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HEAVY METALS IN TISSUES OF THE MUTE SWAN (CYGNUS OLOR)

By

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FRANK, A. and K. BORG: *Heavy metals in tissues of the mute swan (Cygnus olor)*. Acta vet. scand. 1979, *20*, 447—465. — During the period 1973—1977, a number of mute swans (Cygnus olor) were submitted to the National Veterinary Institute in Stockholm and examined post mortem. Organ tissues from 58 swans were chemically analysed for concentrations of lead, cadmium, copper, zinc, iron and manganese. In 10 swans (17 %), elevated residues of lead were found indicating intoxication. Cadmium concentrations were significantly higher in adult than in juvenile swans; also in swans from the Swedish east coast compared with specimens from the west coast. Remarkably high copper concentrations were often found, with one exception yet without any signs of intoxication. High zinc and cadmium concentrations were often found in the same swans. The findings of iron and manganese were not remarkable.

mute swan; lead; cadmium; copper; zinc; iron; manganese.

Since several years, systematic research on diseases and intoxications in Swedish wildlife is performed at the National Veterinary Institute (SVA) in Stockholm (*Borg et al.* 1969, *Borg* 1975 a). For pollution of the aquatic environment as regards heavy metals and other pollutants swans and eider-ducks appeared to be excellent indicator organisms (*Frank* 1976 a). Owing to different diets and perhaps also other conditions, the accumulation pattern is not identical within the two bird species.

During the period 1958—1977, chemical analyses of varying kinds were performed on tissues from about 100 mute swans (Cygnus olor Gmel.) 58 of which are included in this presentation, and about 10 whooper swans (Cygnus cygnus L.). Mercury concentrations in liver tissue from the mute swans were low and with a few exceptions below 2 mg/kg (unpublished):

$$n = 30; \quad r = 0.017—5.44 \text{ mg/kg}; \quad \bar{x} = 0.79 \text{ mg/kg}.$$

A number of analyses concerning residues of DDT and its metabolites, of lindane and PCBs in liver and kidney tissues were performed as well. Low concentrations were found throughout, or as a maximum for sum DDT 0.45 mg/kg, for lindane 0.1 mg/kg, and for PCBs 0.2 mg/kg.

In addition, a number of whooper swans were examined for the presence of the substances mentioned above. The concentrations of these were also low in these swans. The results of the eider-duck (Somateria mollissima L.) examinations will be given in a future paper.

The mute swan is a rather pronounced herbivorous bird and feeds primarily on aquatic plants (Zostera, Chara, Ulva, Myriophyllum, etc). To a lesser extent animal food is ingested, such as frogs, fish and insects. Samples of as well vegetable as animal food were collected at the feeding places of the swans and analysed. Only small residues of mercury, organochlorine pesticides and PCBs were found.

In the swans mentioned, also the presence of other heavy metals than mercury were looked for, viz. lead and copper. Often high concentrations of these metals were found.

The findings initiated more systematic examinations concerning the burden of heavy metals. Since 1973, a new analytical method has been worked out (*Frank*, to be published, a) and applied and this made a more comprehensive analytical activity possible. These analyses concerned lead, cadmium, copper, zinc, iron and manganese.

MATERIALS AND METHODS

Animals examined

Mute swans and whooper swans are totally protected against hunting in Sweden. The investigation material of swans submitted to SVA thus almost exclusively consists of birds, found dead. However, a few swans killed because of weakness are included.

A complete post-mortem examination was performed and tissue samples secured for chemical analysis. As renal cortex and marrow are not separated anatomically in birds, kidney samples consisted of total kidney tissue. During the period 1973—1977, chemical analyses of organ tissues concerning the six metals mentioned, were performed on 58 of the mute swans submitted.

The swans were sexed by inspection of the sex glands. Juvenile and adult birds were differentiated according to the plumage. Young swans are carrying a greyish brown plumage which is changed into an all white plumage at an age of a little more than one year. Later it is very difficult to determine the age.

Sex and age in the 58 mute swans examined were as presented in Table 1.

Table 1. Cadmium concentrations in kidney tissues of mute swans. Grouping of 58 mute swans was performed according to sex, age, season and geographic area. Distribution of age is shown in each group.

Swans of different sexes, ages, seasons and locations	Number	Age			Cd, mg/kg wet weight	
		juv.	ad.	-	range	mean
♂♂	32	8	22	2	0.1—35	6.6
♀♀	26	6	14	6	0.05—42	7.0
juvenile	14				0.5—3.7	0.9
adult	36				0.4—42	9.1
Oct.-Mar.	36	13	19	4	0.1—35	6.5
Apr.-Sept.	22	1	15	6	0.05—42	7.1
A & B counties (east coast)	26	8	15	3	0.2—35	9.5
N & O counties (west coast)	14	2	10	2	0.2—10	2.9

Geographically, the swan material was distributed as demonstrated on the map (Fig. 1). Twenty-six mute swans from the Stockholm area (east coast) and 14 swans from the west coast were examined. Some of the birds were ringed and this made a determination of the movements of the swans possible. Research in recent years has shown that swans are stationary during the breeding and moulting period but might move around considerably during the rest of the year (*Mathiasson* 1970/1973).

