Causes of mortality in Mute Swans *Cygnus olor* in Scotland 1995-1996

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As part of a survey into the causes of mortality of wild birds, the carcases of 41 Mute Swans (Cygnus olor) were examined at S.A.C. Veterinary Science Division, Auchincruive, Ayr, Scotland, between January 1995 and December 1996.

Birds were submitted for post mortem examination directly by members of the general public, by a local wildlife rehabilitation centre, or by other organisations with an interest in wild birds. Fourteen of the birds were received from one site during a five month period and were representative of a greater number of dead swans found on that site. The remaining 27 swans were received over the two years from several different sites as sporadic deaths.

The most significant primary causes of death in the birds from the site with heavy mortality were lead poisoning and heavy parasitic burdens. In contrast, in birds submitted as apparent sporadic deaths, the commonest primary causes of death were trauma, starvation, and aspergillosis.

Mute Swan *Cygnus olor* drawn by Amanda Bradbury, WWT

Key words: Mute Swan, Mortality, Lead Poisoning, Parasitism.

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Pairs or flocks of the Mute Swan *Cygnus olor* are a common and popular sight on the inland waters and estuaries of the United Kingdom, including artificial reservoirs and ponds in urban areas. The size of the population of this conspicuous bird has fluctuated in the past 50 years, partly due to cold winters in the early 1960s (Ogilvie 1967) and partly due to man-made problems such as lead poisoning (Sears & Hunt 1991) and collisions with power lines (Perrins & Sears 1991). The importance of lead poisoning as a cause of death in swans was

Table 1: Major conditions found in 41 Mute Swans

	Group A*	Group B*
No. of birds examined	14	27
Significant parasitism	5	5
Confirmed lead poisoning	8	1
Probable lead poisoning	0	2
Trauma	0	13
Aspergillosis	2	4
Bacterial infections	3	2
Candidiasis	1	1
Visceral gout/kidney failure	1	1
Starvation	0	4

* Multiple diagnoses recorded, therefore the total number of diagnoses exceeds the number of birds examined.

highlighted in two series of post mortem examinations; Brown *et al.* (1992) reported the results of post mortems carried out on Mute Swans (mostly from England) between 1951 and 1989, in which lead poisoning caused the death of 19.7% of the 193 Mute Swans examined. MacDonald *et al.* (1987, 1990) examined the carcases of 133 Mute Swans from Scotland between 1980 and 1987, in which deaths from lead poisoning were 11.3%. Voluntary codes of practice in 1982, followed by legislation in 1987, were introduced to reduce hazards to swans from lead fishing weights in England and Wales, although only the voluntary codes were applicable in Scotland.

Between January 1995 and December 1996, approximately 13 years after the introduction of voluntary codes, the carcases of 41 Mute Swans were examined at S.A.C. Veterinary Science Division, Auchincruive, Ayr, Scotland. The results of these post mortem examinations are discussed, and compared with the findings of the earlier surveys.

Materials and Methods

Origin of birds

Birds were submitted for post mortem examination directly by members of the general public, by Scottish Natural Heritage, or by a local wildlife rehabilitation centre. Fourteen of the birds, referred to as Group A, were received from one site during a five month period (January to June 1996), and were representative of a greater number of dead swans found on that site. This was a relatively small loch with a surface area of about 16 hectares, where the resident population of non-breeding swans had increased in recent years to over 90 birds. The depth of water normally varied between 1.5 and 2 metres, but had been allowed to fall prior to the onset of the mortality problem. During the winter months the flock was heavily dependent on humans for supplementary feeding. The remaining 27 swans, Group B, were received over a two year period from sites on which only sporadic deaths were being

Table 2: Categories of parasites found in 41 Mute Swans

		Group A	Group B	Total
No. of birds examined		14	27	41
No. of birds in which parasites were detected		11	18	29
Tapeworms		1	3	4
Roundworms	- *Amidostomum sp.*	5	9	14
	- *Capillaria sp.*	1	4	5
	- *Echinuria sp.*	0	1	1
Acanthocephalans		6	2	8
Flukes	- Blood	8	10	18
	- Tracheal	7	2	9
	- Intestinal	2	3	5
Coccidia		0	1	1
No. of birds with significant parasitic burdens		5	5	10

