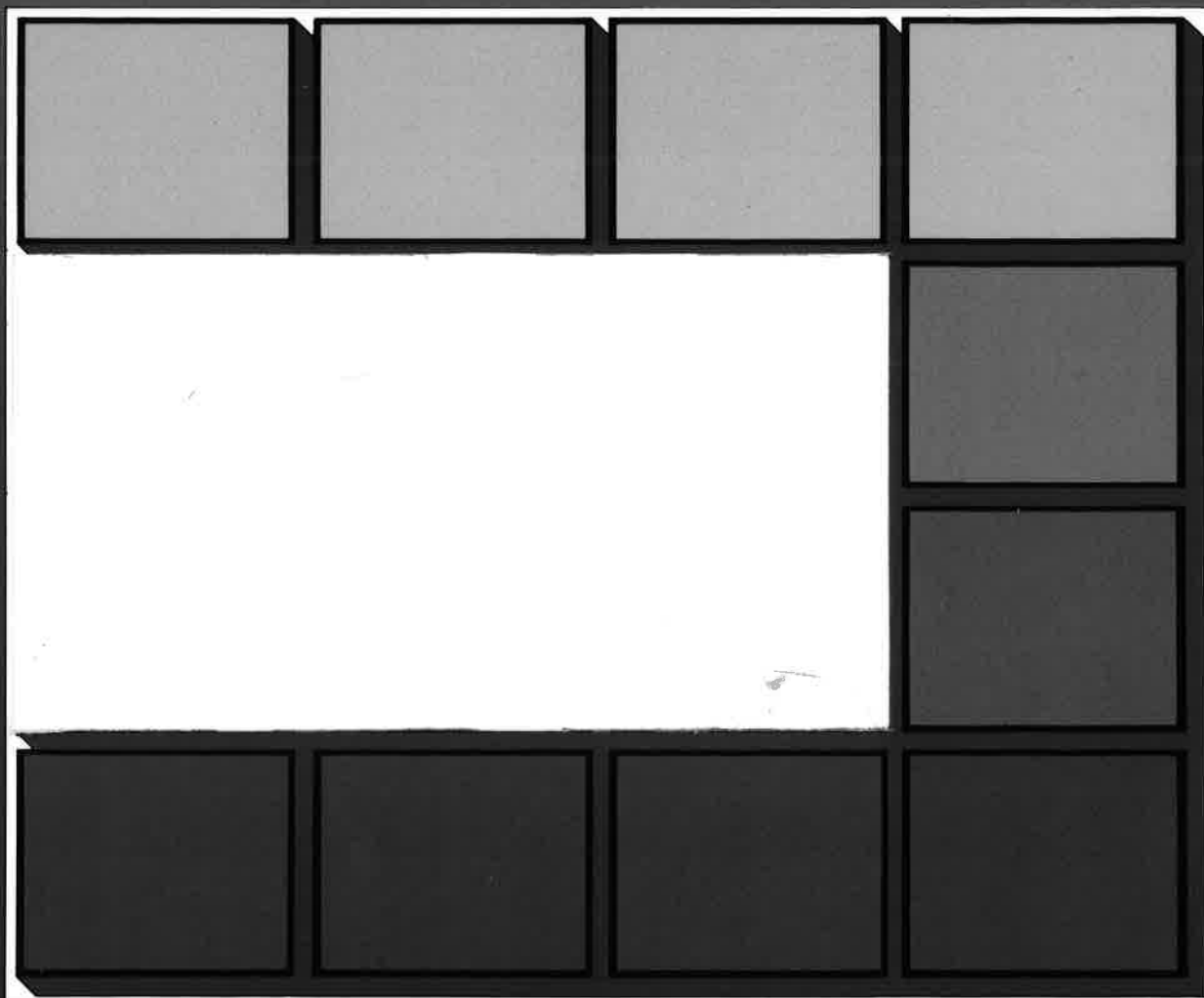


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BIRDS AND POLLUTION

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Annual Report to Nature Conservancy Council

I NEWTON, A ASHER, P FREESTONE, M C FRENCH,
D LEACH, G POLWARTH & I WYLLIE

Monks Wood Experimental Station
Abbots Ripton, Huntingdon
Cambs PE17 2LS

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- Part 1 Organochlorines and mercury in predatory birds
- 2 Sparrowhawk survey
 - 3 Organochlorines and mercury in peregrine eggs
 - 4 Organochlorines and mercury in merlin eggs
 - 5 Mercury in feathers of merlins and their prey
 - 6 Organochlorines and mercury in gannet eggs
 - 7 Rodenticides in barn owls

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Part 1 Organochlorines and mercury in predatory birds

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1 ORGANOCHLORINES AND METALS IN PREDATORY BIRDS

1.1 Recent findings

The main objective of this work was to analyse the carcasses of predatory birds, supplied by members of the public, in order to continue the monitoring of organochlorine and metal residues in livers. The chemicals of interest included DDE (from the insecticide DDT), HEOD (from the insecticides aldrin and dieldrin), PCBs (poly-chlorinated biphenyls from industrial products) and Hg (mercury from agricultural and industrial sources). Throughout this section the levels of organochlorines are given as ppm in wet weight and of mercury as ppm in dry weight.

The main species involved included the sparrowhawk and kestrel, representing the terrestrial environment, and the fish-eating heron, kingfisher and great-crested grebe, representing the aquatic environment. The last major analyses of long-term data were by Cooke *et al.* (1982), on specimens obtained to 1977, and by Newton *et al.* (1986) on specimens obtained to 1985.

During 1988, the livers from 184 birds were analysed, including those from 28 kestrels, 69 sparrowhawks, 16 herons, 9 kingfishers, 4 great-crested grebes and 23 others. These totals included some birds which had died in earlier years, but which were analysed in 1988. The results from all these birds are listed in Table 1, and the geometric means for each chemical from the main species (1988 specimens only) are given in Table 2.

Three significant differences in geometric mean values were found between the 1988 and 1987 results, out of 20 comparisons (Table 3). These included an increase in the mean DDE, HEOD and Hg values for sparrowhawk. It is impossible to say whether these differences reflected real changes in exposure.

1.2 Long-term trends

In order to assess the recent results for each species in the context of the longer-term trends, the individual values of all chemicals are given in Figures 1-20 nationwide for the whole monitoring period. Trends are indicated by 3-year moving averages. Analyses for DDE and HEOD were started in 1963-64, analyses for PCB in 1967-69, and for Hg in 1969-80, depending on species.

In each case the significance of the long-term trend was assessed by regression analyses of individual residue levels on years (Table 4), covering the whole analytical period for each chemical. During the periods concerned, various restrictions were imposed on particular agricultural uses of DDT, aldrin/dieldrin and organo-mercury compounds, so the total amounts of these chemicals applied each year to the land surface of Britain should have declined. Separate regression analyses covered the period 1981-88, in order to examine the most recent trends, independently of earlier results. In 1981 Britain came under EEC regulations, aimed at stopping almost all agricultural uses of these chemicals, and by 1988 only one minor and localised use of aldrin remained. Over the whole analytical period (1963-88), DDE levels declined in all species, though the decline was not significant in the Great-crested Grebe (Table 4). In the fish-eating species, the decline was evident more or less throughout the period of study, but in the raptors chiefly since the late 1970s (Figures 1-5).

The general level of residues differed between species. In the fish-eaters, geometric mean values were mostly in the range 5-10 ppm fresh weight in the early years, declining to 1-2 ppm later. In the raptors, geometric mean levels were generally higher in sparrowhawks (5 ppm declining to around 3 ppm) than in kestrels (1-2 ppm declining to around 0.4 ppm). After 1970 kestrels showed the greatest range of DDE values, and even as late as 1978 and 1982, 2 birds (from Kent) had levels exceeding 1,000 ppm. In the period 1981-88, significant decline in DDE levels was apparent only in sparrowhawks.

Over the whole period 1963-88, significant declines in HEOD levels were evident in all species except Great-crested Grebe (Table 4). In both raptors, levels were similar, declining from a geometric mean of around 1 ppm initially to 0.4 ppm later, but in the heron the decline was steeper, from 1-4 ppm initially to less than 0.5 ppm later (Figures 6-8). In kingfishers and grebes it was hard to discern any long-term trend in HEOD (Figures 9-10): the higher initial levels in kingfishers could have resulted from the small sample in the early years, after which the geometric mean stayed for most of the period around 1-2 ppm; in grebes the geometric means stayed below 1 ppm throughout, but fluctuated considerably, often in association with small annual samples.

In the period 1981-88, significant change in HEOD level was evident only in herons, in this case an increase in geometric mean from 0.2 ppm to around 0.6 ppm.

Over the whole period of PCB analysis (1967-85), regression studies suggested no long term trend in the two raptors, but very slight declines in the levels in the three aquatic species, although only in the great-crested grebe was the trend statistically significant (Table 4). The annual fluctuations in geometric means did not vary in parallel in the different species, and were probably due partly to small or unrepresentative annual samples. In the fish-eaters, annual geometric means were mostly in the range 2-10 ppm, in the sparrowhawk they were mostly in the range 1-3 ppm, and in the kestrel 0.5-2.0 ppm (Figures 11-15). In the most recent period, 1981-88, no significant change in PCB levels was evident in any species.

Over the whole period of Hg analysis (1969/70-88), significant declines in levels were evident in sparrowhawk, kestrel and heron (Table 4). In the remaining species, analyses were started only in 1979-80, and significant change (an increase) was apparent by 1988, only in heron (Table 4).

The heron was much the most contaminated of the species studied, with geometric means of around 40 ppm Hg initially, declining to around 15 ppm latterly (Figure 18). The great-crested grebe had geometric mean levels in 1980-88 mostly around 5-10 ppm, and the kingfisher had mean levels of 1-2 ppm (Figures 19-20).

Of the two raptors, the kestrel had geometric mean levels of almost 5 ppm Hg initially, declining to around 1 ppm later, while the sparrowhawk had levels of 5-6 ppm, declining to around 3 ppm (Figures 16-17).

1.3 Discussion

For any given pollutant, the levels in livers varied between species, as did the extent of change in levels over the study period. This was presumably because of differences in diet between species, and in the ease with which they could metabolise the various chemicals. The fact that the heron accumulated most pollutants to greater levels than the other

fish-eaters, could be attributed to the heron taking generally larger (and older) fish, which generally have more residue than do smaller fish. Herons also eat eels, which are known to accumulate fat and organochlorine residues to greater levels than are some other freshwater fish (Holden 1973; Hider *et al* 1982). To judge from their size, some of the eels eaten by herons could be up to 20 years old (Hussein 1982; Marquiss 1987). Among the raptors, the sparrowhawk had higher levels of most pollutants than the kestrel, and showed less decline in levels during the study period. There were probably three reasons for this difference. The sparrowhawk eats other bird-species (herbivores and carnivores), and hence feeds higher in the food chain than the kestrel, which eats mainly herbivorous voles. Secondly, birds in general are less able to metabolise organochlorines and other pollutants than are mammals (Walker 1983), so for this reason too the bird-eating sparrowhawk would tend to accumulate higher levels than the mammal-eating kestrel. Thirdly, sparrowhawks are less able than kestrels to metabolise organochlorines within their own bodies (Walker *et al* 1987). It was not surprising, therefore, that sparrowhawks suffered a more marked and widespread population decline than kestrels.

In contrast to the other chemicals, mean HEOD levels were not much higher in sparrowhawks than in kestrels. This was possibly because HEOD is much more toxic than the other residues examined, and accumulation at the upper levels observed is likely to cause death. In this case, more heavily contaminated individuals were not likely to occur, either in the population at large or in the samples. For the other organochlorines, most of the values recorded were well below the level expected to kill (Cooke *et al* 1982).

The greater range of DDE levels found in kestrels, compared to other species was largely attributable to individuals from Kent and other parts of the southeast. The two most heavily contaminated individuals, containing 1,480 and 1,500 ppm DDE respectively in liver, came from Kent as recently as 1978 and 1982. The birds concerned were in otherwise good condition and had presumably died of DDE poisoning. Such high levels were probably attributable to the high DDT use in the orchards of Kent. Sparrowhawks have only recently begun to recolonise Kent (Newton & Haas 1984), and too few specimens have been received from there to assess DDE levels in this more sensitive species.

The general declines recorded in the levels of pesticide residues would be expected from declines in agricultural usage, resulting from various restrictions imposed from time to time during the study period. It is surprising, however, that the declines in DDE residues were as slight as they were. This may be due partly to the greater persistence of this chemical in soil and in animal bodies, compared to HEOD. Calculated half-lives of DDE in soil have ranged between 12 and 57 years (Buck *et al* 1983; Cooke & Stringer 1982), compared with only 4-7 years for HEOD (Anon 1964; Edwards 1966). In the bodies of pigeons, DDE had a half-life of 240 days, compared with 47 days for HEOD (Walker 1983).

The increase during the period 1981-88 in residues of HEOD in herons was disconcerting and inexplicable. This increase did not result from a changed geographical spread in the sources of specimens, and presumably reflected increased exposure. If it was due to attempts to use up stocks before the EEC regulations came in, then this should have reflected in the other species, which it was not. It must therefore have been due to some changed exposure which affected mainly this species.

The fish-eaters were likely to obtain their HEOD residues, not only from agricultural sources, but also from industrial ones, as factory effluent in rivers. Indeed, in some regions factory effluent was probably the major source of residues for herons and kingfishers, and improved water quality control is likely to have reduced the input of HEOD from industrial sources over the last 30 years, thus contributing to the decline in overall contamination levels.

Levels of PCBs were generally higher in the fish-eaters than in the raptors. This would probably be expected considering the wholly industrial source of PCBs. The somewhat higher levels in sparrowhawk than kestrel could be attributed to the same factors that influence DDE levels, namely feeding habits and differing ability to metabolise organochlorines.

Much more mercury has been used in industrial processes in Britain than in agriculture (DoE 1976), and industrial processes are likely to have provided the main source of residues for at least the fish-eaters. The use and disposal of mercury has been much more rigorously controlled in recent years, and agricultural uses have also been reduced (ARC 1964), so it was not unexpected that residue levels had declined in all species examined.

1.4 Conclusions

The 5 species examined have continued to show contamination with organochlorine and mercury compounds. All but one have shown significant declines in DDE and HEOD levels over the last 26 years. The decline in DDE levels is perhaps less than would be expected from trends in agricultural usage, and may be due to high persistence. No significant declines have occurred in PCB levels except in Great-crested Grebes. Hg has declined in both raptors and the heron, the only species for which long-term results were obtained. The heron continues to be the most contaminated of the three fish-eaters examined, and the sparrowhawk the most contaminated of the two raptors.

In view of these various findings, it seems prudent to continue the monitoring programme for some further years. For the pesticides, it is especially important to check the effect of the various EEC restrictions on usage.

1.5 Acknowledgements

Thanks are due to all the contributors who sent in specimens during the past year, too numerous for individual mention.

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Table 1. Levels of organochlorines (ppm in wet weight) and mercury (ppm in dry weight) in the livers of predatory birds analysed between April 1988 and March, 1989. ND = None detected.

Spec. no.	Date found	County	Age	Sex	pp'-DDE	HEOD	PCBs	Hg
Kestrel (<u>Falco tinnunculus</u>)								
9178	Sep 86	Gwent	J	M	ND	0.06	0.98	0.60
9155	Oct 87	Orkney	J	M	0.18	0.30	3.46	9.28
9183	Dec 87	Gwent	J	M	ND	ND	7.94	1.08
9222	Dec 87	Orkney	J	F	1.50	0.32	19.19	2.84
9146	Jan 88	Cambs	A	F	0.18	1.10	4.04	0.80
9175	Jan 88	London	A	F	0.22	ND	4.49	1.42
9186	Jan 88	Kirkcud	J	F	ND	5.42	27.86	1.64
9187	Jan 88	D & G	J	F	0.02	0.09	1.69	1.10
9190	Feb 88	Cambs	J	M	0.98	0.63	0.88	1.32
9193	Feb 88	Leics	J	M	0.33	0.43	3.74	0.23
9223	Feb 88	Orkney	J	F	0.03	0.15	6.42	14.21
9224	Mar 88	Orkney	J	M	6.49	0.11	10.87	13.78
9226	Mar 88	Cambs	A	M	0.24	0.92	0.73	1.41
9230	Mar 88	Glos	A	M	0.21	0.39	1.23	0.12
9255	Apr 88	N'hants	A	F	0.21	0.42	ND	1.03
9270	May 88	Cambs	J	F	0.28	1.22	1.06	1.56
9279	Jun 88	Leics	A	M	0.29	0.66	0.68	0.29
9288	Jul 88	Worcs	J	M	0.32	0.55	ND	2.05
9298	Jul 88	G. London	J	F	1.89	0.40	10.09	1.24
9302	Jul 88	Cambs	J	F	ND	0.58	0.55	0.80
9305	Jul 88	N'hants	J	F	0.63	0.68	0.64	1.65
9306	Jul 88	Essex	J	M	0.41	0.20	20.60	0.41
9323	Aug 88	Co. Ferm	J	M	0.22	0.26	5.31	2.12
9368	Oct 88	Staffs	-	-	5.12	ND	45.95	1.04
9374	Oct 88	Gramp	J	F	4.43	0.93	6.05	1.22
9382	Nov 88	Aberdeens	J	M	0.23	0.58	18.42	2.75
9397	Dec 88	Aberdeens	J	M	0.36	0.33	2.18	3.38
9398	Dec 88	Aberdeens	J	M	ND	ND	0.54	1.01
Sparrowhawk (<u>Accipiter nisus</u>)								
9180	Oct 82	Gwent	J	F	0.25	0.08	1.87	1.84
9179	Feb 83	Gwent	J	F	1.54	0.98	4.11	3.85
9181	Nov 83	Gwent	J	F	1.15	0.49	1.36	2.80
9207	May 85	Grampian	A	M	8.72	0.35	2.70	3.93
9208	Apr 86	Grampian	J	M	37.80	0.97	32.43	1.86
9337	Jul 87	Aberdeens	J	M	0.74	0.31	1.11	0.63
9293	Aug 87	Humberside	J	M	ND	0.26	1.29	0.91
9292	Dec 87	Beds	J	F	0.69	0.24	5.57	1.11
9177	Jan 88	Norfolk	J	F	30.11	2.90	26.09	20.02
9182	Jan 88	Gwent	A	M	13.71	0.82	22.89	4.17
9215	Jan 88	N. Yorks	A	F	1.04	0.16	4.44	3.83
9185	Feb 88	S'clyde	J	F	9.07	0.41	9.70	10.72
9194	Feb 88	Suffolk	J	M	3.13	0.23	1.60	2.46

9196	Feb 88	Herefords	J	7 F	1.06	0.07	0.75	2.12
9198	Feb 88	Staffs	J	M	0.76	0.05	1.40	1.54
9202	Feb 88	Beds	A	F	38.65	2.82	19.91	2.55
9203	Feb 88	Somerset	J	M	1.86	0.13	0.68	1.38
9210	Feb 88	Humberside	A	F	2.81	1.04	0.47	2.52
9211	Feb 88	Dyfed	A	M	1.34	0.11	3.18	3.77
9213	Feb 88	D & G	J	M	9.50	0.04	4.86	9.27
9214	Feb 88	Lancs	A	F	0.88	0.25	0.68	5.03
9233	Feb 88	Kent	A	M	208.00	5.00	97.72	1.76
9247	Feb 88	Suffolk	A	F	1.99	0.19	6.87	3.39
9281	Feb 88	Lincs	A	F	55.55	2.61	66.80	7.92
9229	Mar 88	Powys	J	M	1.61	0.27	1.23	4.64
9231	Mar 88	Lincs	A	F	19.10	4.26	18.53	9.41
9232	Mar 88	Aberdeens	J	F	31.23	0.61	12.85	4.85
9235	Mar 88	Fife	A	M	37.28	2.96	47.24	8.33
9237	Mar 88	D & G	A	F	4.18	0.78	8.08	15.90
9238	Apr 88	Kent	A	F	19.48	0.71	22.22	5.45
9239	Apr 88	Lincs	J	F	1.62	1.40	15.37	3.55
9240	Apr 88	Grampian	J	F	3.38	0.29	4.56	7.16
9242	Apr 88	Herts	J	M	10.71	1.29	54.57	5.06
9243	Apr 88	Borders	A	F	44.81	1.51	46.33	9.07
9246	Apr 88	Hants	A	F	8.03	1.62	34.97	3.59
9252	Apr 88	IOW	A	M	12.21	1.83	55.44	7.00
9256	Apr 88	Kent	A	F	128.00	2.25	32.93	5.06
9257	May 88	Aberdeens	A	M	38.58	0.99	31.28	3.28
9258	May 88	Wilts	J	M	3.62	0.52	12.68	4.97
9259	May 88	Humbers	J	M	2.18	0.55	4.29	2.21
9284	Jun 88	Notts	J	M	4.64	0.70	8.68	1.44
9287	Jun 88	Staffs	A	F	1.02	0.48	9.46	2.16
9303	Jul 88	N'hants	A	M	0.74	0.14	5.15	4.51
9310	Jul 88	Leics	A	F	4.67	0.74	8.00	2.71
9311	Aug 88	Perths	J	M	1.48	0.27	2.94	3.65
9315	Aug 88	Worcs	J	F	0.18	0.13	2.22	0.84
9319	Aug 88	Norfolk	J	M	33.44	0.51	21.11	6.79
9324	Aug 88	N'umberland	J	F	0.45	0.13	2.92	2.15
9325	Aug 88	Hants	J	F	0.65	0.18	1.96	3.47
9326	Aug 88	W. Midlands	J	M	0.37	0.22	ND	1.18
9344	Aug 88	Aberdeens	J	M	0.87	0.12	ND	2.32
9330	Sep 88	D. & G.	J	F	0.47	0.22	ND	4.02
9331	Sep 88	Bucks	J	F	0.27	0.13	ND	2.03
9332	Sep 88	Staffs	J	F	0.17	0.16	ND	0.96
9335	Sep 88	Somerset	J	M	1.15	0.51	4.93	1.58
9336	Sep 88	Norfolk	J	F	0.90	0.16	ND	2.31
9363	Sep 88	Devon	J	M	1.24	0.52	3.26	5.69
9361	Oct 88	Bucks	A	M	1.63	0.37	4.80	0.97
9362	Oct 88	Salop	J	F	0.20	0.19	ND	2.27
9364	Oct 88	Staffs	J	F	1.05	0.21	ND	0.79
9375	Oct 88	-	J	F	0.24	0.13	ND	1.29
9377	Oct 88	Powys	J	F	0.09	0.11	ND	4.42
9378	Nov 88	Notts	A	M	1.01	0.17	3.55	1.03
9379	Nov 88	Somerset	J	F	2.64	0.59	8.89	2.17
9381	Nov 88	Cambs	A	F	2.41	0.25	ND	0.93
9387	Nov 88	Avon	A	F	16.96	1.04	12.39	1.74
9388	Nov 88	W. Midlands	J	M	0.47	0.22	ND	0.58
9402	Dec 88	Herts	A	F	2.13	1.02	12.86	1.04
9403	Dec 88	N. Yorks	A	F	8.78	11.43	51.55	7.09

8

Peregrine falcon (Falco peregrinus)

9221	May 86	Orkney	A	M	4.38	ND	6.95	14.31
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Merlin (Falco columbarius)

