

Lead poisoning in Mute Swans, *Cygnus olor*, in England

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Lead poisoning due to the ingestion of anglers' lead weights has been the major cause of death of Mute Swans in England. Various measures have been taken to reduce the problem, including the introduction of legislation prohibiting the sale and use of lead weights used in 'coarse-angling' (fishing for non-game species). This study reports on changes in the incidence of lead poisoning in relation to these measures.

Lead poisoning was monitored by three methods: post-mortem examination of swans found dead, veterinary diagnosis of rescued swans and blood lead analysis of live swans. Dead swans from throughout England were examined at the Sutton Bonington Veterinary Investigation Centre. A more detailed study into lead poisoning amongst the population of swans in the Thames valley in Southern England was initiated in 1979.

The proportion of deaths due to lead poisoning amongst swans from throughout England dropped from 50% in 1980/81 to 40% in the mid-1980s, when alternatives to lead were introduced. In 1987 there was a further reduction to 30% which corresponded to the introduction of the ban on the sale and use of lead weights. The number of cases of lead poisoning amongst swans rescued from the River Thames and adjacent waters has dropped from a peak of 107 in 1984 to 25 in 1988. There has also been an annual reduction in the blood lead levels of swans on the River Thames at Windsor, from a median of 127 µg/100ml in 1984 to 22 µg/100ml in 1987.

Changes in the seasonal pattern of lead poisoning amongst swans in the Thames area during 1987 and 1988 corresponded to predictions made on the basis that swans were ingesting mainly recently used weights. There was no longer a peak in lead poisoning during the summer months and the incidence throughout the year dropped to levels only recorded during the coarse-angling close-season in previous years. Reasons for the continuing occurrence of lead poisoning are discussed.

The first documented cases of lead poisoning in Mute Swans, *Cygnus olor* that were attributed to the ingestion of anglers' lead weights occurred on the River Trent at Nottingham, England in 1973 (Simpson *et al.* 1979). Further investigations into swan mortality throughout England revealed the problem to be widespread amongst swans inhabiting lowland rivers, lakes and ponds (Nature Conservancy Council 1981). Lead poisoning has been implicated in local population declines on the River Avon, Warwickshire in the English Midlands (Hardman & Cooper 1980) and on the River Thames in Southern England (Birkhead & Perrins 1985).

The main source of the lead is split-shot used in coarse angling (fishing for non-game species) which range in size between 1.8 mm and 6.8 mm diameter. They have been found in the gizzards of 77% to 90% of Mute Swans diagnosed as lead-poisoned on autopsy (Simpson *et al.* 1979, Birkhead 1982, Sears

1988). Larger 'leger' weights were found in 50 out of 700 lead poisoned swans examined from throughout England (Hunt 1985). Spent gun-shot, which are the main cause of lead poisoning amongst Whooper swans, *Cygnus cygnus*, in Scotland (Spray & Milne, 1988) and Mute Swans in some areas of Ireland (O'Halloran *et al.* 1988), were responsible for fewer than 2% of cases of lead poisoning amongst swans from England (N.C.C. 1981, Birkhead 1982, Sears 1988). Other sources of lead, such as emissions from car exhausts, contribute to the overall lead burden in swans, but do not account for mortality (Sears 1988).

The incidence of lead-poisoning amongst swans from the Thames valley has varied regionally and seasonally in relation to the pattern of fishing (Sears 1988). The highest incidence occurred in heavily-fished urban areas where large numbers of discarded lead

weights were found on the river banks and in the sediment. There was a distinct seasonal pattern of a peak in lead poisoning in mid-summer and a low during the spring months which appeared to be related to the fishing season. During the coarse-angling close-season from mid-March to mid-June, when fishing is prohibited, the incidence of lead poisoning dropped markedly; despite large numbers of lead weights accumulated on the banks and in the sediment which remained available to swans. The rapid increase in lead-poisoning as fishing re-commenced suggested that the swans were ingesting mainly recently lost weights.

