

Lead poisoning in Mute Swans *Cygnus olor* in Ireland: a review

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Lead toxicity in Mute Swans in Ireland was investigated, firstly by examination of dead swans and secondly by blood sampling live birds. Post mortem examination showed that almost 70% of all Mute Swans examined died from lead poisoning. Two sources of ingested lead resulting in fatalities were identified: spent gunshot and lost or discarded anglers' lead weights. Tissue lead levels were investigated in all dead birds to determine any sub-lethal effects of lead. Three categories of Mute Swan mortality were identified: (1) acute lead poisoning (2) collisions and (3) other reasons. In most cases, lead concentrations in the tissues were highest according to the cause of death in order: lead poisoning > collision > other causes. Blood samples were collected from swans at six sites and the degree of lead contamination established. Over forty per cent of blood samples from one site showed elevated lead. X-ray examination of live swans revealed the source of contamination to be ingested lead pellets. Urban birds were shown to have higher lead levels than rural birds. The blood levels of rural birds were presumed to reflect background levels. Biochemical and haematological aspects of swan blood were also investigated. Reference biochemical and haematological values for 'normal' healthy swans were compared with those which had been contaminated with excess lead.

Avian lead poisoning was first recognised in ducks over 100 years ago (Philips & Lincoln 1930), and has now been recorded in upland game birds (Reiser & Temple 1981), in birds of prey (Wilcove & May 1986) and swans. One of the first reported cases of lead poisoning in swans was that of 13 Tundra Swans *Cygnus columbianus* (Ord) which died from ingesting vegetation contaminated from mining activities (Benson *et al.* 1976). Simpson *et al.* (1979) were one of the first group of workers to identify lead poisoning from anglers' lead weights as a cause of mortality in Mute Swans *C. olor*, (Gmelin). Since then, lead poisoning in Mute Swans has been attributed by many workers (e.g. Anon 1981, Birkhead 1982, O'Halloran *et al.* 1988a, Sears 1988) to the ingestion of anglers' weights. In addition to ingesting discarded anglers' lead weights, Mute Swans have also died from ingesting spent gunshot (Clausen & Wolstrup 1979, O'Halloran *et al.* 1988a).

In Ireland, there are approximately 90,500 people (1987 data) with game endorsements to their shot-gun licences (Department of Justice, pers. comm.). The quantity of lead discharged into the environment is therefore considerable. It is estimated that there are 9,000 resident

coarse-fish anglers, with an additional 16,380 visiting the state each year (Anon 1986). The first record known to us of lead poisoning in wildbirds in Ireland was in 1962, when two Shelduck *Tadorna tadorna* (L) died at Cork Lough. Since then a detailed study has been carried out to investigate fatal lead poisoning and sub-lethal effects of lead on swans in Ireland (O'Halloran 1987a). This paper reviews current knowledge of lead poisoning in Irish Mute Swans.

Study Area and Methods

Study Area

Dead Mute Swans and blood samples from live Mute Swans were collected at six sites in Cork, Galway, Belfast, Dublin, Clare and Kerry (Fig. 1, Table 1) between December 1984 and November 1986. The principal sampling site was a Lough in the western suburbs of Cork city (51°52'N 08°29'W). This fifteen acre freshwater lake is a refuge for a number of wildfowl species including Canada Geese *Branta canadensis* and up to 160 Mute Swans. The



Fig. 1. Map of Ireland indicating the main areas of sampling. 1 - Cork Lough, 2 - Malahide, Co. Dublin, 3 - The Waterworks, Belfast, 4 - Lough Neagh, 5 - Galway Harbour, 6 - Shannon Lagoon, Co. Clare and 7 - Lough Leane, Co. Kerry.

maximum depth is 1.5M in the northern basin. The area is in an internationally renowned coarse angling site. Two other urban sites were selected, Antrim Road Waterworks, Belfast and Galway Harbour (Table 1). The other three rural sites were selected to represent areas with low and presumably background levels of lead (Table 1).

Collection and treatment of corpses and blood samples

Corpses were collected, cause of death recorded and tissue lead levels determined (O'Halloran *et al.* 1989). Blood samples were collected by brachial venipuncture and analysed for lead, protoporphyrin, haemoglobin and other blood constituents. For details see O'Halloran *et al.* (1987b, 1988a, 1988b, 1988c). Mute Swans were X-rayed on a CGR Unimax 500 X-ray machine.

