Lead poisoning and parasitism in a flock of mute swans (*Cygnus olor*) in Scotland

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Increased mortality in a flock of non-breeding mute swans (*Cygnus olor*) on a Scottish loch was investigated. Postmortem examinations were carried out on eight adult and six immature swans. The commonest cause of death, found in eight birds, was lead poisoning associated with the ingestion of large lead fishing weights. Heavy parasitic burdens were found in five immature birds, involving combinations of the gizzard worm *Amidostomum* species, the thornyheaded worms *Polymorphus minutus* and *Profilicollis anatis*, and the tracheal trematode *Orchipedum tracheicola*. Other parasites of lesser significance were the biting louse *Trinoton anserinum*, the tapeworm *Wardoides nyrocae*, the hairworm *Capillaria* species and the intestinal trematode *Echinoparyphium recurvatum*. Eight of the 14 swans carried trematodes of the family Schistosomatidae, which may be involved in human cercarial dermatitis or 'swimmers' itch'. It is suggested that the increased mortality arose through a combination of increased numbers of swans on the loch, and a fall in the water level of the loch which exposed the birds to previously inaccessible lead fishing weights and to the intermediate hosts of a range of internal parasites.

THE mute swan (*Cygnus olor*) is the largest resident breeding wild bird in the UK, adult males occasionally weighing over 15 kg. In the past 50 years there have been marked fluctuations in the population of this conspicuous bird. Numbers of mute swans were high in the late 1940s and 1950s, only to fall back because of the cold winters of the early 1960s (Ogilvie 1967). Numbers recovered, but declined steeply again in the 1970s and 1980s as a result of lead poisoning from the ingestion of lead fishing weights (Ogilvie and Delaney 1993). A combination of mild winters, greater control on the use of lead weights by anglers, and increased numbers of flooded gravel workings providing suitable habitats, led to a recovery in numbers, from about 19,000 birds in 1983 to nearly 26,000 birds in 1990 (Ogilvie and Delaney 1993), at which figure the numbers appear to have stabilised (Cranswick and others 1995).

Juvenile and non-breeding adult mute swans usually form flocks, sometimes numbering hundreds or even thousands, in estuaries, rivers, lakes, reservoirs, shallow coastal waters, shallow freshwater marshes, flooded gravel pits, and ponds in public parks in towns and cities (Birkhead and Perrins 1986). Mute swans feed almost exclusively on plant material, with animal material such as molluscs and crustaceans being ingested accidentally. Many urban flocks become dependent on food, usually bread, provided by the general public, especially in the winter months when natural vegetation dies back. Increases in the size of these non-breeding flocks may make the birds vulnerable to the accidental or intentional acts of the human population, to changes in food availability, and to infectious diseases. For example, a rapid increase and subsequent decrease in the numbers of mute swans at the Loch of Harray, Orkney, was associated with fluctuations in the availability of Canadian pond weed (*Elodea canadensis*) and the cumulative effects of parasites (Meek 1993).

This paper describes the causes of increased mortality in a flock of non-breeding mute swans on a loch at a country park in central Scotland between January and May 1996.

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Materials and methods

Site

The loch has a surface area of approximately 16 hectares and is rich in animal and plant life. It supports a range of waterfowl, mostly tufted duck (*Aythya fuliga*), mallard (*Anas platyrhynchos*) and coot (*Fulica atra*). In recent years the population of non-breeding mute swans has been increasing, and there were over 90 in the winter of 1995/96. The water depth over most of the loch varies between 1·5 metres and 2 metres but had been allowed to fall below this level for several months.

Submission of sick or dead birds

The country park is widely used by fishermen, other members of the public take recreation in the park and feed the swans, and a team of park rangers is active in the park. The high level of human activity increases the chance of any sick or dead birds being observed.

Birds found dead were submitted directly to the Scottish Agricultural College Veterinary Services Avian Health Unit (AHU) by park rangers. Birds which were alive but ill were taken to a local wildlife rehabilitation centre (Hessilhead Wildlife Rescue Trust, Beith) where they were given appropriate treatment. Birds which died were then submitted to the AHU for postmortem examination.