Table 3: Birds with significant parasitic burdens

Group A	
Bird 1	Amidostomum, acanthocephalans, tracheal flukes, tapeworms
Bird 2	Amidostomum, acanthocephalans, tracheal flukes
Bird 3	Acanthocephalans, tracheal flukes
Bird 4	Tracheal flukes
Bird 5	Amidostomum, acanthocephalans, tracheal flukes, intestinal flukes
Group B	
Bird 1	Tapeworms, intestinal flukes
Bird 2	Amidostomum, tapeworms
Bird 3	Amidostomum
Bird 4	Tapeworms
Bird 5	Intestinal flukes

reported. All the swans were recovered from sites in central or western Scotland.

Post mortem examination

The post mortem examination was based on that described by Pennycott (1998). A gross examination was made of the major internal organs, and samples collected for parasitology, microbiology, toxicology and histopathology as appropriate. Birds which still retained some brown feathers were considered to be immature, those which were fully white were described as mature.

Results

The major conditions found in the 41 Mute Swans are summarised in **Table 1**. Multiple diagnoses were recorded in some birds, therefore the total number of diagnoses exceeds the number of birds examined. In **Table 5** the conditions considered to be the primary cause of mortality are listed, and compared with the primary causes of mortality found in other surveys.

1 Parasitism

Internal parasites were found in 29 of the 41 swans examined (**Table 2**). Heavy parasitic burdens caused the death of four birds from Group A, and probably contributed to the death of a fifth bird from this group in which the primary cause of death was lead poisoning.

Parasitism was considered to be the primary cause of death of two birds from Group B, and heavy parasitic burdens were present in another three birds from this group, in which the primary cause of death was aspergillosis, trauma and starvation respectively. All ten birds were immature. The parasites found in these ten birds are listed in **Table 3.**

Significant numbers of flukes were present in the trachea (windpipe) of five birds, all from Group A. The flukes were leaf-shaped, measured approximately 13 mm by 4 mm, and were clearly visible in washings from the trachea. Damage caused by these flukes resulted in inflammatory debris in the trachea, and in some birds an airsacculitis was also present, presumably secondary to the parasitic burdens in the trachea. Some birds with tracheal flukes had been seen alive and had exhibited noisy breathing. Representative specimens of the flukes were identified by the Natural History Museum, London as *Orchipedum tracheicola*, a parasite not previously associated with flock problems in Mute Swans in the U.K.

The gizzard worm *Amidostomum*, a thin parasite measuring 10-20 mm in length and 1 mm in diameter, was found in significant numbers in five birds, three from Group A and two from Group B, resulting in degeneration of the thick lining of the gizzard.

Thornyheaded worms or acanthocephalans are cylindrical in shape, white or yellow in colour, and measure 8-16 mm in length. Significant burdens were found in four birds from Group A, in

Table 4: Concentrations of kidney lead (ppm DM)*

	Number of Birds	
Concentration	Group A	Group B
> 100	8	1
11 - 100	1	4
10 and below	5	11

*Lead estimations only carried out on kidney from 30 birds.

which the parasites caused an enteritis, sometimes bloody, and in some birds perforation of the wall of the intestine led to nodule formation and peritonitis. The acanthocephalans were subsequently identified by the Natural History Museum, London as *Polymorphus (Profilicollis) minutus* and *Filicollis anatis*, and were present either as single species or as mixed species.

Moderate to many tapeworms were recovered from the intestine of four birds, one from Group A and three from Group B. They were clearly visible to the naked eye and were present as part of mixed parasitic burdens in three of the four birds. The birds in which the tapeworms were found were thin and had fluid intestinal contents, and tapeworms appeared to be contributing to the birds' problems. The Natural History Museum, London, identified the tapeworms from one bird as *Wardoides nyrocae*.