Classification of lead poisoned birds

In dead swans, lead levels greater than 31.25 µg/g (wet matter) in the kidney and 12.5 µg Pb/g (WM) in the liver were considered diagnostic of lead poisoning following Clarke & Clarke (1975). Swans with a blood lead level in excess of 3.00 µg Pb/gHb were considered to have exceeded the maximum tolerable limit of lead [equivalent to 2.0 µmoles/l or 40 µg/100ml following Simpson *et al.* (1979)]. This criterion was found by O'Halloran *et al.* (1988b) to be more reliable measure of excess lead exposure.

Environmental lead

At Cork Lough, the quantity of discarded lead weights was estimated by sampling four square metres of the lake bed. Samples were examined using X-ray to detect any lead pellets in the

Table 1. Location and number of live Mute Swans blood sampled and dead Mute Swans collected from October 1984 to April 1987.

Location	Irish Grid Reference	No. live birds blood sampled	No. dead swans examined	Source of Lead contamination
Cork Lough	W665704	870	41	High airborne-lead* and anglish lead Shooting 100 years ago
Lough Neagh	H901661	0	49	Low airborn lead claypigeon shooting
Antrim Road, Belfast	J325775	16	3	High airborne lead angling in the catchment of swans
Galway	M297249	13	0	High airborne lead
Shannon Lagoon	R375601	10	2	Low airborne lead
Malahide	O195475	53	4	Low airborne lead
Lough Leane	V930880	9	0	Low airborne lead
Others		0	2	—

*Airborne lead estimates based on traffic density (after Cabot, 1985).

Table 2. Proportion of lead poisoned swans from those examined from October 1984 to April 1987, with details of other causes of mortality. (from O'Halloran *et al.*, (1988a), with permission from the Zoological Society of London.)

	Cork	Dublin	Location Belfast	Lough Neagh	Others ¹	Total
No. Examined	41	4	3	29 (20) ²	4	101
No. Lead Poisoned	17	0	3	29 (20)	0	69

¹Others = Co. Clare (2), Co. Limerick (1), Co. Mayo (1). ²Prior to this date 20 Mute Swans were lead poisoned at Lough Neagh in 1980 which are also included. Other causes of mortality include hypothermia (6), collisions (9), oiled (3), shot (2), infection (1), choked (1), killed by another swan (1) and unknown (9).

sediment (after Milne & Ramsay 1987). At Lough Neagh, grab and core samples were taken of the lake bed in a region where birds had been found dead, to establish the distribution and density of lead pellets.

Results

Post Mortem examination of dead birds

A total of 101 dead Mute Swans were examined, mainly from Cork Lough and Lough Neagh (Table 2). Of all birds, 68% had tissue lead levels diagnostic of lead poisoning. The birds at Cork Lough had ingested anglers' lead weights while those from Lough Neagh had ingested shotgun pellets. In some instances up to 100 spent shotgun pellets were found in a single gizzard. The pathological features at post mortem were generally similar to those described by other workers (e.g. Simpson *et al.* 1979).

Tissue lead levels for Mute Swans which had

died of lead poisoning, collisions and of natural causes were compared (Table 3). Tissue lead levels in lead poisoned birds were similar to reported levels for acutely poisoned birds. Median lead levels found in birds which had collided were intermediate between those that died from lead poisoning and those that died from natural causes.

Lead concentration in blood

Blood lead levels were related to the degree of exposure to lead, and were significantly lower ($P < 0.001$ Mann Whitney U Test) at 'rural' sites, reflecting the lower background levels of lead. At these sites, 4.5% of blood samples had elevated lead ($> 3.00 \mu\text{gPb/gHb.}$), with 82% of samples less than $2.00 \mu\text{gPb/gHb.}$ (Fig. 2). Blood samples collected at urban sites had increased lead levels, particularly in the group $2.00\text{--}3.00 \mu\text{gPb/gHb.}$ It is presumed that at least one of these urban birds had ingested lead weights since, when collected dead a few months later, ingested lead pellets

Table 3. Numbers of ingested lead weights and median lead levels ($\mu\text{g/g}$) wet matter for three categories of Mute Swans *Cygnus olor*. Group A: Mute Swans which died from acute lead poisoning; Group B: Mute Swans which died from collisions and Group C: Mute Swans which died from other causes. Parenthesis include range of values. (from O'Halloran *et al.* 1989 with permission from the Zoological Society of London.)