Postmortem examination

A standard postmortem procedure was followed, similar to that described by Waine (1996). Cultures for aerobic bacteria, anaerobic bacteria or fungi were made if there was any suggestion that these agents were involved and selected tissues were examined histologically if required. Samples of liver and kidney were analysed for lead by atomic absorption spectrophotometry at the Moredun Research Institute, Edinburgh. Parasitological investigations included an inspection of the plumage for ectoparasites, an examination of the nasal sinuses, a gross examination of the length of the opened alimentary tract from proximal oesophagus to cloaca, and a gross examination of the trachea and airsacs. Scrapings were made from the mucosa of the oesophagus, proventriculus and approximately every 30 cm of the intestine from the duodenum to the cloaca, including the terminal portion of the large intestine and the caeca. The scrapings were examined microscopically at x 100 and x 400. As an aid to identification, the parasites or their eggs were measured using a computer-based veterinary diagnostic system developed for the differentiation of parasitic eggs and oocysts (Mitchell and others 1995). If schistosome eggs were detected on microscopical examination of intestinal smears, various blood vessels throughout the carcase were opened in an attempt to find the adult trematodes. The proventriculus and gizzard and, where appropriate, the oesophagus, were opened under water and, after the removal of any parasites, the contents were thoroughly examined for material such as lead shot or lead weights by repeated washing and inspection of the sediment. The length of the trachea and bronchi was examined grossly in all the birds and, in the majority, the trachea and bronchi were also washed under water to increase the likelihood of recovering parasites. Parasites were identified to genus level by standard parasitological techniques, and some parasites were submitted to the Natural History Museum, London, for more detailed examination.

TABLE 1: Criteria used to allocate scores to the burdens of different parasites recorded in 14 swans

Parasite	Scores
Cestodes*	+ An occasional cestode in affected areas of intestine ++ Many cestodes in affected areas of intestine +++ Many cestodes causing distension of affected areas of intestine, with cestodes oozing out when intestine is opened
Amidostomum	+ 1-10 worms in gizzard ++ 11-50 worms in gizzard +++ More than 50 worms in gizzard
Capillaria	+ Consistently 1-5 loose eggs per microscope field at x 100 magnification in smears from affected areas ++ Consistently 6-50 loose eggs or 1 worm/worm fragments per microscope field at x 100 magnification in smears from affected areas +++ Consistently more than 50 loose eggs or more than 1 worm/worm fragments per microscope field at x 100 magnification in smears from affected areas
Acanthocephala	+ 1-20 acanthocephalans in intestine ++ 21-100 acanthocephalans in intestine +++ More than 100 acanthocephalans in intestine
Blood trematodes	+ Consistently 1-5 eggs per microscope field at x 100 magnification in smears from affected areas of intestine ++ Consistently 6-50 eggs per microscope field at x 100 magnification in smears from affected areas of intestine +++ Consistently more than 50 eggs per microscope field at x 100 magnification in smears from affected areas of intestine
Tracheal trematodes	+ 1-20 trematodes in trachea ++ 21-100 trematodes in trachea +++ More than 100 trematodes in trachea
Intestinal trematodes	+ Consistently no more than 1 trematode per 22 mm x 22 mm microscope cover slip on microscopical examination of smears from affected areas of intestine ++ Consistently 2-5 trematodes per 22 mm x 22 mm microscope coverslip on microscopical examination of smears from affected areas of intestine +++ Consistently over 5 trematodes per 22 mm x 22 mm microscope coverslip on microscopical examination of smears from affected areas of intestine
All categories	0 Parasites or their eggs not seen

* Large species visible without microscopy

Parasitic burdens were graded on the basis of the numbers present. The criteria upon which the scores were allocated varied with the nature of the parasites present, and are summarised in Table 1.

A diagnosis of 'parasitism' was made if the parasites were associated with gross lesions sufficiently severe to cause or contribute to the death of the bird, otherwise the parasites were described as 'insignificant'.

Birds were described as immature if they still retained some brown feathers in their plumage. If they were fully white, which occurs when mute swans are just over 12 months of age (Birkhead and Perrins 1986), they were described as adult.