In three birds burdens of intestinal flukes were considered to have contributed to the deaths of the birds. One bird from Group A had moderate numbers of *Echinoparyphium recurvatum*, and this parasite was also found in a bird from Group B. Unidentified flukes were present in the intestine of another bird from Group B.

Although eggs of blood flukes of the family Schistosomatidae were found in intestinal smears from 18 birds (**Table 2**), they did not appear to be causing any problems. Similarly small numbers of hairworms (*Capillaria* species) in five birds, spirurid worms [*Echinuria (Acuaria) uncinata*] in one bird and coccidial oocysts from the intestine of one bird were not considered to be significant.

2 Lead Poisoning

Lead poisoning was confirmed in nine birds (eight from Group A, one from Group B) on the basis of kidney lead values in excess of 100 parts per million dry matter (ppm DM). Seven of the nine birds were adult, two were immature. Birds with lead poisoning were usually in poor body condition and their proventriculus and oesophagus were often impacted with vegetation. Large lead ledger weights were found in the gizzard of six of the eight birds from Group A, and lead gunshot was present in the bird from Group B with lead poisoning.

In addition to the nine birds with confirmed lead poisoning, a further five birds (one from Group A, four from Group B) had kidney lead levels between 11 and 100 ppm DM (**Table 4**). One bird, with a kidney content of 67 ppm DM, had shown clinical signs of lead poisoning and had been treated accordingly but had subsequently died. At post mortem examination an impacted oesophagus (gullet) and proventriculus (glandular stomach) was present and a few shotgun pellets were found in the gizzard. A second bird (kidney lead level of 23) had also been treated for lead poisoning but was euthanased - post mortem examination revealed an impacted oesophagus with secondary *Candida* (yeast) infection. These two birds, one adult and one immature from Group B, probably also were suffering from lead poisoning but the diagnosis could not be confirmed due to treatment.

Kidney lead levels of 18, 20 and 38 ppm DM were present in birds which died from other causes. These levels probably indicate exposure to lead, but insufficient to cause clinical disease.

3 Trauma

A diagnosis of death from traumatic injuries was made in 13 birds (7 adults, 6 immature), all from Group B. In some instances the cause of the trauma was

Table 5: Primary causes of mortality in Mute Swans

	Brown *et al.* (1992)	MacDonald *et al.* (1987, 1990)	Group A of this study	Group B of this study
Years	1951-1989	1980-1987	1996	1995-1996
No. of birds	193	133	14	27
Mature	66.0%	66.0%	57.0%	52.0%
Immature	34.0%	34.0%	43.0%	48.0%
Heavy parasitic burdens	4.7%	6.0%	28.6%	7.4%
Confirmed lead poisoning	19.7%	11.3%	57.1%	3.7%
Trauma	30.6%	31.6%	0%	48.1%
Aspergillosis	4.7%	7.5%	7.1%	14.8%
Avian Tuberculosis	2.6%	1.5%	0%	0%
Other bacterial diseases	<2.0%	7.5%	7.1%	3.7%
Other/no diagnosis (incl. starvation)	36.0% (approx)	34.6%	0%	22.2%

known, such as collisions with power cables or trees and fatal injuries caused by fishing hooks or fishing line. In other birds the cause of the injuries was not known. Kidney lead analysis was carried out on ten of the birds - all had kidney lead values of 10 ppm DM and below, except for one adult which died following injuries resulting from fishing tackle in the oesophagus, and in which the kidney lead level was 18 ppm DM. No evidence of firearm injuries was found in any of the birds.

4 Aspergillosis

A severe airsacculitis and nodular pneumonia caused by *Aspergillus fumigatus* was found in two swans (one adult, one immature) from Group A, and four birds (three adults, one immature) from Group B. Two of the birds had died after being held on a rehabilitation site for several weeks, in another bird the aspergillosis was most likely secondary to heavy tracheal fluke burdens, but no predisposing factor was found in the remaining three birds.