Category	n	A	n	B	n	C
No. Pellets		7		0		0
Kidney	10	113.00 (40.00-305.00)	6	28.24 (20.50-60.00)	20	8.70 (0.40-19.00)
Liver	8	315.00 (93.15-450.00)	7	33.00 (12.50-194.00)	17	8.00 (1.00-14.00)
Pancreas	7	67.00 (20.00-155.00)	6	21.0 (9.20-97.00)	18	11.00 (1.20-48.00)
Heart Muscle	7	14.50 (6.00-730.00)	4	12.10 (5.20-41.30)	17	6.50 (1.70-17.00)
Gizzard Muscle	9	13.00 (6.00-85.00)	6	12.80 (5.60-43.00)	20	6.80 (0.50-19.00)
Breast Muscle	7	19.00 (8.00-273.00)	5	18.20 (16.00-188.00)	13	9.00 (0.50-15.00)

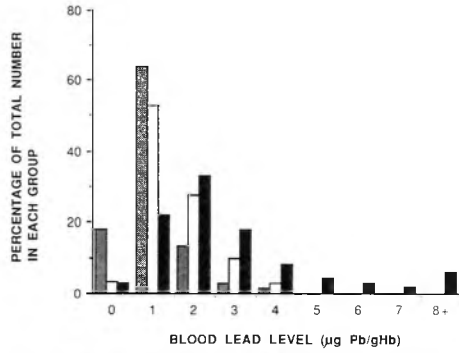


Fig. 2. Blood lead levels (µgPb/gHb) of swans in rural sites (n=72), Urban sites (n=29) and The Lough (n=870). (from O'Halloran *et al.* (1988a) with permission from the Royal Society of London.

were found in the gizzard and it had a blood lead value of 62.00 µgPb/gHb.

At Cork Lough, blood lead values differed ($P < 0.001$ Mann Whitney U Test) from those found at other sites with 44% of Mute Swans having elevated lead. There was no difference

Table 4. Eleven (of 41) X-rayed swans with lead pellets in their bodies. (from O'Halloran *et al.* (1988a) with permission of the Zoological Society of London).

Shot-in		Ingested	
No. of Pellets	Blood lead µmoles/l	No. of Pellets	Blood lead µmoles/l
4	2.60	1	2.50
5	2.00	1	3.60
6	1.45	1	4.84
		1	5.00
		1	9.50
		3	2.60
		7	40.00
		11	80.73

in the proportion of birds with elevated lead from different sex or age classes so the seasonal pattern for the whole population at Cork Lough is presented (Fig. 3). The proportion of swans with elevated lead levels (> 3.00 µgPb/gHb.) was highest in winter (63% and 66%) and lowest in summer (31% and 31%). The median blood lead levels were highest in the winter

