juhl & Latendorf 1997).

Sources of natural mortality are rarely reported. Predation of a nestling by an adult Sea Eagle was described by Uttendörfer (1952). Territorial fights with fatal consequences are also reported (Fischer 1984, Langgemach & Sömmer 1996, Oehme 1961, Struwe-Juhl & Latendorf 1997). The death of a 20 days old nestling was attributed to hypothermia and in another case to a storm which knocked over the nesting tree (Struwe-Juhl & Latendorf 1997). A case of cainism was documented by Görke & Görke (2001). Diseases are virtually disregarded as a source of natural mortality. Oehme (1961) speculated that parasites did not play any role. In a survey conducted later Oehme *et al.* (1993) found a fungal infection (*Aspergillosis*) to be responsible for the death in one of 22 sea eagles. Occasionally, malformation of feathers during development ofin nestlings are noticed (Oehme & Manowsky 1991). A high percentage of diseases in White-tailed

Sea Eagles found dead in the Federal state of Brandenburg, Germany is documented by Langgemach and Sömmer (1996). Lesions in the liver-bile-duct-system were diagnosed most frequently.

MATERIAL AND METHODS

The birds originated from the Federal states of Brandenburg, Mecklenburg Western Pomerania, Saxony-Anhalt and Schleswig-Holstein (Fig. 1) and were collected between 1990 and 2000. A total of 120 carcasses was collected by private persons, nature conservation authorities and the Sea Eagle Working Group Schleswig-Holstein and stored at –20° C until submitted for necropsy. Sixty carcasses were examined at the Institute for Zoo and Wildlife Research (IZW), Berlin including analysis of heavy metals and another 60 at the Institute for Food, Drugs and Animal Diseases (ILAT), Berlin without analysing heavy metals. All birds were measured, weighed, and x-rayed prior to dissection. The age categories adult (5th year and older), subadult (4th year), immature (older than one and younger than 4 years), juvenile (1st year), and nestling (until fledging) were determined using plumage and beak characteristics (Fischer 1984; Forsman 1999, Glutz *et al.*, 1971) and remains of the *Bursa fabricii* (only for immatures). Sexing was performed by gonad type (ovary or testis). The thickness of subcutaneous, body cavity and coronary fat tissue together with the shape of the breast muscle and the weight of the bird were used to evaluate body condition. Post mortem examination concentrated on trauma, organ alterations, diseases and parasites. Parasitological techniques are described previously (Krone 2000). The causes of death were classified into 11 categories by pathological findings along with background information provided by the finder of the carcass.

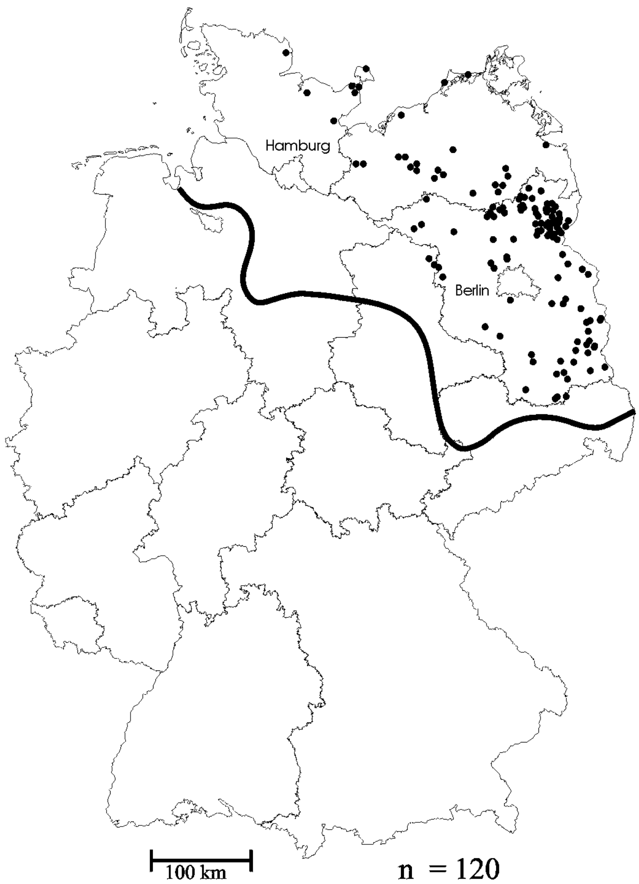

Figure 1. Locations of White-tailed Sea Eagles found dead in Germany (1990 – 2000). The bold line denotes the western edge of the breeding distribution in Germany.

