



Mortality Causes in British Barn Owls (*Tyto alba*), Based on 1,101 Carcasses Examined During 1963-1996

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Abstract.—During 1963-1996, 1,101 Barn Owl (*Tyto alba*) carcasses were received for autopsy and chemical analysis. Much larger numbers were received per month outside the breeding season than within it. A peak in the monthly mortality of first year birds occurred in autumn (November) and a peak in the mortality of adults in late winter (March).

The main causes of recorded deaths were collisions (mostly with road traffic) and starvation. No great seasonal variation occurred in the main causes of recorded deaths. Among accident victims, the mean weight of females (305 g) was about 5 percent greater than that of males (291 g). Most starved birds of both sexes weighed less than 240 g.

Organochlorine pesticide victims formed 20 percent of all dead Barn Owls obtained during 1963-1970, and a decreasing proportion thereafter. None was recorded after 1976 when the use of aldrin/dieldrin was greatly curtailed. During the 1980s and 1990s, increasing proportions of birds contained residues of second generation rodenticides, but relatively few at sufficient level to have caused their death.

Although the numbers of Barn Owls (*Tyto alba*) breeding in Britain and some other parts of western Europe have declined during the present century, there is no consensus view of the underlying causes. Changes in agriculture, notably the reduction in area of rough grassland and its associated *Microtus* voles, have probably greatly reduced the food-supply of the species. At the same time, in some regions, the loss of old trees and farm buildings that provided nest sites might have reduced Barn Owl numbers below the level that the contemporary food-supply would permit, as might the increased mortality imposed by road traffic and pesticides (Bunn *et al.* 1982, de Bruijn 1994, Sawyer 1987, Taylor 1994). In this paper, we present information on the causes of death of 1,101 Barn Owls found dead in Britain during 1963-1996, and sent to Monks Wood Research Station for study. We focus on organochlorine pesticides, notably aldrin and dieldrin, as a major cause of Barn Owl deaths during the

1960s and 1970s, and on new rodenticides as increasing contaminants during the 1980s and 1990s. The rodenticides concerned include difenacoum, bromadiolone, brodifacoum, and flocoumafen, all of which are marketed under several different trade names.

Findings on carcasses analyzed to 1989 were summarized by Newton *et al.* (1991). Since that date the number of Barn Owl carcasses received has increased from 627 to 1,101, and some causes of death have changed in importance. Other studies of mortality in British Barn Owls have been reported by Sawyer (1987), based on 629 specimens found during 1982-1986, and by Glue (1971), based on 320 ring recoveries, covering the period 1909-1970.

PROCEDURE

Carcasses were obtained from most parts of Britain, in response to regular advertisements placed in ornithological magazines and journals. All carcasses were requested, regardless of the cause of death. On receipt, each carcass was weighed, marked and then stored at -20°C until it could be examined, up to several

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months later. Age classes (juvenile or adult) were diagnosed from 1988 onwards, mainly on plumage. Juveniles (in their first year) had unabraded primaries of uniform bloom, more pointed than those of adults, and outermost primary feathers (number 11, small and hidden) that were pure white, not grey. Some also had fault bars running in the same position across all tail feathers. On dissection, juvenile females before the breeding season had a thin straight oviduct, rather than the thicker convoluted oviduct of a bird that had laid. Birds of all ages were sexed by their gonads, but most males had fewer dark spots on the underside than females. Not all specimens could be aged or sexed, however, because some had been previously skinned or were badly damaged. Full information was therefore not obtained from every bird, which is one reason why the totals given in the different tables and figures of this paper vary.

For autopsy, the unfrozen carcass was opened up and examined for any obvious parasites, lesions or other abnormalities. The findings were used, along with information from the sender, to diagnose the cause of death. Typically, collision victims had extensive bruising and broken bones, and many were found at roadsides indicating that they were traffic victims. Starved birds were low in weight, with wasted breast muscles, no body fat, and empty blackened or greenish intestines. Diseased birds showed obvious lesions, particularly in liver, kidneys or lungs, or contained parasites; and many were also thin. Shot birds contained lead pellets or pellet wounds. Diagnosis of other mortality causes, such as drowning and electrocution, was dependent primarily on information from the sender, together with the lack of any conflicting evidence from autopsy. Identification of pesticide or rodenticide victims was dependent mainly on chemical analysis, together with the lack of any other obvious mortality cause. Some organochlorine victims that were found still alive were reported to die in spasms. For certain birds (8.4 percent of the total), in the absence of any evidence, the cause of death was classed as 'unknown'.