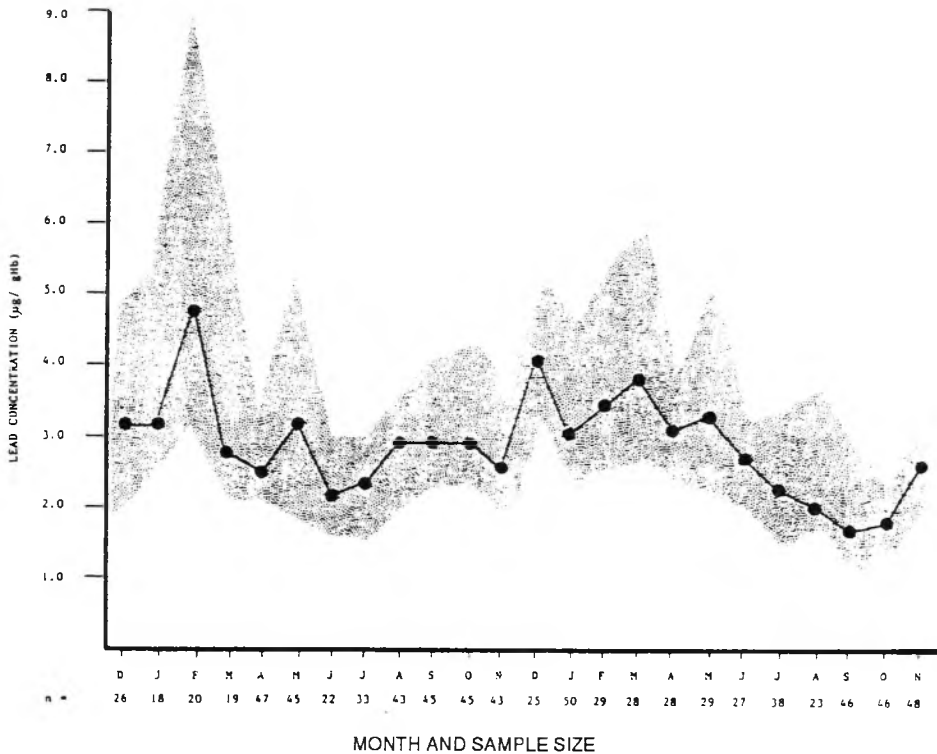


Fig. 3. Median blood lead levels (corrected for haemoglobin) in Mute Swans at Cork Lough over a two year period. Shaded area indicates the 25% percentile above and below the median line (from O'Halloran *et al.* (1988b), with permission from Elsevier Applied Sciences Publishers.

and spring months (Fig. 3). Forty one birds were X-rayed, of which 20% had lead weights in their digestive tracts. The blood lead level varied depending on the number of lead pellets ingested (Table 4), although two birds which were positive for lead on X-ray, showed no increase in blood lead and eight birds which had no lead weights in their gizzards had elevated blood lead ranging from 3.10-5.20 $\mu\text{gPb/gHb}$. Five of the eight swans previously had elevated blood levels indicating chronic lead poisoning.

Blood chemistry and haematology

All blood samples included in the reference population were from apparently healthy Mute Swans. Their blood lead values ranged between 0.46-3.00 $\mu\text{gPb/gHb}$. [equivalent to 0.30-2.2 $\mu\text{moles/l}$], assuming a mean haemoglobin concentration of 13.70gHb/100mls. Reference limits for blood chemistries and haematology are described in detail elsewhere (O'Halloran *et al.* 1988c). Abnormalities in blood chemistry and haematology in lead poisoned swans were identified by comparison with this reference distribution. In summary, all birds had high blood lead levels and were clearly suffering from hypochromic anaemia. Changes in blood chemistry were variable in lead poisoned birds, with eight biochemical variables showing deviations from the reference range (O'Halloran *et al.* 1988c).

Environmental lead

Discarded anglers' lead weights were only found at one sampling site at Cork Lough where five pellets were found (0.02/m²). At Lough Neagh, Co. Antrim, spent gunshot pellets were found along 100m of the lake shore in front of a clay pigeon shooting site and on the lake bed up to 60m from the shore. The pellet density in the top 5cm was 2,400 pellets/m². Numbers of coarse-fish anglers at Cork Lough using lead weights varied with a peak in summer (July-August), and a maximum of 12 rods per day.

Discussion

Lead poisoning in Mute Swans in Ireland was investigated for the first time between October 1983 and November 1986. Though Mute Swans were sampled from many parts of the country (Table 1) it is important to emphasise that detailed knowledge is only available for three sites

namely: Cork Lough, Lough Neagh, Co. Antrim and Malahide, Co. Dublin. The population of Mute Swans in Ireland numbers approximately 5,000-6,000 birds (Hutchinson 1979) thus current studies only give details of a proportion of Irish Mute Swans.

Of the dead Mute Swans examined in Ireland, 68% died from lead poisoning. Three Whooper Swans *C. cygnus* (L.) were also recorded as lead induced mortalities (O'Halloran *et al.* 1988a). Almost all these birds came from Lough Neagh, Co. Antrim and Cork Lough (Table 1). At Cork Lough and Wills' pond Belfast ingestion of anglers' weights was the source of lead poisoning. Anglers' lead weights were identified from gizzards by comparing lead signatures in corpses at post mortem, on X-rays of live birds and also found in the lake sediments. By contrast, at Lough Neagh lead poisoning resulted exclusively from the ingestion of spent gunshot pellets at a clay-pigeon shooting site. Some of the swans had up to 100 gunshot pellets in their gizzards, and this related to density of pellets (2,500/m²) found along the shore. Pathological findings were similar to those found in other studies, except some birds showed impaction beyond the gizzard to the duodenum.