*8956	Apr 87	W. Glam	A	F	49.12	3.42	430.40	26.68
8959	Apr 87	Derbyshire	A	M	1.85	0.44	9.85	1.31
9204	Jul 87	Grampian	J	F	ND	ND	1.33	1.40
9205	Jul 87	Grampian	J	F	0.11	0.01	0.20	1.23
9206	Jul 87	Grampian	J	F	0.19	0.02	0.31	1.12
9158	Aug 87	Orkney	A	F	0.32	0.07	0.53	4.12
9216	Feb 88	S'clyde	J	F	6.32	0.98	8.79	1.45
9220	Mar 88	Orkney	A	F	2.29	ND	6.61	6.04
9322	Aug 88	Humberside	J	M	4.81	0.22	4.94	8.20
9338	Sep 88	Aberdeens	A	F	1.07	0.22	0.74	5.39
9339	Sep 88	Tayside	J	M	1.30	0.42	0.34	1.62
9340	Sep 88	Aberdeens	J	F	0.20	0.09	ND	0.68
9341	Sep 88	Aberdeens	P	-	0.22	0.11	ND	0.88
9342	Sep 88	Aberdeens	P	-	0.84	0.08	0.50	1.48

Buzzard (Buteo buteo)

9234	Mar 88	W. Glam	J	F	0.07	31.87	0.27	0.80
9254	Apr 88	W. Isles	A	M	0.59	ND	ND	2.09
9391	Dec 88	Devon	J	M	0.14	ND	2.37	0.79

Red Kite (Milvus milvus)

9291	Jul 88	S. Humberside	A	F	9.44	ND	71.63	5.28
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European Eagle Owl (Bubo bubo)

9172	Jan 88	Beds	A	M	1.48	ND	0.57	0.13
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Little Owl (Athene noctua)

9275	Jul 87	Norfolk	J	F	3.23	ND	ND	1.35
9300	Jul 88	S Glam	A	F	ND	0.12	0.44	1.05
9301	Jul 88	W Yorks	J	M	0.13	0.14	ND	0.69
9390	Dec 88	Wilts	A	F	0.13	0.15	ND	0.46

Long-eared Owl (Asio otus)

9209	Oct 85	S'clyde	A	F	0.47	0.05	0.55	0.22
9277	Jul 87	Suffolk	J	F	5.81	2.76	5.53	3.57
9309	Apr 88	Aberdeens	-	M	0.61	0.26	ND	0.77
9373	Oct 88	S Clyde	A	M	0.80	0.27	ND	1.41

Short-eared Owl (Asio flammeus)

9272	Mar 88	Orkney	A	M	1.27	ND	1.44	1.41
9271	May 88	Orkney	-	M	ND	0.23	ND	0.30
9283	Jun 88	Cumbria	A	-	85.98	1.07	TO FOL	33.30
9314	Aug 88	Orkney	A	M	0.49	ND	3.95	1.54
9359	Sep 88	Orkney	J	M	0.98	ND	16.31	3.06
9404	Dec 88	Essex	-	-	0.77	0.81	6.23	2.63

Nightjar (Caprimulgus europaeus)

9343	Sep 88	E. Sussex	J	F	ND	0.56	ND	1.13
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Stone Curlew (Burhinus oediconemus)

9261	Sep 88	Derbys	J	M	0.64	0.10	0.92	1.22
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Heron (Ardea cinerea)

9084	Oct 87	H & W	A	M	70.19	1.41	233.80	48.66
9112	Nov 87	Suffolk	A	M	0.68	ND	1.21	2.36
9176	Jan 88	S'clyde	A	F	0.96	0.07	1.12	11.87
9184	Jan 88	H'land	J	M	3.51	0.29	6.39	57.34
9219	Jan 88	Orkney	J	F	0.12	0.26	1.88	27.27
9199	Feb 88	Cambs	J	M	11.49	1.03	21.66	12.70
9212	Feb 88	S'clyde	A	M	0.68	0.03	6.90	9.44
9218	Mar 88	Essex	J	F	3.09	0.44	5.61	41.48
9241	Mar 88	Grampian	A	F	1.46	0.12	0.67	16.90
9249	Apr 88	Essex	A	M	0.89	0.42	2.72	6.98
9250	Apr 88	Berks	A	M	1.43	0.31	16.28	14.40
9273	Apr 88	Orkney	J	F	2.77	0.41	8.41	34.90
9318	Apr 88	Beds	A	F	1.04	ND	51.65	3.00
9299	Jul 88	Gwent	J	F	5.74	20.34	36.23	50.90
9376	Nov 88	Aberdeens	J	F	1.46	ND	2.83	4.49
9401	Dec 88	Herts	J	F	ND	0.10	ND	17.70

Great Crested Grebe (Podiceps cristatus)

9248	Apr 88	Suffolk	A	F	0.28	0.07	0.39	7.19
9280	Jun 88	Staffs	A	M	1.63	0.11	4.68	22.30
9345	Oct 88	G. London	A	F	1.55	0.16	4.52	6.95
9392	Dec 88	W. Yorks	A	F	1.43	0.19	7.21	8.73

Little Grebe (Podiceps ruficollis)

9147	Jan 88	Wilts			0.18	0.09	0.78	8.01
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Kingfisher (Alcedo atthis)

9096	Nov 87	Wilts	A	M	0.35	2.40	2.96	3.13
9227	Mar 88	Staffs	A	M	0.65	0.57	4.89	1.68
9282	Jun 88	Essex	J	F	0.82	0.70	2.43	2.94
9329	Sep 88	Cambs	A	M	0.43	0.54	ND	2.32

*This bird was exceptionally large and pale, with a winglength outside the range of British Merlins, and probably came from Northern Europe. This may account for its very different pollutant load.

Table 2. Geometric mean levels of pollutants in the various species in Table 1, but for 1988 specimens only. *

	pp'-DDE	HEOD	PCBs	Hg
<u>Kestrel</u>				
Mean	0.25	0.30	1.97	1.25
SD	0.82	0.69	0.93	0.47
Range within 1 SE	0.17 - 0.37	0.21 - 0.42	1.26 - 3.07	0.99 - 1.56
<u>Sparrowhawk</u>				
Mean	2.90	0.44	2.07	3.05
SD	0.76	0.53	1.30	0.34
Range within 1 SE	2.33 - 3.62	0.38 - 0.51	1.42 - 3.02	2.76 - 3.36
<u>Merlin</u>				
Mean	1.17	0.14	0.50	2.20
SD	0.55	0.59	1.17	0.41
Range within 1 SE	0.75 - 1.84	0.09 - 0.23	0.19 - 1.29	1.58 - 3.07
<u>Heron</u>				
Mean	1.11	0.19	3.83	15.91
SD	0.75	0.85	0.92	0.39
Range within 1 SE	0.70 - 1.77	0.11 - 0.32	2.17 - 6.78	12.54 - 20.18
<u>Great-crested Grebe</u>				
Mean	1.00	0.12	2.78	9.93
SD	0.37	0.19	0.58	0.24
Range within 1 SE	0.66 - 1.54	0.10 - 0.15	1.43 - 5.39	7.55 - 13.07
<u>kingfisher</u>				
Mean	0.61	0.60	0.49	2.25
SD	0.14	0.06	1.47	0.12
Range within 1 SE	0.51 - 0.74	0.55 - 0.65	0.06 - 3.48	1.92 - 2.65

* The Kestrel figures exclude the Irish bird from Co. Fermanagh, see Table 1 for residue levels, No. 9323

Table 3. Comparison of geometric mean residue levels (log values) from birds collected in 1987 and 1988; t-values are shown. Minus values indicate a decrease and plus values indicate an increase from 1987.

	pp'-DDE	HEOD	PCBs	Hg
Kestrel	$t_{48}=-0.77$	$t_{48}=+0.45$	$t_{48}=+0.33$	$t_{48}=+1.03$
Sparrowhawk	$t_{122}=+3.68^{***}$	$t_{122}=+3.86^{***}$	$t_{122}=+0.45$	$t_{122}=+1.98^{*}$
Kingfisher	$t_8=+0.33$	$t_8=-0.56$	$t_8=-0.06$	$t_8=-0.12$
Great-crested Grebe	$t_7=-0.73$	$t_7=+0.90$	$t_7=+0.27$	$t_7=+0.14$
Heron	$t_{29}=-0.50$	$t_{29}=-1.11$	$t_{29}=+0.44$	$t_{29}=+0.74$

Notes: Zero values were taken as 0.01 for all residues.

* significance of difference $P<0.05$; *** $P<0.001$

Table 4. Trends in pollutant levels in livers of predatory birds during 1963 - 1988 and 1981 - 1988. Figures show linear regression coefficients, with significance levels.

		1963-1988	1981-1988
DDE	Sparrowhawk	-0.020***	-0.031*
	Kestrel	-0.025***	-0.024 ns
	Heron	-0.023***	0.038 ns
	Kingfisher	-0.041***	-0.573 ns
	Great-crested Grebe	-0.009 ns	0.041 ns
PCBs	Sparrowhawk	0.001 ns	0.013 ns
	Kestrel	0.004 ns	0.036 ns
	Heron	-0.002 ns	0.053 ns
	Kingfisher	-0.023 ns	-0.047 ns
	Great-crested Grebe	-0.030*	0.019 ns
HEOD	Sparrowhawk	-0.009*	-0.013 ns
	Kestrel	-0.024***	-0.010 ns
	Heron	-0.034***	0.077*
	Kingfisher	-0.018*	-0.020 ns
	Great-crested Grebe	-0.011 ns	0.076 ns
Hg	Sparrowhawk	-0.032***	-0.011 ns
	Kestrel	-0.064***	-0.017 ns
	Heron	-0.025***	0.063**
	Kingfisher		-0.001 ns
	Great-crested Grebe		0.022 ns

Notes: Analyses for Hg in Sparrowhawk, Kestrel and Heron were started in 1970, in Kingfisher in 1980, and in Great-crested Grebe in 1979.

Analyses for PCBs in Sparrowhawk, Kestrel and Heron were started in 1967, and in Kingfisher and Great-crested Grebe in 1968.

ns = not significant; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Figure 1. Levels of DDE in livers of individual Sparrowhawks, 1963 - 1988.
Line shows 3-year moving geometric mean.

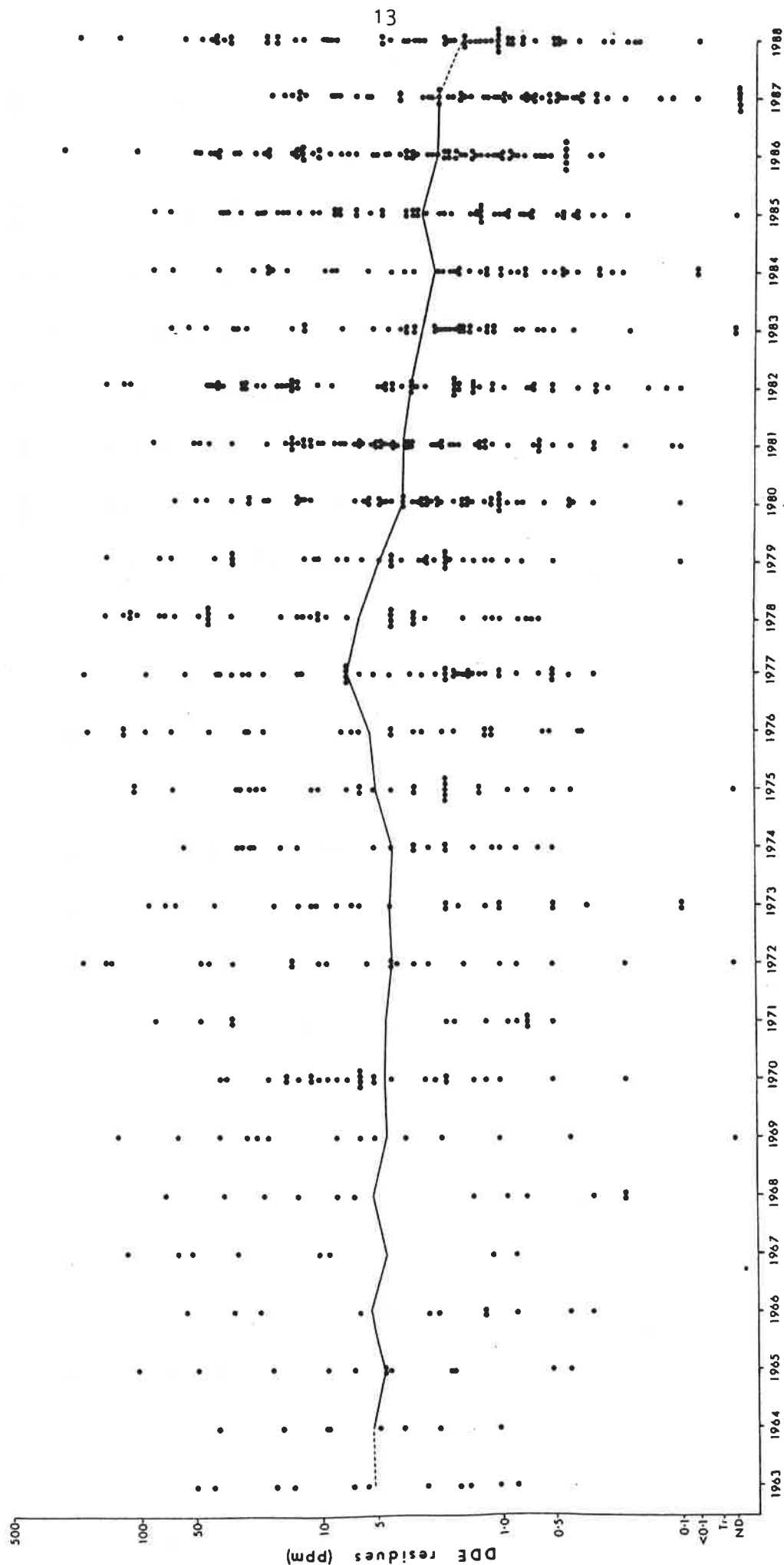


Figure 2. Levels of DDE in livers of individual Kestrels, 1963 - 1988.
Line shows 3-year moving geometric mean.

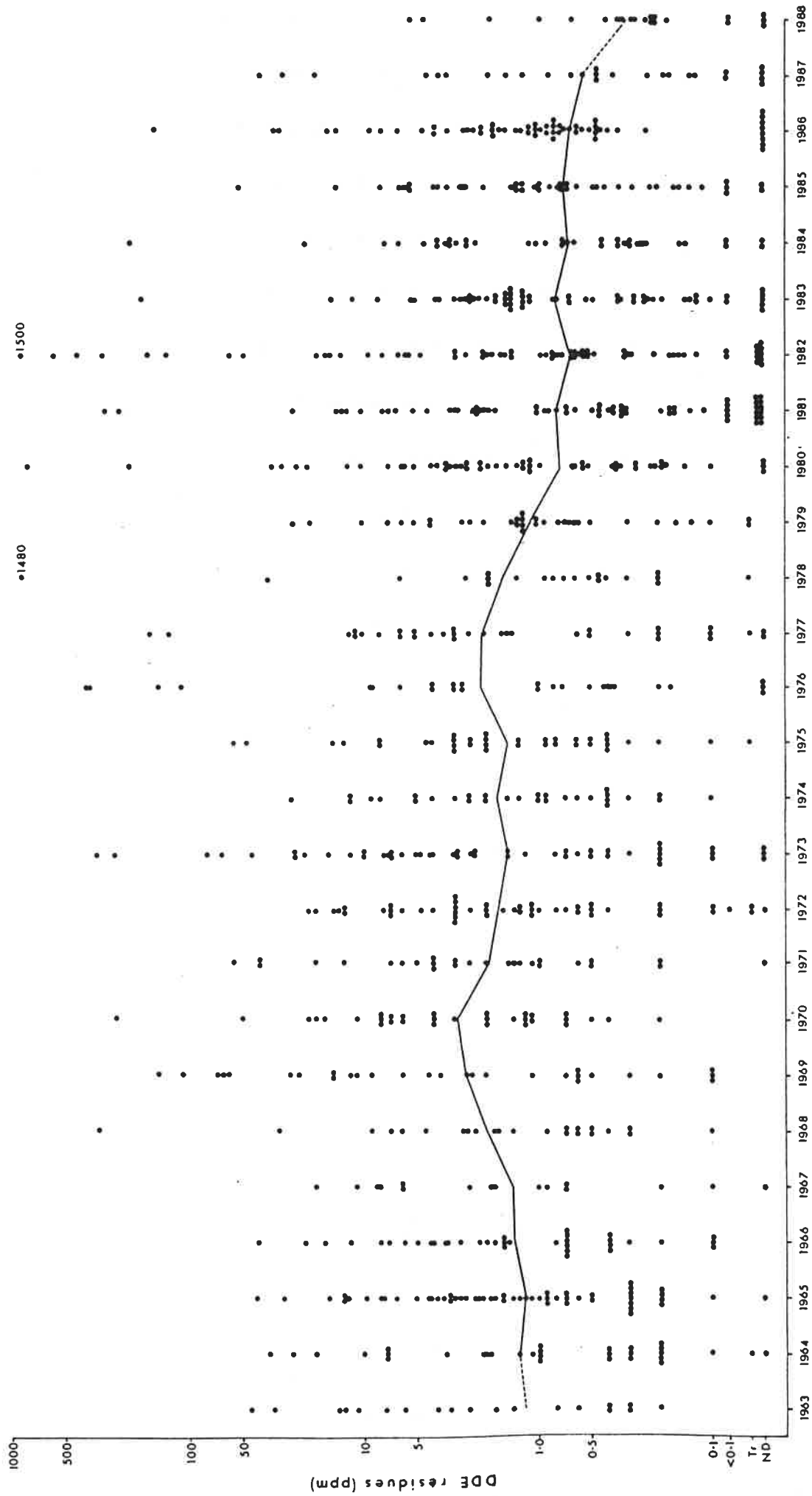


Figure 3. Levels of DDE in livers of individual Herons, 1963 - 1988.
Line shows 3-year moving geometric mean.

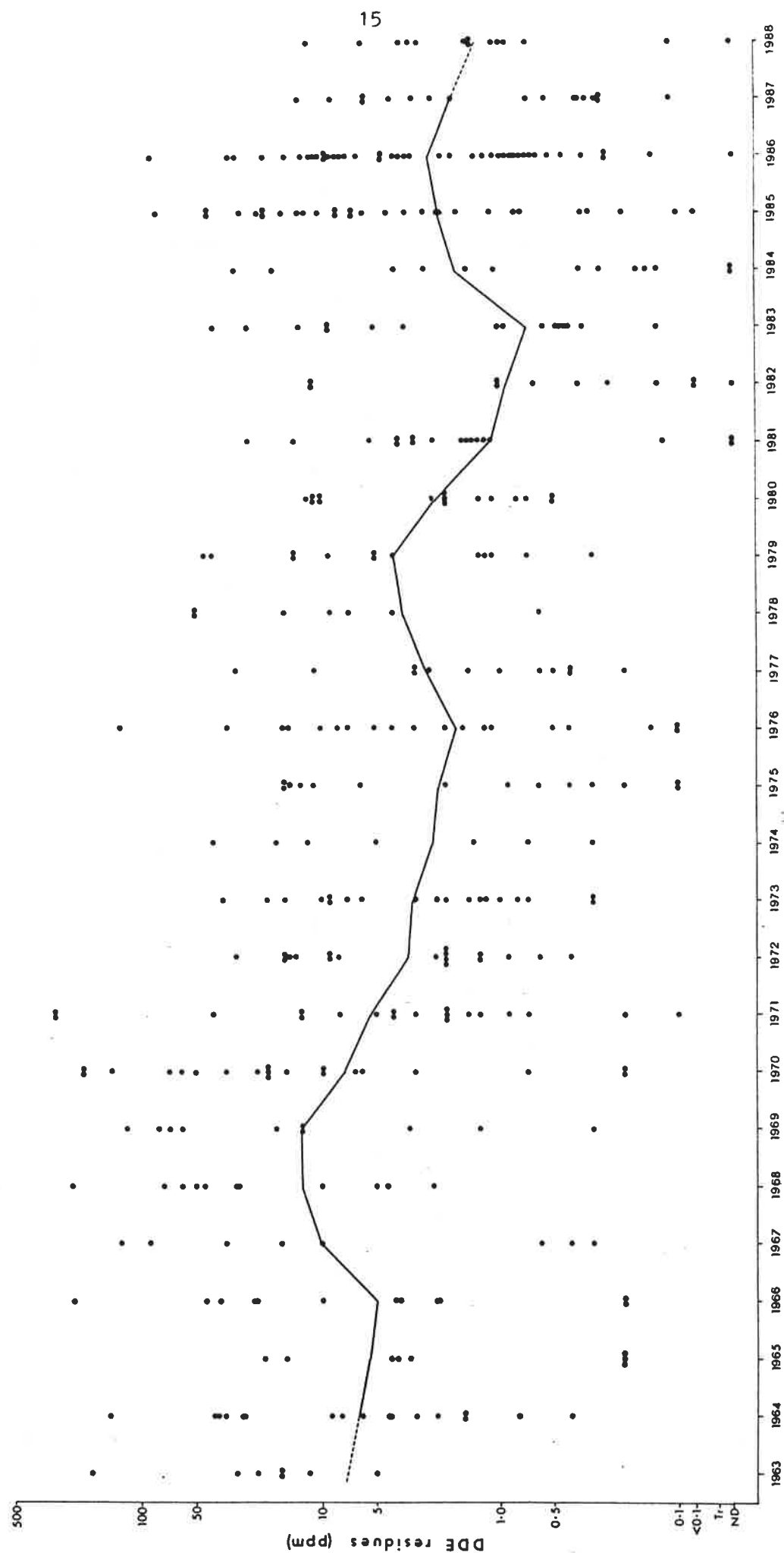


Figure 4. Levels of DDE in livers of individual Kingfishers, 1964 - 1988.
Line shows 3-year moving geometric mean.

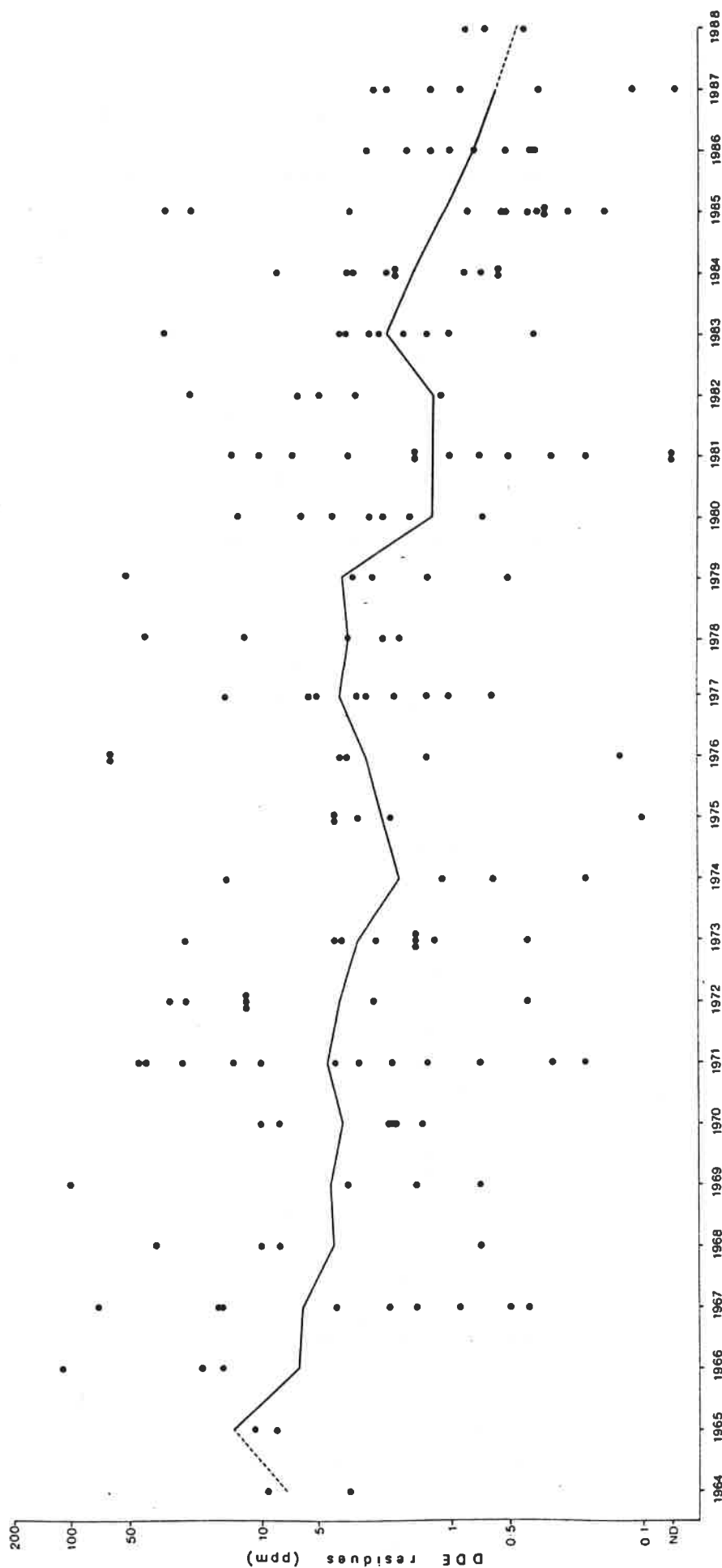


Figure 5. Levels of DDE in livers of individual Great-crested Grebes, 1963 - 1988. Line shows 3-year moving geometric mean.