Many of the birds examined showed signs of hemorrhaging, which differed according to cause of death. Accident victims typically bled heavily around the site of impact, while some organochlorine victims showed hemorrhaging of certain internal organs, including brain, lungs, heart, and foregut (Newton *et al.* 1982).

Other work revealed that rodenticide victims typically showed faint subcutaneous bleeding along the keel and on the skull, and external bleeding around the leg joints and beak (Newton *et al.* 1990). However, some organochlorine and rodenticide victims showed no obvious bleeding. Hemorrhaging was therefore not used as the sole diagnosis of any mortality cause, only along with other evidence, including chemical analysis.

After autopsy, a piece of liver was removed and analyzed for organochlorine residues of DDE (from the insecticide DDT), HEOD (from the insecticides aldrin and dieldrin), HE (from the insecticide heptachlor) and PCBs (polychlorinated biphenyls from various industrial products) (for methods of analysis, see Newton *et al.* 1990). Organochlorine analyses almost ceased after 1977, partly on grounds of cost, when residues fell to small levels after the last major restriction in 1976 in aldrin-dieldrin use. Only a random sample of 50 livers was analyzed for organochlorines after 1977. Although residues were detected in all these livers, the levels were low, invariably less than one tenth of the levels normally associated with death.

After considering the available data, Cooke *et al.* (1981) concluded for various birds of prey that a concentration in liver of 10 ppm or more HEOD (in wet weight) or 100 ppm or more DDE could be taken as indicative of organochlorine poisoning. Little information is available for heptachlor, but De Witt *et al.* (1960) found 6-20 ppm HE in tissues of various birds poisoned by this chemical. Organochlorine victims often showed other symptoms, however, such as internal hemorrhaging (Newton *et al.* 1982) and usually died with muscle tremors or convulsions.

Specific information on the HEOD levels in livers of owls that had died of dieldrin poisoning was obtained at the London Zoo, where in a 30-month period 55 owls of 21 species died (Jones *et al.* 1978). Their deaths were traced to high dieldrin levels in the mice that they were fed, the mice having been kept on sawdust bedding derived from dieldrin-treated timber. Of 22 obvious dieldrin casualties that were analyzed, HEOD levels in liver ranged between 13 and 46 ppm, with a geometric mean of 29 ppm.

Since 1983, liver samples were analyzed for residues of 'second generation' anticoagulant



rodenticides, namely bromadiolone, difenacoum, brodifacoum, and flocoumafen, which have been implicated in Barn Owl mortality (Newton *et al.* 1990, Shawyer 1987). Little information is available on liver residue levels associated with death in Barn Owls, but some figures are given later in this paper, to be added to the figures of 0.5-1.3 ppm given for brodifacoum by Newton *et al.* (1990), 0.3-1.7 ppm given by Wyllie (1996) for bromodialone, and 0.9 ppm for flocoumafen given by Newton *et al.* (1994).

RESULTS

Annual Cycle

Although Barn Owls in Britain can be found breeding in all months from February to November, depending on vole abundance, most breeding activity occurs in April-August (Bunn *et al.* 1982, Hardy *et al.* 1982, Shawyer 1987, Taylor 1989).

Many more carcasses were received outside the breeding season than within it (fig. 1). Monthly numbers rose from August to November and remained at high level until March, declining thereafter to a low in May-July. Birds were aged (as juvenile to 31 July the next year and as adult thereafter) on a systematic basis only from 1988. From then on, in nine complete years juveniles formed 76 percent of 541 birds received (table 1). As an estimate of first-year mortality this is 14 percent higher than the 62 percent calculated by Glue (1971) from 320 ring recoveries from a longer run of years.