RESULTS

Of all White-tailed Sea eagles (n = 120) slightly more females (47%) than males (38%) were found. Sex could not be determined in 15% of the carcasses due to decomposition. Nearly half (47%) of the 120 birds were adult, 8% subadult, 23% immature, 17% juvenile, 1% pulli and in 3% no exact age could be diagnosed. Body condition was determined in 26 females and 17 males indicating that most of the birds were in a good condition (Fig. 2).

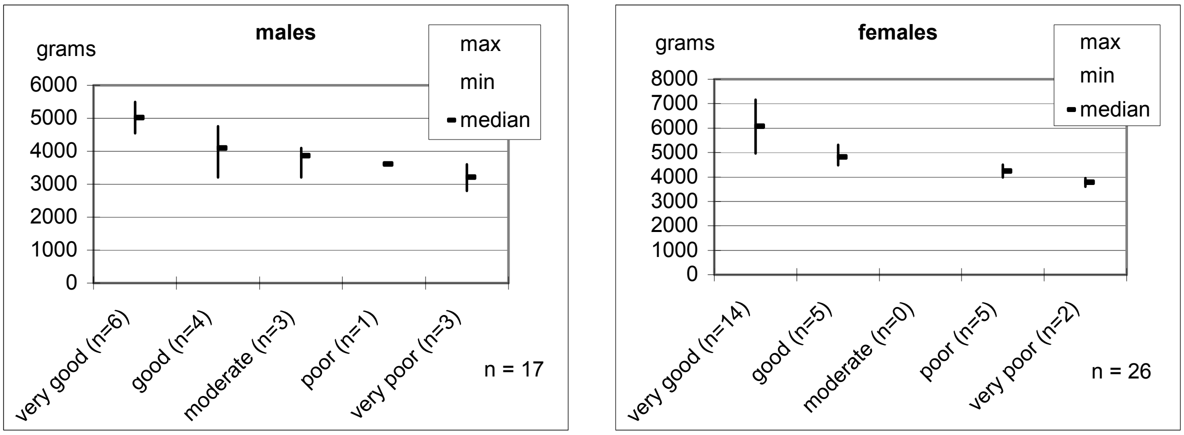

Figure 2. Weight in relation to body condition of female and male White-tailed Sea Eagles.

Among the “natural” causes of death infections were predominant. Six eagles died due to a severe liver fluke infection, four because of a bacterial infection, one due to pneumonia associated with *Klebsiella pneumoniae* and *Pseudomonas aeruginosa.* One eagle died due to a *Chlamydia psittaci* infection resulting in a fibrinous peritonitis, and one eagle that was kept in captivity for 20 days died due to a *Erysipelothrix rhusiopathiae* infection leading to a fibrinous pericarditis. Two birds were heavily infected with *Aspergillus sp*. in the air sacks and lungs, of which one had an additional liver fluke infection. A *Candida albicans* infection was diagnosed in the gall bladder of one eagle. Eagles that died due to “territorial fights” had stab wounds on their feet and died due to hypovolaemic or neurogenic shocks resulting from lacerations of internal organs. Four of these eagles were weakened by severe liver fluke infection and another by high lead level in the liver tissue. Two nestlings were found with the pinching-off syndrome, characterized by abnormalities in the development of the feather. The feathers showed the typical diminution in the *calamus* (Halliwell 1986), and a prolonged growing inside the feather sheath resulting in a very long blood filled rachis. The feather is poorly keratinised resulting in a soft appearance of the vane and shaft. Another nestling died due to starvation.

Among the “artificial” causes of death most birds died due to collisions with trains while feeding on railway kills. Tissue or bone destruction found to be the cause of death were subsummed under “trauma” with unknown origin. According to Wayland & Bollinger (1999) lead concentrations of more than 30 ppm dry weight (8.5 ppm wet weight) in hepatic tissue were judged as potential lethal in fifteen cases. Lethal mercury intoxication was suspected to be the cause of death in two birds with a level of more than 28 ppm (wet weight) in kidney tissue, while no other organ alterations or trauma were detected. Of three eagles that died due to poisoning with chlorinated carbohydrates, carbofuran was found in one bird whereas in the two other birds a metabolic product of parathion was detected. In 29 birds the cause of death could not be determined. Gun shot pellets found in five eagles (n = 58) were detected by radiograph.

