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Health status of otters in southern and south west England 1996-2003

Science Report: SC010064/SR1

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Executive Summary

The reasons for the dramatic decline in the population of otters (*Lutra lutra*) in Europe that started in the late 1950s have been the subject of much debate. Possible explanations that have been proposed include exposure to environmental pollutants, over hunting, habitat destruction, human disturbance, acid rain, competition with introduced American mink (*Mustela vison*) and infectious diseases. In Britain it is widely accepted that exposure to organochlorine pesticides was the principal factor, although until recently this conclusion was based almost entirely on circumstantial evidence. In the rest of Europe the role of organochlorines is less clear and most research has been concentrated on the possible influence of polychlorinated biphenyls (PCBs) and related compounds. This study is a continuation of one started by the author in 1988 to investigate the health of the otter population in south west England and, in particular, to determine whether otters were being adversely affected by environmental pollutants.

Between 1996 and 2003 post mortem examinations were carried out on 341 otters found dead in south west and southern England. The majority of deaths (83 per cent) were due to road traffic accidents and there was a marked seasonal pattern to mortality, with reduced deaths in the summer months. Bite wounds and dental lesions were the other main causes of death (11 per cent). It is thought that most of the bites were inflicted by other otters but some were due to conflict with American mink and others by domestic dogs. The population appears to be expanding, with increased numbers submitted each year and good evidence of recolonisation of some southern counties. However, coincidentally, there has been a marked increase in the proportion of otters suffering from bite wounds, possibly due to competition for territory. There was no evidence of significant reproductive disease and almost half of all adult females showed evidence of recent breeding. No bacterial or viral diseases likely to pose a threat to the otter population were identified. There was evidence of a potentially damaging parasite not previously recorded in British otters, the bile fluke, *Pseudamphistomum truncatum*.

Analysis of livers showed that concentrations of the organochlorine compounds pp'DDE, TDE and dieldrin have continued to decline over time but that there has been no similar decline in overall PCB or hexachlorobenzene (HCB) residues. There was clear evidence that otters accumulate the predominant organochlorine pesticides and PCBs with age, with higher concentrations in adult males than in non-adult males. However, levels in adult females were significantly lower than in adult males and were either equal to, or lower than, those in non-adults of either sex. It is likely that these low levels in adult females are due to them off-loading a significant proportion of their pollutant burden into their cubs, mostly by secretion in colostrum and milk.

Liver vitamin A levels in adult females were significantly higher than in adult males and were lowest in non-adult females. Levels in cubs and non-adults rose significantly over time. Although a statistical relationship to pollutant levels was not proven, the results are consistent with experimental studies showing that animals exposed to organochlorines and PCBs have low levels of vitamin A.

Thyroid size was positively associated with hepatic pp'DDE levels and this is consistent with endocrine disruption. Thyroid size also declined over time as organochlorine levels declined. Reduced penis size and developmental eye defects were associated with high levels of organochlorine pollutants.

The study provides evidence that organochlorine pesticides that were banned decades ago are still present in the aquatic ecosystems of southern and south west England and are associated with physiological and developmental defects. However, their impact appears to be lessening as levels continue to fall. At present the otter population in this part of England is generally healthy and is recolonising former haunts, particularly in an easterly direction. As this expansion continues, and population density increases, the level of conflict between otters, and between otters and mink, is likely to increase. It is also inevitable that there will be an increase in reports of otter predation on trout farms, carp fisheries, koi carp collections and even garden pond fish.

Summary of main findings

1. In the period 1996-2003 post mortem examinations were carried out on 341 otters from southern and south west England. The numbers submitted each year increased, not only from the strongholds of Cornwall, Devon and Somerset but also from counties further east. The previously isolated populations in Hampshire and Dorset now appear confluent with those of Somerset and east Devon.

2. Submissions followed a marked seasonal pattern, with few otters received during the summer months. Monthly mortality was shown to be closely correlated to night length.

3. Almost 70 per cent of the otters were adults and 62 per cent of these were males. There was no evidence of a sex bias in the other age classes.

4. Otters killed by road traffic formed 83 per cent of submissions. The other main causes of mortality were bite wounds and infected dental lesions (11 per cent). Most bite wounds were thought to have been inflicted by other otters but some were believed to be due to American mink and others to domestic dogs. A small number of otters were killed illegally.

5. The proportion of otters suffering from bite wounds increased steadily from 16 per cent at the start of the study to 52 per cent in 2003. The great majority were believed to be due to intraspecific aggression. Both sexes were equally affected. Bites by domestic dogs were a significant cause of mortality in cubs.

6. There was evidence of sampling bias, with adult male mortality during the summer months almost four times that of adult females. Sampling bias may also explain the high proportion of otters suffering from bite wounds, as it is believed that otters engaged in aggressive interactions are more likely to be killed in road accidents.

7. Fractured or missing teeth became increasingly common, particularly in adults. In several cases a fractured upper carnassial tooth developed a root abscess which then led to a fatal bacterial infection.

8. Analysis of stomach contents confirmed that eels were the most common food item. Cyprinids, salmonids and amphibians were also regularly eaten. Remains of large carp and ornamental carp were seen on several occasions.

9. Respiratory tract lesions were very common. Many otters had scattered focal lung lesions caused by inhalation of fungal spores but similar lesions, thought to be due to spread of bacteria from infected bite wounds via the blood, became increasingly common. In a small number of cases lesions resembling those of tuberculosis were seen but cultural and histopathological tests proved negative

10. The predominant bacterial infections were those caused by *Streptococcus* spp., mostly in otters dying from bite wounds or infected dental lesions. Other pathogens isolated included *Listeria* sp., *Yersinia* sp. and *Salmonella* sp. All otters submitted in 2002-03 were screened for *Brucella* sp. and proved negative.

11. There was no serological or pathological evidence of viral infections and no significant parasites were seen. However, thickened, shrunken gall bladders and

fibrosed bile ducts were seen in three otters between 2000 and 2002. Subsequent tests in 2004 indicated that the lesions were almost certainly caused by the bile fluke *Pseudamphistomum truncatum*. This parasite had not been recorded previously in Britain.

12. Renal calculi were not observed in otters in south west England from 1988 to 1996. The first case was seen in 1997 and the annual incidence increased remarkably over the study period, with calculi present in 33 per cent of the adults in 2003. The reasons for this increase are not known.

13. Enlargement of adrenal glands and atrophy of other organs, including thyroids, spleen and thymus, was recorded frequently, particularly in otters which had died or been euthanased due to prolonged illness. Enlarged adrenals were also seen in lactating females and in otters with renal calculi. However, some apparently healthy otters had enlarged adrenals and regressed thyroids.

14. Adrenals in sick otters were significantly heavier than in healthy ones but there was no demonstrable relationship between adrenal mass and pollutant burdens. Otters which had been frozen had significantly lighter adrenals than fresh cases.

15. There was a strong positive correlation between thyroid weight and body length and sex. Thyroid weight was also positively correlated with hepatic pp'DDE levels and there was a decline in thyroid weights over time. Sick otters had lighter thyroids than healthy ones and frozen thyroids weighed less than fresh ones.

16. There were highly significant declines over time in the hepatic concentrations of pp'DDE, TDE and dieldrin. By contrast, no such declines were seen in levels of hexachlorobenzene or PCBs. Toxic Equivalent (TEQ) values also remained constant over the study period

17. Organochlorine and PCB levels showed evidence of accumulation with age, with levels in adult males higher than in non-adult males.

18. Organochlorine and PCB levels in adult males were significantly higher than in adult females. However, levels in adult females were equal or lower than in non-adult females.

19. Few of the otters examined were pregnant but there was good evidence that breeding was occurring, with 40 per cent of all females either lactating or showing signs of having bred.

20. Vitamin A levels were judged to be satisfactory in the majority of otters but in a small number of cases they were low. There were marked differences in levels between age classes, with the highest levels in adult females and the lowest in non-adult females. Vitamin A levels in cubs and non-adults rose significantly over time.

21. There was no correlation between hepatic concentrations of vitamin A and the organochlorine pesticides or PCBs. However, low vitamin A levels and high dieldrin levels appeared to be related to retinal defects in almost 30 per cent of otters examined up to the end of 1999.

22. Although pollutant levels were generally highest in adult males, the animal with the highest pp'DDE level (12,400 μg/kg), and the highest pollutant burden overall, was

an unweaned cub. It is likely that adult female otters reduce their organochlorine burden during reproduction, especially by excretion in to colostrum and milk.

23. A preliminary study showed a significant negative correlation between baculum length in young otters and hepatic organochlorine pollutant levels.

Contents

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Vic Simpson
December 2005

1. Introduction

1.1 Background

The dramatic decline of the otter population that occurred throughout much of Europe during the 1960s and 1970s has been well documented (Mason and Macdonald 1986) but the reasons for it have been the subject of much debate. A report by Chanin and Jefferies in 1978 provided circumstantial evidence linking the decline in Britain to the introduction of dieldrin and related organochlorine pesticides (OCs) in 1957. However, in a review of data from Europe and elsewhere, Mason (1989) concluded that the principal problem was due to polychlorinated biphenyls (PCBs). Other factors that have been considered include the effects of pollution on fish populations, acid rain, habitat destruction, disturbance and persecution by humans, road traffic, competition with American mink (*Mustela vison*) and infectious diseases (Mason and Macdonald 1986).

The main difficulty in assessing the significance of the various proposed factors was that although in Britain and elsewhere in Europe, otter tissues had been analysed for various pollutants, very few otters had been subjected to a detailed post-mortem examination (Keymer 1991). As a result, there was insufficient evidence to relate pollutant burdens or other factors to pathological changes. By the 1980s otters had become extinct from most of England but small numbers remained in the South West. In 1988, the author started to carry out post mortem examinations on otters found dead in Cornwall and Devon to establish whether pathological lesions were present in this population. For the first seven years the work was carried out on an ad hoc basis. From 1996 onward it was carried out under a contract with the Environment Agency. The contract made it possible to extend the study area to include otters from the Southern Region (Somerset through to Kent). The results of the investigations from 1988 to March 1996 are described in an earlier report (Simpson 1998). This report summarises the results obtained between April 1996 and the end of 2003.

1.2 Objectives

The overall objective was to continue to monitor otters in south west and southern England for evidence of disease, whether caused by infectious agents, metabolic, genetic, nutritional or toxic factors. Particular emphasis was placed on the possibility that any such disease might be the result of exposure to environmental pollutants, including organochlorine pesticides and polychlorinated biphenyls.

Additional objectives were to expand the 1988-1996 database of normal biometric parameters, record and analyse data for evidence of reproductive success and to collect samples for peripheral research projects, such as teeth for age determination and kidneys for DNA analysis.

2. Materials and methods

Otters found dead or dying in southern and south west England between April 1996 and the end of 2003 were collected by members of the public, conservation bodies, veterinary surgeons and Environment Agency staff and submitted for post mortem examination. In some cases the specimens were held deep frozen, sometimes for more than a year, until they could be transported. Otters found prior to January 2001 were examined at the Veterinary Laboratories Agency (VLA) Polwhele, Truro. After that date, examinations were made at the Wildlife Veterinary Investigation Centre, Chacewater. On arrival at the laboratory specimens were given a unique identification number. Freshly dead animals were examined on the day of delivery or within 24 hours.

2.1 Post mortem procedure

Specimens were weighed, measured and washed, taking particular care to examine the skin for puncture wounds. The post mortem procedure was as described previously (Simpson 1997, 1998) but was modified periodically to accommodate new lines of investigation or to conserve resources. From 2000 to 2003 the penises of 139 otters were collected. The baculum was cleaned by simmering in an oven at 90°C for approximately 12 hours and the soft tissues then removed.

With the exception of incomplete or badly damaged specimens, a Condition Index (CI) was calculated using the formula: $CI = W/aL^n$ where W = body weight in kilograms and L = nose to tail tip length in metres. The constants a and n were 5.02 and 2.33 respectively for females and 5.87 and 3.39 for males (Kruuk *et al.*, 1987).

Otters were ascribed to one of four age categories: cub, immature, subadult or adult. Cubs were defined as animals with one or more deciduous teeth present. Immatures were animals that ranged from cub size, but without any deciduous teeth, to those considered to be around twelve to fifteen months of age based on body size and immature gonads. Subadults were animals of adult body size but with no obvious dental wear, slender canines and gonads not fully developed.

In some cases otters had injuries consistent with them having been bitten. However, lesions were only recorded as bite wounds if they were open, unhealed punctures or tears or, in a few cases, if there was a pattern of bruising, haemorrhage and associated crush injuries typical of large dog bites.

2.2 Laboratory examinations

Where lesions suggestive of a bacterial infection were seen tissue samples or swabs were inoculated onto five per cent sheep blood agar and MacConkey agar and incubated for up to 48 hours. In selected cases samples were inoculated onto specialist media and, where appropriate, media were also incubated under microaerophilic or anaerobic conditions. Smears from infected wounds and from internal organs showing lesions were stained by Gram's stain and examined microscopically. Lesions suggestive of tuberculosis were cultured by the VLA for *Mycobacterium* sp. During 2000 and 2001 a standard set of tissues from each otter was also screened for possible mycobacterial infection. Following a request in 2002 from the Brucella Research Group, VLA Weybridge, a blood sample and a range of tissues was taken from all otters and screened for evidence of infection with *Brucella* sp.

Between 1996 and 2000 samples from all major organs were routinely taken for histopathological examination. From 2001 onwards, selected tissues, including thyroids, adrenals and uterus were retained in fixative but other tissues were only examined if there was evidence of gross pathology. Samples were fixed in 10 per cent buffered formol saline, embedded in paraffin wax, sectioned at 6μm and stained by haematoxylin and eosin and other stains as appropriate. Where possible, at least two upper incisor teeth were collected and placed in 10 per cent buffered formol saline for use in age determination studies.

Duplicate samples of liver for toxicological examination were wrapped in aluminium foil and stored at -20°C. They were submitted to the Environment Agency laboratory at Exeter or, after 2001, at Starcross where they were examined by gas liquid chromatography and mass spectrometry for a range of pollutants, including halogenated hydrocarbons. Liver samples collected up to September 1998 were also analysed for heavy metals by atomic absorption spectrophotometry. Until the end of 2000, samples of livers received in fresh condition were analysed for vitamin A at VLA Shrewsbury.

The fifth left rib was collected routinely and stored frozen for future heavy metal analysis. Kidney samples were taken and stored at -20°C for use in a DNA typing study at Exeter University. Various additional samples were collected during the study period following requests from other laboratories. These included lung samples for the Mycology Reference Laboratory of the Health Protection Agency (HPA), lung and muscle samples for Dr Karen Laurensen, Glasgow University, muscle samples for Dr John Dallas, Aberdeen University and kidney and blood samples for Dr Richard Birtles at Liverpool University.

3. Results of post mortem examinations

3.1 Numbers and origin of otters submitted

In total, 341 otters were examined between April 1996 and the end of 2003. The number found dead each year is shown in Table 3.1. Most specimens came from Cornwall, Devon and Somerset with lesser numbers from Dorset and Hampshire, a few specimens from Wiltshire, Sussex, Kent and one from Powys. A list of all specimens is given in Appendix I, with their laboratory reference number and the national grid reference showing where they were found.

Table 3.1 Number of otters submitted by year of death

	1996	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**	**2003**
Number	19[1]	25	31	47	57	31	63	63[2]

Notes: Five otters were submitted where the year of death was not stated.
[1]The total for the year was 24 but five occurred before April, and were therefore outside the study period.
[2]This figure does not take in to account the number of otters collected in 2003 but not submitted until 2004.

With the exception of 2001 there was an increase in the number of otters submitted each year. This follows the trend seen during the first eight years of the study (Simpson 1998) (Figure 3.1). The reduced number of submissions during 2001 was possibly due to the impact of the foot and mouth disease outbreak on observer activity. In addition there was a temporary break in the Environment Agency contract in 2001 when carcases had to be stored frozen.

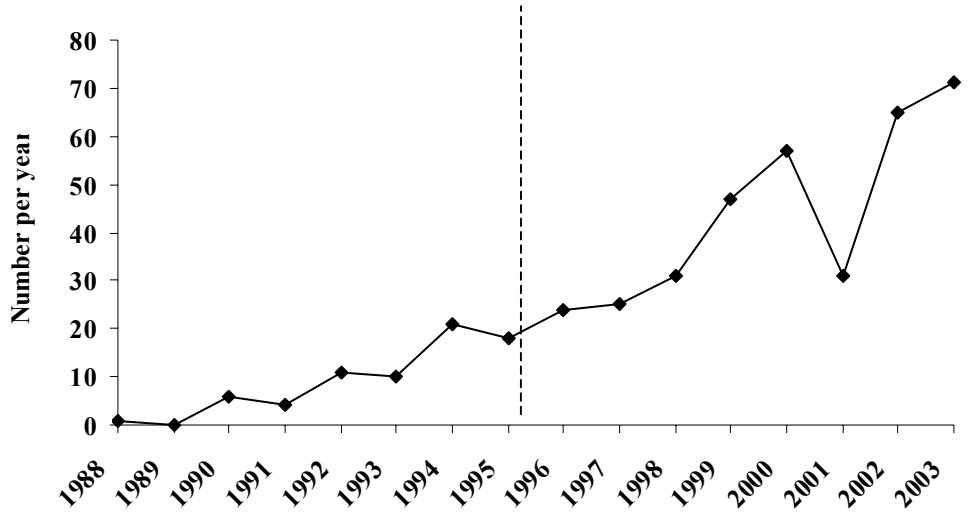

Figure 3.1 **Otter submissions by year of death.** The vertical dotted line shows the start of the present study. The trend recorded from 1988 to 1996 for an increase in the number of deaths per year continued except in 2001.

3.1.1 Geographical distribution of submissions

Although the majority of otters came from Cornwall, Devon and Somerset (see Table 3.2), the geographical distribution of submissions changed significantly between 1996 and 2003. Very few were received from south and east Devon between 1988 and 1996 and the first case was not received from Dorset until December 1996. By 1999/2000 specimens were being submitted with increasing frequency from these and other areas further east and also from north Somerset (Figure 3.3). The cases submitted from east Dorset and west Hampshire show how the main population has become confluent with the formerly isolated population in and around Winchester.

Whilst the increase in submissions from the more easterly counties contributed to the overall annual increase, numbers submitted from the original strongholds of Cornwall, Devon and Somerset also continued to increase (Figure 3.2).

Table 3.2 Numbers of otters submitted by county of origin, 1997-2003

Cornwall	Devon	Somerset	Dorset	Hants	Wilts
95	105	73	20	18	2[1]

Notes: Single submissions were received from Sussex, Kent and Powys.
[1]This number is not a true reflection of otter mortality in Wiltshire as part of the county lies in the West Thames region and otters from there are sent to Cardiff University.

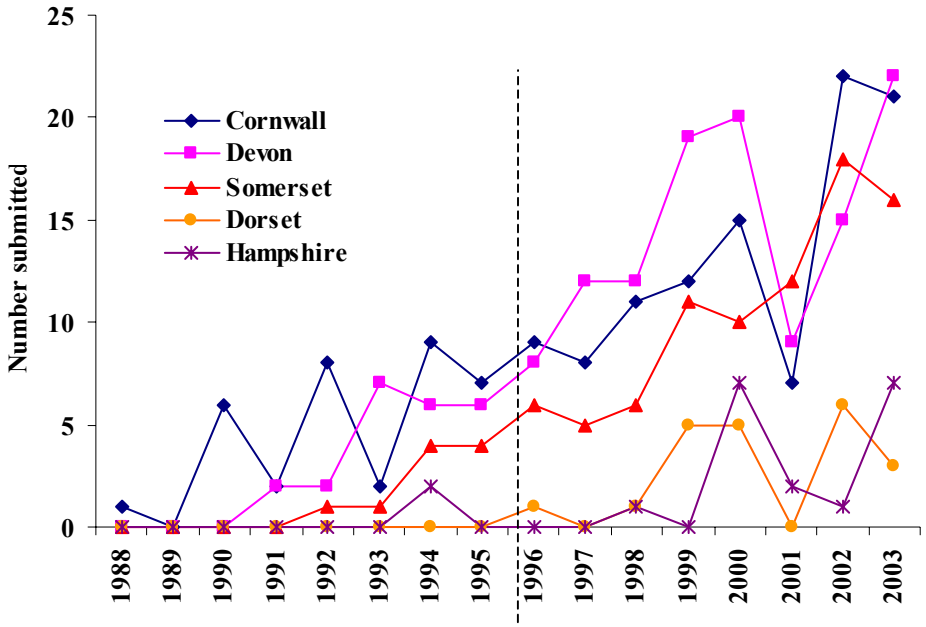

Figure 3.2 Otter submissions by county and year of death. From the start of the present study (vertical line), the pattern of steadily increasing submissions from Cornwall, Devon and Somerset continued. From around 1999 there was a noticeable increase in submissions from Dorset and Hampshire.

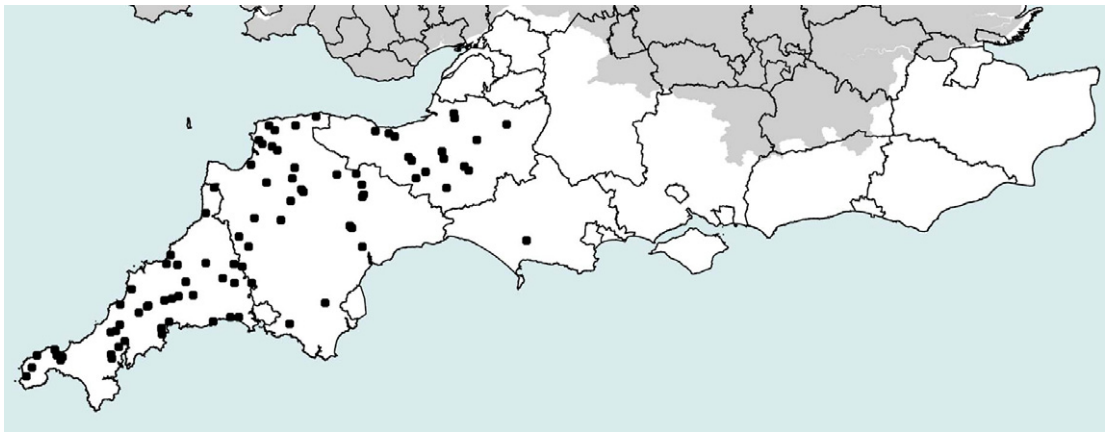

Figure 3.3a

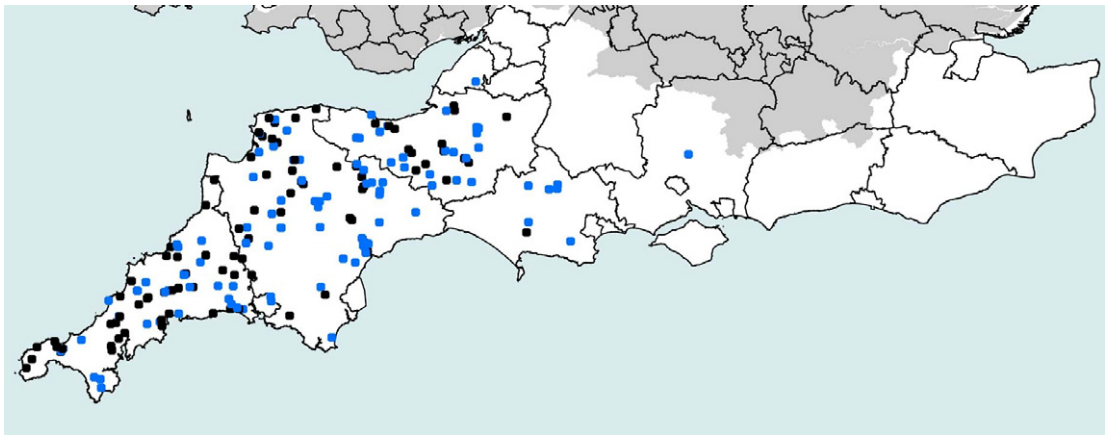

Figure 3.3b

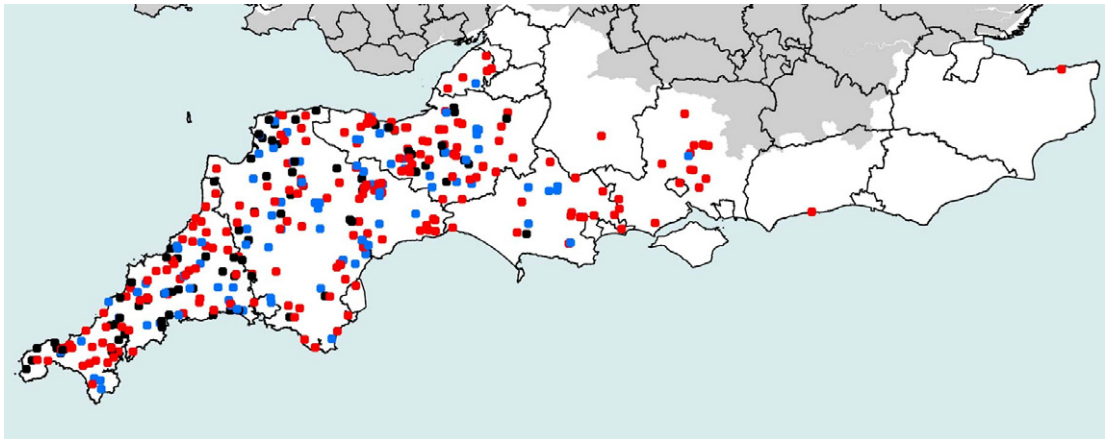

Figure 3.3c

Figure 3.3 **Geographical distribution of otters submitted.** The black spots in Figure 3.3a show the location of cases from 1988 to the end of 1996. In 3.3b the additional blue spots indicate the cases submitted in the period 1997-1999 and in 3.3c the red spots show the origins of cases submitted from 2000 onwards. Note the increase in cases from east Devon, Dorset and Hampshire during the present study period. Two otters from Hampshire in 1994 are not shown as they were submitted without grid references.