In our sample, juveniles predominated in the August-November period but declined in proportion thereafter, so that the two age groups had partly different periods of mortality. The number of juveniles received each month reached a peak in the autumn (November) whereas the number of adults reached a peak in late winter (March). Of 968 owls in which the sex was recorded, 495 (51 percent) were males and 473 (49 percent) were females, a ratio not significantly different from unity. There was no evidence for a change in the sex ratio of casualties through the year (table 1), except that more males than females were received in the breeding season. This could be attributed to the sex difference in breeding roles (the male does the hunting while the female tends the eggs and young).

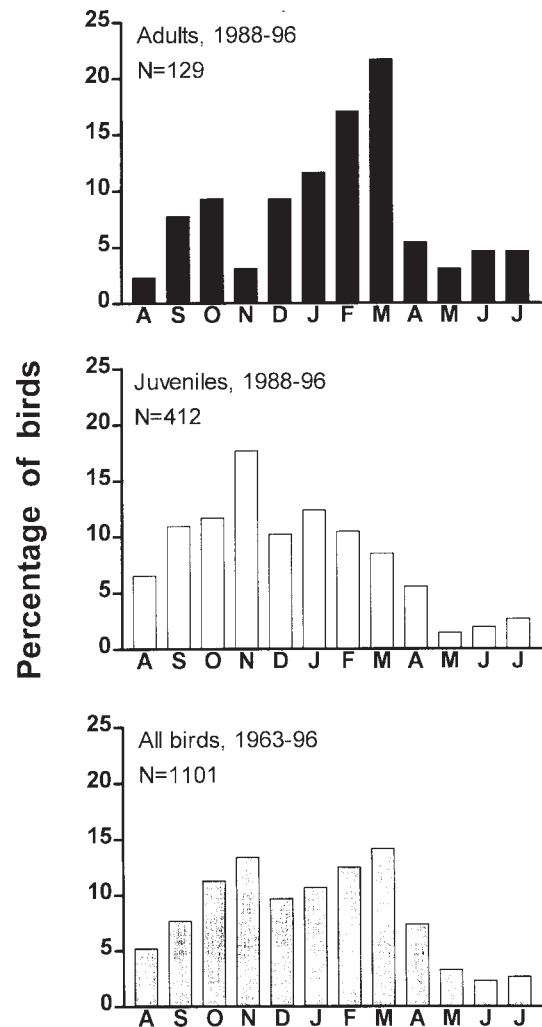


Figure 1.—Percentage of Barn Owl (*Tyto alba*) carcasses found in Britain in different months. The lower diagram includes all birds received over the 34 year study period, and the others refer separately to first-year birds and adults respectively, which were distinguished from 1988.

Because some important prey species (notably *Microtus agrestis*) of the Barn Owl fluctuate in roughly 3-4 year cycles of abundance, one might have expected some cyclic variation in the number of owl carcasses received at Monks Wood. This was not apparent on a national scale, however, and too few carcasses were obtained from particular regions to check for more local cycles in mortality.

Table 1.—Sex and age ratios among Barn Owls (*Tyto alba*) in Britain found dead in different months.

	1963-1996		1988-1996	
	Male	Female	Juvenile	Adult
January	55	48	51	15
February	59	64	43	22
March	70	70	35	28
April	32	32	23	7
May	20	10	6	4
June	13	10	8	6
July	14	9	11	6
August	34	19	27	3
September	37	38	45	10
October	47	62	48	12
November	55	70	73	4
December	<u>59</u>	<u>41</u>	<u>42</u>	<u>12</u>
Total	495	473	412	129
Percent	51%	49%	76%	24%

Mortality Causes

Over the year as a whole, 54 percent of all recorded deaths were attributable to some form of collision or other accident (table 2). Within this category the most prevalent were road traffic victims, which formed at least 45 percent of all deaths. Minor causes included other forms of trauma, drowning in water-troughs, and electrocution.

'Natural' causes accounted for 31 percent of all recorded deaths. The most important was starvation (26 percent of all deaths), followed by disease/parasitism (3 percent), and then predation (2 percent). Most of the diseased birds were also thin, so it was often uncertain whether food-shortage or infection was the primary cause of death. Several birds classed as diseased had extensive lesions on the kidneys, liver or heart, others had infected lungs, while in one the digestive tract was heavily infested with nematode worms. The only predators identified with certainty were domestic cat and dog.