Conservation measures to reduce the problem of lead poisoning were initiated by the Nature Conservancy Council Working Group in late 1981. A voluntary code of practice for anglers encouraging the careful use and disposal of lead weights was introduced in 1982. Several non-toxic alternatives to lead were first marketed during 1984-85, followed by a voluntary ban on the use of lead weights during 1985 and 1986. From 1 January 1987 it has been illegal to sell or import lead weights for fishing of over 0.06 grams (number 8 split-shot) up to and including 28.36 grams (1 ounce leger weights) in England and Wales, under the Control of Pollution (Anglers' Lead Weights) Regulations, 1986. Further to this, the use of the same sizes of lead weights has been prohibited by Water Authorities throughout England and Wales since the summer 1987 (Fisheries Byelaw amendment to the 1975 Salmon and Freshwater Fisheries Act). The aim of this paper is to assess any changes in the incidence of lead poisoning in relation to the measures taken to reduce the problem.

Study area

Carcasses of Mute Swans were collected from throughout England for analysis at the Sutton Bonington Veterinary Investigation Centre. From 1980 up to 1985 initial surveys covered as wide a geographical area as possible. Since 1985 effort has been concentrated on birds from the Midlands and the Thames valley, although swans from other areas were still examined. A detailed investigation into lead poisoning amongst swans inhabiting the Thames valley in Southern England was initiated in 1979 (Birkhead 1982) and continued from 1982 to the present (Sears 1986, 1988, in prep.). The principal area of study was the River Thames between Lechlade, Gloucestershire (51°41'N 1°41'W) and Richmond, Surrey (51°27'N 0°18'W), some of its tributaries and gravel pits within 5 km of the river (Fig. 1). The flock of swans at Windsor on the River Thames (51°29'N



Fig. 1. Map of Southern England showing location of Thames study area between Lechlade and Richmond.

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|-------------------------------|----------------|
| 1. Lechlade, Gloucestershire. | 51°41'N 1°41'W |
| 2. Windsor, Berkshire. | 51°29'N 0°37'W |
| 3. Richmond, Surrey. | 51°27'N 0°18'W |

0°37'W) were regularly caught and sampled for blood lead analysis.

Methods

Dead swans were examined at post-mortem by the method described by Sears (1988). Samples of liver, kidney and bone were analysed for total lead by colorimetry, using dithizone, at the Sutton Bonington V.I. Centre. Diagnosis was made on the basis of elevated tissue lead levels and the signs of lead poisoning, as described by Simpson *et al.* (1979). A kidney lead level of over 100 µg/g dry matter was considered abnormally elevated. Ingested lead pellets and other foreign objects were retrieved from the gizzard grit. Anglers' split-shot lead weights were identified by the presence of a split in intact weights and the flattened discs of eroded weights which had separated into two halves. Leger weights were distinguished by their characteristic shapes.

Sick swans presented for treatment were examined by qualified veterinary surgeons for the clinical signs of lead poisoning (Sears *et al.* 1989). Diagnosis was made on the basis of symptomology and in many cases was confirmed by total blood lead estimation. Blood samples from the brachial or tarsal vein were analysed for total lead, initially by colorimetry but since 1987 by atomic absorption using the method described by Fernandez (1975). Blood lead levels above 40 µg/100ml were considered to indicate an undesirable level of exposure (Sears 1988).

Results

Incidence of lead poisoning

Lead poisoning has been the largest single cause of

Table 1: Incidence of lead poisoning amongst swans from throughout England examined by post-mortem.

Years	Total No. Examined	No. Lead Poisoned	% Lead Poisoned
1980/81	210	105	50%
1983/84	288	115	40%
1985/86	241	93	39%
1987/88	236	70	30%

death amongst Mute Swans in England (Table 1). In 1980/81 it accounted for 50% of the 210 swans examined by autopsy. Since then the incidence of lead poisoning has dropped during two periods. In 1983/84 40% of the 288 swans examined were lead poisoned; a significant reduction when compared with 1980/81 ($\chi^2=4.99$, $df=1$, $P<0.05$) and in 1987/88 the incidence fell to 30% of 236 swans which was significantly lower than in the previous two years ($\chi^2=5.99$, $df=1$, $P<0.05$). Unfortunately it is not valid to compare the total number of cases of lead poisoning between years due to variation in survey effort.