Thirty-six mute swans were submitted during the winter months (October-March) and 22 swans during the summer months (April-September).

Chemical analysis

Animal tissues (liver and kidney) were prepared for chemical analysis by wet ashing using a commercially available ashing apparatus (Tecator AB, Höganäs, Sweden). The apparatus con-

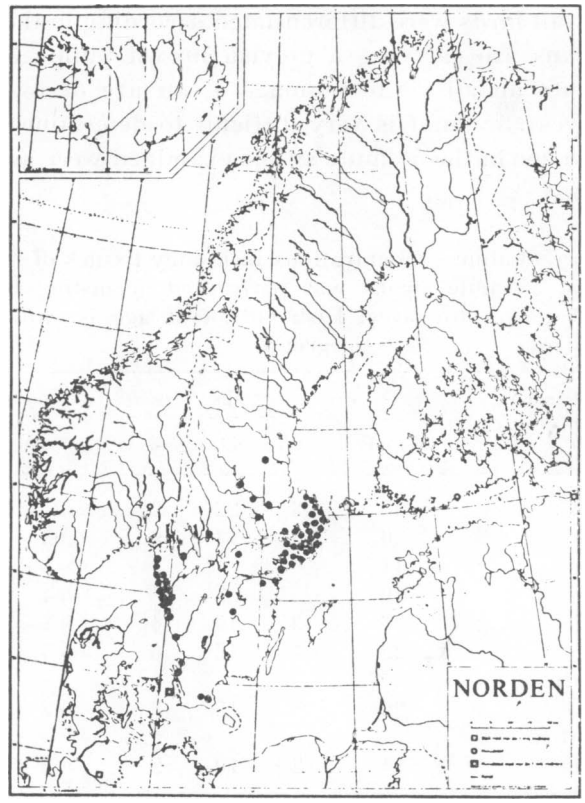

Figure 1. Geographic distribution of 58 mute swans examined during 1973—1977.

sists of two units, viz. an electrically heated aluminium block with space for 40 glass tubes and a thermostate unit. The instrument has been modified to work automatically (*Frank* 1976 b).

The analytical method used may briefly be described as follows. Tissue sample (5 g) is wrapped in a filter paper and placed in the ashing tube. Oxidizing acid (15 ml), a mixture of conc. nitric and 70 % perchloric acid (7:3, by vol.), is added and the samples are kept at room temperature for 5—6 h. The digestion is performed during the night according to the automatic ashing programme. The ashing residue, about 2 ml, is transferred quantitatively to a measuring cylinder (25 ml) and the volume is adjusted to 15 ml with water.

This solution is used for determination of cadmium, copper,

manganese and low amounts of iron by atomic-absorption spectrometry (AAS). For the analysis of copper, zinc and iron the solution is further diluted 1:10 and 1:20 resp., since these metals often occur at high concentrations in the material studied.

Before determining lead and low amounts of copper and cadmium, iron should be extracted from the acid solution by means of cupferron dissolved in MIB-ketone (methyl-isobutyl-ketone) to avoid precipitation of iron as a poorly soluble APDC-complex (1-pyrrolidinedithiocarboxylic acid ammonium salt) during the following steps of the analysis (*Delves et al.* 1971). The aqueous phase is then neutralized with conc. ammonia, pH adjusted with citrate buffer to 7.0, and lead and other metals are extracted as APDC-complexes. The organic extract is used for the determination of metals mentioned above by AAS.

The method is designed to suit wide ranges of concentrations of metals. The tissue metal contents are given on wet weight basis.

RESULTS AND DISCUSSION

The main causes of death among the mute swans examined were inanition, traumatic injuries, certain infectious diseases and heavy metal intoxications (Table 2).

The results concerning each of the six metals mentioned are reported below.

Lead

In the 58 mute swans examined, lead concentrations in liver and/or kidney tissue were below 1 mg/kg in 23 birds, between 1 and 2 mg/kg in 15 birds, between 2 and 5 mg/kg in 9 birds, and more than 5 mg/kg in 10 birds (swan No. 39 was excluded). In the last-mentioned swans (17 % of the material) the lead concentrations were considered rather highly elevated indicating lead poisoning. The average kidney concentration in these birds was about three times as high as the liver concentration (15.9 and 4.8 mg/kg, respectively).