TABLE 3: Concentrations of lead in the kidney and liver of 14 mute swans

Bird	Age	Kidney (ppm DM)	Liver (ppm DM)
1	Adult	869	255
2	Immature	20	5
3	Adult	213	77
4	Immature	4	3
5	Adult	138	61
6	Adult	119	99
7	Adult	359	77
8	Adult	10	2
9	Adult	2	1
10	Immature	708	35
11	Immature	3	1
12	Adult	114	36
13	Immature	10	3
14	Immature	514	30

ppm DM Parts per million in the dry matter

TABLE 2: Main postmortem findings in 14 mute swans

Bird	Age	Main postmortem findings
1	Adult	Lead poisoning; insignificant parasites
2	Immature	Parasitism; airsacculitis
3	Adult	Lead poisoning
4	Immature	Parasitism; visceral gout; fishing hook in oesophagus
5	Adult	Lead poisoning; insignificant parasites
6	Adult	Lead poisoning; fishing hooks in oesophagus and gizzard
7	Adult	Lead poisoning; candidiasis; airsacculitis; insignificant parasites
8	Adult	Necrotic enteritis; fibrinous airsacculitis; fishing hook in oesophagus; insignificant parasites
9	Adult	Aspergillosis; insignificant parasites
10	Immature	Lead poisoning; parasitism
11	Immature	Aspergillosis; parasitism
12	Adult	Lead poisoning
13	Immature	Parasitism; airsacculitis
14	Immature	Lead poisoning; necrotic enteritis; fishing hook in oesophagus; insignificant parasites

Results

General

Fourteen birds were received for postmortem examination between January and May 1996; eight were adult and six immature. A summary of the main findings is given in Table 2.

Lead poisoning

A diagnosis of lead poisoning was made if the concentration of lead in the kidneys was more than 100 parts per million dry matter (ppm DM) (Sears and Hunt 1991). The concentrations in the kidney and liver are given in Table 3. In both the tissues they fell into two distinct ranges, 2 to 20 (mean 8) ppm DM in the kidneys and 1 to 5 (mean 2) ppm DM in the livers of unaffected birds, and 114 to 869 (mean 379) ppm DM in the kidneys and 30 to 255 (mean 84) ppm DM in the livers of the birds considered to be suffering from lead poisoning. The concentration of lead in the kidney exceeded 100 ppm DM in eight birds, two immature and six adult. In six of the eight birds, one or more large lead ledger weights were found in the gizzard. No small split-shot lead weights, and no lead originating from gunshot were found in any of the birds. Many of the affected birds had impactions of the oesophagus and/or proventriculus with vegetation, coarse grit or fine sand.

Parasitism

Parasitism was considered to be the cause of death of three birds, and heavy parasitic burdens contributed to the death of another bird with lead poisoning and another with aspergillosis. All five birds were immature. Smaller numbers of parasites, not considered significant, were found in a further six birds (five adult and one immature). Three adult birds with lead poisoning had no detectable parasites.

The parasites were divided into the following categories: cestodes, nematodes (*Amidostomum* species or *Capillaria* species), acanthocephala (thornyheaded worms), blood trematodes (Family Schistosomatidae), tracheal trematodes (Family Orchipedidae), intestinal trematodes (Family Echinostomatidae and other Families). Tables 4 and 5 list the parasites that were found in the 14 mute swans.

Birds 2, 4 and 13 had sufficient numbers of parasites to be considered pathological. They all had significant numbers of trematodes in the trachea, acanthocephalans in the intestine, and *Amidostomum* species in the gizzard. One of these three birds also had moderate numbers of cestodes, and one also had moderate numbers of an echinostomatid intestinal trematode. Two other

TABLE 4: Categories of parasites found in 14 mute swans

Category of parasite	Number of birds
Cestodes	1
Nematodes: *Amidostomum* species	5
Capillaria species	1
Acanthocephala	6
Trematodes: Blood	8
Tracheal	7
Intestinal	2
None	3