In contrast to studies on the River Thames by Birkhead (1982) and Sears (1988), the peak of lead induced mortality and blood lead levels in Ireland is in winter (Fig. 3). The main angling season at Cork Lough is the same as that on the River Thames and the cause of lead related mortality and elevated lead at Cork Lough is due to ingestion of lead weights as shown by radiology (O'Halloran *et al.* 1988a). A number of factors may be responsible for the different pattern of lead related mortality and blood lead levels at Cork Lough; first, the number of birds present is greater in winter; secondly poor natural feeding may force the swans to forage more and thus ingest lead weights; and thirdly, because of the poor diet in winter, when the birds feed almost exclusively on bread, their susceptibility to absorption of lead may be greater. Trost (1981), also noted a different pattern of lead absorption in captive ducks fed on different experimental diets and grit types, though none of these diets were of bread. However, French (1984) in his study in East Anglia also found a similar pattern to that of Cork Lough.

Though a large number of the swans examined (68%) apparently died directly from lead poisoning others may well have died due to sub-lethal effects. In Irish studies, tissue lead levels of swans which had collided with objects were elevated (O'Halloran *et al.* 1989). Though the

median tissue lead levels in swans following collision (Group B, Table 3) were not diagnostic of acute lead poisoning, they were higher than those recorded for swans dying of natural causes. This is contrast to the findings of Sears (1986) where swans on the Thames area, which collided with power lines, had lower median liver and kidney lead levels than those which died of all other causes, excluding lead. The way lead exerts its effect on the nervous system is poorly understood, but it may be mediated through primary vascular damage (Christian & Tryphonas 1971); direct action on neurones (Bouldin *et al.* 1975) or alterations in porphyrin metabolism (Pentschew & Garro 1966). O'Halloran *et al.* (1989) found anaemia and high tissue lead levels in swans that had collided with objects. These sub-lethal effects may have been responsible for reduced co-ordination and the subsequent collisions in these birds.

The blood lead levels recorded in Irish studies have shown that it is a very sensitive measure of lead contamination. The level of lead found in 'rural' birds reflect background levels of lead, whilst those from urban areas indicate the amount of lead in the environment due primarily to exhaust emissions (Fig. 2). However, Mute

Swan flocks are not sedentary, except at times of full primary moult, and this must be considered when interpreting blood lead values. In Ireland, O'Halloran & Collins (1985) have shown that ringed Mute Swans may travel up to 100km. It is not surprising therefore that some rural birds may come into contact with discarded lead which, in Ireland, is most likely to be spent gunshot. At Cork Lough almost 90% of the Mute Swans in the catchment of the study site (Fig. 1) were blood sampled. Of these 42% had elevated lead due to ingestion of lead weights. Blood chemistry and haematology have also been useful in the study of lead poisoning in Irish Mute Swans. While the documentation of blood lead values is important, it is essential to know what sub-lethal effects lead may be having on the swan population. However, before any changes can be detected it is necessary to know the 'normal' blood chemistry for Mute Swans. Present studies have attempted to define 'normal' reference chemistry and haematology for Mute Swans (O'Halloran, 1988c) against which sub-lethal effects could be detected. It is important that such research on sub-lethal effects should continue to investigate the toxicological effects of lead on swans.