Annual reductions in both the number and percentage of cases of lead poisoning amongst swans rescued within the Thames valley have occurred each year since 1984 (Table 2). The reduction was significant between 1986 and 1987 ($\chi^2=5.867$, $df=1$, $P<0.05$).

Tissue lead levels

The tissue lead levels of swans from the Thames valley diagnosed as lead poisoned are compared for two periods, 1983-86 and 1987-88 (Table 3). The median lead levels of all three tissues were lower in the latter period. There was a very significant reduction in the median

Table 2: Incidence of lead poisoning amongst swans rescued from the Thames valley.

Years	Total No. Examined	No. Lead Poisoned	% Lead Poisoned
1983	144	80	56%
1984	181	107	59%
1985	152	67	44%
1986	137	55	40%
1987	131	28	21%
1988	173	25	15%

Table 3: Tissue lead levels of swans from the Thames area diagnosed as lead poisoned, comparing 1983-86 with 1987-88.

	Tissue lead levels in $\mu\text{g/g}$ dry matter		
	Liver	Kidney	Bone
1983-86 No.	83	83	81
Median	57	439	156
Range	1-404	60-6560	5-1018
1987-88 No.	60	59	60
Median	35	243	137
Range	0.4-293	27-1888	7-2776
Mann-Whitney U-Test, P=	>0.05	0.001	>0.05

kidney lead level from 439 $\mu\text{g/g}$ dry matter to 243 $\mu\text{g/g}$ dry matter (Mann Whitney, $P=0.001$).

Rate of ingestion of lead

A total of 521 pieces of lead were retrieved from the gizzards of 118 lead poisoned swans from the Thames area examined by autopsy between 1983 and 1988. These were identified as anglers' lead weights in all but one case. The exception was a swan which had ingested 125 gun-shot from a clay pigeon shoot. The rate of ingestion is compared for two periods; 1983 to end May 1987, prior to the ban on the use of lead weights, and June 1987-end 1988 when the use of lead weights was prohibited (Table 4). The percentage of lead poisoned swans with ingested lead weights dropped from 75% to 64%

Table 4. The gizzard contents of lead poisoned swans from the Thames, comparing two periods; before and after the ban on the use of lead weights.

	1983- end May 1987	June 1987- 1988
No. lead poisoned swans autopsied	93	25
No. swans with ingested Pb gunshot (percentage)	1 1%	0 —
No. swans with ingested Pb weights (percentage)	70 75%	16 64%
Mean no. pieces of Pb split-shot \pm sd	5.7 ± 5.35	7.63 ± 6.25
Range	1 to 30	1 to 26

Table 5: Blood lead levels of immature swans in the Windsor flock.

Year	Blood lead levels in µg/100ml			
	Number	Median	Range	%>40 µg/100ml
1983	44	107	10 - 2495	84%
1984	46	127	3 - 2116	87%
1985	35	97	25 - 996	89%
1986	56	63	20 - 692	89%
1987	108	22	1 - 635	44%
1988	119	25	3 - 729	24%

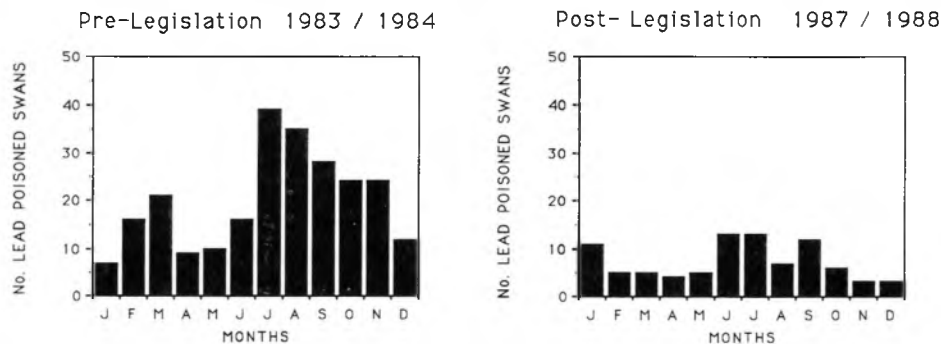


Fig.2. Monthly distribution of lead poisoning amongst swans found dead or rescued from the Thames area comparing 1983-84 and 1987-88. (Monthly data for each two year period have been combined.)