TABLE 5: Burdens of parasites found in 14 mute swans

Bird	Age	Parasites found
1	Adult	+ Blood trematodes
2	Immature	++ Cestodes ++ *Amidostomum* +++ Acanthocephala ++ Tracheal trematodes +++ Blood trematodes
3	Adult	None
4	Immature	++ *Amidostomum* +++ Acanthocephala +++ Blood trematodes +++ Tracheal trematodes
5	Adult	+ Acanthocephala ++ Blood trematodes
6	Adult	None
7	Adult	++ Blood trematodes + Tracheal trematodes
8	Adult	+ Acanthocephala + Blood trematodes
9	Adult	+ *Amidostomum*
10	Immature	++ Tracheal trematodes
11	Immature	+++ Acanthocephala +++ Tracheal trematodes
12	Adult	None
13	Immature	+++ *Amidostomum* + *Capillaria* +++ Acanthocephala ++ Blood trematodes +++ Tracheal trematodes ++ Intestinal trematodes (Echinostomatidae)
14	Immature	+ *Amidostomum* ++ Blood trematodes + Tracheal trematodes + Intestinal trematodes (non-Echinostomatidae)

birds had heavy parasitic burdens; bird 10 which died from lead poisoning had large numbers of tracheal trematodes, and bird 11 (cause of death aspergillosis) had many acanthocephalans and tracheal trematodes. The tracheal trematodes were subsequently identified as *Orchipedum tracheicola,* and the acanthocephalans as *Polymorphus (Profilicollis) minutus, Filicollis anatis,* and/or mixed infections of these two species.

Gross and histological lesions were associated with these parasites of the gizzard, trachea and intestine. When moderate to large numbers of *Amidostomum* were present, the koilin layer of the gizzard was soft, easily disrupted, and discoloured black/brown, with thin parasites measuring approximately 10 to 20 mm in length and 1 mm in width in the disintegrating koilin layer or under the adjacent areas. In the birds with moderate to many tracheal trematodes, a tracheitis was visible grossly, varying from mucoid to haemorrhagic and necrotic. The trematodes were large and easily visible in the trachea, adults reaching approximately 13 mm long by 4 mm wide. The ventral sucker of the trematode was prominent, and there was a constriction in the width of the trematode at the level of the sucker. Histologically there was a chronic tracheitis with loss of cilia and mucous glands, and nodular epithelial hyperplasia and metaplasia to stratified squamous epithelium. Several of the birds with tracheal trematodes also had a moderate to severe airsacculitis. Cultures from the airsacs yielded *Escherichia coli* in three birds, one bird had a typical mycotic airsacculitis from which *Aspergillus fumigatus* was isolated, and in another bird the airsacs were covered by a fibrinous layer 2 to 3 cm thick from which *Pasteurella multocida* was isolated. Smears from the airsacs frequently contained the eggs of the trematodes, which appeared as large golden-brown ovoid structures measuring on average 79 µm by 62 µm.

The presence of large numbers of acanthocephalans was associated with a catarrhal to haemorrhagic enteritis, the cylindrical white to yellow parasites being clearly visible with their heads embedded in the intestinal mucosa. Most of the acanthocephalans were 8 to 16 mm long, with a diameter of 1 to 3 mm. The mid intestine appeared to be the area most heavily parasitised and perforation to the serosal surface resulted in nodule formation and peritonitis.

In contrast with the gizzard worms, tracheal trematodes and acanthocephalans, the presence of trematodes of the family Schistosomatidae did not appear to be associated with lesions. This was the commonest parasite detected, with eggs found in intestinal scrapings from four adult and four immature birds. The eggs were usually found in scrapings from the mid to distal intestine. Most were found clustered in fragments of villi removed when making the smears, although small numbers were found free in the intestinal contents. The majority were oval, measuring approximately 70 µm by 50 µm, with a terminal hook apparent in some but not all of the eggs. Less common was an elongated form measuring approximately 80 µm by 40 µm, again with a terminal hook visible on some eggs. Most birds had both types of eggs. The adult trematodes producing the eggs were not detected by the standard postmortem procedure, but histological examination revealed schistosomes in the veins of the serosa of the small intestine.

In addition to the internal parasites, most of the birds had small numbers of the large biting louse *Trinoton anserinum* on the feathers of the head, neck or body, but the presence of these parasites was not considered to be significant.