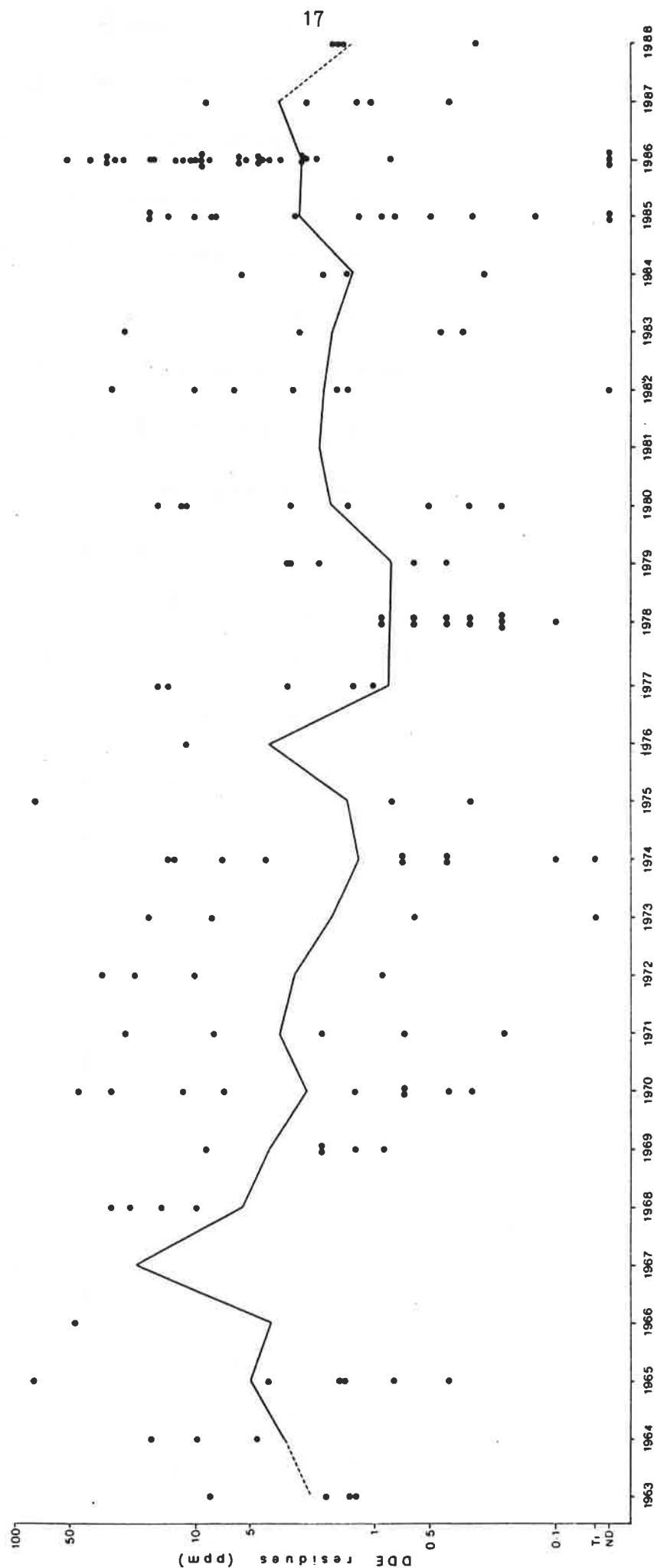


Figure 6. Levels of HEOD in livers of individual Sparrowhawks, 1963 - 1988.
Line shows 3-year moving geometric mean.

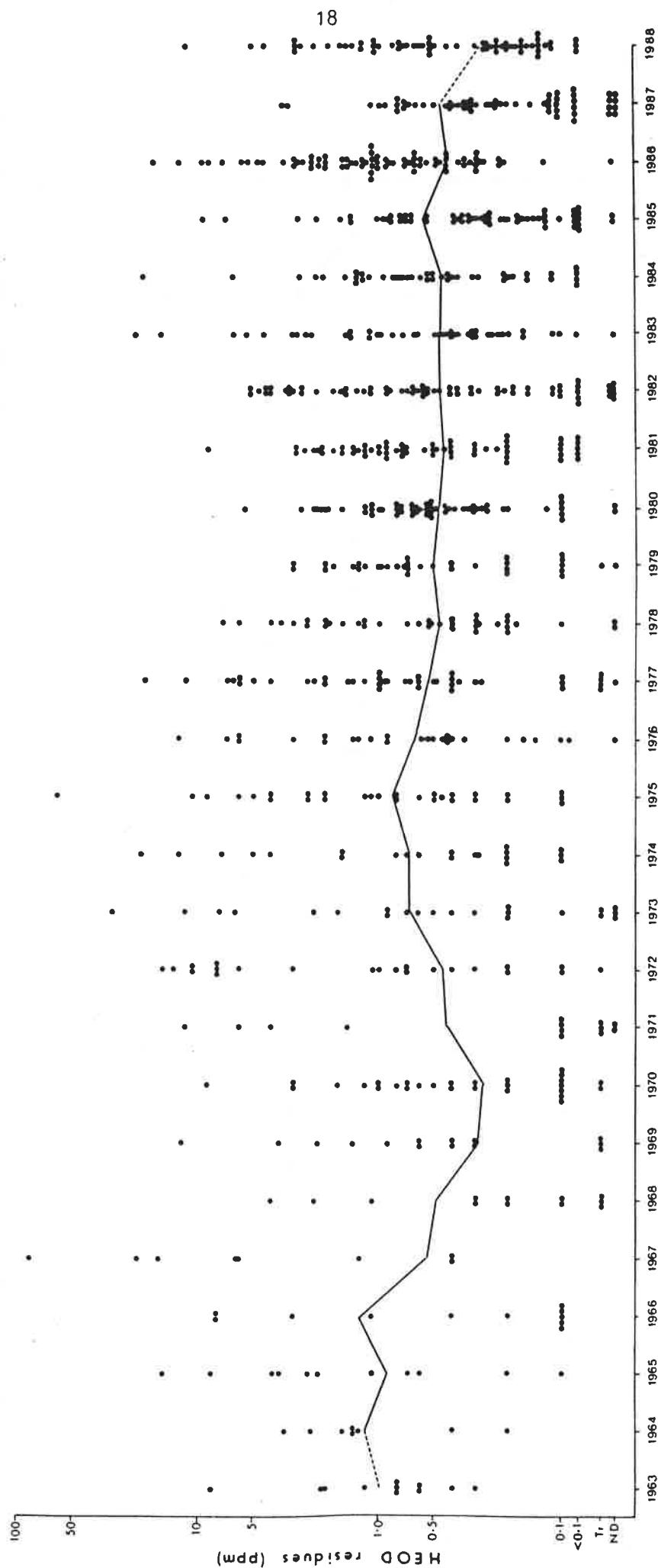


Figure 7. Levels of HEOD in livers of individual Kestrels, 1963 - 1988.
Line shows 3-year moving geometric mean.

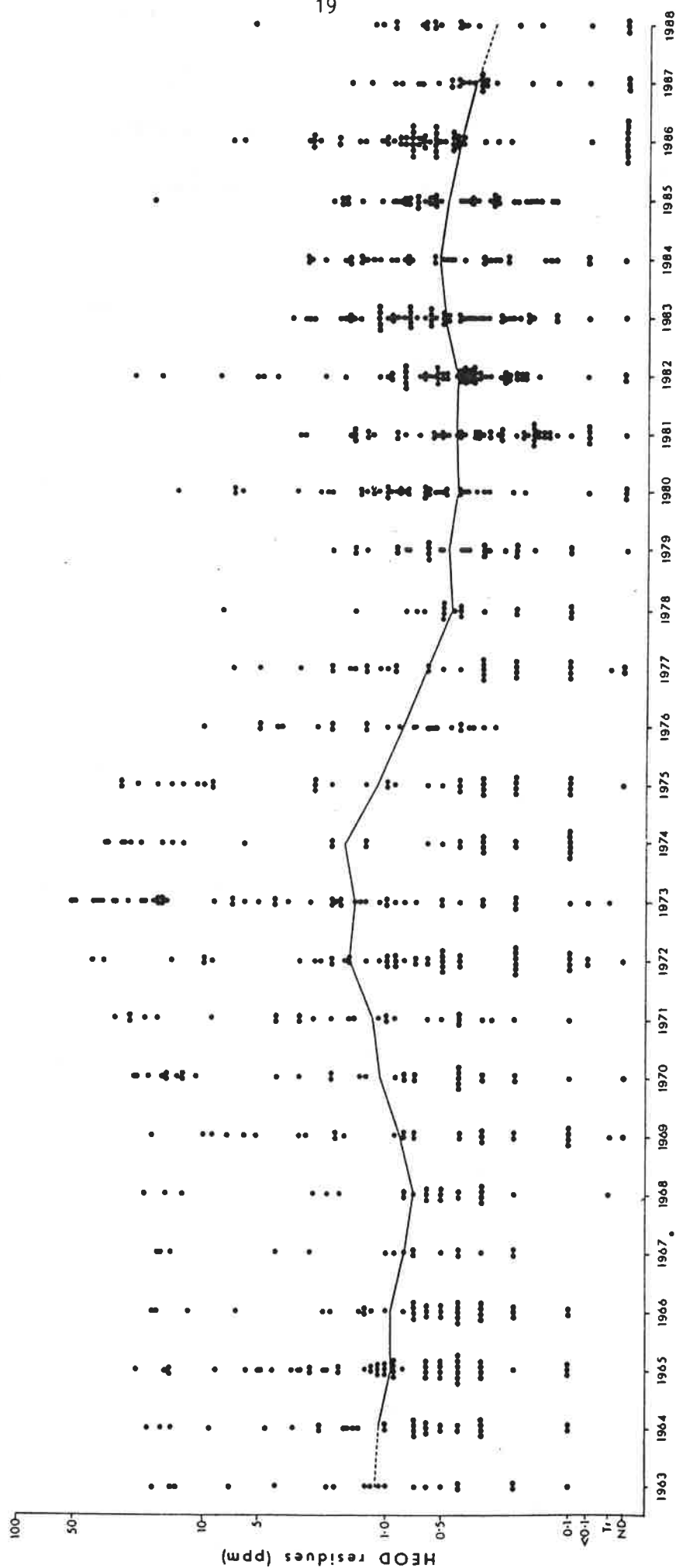


Figure 8. Levels of HEOD in livers of individual Herons, 1963 - 1988.
Line shows 3-year moving geometric mean.

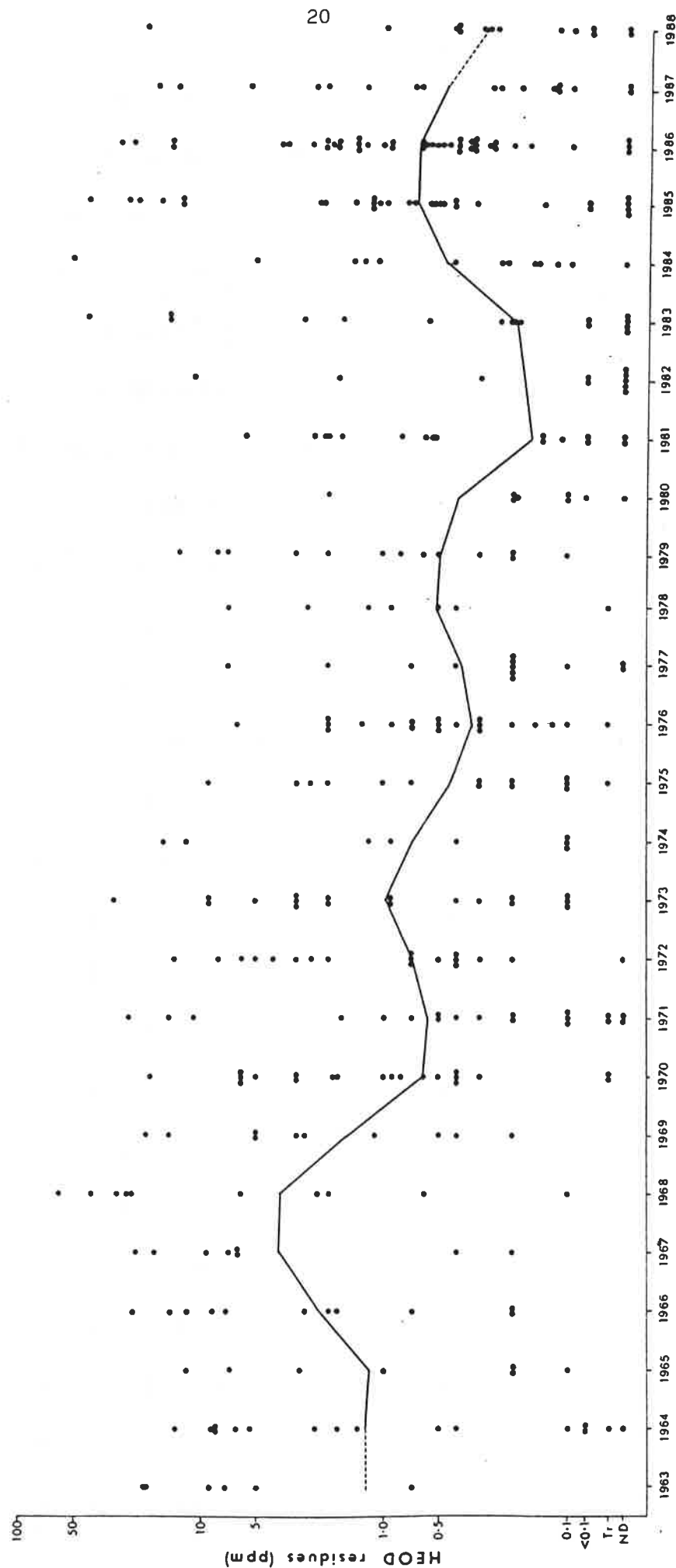


Figure 9. Levels of HEOD in livers of individual Kingfishers, 1964 - 1988.
Line shows 3-year moving geometric mean.

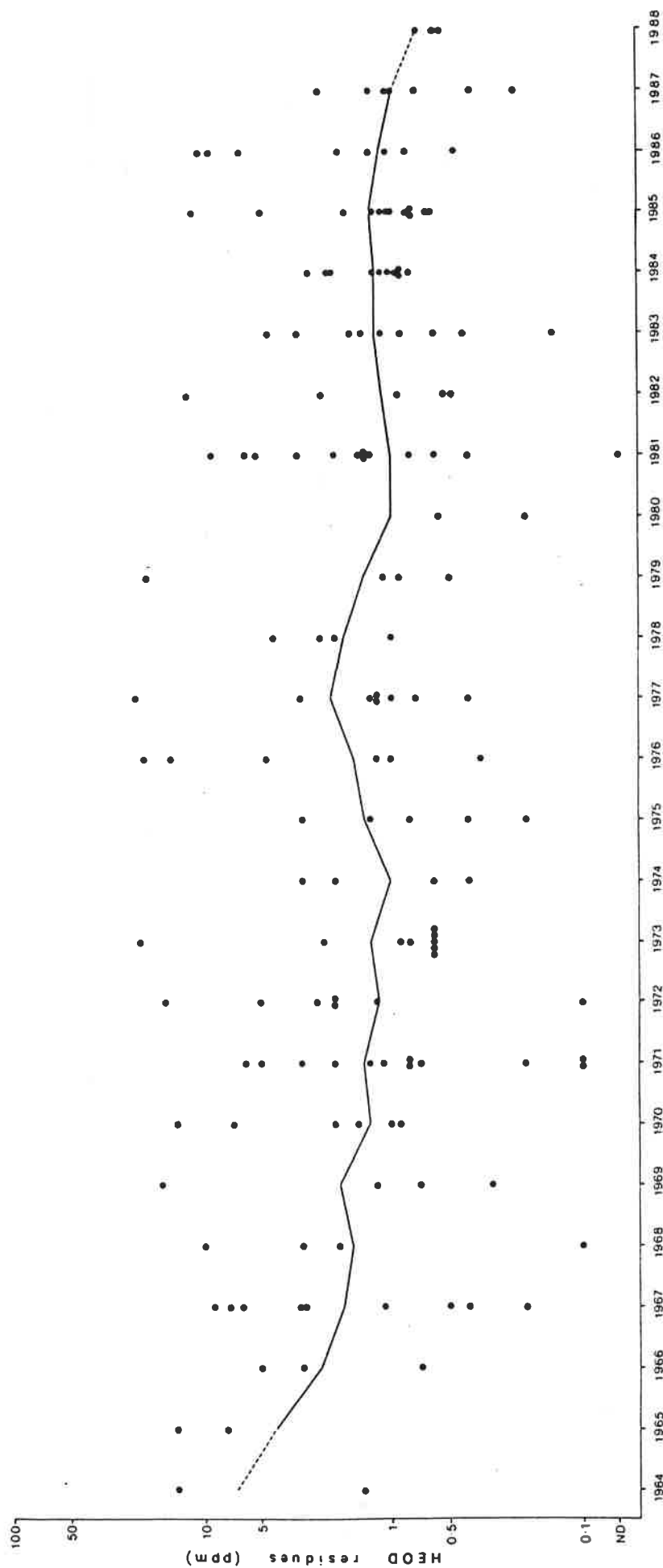


Figure 10. Levels of HEOD in livers of individual Great-crested Grebes, 1963 - 1988. Line shows 3-year moving geometric mean.

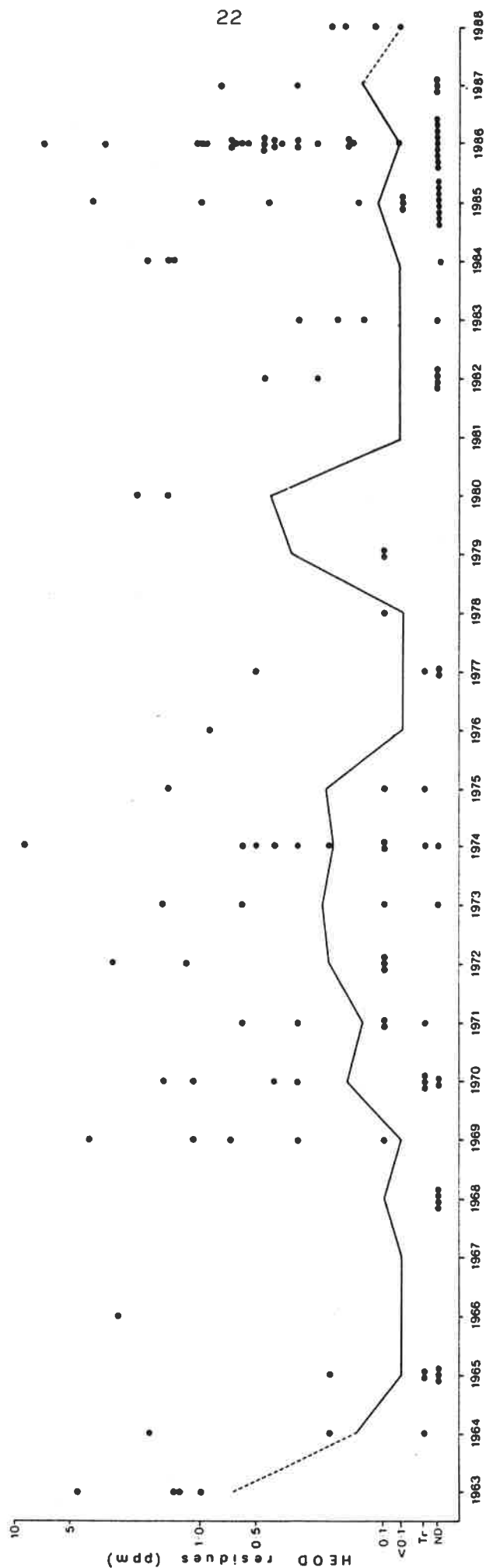


Figure 11. Levels of PCBs in livers of individual Sparrowhawks, 1967 - 1988.
Line shows 3-year moving geometric mean.

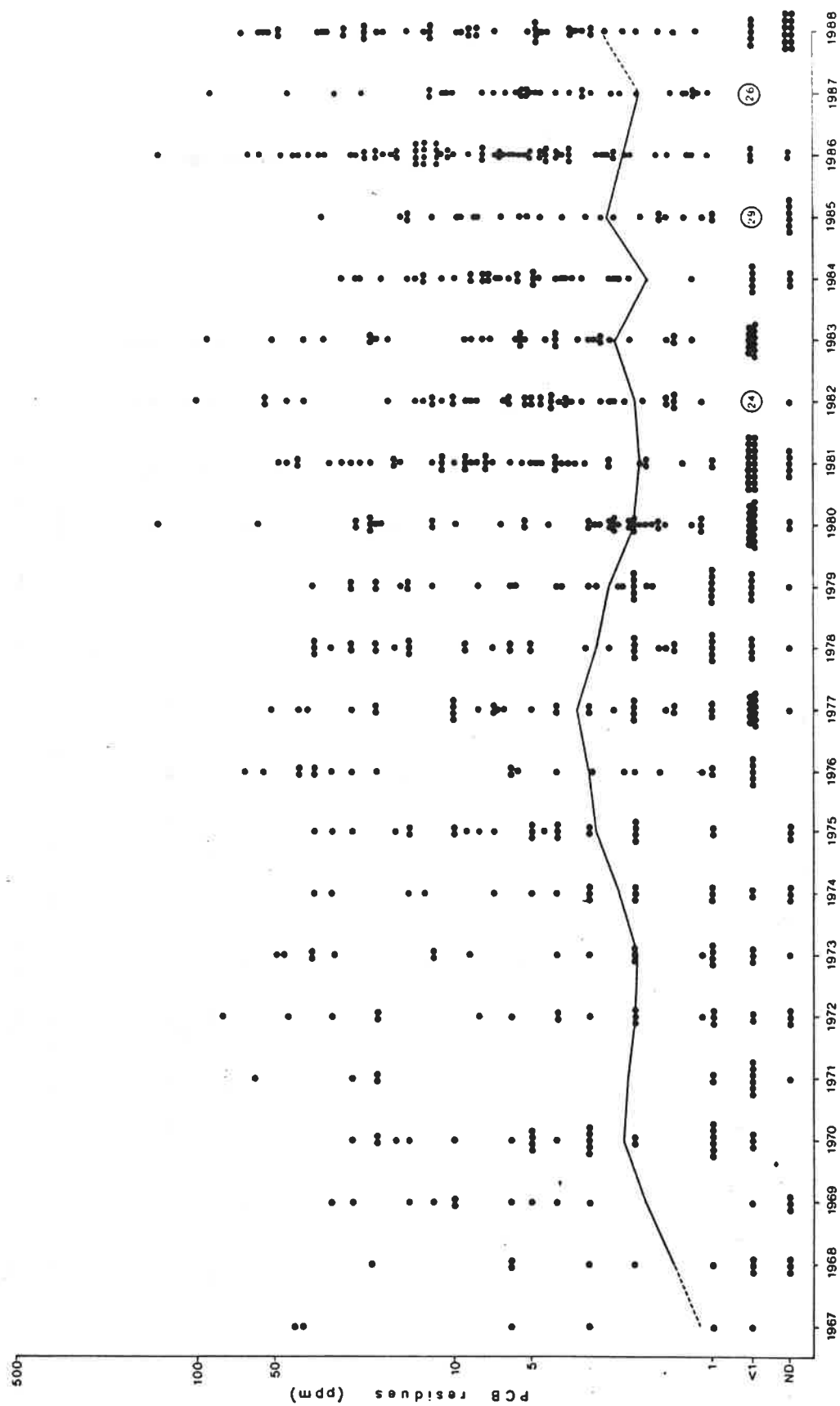


Figure 12. Levels of PCBs in livers of individual Kestrels, 1967 - 1988.
Line shows 3-year moving geometric mean.