3.1.2 Seasonal variation in mortality

Otter mortality was not constant throughout the year and very few were received during the summer months (see section 3.2). The pattern was broadly similar for both sexes but there was a marked peak in male mortality during February and a similar, less pronounced, peak in October. During these months there were corresponding falls in female mortality. The peak of female mortality was between November and January (Figure 3.4).

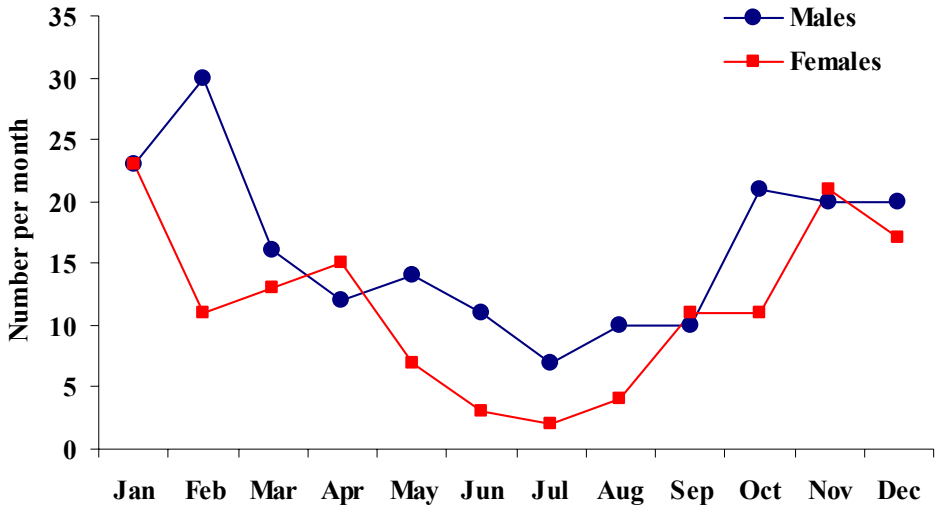

Fig / in both sexes was lowest in the summer months but there was a marked peak in male mortality in February.

3.1.3 Mortality relative to sex and age class

There was an overall sex bias, with 195 males and 141 females submitted (58 per cent males). However, this bias was due entirely to a predominance of males in the adult age class where the male/female ratio was 1.65 to 1. There was no significant difference in mortality between the sexes in any other age class (Table 3.3).

Table 3.3 Number of otters examined by sex and age class

Sex	Adult	Subadult	Immature	Cub	Total
Male	144	23	18	9	195
Female	87	26	16	10	141
Total	231	49	34	19	336

Note: In eight cases the carcases were incomplete or too badly damaged to determine age and/or sex.

The higher level of mortality in males was observed throughout the study period, although it was most marked in the period 2001-2003 (Figure 3.5).

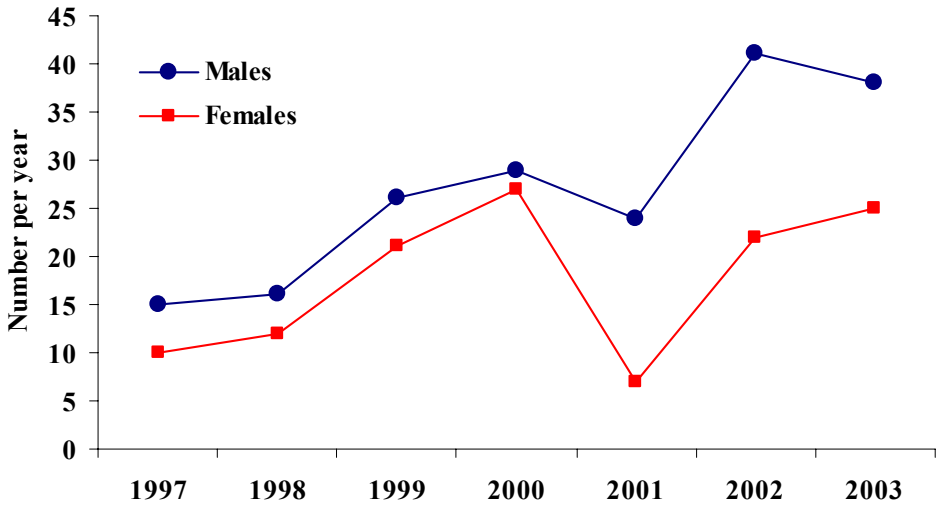

Figure 3.5 **Number of male and female otters submitted per year.** The reason for the reduced proportion of females after 2000 is not known.

The male: female mortality ratio varied according to the season, with proportionally more males dying during the summer and also in the months of February and October (Figure 3.6).

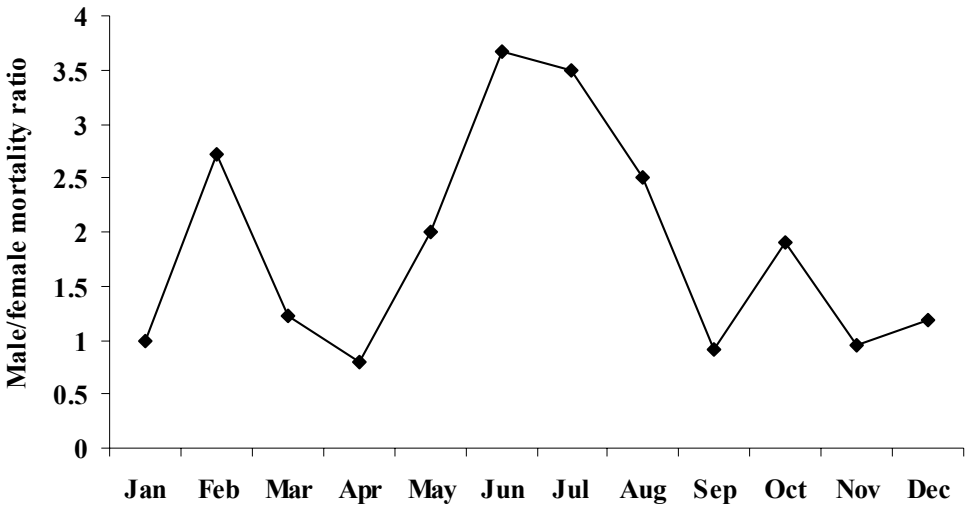

Figure 3.6 **Ratio of male to female deaths per month.** The reason for the high proportion of male deaths during February and the summer months is not known but is possibly related to breeding activity.

3.2. Causes of mortality

3.2.1 Mortality due to road traffic

Mortality due to road traffic accidents (RTA) was 83.6 per cent overall but the proportion of RTA deaths was not constant throughout the year, being lowest in the spring (Figure 3.7).

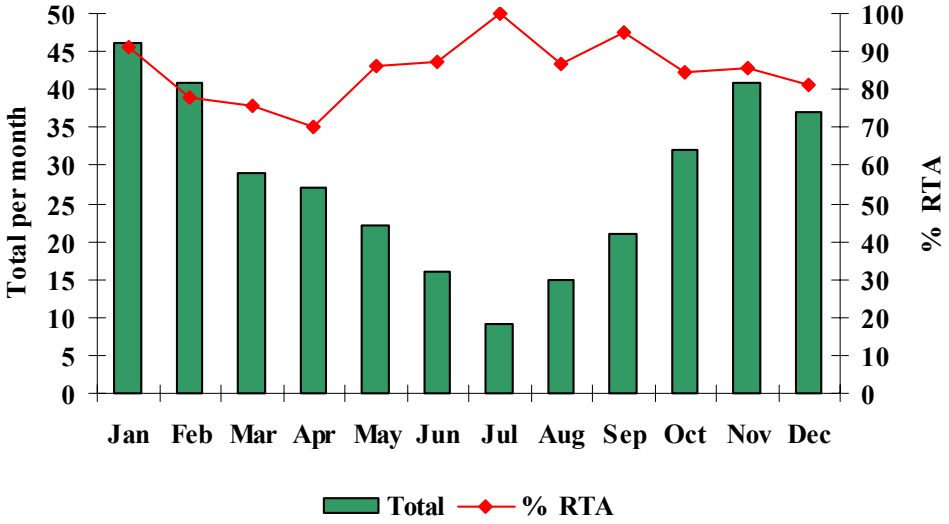

Figure 3.7 **Total otter deaths per month and proportion killed by traffic.** Note that although total mortality fell during the summer months, the proportion killed by traffic was highest at this time. There was a relative decrease in road traffic deaths during the spring months.

As noted in the 1988-96 report (Simpson 1998), otters were often killed in road accidents during or following storms and when rivers were in spate. This mostly occurred during the autumn and winter months. However, it became apparent that these incidents alone did not explain the overall seasonal pattern of mortality. As otters are largely nocturnal, monthly mortality was examined in relation to the hours of darkness. This showed that both total mortality and total RTA deaths per month are closely correlated with night length (Figure 3.8). Statistical analysis of combined data from the present study and the 1988 to 1996 study confirmed a highly significant positive correlation between mortality and night length (Simpson 2006).

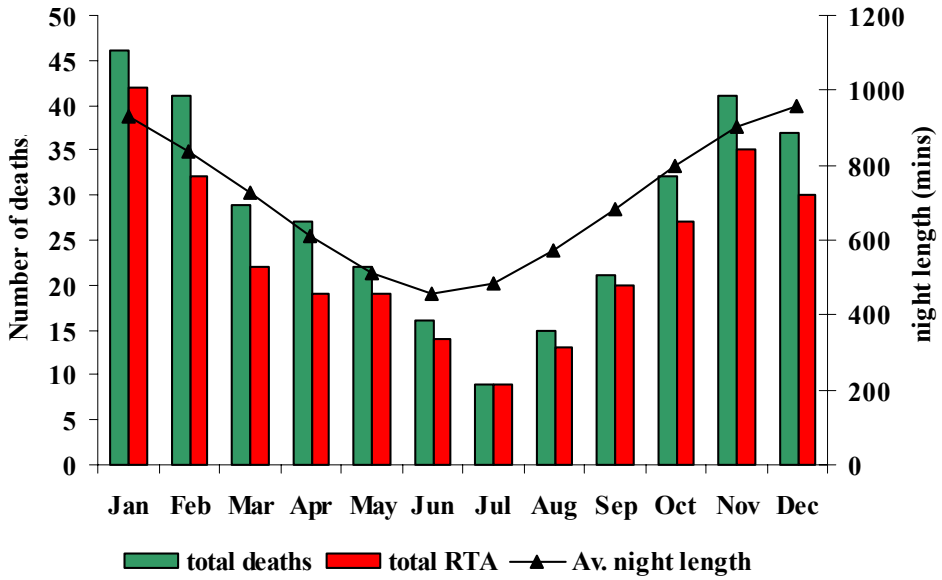

Figure 3.8 **Total otter deaths per month and road traffic deaths in relation to hours of darkness.** Both total mortality and road traffic mortality were closely correlated with monthly average night length.

3.2.2 Mortality due to bite wounds

Irrespective of the apparent cause of death, many otters had injuries that were judged to be recent bite wounds. Some carcases were incomplete or too badly damaged for assessment of bite wounds and 42 were therefore excluded from the dataset. Of the 299 otters remaining, 116 (38.7 per cent) had bite wounds.

The great majority of wounds were punctures and/or tears to the skin consistent with damage caused by canine teeth. As described previously (Simpson 1997; Simpson & Coxon 2000), there was a definite pattern, with almost all of the bites occurring on the head, feet and perineum. In many cases all three sites were bitten. Some of the wounds were minor but in other cases there was extensive tissue damage and some wounds had gone septic. Often it was not possible to estimate the spacing of the aggressor's canines but in some instances there was a clear pattern of a bite. In the most frequently seen pattern the punctures were around 18 to 22 mm apart and these match the spacing of an otter's canines. However, in a few cases, mostly between 2000 and 2003, there were punctures spaced at around 10 mm apart (Figure 3.9). These wounds are consistent with the otter having been bitten by an American mink (*Mustela vison*).

Figure 3.9a

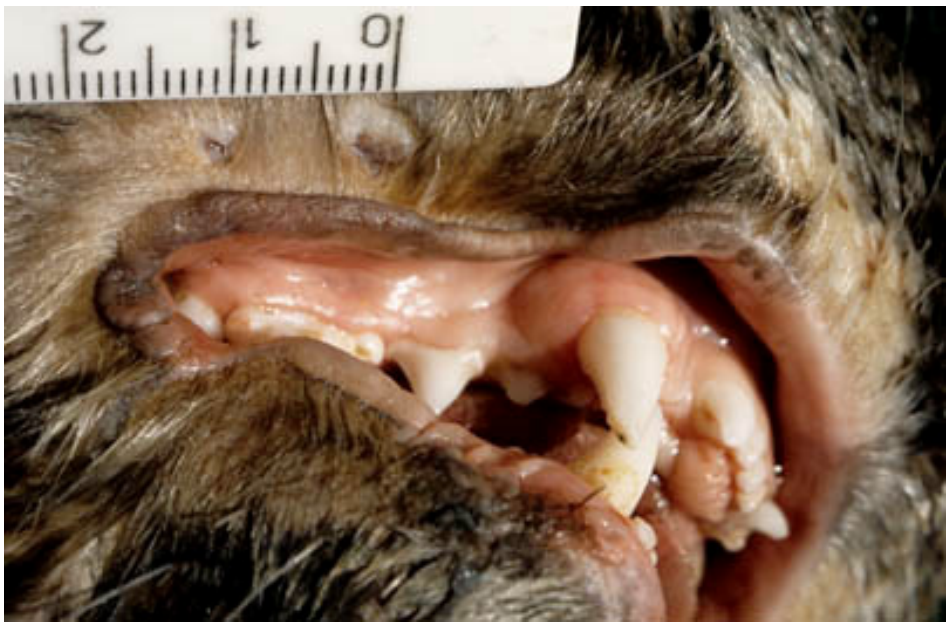

Figure 3.9b

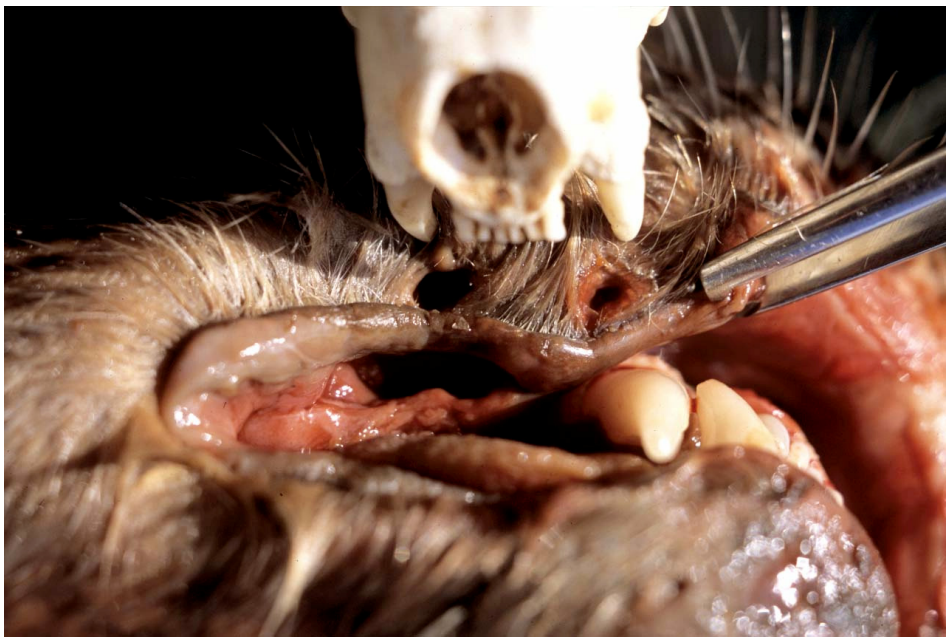

Figure 3.9 **Bite wounds on otters due to American mink.** The spacing of the wounds in Figure 3.9a is approximately 10 mm, consistent with the otter having been bitten by an American mink. In a similar case (Figure 3.9b) the spacing of the wounds can be seen to match that of the canines in a mink skull.

A second type of bite wound occurred only in cubs and a few immatures. These were animals that were either known or suspected to have been bitten by domestic dogs. The lesions in these cases involved the trunk, particularly the thorax, and typically there was severe bruising and haemorrhage, tearing of intercostal muscles, fractured ribs and ruptured organs. Puncture wounds to the skin were not always obvious but where seen they were much larger in diameter and more widely spaced than those in the main bite wound group. A small number of subadults and adults had large bites to atypical sites, such as throat, abdomen or hind limb, that were also probably due to attack by dogs. In a few cases otters had wounds that appeared to have been inflicted by more than one species, for example otter bites followed by dog bites.

3.2.2.1 Changing prevalence of bite wounds

In the period 1988-1996 the prevalence of bite wounds was around 16 per cent and males were almost twice as likely to be bitten as females (Simpson 1997). However, the proportion of otters with bites has increased markedly over time and for the year 2003 the overall figure was 51.6 per cent, with bites equally common in females (Figure 3.10).

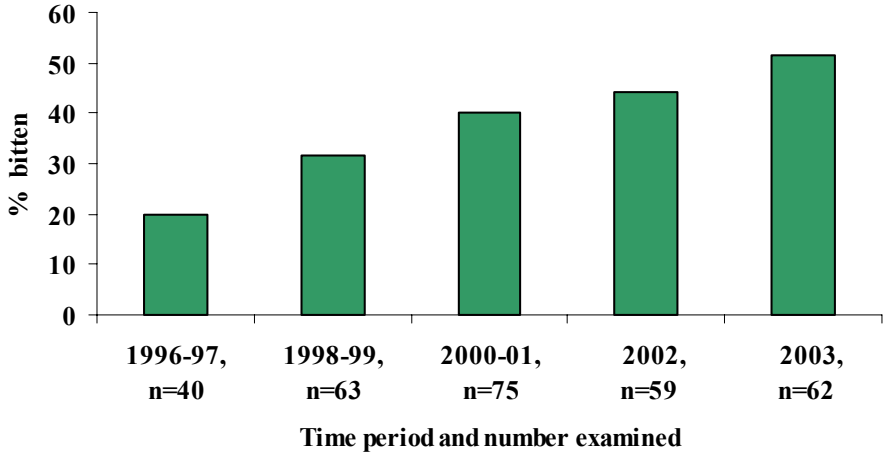

Figure 3.10 Proportion of otters with bite wounds 1996-2003. The number of otters submitted in the periods 1996-1997, 1998-1999 and 2000-2001 were pooled to obtain similar size groups for comparison. Over the eight year study period bite wounds were seen in 116 of the 299 otters that were suitable for examination (38.7 per cent).

The prevalence of bite wounds varied according to the season, with the lowest level in the spring months. Surprisingly, the highest level was in mid to late summer when submissions were at their lowest. During this period bites were much more common in males than females. Figure 3.11 shows the total submissions per month and the percentage bitten, excluding all cases that were known or suspected to have been bitten by domestic dogs. Most of the otters suffering from bites between July and September were adults, with approximately 50 per cent of them affected. By contrast, bite wounds in subadults were confined to the period September to February (Simpson 2006).

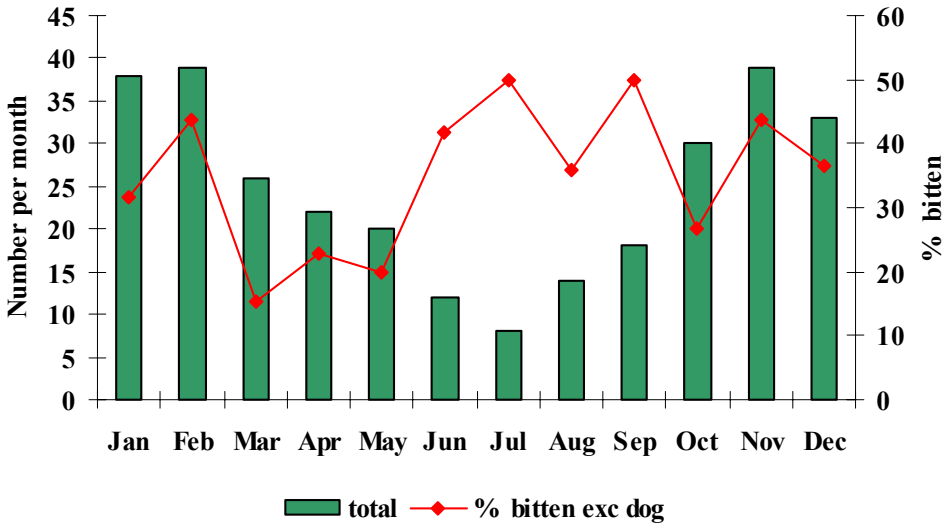

Figure 3.11 Total otter submissions per month and proportion bitten (excluding dog bites). The proportion of otters with bite wounds was lowest in the spring months and highest in the summer.

Between 1996 and 2003 bite wounds accounted for 10.7 per cent of all deaths and, after road traffic accidents, were the second most commonly recorded cause of mortality. In cases where cubs and immatures were bitten by domestic dogs the wounds were usually immediately fatal but otters also died as a result of wounds becoming septic. If deaths caused by domestic dogs are excluded the mortality from bites is approximately 7 per cent.

Otters dying from septic bite wounds were mostly in poor condition, some having lost around an estimated 50 per cent of body weight. Excluding cases believed to have been bitten by dogs, the mean Condition Index for males and females dying of bite wounds was 0.76 and 0.73 respectively. By contrast, males and females without bites both had a mean CI of 1.03.

3.2.2.2 Baculum fractures

Although bite wounds to the scrotum were common, bites to the penis were rarely seen. However, 11 out of the 139 baculi examined (7.9 per cent) showed evidence of a fracture and it is strongly suspected that these arose as a result of the penis being bitten. In some cases the lesion had healed, usually with callus formation, but others had remained fractured or formed a false joint (Figure 3.12).