A number of duplicate determinations were performed, and greatly varying results were sometimes obtained. The higher values were traced to metallic lead as fragments or dust, distributed in the body tissues after shooting. When securing analysis material, this metallic lead had contaminated the samples. There

Table 2. Heavy metal concentrations in liver and kidney tissues of mute swans (Cygnus olor Gmel.) during the period 1973—1977. The metal concentrations (mg/kg) are given on wet weight basis.

Swan No.	SVA No.	Month	County	Sex	Age	Post mortem findings	Pb		Cd		Cu		Zn		Fe		Mn	
							Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn
1	02538/73	Sept.	N	♂	ad	Accumulation of food-stuff in esophagus	0.05	0.05	0.3	0.5	232	3.2	31	17	—	—	—	—
2	02539/73	„	N	♀	ad	Catarrhal enteritis, pulmonary and hepatic congestion	0.2	0.2	0.7	0.8	490	6.0	49	19	—	—	—	—
3	02541/73	„	N	♀	—	Catarrhal enteritis, pulmonary congestion	0.4	0.1	1.7	2.2	217	3.9	40	16	—	—	—	—
4	02542/73	„	N	♂	ad	Catarrhal enteritis, parasit. gastritis (Amidostomum sp)	0.1	0.1	0.5	0.4	37	9.6	40	15	—	—	—	—
5	03388/73	Oct.	B	♂	juv	Helminthiasis intest. (Hymenolepis sp), general anemia, inanition	0.6	0.7	0.4	0.4	440	1.2	79	19	—	—	—	—
6	03745/73	Nov.	O	♀	—	Fresh fracture of cervical vertebra	0.4	0.7	0.2	0.3	323	3.7	57	23	—	—	—	—
7	03816/73	„	B	♂	juv	Old shot wound, inanition	0.4	0.8	1.1	2.5	377	10	128	41	—	—	—	—
8	03826/73	Dec.	M	♂	juv	Chron. parasit. gastritis and enteritis (Amidostomum sp, Hymenolepis sp), inanition	0.1	0.1	0.1	0.1	378	6.6	194	87	—	—	—	—
9	03856/73	„	O	♂	ad	Traumatic injury	1.0	1.4	0.7	2.1	459	4.4	83	26	—	—	—	—
10	03942/73	„	O	♂	ad	Fatty degeneration of liver and kidneys, hepatic and myocardial congestion	0.4	0.5	5.3	6.7	576	4.1	55	30	—	—	—	—
11	04012/73	„	B	♀	ad	Multiple cellular liver necroses, calcification of aorta wall, fibrous poly-arthritis	1.1	1.2	1.1	4.0	452	7.4	100	57	—	—	—	—
12	074/74	Jan.	B	♂	juv	Helminthiasis intest. anemia, inanition	0.7	1.2	0.7	1.5	1160	9.6	145	90	—	—	—	—
13	0589/74	Febr.	B	♂	ad	Fibrous staphylococcal endocarditis, hepatic amyloidosis	0.9	8.5	1.6	7.3	51	9.1	29	89	—	—	—	—
14	0720/74	Mar.	O	♂	ad	Subacute peritonitis (shot wound)	1.9	3.1	2.1	5.9	669	2.4	50	30	—	—	—	—
15	0792/74	„	A	♂	ad	Traumatic injury	1.2	18	1.1	4.9	672	4.4	37	27	—	—	—	—

Table 2 (continued).