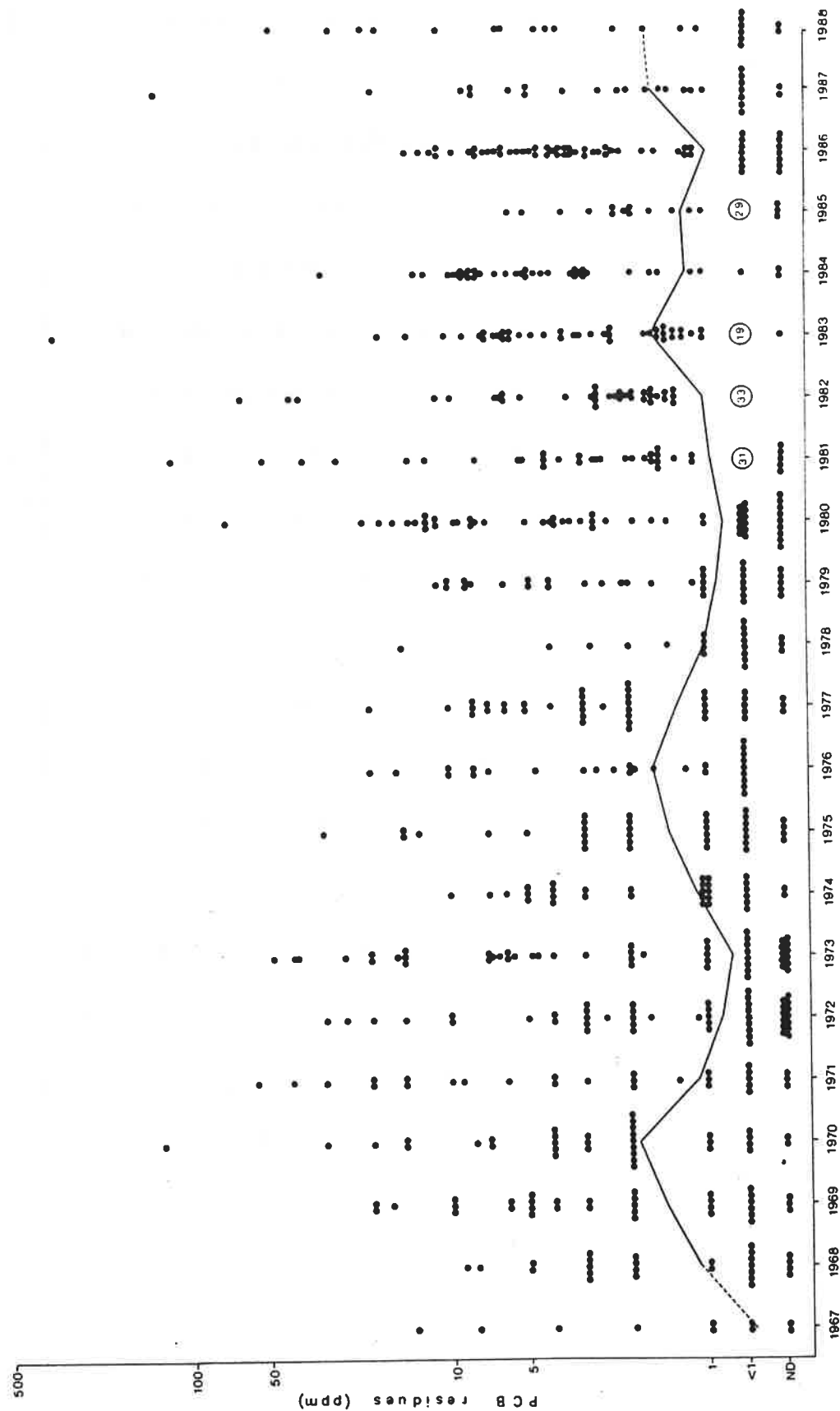


Figure 13. Levels of PCBs in livers of individual Herons, 1967 - 1988.
Line shows 3-year moving geometric mean.

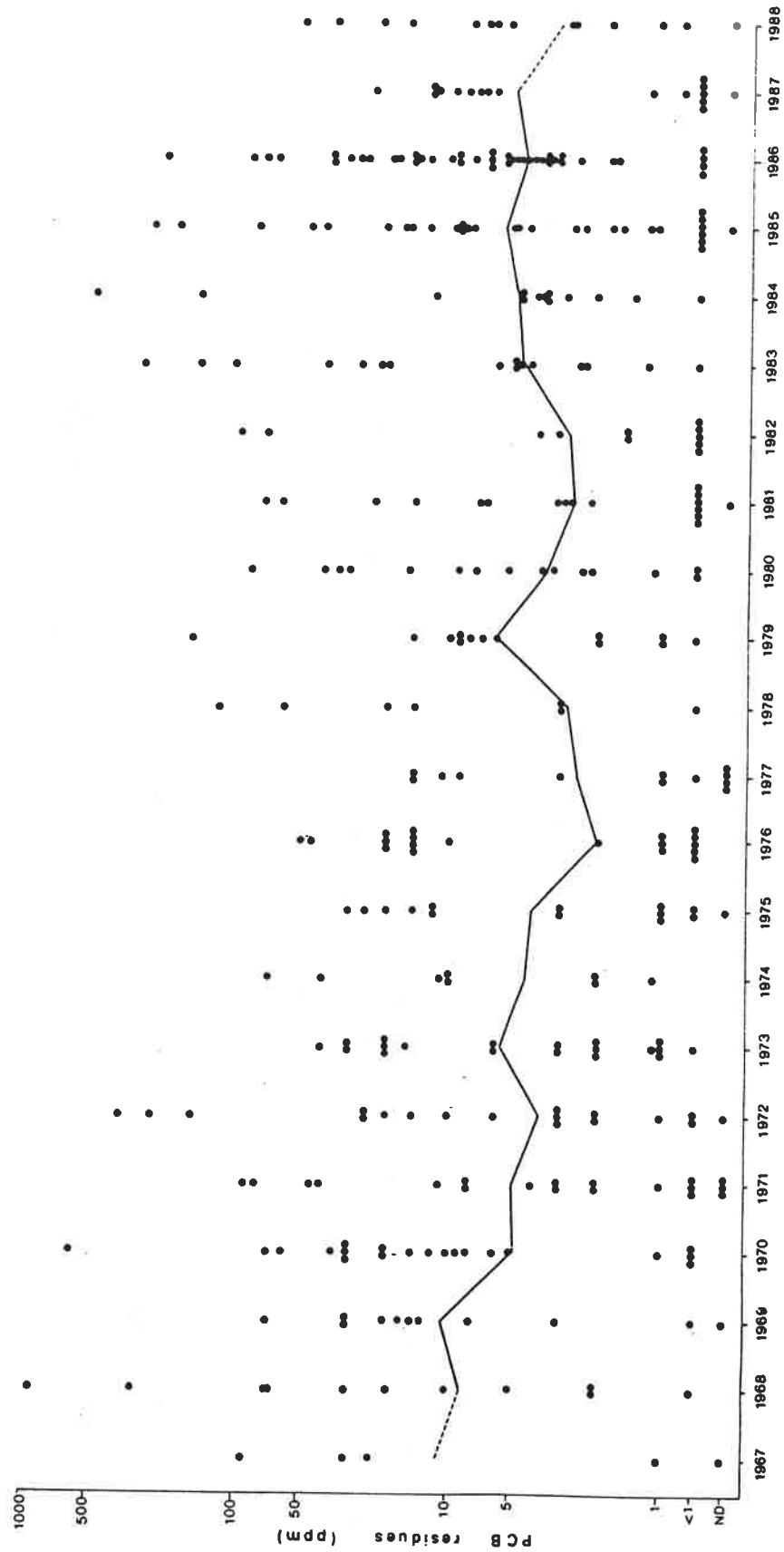


Figure 14. Levels of PCBs in livers of individual Kingfishers, 1967 - 1988. Line shows 3-year moving geometric mean.

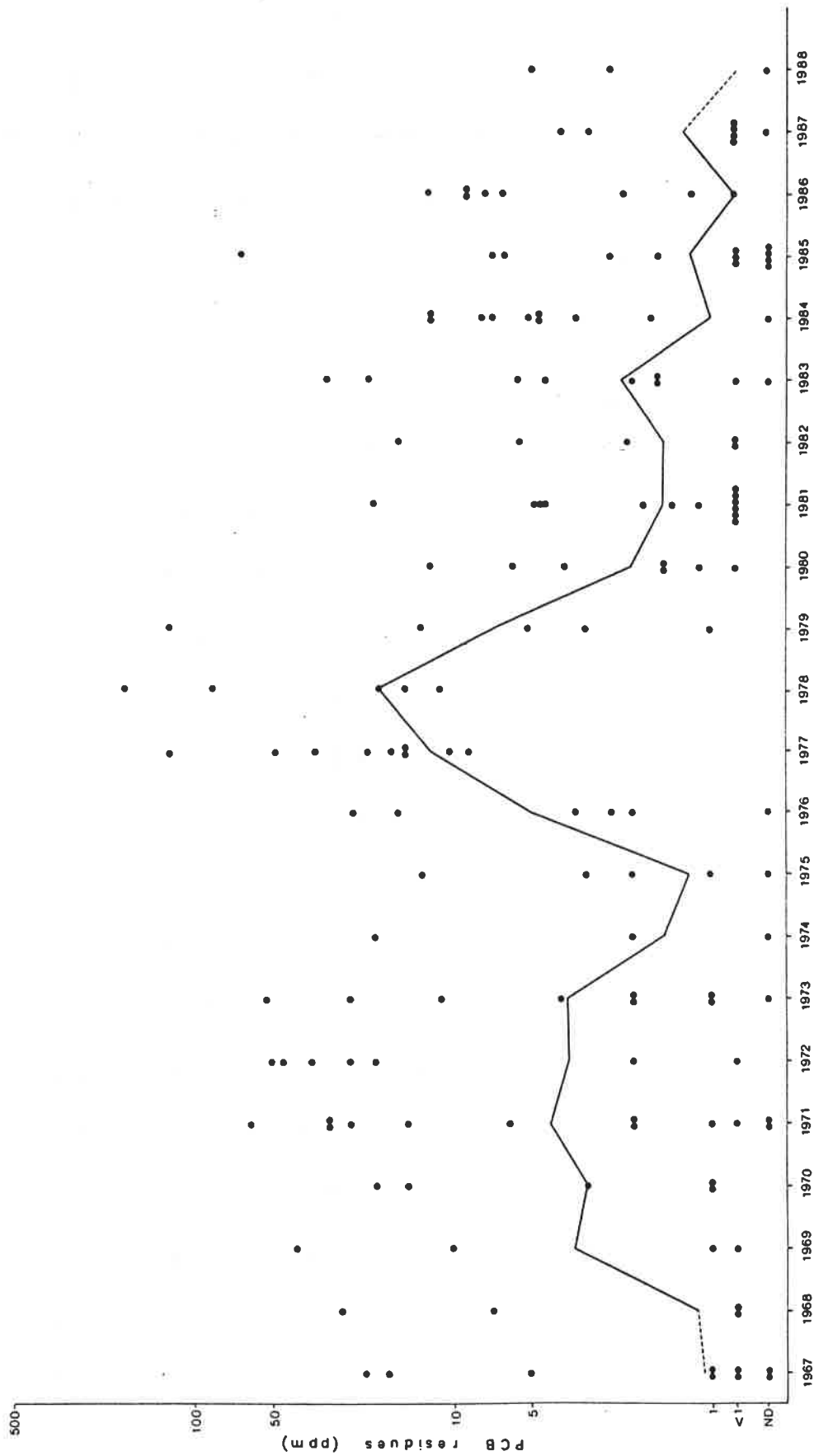


Figure 15. Levels of PCBs in livers of individual Great-crested Grebes, 1968 - 1988. Line shows 3-year moving geometric mean.

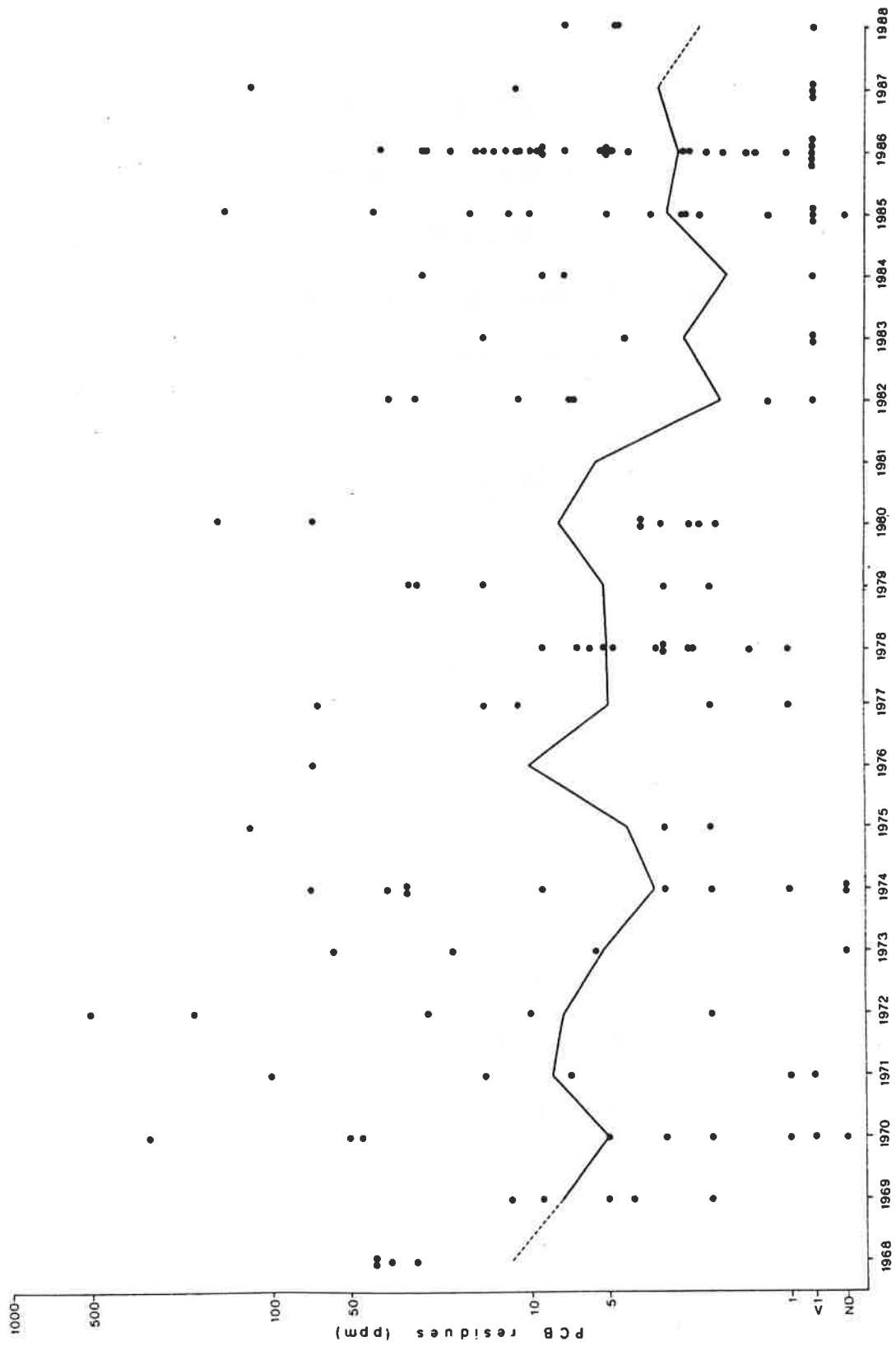


Figure 16. Levels of mercury (Hg) in livers of individual Sparrowhawks, 1970 - 1988. Line shows 3-year moving geometric mean.

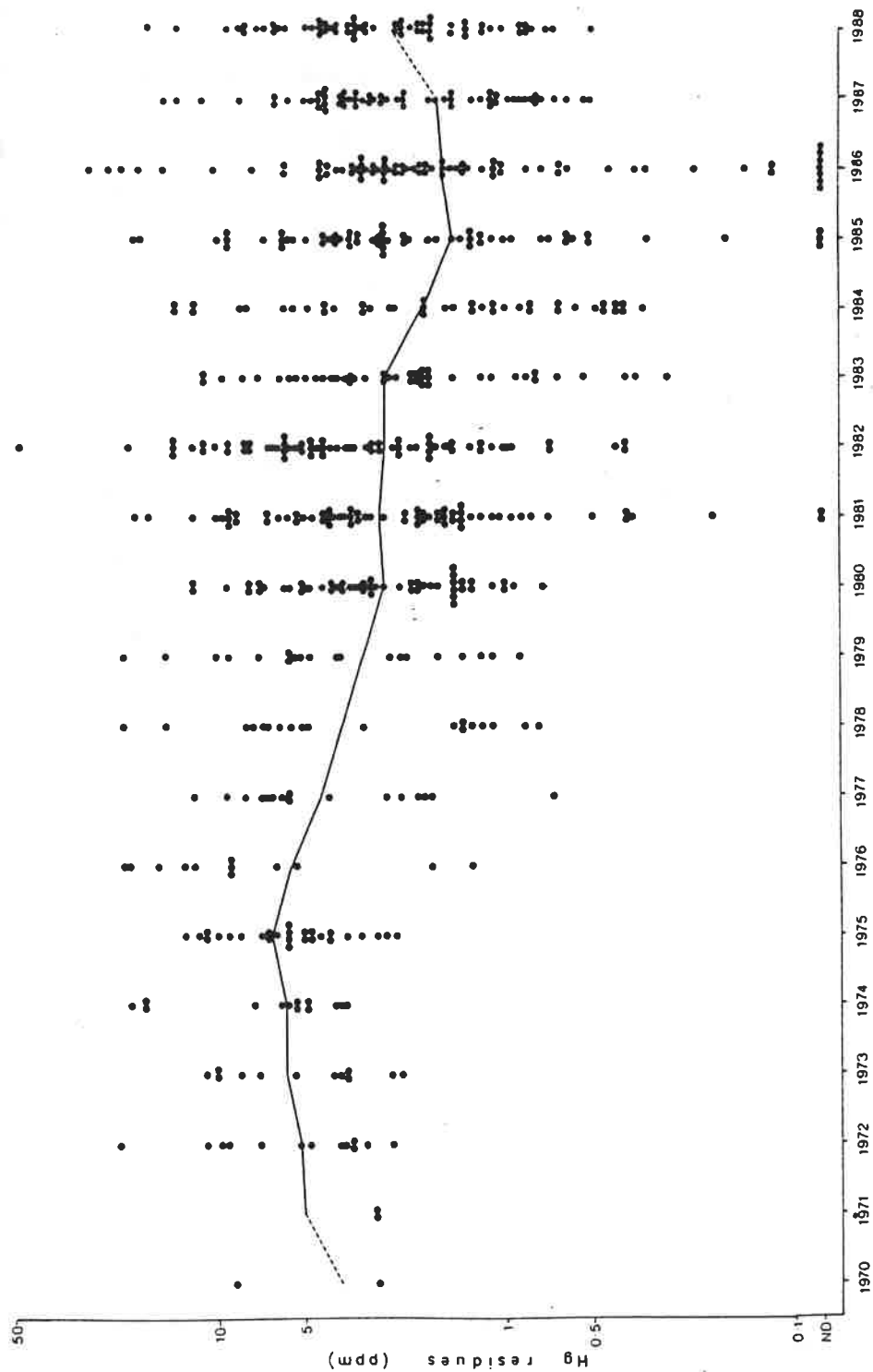


Figure 17. Levels of mercury (Hg) in livers of individual Kestrels, 1970 - 1988.
Line shows 3-year moving geometric mean.

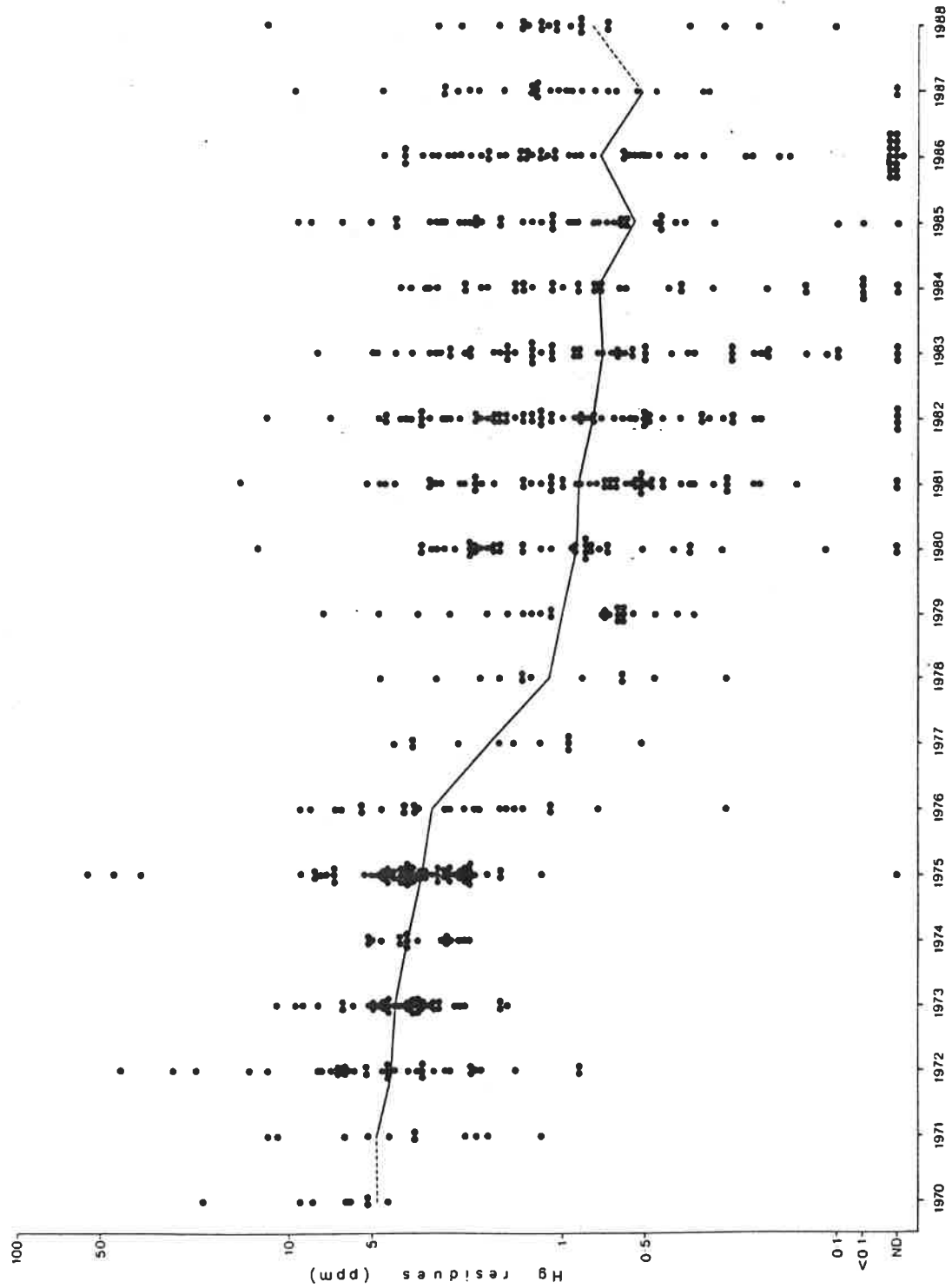


Figure 18. Levels of mercury (Hg) in livers of individual Herons, 1969 - 1988.
Line shows 3-year moving geometric mean.