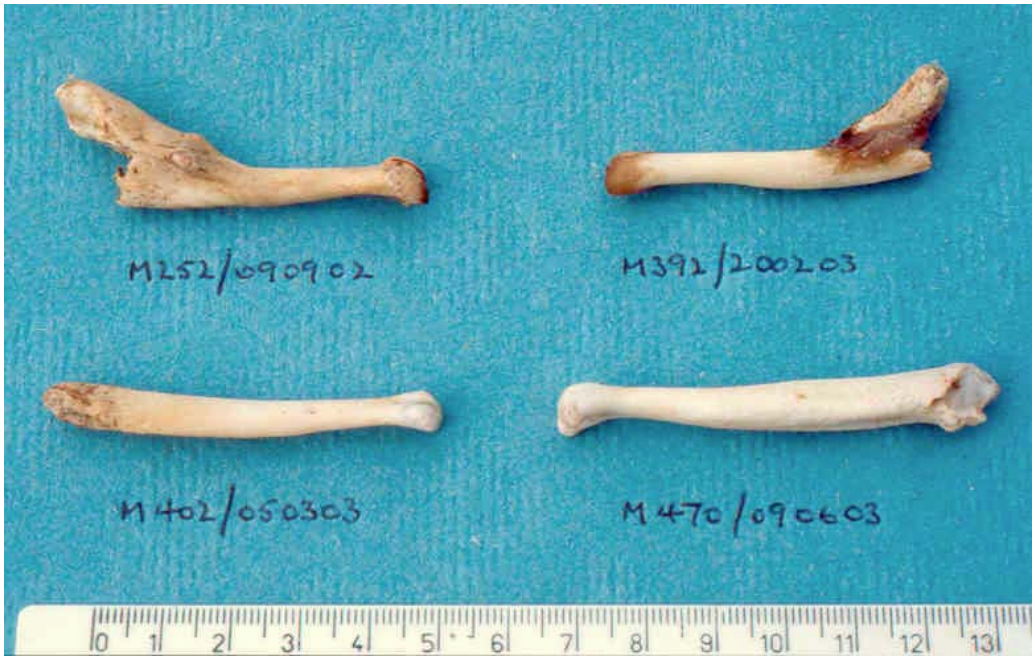

Figure 3.12 **Fractured and normal baculi.** The two upper baculi have been fractured, probably due to the penis being bitten. The one at top left has formed a callus around the fracture and the other has formed a fibrous union. The two lower baculi are normal.

3.2.3 Mortality due to dental lesions

Trauma from road traffic often made detailed examination of teeth difficult and therefore 41 specimens had to be excluded. The following assessment was carried out on the remaining 300 otters.

In general the dental health of the otters was good, with very little calculus or caries present in the majority of cases. Erosion of the enamel in an oval pattern on the rostro-lateral aspect of the lower canines was seen in the majority of adult otters. This did not appear to be of any clinical significance and was thought to be due to abrasion, probably as a result of some aspect of feeding behaviour. However, many adults had damaged or missing teeth (Figure 3.13) and the proportion of otters with such lesions increased over time (Figure 3.14).

The teeth most commonly damaged were the incisors, especially those in the lower jaw (Table 3.4). However, fractures involving the canines and cheek teeth were more significant as these often resulted in a root abscess and osteomyelitis. In four such cases where there was a fracture of an upper fourth premolar – or carnassial tooth – the abscess led to septicaemia and fatal pleurisy and pneumonia. However, some otters killed in road accidents or that died from bite wounds also had infected fractured teeth that, in time, would almost certainly have proved fatal. Dental lesions were slightly more common in otters suffering from bite wounds, with 41 per cent of bitten adult males and 34.7 per cent of adult females affected.

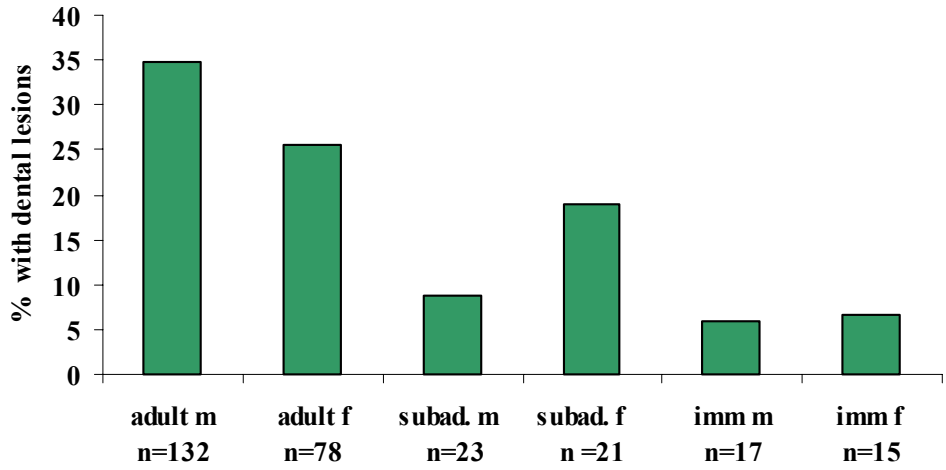

Figure 3.13 The proportion of otters with dental lesions by age class.

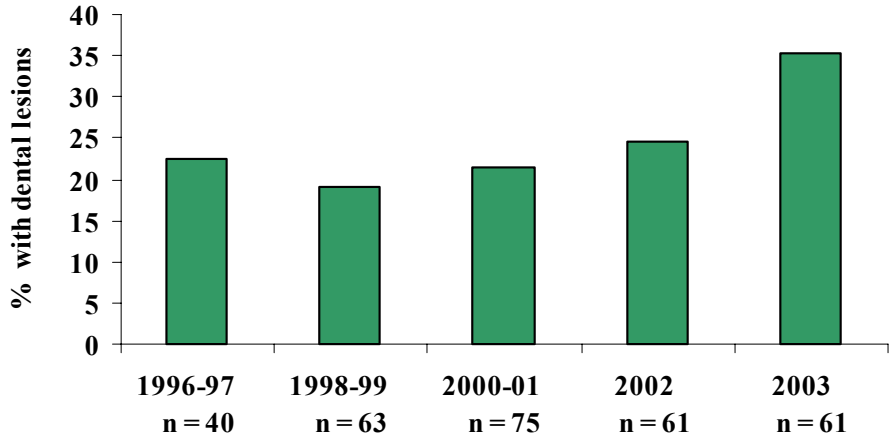

Figure 3.14 Proportion of otters with dental lesions 1996-2003. The proportion of otters with dental lesions has increased over the study period. The reasons for this are uncertain, although they could be related to the increase in intraspecific aggression.

Table 3.4. Distribution of dental lesions by sex and age class

	Adults		Subadults		Immatures	
	males	**females**	**males**	**females**	**males**	**females**
Total otters	132	78	23	21	17	15
Number (per cent) of otters with lesions to:						
Incisors	27 (20.5)	12 (15.4)	1 (4.3)	4 (19.0)	1 (5.9)	0 (0.0)
Canines	18 (13.6)	11 (14.1)	1 (4.3)	0 (0.0)	0 (0.0)	0 (0.0)
Premolar/ Molars	18 (13.6)	4 (5.1)	0 (0.0)	0 (0.0)	0 (0.0)	1 (6.7)

3.2.4 Otters killed illegally

Two otters were found dead in Fyke nets, one on the River Stour in Hampshire and the other at Powderham, Devon. There were other carcases found in water, that may also have died through drowning, including one on the Sussex coast, but which were too autolysed for detailed examination.

One of the most significant cases was an adult female found partway down a cliff on the north coast of Cornwall. The facial bones had been smashed but the cranium had remained intact. The trachea and lungs were full of inhaled blood, indicating that the animal had continued to breathe for a while after it suffered the blow to the head. There were two near-term foetuses in the uterus. The stomach contained bones and scales of large carp. In view of these findings, and the fact that the carcase was found well away from a road, and where the habitat was not typical for a breeding otter, the death was regarded as suspicious.

Some weeks later an animal welfare organisation submitted a steel wire snare which they had collected from a commercial coarse fishery not far from where the otter had been found. There were hairs trapped in the eye of the snare and microscopic examination indicated that they were otter hairs. Samples were submitted for DNA analysis and it was confirmed that the hairs were from an otter. Unfortunately, there was insufficient genomal DNA from the hair sample to provide a match with the DNA from the animal found on the cliff. Nevertheless, it was clear that the snare had been used to catch an otter. It was disappointing, therefore, that no prosecution under the Wildlife and Countryside Act was attempted.

3.3 Gross pathology

The state of carcase preservation varied considerably, with many specimens damaged by autolysis and/or trauma. In addition, 172 bodies had been stored deep-frozen, some for several years, and this often made meaningful interpretation of pathological lesions impossible.

Most otters were in good general condition. The median body weights for adult males and females were 8.2 and 5.7 kg respectively. The two heaviest males both weighed 11.3 kg,

one originating from Hampshire and the other from Dorset. There were three more males weighing over 10 kg, two from Devon and one from Somerset. The three heaviest females, weighing 7.6, 7.3 and 7.0 kg, all came from Devon, with another weighing 7.0 kg from Hampshire.

It was apparent that some otters were considerably underweight due to illness or the stress of rearing cubs. Therefore body length, rather than weight, was taken to be a more reliable indication of size. For adult otters the median lengths of males and females were 115 and 103 cm respectively. The two longest males, one from Dorset and the other from Somerset, measured 122.5 cm. There were six more males with a total length of over 120 cm, two from Hampshire, one from Dorset, two from Somerset and one from Devon. The top ten females ranged from 106.5 to 110 cm, with cases coming from Cornwall (3), Devon (3), Somerset (2), Dorset (1) and Hampshire (1).

The Condition Index could not be calculated for all the otters as measurement of both body weight and length was not possible in every case. The mean CI for the 152 males and 103 females of all ages was the same at 1.00 and the medians were also the same at 1.03. In adults, the mean CIs (medians in parenthesis) were slightly higher, with values of 1.03 (1.05) for males and 1.04 (1.07) for females and respective ranges of 0.59 to 1.28 and 0.56 to 1.39. In many cases a low CI was clearly associated with bite wounds.

3.3.1 External features

In the great majority of otters the coat was in good condition. There was some variation in coat colour, with occasional cases being unusually dark and others very pale. There were five cases of so-called 'Royal Otters'. These had a pattern of white spots in the fur similar to that of fallow deer (*Dama dama*). The spots varied in number and size from case to case but were mostly about 0.5 to 1 cm diameter. One specimen came from Devon and the other four from Cornwall. However, they came from widely separated locations and it seems unlikely that they were related.

Small numbers of ixodid ticks were seen occasionally, mostly around the head and neck. Heavier infestations were recorded in several sick animals but were also seen in four adult males and one immature female killed in road accidents and which were in good condition. In these cases the level of parasitism did not appear to be of clinical significance.

Injuries to the feet were common, particularly puncture wounds and tears to the digits or the pads. In some cases digits had been amputated. These injuries were typically seen in otters showing evidence of fighting. One otter from Hampshire (M12/7/00) had a deformed left front foot with abnormal angulation of digits four and five. The defect could have been a congenital abnormality or due to trauma at an early age. This otter, which had an unusually pale coat, is of interest because another case submitted from Hampshire in 1994 (M290/6/94) also had abnormal feet and was thought to have been a captive release (Simpson 1998).

The only cases of ocular disease visible on gross examination were those associated with trauma. Two adult males, both in emaciated condition and with multiple bite wounds, had a punctured, collapsed eye and one of them also had a torn lid on the opposing eye. Unilateral corneal opacity was seen in a subadult male and one adult female had a healed tear to its right eyelid and a lenticular cataract in the left eye. The lesions in both these animals were related to bite wounds.

3.3.2 Musculo-skeletal system

As many of the otters had been damaged by traffic it was frequently difficult to examine them for musculo-skeletal defects. Muscle condition in diseased animals and those with extensive bite wounds was usually poor but most otters had well developed skeletal muscles and no obvious bone or articular lesions. An adult male captured in very poor condition on a trout farm (M9/4/00) had been badly bitten, probably by a domestic dog, but also had an improperly healed fracture of the left femur and callused fractures of the tibia and the tail. In addition it had ulcerated digits on three feet with the bones exposed. It was thought that the leg injuries were probably the result of an earlier road accident and the other conditions were secondary developments.

Jaw deformities were seen in three cases. The first was an immature (M44/171201) found ill in Devon and submitted to the RSPCA with a bite wound to the right side of the face. Over a two-month period it developed chronic sinusitis and a deformed jaw and was therefore euthanased. On post mortem the original wound had healed but the maxilla, premaxilla and also the mandible on the right side were markedly shorter than those on the left. There were healed rib fractures on both sides of the chest close to the spine and it is probable that when it was originally found the animal had been bitten by a domestic dog. A very similar facial deformity was seen in one of a pair of cubs found abandoned near Truro, although in that case the mandible was normal. In both cases, it was suspected that the deformities had developed as a sequel to trauma or inflammation involving the bones when the animals were younger.

The third case was an immature with an overshot jaw. It was one of two young otters with markedly deformed skulls. They had originally been found as abandoned cubs in the South West Region and submitted to the Vincent Wildlife Trust (VWT) rehabilitation centre in Scotland (See section 3.3.3.12).

3.3.3 Internal organs

All major organs were examined where possible except for the brain, which was only examined routinely until January 2001. Detailed examination of organs was often impossible due to autolysis or decomposition. Many specimens had also been frozen, irrespective of their state of freshness and this reduced their diagnostic value even further. As the great majority were road traffic casualties, there was often severe traumatic damage and in some cases the internal organs were totally crushed and no meaningful examination could be carried out.

3.3.3.1 Alimentary system

Apart from those associated with bite wounds and dental disease, no significant lesions were seen in the mouth or oesophagus of any of the otters. One otter, submitted as a suspect poisoning case, died from starvation caused by a short length of alder stick becoming jammed between the teeth across the roof of the mouth. It is not uncommon for sticks or bones to become lodged in this way in domestic dogs and cats but the author had not seen it previously in a wild animal.

The stomach contents were examined grossly at the time of post mortem and eels and frogs were the most readily identified food items. Prey size varied, some otters having ingested large numbers of very small fish. There were other cases where the stomach contained scales and/or bones typical of large carp. Suspected salmonid remains, including ova, were occasionally seen. Recognisable mammalian prey items were uncommon, although one otter had a large fur ball in the stomach that was thought to be

mink hair. Unfortunately, attempts to have this confirmed by DNA analysis have so far been unsuccessful. An otter from Newquay had a large quantity of domestic chicken feathers in its stomach but there was normally little evidence of birds being eaten.

Stomach contents from 162 otters were submitted to Robert Britton at the Environment Agency Fisheries laboratory, Brampton for specialist identification of prey species and the results are summarised in Figure 3.15.

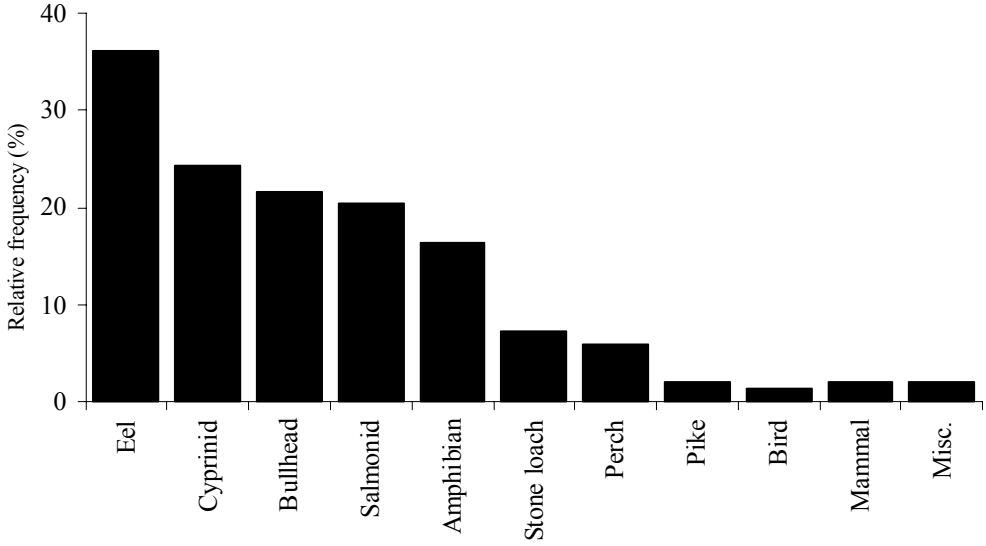

Figure 3.15 Relative abundance of prey species in otter stomach contents 1997-2003. The importance of eels as prey species is evident but the results also show that significant numbers of cyprinids and salmonids are eaten (data provided by Robert Britton).

The gut was not examined in detail for the presence of parasites but no pathology suggestive of parasitism was seen and no nematodes, cestodes or trematodes were seen.

Several otters dying from bite wounds had reddish black or haemorrhagic fluid in the stomach and blackish, thick fluid throughout the intestines which sometimes extended as far as the anus. Similar blackish fluid was seen in cubs found dead that were thought to have starved, probably having been abandoned or orphaned. However, three cubs found alive, which people had tried to hand rear, died after a few days and all had inflammation and ulceration of the stomach mucosa. In one case the ulcer had perforated. It is strongly suspected that these lesions are due to a combination of starvation and stress.

3.3.3.2 Respiratory system

The respiratory tract was the organ system in which pathological lesions were most frequently seen. The commonest lesions were multiple, whitish foci, approximately 1-2 mm diameter, scattered throughout the lungs. These lesions had been seen occasionally during the early years of the study (1988-1995) but they became more noticeable from about 1996. Histopathological examination showed that the majority were cases of adiaspiromycosis, a disease caused by the dimorphic fungus *Emmonsia* sp. This has a free-living, saprophytic, phase where the fungus grows in decaying plant material and produces small spores, 2-4 microns in diameter. If these are inhaled by a mammal they become embedded in lung tissue and develop into adiaspores, growing up to 500 microns in diameter. The life cycle is completed when the mammalian host dies and decays,

whereupon the spores are released. The condition is normally considered to be of little clinical significance but is of importance because the lesions can easily be confused with those of bovine tuberculosis (Figure 3.16).

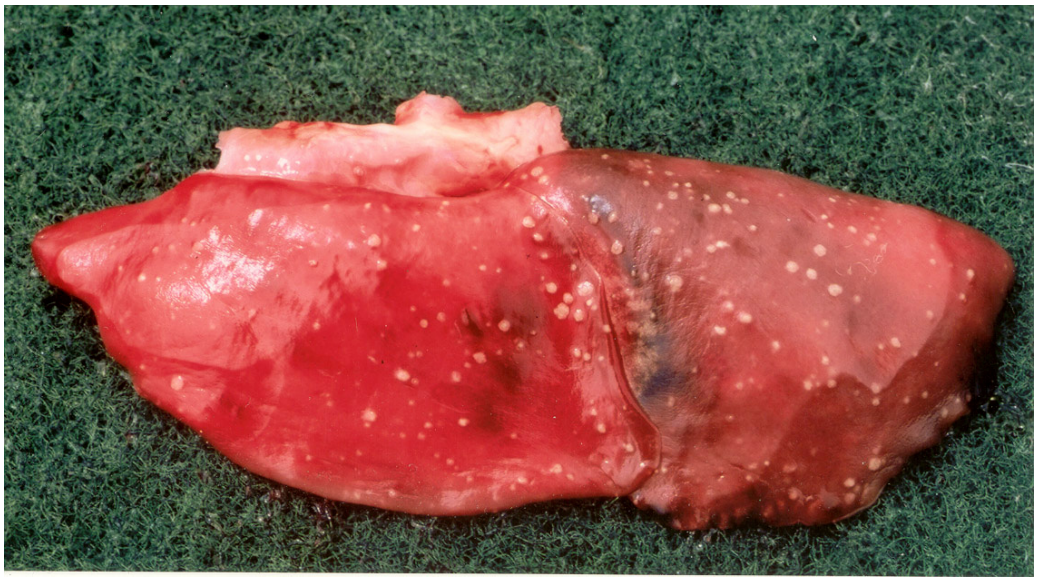

Figure 3.16 **An otter's left lung showing numerous white, focal lesions.** Although the lesions resemble those of tuberculosis, histopathological examination showed they were partially mineralised granulomata caused by fungal adiaspores.

The fungus *Emmonsia* can, however, cause disease in immunosuppressed animals, including humans. In April 1998 an immature otter was found dying on a trout farm in Hampshire. On post mortem examination there was no detectable thymus gland and the lungs were swollen, firm and emphysematous. Histopathological examination of lungs showed a severe inflammatory response to an overwhelming *Emmonsia* infection. The absence of a thymus suggests that the heavy infection could have been due to immunosuppression, especially as the concentration of PCB congener 105 in the otter's liver was much higher than the mean for otters in the south west and this congener is known to affect thymus development (Simpson and Gavier-Widen 2000).

Routine histopathological examination of lungs showed that a second type of focal lesion, similar to that caused by *Emmonsia*, has become increasingly common. These lesions are seen regularly in otters with infected bite wounds and it is thought that they are resolving microabscesses resulting from haematogenous spread of bacteria such as *Streptococcus* spp. In ten cases there was a suspicion that small, whitish foci in the lungs could have been miliary lesions of bovine tuberculosis but smears or sections stained by Ziehl-Neelsen, and specific cultures carried out by the VLA, all proved negative for mycobacteria.

Areas of pulmonary collapse and consolidation were seen occasionally, especially in otters with severe bite wounds or tooth abscesses. These were mostly attributed to haematogenous spread of bacteria but in several cases histopathological examination showed fragments of inhaled plant material within the bronchi or bronchioles. A bitch from Powys, submitted by the RSPCA, Somerset, had bronchopneumonia together with severe, unilateral, mucopurulent sinusitis associated with inhaled duckweed (Lemnaceae). It is suspected that the plant material in cases such as this may have been inhaled whilst the otters were fighting in water.

Four otters died due to severe purulent pleurisy, pneumonia and pericarditis, in each case associated with bites or septic teeth.

3.3.3.3 Cardiovascular system

One animal (M216/190802) had a fibrinous, plaque-like body measuring approximately 2 x 1 x 1 cm, adherent to the endocardial surface of the right atrium. The animal had died from pneumonia, pleurisy and pericarditis as a result of severe bite wounds. *Streptococcus canis* was isolated from purulent fluid in the thoracic cavity and it is likely that the atrial lesion was also caused by this organism. In this, and the other similar cases of pleurisy, pneumonia and pericarditis, the cardiac surface of the pericardium remained normal.

3.3.3.4 Adrenal glands

In 81 otters either one or both adrenal glands could not be examined because of trauma or autolysis. In the remaining 260 otters both glands were examined and weighed.

Most organs increase in size as an animal grows and reach maximum size at maturity. Conversely, they tend to regress in animals that are chronically ill and have lost weight. Adrenal glands, however, are unusual in that they increase in size in response to prolonged stress and therefore may be larger than normal in sick animals. This was evident in the present study, where the adrenals in otters that were sick or otherwise under stress often appeared to be enlarged.

As assessment of whether adrenals are enlarged is subjective, it is preferable to express the adrenal weight relative to size of the otter. The cube root of total adrenal weight in grams was therefore divided by overall body length in metres to give Adrenal Index (AI). The index was based on body length rather than weight because if weight were used, an underweight animal with normal size adrenals would have an erroneously high index. This problem does not occur using body length because animals do not become significantly shorter even when starving.

The AI was calculated in 233 otters. Ten per cent (n=23) had an index greater than 1.00 and in 20 per cent of cases it was greater than 0.95. All age classes were represented within the highest 10 per cent, with seven cubs, one immature, three subadult and 12 adults. Thirteen out of the 23 otters (56 per cent) with an AI greater than 1.00 had died due to disease, mostly with septic bite wounds or dental lesions (classed as 'sick'), and six had renal calculi. Within the top 20 per cent (AI>0.95), 20 otters (43 per cent) were sick and ten had renal calculi. Although most of the otters classed as 'healthy' had died of trauma, many had bite wounds, dental lesions or other conditions of varying severity. It is likely that in some of these cases the AI was also raised.

The distribution of AI was examined within age classes. In the 30 adults with the highest indices (>0.95) there were equal numbers of males and females. Six of the 15 males were sick and eight had renal calculi. Five of the 15 females were sick and two had calculi. In addition, six of the females were lactating or had recently bred. The index was calculated for 13 of the 19 cubs and two of the six males and all seven females had an index of more than 0.95. Many of these were cubs that had died during attempted hand rearing.