Fishing hooks and line

Fishing hooks were found embedded in the wall of the oesophagus of four birds, one of which also had a fishing hook in the web of a foot and one of which in addition had a fishing hook firmly embedded through the koilin layer into the gizzard muscle. In two of the four birds, the presence of the fishing hook was probably incidental, death being the result of lead poisoning and parasitism, respectively. In the other two birds a quantity of nylon line was still attached to the hook or had accumulated lower down the digestive tract, and in both birds a severe necrotic enteritis was considered to be responsible for the death of the bird, with concurrent lead poisoning in one of them. Necrosis and sloughing of the mucosa of the duodenum and much of the intestine had resulted, forming a hollow cast. Gram-stained smears revealed many Gram-positive bacilli, and anaerobic cultures yielded profuse growths of *Clostridium perfringens.*

Fungal and yeast infections

A fumigatus was recovered from the airsacs of an immature swan which had a severe airsacculitis with large fungal plaques, and large numbers of tracheal trematodes. A second swan, an adult, had an airsacculitis and granulomatous pneumonia associated with *A fumigatus,* with a terminal fatal intrapulmonary haemorrhage. This bird had previously been isolated and treated for lead poisoning, had apparently recovered, but then died suddenly.

The distal oesophagus of a third swan was thickened and had raised, white loosely adherent linear plaques, from which *Candida albicans* was isolated. This bird had died from lead poisoning, as a result of which the oesophagus and proventriculus had become impacted with vegetation.

Discussion

The commonest cause of death was lead poisoning which affected six adult birds and two immature birds. Some of the birds with lead poisoning were seen alive, showing the typical signs of lead poisoning – generalised weakness, inability to hold up the head resulting in a kinked neck, loss of weight and green fluid faeces. These birds died despite chelation therapy with 10 to 40 mg/kg sodium calcium edetate intramuscularly twice daily for up to 10 days (Forbes 1996). The failure to respond to treatment was most probably due to the large mass of lead remaining in the gizzard of most of the birds, and found postmortem. This observation reinforces the value of routine radiography of swans with suspected lead poisoning to detect the presence of lead in the gizzard, with a view to its removal by gastric lavage (Forbes 1993) or by surgery (Poole 1986).

The history of lead poisoning in mute swans in England has been well documented (Sears and Hunt 1991). Historically, most of the lead originated from split-shot weights used by anglers, with a smaller number of birds being poisoned by the large ledger weights used by anglers or by lead from gunshot pellets. Deaths from lead poisoning tended to be highest in mid summer, coinciding with the peak of the fishing season. After the report of the Nature Conservancy Council Working Group in 1981 (Nature Conservancy Council 1981), a series of voluntary codes of practice or voluntary bans were implemented to reduce the hazards to swans from fishing weights. This was followed by statutory measures (The Control of Pollution [Anglers' Lead Weights] Regulations 1986; The Salmon and Freshwater Fisheries Act 1975 [as amended]) making it illegal in England and Wales to sell, import or use for fishing lead weights weighing between 0·06 g and 28·36 g. These regulations do not apply in Scotland, but are observed on a voluntary basis.

The cases described here differ from those seen before the changes in legislation in England, in that they occurred outside the peak of the fishing season, and consistently involved large ledger weights rather than split-shot. These ledger weights were within the banned weight range, and have seldom been used by anglers in recent years. It is probable that the weights responsible for the lead poisoning in these swans had been present in the environment for several years, and became available to the swans only because the water level in the loch had fallen, giving the birds access to previously inaccessible debris. The seasonal pattern of the deaths from lead poisoning (January to May) was, however, similar to that described in Scottish swans by McDonald and others (1987) who found that 13 deaths from lead poisoning all occurred in the cold months of the year, although they were associated with shotgun pellets rather than anglers' weights. It is likely that lead poisoning will continue to be a hazard for waterfowl, despite the restrictions on the use of lead weights by anglers, owing to the persistence of ledger weights and shotgun pellets in the environment.

Heavy parasitic burdens were found in five immature birds. The gizzard worm *Amidostomum* was found in moderate to large numbers in three of the five birds, large numbers of acanthocephalans in four of them and moderate to large numbers of tracheal trematodes in all five birds.

The gizzard worm *Amidostomum anserinus* has been found in mute swans suffering from emaciation, weakness and death (McNeil 1970, Brown and others 1992a,b) with postmortem lesions similar to those observed in this study. Healthy swans commonly have small numbers of *Amidostomum* in the gizzard (Bailey and others 1990) and disease caused by this parasite is most likely to be seen in young or debilitated birds (Brown 1992a). This study supports that view, the three birds with significant numbers of *Amidostomum* being immature birds with additional parasitic problems.