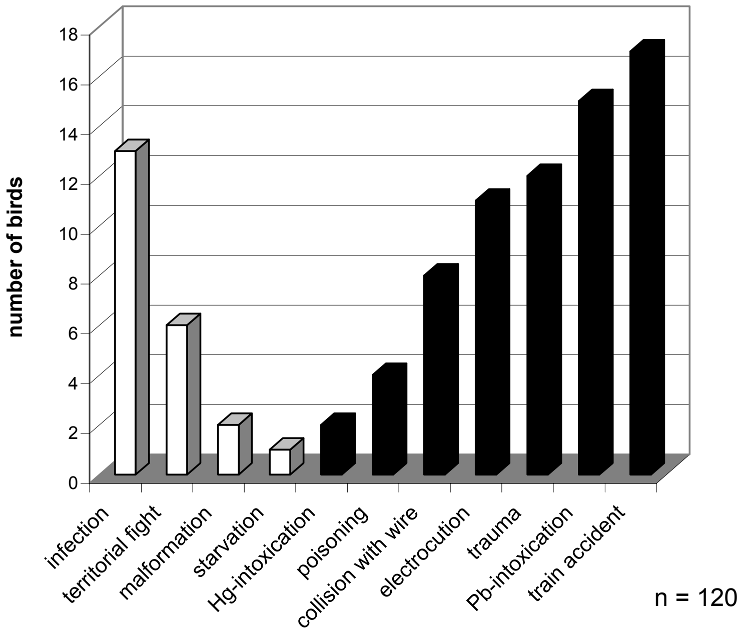

Figure 3. Causes of death in White-tailed Sea Eagles from Germany. The white bars indicate the "natural" and the black bars the "artificial" causes of death (Pb = lead, Hg = mercury).

Table 1. Helminths of the White-tailed Sea Eagle.

Nematoda	*Porrocaecum angusticolle*	Skrjabin (1992)
	P. depressum	Skrjabin (1992)
	Contracaecum milviensis	Rysavý & Ryzhikov (1978)
	Eucoleus dispar	this study
	Capillaraia tenuissima	this study
	Microtetrameres sp.	this study
	Hovorkonema variegatum	this study
Cestoda	*Cladotaenia globifera*	Furmaga (1957)
	Gryporhynchus pusillus	Ryzhikov & Rysavý (1985)
	Digramma interrupta	Ryzhikov & Rysavý (1985)
Trematoda	*Cotylurus platycephalus*	Dubois (1970)
	Neodiplostomum (Conodiplostomum) perlatum	Dubois (1970)
	Metorchis bilis	Zablokij (1962)
	N. (C.) spathula	Dubois (1970)
	N. (Neodiplostomum) spathoides	Dubois (1970)
	Metagonimus yokogawai	Bychovskaja-Pavlovskaja (1962)
	Paracoenogonimus ovatus	Sitko (1994)
	Pygidiopsis genata	Bychovskaja-Pavlovskaja (1962)
	Sobolevistoma graciosa	Zablokij (1962)
	Strigea falconispalumbi	Dubois (1970)
	S. strigis (side host)	Dubois (1970)
	Apophallus sp.	this study
Acanthocephala	*Sphaerirostris turdi*	Ryzhikov & Rysavý (1985)
	Polymorphus striatus	Ryzhikov & Rysavý (1985)

Four nematode and one trematode species were diagnosed as new parasites for the White-tailed Sea Eagle from 58 specimens examined in detail (Table 1). *Eucoleus dispar* lives in the mucosal layer of the oesophagus, *Capillaria tenuissima* in the small intestine and *Hovorkonema variegatum* in the air sacks or the trachea. Only females of *Microtetrameres sp*. were found embedded in the glands of the proventriculus, whereas no male parasites were found in the lumen of the proventriculus where they live. *Apophallus sp.* (trematodes) was diagnosed from the small intestine. *Coccidia* of the genera *Sarcocystis/Frenkelia* can not be separated from each other on the basis of oocysts found in the small intestine of the eagles.