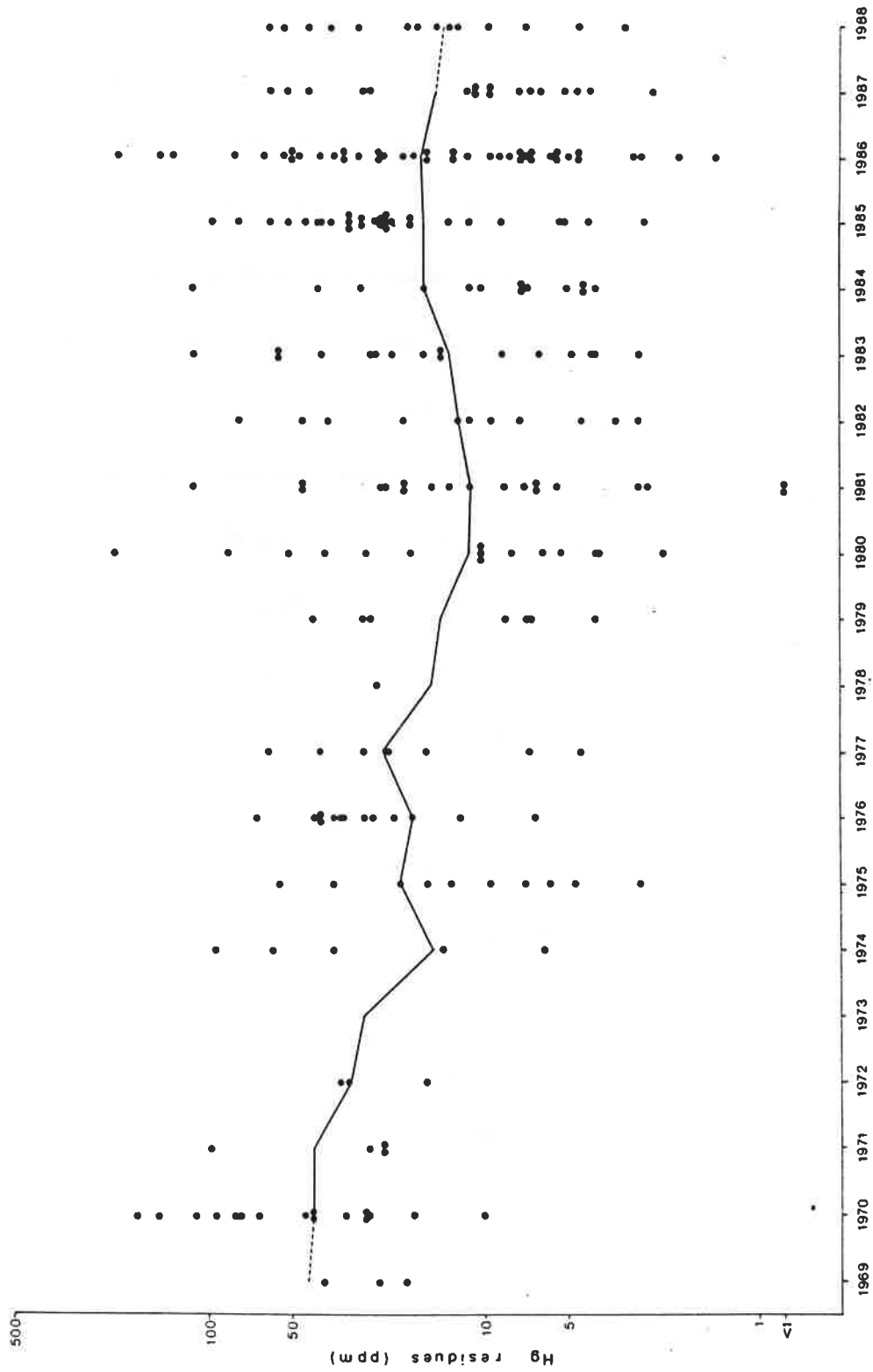


Figure 19. Levels of mercury (Hg) in livers of individual Kingfishers, 1980 - 1988. Line shows 3-year moving geometric mean.

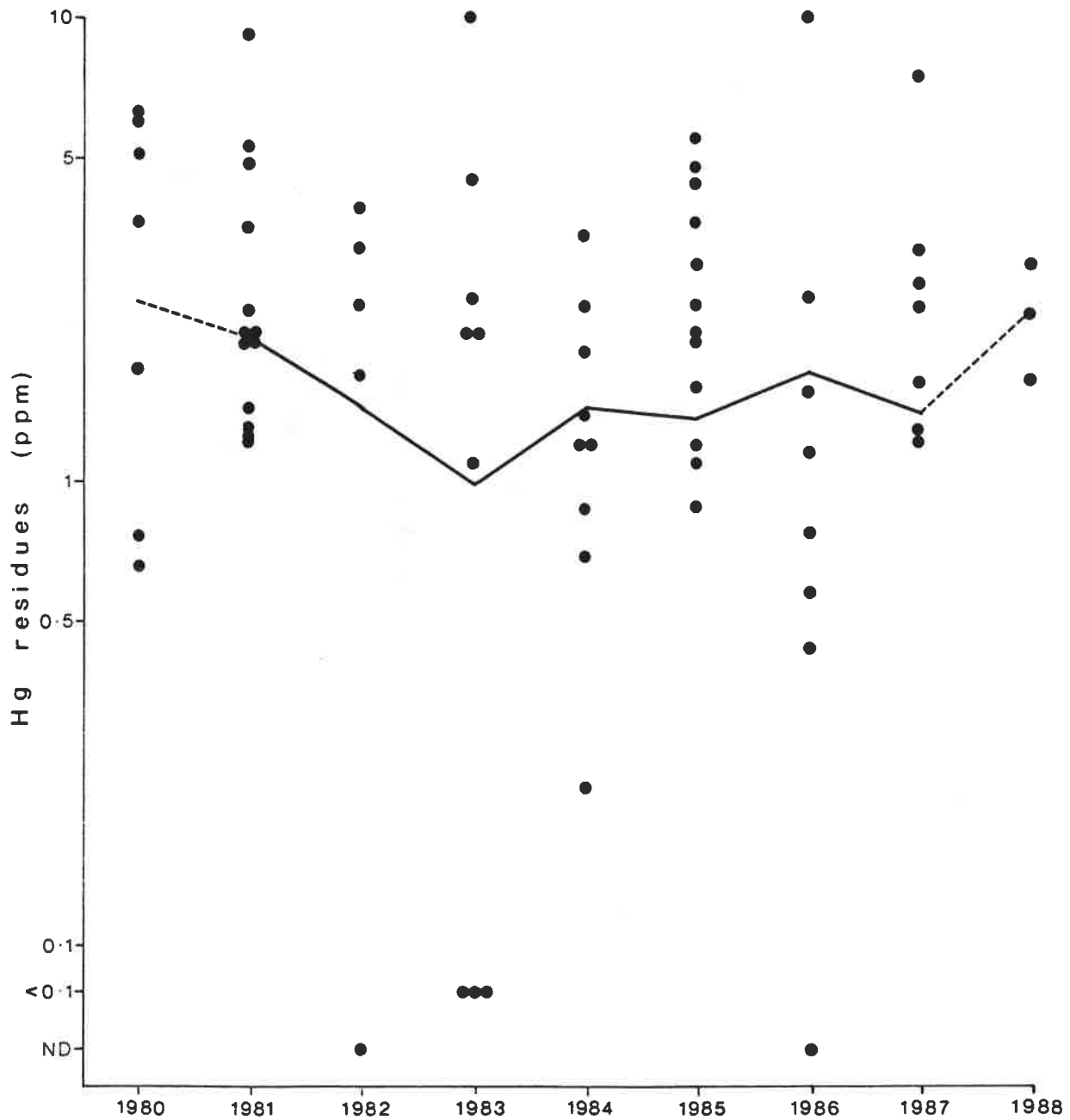
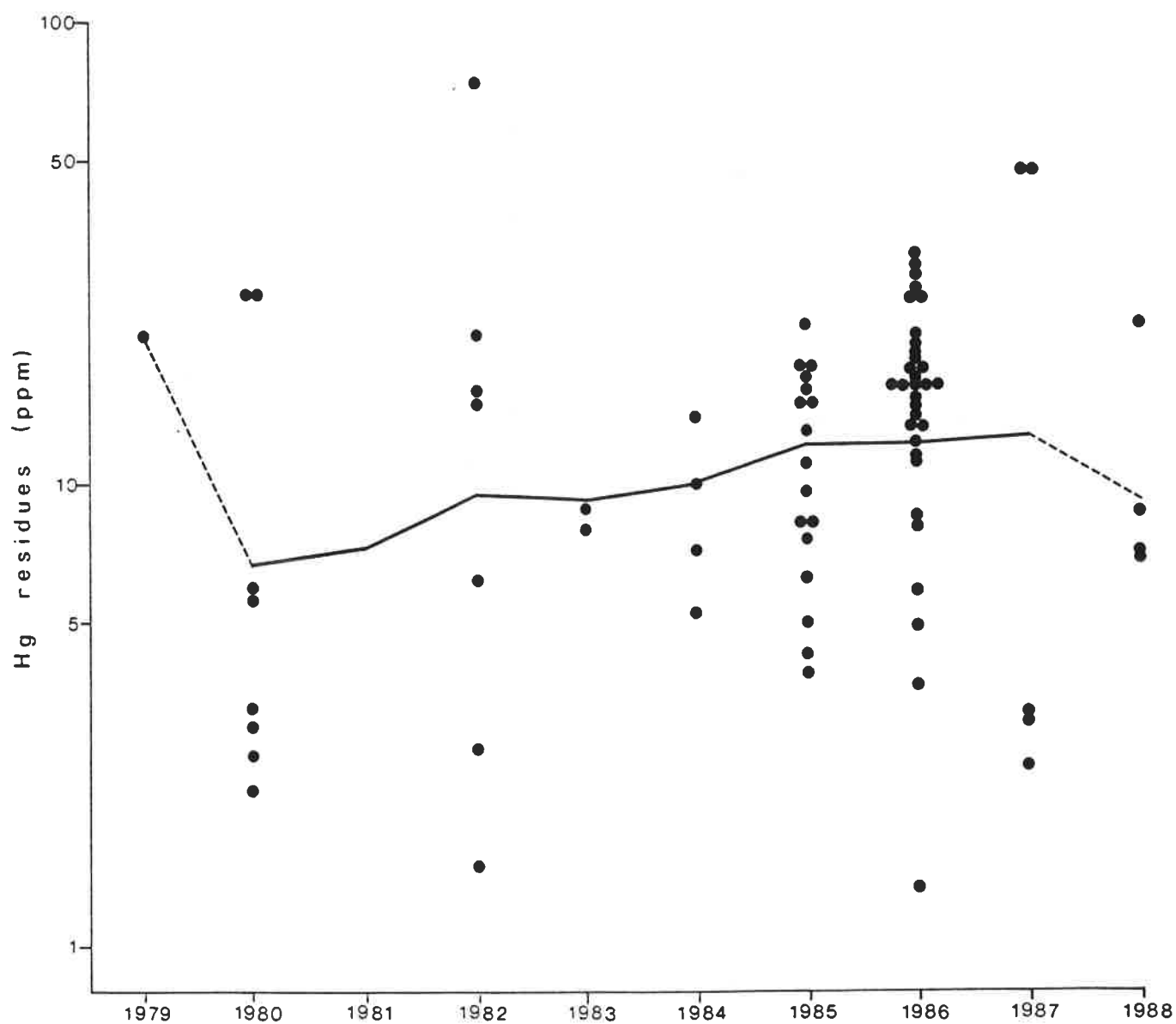


Figure 20. Levels of mercury (Hg) in livers of individual Great-crested Grebes, 1979 - 1988. Line shows 3-year moving geometric mean.



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BIRDS AND POLLUTION

Part 2 Sparrowhawk Survey

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August 1989

2 SPARROWHAWK SURVEY

2.1 Introduction

The sparrowhawk Accipiter nisus suffered a marked population decline in the late 1950s, following the widespread use of cyclodiene pesticides in agriculture. Beginning in 1964, in each of seven study areas, potential territories were checked periodically for occupation and breeding success. As the national population recovered, west to east, the different study areas were progressively dropped from the scheme, as they became re-occupied. The last study area to be re-colonised was the East Midlands area, around Monks Wood.

2.2 Recent developments

Evidence for Sparrowhawks in the East Midlands area was first obtained in 1982 and the first nests were found in 1984 after an absence of nearly 25 years. The area was last surveyed thoroughly in 1987, when recent or active nests were found on ten of 15 potential territories that were checked, and other signs of Sparrowhawks were found on the remaining five territories (Bell 1988).

Since then it has become clear that Sparrowhawks have fully re-occupied the area. They were reported from many local woods in 1988, and no less than five nests were found in Woodwalton Fen and three in Monks Wood.

2.3 Recommendations

For this reason, we recommend that the recolonisation of all study areas is regarded as complete, and that no further survey is done as part of the present scheme. Sparrowhawks in various parts of the country are still being studied by ITE and numerous amateur observers, so that if a new problem arises, it should soon come to light. A full discussion of the recovery, in the light of declining organochlorine use, may be found in Newton & Haas (1984) and Newton (1986).

2.4 References

- BELL, A.A. 1988. Sparrowhawk Survey. Birds and pollution (Part 2). NERC annual report to the NCC. Abbots Ripton: Institute of Terrestrial Ecology.
- NEWTON, I. & HAAS, M.B. 1984. The return of the Sparrowhawk. Brit. Birds 77: 47-70.
- NEWTON, I. 1986. The Sparrowhawk. Calton: Poyser.

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BIRDS AND POLLUTION

Part 3 Organochlorines and mercury in peregrine eggs

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August 1989

3 ORGANOCHLORINES AND MERCURY IN PEREGRINE EGGS

3.1 Introduction

The findings from all peregrine eggs analysed between 1961 and 1986 have recently been summarised in Newton et al (1989); those from eggs analysed in 1987 are given in Newton et al (1988), and those from eggs analysed in 1988 are given in Table 5. Other peregrine eggs from these years are awaiting analysis at the Glasgow University Veterinary School, and are outwith our programme.

3.2 Results

The eggs from 28 clutches analysed in 1988 add little to the findings of previous years, except to confirm the continuing contamination of peregrines with organochlorines and mercury (Table 5). All the values found were within the range of previous figures, and no particularly high values were found, even in the coastal eggs.

3.3 References

- NEWTON, I., BOGAN, J.A. & HAAS, M.B. 1989. Organochlorines and mercury in British Peregrine eggs. *Ibis* 131: 355-376.
- NEWTON, I., HAAS, M.B., ASHER, A., LEACH, D. & POLWARTH, G. 1988. Organochlorines and mercury in peregrine eggs. *Birds & Pollution* (Part 3). NERC report to the MCC. Abbots Ripton: Institute of Terrestrial Ecology.

Table 5. Residue levels (organochlorine ppm wet weight; mercury ppm dry weight) and shell-indices for Peregrine eggs analysed in 1988. ND = none detected.

Year	County	Shell Index	pp'DDE	HEOD	PCBs	Hg
<u>SOUTH WEST ENGLAND</u>						
1988	Cornwall (C)	1.71	1.23	0.23	6.14	0.62
	Devon (C)	2.01	1.58	0.16	3.03	0.39
	Devon (C)	1.93	1.64	0.26	3.86	1.24
<u>WALES</u>						
1988	Gwent	1.76	1.52	0.23	4.90	0.62
	Gwent	1.86	1.35	0.34	0.77	0.27
	Gwent	1.95	2.55	0.39	11.35	0.78
<u>NORTHERN ENGLAND</u>						
1987	Cumbria	1.59	1.39	0.06	2.40	0.99
	Cumbria	1.77	0.72	0.10	2.60	0.57
	Cumbria	1.56	5.88	0.21	14.28	2.42
	Cumbria	1.60	1.48	0.05	3.59	1.00
1988	Cumbria	1.59	0.90	0.30	0.63	0.19
	Cumbria	1.62	4.01	0.42	5.57	0.83
	Cumbria	1.58	3.42	0.37	16.98	ND
	Cumbria	1.87	1.68	0.33	15.90	0.61
	Cumbria	1.62	1.89	0.31	3.76	1.20
	Cumbria	-	1.16	0.26	1.38	0.18
	Cumbria	1.44	1.47	0.21	1.19	0.74
	Cumbria	2.06	0.95	0.10	0.98	0.24
	Cumbria	1.88	0.38	0.16	1.50	0.37
	Cumbria	1.76	1.34	0.42	3.14	0.44
	Northumberland	2.04	0.52	0.15	8.65	2.51
	Northumberland	2.00	1.93	0.29	13.25	1.17
<u>SCOTLAND - CENTRAL AND EASTERN HIGHLANDS</u>						
1988	Grampian	1.73	1.57	0.20	5.66	0.89
	Grampian	2.07	0.37	0.30	2.28	0.63
	Grampian	1.53	0.12	0.20	0.82	ND
	Grampian	1.03	0.32	0.18	3.16	ND
	Tayside	1.96	0.22	0.34	1.28	0.22
	Tayside	1.60	0.11	0.29	0.52	ND

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BIRDS AND POLLUTION

Part 4 Organochlorines and mercury in merlin eggs

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August 1989

4 ORGANOCHLORINES AND MERCURY IN MERLIN EGGS

4.1 Introduction

The findings from all previous analyses of merlin eggs were given in Newton & Haas (1988), those from 1987 in Newton et al (1988), while those from 1988 are summarised in Table 6.

4.2 Results

As in the peregrine, the results from these additional merlin eggs add little to the findings from previous years, except to confirm a continuing contamination of British merlins with organochlorines and mercury. One egg from Co. Durham contained exceptionally high levels of organochlorines (347 ppm DDE, 35 ppm HEOD and 147 ppm PCBs in lipid) while one egg from Orkney contained an amazingly high level of mercury (27 ppm in dry weight). This last value was the highest recorded in the egg of any British raptor since the start of the scheme. Several eggs from Shetland also contained high mercury levels, in line with earlier findings on eggs from these islands.

4.3 References

- NEWTON, I. & HAAS, M.B. 1988. Pollutants in Merlin eggs and their effects on breeding. *Brit. Birds* 81: 258-269.
- NEWTON, I., HAAS, M.B., ASHER, A., LEACH, D. & POLWARTH, G. 1988. Organochlorines and mercury in Merlin eggs. *Birds and Pollution* (Part 4). NERC report to the NCC. Abbots Ripton: Institute of Terrestrial Ecology.

Table 6. Residue levels (organochlorine ppm in lipid; mercury ppm in dry weight) and shell indices for Merlin eggs analysed in 1988.

C=clutch size; F=brood size; ND=none detected.

Year	County	C	F	Shell index	pp'-DDE	HEOD	PCBs	Hg
<u>WALES</u>								
1988	Cardigan	5	0	1.12	42.46	9.47	167.37	3.05
	Montgomery	4	0	0.98*	91.99	5.26	149.43	2.44
	Denbyshire	-	-	0.99	23.74	4.24	29.71	4.21
<u>NORTHERN ENGLAND</u>								
1988	Westmorland	4	3	-	34.21	9.77	132.71	5.66
	Yorkshire	5	4	1.09	146.83	7.25	171.30	2.93
	Northumberland	5	0	1.17	80.59	2.96	71.05	1.90
	Durham	3	0	0.97	255.31	8.44	207.81	1.85
	Durham	5	3	1.23	54.59	6.63	94.90	1.34
	Durham	3	2	1.10	89.62	5.66	111.64	2.63
	Durham	4	0	1.01	347.00	35.25	147.00	3.12
<u>GALLOWAY AND SOUTHERN UPLANDS</u>								
1988	Lothians	4	3	1.08	43.79	6.80	46.15	2.63
	Lothians	5	4	1.11	87.77	5.64	58.93	6.55
	Lothians	4	3	-	72.84	3.67	43.49	2.05
	Lothians	3	0	1.19	115.65	5.75	41.21	2.00
	Lothians	4	1	-	179.75	16.26	88.04	3.14
	Dumfries	4	0	1.25	79.84	6.45	37.50	4.18
	Dumfries	3	2	1.19	122.34	9.89	63.00	3.15
	Dumfries	4	3	1.12	229.61	5.92	194.74	3.87
<u>HIGHLANDS</u>								
1988	Aberdeenshire	4	0	1.03	152.20	7.66	219.49	4.30
	Aberdeenshire	5	4	1.17	124.11	2.19	99.73	2.98
	Aberdeenshire	5	4	0.98	105.79	3.58	116.01	3.68
	Aberdeenshire	5	3	1.17*	98.77	6.17	117.78	1.94
	Aberdeenshire	4	0	1.06	121.35	0.65	51.89	3.07
	Kincardines.	-	0	1.20	102.96	11.51	55.59	2.56
	Kincardines.	5	3	1.21	166.09	8.62	165.23	5.15
	Morayshire	5	0	1.33	37.21	4.65	100.78	5.39
	Morayshire	-	-	1.21	47.09	3.88	86.70	3.38
	Perthshire	4	0	1.13	96.13	8.84	154.42	5.56
	Nairnshire	-	-	1.18	33.33	2.65	108.99	4.52
	Angus	-	-	1.22	50.63	3.15	98.42	1.68
<u>ORKNEY</u>								
		-	-	1.18	39.15	3.10	100.56	27.80

SHETLAND

1987	-	-	1.07	53.35	2.74	191.16	6.88
	-	-	1.15	26.18	1.82	117.45	9.87
	-	-	1.22	25.40	2.22	88.25	12.80
	-	-	1.13	61.19	2.61	95.15	13.80
1988	-	-	1.04	28.67	4.67	66.33	7.58
	4	-	1.29	28.88	3.25	103.25	5.95
	-	-	1.07	34.13	5.12	82.59	7.38
	-	-	0.98	43.08	3.16	143.87	5.16
	-	-	1.23	38.59	2.01	120.81	6.90
	-	-	1.26	36.51	2.07	182.16	8.00
	-	-	1.10	47.03	3.96	163.37	7.10
	-	-	1.10	26.38	1.63	86.32	13.80
	-	-	1.19	33.46	1.97	101.57	8.33
	-	-	1.18	23.20	ND	141.33	13.30

* Shell cracked, approximate Shell index only.