Deaths in mute swans resulting from large burdens of the thornyheaded worm *Polymorphus (Profilicollis) minutus* (synonym *Polymorphus boschadis*) have been reported by Sanford (1978), McDonald and others (1978, 1987) and Meek (1993). The intermediate hosts for these acanthocephalans are the freshwater shrimp (*Gammarus pulex*), possibly the crayfish (*Potamobius astarus*) and the water louse (*Asellus aquaticus*) (Soulsby 1982). The large number of acanthocephalans in the swans may have been the result of the drop in water level and the shortage of natural vegetation in the winter months, resulting in an increased accidental consumption of these intermediate hosts. Parasite counts of 35 and 50 were considered to be significant by McDonald and others (1987). In this study four of the five birds with parasitism had more than 100 acanthocephalans.

Five immature birds had moderate to large numbers of the tracheal trematode *Orchipedum tracheicola*. Some of the birds were seen before they died and were breathing noisily. This is believed to be the first report of this parasite causing clinical disease in several mute swans in the UK, and the trematode should be added to the list of conditions causing respiratory disease in swans. The intermediate host is probably a small crustacean such as the freshwater shrimp (*Gammarus* species) (E. Harris, personal communication) and again it is possible that access to these crustaceans was increased by the drop in water level.

The occurrence of schistosomes in eight of the 14 swans was unexpected. Schistosomes inhabit various blood vessels depending on the species of trematode, and their eggs migrate from the blood through the tissues to the intestine or other organs, from which they are excreted. An intermediate host, for example freshwater snails of the genus *Lymnaea*, are required for completion of the life cycle (Soulsby 1982). Inflammatory changes can result after the eggs, which may have a terminal spine, pass through the tissues. Avian schistosomes are also important because the cercariae can cause a dermatitis if they migrate through the skin of human beings. Repeated exposure to water containing the cercariae may result in a chronic dermatitis referred to as 'swimmers' itch' and other names describing the occupations at risk (Soulsby 1982). The frequent occurrence of schistosomes in the swans on this loch, while not apparently causing disease in the birds, may therefore pose a hazard to members of the general public using the water for recreational purposes.

One bird had moderate numbers of the echinostomatid trematode *Echinoparyphium recurvatum* in the duodenum, as part of a heavy burden of many species of parasite. A catarrhal enteritis, diarrhoea, emaciation and death have been described in mute swans with this parasite (Soulsby 1955), but in the present study the other parasites present (*Amidostomum*, acanthocephala, *Orchipedum*) were considered to be of greater importance. Also as part of a generalised parasitic burden, one bird had moderate numbers of tapeworms identified as *Wardoides nyrocae* (E. Harris, personal communication).

It is apparent, therefore, that parasitism was a major problem in the immature birds. The large numbers of parasites may have resulted from a combination of an increase in the number of swans on the loch, a reduction in natural food supplies in the winter months, and a fall in the water level allowing increased access to the intermediate hosts. The potential consequences of human activities such as the lowering of water levels should therefore be considered when dealing with large numbers of vulnerable wild birds.

The direct impact of human activities was also underlined by the recovery of portions of fishing tackle from four of the birds. Nylon fishing line (of which much was in evidence, discarded around the shores of the loch) caused more problems than fishing hooks themselves. Tangled masses of fishing line were found in the digestive tract of two birds, both of which also had a necrotic enteritis associated with *C perfringens*. Necrotic enteritis, reported in mute swans by McDonald and others (1987, 1990) is not uncommon in broiler chickens in which the disease may be the result of dietary factors and/or damage to the intestine causing an increase in the numbers of *C perfringens* in the digestive tract (Truscott and Al-Sheikhly 1977, Ficken 1991). In the two swans which had necrotic enteritis, the tangled masses of fishing line may have been the trigger factor.