DISCUSSION

Causes of death in White-tailed Sea Eagles have mainly been attributed to "anthropogenic" factors in the past, while diseases and territorial fights are reported only sporadically. As Langgemach and Sömmer (1996) previously pointed out earlier publications often miss diseases as a "natural" mortality factor. Compared with numbers of shot White-tailed Sea Eagles in the past Struwe-Juhl and Latendorf (1997) reported an increase of anthropogenic causes of death (collisions with trains, cars and wire, electrocution) in White-tailed Sea Eagles from Schleswig-Holstein, Germany, from 1980 onwards. The most common causes of deaths for Bald Eagles *Haliaeetus leucocephalus* from Florida, USA examined from 1963 to 1994 were trauma due to collision (59%), followed by electrocution (16%), poisoning (10%), infectious diseases (7%), and emaciation of unknown causes (5%) (Forrester & Thomas 1998). Mortality of 49 Bald Eagles found in the Yellowstone National Park, USA were determined as unknown (31%), electrocution or collision with power lines (20%), known or suspected poisoning (16%), gunshot wounds (14%), collision with vehicle (8%), "conspecific encounter" (6%), and captured in leg-hold traps (4%) (Harmata *et al.* 1999).

The present study documents infections as an important source of mortality in White-tailed Sea Eagles. The liver fluke *Metorchis sp.* is diagnosed as a pathogenic parasite that can cause death in Sea Eagles, four of six birds killed in fights by competitors had severe liver fluke infections that might have weakened the loser. Infections caused by bacteria and fungi are probably underrepresented because of the decomposed condition of many carcasses and storage at –20°C prior to necropsy, which prevents the detection of kryo-labile bacteria. It still remains unclear if the eagle that died due to the *E. rhusiopathiae* acquired the infection in the wild or in captivity. The source of *E. rhusiopathiae* may have been fish as in the case of an infected Bald Eagle (Franson *et al.* 1994).

Among eagles that died due to human influences ("artificial" causes of death), most were killed by trains. These birds used the railway kills of game animals for food and while feeding on the carrion they were themselves hit by the train. A large number of birds still die due to electrocution. When perching on a traverse between standing isolators the eagle bridges two power lines or one power line and the ground and gets electrocuted (Fig. 4).

Lead intoxication is the most important cause of death in White-tailed Sea Eagles from Germany (Kenntner *et al.* 2001). The main sources of lead in White-tailed sea eagles are probably waterfowl and game animals shot with lead ammunition or their remains (organs left in the field after disembowelling). The eagles ingest the lead shot or bullet fragments embedded in the carcasses of shot animals or those captured as handicapped prey. Trauma of unknown origin could stem from accidents with traffic and trains or collisions with power lines and aircraft. Three birds presumably died due to poisoning with pesticides as non target species. Also Tataruch *et al.* (1998) diagnosed death due to carbofuran poisoning of two wintering White-tailed Sea Eagles from Austria. The gunshot pellets found in 5 eagles indicate that illegal persecution still occurs.

Figure 4: Typical signs of electrocution: a) curled barbules. b) skin lesion.

The list of helminths of the White-tailed Sea Eagle now contains 24 species. The only protozoan found belongs to the coccidia *Sarcocystis/Frenkelia*. The most pathogenic parasite is the trematode *Metorchis* which lives in the gall bladder and bile ducts, where complete obstruction can lead to liver failure. From the North American Bald Eagle *Haliaeetus leucocephalus* 21 helminth (Kocan & Locke 1974, Nickol & Kocan 1982, Smith, 1978, Tuggle & Schmeling 1982) and two protozoan species (Tuggle & Schmeling 1982, Rettig 1978) have been reported. Only in cases of the *Trichomonas gallinae* (Stone & Nye 1979, Rettig 1978) and the *Cryptocotyle lingua* (Smith 1978) infections, has this been shown to be fatal for Bald Eagles.

The causes of diseases and death of the White-tailed Sea Eagles documented in this study probably do not represent the mortality factors of the population, due to a lack of a randomly chosen sample. Carcasses found near human settlements are possibly over represented, because these are more likely to be found than birds that die in remote areas which are less frequently visited by humans. However, the research on mortality factors enhances the knowledge of the biological understanding of the White-tailed Sea Eagle and points out important risk factors for this still threatened species in Europe.

