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## Pathology of Common Eiders in the Dutch Wadden Sea in December 1999

In December 1999, there was an unexplained high mortality of Common Eiders (*Somateria mollissima*) in the Dutch Wadden Sea. Gross necropsies, supplemented by histological, virological, bacteriological, and parasitological analyses were performed on 13 eiders. All birds were severely emaciated and had multifocal enteritis caused by infection with the acanthocephalan parasite *Profilicollis botulus*. The abundance of this parasite varied from 20 to 1833 (average: 865). The primary cause of death may have been starvation, i.e., lack of adequate food, or the *P. botulus* infection, although the latter was considered less likely. There was no evidence of other diseases, including those caused by viral or bacterial infections, as the primary cause of mortality in these eiders. Toxicological analyses have yet to be performed to rule out the role played by toxins in the mortality of these eiders.

### Introduction

At the beginning of December 1999, the bird rehabilitation center "Fûgelpits," Anjum in The Netherlands, reported approximately 1000 eiders *Somateria mollissima* from an estimated total of 10 000 to 12 000 eiders in the Dutch Wadden Sea as being dead or dying (Wiersma, bird rehabilitation center "Fûgelpits", pers. comm.). The cause of mortality was unknown.

In this report, the results of the pathological examination of 13 eiders found dead or dying in the Dutch Wadden Sea in the first half of December 1999 are presented. The principal aim of the study was to describe the main lesions on these birds and to determine the cause of death.

### Materials and methods

Thirteen eider carcasses were obtained for necropsy: nine were found dead and four were found alive and died after arrival at the "Fûgelpits" bird rehabilitation center. The ducks were weighed and a standard necropsy was carried out. The tissues from all major organs and from any lesions were fixed in formalin, stained with haematoxylin and eosin and examined by light microscopy. Virological analysis was performed at the Department of Virology at the Erasmus University of Rotterdam in The Netherlands.

The pooled samples of liver and spleen from each eider, the samples of oesophageal lesions from birds # 10 and #12 and a sample of an oral lesion found on bird #6 were used in procedures capable of isolating the herpesvirus of duck viral enteritis. The tissue samples from each eider were tested for the presence of influenzaviral RNA and herpesviral DNA by PCR. A sample of lung showing evidence of much inflammation was submitted for routine aerobic bacterial culture to the Veterinary Microbiologic Diagnostic Center, Faculty of Veterinary Medicine of the University of Utrecht in The Netherlands. 3-cm lengths of intestine were cut open at 30 cm intervals along the intestine, thus allowing the acanthocephalans to be counted. The total acanthocephalan burden was calculated as the product of the average number of acanthocephalans per cm of intestine and the total length of intestine. Samples of each type of parasite found were identified at the Veterinary Microbiological Diagnostic Center, Faculty of Veterinary Medicine at the University of Utrecht in The Netherlands.

### Results

All birds were severely emaciated, and also characterized by a low body weight, severe atrophy of skeletal musculature, absence of fat depots, serous atrophy of epicardial fat, a small, dark-tan liver with sharp edges and a distended gall bladder.

All birds had acanthocephalan parasites, identified as *Profilicollis botulus*, in the intestine. They were generally located in the second half of the jejunum, from about 10 cm anterior to the vitelline diverticulum to the ileo-caecal junction. The heads of most *P. botulus* were embedded in the intestinal wall and were visible from the serosal side as white to yellow foci, about 5 mm long and 1 mm wide. The total acanthocephalan burden per bird varied greatly, from 20 to 1833 (average: 865). Nine birds had cestodes, identified as *Microsomacanthus microsoma*, in the first 30 to 60 cm of the small intestine. Most of these cestodes were not attached to the mucosa and no associated gross lesions were seen. There were multiple yellow foci (1 to 5 mm in diameter) in the mucosa of the oesophagus (#10 to #13) or palate (#6).

The *Profillicollis botulus* infection was associated with multifocal enteritis, which varied from suppurative to granulomatous to fibrosing. The inflammation was limited to the tissue immediately adjacent to the parasite's head, which was located in the mucosa, submucosa, or muscularis (Fig. 1). The oral and oesophageal lesions were histologically similar, and consisted of well-demarcated areas of epithelial necrosis, with hyper-eosinophilia and karyolysis. Aggregates of cocci were abundant in and on the necrotic epithelium. Depending on the age of the lesions, the underlying submucosa contained either granulocytes or epithelioid macrophages and multinucleated giant cells.

Other histological findings included renal coccidiosis (3 ducks), bursal trematodes (2 birds), pulmonary haemorrhage (2 birds), proventricular haemorrhage (1 bird), and aspiration of foreign material (1 bird), myocardial sarcocysts (1 bird), and proventricular parasites (1 bird).

No viruses were isolated from any of the birds. The results of RT-PCR, which was used to detect influenza viruses and of the PCR used to detect herpesviruses, were found to be negative. A mixture of six bacterial species was isolated from a lung sample from bird #8, which had aspirated foreign material.

## Discussion

The two most common and most noticeable changes in these 13 eiders were emaciation and *P. botulus*-associated enteritis. By gross necropsy, the degree of emaciation of these Common Eiders was considered to be sufficiently severe to be fatal. A more objective parameter for emaciation is the percentage of weight loss compared to the original body weight. From data gathered from laboratory experiments and field studies on food deprivation, Wobeser (1981) concluded that in general, waterbirds lose about 40% of their body weight before dying. This is slightly more than the weight loss in these eiders, which was 7 to 52% (average 29%) of their body weight before death. However, it should be noted that expected body weights were derived from data of eiders from 1926 in Denmark, and that the body weight of nine birds was more than likely overestimated because they were weighed while their plumage was wet.