Aspergillosis was the cause of death of two of the swans, and candidiasis was a secondary complication in a third bird. Although Brown and others (1992a,b) commented that swans are not particularly susceptible to aspergillosis, in their studies it was the commonest non-parasitic infectious cause of death of imma-

ture captive or wild swans, and was second only to tuberculosis in adult swans. Aspergillosis was also found by McDonald and others (1987, 1990) to be the commonest non-parasitic infectious cause of death in their studies of mortality in Scottish swans. In the present study, aspergillosis appeared to be secondary to tracheal trematodes in one bird, and the other bird had been held in captivity while undergoing treatment for lead poisoning, a situation likely to stress the bird and predispose it to aspergillosis.

The oesophageal candidiasis observed in one bird with lead poisoning was most likely to have been a consequence of the debilitated state of the bird and the effect of the vegetative mass impacting the oesophagus.

One immature bird with a heavy parasite burden had extensive lesions of visceral gout, with urates on the surface of the epicardium, liver, kidney and other abdominal organs, and under the skin. Histological examination demonstrated nephrotic changes, and the renal damage was probably secondary to the parasitism. Visceral gout was described by McDonald and others (1987, 1990) in swans with histological changes of nephritis and sometimes amyloidosis. Brown and others (1992a,b) also found visceral gout and renal failure to be common in swans, frequently in birds dying of other conditions or with renal coccidiosis. It is apparent, therefore, that several different conditions can result in visceral gout.

There were several causes of death in this flock of swans, but certain conditions which have been recorded in other studies, notably trauma, avian tuberculosis and acuariasis, were absent. Trauma, especially flying accidents, caused 20 to 30 per cent of all deaths in wild swans in the surveys by McDonald and others (1987, 1990) and Brown and others (1992b), but it was not recorded in this study, suggesting that this was a fairly static flock in January to May. Avian tuberculosis was recorded in 1 to 6 per cent of wild swans (McDonald and others 1987, 1990, Brown and others 1992b) and in 33 per cent of captive adult swans (Brown and others 1992a), but was not diagnosed in swans in the current study, possibly because they had not gathered in large numbers on this loch until recent years. In contrast to the results reported by Brown and others (1992a,b) in which heavy burdens of *Acuaria uncinata* resulted in mortality rates of 12·5 per cent of immature wild swans and 26·8 per cent of immature captive swans, none of the birds in this study or in the survey of Scottish swans by McDonald and others (1987, 1990) had detectable burdens of *Acuaria.* Swans acquire the larval stages of *Acuaria* by ingesting the common waterflea (*Daphnia* species) from the surface of the water as they feed.

Unlike several other species of Britain's wild birds, the numbers of which are in decline (Gibbons and others 1996), the mute swan population appears to have stabilised, after fluctuating for many years. However, the close association between this bird, especially when in large non-breeding flocks, and the human population, makes it vulnerable to man's activities. Lead poisoning remains a threat to swans, despite measures to reduce the impact of anglers' lead weights, and carelessly discarded fishing line can also result in swan deaths. Large non-breeding flocks may become dependent on people for winter feeding, and accidental or intentional manipulation of their environment, for example by reducing water levels, may precipitate parasite problems.

To the list of diseases which should be considered when the veterinary profession and wildlife rehabilitators are investigating infectious diseases of swans, the study has added respiratory disease caused by the tracheal trematode *O tracheicolum.* The role of swans in carrying trematodes of the family Schistosomatidae, with the possible zoonotic implications of cercarial dermatitis or 'swimmers' itch' has also been identified. Work remains to be done, however, to find appropriate solutions to these problems, both in terms of suitable therapeutics, and in ameliorating the deleterious effects of the human population on wild birds.

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Abstract

Risk factors for feline infectious peritonitis in catteries

FELINE infectious peritonitis (FIP) virus is a mutant strain that arises during the replication of endemic feline enteric coronavirus (FECoV). The risk factors for FIP include age (most affected cats are four months to three years old), being born in a multiple-cat household and a positive titre against FECoV. In this study the cats in seven catteries and a cat shelter in which FECoV was endemic were studied for a year. The most important individual risk factor for FIP was age (the mean age at death of the 24 cats that died was 9·8 months); the most important risk factors associated with the cats' accommodation were the proportion of cats that regularly shed FECoV in their faeces and the overall prevalence of shedding of FECoV. The factors not associated with FIP were; the numbers and sex of the cats, the husbandry and quarantine practices, the caging and breeding practices and the prevalence of other diseases. More of the cats died of FIP in the autumn and winter than in the spring and summer.

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