CONCLUSIONS

To reduce the mortality of White-tailed Sea Eagles in Germany refitting of dangerous pylons should be continued, the use of lead ammunition should be banned completely, and carcasses of large animals killed by trains should be removed from the railway by railway companies. Finally every single Sea Eagle found dead or moribund should be examined to document the causes of death.

ACKNOWLEDGEMENTS

The authors wish to thank U. Wittstatt and A. Aue for contributing the diagnostic results of the necropsies performed at the Institute for Food, Drugs and Animal Diseases (ILAT), Berlin, F. Tataruch, Research Institute of Wildlife Ecology (FIWI) Vienna for analysing the toxins of the poisoned eagles, S. Thiede for diagnosing the bacteria, and B. Seidel for performing several of the radiographs. We are also grateful to K. Ernst for her technical assistance and to L. Wölfel for organising the logistics of the sea eagle collection in Mecklenburg-Vorpommern and to the members of the Sea Eagle Working Group Schleswig-Holstein, especially to B. Struwe-Juhl. This study was supported by the State Office of Environment, Nature Conservation and Geology, Mecklenburg-Vorpommern.

REFERENCES

Bychovskaja-Pavlovskaja, I. E. 1962. *Bird trematodes of the fauna of the USSR.* [In Russian]. Verl. Akad. Wiss.
USSR, Moskau, Leningrad.

Dubois, G. 1970. Synopsis des Strigeidae et des Diplostomatidae (*Trematoda*). *Mém. Soc. Neuchâtel. Sci. Nat.* 10:
259-727.

Fischer,W. 1984. *Die Seeadler*. Die Neue Brehm-Bücherei. A. Ziemsen Verlag, Wittenberg Lutherstadt, Germany.

Forrester, D..J. & Thomas, N.J. 1998. Causes of morbidity and mortality in bald eagles from Florida. *Abstr. Annu.*
Wildl. Dis. Assoc. Conf. 47: 92.

Forsman, D. 1999. *The raptors of Europe and the Middle East.* A handbook of field identification. T & AD Poyser,
London.

Franson J.C., Galbreath, E.J., Wiemeyer, S.N. & Abell, J.M. 1994. *Erysipelothrix rhusiopathiae* infection in a
captive bald eagle *Haliaeetus leucocephalus. J. Zoo Wildl. Med.* 25: 446-448.

Furmaga, S. (1957) The helminth fauna of predatory birds (*Accipitres & Striges*) of the environment of Lublin. *Acta*
Parasitol. Polon. 5: 215-297.

Glutz von Blotzheim, U. N., Bauer, K.M. & Bezzel, E. 1971. *Handbuch der Vögel Mitteleuropas.* [Vol. 4].
Akademische Verlagsgesellschaft, Frankfurt am Main.

Görke, P. & D. Görke 2001. Kainismus beim Seeadler. *J. ber. Projektgruppe Seeadlerschutz Schleswig-Holstein*
2001: 8-9.

Halliwell, W. H. 1986. Toxic and metabolic conditions in birds of prey. Pp. 430-433 in: Fowler, M. E. (ed.). *Zoo &*
wild animal medicine. W. B. Saunders Company, Philadelphia, USA,

Harmata A.R., Montopoli, G.J., Oakleaf, B., Harmata, P.J. & Restani, M. 1999. Movements and survival of bald
eagles banded in the Greater Yellowstone Ecosystem. *J. Wildlife Manage.* 63: 781-793.

Hauff, P. 1998. Bestandsentwicklung des Seeadlers *Haliaeetus albicilla* in Deutschland seit 1980 mit einem
Rückblick auf die vergangenen 100 Jahre. *Die Vogelwelt* 119: 47-63.

Kenntner, N., Tataruch, F. & Krone, O. 2001. Heavy metals in soft tissue of White-tailed eagles found dead or moribund in Germany and Austria from 1993 –2000. *Environ. Toxicol. Chem.* 20: 1831-1837.

Kocan, A. A. & Locke, L.N. 1974. Some helminth parasites of the American bald eagle. *J. Wildlife Dis.* 10: 8-10.

Krone O. 2000. Endoparasites in free-living birds of prey from Germany. Pp. 101-116 in: Lumeij, J.T., Redig P. T., Remple D., Lierz, M. & Cooper J. E. (eds.). *Raptor Biomedicine III.* Zoological Education Network, Florida, USA.