Swan No.	SVA No.	Month	County	Sex	Age	Post mortem findings	Pb		Cd		Cu		Zn		Fe		Mn	
							Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn
16	01443/74	May	S	♂	ad	Fresh fracture of cervical vertebra, hypoplasia of right testicle	0.6	0.8	3.7	5.2	724	3.7	51	22	—	—	—	—
17	02074/74	July	E	♀	ad	Inanition, pulmonary, hepatic and renal	1.1	0.8	11.5	42	395	16	134	62	—	—	—	—
18	03236/74	Sept.	W	♀	ad	Dilatation of small intestines, hepatic and renal congestion	1.3	0.2	1.2	4.7	2300	3.1	44	30	1000	209	2.9	5.0
19	03242/74	„	E	♀	juv	Helminthiasis intest. inanition	0.05	0.1	0.05	0.05	126	3.2	34	20	371	140	2.1	1.7
20	03243/74	„	O	♀	ad	Fracture of right tibia, poor condition	0.7	1.2	0.9	10	1200	8.7	41	39	1250	243	1.5	1.7
21	03244/74	„	O	♂	ad	Fracture of left radius and ulna	0.1	1.4	2.8	3.7	236	5.6	32	24	1120	201	3.0	2.8
22	03931/74	Nov.	B	♂	ad	Accumulation of foodstuff in mouth cavity, fatty degeneration of liver	0.9	2.7	0.5	3.7	168	2.2	24	19	1790	195	1.7	1.9
23	04214/74	Dec.	C	♀	juv	Fresh fracture of right wing	0.8	2.7	0.3	0.3	2980	5.2	64	29	200	125	5.9	3.5
24	04216/74	„	M	♀	ad	Chronic purulent aerocystitis, confluent spleen necroses	0.4	0.4	0.05	1.9	104	7.8	48	35	5620	470	2.3	2.6
25	P5011/74	„	O	♂	ad	Negative section	0.3	0.6	2.4	4.4	711	3.3	61	23	447	81	2.4	3.4
26	P5012/74	„	P	♀	ad	Negative section	0.9	1.9	3.1	12	145	7.7	196	139	1910	231	1.6	2.8
27	04396/74	„	B	♂	ad	Plumage contaminated with mineral oil	2.5	7.4	3.6	35	696	6.5	94	57	983	248	1.8	2.8
28	0332/75	Jan.	A	♀	ad	Inanition, hemorrhagic erosions of gizzard mucosa	2.7	3.3	4.9	16	273	4.9	173	93	3590	282	1.4	1.7
29	0333/75	„	A	♀	ad	Inanition hemorrhagic erosions of gizzard mucosa	3.0	3.4	5.5	11	932	12	220	76	3970	195	1.4	1.9
30	0371/75	Febr.	A	♂	ad	Hepatic amyloidosis, rupture of liver, hemocoeli, hemosiderosis	4.1	13	1.3	7.1	2420	19	34	21	929	111	1.4	3.1
31	0972/75	Mar.	O	♀	juv	Inanition	0.7	1.4	0.1	0.2	1370	5.4	67	23	990	197	3.5	3.6
32	0973/75	„	O	♂	ad	Traumatic injury, parasit. gastritis (Amidostomum sp)	0.2	0.4	0.8	3.2	458	3.7	33	27	649	104	2.4	4.4

Table 2 (continued).

Swan No.	SVA No.	Month	County	Sex	Age	Post mortem findings	Pb		Cd		Cu		Zn		Fe		Mn	
							Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn
33	0974/75	„	O	♂	juv	Inanition, parasit. gastritis (Amidostomum sp)	0.9	1.2	0.1	0.2	455	2.8	65	24	841	192	1.4	2.2
34	02332/75	July	T	♂	ad	Necroses in esophagus, inanition	0.9	0.5	3.9	3.8	800	9.0	162	61	3170	311	2.3	1.2
35	03793/75	Aug.	B	♂	ad	Subacute staphylococcal endoaortitis and splenitis, serous hepatitis	0.1	0.6	0.9	12	256	5.9	28	37	195	123	2.5	1.6
36	04598/75	Sept.	W	♂	—	Mycotic aerocystitis (Aspergillus fumigatus)	0.7	1.9	3.7	8.6	576	13	568	141	1410	266	1.6	2.2
37	05079/75	Oct.	B	♀	ad	Catarrhal enteritis, non-purulent myocarditis, interstitial nephritis	0.1	0.2	1.9	4.0	256	3.1	26	18	645	296	1.8	1.3
38	05167/75	„	B	♂	ad	Nephrosis, fatty degen. and cirrhosis of liver	3.1	2.6	2.9	12	1920	6.7	89	39	782	186	5.7	3.8
39	05924/75	Dec.	D	♂	juv	Pericarditis (shot)	(4.1	41)	0.2	1.1	95	6.9	263	63	889	245	2.6	2.6
40	05991/75	„	A	♀	ad	Traumatic injury	2.7	8.2	2.1	9.6	383	7.3	86	35	691	125	2.1	1.7
41	0458/76	Febr.	B	♀	juv	Catarrhal enteritis, fatty degen. of liver	1.5	2.8	0.8	1.4	1270	2.3	40	13	693	338	1.5	1.1
42	0583/76	Febr.	B	♀	juv	Encephalitis, parasit. gastritis (Amidostomum sp)	5.7	26	2.2	3.7	1370	7.5	86	31	1310	218	1.5	1.4
43	0591/76	„	F	♀	ad	Traumatic peritonitis, helminthiasis intest., nephritis urica	0.2	0.7	1.4	1.5	1500	5.3	133	39	504	140	4.5	2.6
44	0763/76	„	A	♀	ad	Inanition, hemosiderosis of Kupffer cells and hepatic endothelial cells, old shot wounds (lead shots in body)	0.05	5.8	11	22	2220	15	148	68	3680	170	1.2	1.6
45	01714/76	Apr.	B	♂	—	Catarrhal-hemorrhagic enteritis, inanition	2.2	4.0	8.1	24	992	8.8	228	107	2730	262	3.6	4.6
46	01862/76	May	X	♂	ad	Hemorrhagic enteritis, periportal cell infiltrations in liver	0.3	0.4	2.7	6.1	436	2.5	53	21	1330	268	2.3	3.3
47	02258/76	June	L	♀	—	Mycotic aerocystitis (Aspergillus fumigatus)	0.3	0.1	1.4	1.6	63	2.4	66	26	1600	79	1.4	0.6
48	02277/76	„	W	♂	ad	Piece of copper in gizzard, inanition, anemia, helminthiasis intest.	—	1.0	3.4	7.9	3820	51	190	134	1510	158	2.0	1.6
49	02942/76	July	W	♀	—	Purulent and necrotic tracheitis, pulmonary congestion and edema	0.5	0.3	3.5	5.2	1120	3.7	144	39	2920	343	3.9	1.6

Table 2 (continued).