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BIRDS AND POLLUTION

Part 5 Mercury in feathers of merlins and their prey

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August 1989

5 MERCURY IN FEATHERS OF MERLINS AND THEIR PREY

5.1 Introduction

Previous analyses showed that merlin eggs from Shetland and Orkney contained high levels of mercury, enough in some clutches to reduced breeding success (Newton & Haas 1988). Some eggs obtained from these islands in 1987 and 1988, and discussed in the previous section of this report, also contained high mercury levels.

The reason why Merlins on Shetland and Orkney are more contaminated than those nesting in parts of mainland Britain is not known. In the hope of shedding light on the problem, feathers from merlins and their common prey species were collected in Shetland and Orkney in 1988, and analysed for mercury content. Feathers were also obtained from Aberdeenshire and acted as a standard for comparison. In each area, merlin feathers were found at nest sites (having been shed during moult) or were taken from birds trapped at nests, while prey feathers were obtained from plucking posts. In addition, a few whole carcasses of Merlins were obtained, enabling many feathers to be examined from the same individuals.

Not all feathers have yet been analysed, but Figure 21 and Table 7 summarise the results from those that have.

5.2 Results

Among the merlins contamination was not uniform across all primary feathers. Primary numbers 3-7 (numbering from the innermost outwards) contained mercury at higher concentration than the others (Figure 21). The most contaminated primary feathers were the first to be moulted, so for this reason may receive more mercury from the body than feathers moulted later in the sequence. None of the adults examined were in their first year of life, so none would have grown all their primaries at the same time, while in the nest.

In general, feathers from Shetland seemed to contain more mercury than equivalent feathers from Orkney. Feathers from the one adult merlin from Aberdeenshire were fairly low in mercury, but not the lowest found.

For the various prey species, several flight feathers from each prey-bird were pooled for analysis, as individual feathers were extremely light in weight, which would have made their mercury content difficult to quantify. Feathers from only three species were obtained in large enough numbers from all three areas to permit useful comparison (Table 7). Meadow pipit Anthus pratensis and skylark Alauda arvensis feathers from Orkney and Shetland contained more mercury than did those from Aberdeenshire. The differences were statistically significant for pipits from Shetland, and for larks from both Orkney and Shetland. In contrast, the feathers of wheatears Oenanthe oenanthe showed no significant variation in mercury levels between the three areas.

In addition to the main prey species, some others were obtained in good numbers from only one or two of the three areas. Six snipe Capella gallinago from Aberdeenshire had more mercury in their feathers (mean (\pm SE) = 3.24 ± 0.86 ppm) than any of the passerines from that area. Seven dunlin Calidris alpina from Orkney contained 2.36 ± 0.62 ppm mercury, while ten dunlin from Shetland contained 1.94 ± 0.32 ppm. In contrast, 20 house sparrows Passer domesticus from Orkney contained only 0.22 ± 0.12 ppm mercury, while in 12 linnets Carduelis cannabina from Orkney no mercury could be detected.

5.3 Discussion

With so few analyses, it is difficult to draw firm conclusions. However, as two out of three common prey species had more mercury in Shetland and Orkney than in Aberdeenshire, there may be a difference in the general environmental levels of mercury between these areas. The fact that, on Orkney, house sparrows and linnets contained so little mercury would seem to rule out seed-dressings as a major source of contamination. Further discussion of the findings will await further analyses.

5.4 Reference

NEWTON, I. & HAAS, M.B. 1988. Pollutants in merlin eggs. Brit. Birds 81: 258-269.

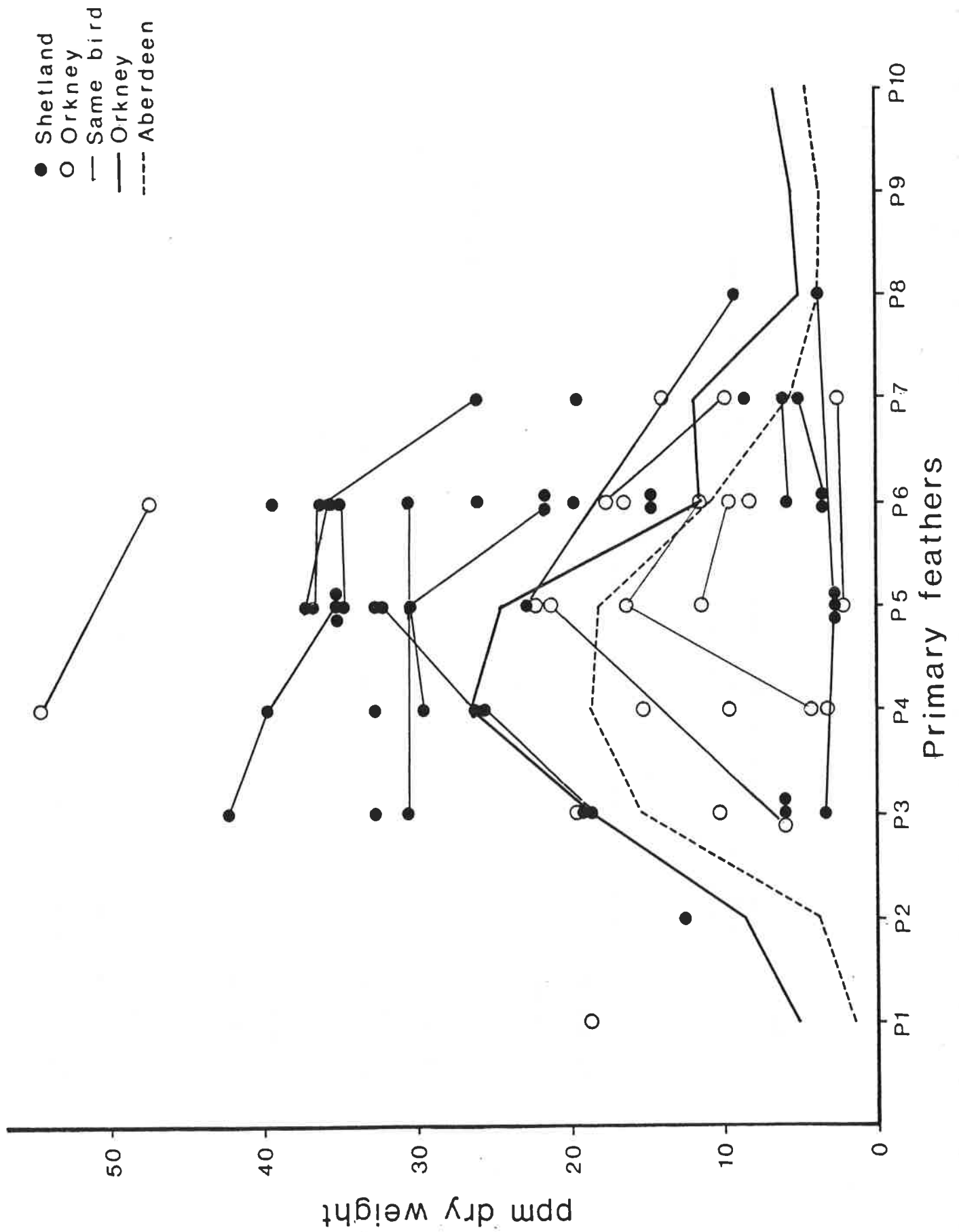
Table 7 . Comparison of mercury in the feathers of three main Merlin prey species from Aberdeenshire with those of Orkney and Shetland. t-values refer to the difference between that sample and the Aberdeenshire one.

	Aberdeenshire	Orkney	Shetland
Meadow Pipit			
Mean	1.27	1.68	2.58
Range	0.56 - 3.36	0.01 - 7.13	0.85 - 5.25
		$t_{121} = -1.11$	$t_{45} = -4.48^{***}$
Skylark			
Mean	0.74	2.12	2.20
Range	0.40 - 1.30	0.39 - 8.33	0.32 - 9.52
		$t_{66} = -3.63^{***}$	$t_{50} = -2.98^{**}$
Wheatear			
Mean	1.65	0.99	1.88
Range	0.53 - 5.78	0.01 - 3.70	0.42 - 8.48
		$t_{21} = 1.07$	$t_{44} = -0.38$

0.01 = None detected

Significance of difference * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Figure 21. Mercury levels in primary wing feathers of Merlins. Feathers from the same bird are joined by lines.



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BIRDS AND POLLUTION

Part 6 Organochlorines and mercury in gannet eggs

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August 1989

6 ORGANOCHLORINES AND MERCURY IN GANNET EGGS

6.1 Introduction

Since 1971, Gannet Sula bassana eggs have been collected from several British colonies, and analysed for pollutant residues. The chemicals of interest included DDE (from the insecticide DDT), HEOD (from the insecticides aldrin and dieldrin), PCBs (industrial polychlorinated biphenyls) and Hg (mercury, derived from natural, industrial and agricultural sources). Parslow & Jefferies (1977) described the analytical findings to 1976, and examined the relationship between DDE and eggshell thickness; while Cooke (1979) explored in detail the relationship between DDE and various shell features. In this paper we incorporate the additional data that have accrued since these previous studies in order to assess trends in residue levels over the years.

We also re-examine the relationship between DDE levels and shell thickness indices on the enlarged samples.

Eggs provide a convenient sampling unit, because they are highly consistent in composition (notably lipid content), and are produced by the same sector of the population (breeding adult females) each year. Moreover, organochlorine levels in eggs reflect the levels in the female body (Vermeer & Reynolds 1970, Enderson & Berger 1970, Henny 1977), which in turn reflect dietary intake (Lincer 1975).

6.2 Procedure

During 1971-87, eggs were collected from two colonies, at Ailsa Craig (Firth of Clyde) and the Bass Rock (Firth of Forth) every 1-2 years, and from five other colonies periodically, as opportunity allowed (Table 1). Thus eggs were obtained in 11 different years from Ailsa Craig and the Bass Rock, in five years from Scar Rocks (Solway Firth), in four years from Hermaness (Shetland) in three years from St Kilda (northwest Scotland), in two years from Grassholm (southwest Wales), and in one year from Little Skellig (southwest Ireland). On each occasion, a colony was visited during the laying or early incubation periods, and around ten eggs were taken (Gannets lay only one egg per clutch). In all 366 eggs were analysed.

Organochlorine levels were determined using gas-liquid chromatography, with an electron-capture detector, after extraction from the egg-contents with hexane and cleaned up using de-activated alumina. Throughout the programme, for every batch of 25 samples, standards of known concentration were used to check the efficiency of recovery for each chemical, thus ensuring consistency in analytical accuracy over the years. Recovery of organochlorines was generally greater than 98%. All organochlorine, except for the various pesticides, was counted as PCBs, but identification was checked against five different PCB standards, including Aroclor 1254. Results are expressed as $\mu\text{g g}^{-1}$ in wet weight. Mercury levels were determined by digestion in nitric acid, followed by atomic absorption spectrophotometry (Hatch & Ott 1968). Residues are expressed as $\mu\text{g g}^{-1}$ in dry weight. Minimal detectable levels of organochlorines were about $0.01 \mu\text{g g}^{-1}$ in wet weight, and of mercury about $0.01 \mu\text{g g}^{-1}$ in dry weight. For the calculation of mean residue levels, values below the limit of detection were taken as $0.01 \mu\text{g g}^{-1}$ for all chemicals. Gannet eggs contain about 4.6% lipid and 83% water, so that organochlorine levels given here could be converted to an approximate lipid basis by multiplying them by 21 and to a dry weight basis by dividing by five. As the eggs that were analysed were obtained fresh (or nearly so) water loss had been

slight and no correction for this was made. Any bias thereby introduced would have had no important impact on the results. Because of the non-normal distribution of data, all residues levels were \log_{10} transformed for statistical analyses. Shell indices, reflecting shell thickness, were taken as the dry shell weight (mg)/shell length x breadth (mm), after Ratcliffe (1970).

6.3 Results

General residue levels

DDE was found in Gannet eggs at up to $15.1 \mu\text{g g}^{-1}$ in wet weight, HEOD at up to $1.9 \mu\text{g g}^{-1}$, PCBs up to $28.0 \mu\text{g g}^{-1}$, and Hg up to $18.2 \mu\text{g g}^{-1}$ in dry weight. Over the years, one egg had no DDE at detectable levels, 15 had no HEOD, and three had no PCB. All eggs had detectable amounts of Hg.

In most years, the levels of DDE and HEOD varied significantly between eggs from different colonies (Table 1). However, the pattern was not consistent between years, as no one colony yielded the highest or lowest levels throughout. The same was true for PCBs, except that the levels in Hermaness eggs were the lowest in all three years in which this colony was sampled.

Colony differences in mercury levels were more consistent (Table 1). The highest levels were found in eggs from Scar Rocks, especially in 1972 and 1973. In all nine years in which the Ailsa Craig and Bass Rock colonies were sampled together, mercury levels were higher on Ailsa Craig. Unusually low Hg levels were found in eggs from St Kilda in 1979, but not in 1985 and 1987. The colony on Little Skellig was sampled only once (in 1973), when levels of all pollutants were not dissimilar from those in eggs from other colonies that year.

Time trends in residue levels

At Ailsa Craig and Bass Rock, which were studied over the longest period, levels of organochlorines were highest in the early 1970s, declined to around 1983, and then increased again (though not to the initial levels) (Figure 1). Other colonies were sampled over only part of this period, but the changes observed in organochlorine levels were largely consistent with this pattern. Thus declines in organochlorines recorded at Scar Rocks (1971-73 to 1980) and Hermaness (1980 to 1983) coincided with the decline phase at Ailsa Craig and Bass Rock, while increases at St Kilda (1979 to 1987) and Grassholm (1980 and 1984) coincided with the increase phase at Ailsa Craig and Bass Rock. There may therefore have been a general decline in the organochlorine contamination of British Gannet eggs to around 1983, followed by a rise.

Mercury levels followed different trends, which were less consistent between colonies. Levels declined significantly at Ailsa Craig (1971-87) and Scar Rocks (1972-83), and rose significantly at Bass Rock (1973-87), Hermaness (1979-83), St Kilda (1979-87) and Grassholm (1980 and 1984) (Table 1).

Shell indices and pollutant levels

Significant relationships were found between shell indices and levels of all four pollutants (Table 3). Overall DDE explained 19% of the variance in shell index, PCBs explained 18%, HEOD 13% and Hg 16%. However, the levels of these various chemicals in eggs were inter-correlated, and when this was allowed for in a multiple regression analysis, none of the other chemicals explained significantly more of the variance in shell-index than did DDE alone (Figure 2).

The relationship between shell-index and DDE was consistent with the earlier findings of Parslow & Jefferies (1977) and Cooke (1979); this was not surprising as their studies were based on sub-sets of the eggs available to us. The actual equations differ, however, partly because these previous authors expressed DDE on a lipid (rather than wet weight) basis. The overall findings are also consistent with those in many other species, implicating DDE as the main causal agent of reduced shell index (Cooke 1973, Newton 1979, Newton & Haas 1988).

6.4 Discussion

Geographical and time trends

Because only two of the seven colonies studied were sampled over the whole period, it is difficult to draw firm conclusions on time trends in residue levels, and the degree of consistency between colonies. We could not exclude the possibility that time trends in residues varied between colonies. However, the organochlorine data from all colonies were largely consistent with the patterns at Ailsa Craig and Bass Rock, which showed a general decline between the early 1970s and early 1980s, followed by a rise. Trends in mercury levels were much less consistent between colonies, with declines recorded at two colonies (including Ailsa Craig), and increases at four (including Bass Rock).

The trends observed could reflect changes in the general contamination of the marine environment over the years concerned, or simply changes in the foraging areas or diets of the birds. Although Gannet colonies are at fixed locations, the birds can forage up to 150 km away from the colonies in the breeding season (Nelson 1966, Tasker et al 1985), and range even more widely in winter (Nelson 1978, Tasker et al 1985). The diet consists of various species of pelagic shoaling fish.

The fact that the three organochlorine chemicals changed in parallel with one another suggested that changes in diet or food intake may have been at least partly responsible for the changes in Gannet egg residues. There is some other slight evidence for this view. On Ailsa Craig Gannets fed their chicks mainly on Mackerel Scomber scombrus in 1975-76 and 1979-82, but turned mainly to sand-eels Ammodytes spp. in 1983 (Wanless 1984). Moreover, the fat content of Mackerel is much higher than that of most other fish, so Mackerel would be expected to contain higher levels of the fat-soluble organochlorines. Other fish, such as Pollock Pollachius pollachius, which formed about 30% of the diet by weight in 1975-76, were almost entirely lacking by 1981-83. These changes in diet composition coincided with the time when the organochlorine contents of Gannet eggs from Ailsa Craig reached their lowest levels.

At Hermaness, sandeels formed 90% of the chick diet in 1981, but had declined to 14% by 1987, with Mackerel and Herring Clupea harpengus increasing in importance (T. Martin, pers. comm.). This coincided with a rise in organochlorine levels at several colonies, but no eggs were collected at Hermaness after 1983. It is worth adding, however, that all this information on diet is based on food fed to chicks, which is not necessarily the same as that eaten by adults prior to egg-laying.

Long-term declines in organochlorine pollutants in Gannet eggs have become apparent, not only in some British colonies, but also in a Norwegian colony (Fimreite et al 1980), and on Bonaventure Island, off the Gaspé Peninsula, in eastern Canada (Chapdelaine et al 1987). In this latter colony the decline in DDE was substantial, and associated with known reductions in input to the sea.

It is much more difficult to interpret the variable trends in mercury levels between colonies. In contrast to the organochlorines, the mercury in Gannet eggs may be partly natural in origin. However, the exceptional levels in eggs from Scar Rocks in 1972-73 were almost certainly derived from some local industrial source, which has since reduced its output.

Pollutants and breeding success

In the Gannet the association between shell-index and DDE is mainly due to effects on the chalky vaterite layer of the shell, with much less effect on the underlying palissade and mammillary layers (Cooke 1979). The fact that DDE explained only 19% of the variance in shell index in our sample - a much smaller proportion than in some other birds (Newton 1979) - was perhaps because other factors, such as general wear, also affect the chalk layer. It can easily be scraped off by hand, so is presumably also abraded by the feet of the incubating bird. By analogy with other species, the reduction in shell index recorded in British Gannets was not sufficient to result in widespread egg-breakage and breeding failure, and to our knowledge, this has not been recorded.

The DDE levels in our eggs, mostly less than $5 \mu\text{g g}^{-1}$ in wet weight, were much lower than those associated with breeding failure in Gannets on Bonaventure Island (Chapdelaine et al 1987). In 1969-74, eggs from this colony contained around $10-25 \mu\text{g g}^{-1}$ DDE (converted by us from the lipid values given) and showed a hatching success of only 36-40%. By 1978 DDE levels had dropped to $5-15 \mu\text{g g}^{-1}$ and hatching success had risen to 58%, and by 1976-84 levels had dropped to less than $5 \mu\text{g g}^{-1}$ (similar to those in British colonies) and hatching success varied annually between 78% and 89%. The only British colony examined during our study period was Ailsa Craig, where Wanless (1979) found a hatching success in 1975-76 of 82%, within the range of the most recent (normal) Canadian values. Taking these findings as a whole, therefore, it seems unlikely that the productivity of British Gannets was reduced by DDE during 1971-87, the period covered by our samples. In view of the high success, productivity was probably not reduced by the other pollutants either. The fact that almost all British colonies, except St Kilda, have increased in that time (Wanless 1987) also suggests that productivity has not been seriously affected.

The levels of organochlorine pesticide residues in gannet eggs may well have been higher in the 1960s than during our study. Even then, however, Nelson (1964) recorded a hatching success of 82% on Bass Rock in 1961-63. Possibly, therefore, the breeding success of Gannets around Britain has not been seriously reduced by organochlorine pollutants at any time.

6.5 Acknowledgements

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6.6 Summary

Levels of DDE, HEOD, PCBs and mercury were measured in gannet eggs from several colonies around Britain during 1971-87. In two colonies (Ailsa

Craig and Bass Rock) sampled throughout this period, organochlorine levels declined between the early 1970s and 1983, and then increased again. The trends at other colonies, sampled over shorter periods, were generally consistent with those at Ailsa Craig and Bass Rock. Trends in mercury levels were more variable, and increases were observed at four colonies and declines at two. Some shell-thinning occurred in association with DDE contamination. However, levels of this chemical were too low to affect breeding success in the years concerned. The same was probably true of the other chemicals.

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Table 8. Geometric mean pollutant levels ($\mu\text{g g}^{-1}$) in Gannet eggs from different years and different colonies. Organochlorine levels are on a wet weight basis and mercury levels on a dry weight basis.

(Egg Nos.)		1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1987	Net increase (%) or decrease (D) ²
HEOD																		
Ailsa Craig	(98)	0.86	0.49	0.27	0.73	0.64	-	0.44	-	0.20	-	0.09	-	0.09	-	0.29	0.18	D***
Bass Rock	(141)	-	-	0.57	0.54	0.23	0.22	0.32	0.68	0.04	-	0.11	-	0.06	-	0.21	0.12	D***
Scar Rocks	(42)	0.04	0.59	0.36	-	-	-	-	-	-	0.01	-	-	0.19	-	-	-	D**
Hermaness	(34)	-	-	-	-	-	-	-	-	-	0.05	0.33	0.04	0.01	-	-	-	D**
St Kilda	(24)	-	-	-	-	-	-	-	-	0.03	-	-	-	-	-	0.14	0.86	I***
Grassholm	(20)	-	-	-	-	-	-	-	-	-	0.01	-	-	-	0.14	-	-	I*
Little Skellig	(7)	-	-	0.22	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Significance of variation between colonies ¹		***	ns	**	ns	***	-	ns	-	***	-	ns	-	**	-	**	***	
DDE																		
Ailsa Craig	(98)	2.06	2.13	0.49	1.55	2.50	-	0.87	-	1.43	-	0.57	-	0.19	-	0.39	0.26	D***
Bass Rock	(141)	-	-	1.53	1.01	1.10	0.52	0.51	0.81	0.43	-	0.32	-	0.17	-	0.34	0.32	D***
Scar Rocks	(42)	0.16	2.48	1.23	-	-	-	-	-	-	0.80	-	-	0.31	-	-	-	D***
Hermaness	(34)	-	-	-	-	-	-	-	-	-	0.42	0.50	0.28	0.14	-	-	-	D**
St Kilda	(24)	-	-	-	-	-	-	-	-	0.25	-	-	-	-	-	0.28	0.36	I**
Grassholm	(20)	-	-	-	-	-	-	-	-	-	0.59	-	-	-	0.28	-	-	D**
Little Skellig	(7)	-	-	1.25	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Significance of variation between colonies ¹		*	ns	**	ns	***	-	*	-	***	*	ns	-	**	-	ns	ns	
PCBs																		
Ailsa Craig	(98)	11.94	4.83	2.47	11.37	1.55	-	7.55	-	7.19	-	1.04	-	0.37	-	3.20	1.19	D***
Bass Rock	(141)	-	-	7.25	7.42	1.71	3.86	4.49	7.75	2.85	-	0.90	-	0.26	-	2.63	4.35	D**
Scar Rocks	(42)	10.70	12.90	5.16	-	-	-	-	-	-	2.33	-	-	1.01	-	-	-	D***
Hermaness	(34)	-	-	-	-	-	-	-	-	-	1.28	0.42	0.29	0.14	-	-	-	D***
St Kilda	(24)	-	-	-	-	-	-	-	-	0.67	-	-	-	-	-	2.39	4.90	I**
Grassholm	(20)	-	-	-	-	-	-	-	-	-	1.81	-	-	-	6.04	-	-	I***
Little Skellig	(7)	-	-	4.53	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Significance of variation between colonies ¹		ns	**	ns	ns	ns	-	*	-	***	ns	**	-	**	-	ns	***	
Hg																		
Ailsa Craig	(98)	5.07	3.16	3.62	4.56	3.86	-	3.43	-	3.22	-	3.62	-	2.85	-	2.04	2.97	D***
Bass Rock	(141)	-	-	1.56	2.52	1.24	1.52	0.80	0.89	2.01	-	2.74	-	1.99	-	1.75	2.05	I**
Scar Rocks	(42)	-	10.47	9.57	-	-	-	-	-	-	4.01	-	-	6.12	-	-	-	D***
Hermaness	(34)	-	-	-	-	-	-	-	-	-	1.16	2.49	2.11	2.48	-	-	-	I***
St Kilda	(24)	-	-	-	-	-	-	-	-	0.44	-	-	-	-	-	2.31	2.09	I***
Grassholm	(20)	-	-	-	-	-	-	-	-	-	1.45	-	-	-	2.92	-	-	I***
Little Skellig	(7)	-	-	3.10	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Significance of variation between colonies ¹		-	***	***	***	***	-	***	-	***	***	ns	-	***	-	ns	**	

Notes: 1 In years when only two colonies were sampled, the difference in \log_{10} residues between them were examined using a t-test, and where more than two colonies were sampled, differences were examined by analysis of variance.
* P[0.05]; ** P[0.01]; *** P[0.001].