Langgemach, T. & Sömmer, P. 1996. Zur Situation und zum Schutz der Adlerarten in Brandenburg. *Otis* 4: 78-143.

Nickol, B. B. & Kocan, A.A. 1982. *Andracantha mergi: (Acanthocephala: Polymorphidae)* from American bald eagles, *Haliaeetus leucocephalus*. *J. Parasitol*. 68: 168-169.

Oehme, G. 1961. Die Bestandsentwicklung des Seeadlers, *Haliaeetus albicilla* (L.), in Deutschland mit Untersuchungen zur Wahl der Brutbiotope. In: Schildmacher, H. (ed.) *Beiträge zur Kenntnis deutscher Vögel.* Gustav Fischer Verlag, Jena, Germany.

Oehme, G. 1987 a. Seeadler - *Haliaeetus albicilla* (L., 1758). In: Klafs, G. & J. Stübs (eds.) *Die Vogelwelt Mecklenburgs.* 3. Aufl., Gustav Fischer Verlag, Jena, Germany.

Oehme, G. 1987 b. Zum Phänomen der Eidünnschaligkeit allgemein sowie am Beispiel des Seeadlers, *Haliaeetus albicilla* (L.), in der DDR. *Populationsökologie Greifvogel- und Eulenarten* 1: 159-170.

Oehme, G. & Manowsky, O. 1991. Entwicklung und Reproduktion des Seeadlerbestandes im ehemaligen Bezirk Frankfurt/O. unter besonderer Berücksichtigung der Schorfheide. *Populationsökologie von Greifvogel- und Eulenarten* 2: 167-182.

Oehme, G., Franke, E., Hauff, P. & Scharnweber, C. 1993. Der Seeadler in Mecklenburg-Vorpommern 1990 und 1991 – Bestand, Reproduktion, Gefährdung und Schutz. *Orn. Rundbrief Mecklenburg-Vorpommern* 35: 3-8.

Rettig, T. 1978. Trichomoniasis in a bald eagle *Haliaeetus leucocephalus*. Diagnosis and successful treatment with Dimetridazole. *J. Zoo Animal Medicine* 9: 98-100.

Rysavý, B. & Ryzhikov, K.M. (eds.) 1978. *Helminths of fish-eating birds of the Palearctic region I. Nematoda.* Czechoslov. Acad. Sci., Moscow/Prague.

Ryzhikov, K. M. & Rysavý, B. (eds.) 1985. *Helminths of fish-eating birds of the Palearctic region II. Cestoda and Acanthocephales*. Czechoslov. Acad. Sci., Moscow/Prague.

Sitko, J. 1994. Helminths of birds of prey (*Falconiformes*) and owls (*Strigiformes*) in the Czech Republic and their influence on health condition of caged birds. *Zprávy MOS* 52: 53-84.

Skrjabin, K. J. [ed.] 1992. *Key to the Parasitic Nematodes*. 4. E. J. Brill, Leiden.

Smith, H. J. 1978. Cryptocotyle lingua infection in a bald eagle *Haliaeetus leucocephalus*. *J. Wildlife Dis.* 14: 163-164.

Struwe-Juhl, B. & Latendorf, V. 1997. Todesursachen von Seeadlern *Haliaeetus albicilla* in Schleswig-Holstein.
Vogelwelt 118: 95-100.
Tataruch, F., Steineck, T. & Frey, H. 1998. Vergiftungen durch Carbofuran bei Wildtieren (Greifvögel, Singvögel
und Carnivoren) in Österreich. *Wien. Tierärztl. Wochenschr.* 85: 12-17.
Tuggle B. N. & Schmeling, S.K. 1982. Parasites of the bald eagle (*Haliaeetus leucocephalus*) of North America. *J.
Wildlife Dis.* 18: 501-506.
Uttendörfer, O. 1952. *Neue Ergebnisse über die Ernährung der Greifvögel und Eulen.* Eugen Ulmer Verlag,
Stuttgart, Germany.
Stone W.B. & Nye, P.E. 1981. Trichomoniasis in bald eagles. *Wilson Bull.* 93: 109.
Zablockij, V. I. 1962. Material on the helminth fauna of the birds of prey of the riparian region of the Caspian Sea
[In Russian]. *Trudy Astrachan. Zapovedn.* 6: 91-114.