Swan No.	SVA No.	Month	County	Sex	Age	Post mortem findings	Pb		Cd		Cu		Zn		Fe		Mn	
							Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn
50	04214/76	Nov.	A	♂	juv	Fatty myocardic degen. liver necroses, helminthiasis intest.	0.5	0.7	0.3	0.3	1140	2.0	68	14	638	309	2.0	1.2
51	04215/76	„	A	♀	juv	Helminthiasis intest. (Amidostemum sp, Hymenolepis sp)	3.9	8.4	0.3	0.6	739	5.8	59	22	411	189	2.1	2.3
52	019/77	Jan.	B	♂	juv	Fishing hook perforating wall of esophagus and trachea, inanition	0.8	1.2	0.1	0.2	1480	8.9	237	77	2330	160	2.0	1.7
53	0881/77	Mar.	A	♂	ad	Fibrinous pericarditis, inanition	11	34	13	29	1130	15	249	86	3030	119	2.5	2.2
54	01214/77	„	B	♀	—	Helminthiasis intest. (Amidostomum sp, Hymenolepis sp), inanition	16	30	8.1	19	2240	24	228	85	5530	233	1.7	1.6
55	01461/77	Apr.	B	♀	—	Traumatic injury	0.7	1.7	2.1	6.0	1400	4.1	51	28	495	132	4.3	2.7
56	01651/77	„	B	♂	ad	Gout, parasit. gastritis (Amidostomum sp)	1.6	2.0	3.4	8.8	330	3.4	72	30	774	211	2.7	1.4
57	01898/77	May	L	♀	ad	Indurative myocarditis, hepatic, renal and cerebric congestion	0.5	1.2	0.5	1.0	823	3.1	83	20	847	178	4.7	2.3
58	01926/77	„	E	♂	ad	Traumatic injury	0.2	0.4	1.1	1.9	1070	2.2	46	19	338	227	2.2	2.1

Table 3. Heavy metal concentrations in liver and kidney tissues of whooper swans (Cygnus cygnus L.) during the period 1973—1977. The metal concentrations (mg/kg) are given on wet weight basis.

Swan No.	SVA No.	Month	County	Sex	Age	Post mortem findings	Pb		Cd		Cu		Zn		Fe		Mn	
							Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn	Liv	Kidn
1	03799/74	Nov.	BD	♀	ad	Giant cell granulomas containing acid fast rods in intestinal wall	0.2	0.3	0.04	0.8	102	3.4	15	26	388	152	0.8	1.5
2	0518/75	Feb.	P	♀	juv	Accumulation of foodstuff in esophagus, inanition, anemia	2.9	3.3	0.4	0.5	337	9.2	86	58	3570	35	4.2	6.9
3	01750/76	Apr.	R	♂	ad	Accumulation of foodstuff in esophagus, liver necroses	32	76	0.8	1.0	161	2.3	121	18	2650	140	1.7	2.1
4	04096/76	Nov.	Y	♂	ad	Interstitial nephritis, fresh fracture of cervical vertebra	0.06	0.2	1.0	4.4	26	3.5	35	22	531	277	2.3	3.4

is thus need for great care in taking samples and evaluating analytical results in game suspected to have been hit by bullets (*Frank*, to be published, b). (Swan No. 39 in Table 1 was shot, consequently the values were not included in the following calculations). Parenterally deposited metallic lead is not absorbed and does not induce lead poisoning.

In some of the swans with highly elevated lead concentrations, there was a greenish discoloration of the feathers around the cloaca. Dilatation of the oesophagus and proventriculus as a cause of accumulation of foodstuff was earlier seen in a few lead intoxicated swans. Among the swans presented here, such foodstuff accumulation was seen in two mute swans and two whooper swans, only one of the last-mentioned, however, with highly elevated lead residues (cf. Table 3, see also Note below.)

In lead-intoxicated ducks, acid-fast intranuclear inclusion bodies were found in the proximal tubular epithelium of the kidney (*Locke et al.* 1966, *Bates et al.* 1968, *Clemens et al.* 1975). Our investigation material was not suitable for more detailed histological studies because it was not completely fresh.