The remaining birds had been shot (1 percent) or were diagnosed as victims of organochlorine pesticide (mostly dieldrin) poisoning (5 percent), or rodenticide poisoning (1 percent). All the birds tested for organochlorines had residues in their livers but mostly at levels considered to be sublethal. Of 51 birds diagnosed as aldrin/dieldrin victims, 10 had HEOD levels of 6-10 ppm in liver, 29 had levels of 10-20

Table 2.—Recorded causes of deaths in Barn Owls (*Tyto alba*) found dead in Britain during 1963-1996.

	Number	Percent
Natural causes	328	30.7
Starvation	275	25.8
Disease	35	3.3
Predation	18	1.7
Accidents	573	53.7
Road casualties	477	44.7
Other trauma	80	7.5
Drowned	12	1.1
Electrocuted	4	0.4
Other human-related causes	76	7.1
Poisoned	65	6.1
Shot	11	1.0
Unknown causes	90	8.4

ppm, 8 had levels of 21-30 ppm, 3 had levels of 31-40 ppm, and 1 had 44 ppm (Newton *et al.* 1991). All these birds (including those with less than 10 ppm HEOD in liver) had other symptoms of organochlorine poisoning (most often convulsions prior to death), and no other obvious cause of mortality. In addition, two road traffic victims had 11 and 14 ppm HEOD, in their livers, so in the absence of the collision, they might have died anyway from poisoning.

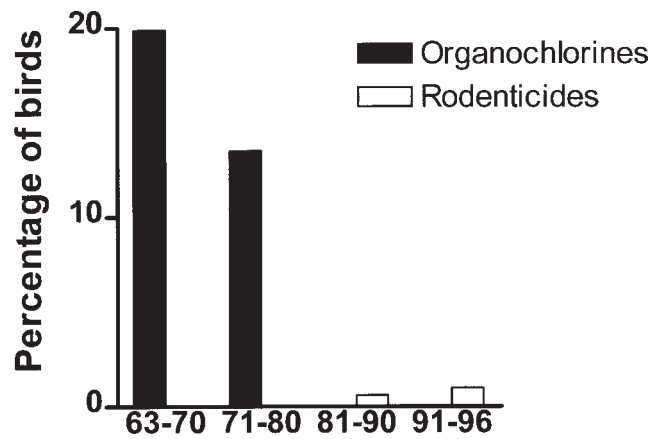


Figure 2.—Proportion of Barn Owls (*Tyto alba*) examined in Britain whose deaths were attributed to organochlorine or rodenticide poisoning in four successive periods.

The geometric mean HEOD level in all these birds was 14.3 ppm. Two other deaths were attributed to poisoning by heptachlor epoxide, and were associated with liver levels of 14.4 and 26.0 ppm HE, and two to poisoning by DDE (130 ppm and 270 ppm). The latter also contained 55 ppm of TDE, another breakdown product of DDT. In addition, one of the birds classed as a HEOD victim also contained 700 ppm DDE. Some of these apparent pesticide victims were thin, so that loss of body fat may have contributed to high residue levels in the liver (Newton *et al.* 1991).

Of 557 birds examined during 1983-1994, 132 (24 percent) were found to contain residues of rodenticides, either difenacoum, brodifacoum, bromadiolone, flocoumafen or more than one of these compounds (table 3). Moreover, the proportion of birds in which residues were detected increased over the years, reaching around 32 percent in 1993-1994. This reflected the increasing use of these chemicals as warfarin replacements and showed that Barn Owls have become increasingly exposed to them.

In total, however, only eight birds were diagnosed as having died of rodenticide poisoning. In the seven that showed typical hemorrhage symptoms, the following residues (mg kg^{-1}) were detected in livers: (1) 0.13 bromadiolone, (2) 0.05 bromadiolone plus 0.003 flocoumafen plus 0.002 brodifacoum, (3) 0.17 difenacoum, (4) 1.07 bromadiolone, (5) 0.87 brodifacoum, (6) 1.72 bromadiolone plus 0.07 brodifacoum, (7)

Table 3.—Percentage of Barn Owls (*Tyto alba*) from Britain that contained rodenticides in different periods.