in the second period. There was an unexpected, although not significant, increase in the average number of pieces of lead per swan from 5.70 (+/- 5.35 sd) to 7.63 (+/- 7.63 sd) during this time.

Blood lead levels

The blood lead levels of immature and non-breeding swans of mainly 1-4 years old which were sampled throughout each year in an urban flock at Windsor on the River Thames are compared for the years 1983 to 1988 (Table 5). Repeat results for swans sampled more than once and the results for swans recently released after treatment have been discounted. There has

been an annual reduction in the median blood lead level from 127 µg/100ml in 1984 to 22 µg/100ml in 1987 with significant reductions occurring between 1985 and 1986 (Mann Whitney, $P=0.002$) and particularly between 1986 and 1987 (Mann Whitney, $P<0.001$). The percentage of swans with elevated blood lead levels (above 40 µg/100ml) was reduced by half between 1986 and 1987 ($\chi^2=30.835$, $df=1$, $P<0.001$) and fell again between 1987 and 1988 ($\chi^2=10.179$, $df=1$, $P<0.01$) (Table 5).

Seasonal pattern of lead poisoning

The seasonal pattern of lead poisoning amongst

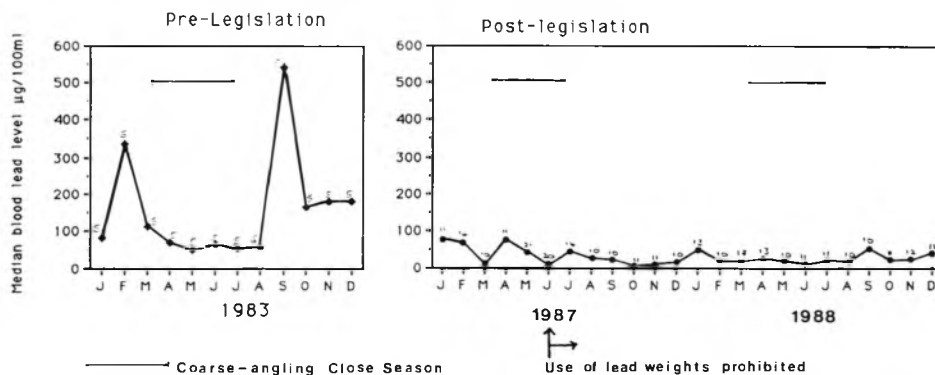


Fig. 3. Monthly median blood lead levels of immature swans in the urban flock on the River Thames at Windsor, comparing 1983 and 1987-88. (Sample sizes given above points).

the swans found dead or rescued from the Thames valley is compared for the years 1983-84 and 1987-88 (Fig. 2). (Monthly data for each two year period have been combined). There was a more even monthly distribution of cases of lead poisoning during 1987-88. In particular the summer peak of lead poisoning during July to September has been reduced.

The monthly median blood lead levels of samples of between 5-20 immature swans caught in the flock at Windsor are compared for 1983 and 1987-1988 (Fig. 3). There has been a significant change in the seasonal pattern (comparing 1983 and 1987, Kolmogorov-Smirnov, $D=0.428$, $P<0.001$). The summer peak in blood lead levels was not apparent in 1987 or 1988. Since May 1987 all median blood lead levels were below $50 \mu\text{g}/100\text{ml}$ (with the exception of $53 \mu\text{g}/100\text{ml}$ in Sept. 1988).