The source of the lead poisoning in the swans may have been metallic lead, for instance as lead shots which during hunting were deposited in shallow waters and ingested by the swans when feeding in the area. French research (*Hovette* 1971) indicates that this ingestion might be intentional. Other lead sources may also occur, such as lead-containing paints or lead compounds of other kind. Swans are obviously more likely to be lead-poisoned than eider-ducks because of their different diets.

Lead concentrations of 6—20 mg/kg in the liver and more than 20 mg/kg in the kidneys may according to *Longcore et al.* (1974) indicate a recent acute lead exposition in the mallard.

In a material of 29 mute swans, submitted to the SVA during the period 1958—1968 from different parts of Sweden, lead concentrations in liver and kidney tissue exceeded 10—20 mg/kg in 50 % of the birds (*Erne & Borg* 1969). One of the swans was

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Note: Lead poisoning in whooper swans was recently reported by Norwegian authors (*Holt et al.* 1978). Lead contents more than 5 mg/kg liver tissue were found in 8 of 34 swans investigated. Only 2 of them showed clinical signs and pathological lesions of "typical" lead poisoning. Lead shots in the gizzard were found in 3 swans.

found dead quite near a skeet-range located close to a small lake and a great number of lead shots were found in the gizzard. The frequency of swans suspected to have been lead-poisoned in that material was thus considerably higher than in the present investigation material (17 %).

Recently, mortality among Danish swans seems at least partly to be associated with certain conditions causing the swans to ingest lead shots to an unusual high degree (*Clausen et al.* 1975).

There are varying reports on the possibility of gallinaceous birds being poisoned by metallic lead. *Shifrine et al.* (1964) state that in chicken which ingested lead shots, 125—510 mg/kg (dry weight) of lead was found in liver tissue (these levels might correspond to about 40—150 mg/kg wet weight). *Silvén* (1967) did not succeed, however, to induce clinical signs of intoxication in chicken, fed lead sand and lead shots during a prolonged period of time. Subsequent analyses revealed only moderately high lead residues or as a maximum 2.0 mg/kg in liver and 6.6 mg/kg in kidney tissue. There is also a minor number of reports on lead poisoning in pheasants, partly based on the findings of lead shots in the gizzard, partly on elevated tissue concentrations of lead found at chemical analysis. As mentioned above, findings of lead in game ought to be evaluated with care.

The absorption of metallic lead from the digestive tract is influenced by a number of factors, e.g. the composition of the diet. High fiber content induces a more rapid absorption and intoxication than does a low fiber content (*Clemens et al.* 1975). The accumulation of lead in the organs furthermore increases at a low calcium diet (*Longcore et al.*). The presence of vitamin D_2 facilitates the absorption of lead (as well as that of calcium). In man, most lead intoxications occur during the summers which is considered to depend on the formation of vitamin D in the skin at sunshine. In swans, however, the most elevated lead residues were found during the winter months. Lead absorption is much higher, up to 50 % higher, in young than in old people (*Alexander et al.* 1973). Starvation also increases gastrointestinal absorption of lead. *Wetherill et al.* (1975) showed that while fasting, experimental human subjects absorbed as much as 50 % of the amount of lead given with the food, whereas subjects in the non-fasted state absorbed 6—14 %.

Cadmium

The environmental pollution of cadmium is worldwide. It has unusual biological properties e.g. great accumulation tendency in kidney tissues of mammals and birds, low rate of excretion and consequently long biological half life. These properties make the metal especially interesting for closer studies.

The mean cadmium concentrations in kidney tissue of the 58 mute swans included in the present investigation material are shown in Table 1 (see also Table 2). The material is divided according to sex and age, seasons of the year and to some extent locations as well. This division makes every group rather small and even if there is a clear tendency in the material, the results ought to be evaluated with certain care.

As is obvious from Table 1, there is no great difference in kidney cadmium concentration between the sexes, nor between the seasons. This is hardly to be expected either, in the latter case because of the long biological half life of cadmium.

Not surprisingly, however, the cadmium burden was considerably greater in adult than in juvenile birds, mean renal concentrations being 9.1 and 0.9 mg/kg, respectively. The difference is statistically significant ($P = 0.01—0.001$).

The highest renal cadmium concentrations in the swan material, 35 and 42 mg/kg, were found in an adult male from the Stockholm archipelago and in an adult female from Vikbolandet in the county of Östergötland. In none of these two swans, nor in any of the other swans were any lesions indicating harmful effects of cadmium found in the kidneys or other organs or skeleton. Yet, non-specific kidney lesions not possible to establish histologically, but leading to tubular dysfunction, may not be excluded.

Cadmium accumulates primarily in the renal cortex, and analysis of human and equine kidneys also usually refers to cortex concentrations. The first signs of the toxic action of cadmium (tubular dysfunction) in man start to be detectable at cadmium levels of about 200 mg/kg kidney cortex. Such concentrations are not reached in the present material. In lack of knowledge concerning the sensitivity of avian kidneys against cadmium, it is not possible to evaluate the importance of avian renal cadmium concentrations.