2 To test for net changes in residues over the sampling period at each colony a linear regression model was used, with individual log residue levels as the dependent variable and years as the independent variable. At some colonies a linear model did not give the best fit to the data, but the objective was simply to check for long-term net changes. At Grassholm, sampled in only two years, the differences in mean residues were examined by t-tests.
* P[0.05]; ** P[0.01]; *** P[0.001].

Table 9. Linear regression relationships between shell index and pollutant levels in individual eggs (N = 326).

Relationship between shell index and:-	Constant (a)	Regression coefficient (b)	Correlation coefficient (r)	Probability (P)
DDE	2.90	-0.12	-0.19	***
PCBs	2.95	-0.08	-0.18	***
HEOD	2.87	-0.07	-0.13	*
Hg	2.97	-0.13	-0.16	**

Correlations between levels of different pollutants: DDE and PCBs: $r=0.67^{***}$; DDE and HEOD: $r=0.58^{***}$; DDE and Hg: $r=0.27^{***}$; PCBs and HEOD: $r=0.55^{***}$; PCBs and Hg: $r=0.17^{**}$; HEOD and Hg: $r=0.20^{***}$.

* $P<0.05$; ** $P<0.01$; *** $P<0.001$.

Figure 22. Pollutant residues ($\mu\text{g g}^{-1}$ in fresh weight for organochlorines and in dry weight for mercury) in Gannet eggs from Ailsa Craig (upper) and Bass Rock (lower) from 1971 to 1987.

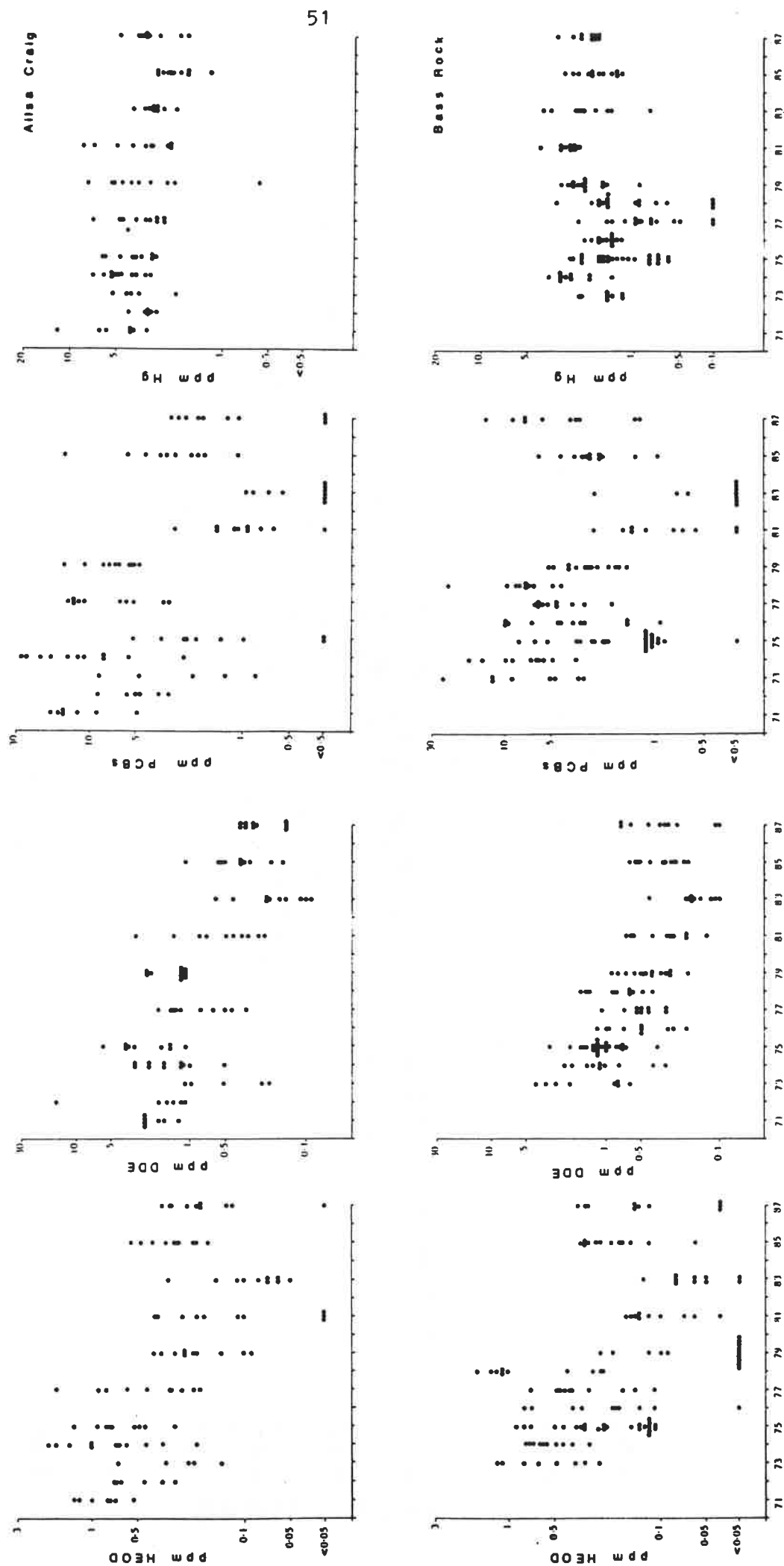
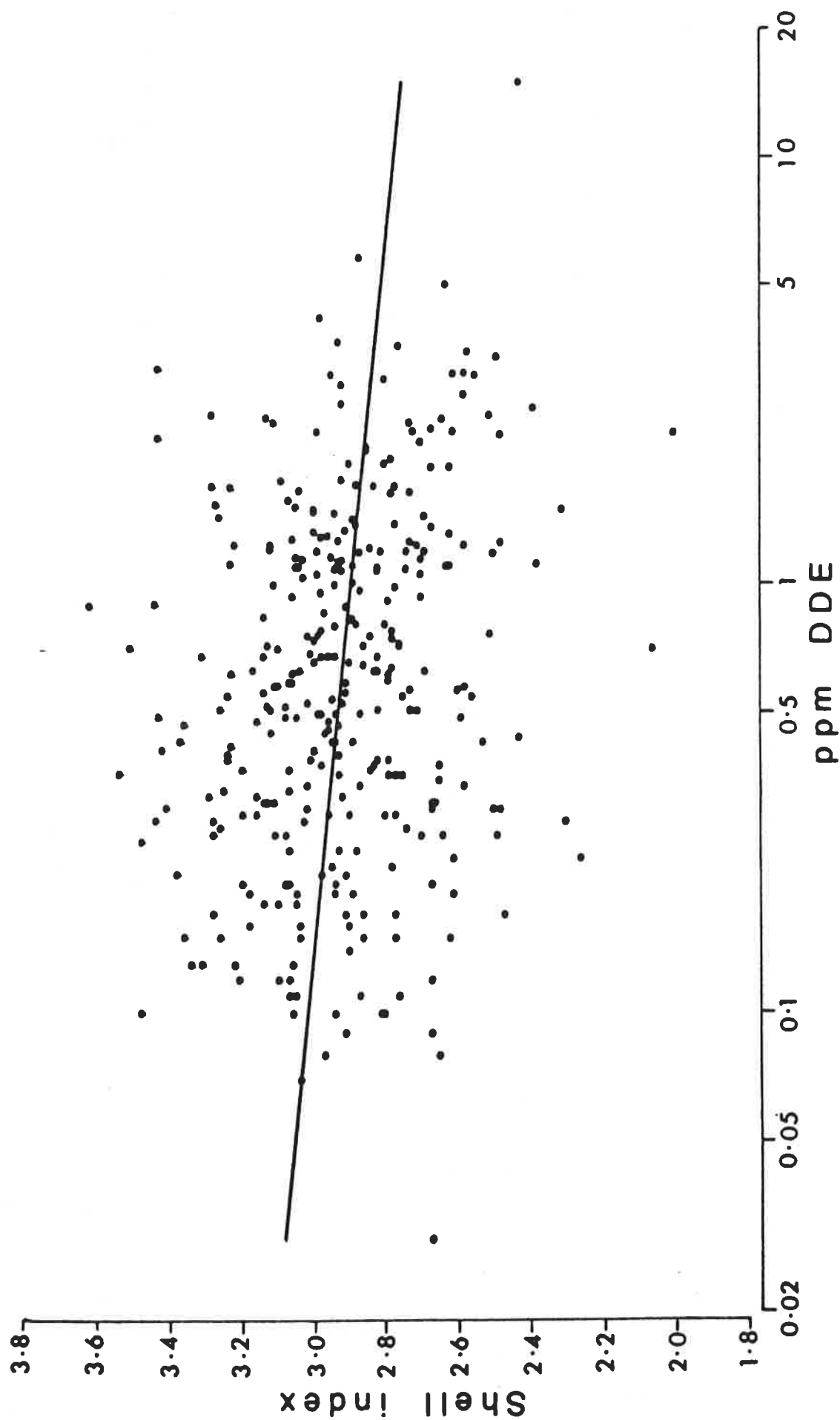


Figure 23. Relationship between shell indices and DDE levels (in fresh weight) in Gannet eggs.



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BIRDS AND POLLUTION

Part 7 Rodenticides in Barn Owls

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7 RODENTICIDES IN BRITISH BARN OWLS

7.1 Introduction

This section has two aims. The first is to report the incidence of residues of two rodenticides, difenacoum and brodifacoum, in the bodies of Barn Owls *Tyto alba* found dead in various parts of Britain. The second is to report the effects of feeding captive Barn Owls on mice killed by these two rodenticides. The chemicals concerned are two of the commonest, "second-generation" rodenticides, developed as replacements to warfarin, to which rodents in some regions have become resistant. Like warfarin, both chemicals are coumarin derivatives and they act as anti-coagulants. They are much more potent than warfarin, however, and have been reported to cause secondary poisoning in various rodent-predators, including owls (Mendenhall & Pank 1980, Hegdal & Colville 1988). The work described here was intended to help in assessing the impact of these new rodenticides on the Barn Owl population of Britain.

Difenacoum has been available in Britain since 1974, and has been used both indoors and outdoors. Brodifacoum has been available since 1984, its use was initially restricted to within buildings by professional operators; but more recently has been extended outdoors, where rats have become difenacoum-resistant. Both chemicals have been commonly used around farmsteads and other sites, where rodents are a problem. As farm buildings provide some of the most frequently used nest-sites for Barn Owls in Britain, these owls would be expected to be at special risk, as they eat affected rats and mice. Indeed, Shawyer (1987) listed four Barn Owls found dead or dying, associated with local difenacoum use, and four others following brodifacoum use; all showed symptoms of haemorrhaging. He suggested that these chemicals may have contributed to a decline in Barn Owl numbers in parts of Britain. Similarly, Duckett (1984) attributed the collapse of a Barn Owl population in a Malayan rubber plantation to use of brodifacoum and coumachlor against rats. Meanwhile the high toxicity of brodifacoum to wild or captive owls of various species has been confirmed by Mendenhall & Pank (1980), Merson & Byers (1984) and Hegdal & Colvin (1988).

7.2 Methods

Survey. Advertisements were placed in various bird journals asking for the bodies of Barn Owls found dead. All carcasses were requested, irrespective of mode of death. On receipt, the bodies were stored deep frozen until they could be examined, usually some months later. After thawing, a post-mortem was conducted (by visual inspection only), and where possible, the findings were used, together with information from the sender, to determine the cause of death. Careful search was made for signs of haemorrhaging, which often follow an exposure to anticoagulants. A part of the liver was removed from each bird and analysed for residues of difenacoum and brodifacoum.

The method of analysis was that of Hunter (1985) modified in minor respects. Liver samples were extracted with chloroform-acetone, and the extracts were cleared of fat using Bond-Elut columns. The concentrated samples from the columns were then analysed by Varian High Pressure Liquid Chromatography, using a MCH-5M micropack column and spectrofluorometer, against a standard for each compound. When difenacoum or brodifacoum was detected, a recovery test was done from a spiked sample to validate the identification and to correct the estimate of mass present. Recoveries from most batches were in the range 75-95%. The lower limit of detection for both compounds was around 0.01 µg, which was equivalent to 0.005-0.01

$\mu\text{g g}^{-1}$, depending on the weight of the sample. For calculation of average concentrations in known contaminated mice, nil detected values were taken as $0.0075 \mu\text{g g}^{-1}$. Concentrations are given as wet weight values, but can be converted to dry weight values by multiplying by 3.64 for liver of owls by 4.30 for liver of poisoned mice and by 3.29 for the whole carcasses of poisoned mice (see later). The values from wild owls were corrected to allow for water loss, assuming a wet to dry conversion factor in fresh liver of 3.64, the value found for captive owls.

Toxicity trials. Owls were dosed with rodenticide-poisoned mice. The owls were kept individually in cages measuring 60 x 50 x 40 cm high, and were thus much less active than wild owls, and were fed on white laboratory mice, rather than on wild rodents. As owls do not need to drink, no water was provided.

Some days before a feeding trial, mice were fed for one day on a proprietary dosed-food mixture containing either 0.005% difenacoum or 0.002% brodifacoum. After dying, some days later, most mice were fed to owls. The mean weight (\pm SE) of the dead mice was $30.0 \pm 1.1\text{g}$. Other mice, from the same batches, were analysed to determine the total content of rodenticide in their bodies. The liver was analysed separately from the rest of the carcass which was first homogenised in a 'blender'.

All the owls were captive-bred, very tame, and in their first year of life. They had not previously been exposed to rodenticides, and at the start of the trials in September they were in good condition, weighing 280-350 g. They were allocated at random into two groups of six, one fed on difenacoum-treated mice and the other on brodifacoum-treated mice. Owls in each group were fed initially for one day on dosed mice (3 per owl). Those which survived this treatment were later fed for three successive days on dosed mice, and any which survived this treatment were later fed for six days on dosed mice. The livers of owls which died were analysed for rodenticide residues in the same way as the livers from wild owls.

Before the trials, and at various stages during it, small blood samples were taken from the brachial vein in some of the owls. In each case the coagulation time was measured, while tipping the blood back and forth in a 75 mm glass capillary tube of 1 mm internal diameter. The tube contained a 1 cm metal rod (a 'flea'), and coagulation time was taken when the rod would no longer slide around within the blood. All such sampling was done at the same time of day, at 1600 h. The results of these tests were used to indicate the recovery time after exposure to the rodenticide, and when another series of trials could safely be started. However, in view of the risks in blood-sampling birds treated with anticoagulant, such sampling was kept to a minimum. No problems were encountered, as the vein closed itself soon after the needle was withdrawn.

7.3 Results

Survey. In the period 1983-89, a total of 146 Barn Owls was received from various parts of Britain for analysis. Results of analyses up to 1987 were given in Newton et al (1988), and those from 1988-1989 are listed in Table 10. On visual inspection of the opened carcass, together with information from the sender, most dead owls were diagnosed as victims of accidents or starvation (Table 1). None was submitted or diagnosed as an obvious rodenticide victim, although slight haemorrhaging, not obviously associated with trauma, was noticed in two birds, in both around the heart.

On later chemical analysis difenacoum was detected in the livers of 7 individuals, brodifacoum in 4, and both chemicals together in another 4, making a total of 15 (10%) contaminated birds. There was no obvious sex bias, as 10% of males had residues and 11% of females, and no seasonal bias, as contaminated birds were found scattered through the year. Levels of difenacoum were in the range 0.010-0.088 $\mu\text{g g}^{-1}$, while levels of brodifacoum were in the range 0.020-0.058 $\mu\text{g g}^{-1}$, with one higher value at 0.593 $\mu\text{g g}^{-1}$. The haemorrhaging occurred in one bird with 0.019 difenacoum and in one with 0.02 brodifacoum.

The proportion of contaminated birds may have increased through the study period, as the use of these chemicals increased, but the trend was not significant statistically (Table 2). Over the years, contaminated owls were received from various parts of Britain (Figure 22), and were not restricted to warfarin-resistance areas depicted by Shawyer (1984).

Toxicity trials: mice. All dosed mice died, mostly 3-8 days after dosing, with no significant difference between difenacoum and brodifacoum-treated animals (Figure 23). Chemical analysis of a subsample showed that, for both chemicals, the residue was much more concentrated in the liver than in the rest of the carcass (Table 13). So great was this difference that, even allowing for the fact that the liver forms only about one-fifteenth of the weight of a mouse, the total mass of rodenticide was still greater in the liver than in the rest of the carcass (Table 13). On the basis of our results a 30 g mouse on the day of death contained a total of 1.86 μg of difenacoum, on average, or a total of 2.36 μg of brodifacoum.

Toxicity trials: owls. All the owls fed on difenacoum-dosed mice survived the 1-day, 3-day and 6-day treatments (Table 4), and none showed external bleeding. After the 1-day treatment, all six owls were blood-sampled 5-9 days later, and coagulation times were 'normal' (Table 15). After the 3-day treatment, the blood of one bird sampled three days later would not coagulate, even after 24 hours, but all birds seemed to have become normal in this respect 21-23 days after treatment. With the method used, no significance could be given to the fact that, in five of the six birds, coagulation times were somewhat shorter after recovery from difenacoum than before exposure to it (Table 15).

Of the six owls fed on brodifacoum-dosed mice, four died 6, 10, 11 and 17 days after the 1-day treatment (Table 14), with 0.63-1.24 $\mu\text{g g}^{-1}$ brodifacoum in their livers. The weight changes in three of these birds between treatment and death were small (+1%, 0%, -6%), but the fourth bird lost 23%, declining from 332 g to 257 g. The two survivors also survived the later 3-day and 6-day treatments. Again, however, they were affected, as all showed prolonged bleeding from the mouth or feet for up to 30 days post treatment. Blood taken from two birds 9 days after treatment would not coagulate, and nor would blood taken from one of these birds (a survivor) after 38 days. However, blood from the other survivor seemed normal 16 days after treatment (Table 15). It seems, therefore, that brodifacoum is more toxic than difenacoum to Barn Owls, and in survivors, has much more prolonged effects. Probably it persists for longer in the body than difenacoum, as found in rodents (Rammell et al 1984).

From knowledge of the number of mice consumed by the owls, and the average weight of rodenticide in their bodies at the time, the total weight of rodenticide consumed by the owls in the 1, 3 and 6-day treatments could be calculated (Table 6). Individuals consumed an estimated 5.6 μg , 11.2 μg , and 9.3-18.6 μg difenacoum in 1, 3 and 6 days respectively, and an estimated 7.08 μg , 14.16 μg and 28.32 μg brodifacoum. The 7.08 μg 1-day dose of brodifacoum was enough to kill four of the six owls, so could be

regarded as approximately the LD₆₇. This was equivalent to a dose of 0.023-0.028 mg kg⁻¹ body weight for the birds concerned. If the nil detected values in mice (excluding liver) were in fact nil, rather than the 0.0075 µg g⁻¹ used to calculate averages, this would have reduced the estimated lethal dose to owls very slightly to 0.021-0.025 mg kg⁻¹.

The analysis of regurgitated pellets (of fur and other undigested parts of food items) from each group of owls revealed that some of the rodenticide that was consumed was excreted unchanged in pellets. In other words it had not passed through the gut. Five pellets collected from one group of owls during the breeding trial contained a mean of 0.60 µg difenacoum (range 0.06-1.19 µg), while six pellets from the other group contained a mean of 0.97 µg brodifacoum (range 0.08-3.86 µg). Assuming a regurgitation rate of two pellets per day, this would have reduced the total rodenticide consumption to 4.4 µg, 7.6 µg and up to 11.4 µg in the 1, 3 and 6 day difenacoum trial, and to 5.1 µg, 8.4 µg and 16.8 µg in the 1, 3 and 6 day brodifacoum trial. These estimates can only be rough, however, because of the great variation in the rodenticide content of particular pellets.

7.4 Discussion

Survey About 10% of the 146 Barn Owls examined in 1983-89 had residues of difenacoum or brodifacoum in their bodies. It is most unlikely that this sample reflects the true exposure of owls to these rodenticides in the areas concerned. Almost all the carcasses were found in the open, and all were victims of road traffic and other accidents, or of apparent starvation. Death from rodenticide poisoning is delayed, and is preceded by lethargy, so poisoned owls would be most likely to die in their roosts, in roof-cavities or hollow trees, where they would be unlikely to be found by the casual observer. When they are found in such situations, moreover, they are seldom fresh enough for analysis. In addition, sampling at one point in time (ie death) takes no account of multiple exposures, and some of the owls in which no residues were detected may have contained residues at an earlier date, which they subsequently metabolised or excreted. Nonetheless, the findings confirm that the exposure of Barn Owls in Britain to difenacoum and brodifacoum is now frequent and widespread, and is not confined to areas where rats are resistant to warfarin. Even if brodifacoum use were restricted to the inside of buildings, as recommended, this would not protect Barn Owls, which will sometimes hunt within buildings, and at the same time could take contaminated rodents which wander outside. It has been found that mammals which receive sublethal doses of brodifacoum can retain residues in their bodies for some months (Rammell et al 1984).