5 Bacterial infections

Three birds in Group A and two birds in Group B had died from bacterial infections, often secondary to other conditions. An immature swan from Group A with a heavy burden of tracheal flukes had a severe airsacculitis from which *Pasteurella multocida* was isolated. The airsacs were covered by a layer of white inflammatory debris 2-3 cm thick. The remaining four birds (three adults, one immature) died from enteritis associated

with *Clostridium perfringens*, in which the lining of the digestive tract had become necrotic and fissured, eventually being sloughed to form a cast within the intestine. In two of these birds the presence of tangled masses of fishing line in the digestive tract was probably the trigger factor, and the other two birds had been stressed by being admitted to a rehabilitation centre for unknown reasons.

6 Candidiasis

Secondary infection with the yeast *Candida albicans* was found in one bird from each group. In both birds the lining of the oesophagus was thickened and covered by loosely adherent plaques of white material. Both birds had a mass of vegetation impacting the oesophagus and proventriculus. Lead poisoning was confirmed in one bird and suspected in the second bird which had died despite treatment for lead poisoning.

7 Kidney failure/visceral gout

An immature bird from Group A and a mature bird from Group B had extensive deposits of urates (white waste products from the kidneys) under the skin and on internal organs such as liver, kidney and heart. This combination of lesions, referred to as visceral gout, is indicative of kidney failure. In the immature bird it appeared to be secondary to parasitism, in the mature bird there was also severe damage to the gizzard arising from a penetrating fishing hook.

8 Starvation

A diagnosis of starvation was made in four birds (all immature) from Group B. These birds were thin, had no food in the digestive tract, but no pathological lesions were detected to explain these findings. Two of the birds still had their downy plumage, and may have been abandoned by their parents, one immature bird died from starvation during a spell of very cold weather in January, and another immature bird died from starvation in December. Kidney lead content of the latter two birds was under 10 ppm DM.

Discussion

The size of the Great Britain population of Mute Swans appears to have increased by over 70% between 1986 and 1996, and has reached around 26,000 birds (Cranswick *et al.* 1997). One feature of this expansion in numbers has been the increased size of flocks of non-breeding birds in many urban areas, often dependent on members of the public for supplementary feeding in winter. Increased bird density under such unnatural conditions may however prove to be detrimental to the health of the birds on some occasions. A comparison was therefore made between birds from one such high density site (Group A) with birds from sites on which only sporadic mortality was occurring (Group B). The findings were also compared with earlier surveys. Several birds had multiple problems found at post mortem examination (**Table 1**), but for each bird the primary cause of death was identified and the results compared in **Table 5** with Brown *et al.* (1992) and MacDonald *et al.* (1987, 1990).

Parasitism was a significant problem in Group A. More details of the site from which Group A birds originated can be found in Pennycott (1998), but it is likely that parasitism occurred because of increased numbers of swans on the loch, and a drop in water level which allowed access to the intermediate hosts of some

of these parasites. Parasitism was recorded less frequently by Brown *et al.* (1992) in which the major parasitic problem was *Echinuria (Acuaria) uncinata* in young swans, and by MacDonald *et al.* (1987, 1990) in which un-named tapeworms, flukes and acanthocephalans in the intestines were the commonest parasites detected. The present study reinforces the findings of other authors as to the importance in Mute Swans of parasites such as *Amidostomum* (MacNeil 1970), acanthocephalans (Sanford 1978; Meek 1993) and intestinal flukes (Soulsby 1955). It has also highlighted the presence of another parasite, the tracheal fluke *Orchipedum tracheicola* which can clearly become a major problem in Mute Swans on some sites.