As regards cadmium residues in swans from different places, there is a great difference in swans from the Swedish east and

west coast, mean renal concentrations being 9.5 and 2.9 mg/kg, respectively. The difference is statistically significant ($P = 0.01—0.001$). The difference is even more obvious than shown by the figures as a larger proportion of juvenile swans is included in the east coast material than in the west coast material (cf. Table 1). In man, the biological half life of cadmium is very long, 30 years or more, and if conditions are similar in swans, the birds may not necessarily have ingested the essential part of their cadmium burden at the place where the body was found. Records from ringing do not indicate, however, that mute swans might migrate to some great extent between the coastal areas of east and west Sweden.

Similarly to the findings in swans, more elevated residues of mercury were found in wild mink and seals from the Swedish east coast than in the same species from the west coast (*Borg* 1975 a, b). In mussles (Mytilus edulis) from the Baltic sea (east coast), higher zinc and cadmium concentrations were demonstrated than in mussles from the Kattegatt (west coast) (*Phillips* 1977). Higher biological availability of both metals in low salinity waters was suggested as the explanation for the difference. It might be added that the zinc concentration in the water of the Baltic sea is in the range of 6—8 µg/l, in the ocean waters of 0.5—4 µg/l (*Gustavsson* 1977).

The correlation between cadmium and zinc is discussed below.

Copper

Remarkably high copper contents or more than 1000 mg/kg were found in liver tissue of roughly 30 % of the swans (Table 2 and diagram, Fig. 2). With one exception (No. 48), no signs of harmful effect of the copper were found, however.

The capacity for hepatic copper storage, i.e. the liver copper levels that can be tolerated without signs of copper toxicosis, varies greatly among species.

In copper-poisoned sheep, *Nilsson* (1968) reports copper concentrations of 61—860 mg/kg in the liver. High amounts of copper in the diet was obviously the source of the poisoning, but also other conditions might have contributed, viz. low amounts of molybdenum and sulphur in the diet.

In diagnosed chronic copper poisoning of sheep, 219—612 mg/kg of copper in liver and 26—104 mg/kg in kidney, were

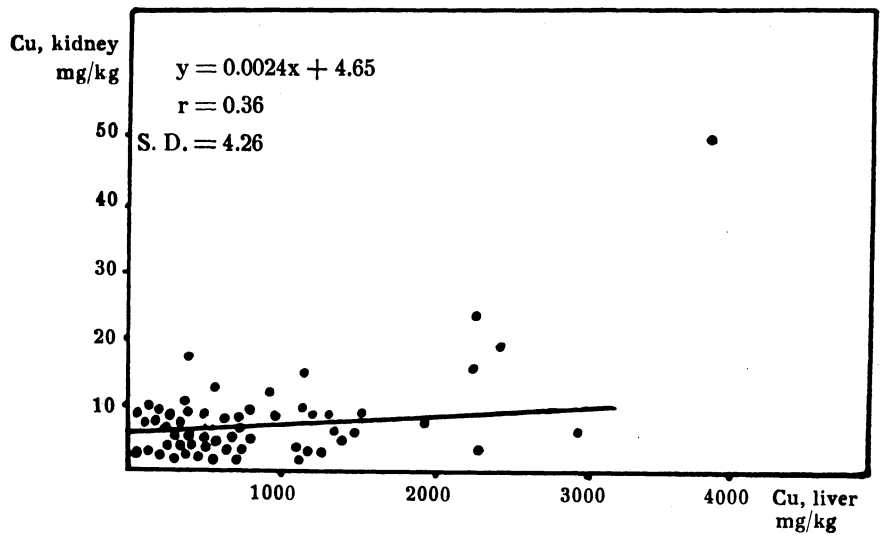

Figure 2. Kidney versus liver copper concentrations in 58 mute swans. Calculating the regression line, the swan with extreme copper values was not included (n = 57).

reported recently (*Arora et al.* 1977). The average liver copper contents in the cited investigation were 418 mg/kg which is in good agreement with the corresponding value of 420 mg/kg given in a Norwegian report on liver copper contents in sheep dead from chronic copper toxicosis. However, the range was considerably greater in the latter investigation or 150—1900 mg/kg (*Frøslie* 1977).

Also clinical and patho-anatomical findings vary greatly in different species. As mentioned above, copper residues of 1000 mg/kg in the liver of swans are not unusual and seem to be well tolerated as reported earlier (*Erne & Borg*). In chronic copper poisoning of sheep, some conditions start catastrophic liberation of copper into the blood resulting in hemolysis and jaundice, the latter not being reported in rat, rabbit and pig, however. Nor was hemolytic jaundice reported in birds (*Underwood* 1971).