Toxicity trials. The feeding trials on captive owls were done in the hope that they would help in interpreting the significance of residue levels found in wild owls. Brodifacoum levels in the four captive birds that died were at least an order of magnitude higher than those in most of the wild birds with brodifacoum (one wild bird had a level of 0.59 ppm, close to the lowest level of 0.63 ppm in a captive bird). There could have been two reasons for this difference. First, the sample of wild owls may not have contained any individuals that had died of rodenticide poisoning, only some which had been exposed to sublethal levels. This explanation would be consistent with the low likelihood of poisoned owls being found (see above), and with the fact that other (non-rodenticide) causes of death were diagnosed on visual inspection. An alternative explanation would be that much lower levels of rodenticides are needed to kill wild owls than captive ones. Our birds, kept in cages, remained still and inactive almost all the time, moving only when disturbed or to eat the

mice provided. Yet they still showed prolonged external bleeding. Wild birds, which must remain highly active, are presumably much more likely to suffer severe haemorrhaging from the exertion involved. Another likely limitation of our feeding trial is that it ran overwinter, when the owls were not moulting (apart from one which started during the 6-day brodifacoum trial). If they had been growing feathers throughout, the opportunities for haemorrhaging may have been much greater (though this did not occur in warfarin-fed moulting Tawny Owls *Strix aluco*, Townsend et al 1984). Finally, there remains the possibility that sub-lethal levels of rodenticide may predispose death from other causes, or reduce the chance of recovery from accidents.

Despite this limitation, our results clearly imply that difenacoum is much less toxic to Barn Owls than brodifacoum. None of the six birds fed difenacoum died, and despite some bleeding, coagulation times were back to 'normal' within 9-14 days. In contrast, four of the six birds fed brodifacoum died within 6-17 days after a 1-day dose, and blood from survivors would still not coagulate at 13 days post-treatment. Birds that died ate three mice, with a combined weight of about 90 g and an estimated total brodifacoum content of 7.08 μg . The fact that two other birds of similar weight survived on the same 1-day intake, together with the later 3-day and 6-day treatments, could have been due to individual differences in sensitivity. It was also possible that, owing to limited carrying capacity of the liver or to continuing metabolism and excretion of residues, birds on 3 and 6 day treatments did not accumulate correspondingly higher concentrations in their tissues than did birds on the 1-day treatment.

These findings on the differential toxicity of the two compounds are consistent with those of Mendenhall & Pank (1980), who found that five out of six Barn Owls fed brodifacoum died, compared with none out of six fed difenacoum. The high toxicity of brodifacoum was confirmed in a field trial with voles and Screech Owls *Otus asio* in Virginia (Hegdal & Colvin 1988). Five owls found dead 5-37 days post treatment contained 0.4-0.8 $\mu\text{g g}^{-1}$ brodifacoum in their livers, while four caught and killed 34-43 days later contained 0.3-0.6 $\mu\text{g g}^{-1}$. Two others in each group had no detectable residues. The levels in the dead Screech Owls were comparable to those in our Barn Owls of 0.63-1.24 $\mu\text{g g}^{-1}$, suggesting a similar pre-death residue accumulation in the two species. Published values for the acute LD_{50} for brodifacoum in different domesticated birds and mammals span two orders of magnitude: from 0.27 mg kg^{-1} body weight in the rat to about 25 mg kg^{-1} in the cat (Worthing & Walker 1987). These figures imply a huge species variation in sensitivity. The lethal dose of 0.023-0.028 mg kg^{-1} brodifacoum which killed our owls, was a further order of magnitude less than any published figures, making the Barn Owl the most sensitive of the species yet investigated. Our owls obtained this dose from three mice (total biomass 90 g), which was nicely within the range of 1-5 'rodents' estimated by Shawyer (1987) to be necessary to kill an owl.

Published figures for the acute LD_{50} difenacoum range between 0.8 and 100 mg kg^{-1} in different species, but for any one species the dose was higher than for brodifacoum (Worthing & Walker 1987). Hence, the greater toxicity of brodifacoum over difenacoum, suggested by our results, was consistent with previous laboratory findings on other species.

In conclusion, there is little doubt that second generation rodenticides could present a threat to Barn Owls and other predators of rodents. The several-day period between dosing and death in rodents, their lethargic behaviour for some hours before death, and the several month persistence of sublethal brodifacoum residues within the mammalian body (Rammell et al

1984), all facilitate the contamination of owls. Moreover, there is little doubt that rodents which are commensal with man are regularly eaten by Barn Owls. House mouse Mus musculus remains were present in 47% of 188 pellet samples examined by Glue (1974) from various parts of Britain, while Brown Rat Rattus norvegicus remains were present in 48% of samples. In a recent Irish study, House mice remains were found in 10 out of 15 pellet samples, while Brown Rat remains appeared in 12 (Smal 1987). The incidence of both species in the diet may well increase during hard weather or when voles are scarce. Field trials have given mixed results but, where no owl mortality was recorded, this could be attributed to the radio-marked owls not hunting near baiting sites or taking mainly non-target prey (Kaukeinen 1982, Hegdal & Blaskiewicz 1984). Other field studies have shown mortality in owls (Merson, Byers & Kaukeinen 1984, Hegdal & Colvin 1988) and even substantial population decline (Duckett 1984).

7.5 Acknowledgements

We are grateful to Dr P. Anderson, Dr S. Dobson and Mr C. Shawyer for helpful advice at the outset. The toxicity trials were undertaken under Home Office Licence.

7.6 Summary

Residues of the rodenticides, difenacoum or brodifacoum, were detected in the livers of 10% of 146 wild Barn Owls, found dead in various parts of Britain during 1983-89. Difenacoum was in the range 0.010-0.088 $\mu\text{g g}^{-1}$ fresh weight, and brodifacoum was in the range 0.020-0.058 $\mu\text{g g}^{-1}$, apart from one exceptional level of 0.593 $\mu\text{g g}^{-1}$.

Mice fed for one day on food containing difenacoum and brodifacoum died after 3-8 days. Within these mice residues were present at greater concentration and mass in the liver than in the rest of the carcass. The mean mass of brodifacoum in a whole mouse was estimated at 1.86 μg for difenacoum and 2.36 μg for brodifacoum.

Such poisoned mice were fed to Barn Owls for successive periods of 1, 3 and 6 days. All six owls fed on difenacoum-dosed mice survived all three treatments, and the coagulation times of their blood returned to near normal in less than 5-22 days. Four of the six owls fed on brodifacoum-dosed mice died after the 1-day treatment, but the survivors also survived the 3-day and 6-day treatments. Those that died had each eaten 3 mice, with a combined weight of about 90 g and a total brodifacoum content of about 7.08 μg which was equivalent to a dose of 0.023-0.028 mg kg^{-1} body weight. After death these owls had 0.63-1.24 $\mu\text{g g}^{-1}$ of brodifacoum in their livers. Blood from the survivors would not coagulate at 9 days post-treatment, but did so at 16 days in one bird and between 38 and 78 days in the other.

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Table 10. Levels of rodenticides (ppm in wet wt) in the livers of wild barn owls (*Tyto alba*) analysed from April 1, 1988 to 30 April, 1989. J = juvenile in first year; A = adult older than one year; M = male, F = female.

No.	Date Found	County	Age	Sex	Brodifacoum	Difenacoum
8432	Mar 84	Lincs	-	F	ND	ND
8183	Aug 84	Devon	J	F	ND	ND
8028	Sep 84	Northants	J	M	ND	ND
8029	Sep 84	Northants	J	F	ND	ND
8032	Oct 84	Gwynedd	J	M	ND	ND
8037	Oct 84	Gwent	J	F	ND	ND
8043	Oct 84	Cumbria	J	F	ND	ND
8094	Oct 84	Borders	A	F	ND	ND
8120	Nov 84	S'clyde	A	F	ND	ND
8430	Nov 84	Hants	J	F	0.593	ND
8227	Feb 85	Norfolk	J	M	ND	ND
8252	Mar 85	Wilts	A	F	ND	ND
8294	May 85	Kent	A	F	ND	0.054
8302	May 85	W. Sussex	A	F	0.036	0.025
8306	Jun 85	Essex	J	F	ND	ND
8307	Jun 85	Essex	A	M	ND	ND
8398	Aug 85	Wilts	J	M	ND	ND
8433	Aug 85	Surrey	A	M	ND	ND
8403	Oct 85	Wilts	A	M	ND	ND
8445	Nov 85	Leics	-	-	ND	ND
8487	Jan 86	Powys	J	F	ND	0.011
8897	Aug 86	Wilts	A	M	0.045	0.065
8808*	Sep 86	Humbers	A	F	ND	0.019
8852	Oct 86	Suffolk	-	M	ND	ND
8854	Nov 86	Dyfed	-	M	ND	ND
8856	Nov 86	IOM	-	M	0.058	0.088
8883	Jan 87	Cambs	J	M	ND	ND
8892	Jan 87	E. Sussex	J	M	ND	ND
8900	Jan 87	S. Yorks	A	M	ND	ND
8923	Jan 87	Norfolk	A	M	ND	ND
8942	Feb 87	W. Sussex	J	F	ND	ND
8950	Mar 87	Norfolk	A	M	ND	ND
8961	Apr 87	Cambs	A	M	ND	ND
8974	May 87	Beds	A	M	ND	0.022
8983	Jun 87	Cambs	A	M	ND	ND
9022	Sep 87	Cornwall	J	M	ND	ND
9031	Sep 87	Dyfed	J	F	ND	ND
9394	Nov 87	Beds	-	F	ND	ND
9316	Dec 87	Devon	J	M	ND	ND
9274	Apr 88	Cambs	A	F	ND	ND
9317	May 88	Devon	A	F	ND	ND
9276	Jun 88	Cambs	A	M	ND	ND
9367	Sep 88	Surrey	J	M	ND	ND
9419	Sep 88	Bucks	J	F	ND	ND
9365	Oct 88	Essex	J	M	ND	ND
9370	Oct 88	Norfolk	J	M	ND	ND
9371	Oct 88	Borders	J	F	ND	ND
9383	Nov 88	Hants	J	F	ND	ND
9389	Nov 88	Grampian	-	M	ND	ND
9393	Dec 88	Norfolk	A	F	ND	0.029

9394	Dec 88	Beds	-	F	ND	ND
9395	Dec 88	Bucks	A	F	ND	ND
9396	Dec 88	Bucks	A	F	ND	ND
9575	Dec 88	Cumbria	J	F	ND	ND
9421	Jan 89	D & G	J	M	ND	ND
9423	Jan 89	Northants	J	M	ND	ND
9425	Jan 89	Wilts	A	F	ND	ND
9430	Jan 89	D & G	A	F	ND	ND
9433	Jan 89	Northants	J	M	ND	ND
9437	Feb 89	Oxon	J	F	ND	ND
9443	Feb 89	Wilts	J	F	ND	ND
9444	Feb 89	Wilts	J	M	ND	0.01
9453	Feb 89	Wilts	A	F	ND	ND
9550	Feb 89	Yorks	A	M	ND	ND
9559	Mar 89	Kent	A	M	ND	ND
9563	Mar 89	S'clyde	J	F	ND	ND
9569*	Mar 89	Gwynedd	A	M	0.02	ND
9574	Mar 89	Somerset	A	F	ND	ND
9578	Mar 89	Essex	J	M	ND	ND

*haemorrhage around heart

Table 11. Causes of death diagnosed on post-mortem examination of 146 wild Barn Owls.

	Number	Number with rodenticide residues
Trauma (road accident)	85	7
Trauma (other accident)*	9	2
Starvation	48	6
Shot	3	0

* Includes mainly collision victims, two drowned and one electrocuted.

Table 12. Numbers of wild Barn Owls tested for rodenticides each year from 1983 showing numbers containing brodifacoum and difenacoum.

Year	Number tested	Difenacoum only	Brodifacoum only	Both rodenticides	% affected
1983	3	0	0	0	0
1984	15	0	1	0	7
1985	31	1	1	1	10
1986	36	2	0	2	11
1987	22	2	0	0	9
1988	24	1	1	1	13
1989*	15	1	1	0	13
Overall	146	7	4	4	10

* To 31 March only

Table 13. Difenacoum and brodifacoum levels in livers and the remaining carcase of dosed mice analysed on the day of death ($n = 11$ in each case).

Mouse	<u>Difenacoum</u>			<u>Brodifacoum</u>		
	μg	Liver only $\mu\text{g g}^{-1}$	Carcase minus liver $\mu\text{g g}^{-1}$	Liver only $\mu\text{g g}^{-1}$	Carcase minus liver $\mu\text{g g}^{-1}$	
1	1.58	2.23	2.21	1.43	1.11	ND
2	0.72	0.41	2.60	1.50	1.44	0.04
3	1.56	1.01	1.85	2.23	1.67	0.14
4	0.53	0.47	0.34	3.00	2.13*	TR
5	0.78	0.59	0.37	1.02	0.58*	ND
6	0.36	0.33	1.22	0.83	0.65	ND
7	1.36	1.04	0.17	1.77	1.09	ND
8	1.66	0.98	0.22	1.17	0.84*	ND
9	0.78	0.58	0.21	0.69	0.41	ND
10	1.03	0.66	0.60	0.59	0.37*	0.09
11	0.07	0.10+	0.21	0.79	0.67	ND
Mean \pm SE	0.95 ± 0.16	0.76 ± 0.17	$0.91 + 0.27$	1.37 ± 0.22	1.00 ± 0.16	0.99 ± 0.44 0.03 ± 0.01

ND = None detected in the sample, taken as $0.01 \mu\text{g}$ and $0.0075 \mu\text{g g}^{-1}$ in the calculation of means.

The value of 0.02 in the sample was corrected to produce a value for the whole liver or carcase.

TR = Trace detected in the sample, taken as $0.05 \mu\text{g}$ and $0.0075 \mu\text{g g}^{-1}$ for the sample and corrected to produce a value for the whole liver or carcase.

+ 8 days after death

* Also contained small amounts of difenacoum

Table 14. Results of feeding rodenticide-poisoned mice to Barn Owls.

	Period of dosing (days)	Mice eaten	Number of owls dosed	Number surviving
<u>Difenacoum</u>	1	3	6	6
	3	6	6	6
	6	5-10	6	6
<u>Brodifacoum</u>	1	3	6	2
	3	6	2	2
	6	12	2	2

Notes: The brodifacoum levels in the livers of the four owls which died were calculated at 0.63, 0.77, 1.02 and 1.25 $\mu\text{g g}^{-1}$.

The gaps between the start of successive dosing periods were 11-14 and 33-35 days for difenacoum, and 77-79 and 75 days for brodifacoum.

Table 15. Coagulation times (minutes, seconds) for Barn Owl blood samples taken on different dates after end of dosing (N.C. = no coagulation within 24 hours).

Difenacoum

Owl no.:	1	2	3	4	5	6
Pre-treatment	2,00	2,30	4,45	4,15	2,45	1,45
After 1-day dose						
5d	-	-	-	-	-	1,00
7d	-	-	-	-	1,30	-
8d	-	-	1,30	3,45	-	-
9d	2,30	1,45	-	-	-	-
After 3-day dose	N.C.	-	-	-	-	-
3d	-	-	-	-	-	-
9d	-	-	-	-	-	5,00
10d	-	6,15	-	-	-	-
21d	-	-	-	2,30	6,00	2,00
22d	-	1,30	1,45	-	-	-
23d	1,30	-	-	-	-	-
Owl no.:	7	8	9	10	11	12
Pre-treatment	1,15	1,45	1,15*	3,30*	4,00	1,15*
After 1-day dose						
9d	N.C.	N.C.*	-	-	-	-
16d	-	-	-	-	6,00	-
24d	-	-	-	-	8,30	-
36d	-	-	-	-	5,30	-
38d	N.C.	-	-	-	-	-
78d	4,30	-	-	-	-	-
After 3-day dose						
60d	5,30	-	-	-	6,15	-

* Died before further blood samples could be taken.

Table 16. Calculation of weight of difenacoum and brodifacoum consumed by owls in the feeding trials.

Days of Dosing	Number of mice eaten	<u>Difenacoum</u>		Number of mice eaten	<u>Brodifacoum</u>	
		Mean weight of difenacoum per mouse (μg)	Total weight of difenacoum eaten per owl (μg)		Mean weight of brodifacoum per mouse (μg)	Total weight of brodifacoum eaten per owl (μg)
1	3	1.86	5.58	3	2.36	7.08
3	6	1.86	11.16	6	2.36	14.16
6	5-10	1.86	9.3-18.6	12	2.36	28.32

Figure 24. Map showing locations of Barn Owls found dead and received for analysis.

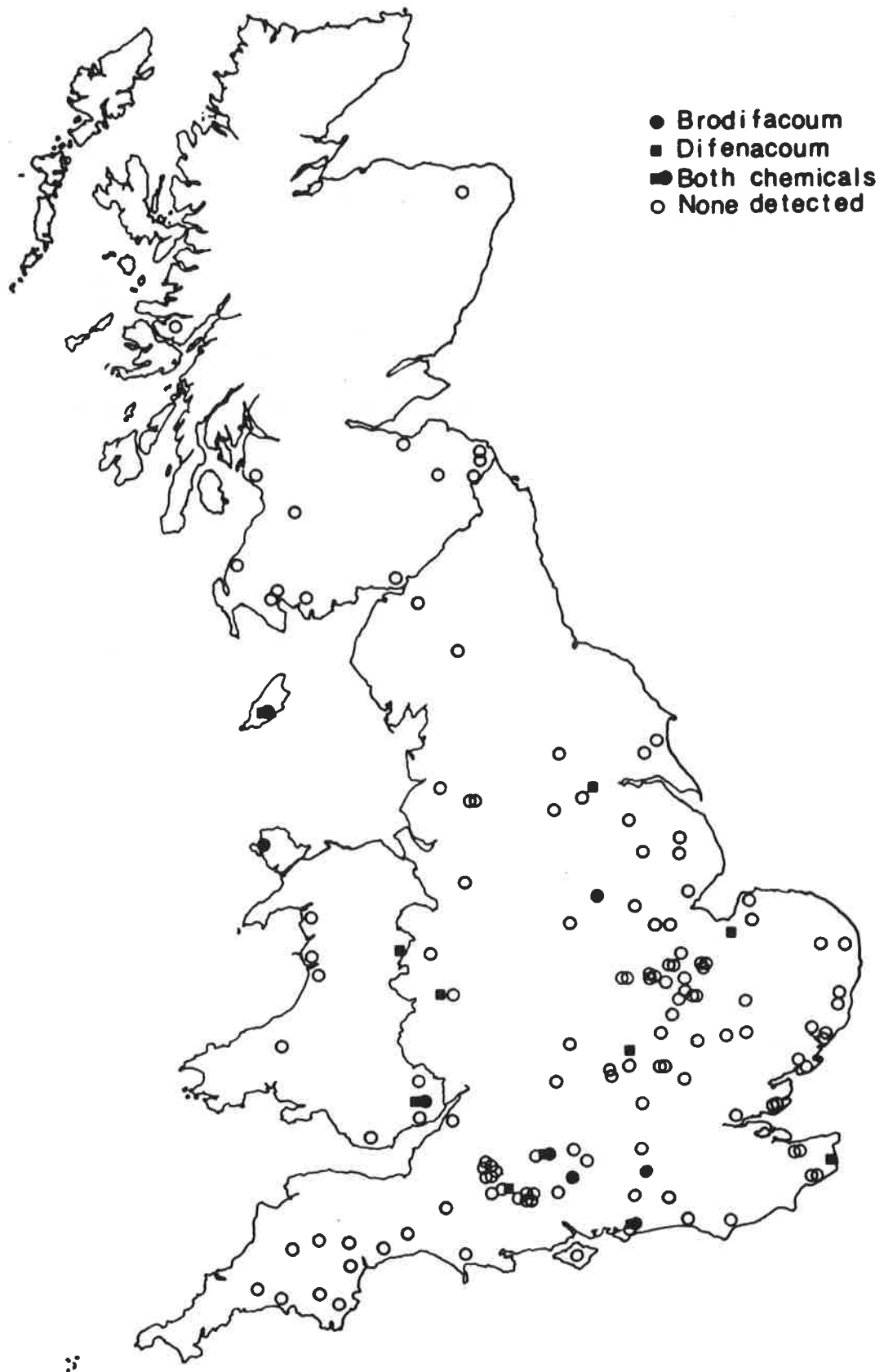
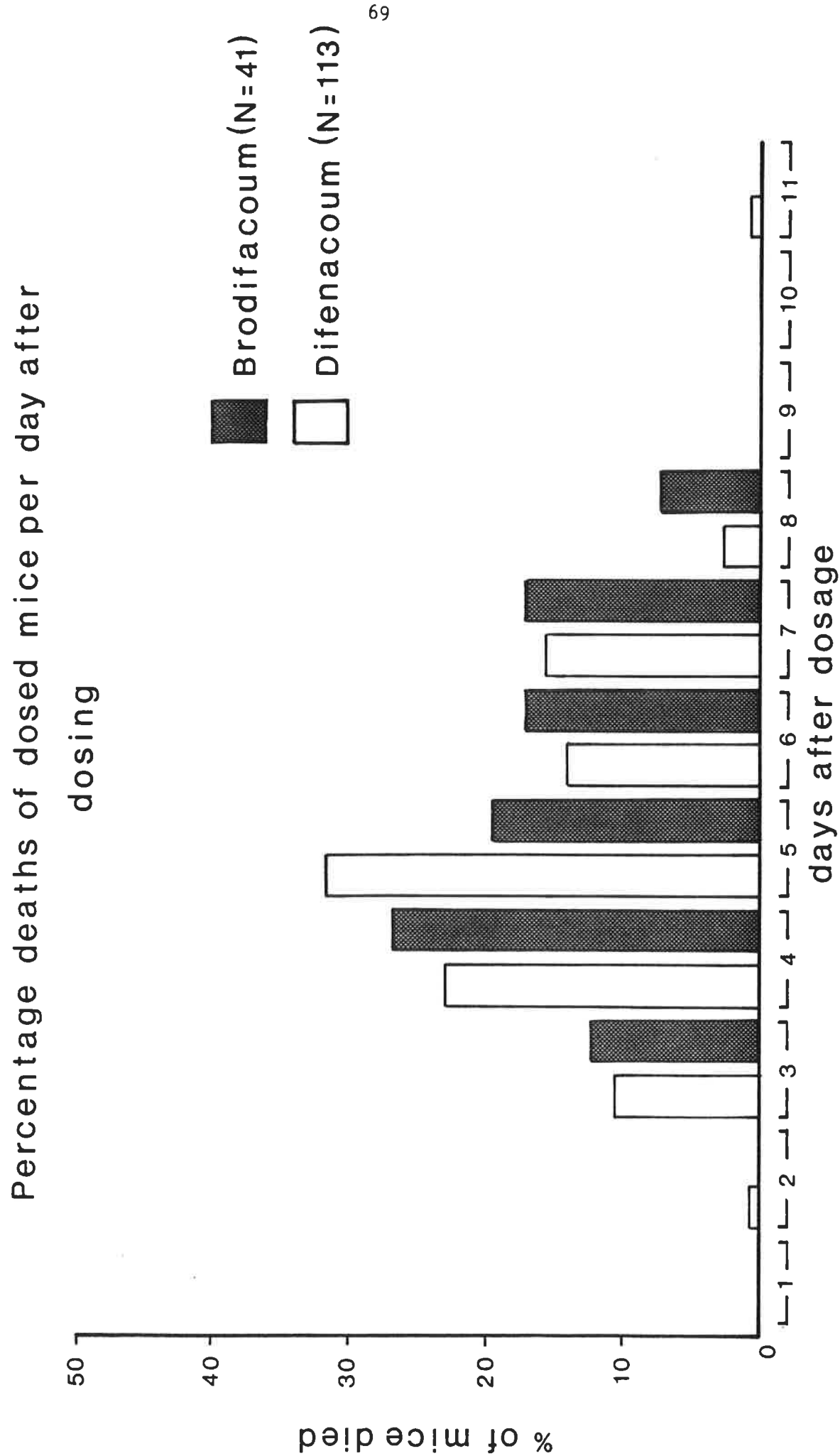


Figure 25. Time to death in mice dosed with difenacoum and brodifacoum



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