Identifying the cause of emaciation can be difficult. In primary malnutrition, the diet is lacking in quantity (simple starvation) or quality (specific nutritional deficiency). By contrast, in secondary mal-

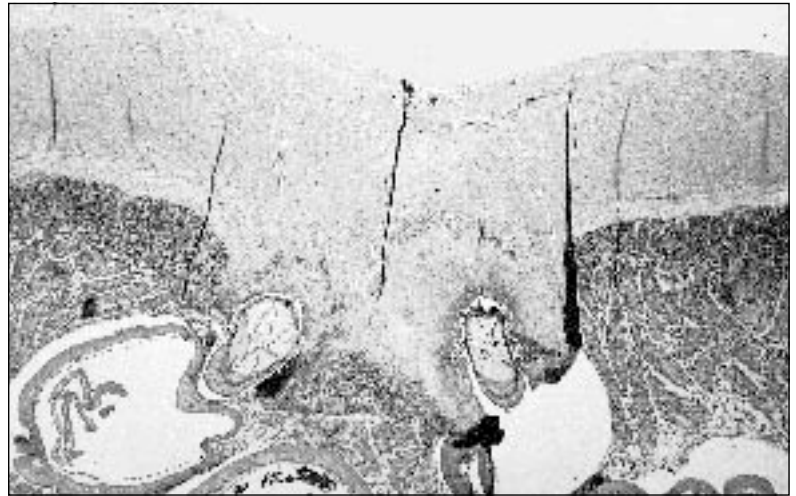


Figure 1: Microphotograph of the small intestine of an eider infected with *Profillicollis botulus*. Note the inflammatory reaction in the tissue immediately around the heads of the two parasites. Haematoxylin and eosin, original magnification 25 X.

nutrition, the supply of nutrients is adequate, but malnutrition may result from nutrient malabsorption, impaired nutrient use or storage, excess nutrient losses, or an increased need for nutrients (Cotran et al., 1999). Before deciding that emaciation is the result of lack of adequate food (i.e., simple starvation), the presence of diseases that might cause secondary malnutrition has to be ruled out.

The only disease affecting all 13 eiders was *P. botulus*-associated enteritis. This inflammation was restricted to small areas where the heads of the parasites penetrated the mucosa, so that a considerable area of intestinal mucosa was not inflamed. Perforation of the intestinal wall with resulting generalized peritonitis, as reported in a Mute Swan *Cygnus olor* infected with a related acanthocephalan, *P. minutus* (Sanford, 1978), was not found in any of these eiders.

It was not possible to determine whether the abundance of *P. botulus* in these eiders differed from that of previous years because historical data is lacking. The abundance of *P. botulus* in eiders is highly variable and depends on different factors. In eiders studied between 1955 and 1982 at one location in Scotland, the average parasite abundance varied with the age of the bird (juveniles in their first winter had ten times higher levels than adults), season (there was a sharp peak in November in juveniles) and year (the abundance in some years was up to seven times higher than in other years) (Thompson, 1985). The average *P. botulus* abundance in juvenile eiders in Thompson's (1985) study was 580 in November and 400 in December, in comparison with an average of 907 in the juvenile/immature eiders reported in this study.

*P. botulus* has been reported as being responsible for the eider mortality (reviewed in Thompson, 1985). Most of these studies, however, assumed that a bird found dead and carrying the infection actually died from the infection and full necropsies of the affected eiders were therefore not performed. Thompson (1985) found no significant difference in *P. botulus* abundance in juvenile eiders taken alive or found dead during winter and concluded that there was no evidence that *P. botulus* increases the mortality rate of eiders.

The pathogenic effect of *P. botulus* on eiders has not been investigated. In a study on the related parasite, *P. minutus*, Hollmén et al. (1999) found that eider ducklings, which had been infected for experimental purposes, grew more slowly and had a lower total protein level in the serum than control birds. It was concluded that this parasite might have, either alone or together with other factors, contributed to the low survival rate of eider ducklings observed in the Gulf of Finland during the past decade.

It is certain that the *P. botulus* infection was at least a contributory cause of emaciation in these eiders, e.g., through direct absorption of amino acids by the parasites, decreased absorption of nutrients by the damaged intestinal mucosa, and reduced appetite (Hollmén et al., 1999). On the basis of current evidence, it cannot be denied that the infection was severe enough to be the primary cause of emaciation –and therefore death– of these animals. However, this is considered unlikely, based on the moderate nature of the lesions, the moderate increase in parasite abundance compared to available reference data and the absence of convincing publications of eider mortality caused by *P. botulus*.

Taking the above into account, starvation, i.e., lack of adequate food, is considered to be the most likely primary cause of emaciation in these ei-

ders, with the *P. botulus* infection as a contributory cause. Regardless of the fact whether the *P. botulus* infection or starvation was the primary cause, there was undoubtedly interaction between both factors. Starvation would have decreased the eiders' ability to resist parasitic infection for example, and the parasite would have exacerbated the malnutrition by damaging the intestine and competing with the host for nutrients (Wobeser, 1981).

The role played by toxins in the death of these eiders cannot be ruled out without further analyses. Chronic lead poisoning, in which there are no specific lesions, is a common primary cause of death in waterfowl found dead in an emaciated condition (Wobeser, 1981). In 1988, high concentrations of nonylphenol were found in the plumage of dead eiders from the Dutch Wadden Sea (Zoun et al., 1991). The toxicological analysis on tissues from these eiders to rule out chronic intoxication, including by above-named substances, is pending.

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