Discussion

Significant decreases in the proportion of swan deaths due to lead poisoning in England have occurred as efforts have been made to reduce the problem. Within the Thames valley the most significant reduction in lead poisoning occurred between 1986 and 1987 when the ban on the sale and use of lead weights was introduced.

Once lead weights were no longer in use it was predicted that there would be a rapid decrease in the incidence of lead poisoning, on the basis that swans from the Thames appeared to be ingesting mainly recently lost weights (Sears 1988). Conditions should resemble those of previous close-seasons, when large numbers of lead weights remained accessible to swans and yet there was a marked seasonal drop in the incidence of lead poisoning. It was expected that the number of cases of lead poisoning per month and the average monthly blood lead levels would fall to levels previously recorded during the close-seasons.

These predictions have held true. In 1983-86 during the close-season months of April and May there was an average of 4.4 (± 2.5 sd) cases of lead poisoning per month amongst the swans found dead or rescued from the Thames area, whereas during the other ten months the average was 8.93 (± 5.3 sd). During the whole of 1987-88 the average incidence had dropped to 3.7 (± 2.7 sd) cases per month. Similarly the monthly median blood lead levels of swans in the Windsor flock dropped to below $50 \mu\text{g}/100\text{ml}$ from June 1987 onwards whereas in previous years such levels had only been re-

corded during the close-seasons.

The introduction of legislation against the sale and use of lead weights in England and Wales has not totally eradicated the problem of lead poisoning however. Lead was still responsible for around 30% of swans found dead throughout England examined by autopsy during 1987-88. Chronic lead poisoning as a result of previous exposure to lead may partly be responsible. It is difficult to categorise cases of lead poisoning as chronic or acute since they form a continuum. There has been an increase in the proportion of swans diagnosed as lead poisoned which have not had lead pellets in their gizzards at post-mortem from 25% in 1983-end May 1987 up to 36% in June 1987-end 1988. Some of these birds may have recently voided ingested pellets prior to their death but others may have been suffering chronic lead poisoning as a result of previous ingestion of lead weights.

Specific incidences of lead poisoning can sometimes be attributed to the ingestion of old lead weights which have accumulated on the beds of rivers and lakes. If these become exposed, for example when the water level is lowered during periods of dry weather or due to dredging activity, swans may then ingest them with or instead of grit. Large numbers of lead weights are known to have accumulated on the bed of the River Thames (Sears 1988). There has been no reduction in the average densities of lead weights found in sediment samples taken every alternate year from 1983 to 1989 (Sears in prep). Since most of these weights are individually scattered throughout the sediment it seems likely that if swans accidentally ingested them with their grit they would only pick up one or two weights at a time. If there were no other source of lead weights one would expect a decrease in the average number of pellets found in the gizzards of lead poisoned swans. Instead there has been an increase in the average number per lead poisoned swan from 5.7 in 1983-May 1987 up to 7.6 in June 1987-1988. This suggests that many swans are still ingesting several weights at one time, possibly as groups of weights attached to line.

Leger weights of over 28.36g (1oz) may still be legally used but are more suitable for sea fishing than coarse-angling and have not been found in swans' gizzards. Small lead 'dust-shot', of 2.2 mm diameter (0.06 grams) and under, also remain legal. If sufficient quantity were ingested at one time and were retained in the gizzard they would cause lead poisoning. Although the original size of weights cannot be determined from the size of pellets retrieved

from swans' gizzards due to the variable amount of erosion since ingestion, it can be assumed that the original weights would have been as large or larger than the remaining pieces of lead. On this assumption, over 97% of the lead weights found in the gizzards of swans from the Thames area which died during 1988 were within the illegal size range (Sears 1989).

To conclude, recent measures to control the use of lead weights appear to have been suc-

cessful in reducing the incidence of lead poisoning, particularly within the Thames area. The problem is unlikely to disappear completely while there are still lead weights accessible for swans to ingest and is likely to re-occur during periods of drought. However, the number of lead weights ingested by swans should decrease with time as old weights gradually sink out of reach and any illegal use of lead diminishes as stocks are used up.

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