	Number of owls analyzed	Number (percent) containing residues
1983-1984	18	1 (6)
1985-1986	75	9 (12)
1987-1988	61	8 (13)
1989-1990	133	31 (23)
1991-1992	139	41 (29)
1993-1994	131	42 (32)

Significance of variation between periods: $\chi^2_5 = 20.4$, $P < 0.001$.

0.33 bromadiolone. The eighth bird, that showed no hemorrhage symptoms, contained 0.42 mg kg^{-1} brodifacoum. It was classed as a rodenticide victim because of the relatively high brodifacoum level present and because it showed no other obvious cause of death.

Surprisingly, there was little seasonal variation in the prevalence of different forms of mortality, apart from the HEOD victims which came mainly in spring. Road and other accidents were the main form of loss throughout the year, and starved birds were found in every month, even in May-July. Although the birds examined might have lost weight through water loss by the time they reached us, the recorded weights should be comparable between different categories. Restricting analysis to the period October-March, outside the main breeding season, when weights of live birds are relatively stable (Taylor 1989), accident victims were the heaviest, while not unexpectedly those diagnosed on appearance as starved were the lightest (fig. 3). Among accident victims, males averaged 291 g and females averaged 5 percent heavier at 305 g. Most birds of both sexes that were classed on autopsy as starved weighed less than 240 g, and the average weights of starved birds of each sex was about 30 percent less than accident victims. Birds diagnosed as aldrin-dieldrin casualties were generally intermediate in weight between accident birds and starved birds (Newton *et al.* 1991), possibly because they had become immobilized sometime before their death. There was no obvious sex bias in any form of mortality, including drowning, which in Shawyer's (1987) sample was confined to females.