It was concluded that a high AI is often seen in otters dying from disease, in lactating females and in those with renal calculi. However, high indices may also occur in animals with no obvious pathology. Factors other than disease or stress that could affect adrenal size are considered in section 10.1.

3.3.3.5 Kidneys

Renal calculi are reported to be common in otters in some countries (Weber 2001) and are frequently seen in captive animals. However, despite systematic examination, none were seen during post mortem examinations of 77 otters from south west England in the period 1988-1996 (Simpson 1998). A single case occurred in 1997 and further cases were seen with increasing frequency from then on. In 2003, a third of all adults had renal calculi (Figure 3.17).

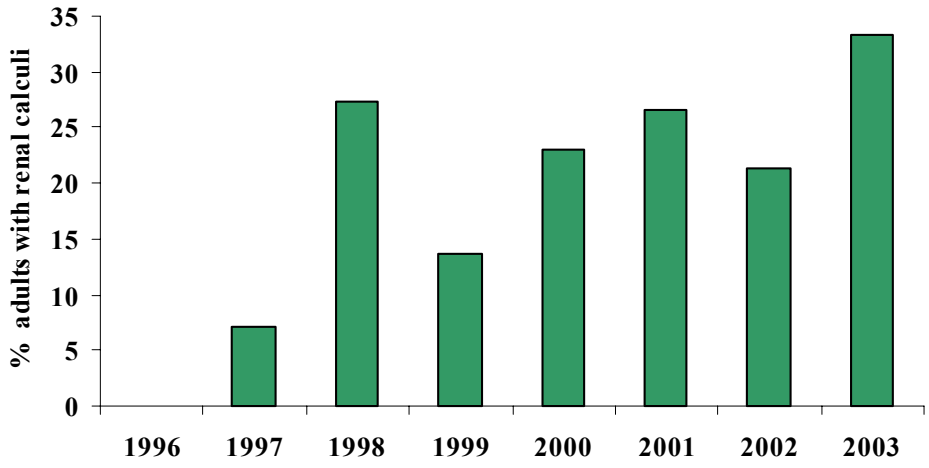

Figure 3.17 Annual prevalence of renal calculi in adult otters 1996-2003.

Two of the 33 affected otters were subadults and the rest were all adults. In most cases the calculi were small and there was minimal pathology. In two otters the calculi were associated with shrunken, fibrosed lobules and cystic dilation but even these lesions were considered to be of limited clinical significance.

Calculi were associated with large adrenals, with almost half the calculi cases (15 of 33) occurring in the 30 otters with the heaviest combined adrenal weights (>1.2 g). Adult otters with calculi had a higher median AI (0.938) than those without (0.875) and the difference between the indices in these two groups was highly significant (Student's t test, p = 0.00035).

Renal calculi were seen most frequently in animals that died as a result of fight wounds or dental abscesses (33 per cent of cases). However, although conditions such as these may have influenced the formation of calculi, it was also observed that the prevalence of calculi was highest in Dorset and Hampshire and was lowest in Cornwall (Table 3.5).

Table 3.5 Number and proportion of otters with renal calculi by county

	Cornwall	Devon	Somerset	Dorset	Hampshire
Number with calculi	5	11	8	5	3
Total adults	64	78	51	18	13
Per cent affected	7.8	14.1	15.7	27.8	23.1

3.3.3.6 Thyroid glands

No gross abnormalities of the thyroids were seen, although they varied in colour and also in size. Whilst thyroid size, and therefore weight, was closely related to body length, some animals had relatively undersized glands (Figure 3.18). Often, these were animals with severe bite wounds, dental abscesses or bacterial infections but small glands were also seen in bitches that were in late pregnancy or that had recently bred. Undersize thyroids were also seen in some apparently healthy animals.

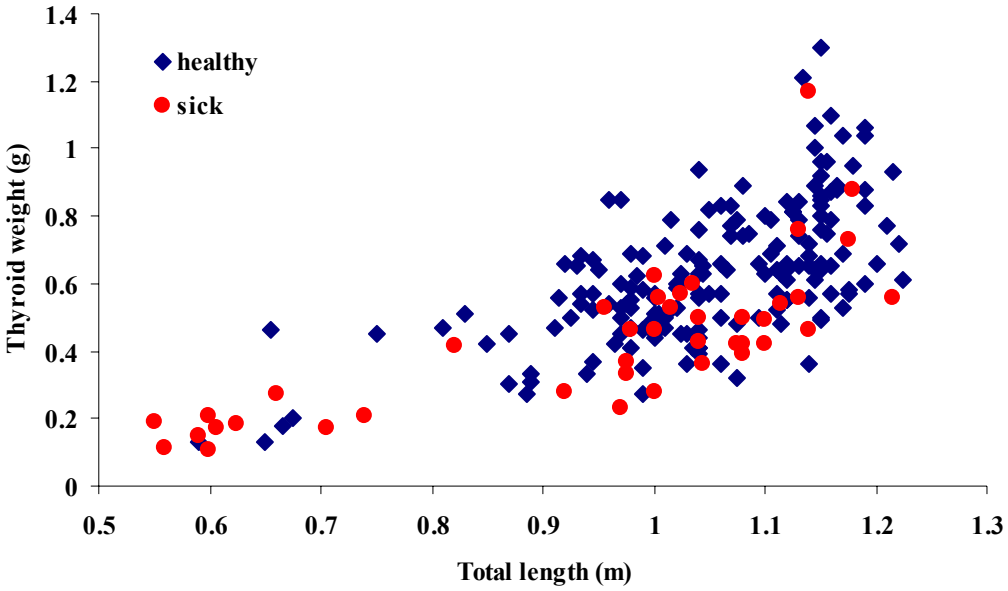

Figure 3.18 Total thyroid weight relative to body length in otters of both sexes. Relatively small thyroids were seen most frequently in adult otters. Although these were often animals that had died in debilitated condition, usually as a result of an infectious process, abnormally small thyroids were also seen in some apparently healthy otters.

In order to assess whether thyroids were regressed, the cube root of total thyroid weight (g) was divided by body length (m) to give Thyroid Index (TI) in 225 otters. The TI ranged from 0.525 to 1.178 with a median value of 0.8. In the 20 per cent of animals (n=53) with the lowest TI values, 18 (34 per cent) were sick. Mean TI values were significantly lower in sick adults, most of which had died naturally or been euthanased, compared with adults that were classed as healthy (Student's t test, $p \leq 0.0046$). Other factors that influence the size of thyroid glands are considered in Section 10.2.

3.3.3.7 Thymus gland

In 95 out of the 341 cases submitted, the thymus gland could not be examined due to severe trauma or decomposition. In 17 of the remaining 246 otters no thymus was detectable and in many others it was reduced to a vestige. This was most apparent in the 54 adult females. Of the 25 per cent (n=13) with the lowest thymus weight, seven had no visible thymus and the rest all had glands weighing less than 0.7 g. Ten of the females in this category had bred recently or were pregnant and more than half the group also had severe bite wounds.

Thymus development is at its peak in the young, growing animal but the gland regresses in later life. This is evident in Figure 3.19, where a higher proportion of cubs/immatures and subadult otters had thymus glands weighing more than 9 g compared with adults. Almost 10 per cent of adults had no visible thymus but some of the cubs/immatures and subadults also had no detectable, or very small, thymus glands. Many of these adults were sick and nearly all the cubs were ones that had died from exposure, abandonment or during attempted hand rearing. These observations are consistent with the fact that inadequate nutrition or debilitating disease will result in either poor development or atrophy of the thymus. However, exposure to pollutants, such as PCBs, will also inhibit development, particularly in the juvenile animal.

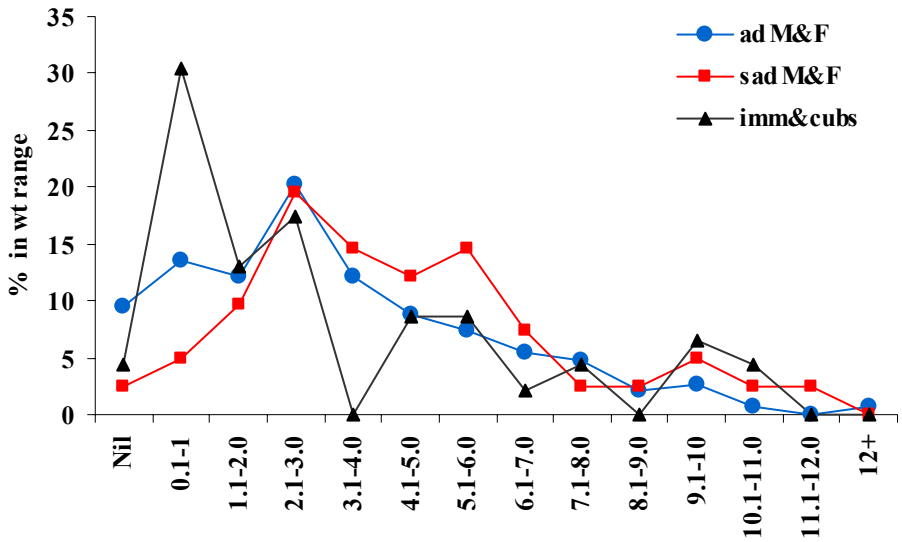

Figure 3.19 **Proportion of otters in each age class with thymus glands in a given weight range.** In 14 adults there was no detectable thymus and these were mostly sick animals in debilitated condition. Well developed thymus glands, weighing as much as 10 or 11 g were seen relatively more frequently in non-adults than adults.

3.3.3.8 Spleen

Trauma or autolysis meant that the spleen could only be examined in 226 cases. Although there was wide variation in size there were few cases where specific lesions were seen. In general spleen size was closely correlated with body length (Figure 3.20). In some animals it was enlarged, apparently due to being distended with blood, but in other cases it appeared shrunken and regressed. In those animals where the spleen was noticeably small there was often no visible thymus or it too was small.

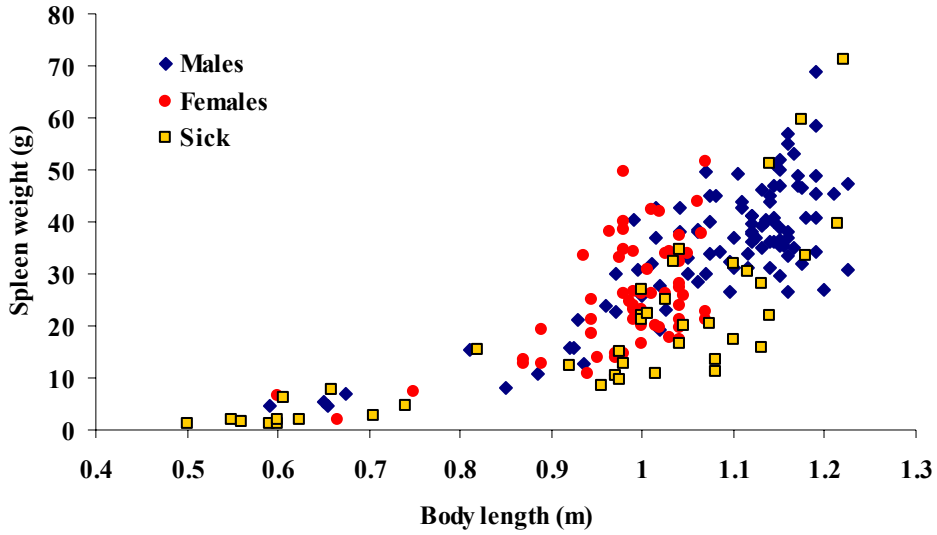

Figure 3.20 **Spleen weight relative to body length.** Yellow symbols denote otters of both sexes that died or were euthanased because they were sick, mostly due to septic wounds or dental lesions. Note that many of the sick animals in the adult size range have low spleen weights.

Small spleens were most apparent in the adult females and in 13 of the total of 54 (25 per cent) the organ weighed less than 20 g. Within this group, eight were suffering from conditions associated with septic bite wounds, one had pneumonia and pleurisy and one had leukoencephalopathy of unknown aetiology. In addition, two had recently given birth and were lactating. There was a similar, but less obvious, association with illness or stress in adult males.

Two otters, an adult male and a subadult female, had similar gross lesions in the spleen. The male was found alive in emaciated condition with multiple, severe bite wounds and blind in one eye. It was euthanased on humane grounds and post mortem examination revealed a range of pathological conditions including a well circumscribed, subcapsular lesion, approximately 12 mm in diameter, in the spleen. Within this there were several gritty, focal lesions less than 1mm diameter but the rest of the organ appeared unremarkable apart from being small. The subadult female had also been submitted alive, having been captured on a canal towpath in emaciated condition. It died and was found to be suffering from pleurisy and pericarditis associated with a fractured rib that had penetrated one lung. There was a dark subcapsular lesion approximately 15 x 7 mm in the spleen. In both cases histopathological examination showed that the subcapsular lesions were organised haematomas resulting from trauma, probably as a result of being bitten by a domestic dog.

3.3.3.9 Pancreas

Abnormalities of the pancreas were rarely seen, although examination of the gland was often difficult due to autolysis or traumatic damage. In one adult male the pancreatic lobules were separated by gelatinous oedema but this was almost certainly a secondary lesion as the animal had been found dying from septic bite wounds. Thickening of the pancreatic ducts was seen in an otter from Somerset that also had thickened bile ducts and an enlarged hepatic lymph node (see section 3.3.3.10 below).

3.3.3.10 Liver

In many otters the liver was ruptured or crushed as a result of road traffic damage. Apart from trauma, there was little evidence of hepatic pathology. Small, shrunken livers weighing only 110-130 g were seen in several otters dying from septic bite wounds. In one such case there were two yellowish, focal, areas of necrosis in a central lobe and *Pasteurella haemolytica*, and a Group D *Streptococcus* sp. were isolated in mixed culture. In a more unusual case, an adult bitch which died after being found in a weak state on a north Cornish beach, had multiple, pin-head, whitish lesions throughout the liver plus a larger yellowish necrotic lesion in a lateral lobe. Although no bite wounds were seen, the animal had multiple small lesions throughout the lungs. *Streptococcus dysgalactiae* was isolated from the lungs but *Listeria monocytogenes* and *Yersinia pseudotuberculosis* were both isolated from the liver (see section 4).

An adult male, killed in February 2000 in a road accident near Wimborne, Dorset, was suffering from bite wounds and an old fractured baculum. It also had a small, thick-walled gall bladder full of thick, creamy fluid. Similar shrunken, thickened gall bladders plus greatly enlarged hepatic lymph nodes and thickened bile ducts were seen two years later in two otters killed on roads in Somerset. Bacteriological examinations on these cases were negative. However, in 2004, when further similar cases were submitted from Somerset, the lesions were shown to be due to infection by the bile fluke, *Pseudamphistomum truncatum* (Simpson *et al*. 2005). This parasite is common in Eastern Europe and Russia but had not been recorded previously in the UK. As the characteristic gall bladder lesions were not seen in the 200 or so otters examined before 2000, it is thought that the fluke has been introduced into England in recent years.

3.3.3.11 Reproductive system

In addition to fractures of the baculum (see Figure 3.12), abnormally small or distorted baculi were seen in seven otters. One was an immature from Cornwall, one a subadult from Hampshire and the others were all adults, two each from Somerset and Devon and one from Hampshire. The latter animal (M608/271103) was well grown and weighed 8.4 kg. Apart from a small penis, it also had unusually small testes. These measured 17-18 mm long and had a combined weight of only 1.8 g. A healthy adult male's testes would measure around 21-22 mm and weigh about 4.2-5 g.

Overall there was little significant pathology of testes although in one otter (M9/4/00) the right testis was missing, apparently having been bitten off. Even in cases where the scrotum was badly bitten, the testes were normally undamaged (See section 10.3 regarding baculum development).

Out of 141 female otters received, 129 were in suitably good condition for assessment of reproductive status. Animals were placed into one of four categories based on the size and development of the uterine horns, the presence of foetuses or placental scars and the degree of mammary development, including nipple size and evidence of lactation.

Category 1 These otters had an immature or undeveloped uterus indicating that they had not yet bred. The group of 75 comprised 10 cubs, 16 immatures, 24 subadults and 25 adults. At the time of post mortem examination eighteen of the latter group were considered to be young adults.

Category 2 In these cases the uterus was mature and/or there was mammary development but no evidence of recent parturition. There were 17 cases and all were classed as adults.

Category 3 The 35 otters in this category were either pregnant or lactating and many had placental scars, indicating that they were actively breeding. All were classed as adults.

Category 4 There were two otters with unusual uteri. In one case both the uterine horns were in a 'Z' configuration and there were several, indistinct, reddish, nodular swellings, mostly at the bends in the horns. In the second case the horns were symmetrically convoluted. The uteri from these cases have been archived pending histopathological examination but it is suspected they may be early stage pregnancies.

The otters that were yet to breed (category 1) comprised over half (58 per cent) of all female submissions while those showing evidence of current or past breeding (categories 2 and 3), formed 40 per cent of submissions. Four of the 35 animals in category 3 were pregnant and 16 were lactating, confirming active breeding in 15.5 per cent of submissions. Of the 79 females classed as adult, 35 (44 per cent) were either pregnant or had bred recently and 52 (66 per cent) were judged to have bred at some time.

Seasonality of breeding

The duration of lactation in otters is uncertain but is probably about eight to ten weeks (Kruuk 1995; Stephens 1957; Wayre 1972). This figure, together with an estimate of the stage of lactation, was employed to give an approximate date of parturition in bitches that had recently bred. In bitches that were pregnant the foetal age was estimated, which made it possible to predict the month of birth. The evidence from these two sources was combined and the probable month of parturition for 20 actively breeding animals estimated (Figure 3.21). This shows that cubs were born in every month of the year except March. Although the numbers involved are too low for definite conclusions to be drawn, it does appear that more births occurred in the winter months than in the summer. However, as submissions in the summer months were very low, it means that if births had occurred in the spring they would be underrepresented.

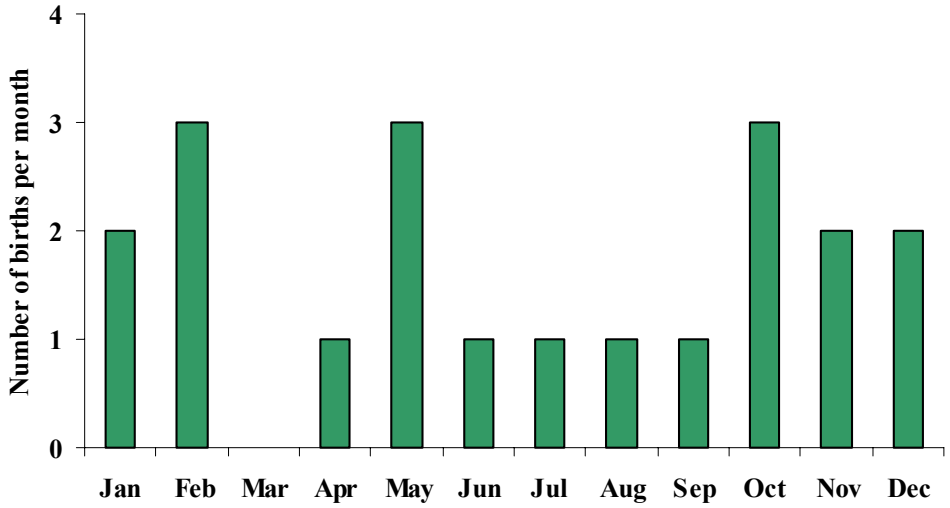

Figure 3.21 Seasonality of parturition, 1996-2003. The pattern of births per month suggests more breeding during the winter months. However, the low number of submissions during the summer means that spring births would be under recorded.

3.3.3.12 Nervous system

Gross brain lesions were seen in two cubs, both of which had marked doming of the cranium. The cerebral hemispheres appeared enlarged with flattened sulci. There was marked thinning of the cortex and distension of the ventricles by reddish fluid. In one case the cerebellum was compressed against the foramen magnum but in the other it had prolapsed through into the spinal canal. On the basis of these gross lesions both were diagnosed as cases of hydrocephalus. The cubs had been found abandoned, separately, in south west England and transferred by the RSPCA to the VWT Rehabilitation centre in Scotland. Here they had shown an increasing lack of co-ordination, and in the worst affected case, fits, before they were euthanased.

4. Bacteriology results

Tissues were cultured from 59 otters showing lesions suggestive of a bacterial infection. The majority were animals with severe bite wounds but there were also cases affected by conditions such as pneumonia, enteritis and stomach ulcers.

Bacteria were isolated from 35 cases, often in mixed culture. The commonly recovered bacteria were coliforms, *Staphylococcus* spp., *Aeromonas hydrophila*, *Corynebacterium ulcerans* and *Streptococcus* spp.. The streptococcal infections were the most significant and 14 of the 19 cases infected with streptococci were animals that had been badly bitten. The commonest were Lancefield Group C *Streptococcus* spp., mostly *Streptococcus. dysgalactiae* subsp. *equisimilis* (12 cases), followed by Group L (4) and Group G species (3).

As might be expected, the isolation rates from internal organs were much lower than from the bite wounds but the range of organisms was similar. No organisms were isolated from the heart, lungs or liver of several otters that had died as a result of septic bite wounds and from which Group C *Streptococcus* spp. were isolated. It is possible that streptococcal toxins in these cases may be significant.

Streptococcus canis (a Group G species) was isolated from three otters dying directly as a result of bite wounds. In one case the organism was recovered only from a purulent bite wound and in the second it was isolated from lung and liver. The third case was an otter that had a fibrinous plaque adherent to endocardial surface of the right atrium. The organism was not recovered from a heart blood sample but from purulent pleural adhesions.

A bitch otter found dying on a beach in Cornwall had multiple, small, focal lesions throughout the lungs and liver. *St. dysgalactiae* was isolated from the lungs but *Listeria monocytogenes* and *Yersinia pseudotuberculosis* were both isolated from the liver. *Y. pseudotuberculosis* was also recovered from the thorax of an immature female dying from pleurisy and pericarditis, although it was actually killed by a domestic dog.

Tissues from 16 otters showing lung lesions suggestive of tuberculosis were cultured for *Mycobacterium* sp. and all proved negative. In addition, during 2000 a range of tissues from 18 otters, including lung and respiratory lymph nodes, were cultured for mycobacteria irrespective of whether lesions were present and these also proved negative.

Pasteurella multocida was isolated twice, once from the lungs of a subadult that had possibly been bitten by a dog and once from the pleura of an adult with pneumonic lesions secondary to severe bite wounds. In a third case organisms resembling *Pasteurella* sp. were seen in a Gram stained smear from a bite wound.

Three otters from Somerset, one in 2000 and two in 2002, had thickened gall bladders and/or enlarged hepatic lymph nodes. Cultures of these sites and of liver, on a range of media all proved negative (see section 3.3.3.10).

Attempts to demonstrate a pathogen in stomach ulcers in cubs were unsuccessful. In addition to routine cultural examinations, samples were submitted to HPA Colindale, where they were examined for *Helicobacter* spp. using specialist media and polymerase chain reaction (PCR). Duplicate samples were also examined by PCR at Liverpool University, again with negative results (Harris 2004). *Salmonella ajoba* was recovered

from the intestines of an otter with enteritis but this was thought to be an incidental finding as the animal also had multiple septic bite wounds and streptococcal septicaemia.

Impression smears from areas of cellulitis associated with bite wounds usually showed Gram-positive cocci and often Gram-negative coccobacilli but in addition there were, in several cases, large numbers of slender, filamentous Gram-negative bacilli resembling fusiforms. These organisms were also seen in samples of pleuritic pus but were never isolated on culture.

In the period 2002-2003, samples of lung, liver, spleen and mesenteric lymph node were submitted to the VLA where they were screened for possible *Brucella* sp. infection. All samples proved negative.

Samples of 70 archived, frozen kidneys were made available in 2003 to Liverpool University Veterinary Department for use in a project to study *Leptospira* spp. in free-living wildlife. A PCR assay was employed to detect DNA extracted from the kidneys and three of the otter samples proved positive (prevalence 4.6 per cent). The DNA from one otter was sequenced and closely resembled that of *L. interrogans* serogroup canicola (Jones, 2003). This is believed to be the first time that PCR has been used to examine otter kidneys for leptospires.