The devastating effects of lead poisoning in swans are well known (Sears & Hunt 1991), mostly arising from the intake by swans of anglers' lead fishing weights. In the study by Brown *et al.* (1992), before full implementation of voluntary codes and regulations banning the use of lead fishing weights between 0.06 g and 28.36 g, lead poisoning caused by lead fishing weights or shotgun pellets was confirmed in 19.7% of the Mute Swans. MacDonald *et al.* (1987, 1990) found 11.3% of Mute Swans had lead poisoning, but commented that lead poisoning in their study was most often associated with shotgun pellets. In Group B of the present study, confirmed lead poisoning was at the much lower level of 3.7%, and was caused by lead of gunshot origin, suggesting that in general the activities to control lead poisoning from fishing weights may be succeeding. However, the results from Group A serve as a reminder that lead poisoning can still be a major cause of mortality on some sites in certain circumstances. Six of the eight birds from Group A with lead poisoning had large ledger lead weights, much bigger than the split-shot lead weights often involved in lead poisoning although still within the banned weight range, and of a type seldom used by anglers in recent years. A drop in the water level at this site had occurred, and this may have given the swans access to the weights on the bottom of the loch, resulting in lead poisoning. Given the persistence of such weights in the environment, lead poisoning will continue to be a hazard.

The commonest cause of death recorded in the surveys of Brown *et al.* (1992) and MacDonald *et al.* (1987, 1990) was trauma, and this was also true for the birds in Group B in the present study. No birds in Group A were considered to have died directly from traumatic injuries, but this probably reflects a degree of selection in the carcasses submitted from this site.

Other than parasitic disease, the commonest infectious disease found in the surveys of Brown *et al.* (1992) and MacDonald *et al.* (1987, 1990) was aspergillosis. A similar situation was encountered in the birds from Groups A and B, and Mute Swans are clearly susceptible to this very common fungus.

Avian tuberculosis was diagnosed in small numbers of swans in the surveys by Brown *et al.* (1992) and MacDonald *et al.* (1987, 1990), but not in the current study. The commonest bacterial condition diagnosed in Groups A and B, and also by MacDonald *et al.* (1987, 1990), was disease associated with *Clostridium perfringens*. The post mortem lesions were identical to those described by Wobeser (1997) in migrating wild geese in Canada, associated with an abrupt change of diet. In the birds in the present study there was evidence to suggest that the enteritis followed initial physical damage to the intestine or arose

in birds after admission to a rehabilitation centre.

The commonest primary causes of death in Group B were trauma, aspergillosis and starvation, with smaller numbers of parasitism, lead poisoning and bacterial disease. These were also the main problems diagnosed by Brown *et al.* (1992) and MacDonald *et al.* (1987, 1990). There was therefore broad agreement among the surveys, with the exception of the encouraging drop in the prevalence of lead poisoning in Group B. In contrast to the causes of sporadic deaths in Group B, the main causes of death in Group A, where significant mortality was occurring, were lead poisoning and parasitism.

The overall increase in Mute Swan numbers in Great Britain is encouraging, but brings with it potential problems, especially for birds at high densities in urban situations where they are vulnerable to changes in food availability, to infectious /parasitic diseases, and to the activities of the human population. It is therefore essential that incidents of increased mortality in Mute Swans be thoroughly investigated, and that the health of the Mute Swan population should continue to be monitored in the future.

The surveys described in this paper are useful because they describe and provide information about a range of infectious and non-infectious causes of death of Mute Swans, and will therefore be of value to people working in this field in the future. They can also highlight particular problems on specific sites, such as lead poisoning and parasitism in birds in Group A. However the birds examined in such surveys cannot be considered to be a representative sample of the Mute Swan population as a whole, because of the high degree of bias in the selection of birds for examination. Similar concerns were expressed by Perrins & Sears (1991) when discussing the incidence of swans killed after colliding with power-lines, because swans which hit wires are more likely to be found (for example by repair engineers) and the cause of death reported, than may be the case for birds dying from other reasons.

Nevertheless there would be great merit in designing and conducting a larger nationwide surveillance programme, in which the samples examined were indeed representative of the whole population of Mute Swans. Such a survey could then examine other issues, such as the relationship between different causes of death and swan density, human density, the frequency of overhead cables, the frequency of coarse fishing and the frequency of game shooting. Such a nationwide survey could also be used to build up a bank of frozen tissues from swans, which could then be examined in the future for heavy metals such as lead, for pesticides, and for other parameters as required.

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