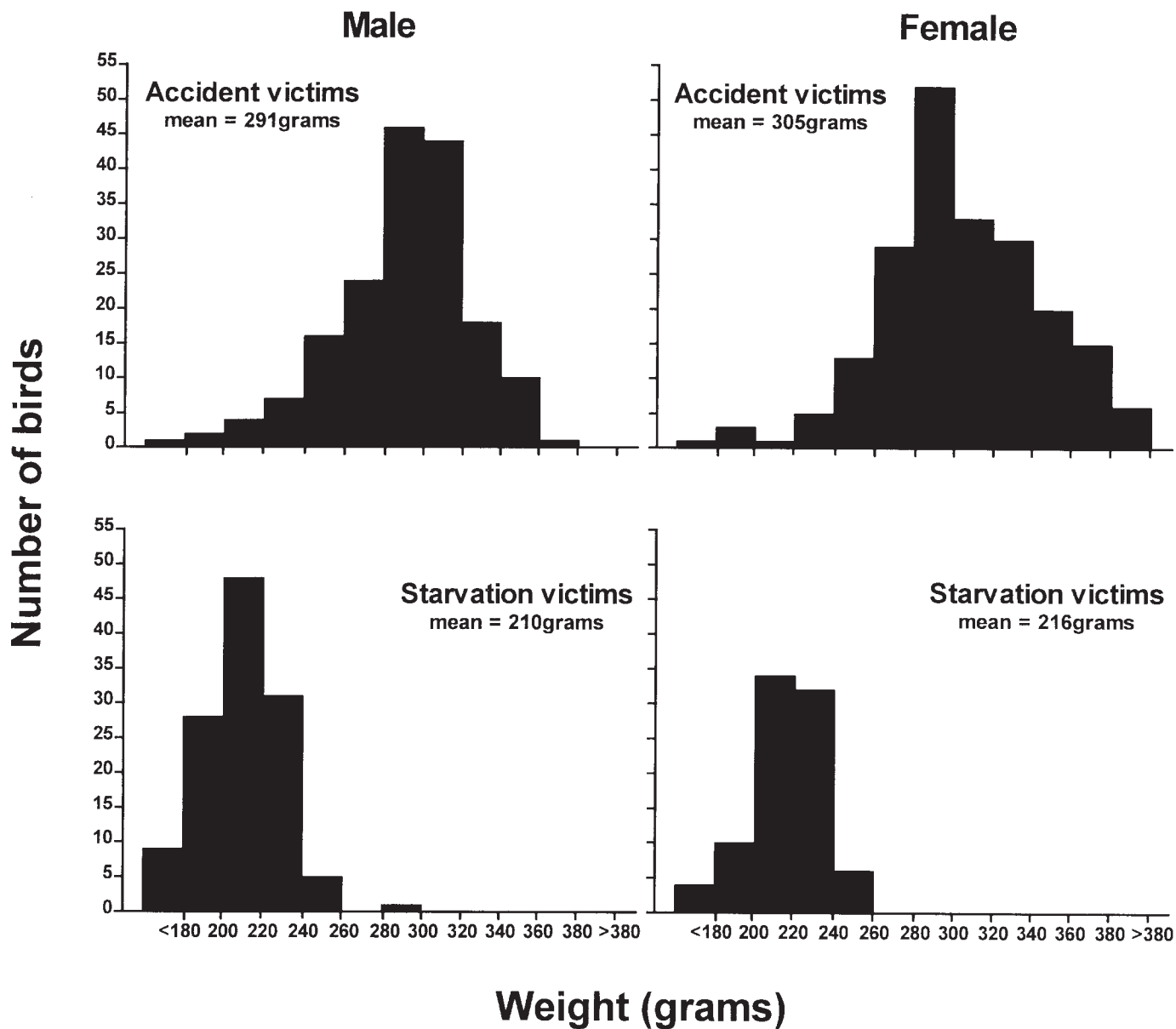


Figure 3.—Body weight during October-March of Barn Owls (*Tyto alba*) in Britain whose deaths were attributed by autopsy to accident (trauma) or starvation.

Some causes of death changed progressively in frequency over the years. Road casualties formed only 35 percent of the sample in 1963-1970 but had increased to 50 percent in 1991-1996. Organochlorine pesticide casualties declined from 20 percent in 1963-1970 to 14 percent in 1971-1980 and to nil in 1978-1996, although only 50 randomly-related birds were analyzed after 1977. Other causes of mortality changed in proportion, but with no consistent trends.

Frequency of Rodenticide Contamination

The dates from which various second generation rodenticides were used in Britain are given in table 4, along with their toxicities to rats and mice, compared with warfarin. In terms of LD_{50} values (lethal dose for 50 percent of a sample, expressed as $mg\ kg^{-1}$ body weight), the new chemicals are roughly 100-1,000 times more toxic than warfarin. It is the combination of greater toxicity and greater persistence which gives the potential for secondary poisoning of rodent predators.



Table 4.—*Toxicities of some rodenticides.*

	Year of introduction to Britain	Lethal dose (mg kg ⁻¹)	
		Rat	Mouse
Warfarin	1952	185	375
Difenacoum	1975	1.80	0.80
Bromadiolone	1980	0.55	0.99
Brodifacoum	1982	0.26	0.40
Flocoumafen	1986	0.25	1.13

Their increased frequency in Barn Owl livers over the period 1983-1994 follows from their increased usage, as they have gradually replaced warfarin and other 'first generation' rodenticides. Moreover, the different chemicals have appeared in Barn Owls in proportion to their usage (table 5). It seems that our monitoring of residues has given a good reflection of changes in usage, and hence in exposure, of the British Barn Owl population.

DISCUSSION

The carcasses received probably did not represent a random cross-section of Barn Owl deaths but were biased towards those forms of mortality most readily detected by people. Ringing recoveries, which are often used in mortality studies, are biased in the same way, but our records had the additional information provided by autopsy and chemical analysis, thus revealing the significance of certain pesticides. They also revealed changes in the relative importance of different types of mortality over the years.

In important respects, our findings agree with those of Glue (1971), Shawyer (1987) and Hardy *et al.* (1982), also based on carcasses found by people. Similarities include (1) the marked seasonal pattern in recorded deaths, with peaks in autumn (due mainly to juveniles) and in late winter (due to both adults and juveniles), (2) the importance of collisions, especially with road traffic, as the major cause of reported deaths, followed by starvation, and (3) the lack of seasonal variation in the relative importance of the main causes of reported deaths.