A brownish discoloration of organ tissues, frequently observed in copper-poisoned sheep, is caused by retention of hemoglobin and methemoglobin in the liver and kidney after hemolysis. This is a consequence of sudden release of copper from the liver during the terminal crisis of chronic copper poisoning

(*Søli & Frøslie* 1977). Consequently, the detection of elevated concentrations of iron in liver and kidney supports the diagnosis. Elevated copper contents in the kidney as well are characteristic in copper poisoning and useful for diagnosis (*Clarke & Clarke* 1975, *Arora et al.*).

Adult birds exhibit a marked loss of body weight and anemia but no evidence of intravascular hemolysis (*Underwood*). In the swan in our material, suspected to have been copper poisoned, similar findings were observed. This also means that there was no brownish discoloration as a result of a hemolytic crisis nor any elevated iron concentrations in liver and kidney.

A regression line was calculated for the relation between copper values in the liver and the kidney of swans. Our data do not necessarily support the existence of a linear relationship (Fig. 2).

For some unknown reason, mute swans may accumulate considerable amounts of copper in the liver, very much more than mammals without any signs of toxic influence. However, in all but one swan there was no particular accumulation of copper in the kidneys.

In our material, one swan (No. 48) presented signs of copper intoxication, very high copper contents being found in the liver and to some extent also in the kidneys, viz. 3820 and 51 mg/kg, respectively. As compared with other swans, both values were considered elevated. The swan had ingested some metallic copper piece, attached to a fishing hook. Post-mortem examination of the swan revealed general anemia and inanition in good agreement with what has been postulated for copper poisoning in birds.

The results support the observations which have recently been presented by Danish authors (*Clausen & Wolstrup* 1978).

The whooper swan does not seem to accumulate copper to the same degree as the mute swan as seen from Table 3.

The disparity in copper accumulation between the mute swan and the whooper swan might depend on a different choice of food, or maybe different copper metabolism in the two bird species.

Zinc

Cadmium is generally present as a trace element in zinc ore (metalliferous rock) and zinc salts, and these two metals thus

often occur simultaneously. They might pollute the environment from different industries etc. Industrial waste containing merely cadmium pollutes the environment as well.

In the present material, the majority of mute swans had a cadmium concentration in liver and kidney not exceeding 6 and 12 mg/kg, respectively, and a zinc concentration in those organ tissues below 100 and 50 mg/kg, respectively.

Adult mute swans with elevated cadmium concentration in liver or kidney had elevated concentrations of zinc too. In contrast, adult and young swans with elevated zinc concentrations had cadmium concentrations ranging from very low to elevated. (No young swans had elevated cadmium contents). — Zinc concentrations were thus higher in organs from swans with higher cadmium concentrations.

These findings are in general agreement with reported increase of zinc in renal cortex of higher mammals (such as man, horse and pig) at increasing cadmium concentrations (*Elinder & Piscator* 1978). In humans the highest molar ratio between zinc and cadmium has been found to be 1 and is believed to be compensatory to the increase of cadmium. In other species, such as goat, rabbit, guinea-pig, mouse and hen, limited or no increase of zinc was reported at higher cadmium levels.

According to statistical calculations (in preparation) the slope of the regression line between zinc and cadmium in whole kidney tissue from swans was unexpectedly high ($\lesssim 3$ on the molar basis, Zn/Cd). It is conceivable that this difference found between mute swans and other species is due to whole kidney tissue being taken to analysis instead of kidney cortex.

In two juvenile swans with low cadmium concentrations (Nos. 8 and 52), the zinc concentrations were remarkably high (Table 2). The two swans originated from the Stockholm and Malmöhus counties.

Iron and manganese

The findings of these two metals in 41 mute swans were not remarkable, cf. what is mentioned in connection with copper. In comparison with iron or other metals discussed above only small variations of manganese concentrations were found in liver and kidney. They had a range of 1.2—5.9 mg/kg in liver and 0.6—4.6 mg/kg in kidney. The mean values were 2.5 and 2.3 mg/kg, respectively.

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SAMMANFATTNING

Tungmetaller i vävnader från knölsvan (Cygnus olor).

Under tidsperioden 1973—1977 insändes ett antal döda knölsvanar (Cygnus olor) till Statens veterinärmedicinska anstalt i Stockholm för undersökning. Organ från 58 av dessa svanar analyserades kemiskt på halter av bly, kadmium, koppar, zink, järn och mangan. Hos tio svanar (17 procent) påvisades starkt förhöjda blyhalter, tydande på förgiftning. Kadmiumhalterna var signifikant högre hos vuxna än hos unga svanar och också hos svanar från den svenska ostkusten jämfört med svanar från västkusten. Anmärkningsvärt höga kopparhalter påträffades ofta, dock med ett undantag utan tecken på förgiftning. Höga zink- och kadmiumhalter påvisades ofta hos samma fåglar. Inga speciella kommentarer kan göras beträffande fynden av järn och mangan.

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