5. Serology results

Blood samples collected from 85 otters during 2002-2003 were examined by enzyme-linked immunosorbent assay (ELISA) at VLA Weybridge for antibodies to *Brucella* sp. An adult male (M392/200303), killed on the road near Axminster, Devon, gave a weak positive result but the tissue samples from the case were culturally negative and there was no pathology suggestive of *Brucella* infection. An otter tested previously, from Winkleigh in Devon in 1994, had also given a positive result. The significance of these results is uncertain, although they suggest that on rare occasions otters may be exposed to *Brucella* sp. or to related bacterial species.

As it is seldom possible to obtain good quality serum samples from dead animals, tissue fluid, extracted from frozen samples of lung and skeletal muscle, can be used as an alternative. Using this approach, samples from 45 otters were screened for antibodies to canine distemper virus, canine parvovirus and canine adenovirus by Glasgow University Veterinary Department. All proved negative.

6. Histopathology results

Tissues from 95 otters were examined. During 1996-1998, as in the earlier stage of this project (Simpson 1998), most major organs were routinely examined irrespective of whether lesions were apparent. This enabled familiarity with the normal appearance of otter tissues to be established. From 1999 onwards tissues were only examined where there was evidence or suspicion of pathology.

6.1 Lungs

Lesions were seen in 61 (81 per cent) of 75 lungs examined. Some of these were minor but in over half the cases the lesions were more extensive. In 11 cases they were either the cause of, or contributed to, the animal's death.

During the first two or three years of this study alveolar macrophages often contained crystalline material, thought to be silica particles, but these became less frequent in later years. Similar particles are regularly seen in badgers' (*Meles meles*) lungs and are probably due to inhalation of soil during digging.

The predominant lesions were granulomata and in the majority of these there was a central, thick walled spore, often measuring 200 to 300 μm and staining strongly positive by periodic acid Schiff (PAS). The spore was usually surrounded by cell debris, a thick zone of epithelioid cells and macrophages and smaller numbers of eosinophils, neutrophils and multinucleate giant cells. The condition, known as adiaspiromycosis, is caused by inhalation of spores of the fungus *Emmonsia* sp. and is common in burrowing mammals. Although in most otters the lesions were considered to be of limited clinical significance, in one case (see section 3.3.3.2) the infection was severe and was directly responsible for the animal's death due to respiratory insufficiency (Simpson and Gavier-Widen 2000).

Granulomatous lesions were also seen where no fungal spore was evident and which resembled miliary lesions of tuberculosis. However, no acid-fast organisms were seen in Ziehl-Neelsen stained sections and specific cultures for *Mycobacterium* sp. proved negative. Smaller, less distinct granulomata, with a predominantly subpleural distribution, were seen with increasing frequency during the latter part of the study period. These lesions were less cellular, often containing amorphous material and with a predominance of neutrophils. They were seen mostly in otters that had suffered severe bite wounds and were thought to be resolving microabscesses resulting from haematogenous spread of bacteria, such as *Streptococcus* sp., from infected wounds. Small, focal, fibrous lesions centred around a fragment of bone or mineralised material were occasionally seen and were thought to be due to nodular ossification of granulomata.

In the cases of purulent pleurisy and pericarditis the pleura were thickened and covered with a layer of amorphous material, macrophages and numerous bacteria. The pulmonary parenchyma was intensely congested and collapsed but there was usually little evidence of pneumonia other than in tissue adjacent to the pleural surface.

Several abandoned or orphaned cubs had lesions of bronchopneumonia associated with globules of eosinophilic material in the airways. It is thought that this may have been due to inhalation of food material. Inhalation of foreign material was also thought to have contributed to purulent bronchopneumonia in several badly bitten adults. These cases had discrete areas of consolidation and necrosis of the apical lobes and within these there were fragments of plant material surrounded by mononuclear cells and neutrophils. It is

thought that the plant material may have been inhaled whilst the otters were fighting in water.

Apart from a single, slender nematode in one case there was no evidence of parasites, either in the lung tissue itself or within the pulmonary vessels.

6.2 Liver

Few of the 39 livers examined had significant lesions. The most significant was one of the three cases from Somerset (see 3.3.3.10 Liver) that had enlarged hepatic lymph nodes associated with thickened, shrunken gall bladders. The bile ducts were markedly hyperplastic, thickened, fibrosed and infiltrated with eosinophils. Most ducts contained inspissated bile and, although no parasites were seen, subsequent studies suggest that this was a case of cholecystitis caused by the bile fluke *Pseudamphistomum truncatum* (Simpson *et al.* 2005).

6.3 Kidneys

Kidneys were examined in 54 cases but most were unremarkable. Significant lesions were seen in only nine cases and these included one case of mild, focal interstitial nephritis and two with early renal calculi, one of which also had amyloid around cortical blood vessels. In four cases dilated or degenerate collecting tubules appeared to be associated with small basophilic bodies in the lumen of medullary tubules. These were of variable size and were laminated. They were also present in smaller numbers in approximately half the kidneys examined. The composition of the bodies could not be established but it is suspected they may be urate. There was no evidence that the presence of the bodies, or of calculi, was related to low vitamin A levels.

6.4 Thyroid gland

The left thyroid gland from 48 otters was examined and these showed great variation in histological appearance. Although some otters had apparently normal thyroids, in many cases the follicles appeared small and the colloid was sparse and pale staining. The lining epithelial cells were mostly cuboidal or low columnar but in one otter the cells were markedly hyperplastic, with the epithelium forming invaginations into the follicular lumen. Typically, there was variation between the lobules in follicle size and colloid content but in some otters the follicles were uniformly very small, with almost no visible lumen, and colloid was minimal or absent. However, small numbers of exceptionally large follicles were present in many glands and these contained dark, often irregular staining colloid and were lined by flattened squamous epithelial cells. Most thyroids were well vascularised and in some cases the capillary supply to the follicles was prominent.

The histological features of the glands were not quantified but in 30 otters they were considered to be within acceptable limits and in 18 cases they were considered abnormal in some respect. Sixteen glands were made available for an MSc project at the Royal Veterinary College, London in 2005. Eight of these were from otters with high OC burdens (median dieldrin concentration 451μg/kg) and eight from a low burden group (median dieldrin concentration 16.8μg/kg). The study found no statistically significant differences in thyroid structure when assessed by quantitative and semiquantitative histological techniques (Millins *et al.* 2005). However, the study was severely constrained by the small sample size.

6.5 Adrenal gland

In many otters the adrenal glands appeared enlarged and/or had an irregular nodular surface. The left adrenal from 49 otters was examined and in only six was the morphology considered to be within normal limits. Various degrees of nodular hyperplasia of the cortex were common and in eight otters the changes were severe. There was hyperplasia of the cells forming the zona glomerulosa – the outer layer of the cortex – to form large nodular lesions. In the most advanced cases the zona glomerulosa cells appeared metaplastic. These were partially or totally enclosed in pockets of fibrous tissue derived from the adrenal capsule. In some cases these fibrous divisions extended deep into the cortex. Within the fibrous tissue there were normally focal accumulations of lymphocytes. The normal columnar arrangement of the middle layer - the zona fasciculata – was frequently unrecognisable and the cells were small, pale and without their normal lipid droplets. There was often a zone of haemorrhage between the inner cortical layer - the zona reticularis - and the medulla. Lesions seen in the medulla were difficult to determine but included focal mineralisation and necrosis.

Of the eight otters with the most obvious cortical lesions, five were adult males, one was an adult female and two were subadults, one male and one female. The adult female had recently bred (it had placental scars), one male had renal calculi, one had dental lesions and three had minor bite wounds. However, all of them were road traffic casualties with high condition indices and, as a group, they were not obviously sick animals. The median pp'DDE and dieldrin levels were high (464 and 193 μg/kg respectively) but not significantly different from the median values of adult males (see section 8).

6.6 Eyes

The eyes of 62 otters collected between April 1996 and the end of 1999 were examined as part of a larger histopathological study of otters from southern and south-west England. A number of pathological conditions were seen but the most significant was retinal dysplasia in almost 30 per cent of the otters. This is a developmental abnormality occurring early in life and characterised by the formation of folds and rosettes of the retina. Vitamin A deficiency is a well recognised cause of retinal dysplasia and the vitamin A levels in the otters with lesions were significantly lower than in unaffected otters. The halogenated hydrocarbons are known to adversely affect vitamin A metabolism and it was observed that the mean hepatic concentration of dieldrin in otters with dysplastic lesions was over three times higher than in those without lesions (Williams *et al*. 2004).

6.7 Other organs

Other organs examined were footpad, thymus, spleen, heart, stomach, pancreas, testes, uterus, mammary gland, salivary gland, anal gland, urinary bladder and brain. Although a variety of pathological lesions were seen in these organs, none were sufficiently distinctive to be indicative of a specific condition. In most cases no significant changes were seen. Constraints of time and space mean that it is not practical to detail the findings in all these organ systems in this report.

7. Dental age determination

Whilst physical characteristics, such as body length and mass, can help when making an assessment of an otter's age, it is clearly preferable to determine age more precisely. In many species this can be achieved by removing a tooth, cutting histological sections and counting the annular rings in the cementum layer. Unfortunately, it appears that this method does not work particularly well with otters, especially when applied to incisor teeth. Age estimation was attempted on archived teeth by two laboratories using different methods.

7.1 Exercise 1

The first batch of 31 teeth, collected up to September 1997, was examined at VLA Lasswade using wax embedded sections. Unfortunately, the results were judged to be unreliable as, in some cases, the recorded dental age was clearly not compatible with the animal's physical characteristics.

7.2 Exercise 2

Following an approach by York University in Spring 2003, archived incisor teeth were made available for another attempt at age determination. An initial batch of 40 formalin fixed teeth was submitted, comprising between one and four teeth per otter. The samples were given random identifying numbers and, although the researcher was aware that there were replicates in the set, they had no means of knowing which otter the samples came from. The teeth were decalcified, sectioned on a freezing microtome and stained. The researcher counted the maximum number of cementum lines per tooth and, in addition, the number seen most frequently. The results of this initial trial were encouraging and therefore a second batch of 60 teeth, with duplicates from five otters, was submitted in December 2003 and a third batch of 118 (11 duplicates) in January 2004. Although the results of the second batch appeared generally satisfactory, there were several cases where the dental age was not compatible with the estimated age class at the time of post mortem examination. Regrettably, constraints on the researcher's time meant that only 40 samples - and none of the duplicate pairs - in batch 3 were examined.

This was the most rigorous exercise to date on teeth collected by the author and it is difficult to see how the procedure could have been improved. However, there remain concerns about the validity of the results in all cases and therefore it is proposed to have unused duplicate teeth examined by another laboratory in the near future.

8. Toxicology results

Liver samples from 271 otters, collected between April 1996 and the end of 2003, were submitted to the Environment Agency laboratories where they were analysed in seven batches. Determinands included organochlorine pesticides and their metabolites, industrial organochlorines and polychlorinated biphenyls (PCBs). The analytical values for all determinands in this section are expressed in micrograms per kilogram of liver on a wet weight (μg/kg ww) basis. The PCB congeners are referred to by their IUPAC (International Union of Pure and Applied Chemistry) numbers (Ballschmiter and Zell, 1980).

Pollutant levels in the otters were examined for evidence of change over time and for differences between males and females within and between age classes. As the numbers of results for subadult and immature otters were small they were combined to form a non-adult set for comparison with adults.

8.1 Organochlorine compounds

The analytical results showed variation in the consistency and sensitivity of detection limits for some organochlorine compounds. Typically detection limits were higher in batches analysed in earlier years. As it is only worth examining differences in concentrations between ages, sexes and years for those compounds with substantial numbers of detected values, an initial screening of the data was carried out. This was restricted to a subset of 211 otters that were analysed in batches that had consistent detection limits. The number of samples in which compounds were either detected or not detected is shown in Table 8.1. The concentrations of some organochlorine compounds were consistently below the detection limit and therefore further analysis of the whole data set was confined to pp'DDE, dieldrin, hexachlorobenzene (HCB) and TDE .

Table 8.1 Organochlorine compounds detected and maximum values in 211 otters.

Compound	Number of detecteds	Number of non detecteds	Total otters analysed for compound	Max value μg/kg ww
Aldrin	0	211	211	0
Dieldrin	211	0	211	1080
Endrin	2	209	211	2.06
op'DDE	1	210	211	1.48
pp'DDE	210	1	211	12,400
pp'TDE	130	80	210	530
op'DDT	2	209	211	2.02
pp'DDT	9	202	211	16.8
alpha HCH	24	187	211	6.03
beta HCH	37	165	202	62.5
delta HCH	2	209	211	33.4
gamma HCH	9	202	211	11.6
HCB	176	24	200	190
Isodrin	70	141	211	52.1

8.1.1 pp'DDE

The extent to which DDE varied with sex and age class and between years was analysed using a general linear model (GLM) in which rank scores were assigned to liver DDE concentration and year, and year was included in the model as a covariate. Data were ranked so that the underlying assumptions of the statistical model were met. An interaction term between age and sex was also included in the model.

Hepatic DDE concentrations in the otters declined significantly between 1996 and 2003 ($F_{1,243}$=20.85, p<0.001; Figures 8.1, 8.2 and 8.3). The other major factor explaining variation in liver DDE was the interaction between age and sex ($F_{1,243}$=12.06, p=0.001). This indicates that differences between males and females in liver DDE concentrations varied with age class.

Adult male otters typically had the highest liver DDE concentrations. Residues in adult males were greater than those in adult females (Figure 8.1) and non-adult males (Figure 8.2). In contrast, there was little or no difference in DDE levels between non-adult males and females (Figure 8.3). DDE concentrations in non-adult females were typically similar to, or higher than, those in adult females.

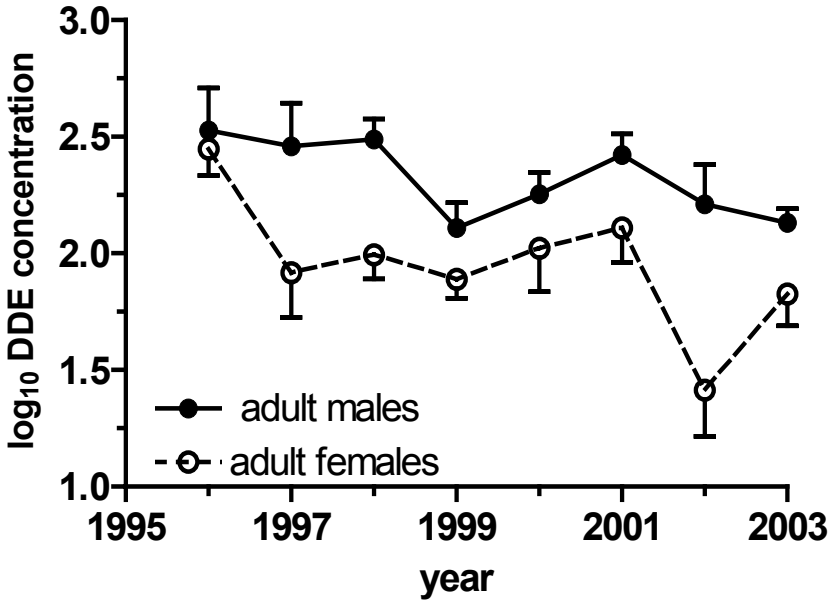

Figure 8.1 **Annual mean log_{10} hepatic pp'DDE values in male and female adult otters.** DDE levels were highest in adult males but declined significantly in both sexes over the study period.

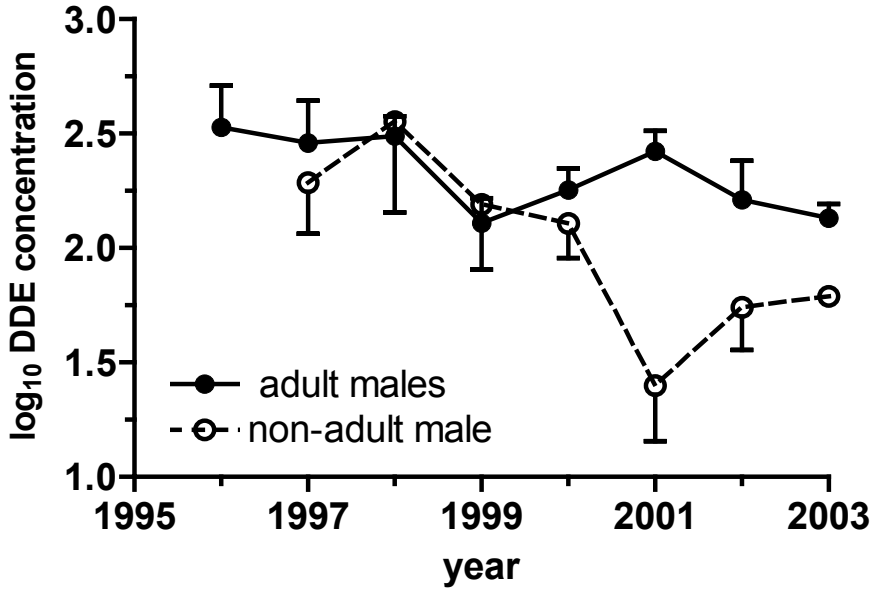

Figure 8.2 **Annual mean log_{10} hepatic pp'DDE values in adult and non-adult male otters.** The higher level of DDE in adult males compared with non-adult males indicates accumulation with age, despite a significant decline over time.

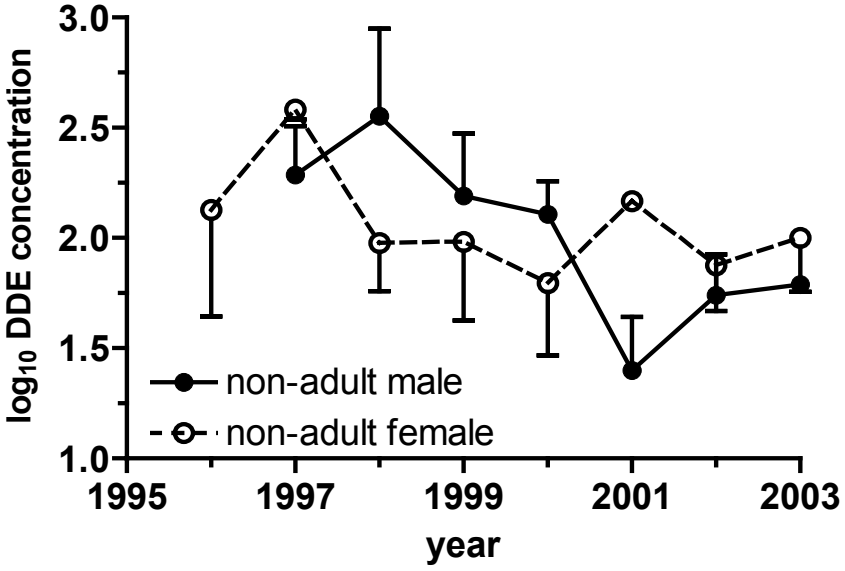

Figure 8.3 **Annual mean log_{10} hepatic pp'DDE values in male and female non-adult otters.** There was little difference in DDE levels in male and female non-adults but levels within the age class did fall over time.

8.1.2 Dieldrin (HEOD)

As for DDE, a GLM analysis was carried out on the data. In this case, it was possible to analyse unranked data for both adults and non-adults together without violating the basic assumptions of the statistical model. Year was included as a covariate and the model again included an interaction term between age and sex, as this had been found to be important in explaining variation in liver DDE levels.

There was a highly significant decline in liver HEOD concentrations over time ($F_{1,243}$=17.3, p<0.001). On average, both adult and non-adult males had higher liver HEOD concentrations than females in their respective age classes (age term: $F_{1,243}$=5.51, p=0.02; Figures 8.4 and 8.5). The difference in liver HEOD concentrations between adults and non-adults was more marked in males (adults had higher concentrations) than females (similar mean concentrations in both age classes). However, neither the age term nor the sex*age interaction terms in the statistical model quite achieved significance (age: $F_{1,243}$=2.04, p=0.155; age*sex: $F_{1,243}$=2.37, p=0.125).

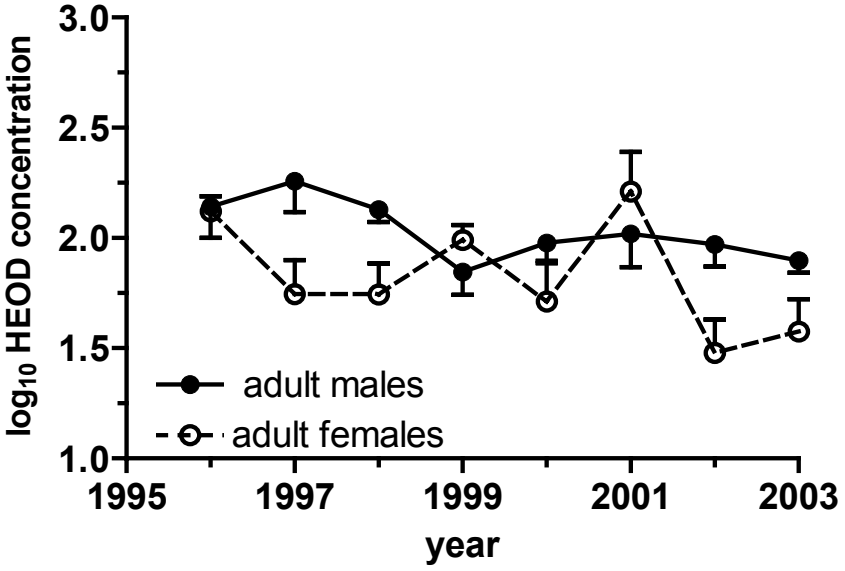

Figure 8.4 **Annual mean log_{10} hepatic dieldrin (HEOD) values in male and female adult otters.** The dieldrin levels in adult males were, on average, significantly higher than in adult females. Levels in both sexes declined over time.

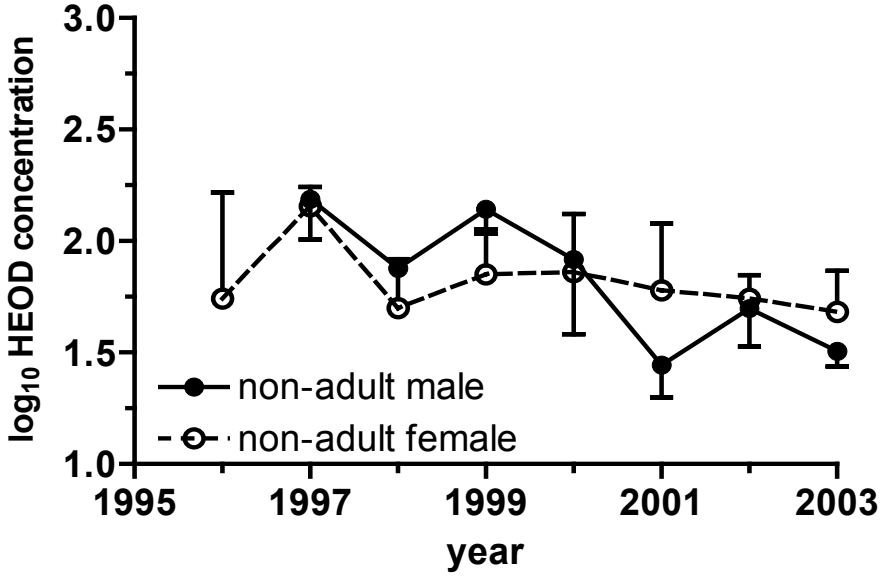

Figure 8.5 **Annual mean log_{10} hepatic dieldrin (HEOD) values in male and female non-adult otters.** Non-adult males had higher dieldrin levels than non-adult females and levels in both groups declined over time.

8.1.3 Correlation between DDE and dieldrin

Previous studies in many species have shown that animals which have a high concentration of one halogenated hydrocarbon tend also to have high concentrations of related compounds. Log_{10} DDE levels in the adults, non-adults and cubs in this study were therefore plotted against log_{10} dieldrin levels. The correlation between DDE and dieldrin was highly significant and this was evident in all age classes (Table 8.2, Figure 8.6).