Over the years, the proportion of recorded deaths attributed to road traffic increased: from 6 percent in 1910-1954 and 15 percent in 1955-1969 (Glue 1971, based on band recoveries), to 35 percent in 1963-1970 and 50 percent in 1991-1996 (our data). This is most readily attributed to the increasing numbers of roads, and the increasing number and speed of road vehicles over the period concerned. Associated declines in the incidence of other forms of reported mortality are notable for organochlorine poisoning, which fell from 20 percent in 1963-1976 to nil in 1981-1996, and for shooting, which fell from 12 percent in 1910-1954 to 5 percent in 1955-1969 (Glue 1971), to 1 percent in 1963-1996 (our study). The fact that few owls ($n=5$ in our study) were reported from railways is presumably because the tracks carry less traffic than roads and are less frequented by people able to pick up carcasses.

Accident victims were presumably over-represented in all these studies, because of ease of finding, while deaths from natural causes (especially predation) were under-represented. Some of our owls which died

Table 5.—*Rodenticide use and Barn Owl (Tyto alba) contamination in Britain.*

	Arable farms ¹	Livestock farms ¹	Barn Owls ²
Number examined	565	459	449
Number with rodenticide	431	404	120
Difenacoum	62%	54%	63%
Bromadiolone	32%	37%	40%
Brodifacoum	5%	7%	14%
Flocoumafen	0.5%	1.5%	5%

¹ Based on a questionnaire survey of randomly selected farms, 1988-1989 (Olney 1991a, 1991b).

² Based on Barn Owls examined in 1988-1994.

from accidents were of normal weight and good condition. But others were thin, and at least 4 percent of male and 7 percent of female trauma victims weighed as little as starved birds. Poor condition may pre-dispose Barn Owls to accidents if it (a) leads them to spend more total time hunting, (b) leads them to spend relatively more time hunting in places where accidents are likely, such as road verges, or (c) makes them less able to avoid collisions. For such birds accidents are the secondary, rather than the primary, cause of death. The distinction is important because it implies that accidents have less effect on the population than their reported frequency would suggest. Nonetheless, most accident victims were of normal weight, so would presumably have lived considerably longer without the accident.

The seasonal pattern of reported mortality was more or less as expected from seasonal changes in population and food-supply (Taylor 1989). The May-July trough in recorded deaths coincides with the main period of breeding, when food is readily available and females are largely confined to their nests. The rise in mortality, from August to November, occurs when the Barn Owl population reaches its annual peak, through breeding. Such mortality falls mainly on the juveniles in the period when they become independent and disperse. As the breeding season extends in some years into November, the transition-dispersal period for the young is also spread over several months. Reported mortality remains high throughout the winter, but adults form a much greater proportion of casualties in February-March. By then the owl population is lower but the food-supply is also approaching its annual trough, perhaps deepened in some years by snow cover, and evidently leading to more frequent starvation and collisions.

Chemical analyses confirm that aldrin-dieldrin poisoning accounted for a large proportion of recorded Barn Owl deaths in the 1960s and 1970s. The owls examined contained some of the highest aldrin-dieldrin and heptachlor levels found in any birds of prey in Britain, including some on a par with the highest levels found in Sparrowhawks (*Accipiter nisus*) (Newton 1986). Lethal HEOD levels, found in owls which had other symptoms of organochlorine poisoning, but no evidence for any other cause of death, were in the range 6-44 ppm, with a geometric mean of 14 ppm. That these pesticides may have caused a decline in

Barn Owl populations in such regions is supported by survey results. Thus, Prestt (1965) found evidence for a decline in Barn Owl numbers, which was most marked in eastern counties, in the period 1956-1962, following the introduction of heptachlor, aldrin and dieldrin in agriculture. Heptachlor was withdrawn in 1964, and few Barn Owl livers examined after 1966 contained more than 0.5 ppm of residue. But aldrin and dieldrin remained in common use until 1976, after which they were much restricted. All the organochlorines mentioned in this paper were banned completely from 1986, so this source of mortality can be assumed to have disappeared.

The increasing contamination of Barn Owls with second generation rodenticides over the past 15 years was expected from the increasing use of these chemicals, which are gradually replacing warfarin and other first generation rodenticides. In 1993-1994, about one-third of all Barn Owls received contained measurable residues of one or more compounds, but only a small proportion of birds (up to 3 percent of the total) contained residues large enough to have killed them. With yet further increases in usage, however, these chemicals could become a more important cause of mortality in the future.

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