References

- Anon. 1981. *Lead poisoning in swans*. Report of Nature Conservancy Council.
- Anon. 1986. *Inland fisheries, strategies for management and development*. Central Fisheries Board of Ireland, Dublin.
- Benson, W. W., Brock, D. W., Gabrica, J. & Loomis, M. 1976. Swan mortality due to certain heavy metals in Mission Lake area Idaho. *Bull. Envir. Contam. Toxicol.* 15:171-174.
- Birkhead, M. 1982. Causes of mortality in the Mute Swan *Cygnus olor* on the River Thames. *J. Zool. Lond.* 198:15-25.
- Birkhead, M. 1983. Lead levels in the blood of Mute Swans *Cygnus olor* on the River Thames. *J. Zool. Lond.* 199:59-73.
- Bouldin, T. W. Muschak, P. O'Tuama, A. & Krigman, M. R. 1975. Blood brain barrier dysfunction in acute lead encephalopathy: a reappraisal. *Envir. Hlth. Perspect.* 12:81-88.
- Christian, R. G. & Tryphonas, L. 1971. Lead poisoning in cattle: brain lesions and haematologic changes. *Am. J. Vet. Res.* 32:203-216.
- Cabot, D. 1985. The state of the environment. Dublin. *An Foras Forbatha*.
- Clarke, E. G. & Clarke, M. L. 1975. *Veterinary Toxicology*. 3rd Edition. Bailliere, Tindall, London.
- Clausen, B. & Wolstrup, C. 1979. Lead poisoning in game from Denmark. *Danish Rev. Game. biol.* 1-22.
- French, M. C. 1984. Lead poisoning in Mute Swans: an East Anglian Survey. In: *Metals in animals*: 25-29. D. Osborn, (Ed). I.T.E. Symposium No. 12 I.T.E., Abbots Ripton.
- Hutchinson, C. D. 1979. *Ireland's Wetlands and their Birds*. I.W.C., Dublin.
- Milne, H. & Ramsay, N. F. 1987. The use of X-ray for quantifying lead shot pellets in substrate samples. *Wildfowl* 38:150-152.
- O'Halloran, J. 1987a. Lead toxicity in Mute Swans *Cygnus olor* (Gmelin). Unpublished PhD. thesis. National University of Ireland.
- O'Halloran, J. & Collins, R. 1985. Preliminary analysis of ringing Mute Swans in Ireland. *Irish birds* 4:85-89.

- O'Halloran, J. & Duggan P. F. 1984. Lead levels in Mute Swans in Cork. *Irish Birds* 2:501-514.
- O'Halloran, J., Duggan, P. F. & Myers, A. A. 1987. Determination of haemoglobin in birds by a modified alkaline haematin (D-575) method. *Comp. Biochem. Physiol.* 86B. 710-704.
- O'Halloran, J., Duggan, P. F. & Myers A. A. 1988c. Biochemical and haematological values for Mute Swans (*Cygnus olor*): effects of acute lead poisoning. *Avian Path.* 17:667-678.
- O'Halloran, J., Myers, A. A. & Duggan, P. F. 1988a. Lead poisoning in swans and sources of contamination in Ireland. *J. Zool. Lond.* 216:211-223.
- O'Halloran, J., Myers, A. A. & Duggan, P. F. 1988b. Blood lead levels and free red blood cell protoporphyrin as a measure of lead exposure in Mute Swans. *Environ. Poll.* 52:19-38.
- O'Halloran, J., Myers, A. A. & Duggan, P. F. 1989. Some sub-lethal effects of lead on Mute Swans *Cygnus olor*. *J. Zool. Lond.* 218:627-632.
- Philips, J. C. & Lincoln, F. C. 1930. American Waterfowl. cited in Bellrose, F. C. (1959). *Bull of Illinois. Hist. Surv.* 27:235-288.
- Reiser, M. H. & Temple, S. A. 1981. Effects of chronic lead ingestion on birds of prey. *Recent advances in the study of raptor diseases*. J. E. Cooper & A. G. Greenwood (eds.), Chiron Publications, Amsterdam.
- Sears, J. 1986. A study of Mute Swans *Cygnus olor* in relation to lead poisoning. Unpublished D.Phil. thesis, University of Oxford.
- Sears, J. 1988. Regional and seasonal variation in lead poisoning in the Mute Swan, *Cygnus olor*, in relation to the distribution of lead and lead weights in the Thames area, England. *Biol. Cons.* 46:115-134.
- Simpson, V. R., Hunt, A. E. & French, M. C. 1979. Chronic lead poisoning in a herd of Mute Swans. *Environ. Poll.* 18:187-202.
- Sturkie, P. D., 1965. *Avian Physiology*. 2nd Edition. Bailliere, Tindall and Cassell, London.
- Trost, R. E. 1981. Dynamics of grit selection and retention in captive Mallard. *J. Wildl. Manage.* 45:64-73.
- Wilcove, D. S. & May, R. M. 1986. The fate of the California Condor. *Nature Lond.* 319:16.

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