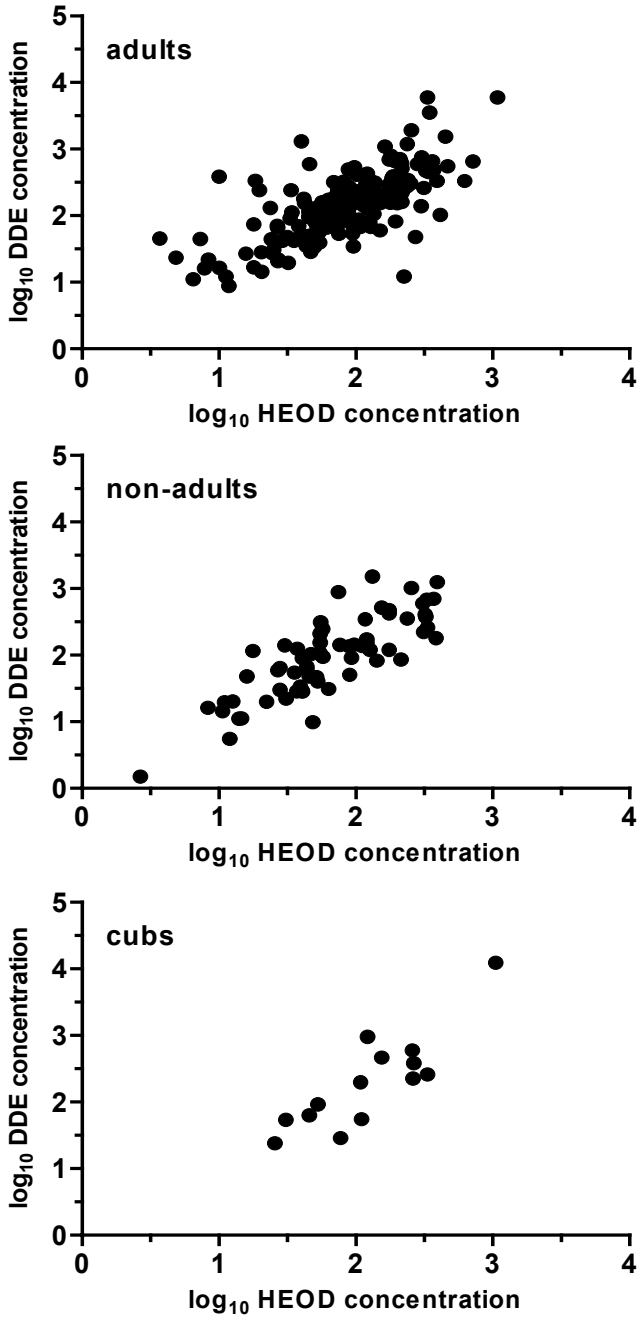

Figure 8.6 Correlation between DDE and dieldrin (HEOD) levels in otters' livers in three age classes.

Table 8.2 Statistical analysis of correlation between DDE and dieldrin levels.

	Adults	Non-adults	Cubs
Number pairs	183	66	14
Pearson r	0.6842	0.8231	0.8478
95 per cent confidence interval	0.5985 to 0.7544	0.7256 to 0.8882	0.5764 to 0.9507
p value (2-tailed)	<0.0001	<0.0001	0.0001

8.1.4 HCB

Liver HCB residues were analysed using GLM and ranked values for HCB and year, with year included in the model as a covariate. Data for adults and non-adults were analysed separately as the assumptions of the model were violated when data for all otters were analysed together. HCB residues in adults showed no consistent upward or downward trend over time ($F_{1,173}$=0.33, p>0.05). However, as observed with DDE and dieldrin, there was a pronounced effect of sex, with higher residues in males than females ($F_{(1,173}$=9.96, p=0.002) (Figure 8.7). By contrast, analysis of data for non-adults showed no significant change over time and no detectable differences between the sexes (Figure 8.8).

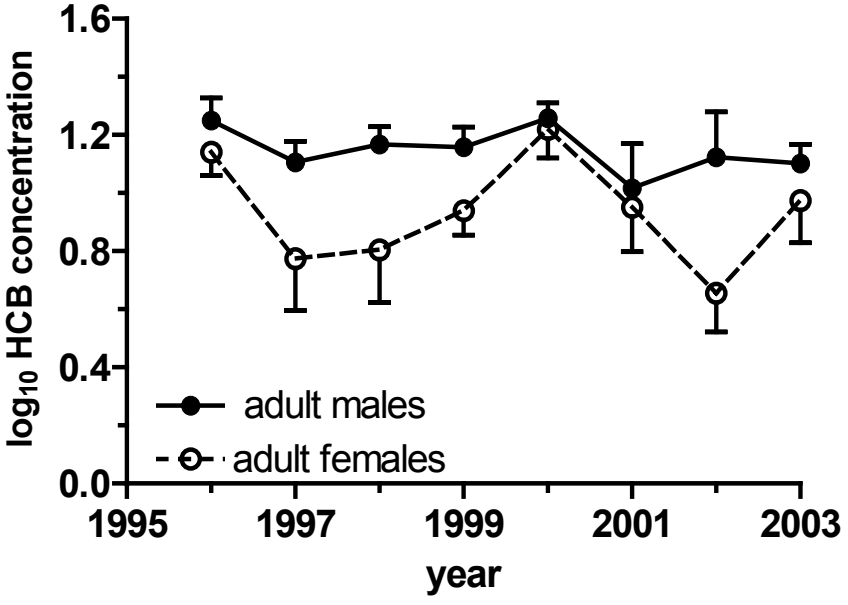

Figure 8.7 Annual mean log_{10} hepatic HCB values in male and female adult otters. Residues in adult males were significantly higher than in adult females. However, there was no trend over time.

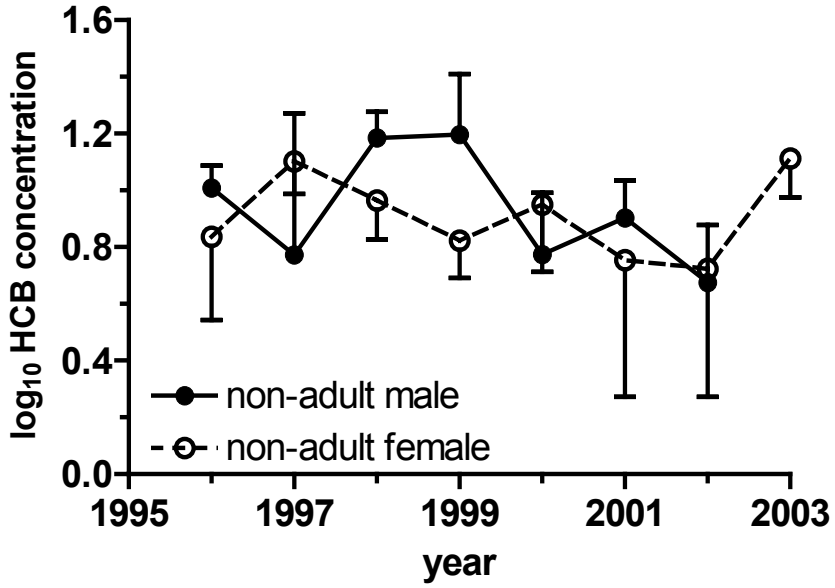

Figure 8.8 **Annual mean log_{10} hepatic HCB values in male and female non-adult otters.** There was no significant difference in HCB levels between the sexes and no evidence of change over time.

As there was no significant variation between years in liver HCB residues, data for otters that died in different years were pooled so that the significance of potential age-related differences could be determined using larger sample sizes. HCB residues in adult and non-adults were compared by student t test using log-transformed data. Data for males and females were analysed separately. In males, adults had significantly greater hepatic HCB residues than non-adults (t_{144}=2.75, p=0.007) but there was no significant difference in HCB liver residues between adult and non-adult females (t_{94}=0.17, p >0.05).

8.1.5 TDE

TDE concentrations were lower overall than those of DDE and there were more non-detected values in the dataset. In order to conform to the assumptions of the statistical model, log transformed TDE liver residue data for adults and non-adults were analysed separately. Year was included as a covariate in the statistical models. In adults, there was a highly significant decline in TDE levels over time, ($F_{1,178}$=20.82, p<0.001) and liver TDE concentrations were on average higher in males than females ($F_{1,178}$=1.92, p<0.05) (Figure 8.9). Liver TDE concentrations also declined significantly over time in non-adults ($F_{1,63}$=23.05, p<0.001) but did not differ between males and females ($F_{1,63}$=1.92, p>0.05) (Figure 8.10).

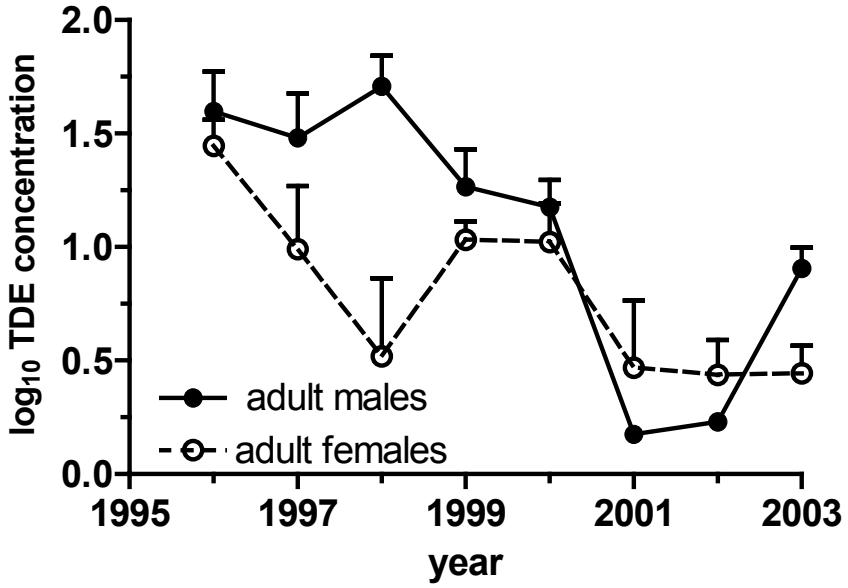

Figure 8.9 **Mean annual log_{10} TDE levels in adult otters.** The TDE levels in adult otters declined significantly over time. Levels in adult females were significantly lower than in adult males.

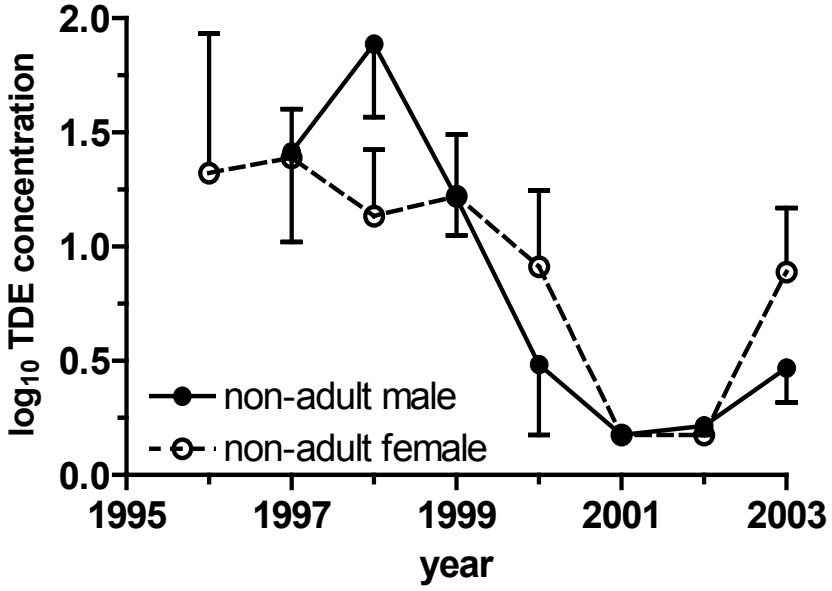

Figure 8.10 **Mean annual log_{10} TDE levels in non-adult otters.** TDE levels in non-adults of both sexes declined over time but residues in males and females were similar.

8.2 PCBs

The first three batches of livers were analysed for ten PCB congeners: 28, 31, 52, 101, 105, 118, 138, 153, 156, 180. From June 2003 onward, analysis was modified to include 13 more congeners: 8, 18, 20, 35, 44, 66, 77, 126, 128, 149, 169, 170, 187. The variation in the limits of detection in the earlier batches meant that the data from those batches had to be excluded from the analysis. Data from cubs were also excluded, giving a dataset for 209 otters. Most of the animals in this dataset died between 1998 and 2003

8.2.1 Congener composition

The sum of the individual congener concentrations in each animal was calculated to give a congener sum total PCB concentration. The overall congener composition of this total is shown in Figure 8.11 where the contribution of each congener is expressed as the median percentage of the total. The predominant congeners in descending order were 153, 138, 187, 180, 170, 118, 128, 156 and 105. The other congeners (8, 18, 20, 28, 31, 35, 44, 52, 66, 77,101, 126, 149, 169) had median percentage values of zero.

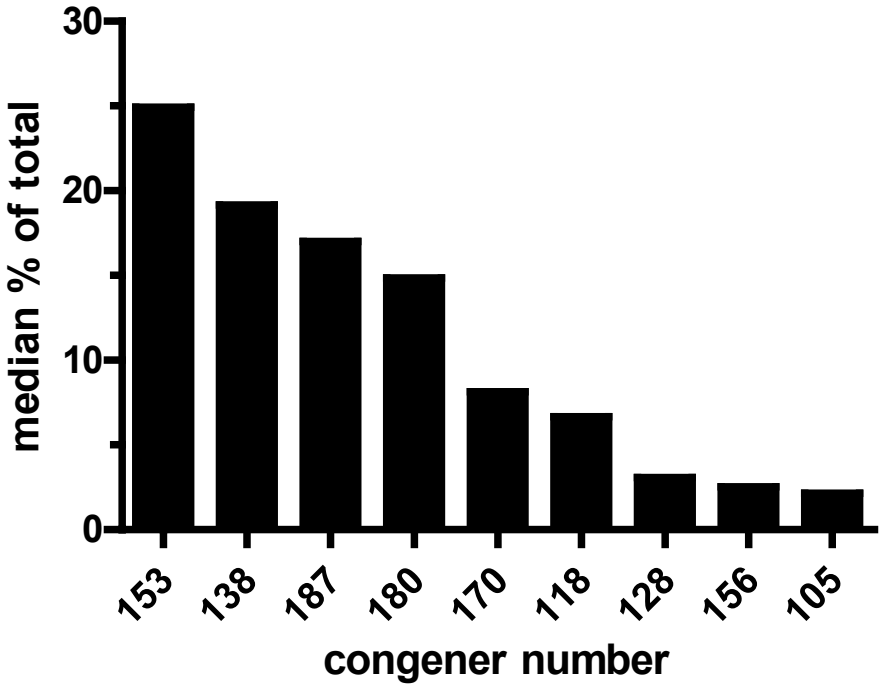

Figure 8.11 Congener composition of the sum total PCB concentration.
The 14 congeners not shown had median per cent values of zero.

The effects of year, age and sex on liver sum PCB concentrations were analysed using a GLM where the data were log-transformed and year was included as a covariate. There was no significant change in liver PCBs over time and no overall consistent effect of sex ($F_{1,193} \leq 0.46$, $p>0.05$ in both cases) but the terms in the model for age and for age*sex interaction were both significant ($F_{1,193}=11.63$, $p=0.001$, and $F_{1,193}=6.59$, $p=0.011$, respectively). Age was a significant term in the model because liver PCB

concentrations were consistently higher in adults than non-adults for both males and females. Although this difference is not obvious when data are summarised by year (Figures 8.12 and 8.13), it is clear when median values are plotted for sex and age class (Figure 8.14). The age*sex interaction term was significant because liver PCB concentrations varied between males and females, but the nature of this variation differed between adults and non-adults. In adult otters, males had greater liver PCB residues than females (Figure 8.15) but in non-adults, females usually had higher liver PCB concentrations than males (Figure 8.16).

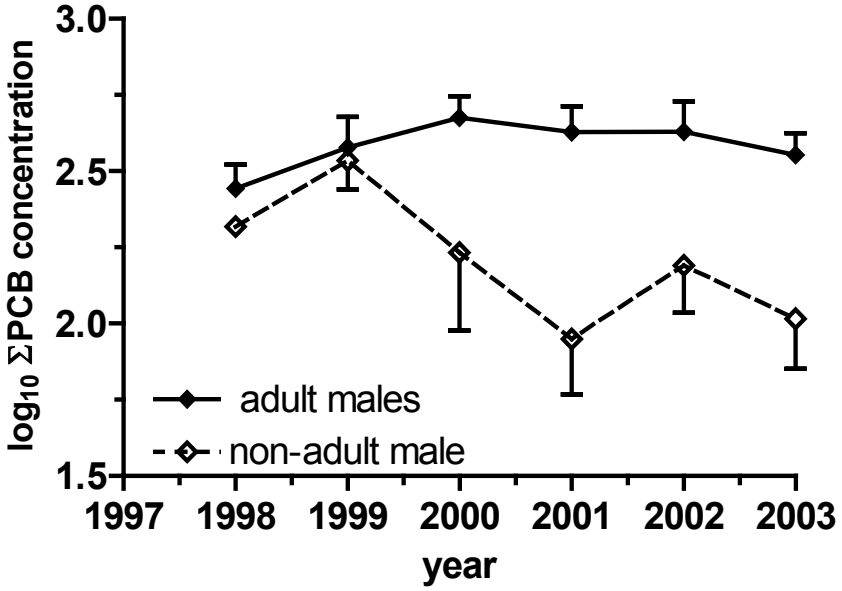

Figure 8.12 Hepatic log $_{10}$ sum PCB congener levels in adult and non-adult male otters. Sum PCB levels in adult male otters were significantly higher than in non-adults.

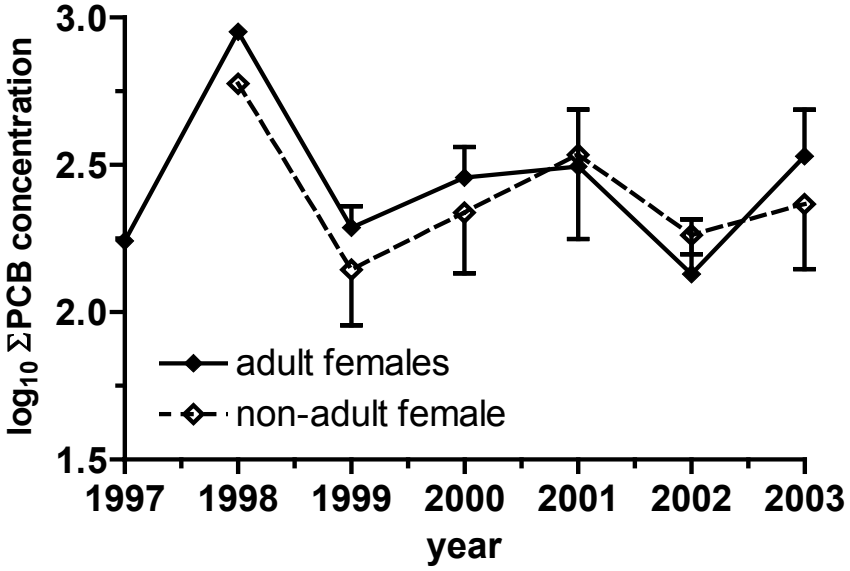

Figure 8.13 Hepatic log $_{10}$ sum PCB congener levels in adult and non-adult female otters. Sum PCB levels in adult female otters were higher than in non-adult females, although this is not apparent in the figure.

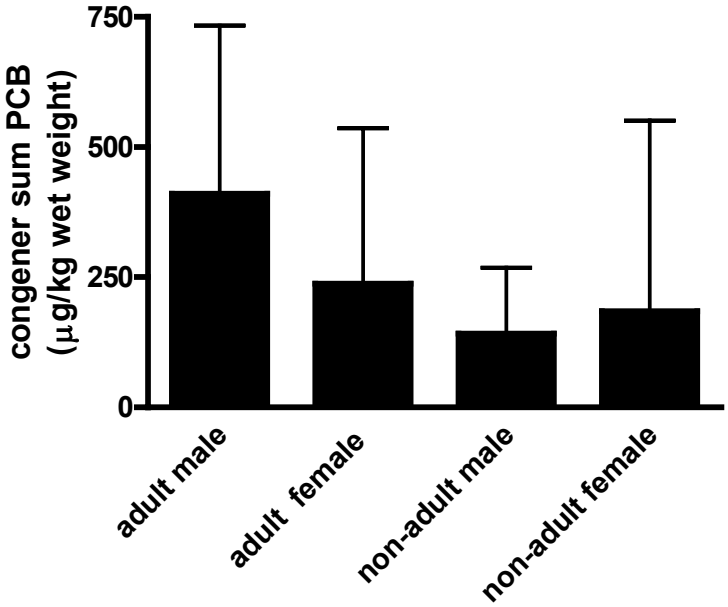

Figure 8.14 Median sum PCB congener values by sex and age class with interquartile ranges. Note that non-adult females have higher PCB levels than non-adult males.

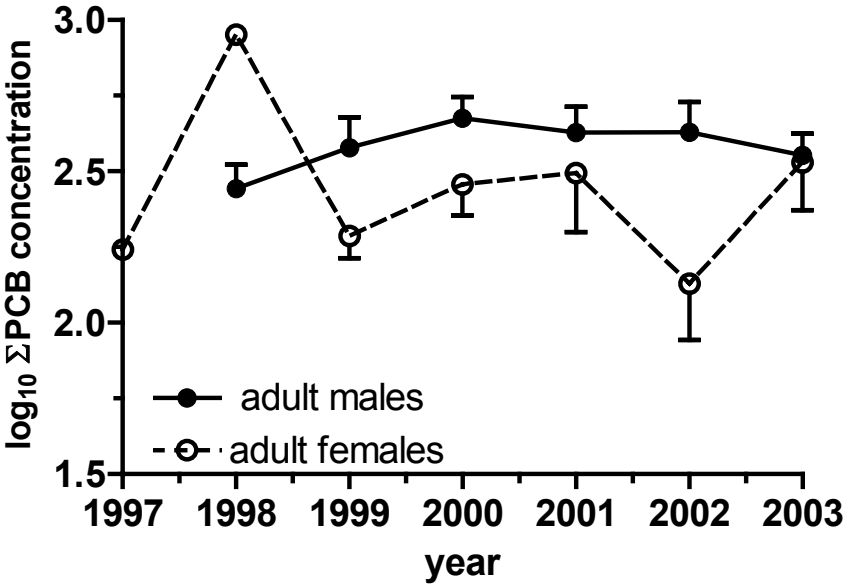

Figure 8.15 **Hepatic log $_{10}$ sum PCB congener levels in male and female adult otters.** Sum PCBs levels were higher in adult males compared to females but there was no significant change over time in either sex.

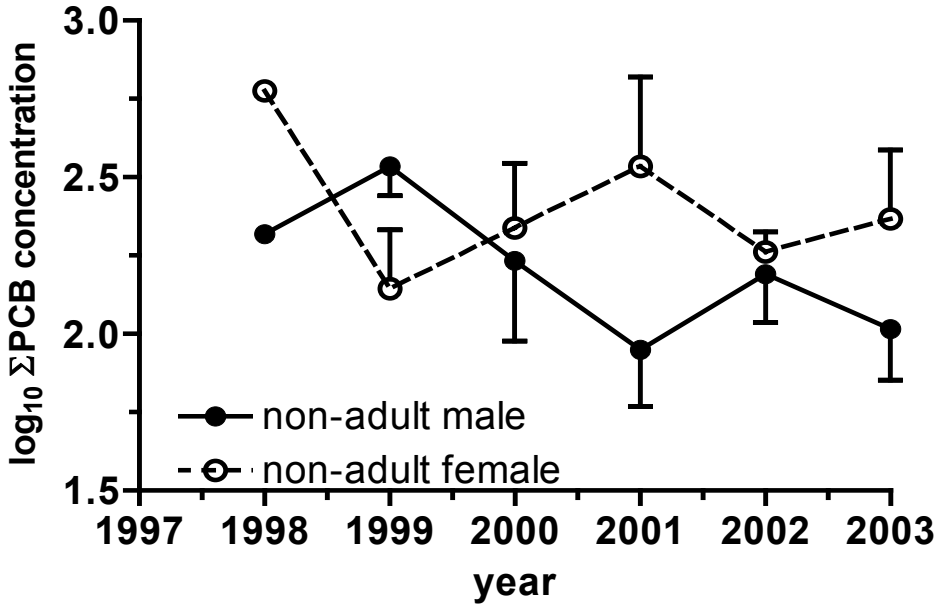

Figure 8.16 **Hepatic log $_{10}$ sum PCB congener levels in male and female non-adult otters.** On average, sum PCB levels in non-adult females were higher than in males but there was no significant change in non-adults of either sex over time.

8.2.2 PCB congener 153

Otter livers from south west England examined over the period 1988-96 had shown small but significant declines in the concentration of several congeners, including PCB153, and that levels were lower in females than males (Simpson 1998). Data for the 1996-2003 period were therefore examined using a GLM for evidence of a continued downward trend in congener 153. Concentration data for PCB 153 was log transformed and year was included in the model as a covariate.

The pattern of results for PCB 153 closely follows those for sum PCB congeners, with no demonstrable decline over time but with much higher levels in adults compared with non-adults ($F_{1,193}$=11.89, p=0.001). Again, although there was no consistent effect of sex across adults and non-adults, residues in adults were higher in males than females whereas in non-adults residues were higher in females than males (age*sex interaction term: $F_{1,193}$=7.46, p=0.007). These results are not shown graphically as they are essentially the same as for sum PCB congeners.

8.2.3 PCBs and toxic equivalents

There is a wide spectrum of toxicity within the 209 theoretically possible PCB congeners but the dioxin-like congeners are widely regarded as the most toxic. These compounds exert their effect through the same mechanism as the highly toxic tetrachlorodibenzodioxin (TCDD) and therefore each has been given a toxicity rating relative to that of TCDD. It is referred to as the Toxic Equivalent Factor (TEF). The concentration of each dioxin-like PCB congener can be multiplied by the TEF for that congener to give a congener-specific Toxic Equivalent (TEQ) value; the various TEQ values for different dioxin-like congeners are then summed to give the sum TEQ in the tissue due to PCBs (Kannan *et al.* 1988; Safe 1990). TEFs have only been established for a limited number of congeners and do not exist for many of the most abundant congeners.

Sum TEQs were calculated for 209 otters, most of which died between 1998 and 2003. The data came from analytical batches that all had the same limits of detection. The congeners for which TEFs were available were 77, 105, 118, 126, 156 and 169. The TEQ values for adults and non-adults were assigned ranked scores and used in a GLM with year included as a covariate. There was no overall consistent effect of year or sex (summary statistics for both year and sex: $F_{1,192} \leq 0.58$, p>0.05 in both cases) but the terms in the model for age and for age*sex interaction were significant or close to significance (age: $F_{1,192}$=5.91, p = 0.016; age*sex interaction: $F_{1,192}$=3.11, p=0.08). Age was a significant term in the models because TEQ concentrations were consistently higher in adults than non-adults for both males and females. An interaction between age and sex was evident because TEQs tended to be higher in adult males than in adult females (Figure 8.19) but higher in non-adult females than in non-adult males (Figure 8.20). As was seen with sum PCB levels, the difference between age groups or sex was not apparent when data were summarised by year.

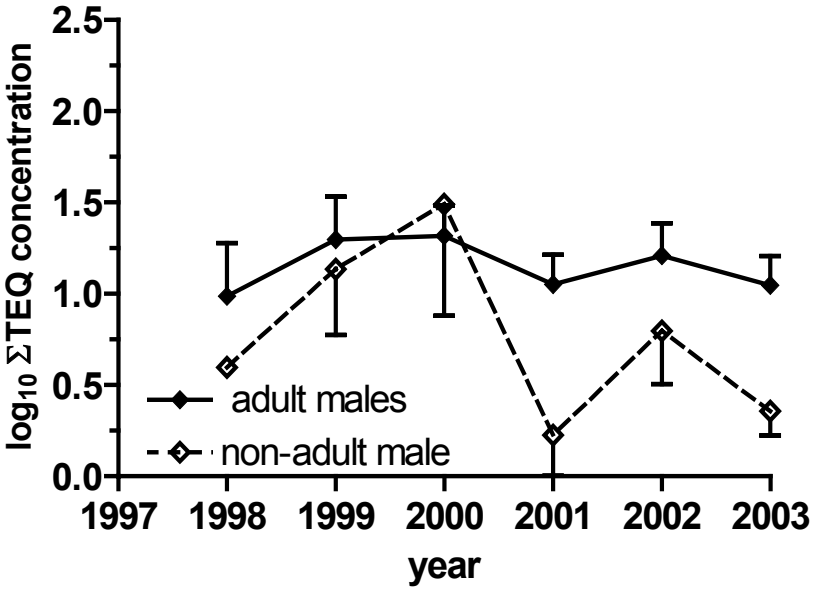

Figure 8.17 **Hepatic log $_{10}$ sum TEQ levels in male adult and non-adult otters.** Higher TEQs in adult males compared to non-adults suggest PCB accumulation with age.

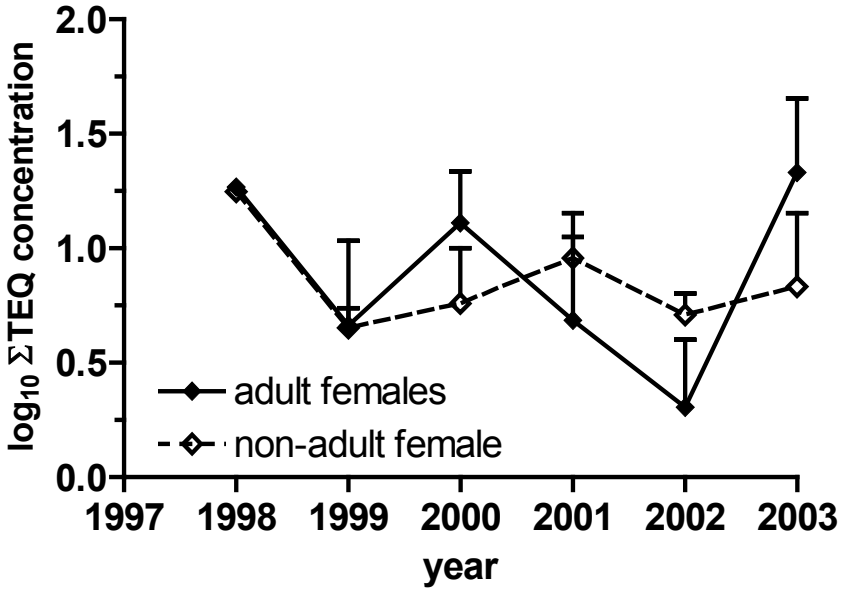

Figure 8.18 **Hepatic log $_{10}$ sum TEQ levels in adult and non-adult female otters.** Adult females had higher TEQs than non-adults although this is not apparent in this graph.

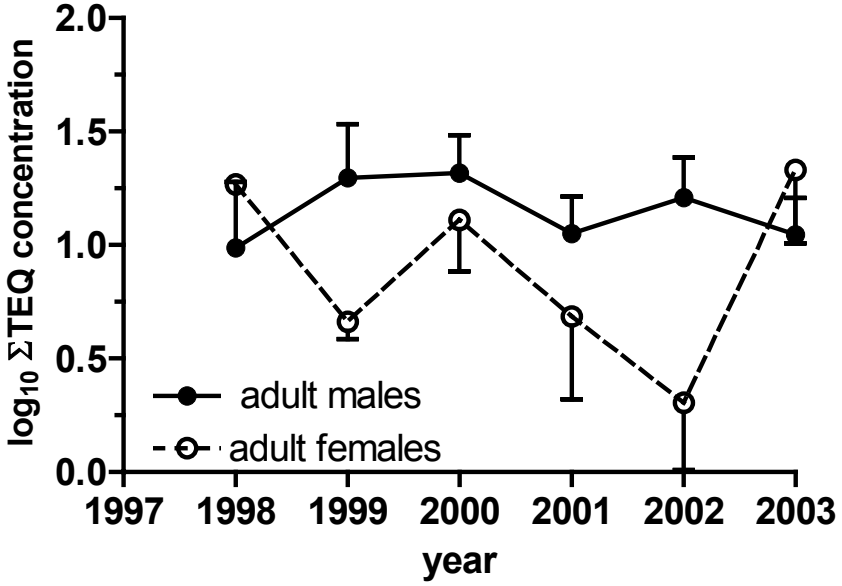

Figure 8.19 Hepatic log $_{10}$ sum TEQ levels in male and female adult otters. The higher TEQs in adult males compared with adult females mirrors the pattern seen with sum PCBs.

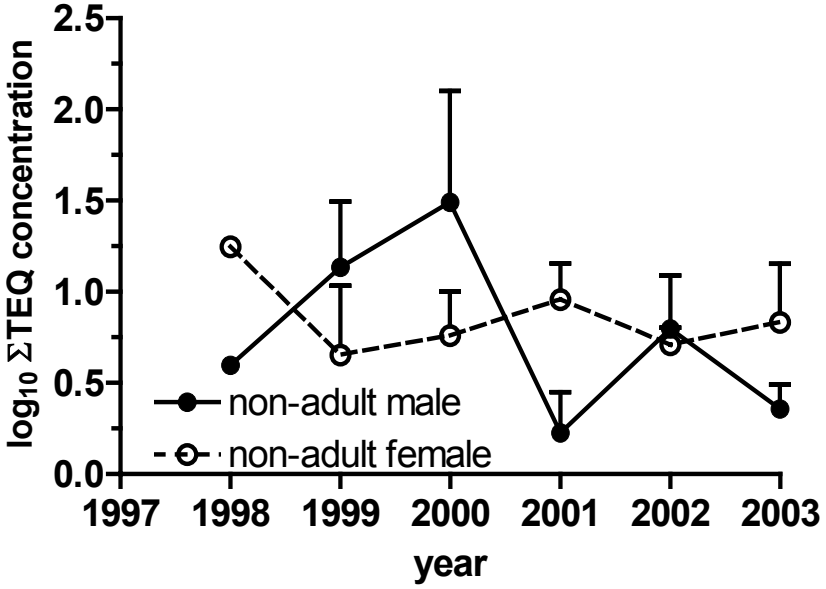

Figure 8.20 Hepatic log $_{10}$ sum TEQ levels in male and female non-adult otters. Overall, TEQs were higher in non-adult females than in males, again resembling the situation with sum PCBs.

9. Biochemistry results

9.1 Vitamin A analysis

Livers were analysed for vitamin A in 100 out of the 181 otters submitted between April 1996 and the end of 2000. The values ranged from nil detectable in five otters to one at 2684 μmol/kg. Almost half the otters had vitamin A values less than 200 μmol/kg, which is regarded as the threshold for normal domestic livestock (Blood *et al.* 1983), and 14 were below the quoted critical level of 7 μmol/kg.

There was a markedly skewed distribution, with 39 per cent of otters having values of less than 100 μmol/kg and a second, smaller, peak at around 400-500 μmol/kg (Figure 9.1).

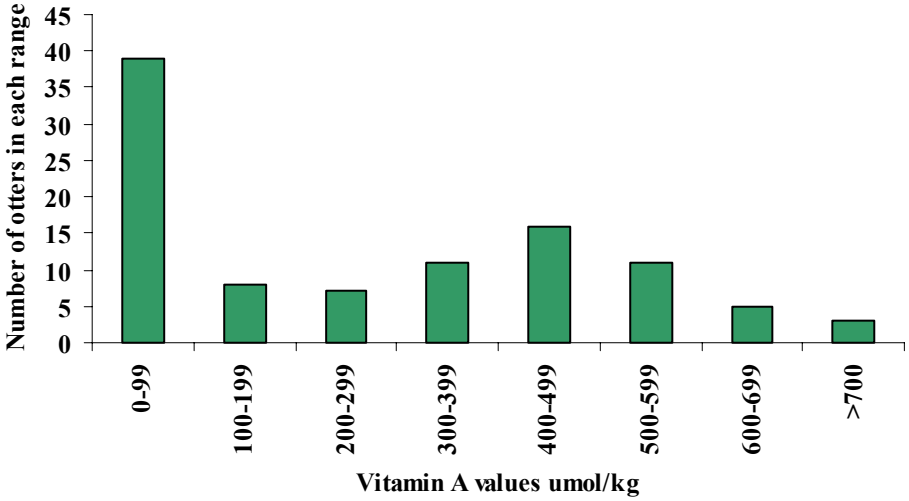

Figure 9.1 **Distribution of hepatic vitamin A levels in otters.** The group of otters with vitamin A values greater than 700 μmol/kg included three with values in excess of 1,000 μmol/kg.

The reasons for the bimodal distribution are unclear. The proportion of otters in a calendar month with values less than 100 μmol/kg varied according to the time of the year, with the highest proportion of low values occurring in the summer months (Figure 9.2). The pattern of monthly median values for all otters was the inverse of that in Figure 9.2, with the highest values in the winter months.

Since the highest proportion of low vitamin A values occurred during the warmer months of the year, it is possible that samples collected at that time may have been affected by autolysis. However, 11 of the 14 samples (78 per cent) with the lowest vitamin A values (<10 μmol/kg) were collected between September and January. It was concluded, therefore, that additional, unidentified, factors are responsible for low vitamin A levels.

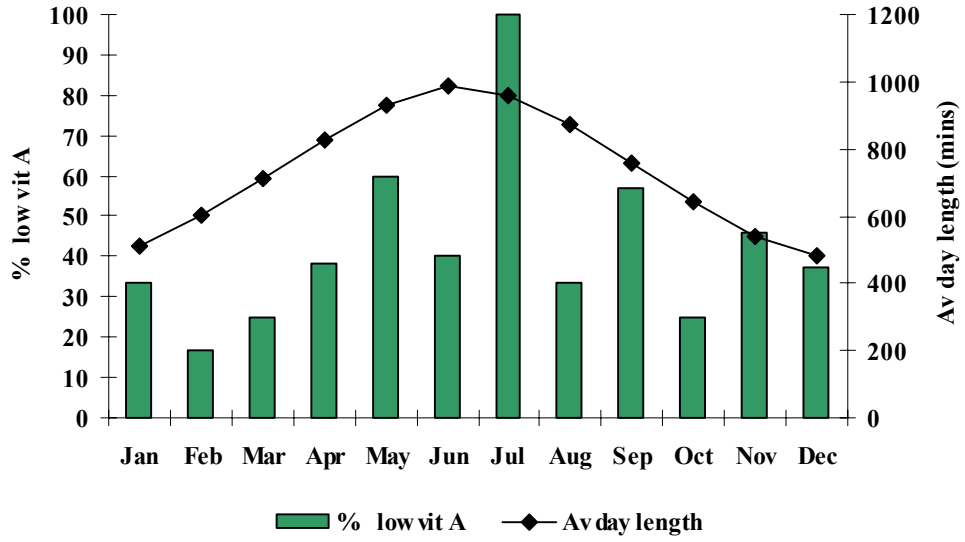

Figure 9.2 The proportion of otters in a month with vitamin A values of less than 100 μmol/kg shown in relation to day length. Low vitamin A levels occurred most frequently during the summer months.

Vitamin A levels were analysed for evidence of variation between sexes and age classes. Data for otters that died in different months were pooled and analysed using a GLM that included age and sex as factors and an interaction term between age and sex. Age and sex were not significantly related to vitamin A level, but the interaction term between age and sex was significant ($F_{1,87}$=6.03, p=0.016). This was because vitamin A levels tended to be higher in adult females than adult males but lower in non-adult females than non-adult males (Figure 9.3).

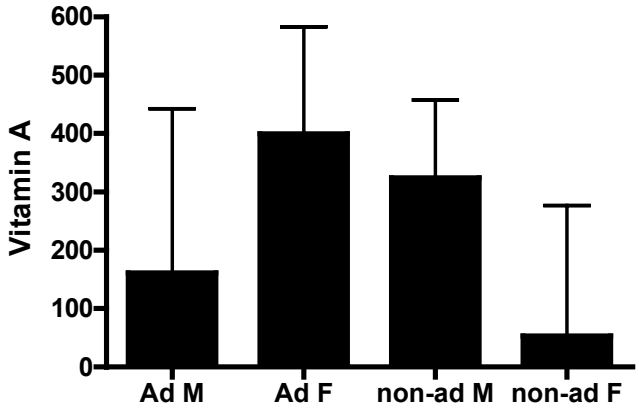

Figure 9.3 Box plot with interquartile ranges of median vitamin A levels in adult and non-adult otters.

A scattergraph of vitamin A levels plotted against year for all otters showed no obvious change in vitamin A values over the study period (Figure 9.4). However, when the data were analysed separately for adults and non-adults using a GLM that included year as a covariate and sex as a variable factor, there was no statistically significant change in Vitamin A levels over time in adults, but levels in non-adult otters rose significantly over time (p=0.015) (Figure 9.5). When cubs were included in the model, the significance increased ($F_{1,28}$=7.77, p=0.009).

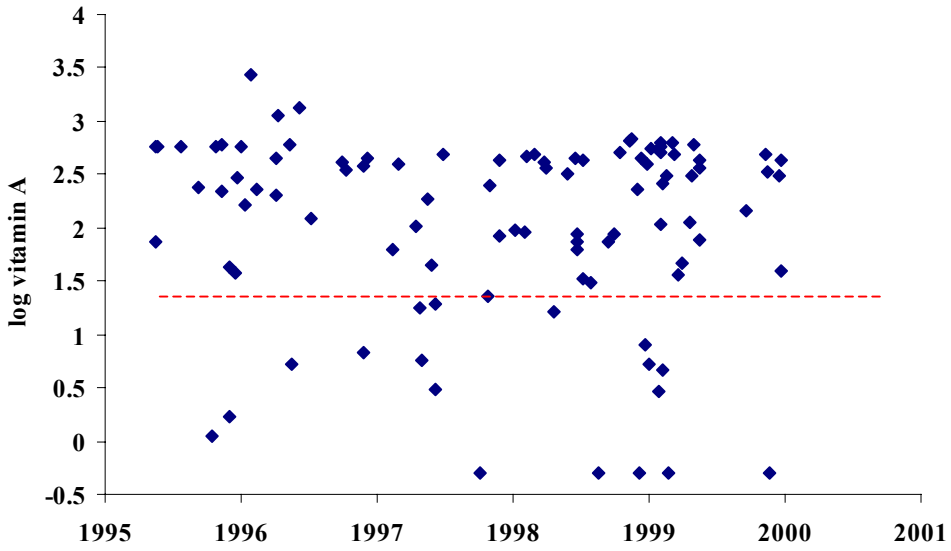

Figure 9.4 **Log vitamin A values over time.** There were 14 otters with hepatic vitamin A levels below 7 μmol/kg (dotted red line). This is regarded as a critical level in domestic species. Livers from five otters that had no detectable vitamin A were given a nominal value of 0.5 μmol/kg.

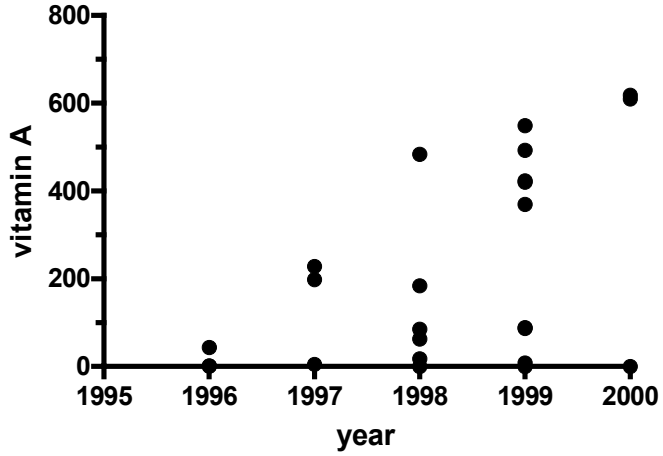

Figure 9.5 **Vitamin A values (μmol/kg) in non-adults over time.** The vitamin A levels in non-adult otters rose significantly over a five year period of the study. There was no significant change in adults over this same period.

10. Effect of pollutants and other factors on organ size

10.1 Adrenal glands

The earlier study of otters from south west England showed that adrenal weight was not only strongly correlated to body weight but also to hepatic concentrations of PCB congeners 138, 153 and 180 (Simpson 1998). It became apparent in the present study (see section 3.3.3), that adrenal mass is affected by the otter's health status and possibly other factors. A backward, stepwise, GLM approach was used to investigate whether a number of factors might influence adrenal weight. These included body length, concentrations of PCB 153, DDE, HEOD, sum PCBs and TEQ (all entered as covariates in the model), health status, whether the carcase was fresh or frozen, sex, year of death and age class. Covariate data were ranked so the model would fit underlying statistical assumptions. To study the effect of health status, adrenal weights from healthy otters were compared with those from 45 cases showing clear evidence of disease.

The factors eliminated from the full model were, in descending order, DDE, sum TEQ, PCB 153, HEOD, sum PCB, year and sex. The factors remaining in the final model are shown in Table 10.1.

As might be expected, body length and age were highly significant predictors of adrenal weight and adrenals in sick otters were significantly heavier than in healthy ones. However, the analysis also showed that freezing has a marked effect, with adrenals from frozen carcases being lighter than from fresh ones. None of the contaminants examined had a demonstrable effect on adrenal weight.

Table 10.1 Results of analyses of variance for rank adrenal weight

Variable	DF	F value	p value
Rank length	1	92.91	0.000
Sick/healthy	1	10.61	0.001
Fresh/frozen	1	16.50	0.000
Age class	2	7.86	0.001
Error	212		
Total	217		

10.2 Thyroid glands

Thyroid glands, like most organs, show a strong positive correlation with body weight but the 1988-96 study (Simpson 1998) also indicated a correlation between thyroid weight and DDE levels. A backward, stepwise regression model, as used for adrenal glands, was employed to investigate the effect of a number of factors on thyroid weights from the present study. Data were ranked and cubs excluded so that the model would fit underlying statistical assumptions.

The factors eliminated from the full model were, in descending order, sum TEQ, age class, sum PCB, HEOD and PCB 153. The factors remaining in the final model are shown in Table 10.2.

Table 10.2 Results of analyses of variance for rank thyroid weight

Variable	DF	F value	p value
Rank length	1	23.10	0.000
Sick/healthy	1	14.47	0.000
Fresh/frozen	1	24.86	0.000
Sex	1	30.39	0.000
Year of death	7	2.57	0.015
Rank DDE	1	6.07	0.015
Error	185		
Total	197		

There was a strong positive relationship between thyroid weight and body length and between thyroid weight and sex. As observed for adrenals, thyroids from frozen otters were significantly lighter than from fresh ones. The observation recorded in section 3.3.3.6 that sick otters have smaller thyroids than healthy ones was confirmed. Thyroid weight was also influenced by year of death, with weight falling over time, and by DDE concentration, where there was a positive correlation.

10.3 Baculi

There is widespread concern in both human and veterinary medicine about the ability of some classes of environmental pollutants to interfere with normal development of genital organs. As predators at the top of a food chain, otters will be exposed to compounds such as OCs and PCBs and a study on Canadian river otters (*Lutra canadensis*) in North America indicated that high levels of halogenated hydrocarbons were linked to poor baculum development in young animals (Henny *et al*. 1996). In view of this, 139 baculi from otters in the present study were made available to the Royal Veterinary College, London, for a student pathology project.

At that time pollutant analysis for the total data set was incomplete but results were available for 58 otters from which baculi had been collected. PCB levels were lower and pp'DDE levels similar to those recorded in the polluted river system in the North American study but dieldrin levels were higher (Henny *et al.* 1996, Zinkann, 2004). Otters in the cub and immature age classes were combined to form a juvenile age class. The study showed a significant negative correlation between juvenile otter baculum length and hepatic concentrations of both dieldrin and the sum of DDT derivatives. There was no such correlation in the subadult or adult age classes. The smallest baculum came from a cub with the highest burdens of DDT metabolites, dieldrin and sum PCB congeners (Zinkann, 2004).

11. Discussion

The overall impression from this study is that otters in southern and south west England are doing well. The numbers submitted each year are increasing, most specimens are well nourished, there is no evidence of significant infectious diseases and otters are breeding successfully. These observations accord with the results of the most recent national otter survey (Crawford 2003). It was a different story 25 years ago when many informed commentators were concerned about the status of the otter population, not only in Britain but elsewhere in Europe (O'Connor *et al.* 1977; Lenton *et al.* 1980; Olsson *et al.* 1981; Mason and Macdonald 1986). It is pertinent, therefore, to examine these latest results to try and identify factors that may have brought about the change in the otter's fortunes, or that may threaten them in the future.

11.1 Otter population size

The steady increase in the numbers of otters submitted each year suggests that the population is increasing. However, as more than 80 per cent of these cases were road traffic casualties, it could be argued that more otters are killed on the roads because the volume of road traffic has increased. Although this may be partly true, the volume of traffic on trunk roads in south west England increased over the study period by approximately 3 per cent per year (D. Mayhew personal communication 2005). This is much less than the annual increase in otter submissions.

It has also been suggested that increased public awareness of the study and increased involvement of Environment Agency and Wildlife Trust personnel may have resulted in more otters being submitted. Although this may be the case, it is a fact that, as otter deaths have become increasingly common events, they have ceased to generate the interest that was evident in the past. As a result, the enthusiasm to collect and deliver bodies has tended to diminish. Other reasons for reduced submissions include the growing number of people who retain a corpse for taxidermy and the increased volume of traffic which makes people reluctant to stop and collect a corpse on a busy road.

It is impossible to know which of these factors has had the most influence on the number of otters submitted. However, the progressive increase in submissions by county in an easterly direction over time, together with submissions from areas that until recently were without otters, suggest a population expanding outwards from its stronghold in the west. These observations, together with the remarkable increase in reported sightings, and increased signs of spraints during surveys (Crawford 2003), provide strong evidence for a significant increase in the otter population in southern and south west England. Cornwall and Devon had the highest proportion of positive sites of any region in the 2000-2002 survey (Crawford 2003) and the otter population in the south west is now probably close to what it was before the crash some 40 years earlier.

11.2 Patterns of mortality

The clear seasonal pattern of mortality seen in this study is very similar to that seen in Norway (Heggberget and Christensen 1994). Whilst it has been observed that road casualties increase when rivers are in spate (Simpson 1997), this alone is not a sufficient explanation. The Norwegian workers considered that darkness, snow, ice and the setting of fyke nets in winter were significant factors. However, the combined results of the 1988-96 study (Simpson 1998) and the current one show that there is

strong positive correlation between monthly mortality and hours of darkness (Simpson 2006). As otters are largely nocturnal it would seem logical that longer nights make it possible for them to travel further, increasing the probability of them crossing a road or encountering another otter.

11.3 Road casualties and bite wounds

The proportion of otters killed by traffic remained remarkably constant at around 83 per cent per year from 1988 to 2003. Over the same period, the proportion with bite wounds increased dramatically, rising from around 16 per cent to 52 per cent. Overall, nearly 39 per cent of otters had been bitten and 10.7 per cent died as a direct result.

In previous reports, bite wounds seen in otters at post mortem have been attributed to domestic dogs (Kruuk and Conroy 1991; Mason and O'Sullivan 1992). Whilst there was evidence of this in the present study, dog bites were mostly confined to young otters or ones that were already sick. There were also mink bites, consistent with other reported evidence of aggressive interactions between otters and mink (Bonesi and Macdonald 2004) and eyewitness accounts of otters attacking mink (K O'Hara, personal communication 2005). The majority of the bite wounds were considered to be the result of fighting between otters.

The author has published previous evidence of intraspecific aggression in otters (Simpson 1997; Simpson and Coxon 2000) and in recent years cases have been recorded in Wales (Bradshaw and Slater 2001) and the west coast of Scotland (A. Patterson, personal communication 1999, V. Simpson, unpublished data). There are also reliable historical accounts of otters fighting, often suffering serious injuries as a result (Stephens 1957; Erlinge 1968). Despite this evidence, some commentators have expressed the opinion that bite wounds in otters are rare or of little significance and that physical aggression plays a minor role in otter social behaviour (Laidler 1982; O'Hara 2004).

The present study has shown that the prevalence of bite wounds is influenced by a number of factors including age, sex and month of death. The combined data from the 1988-96 and the 1996-2003 studies shows that the prevalence of bite wounds in adults of both sexes reached a peak in the summer months. By contrast, none of the subadults examined between April and August had bite wounds, even though more than 50 per cent of them examined between November and February had been bitten (Simpson 2006). These patterns of fighting are likely to relate to defence of territory or breeding activity, especially as there was a 1.6 to 1 male to female mortality ratio in adults but no sex bias in the younger classes.

Observations by experienced biologists on the territorial behaviour of otters in both Sweden and the Shetland Isles suggest that fighting is uncommon and that otters generally avoid one another rather than engage in conflict (Erlinge 1968, Kruuk 1995). These findings appear incompatible with those in the present study where, depending on the season, between 20 and 50 per cent of the otters had recent bite wounds. Accurate aging of the wounds was not possible but, by comparison with bites seen in veterinary practice in domestic dogs and cats, the great majority would have been less than two weeks old. This implies that, at any time in late summer, for example, over half the otter population in southern and south west England is engaged in fighting. This is highly improbable.

The high proportion of otters suffering from bites is most unlikely to be a true reflection of the level of conflict in the population. It is more probable that the otters submitted for

examination represent a biased sample. Otters engaged in some form of interaction, either with another otter or another species such as a domestic dog, are more likely to be killed by traffic. This would be particularly the case when they are engaged in a territorial dispute, or when actually fighting. One might expect a high proportion of these animals to have bite wounds.

The dramatic rise in the proportion of otters with bite wounds over the study period is almost certainly indicative of an increased level of conflict in the population. This would be consistent with increased competition for territory as the otter population has expanded. However, the bite wounds in most cases are unlikely to be the result of a single interaction. Many of the otters killed by traffic had multiple, different age, bites indicative of repeated fighting. In addition, otters with extensive bites usually had a low condition index and this weight loss would have occurred over a period of days or weeks.

It is the author's opinion that many, possibly the majority, of road casualties are a direct result of aggressive interactions between otters or, less frequently, other species.

11.4 Dental lesions and bite wounds

It was apparent that some otters with fresh bite wounds also had recent dental lesions, such as missing or fractured incisors or fractured canines. It is suspected, therefore, that many lesions involving incisor or canine teeth are due to otters fighting and the increase in the proportion of otters with dental lesions over time is a consequence of an increased level of conflict. Alternatively, as older animals are more likely to have accumulated dental lesions, it could be that otters are living longer than they were 10 or 15 years ago. Although the results of dental age determination need to be viewed with caution, they do not support this suggestion. In the period 1988-96 the mean age was two years and the oldest was four years. Based on teeth examined during the present study, the median age was 2 years and the oldest was 8 years (Brickhill 2004).

11.5 Illegal killing

On several occasions people who submitted otters suspected they had been killed illegally, especially where the otter had been found away from a road but close to a commercial fishery or an ornamental fish collection. However, post mortem examinations showed that most of these suspicions were unfounded. This was well illustrated by the case of the large dog otter found dead in the River Crane, Dorset, close to a large fishery. It had a short length of alder stick jammed between the carnassial teeth across the roof of its mouth and this had prevented it from feeding. In a small number of cases there was evidence of deliberate, illegal killing. However, even with strong forensic evidence it can be difficult to effect a prosecution. This was well illustrated in a case where no action was taken after it was proved that a snare recovered from a carp fishery had been used to catch an otter, and where a pregnant bitch was found nearby with its head smashed and carp remains in its stomach. As the otter population expands, it is inevitable that more cases like this will occur.

11.6 Body condition and diet

In general, the nutritional state of the otters was good. Although analysis of stomach contents has yet to be completed, the study has reinforced the fact that eels and amphibians are important components of the otter's diet. It is a matter of concern therefore, that populations of eels and frogs appear to be in decline (Anon. 2004;

Cunningham *et al.* 1993). Further analysis of the data is needed to show the distribution of the prey items by catchment or county. Species such as bullheads, loach and salmonids may be common in north Cornwall but in the Somerset Levels cyprinids will be predominant. This information could assist in the interpretation of pollutant burdens and vitamin A levels in the otters, especially if it were shown that otters of different age class or sex preferred prey of a certain size or species.

11.7 Captive bred and rehabilitated otters

Otters from Dorset and Hampshire were significantly larger than those from counties further west. Whether this is due to dietary factors or genetic differences is currently unclear. Otters bred in captivity have been released in Hampshire, and one road casualty submitted from Hampshire had a DNA profile matching that of an otter in a zoological collection in Cornwall (P. Chanin, personal communication 2003). The captive otter had been purchased from the organisation responsible for releasing otters in Hampshire. Two otters from Hampshire had what appeared to be congenitally deformed front feet, lesions that can be indicative of inbreeding. The contribution of captive bred otters to the population in southern and south west England is unknown. However, between 1996 and 2001 all otters submitted for post mortem examination were scanned for the presence of a microchip and, with the exception of a few cases re-released by the RSPCA, all proved negative.

The rearing and subsequent release of abandoned or orphaned cubs presents a number of problems. When young cubs are being hand reared they often develop stomach ulcers, almost certainly due to stress, and even if they are successfully reared, there remain issues relating to disease control and welfare. To the best of the author's knowledge there are no rehabilitation centres in England dealing exclusively with otters. The practice of keeping them alongside other species, especially exotics, means an increased risk of disease transmission and the potential to introduce a disease into the wild when the animal is released. There are also welfare issues, as an otter released in to an already well-populated area is very likely to be attacked by the resident otters.

When, during the 1970s or 80s, the survival of the otter population was in question, the release of captive bred specimens and the rearing and rehabilitation of wild ones was probably justified as a conservation measure. This is no longer the case.

11.8 Reproduction

The pioneering work of Aulerich and Ringer (1977) in the USA showed how PCBs in the diet of mink had a dramatically adverse effect on reproduction. This resulted in many scientists linking PCBs with the decline of Eurasian otters (Mason and Macdonald 1986; Broekhuizen 1987; Olsson *et al.* 1981; Smit *et al*. 1998). However, despite much research and chemical analysis of otter tissues in many laboratories, there is no proof that PCBs cause reproductive failure in otters. A major difficulty is that it is almost impossible to obtain liver samples from otters that are known to have aborted or suffered foetal resorption.

An otter foetus has a zonal placenta, that is, there is a band around the placenta where it is attached to the lining of the uterus. After parturition the site of detachment remains visible, usually as a dark greyish transverse bar. These are known as placental scars and they can be used to determine how many foetuses were in the uterus. As they are transient they can also be used to decide if a bitch has bred recently. The period they

persist for does not appear to have been accurately established but it is probably about three months (Elmeros and Madsen 1999; Heggberget and Christensen 1994).

Of the 129 females suitable for assessment in this study 79 were adult. Thirty five of these were either pregnant or had placental scars and/or were lactating. A further 17 had evidence of earlier breeding, such as a large uterus or large nipples. Therefore, 44 per cent of the adult females were either pregnant or had bred recently and 66 per cent were judged to have bred at some time. At first sight this appears to indicate a very satisfactory state. However, in some cases an otter had well-developed mammary glands full of milk but no placental scars. In others the reverse was true, with prominent placental scars and a large uterus but no evidence of lactation. There were also a few cases where some of the placental scars were the normal grey colour but one or more were yellow or orange.

Otters are not known to have phantom pregnancies but other than this it is difficult to explain how an otter can lactate if it has not given birth. A possible explanation for cases with placental scars but no sign of lactation is that the foetuses or cubs had been aborted, or had died or been killed. In the absence of young, lactation would cease but the scars would remain. Where a yellow placental scar was present in the same uterus as dark grey ones it seems likely that the foetus had died and been resorbed. Although at present there is no evidence to confirm this suggestion, investigators in Denmark have drawn the same tentative conclusion from similar cases seen there (Elmeros and Madsen 1999). In Norway Heggberget and Christensen (1994) found evidence of significant intrauterine and also postnatal mortality. Without being able to examine the foetuses or cubs, the causes remain a matter of speculation but premature foetal death and poor kit survival are features of PCB toxicity in mink (Aulerich and Ringer 1977). However, in addition to the potentially lethal effects of pollutants, or infectious agents, some very young cubs almost certainly die due to predation or male infanticide (Simpson and Coxon 2000).

11.9 Lung lesions

Significant pathological lesions were seen most frequently in lungs. The commonest were those resulting from fungal or bacterial infections and although these were mostly of a minor nature, in some instances the infection resulted in fatal pneumonia and/or pleurisy. Many of these were animals with septic bite wounds but there were several cases where histopathological examination showed that the focus of infection was centred on inhaled foreign material. In adults this was thought to be plant debris but in several cubs the material was amorphous and could have been food particles. Inhalation pneumonia is uncommon in other species of wild mammals and, in adult otters at least, raises the question of whether the foreign material was inhaled along with water when otters were fighting.

In many cases the nature of the lesions could only be established by a combination of bacteriological and histopathological examinations. However, these procedures cannot be carried out satisfactorily on frozen or autolysed specimens. This illustrates the point that for effective monitoring of the health status of otters, or any other wildlife species, bodies need to be examined in fresh condition.

11.10 Thymus and spleen

The thymus glands in most sick otters were regressed and histopathology in these cases typically showed marked lymphocyte depletion. Similarly, in the regressed spleens there was atrophy of white pulp and loss of lymphocytes. Although it is well

known that changes such as these are seen in animals suffering from severe stress, malnutrition or infectious disease, they can also be readily induced by exposure to toxins, especially TCDD and some of the PCB congeners (McConkey *et al*. 1988; Schuurman *et al*. 1991; Tanabe *et al.* 1987). Other lymphoid organs are also affected, and as a result the immune system is suppressed and the animal is less able to resist infection.

Although lymphoid organs such as thymus and spleen may have been affected by PCBs, particularly during the early stages of an otter's life, these organs were often regressed in sick or severely stressed otters. PCB levels in fatty tissues can rise significantly during stress (Hornshaw *et al.* 1983), which could obscure any relationship between pollutant levels and lymphoid pathology. In view of this, no attempt was made to correlate them. However, this is an area worthy of further study in otters, especially in relation to immune status and vitamin A levels.

11.11 Renal calculi

Renal calculi are common in otters kept in captivity, especially in Asian small clawed otters (*Aonyx cinerea*). However, they also occur in wild populations and Weber (2001) found calculi in the kidneys of 23.4 per cent (105 of 449) of Eurasian otters from a number of northern and central European countries. The prevalence varied, with 9.2 per cent positive in Germany, 16.3 per cent in Denmark, 22 per cent in Austria and 32.5 per cent in Scotland. None were seen in a small sample (n=6) from the Czech Republic.

Calculi have also been recorded in wild otters in England and Wales (Stephens 1957; Keymer *et al*. 1981; Bradshaw and Slater 2002) but none were seen in the 77 otters examined in south west England between 1988 and 1996 (Simpson 1998). It is remarkable, therefore, that they became increasingly common during the present study. The overall prevalence in southern and south west England between 1996 and 2003 was 9.7 per cent and this is similar to the 7.9 per cent prevalence in specimens recovered predominantly from Wales and, to a lesser degree, England during 1992-2000 (Bradshaw and Slater, 2002).

The overall prevalence figure of 9.7 per cent is misleading, as calculi were never seen in cubs or immatures and only twice in subadults. If these age classes are excluded, the prevalence in adults was 13.4 per cent. However, this figure is also misleading as it ignores the rise in prevalence over the 16 year period of the study. In 2003, 33 per cent of adults had calculi. It is clear that when comparing the prevalence of calculi in different populations, the age structure of the sample must be similar.

A number of factors can initiate the formation of calculi in the kidneys and the reasons for the marked rise in the annual prevalence between 1988 and 2003 are not known. It may be linked to the increased incidence of bite wounds and dental lesions in this period and, possibly, to an increase in the proportion of older animals in the population. The marked increase in focal lesions seen in lung tissue during the study is thought to be due to bacteria entering the blood stream, mostly from infected bite wounds. It is tempting to speculate that a similar process may be happening in kidney parenchyma, each lesion providing a nidus, or starting point, for the formation of a calculus. This would be consistent with the observation that calculi were frequently present in otters that had severe bite wounds or septic dental lesions.

One final consideration concerns the apparently higher prevalence of calculi in otters from Dorset and Hampshire. Studies on urolithiasis in various species of otters in

captivity, including Eurasian and small clawed otters, have shown that calculi are mostly composed of calcium oxalate, calcium urate or calcium phosphate (Keymer *et al.* 1981). As the rivers in Dorset and Hampshire have much higher calcium levels than those in the South West, it is possible that this might predispose to the formation of calculi. However, the study by Weber (2001) showed that calculi in wild Eurasian otters were predominantly ammonium urate, not calcium urate. It seems unlikely therefore, that high calcium levels in water influence the formation of calculi in wild otters. A second possible explanation for the high prevalence in Dorset and Hampshire is that some of these otters were captive release cases that had developed calculi whilst in captivity.

11.12 Infectious diseases

There is little evidence of significant bacterial disease in wild otters in the UK (Simpson and King 2003) and most of the organisms identified in this study had either originated from the oral cavity of an aggressor or were opportunistic invaders.

There is one historic record of tuberculosis in an otter from Cornwall (Stephens 1957) but the organism was not identified to species level. More recently, a similar case from Scotland was shown to be due to *Mycobacterium avium* subsp. a*vium* (A. Patterson, personal communication 1999). Although there is potential for *M. bovis* in badgers to spill over into otters, and a number of otters in this study had lung lesions suggestive of tuberculosis, all proved negative by culture and histopathology.

During the study there were occasional cases of salmonellosis and yersiniosis. These infections have been recorded in otters previously (Keymer 1992) but do not appear to be of significance at a population level. A number of authors have suggested that otters may suffer from leptospirosis (Wayre 1979; Laidler 1982; Chanin 1985; Keymer 1992). However, there was little pathological evidence to support this in either the present or the previous study (Simpson 1998). It is concluded that leptospirosis in otters is uncommon. This was confirmed by the examination of kidneys by PCR assay, which showed a low prevalence of leptospires (Jones, 2003).

An unnamed species of *Brucella* was isolated from a coastal otter in Scotland (Foster *et al*. 1996). What is believed to be the same organism has been identified from a variety of marine mammals, including a harbour porpoise from Cornwall (Dawson *et al*. 2004). Although one otter in this study had antibody to *Brucella* sp. this could have been due to a cross reaction with a related species. All cultures for the organism were negative and it is apparent that, if the infection occurs at all in otters in the region, it is at an extremely low level.

In human medicine gastric ulcers are commonly due to infection with *Helicobacter pylori*. A closely related organism, *Helicobacter mustelae*, is known to infect stoats (*Mustela erminea*), American mink and ferrets (*M. putorius*). Therefore the possibility that this organism was associated with gastric ulcers in otters was investigated. The collaborative study, carried out partly as a student project at Liverpool University and partly at the Health Protection Agency, Colindale, found no evidence of *H. mustelae* in the otters, although the organism was found in stoats and mink (Harris 2004). These results are consistent with the absence of organisms resembling *Helicobacter* in histology sections and endorse the conclusion that pyloric ulcers in otters are principally due to stress.

As the population expands, otters are being found with increasing frequency in cities, and this must increase their exposure to viruses that affect domestic dogs and cats.

Canine distemper virus has been diagnosed in otters in Germany (Geisel 1979) and it has been speculated that a case occurred in Norfolk (Jefferies 1984). However, there is no evidence to support the latter claim and it is refuted by Keymer (1991). In the present study, serological screening of 45 otters for canine distemper antibody by Glasgow University Veterinary Department all gave negative results. Similarly, all the samples proved negative for antibodies to canine parvovirus and canine adenovirus.

Aleutian disease is a common viral infection of American mink and a study from the upper Thames region of England showed a high prevalence of antibody in feral mink (Yamaguchi and Macdonald 2001). Otters are believed to be susceptible and the disease has once been suspected in an otter in England (Wells *et al.* 1989). In addition, Aleutian disease viral DNA was detected in a free-living otter in Spain (Manas *et al.* 2001). Only one otter in the present study had pathological lesions suggestive of Aleutian disease and examinations for antigen and antibody proved negative.

There was no evidence of significant pathology associated with confirmed parasite infections. However, retrospective examination of records in 2004 indicted that several otters from Somerset that had shrunken, thickened gall bladders were almost certainly infected with the bile fluke *Pseudamphistomum truncatum*. This parasite had not previously been described in Britain and is likely to have been introduced by the importation of infected fish from Eastern Europe or Russia (Simpson *et al.* 2005). The potential impact on the otter population is not known but the author is concerned that the infection is likely to spread to other lowland areas, such as the Norfolk Broads, by unregulated movements of fish from the infected area.

Adiaspiromycosis is a common fungal disease of small mammals worldwide but can also affect humans, particularly if they are immunocompromised. Members of the family Mustelidae appear to be particularly susceptible and infection has been described in otters in the former Czechoslovakia (Krivenec *et al*. 1976), Finland (Rudback and Sternberg 1998) and England (Simpson 1998). Although some of the adiaspiromycosis affected otters in this study had quite extensive granulomatous lesions throughout the lungs, and one died from an overwhelming infection (Simpson and Gavier-Widen 2000), most infections are believed to be subclinical. No other fungal infections were diagnosed.

11.13 Organochlorines and PCBs

Hepatic residues of DDE, TDE and dieldrin in otters in south west England all declined significantly between 1988 and 1996 (Simpson 1998; Simpson *et al.* 2000) and the results of this later study show a similar pattern of decline. These findings are consistent with the progressive withdrawal of organochlorine pesticides over the period 1962 to 1989. By contrast, although levels of HCB declined between 1988 and 1996, no such decline was detected in the present study. HCB is a by-product of the constantly expanding petrochemical industry and is distributed primarily by atmospheric fallout. These results, therefore, are not unexpected.

Although total PCB did not decline in the period 1988-96, there were downward trends in hepatic concentrations of some PCB congeners (Simpson 1998, Simpson *et al.* 2000). In the present study trends over time were only calculated for one of these congeners, PCB 153, but no downward trend was seen. Sum PCB was employed in this study to represent overall PCB burden but, as observed with total PCB in the period 1988-96, there was no evidence of a decline. Similarly, there was no decline in sum TEQs between 1996 and 2003. These findings are consistent with those of Bradshaw and Slater (2002), where no decrease in PCBs was seen in otters from elsewhere in England and Wales.

In the period 1988-96, pollutant levels were seen to be generally lower in female otters than males (Simpson 1998). However, the size of the dataset in the present study permitted examination of pollutant levels according to sex and age class and this has yielded some of the more valuable results.

Organochlorine and PCB concentrations were consistently higher in adult male otters than in non-adult males (combined subadults and immatures), which suggests bioaccumulation of these pollutants with increasing age. However, residues of both groups of compounds were markedly lower in adult females than in adult males. Although this could be attributed to possible differences in prey selection, adult females tended to have pollutant burdens that were equal to, or less than, those in non-adult females. A similar observation was made by Bradshaw and Slater (2002), where lactating females had lower burdens than quiescent ones.

The halogenated hydrocarbon pollutants can cross the mammalian placenta and accumulate in developing foetuses (Perry *et al*. 1984). However, as the compounds are strongly fat soluble, they are also transferred via colostrum and milk (Tanabe 1988; Bernhoft *et al.* 1997) and this is the major route of excretion. In humans, PCB levels in maternal tissues decrease by 30 to 50 per cent in the first six months of lactation but in dolphins PCBs may decrease by over 90 per cent during lactation (Yakushiji 1988; Tanabe 1988). Human milk is around two to three per cent fat (Yakushiji 1988) but in cetacean milk the fat content may be as high as 30 per cent. This lipid-rich milk is believed to be the reason for the high level of transfer in cetaceans. Otters' milk is 24 per cent fat (Ben Shaul 1962) and it can be reasonably assumed that excretion in milk is the main reason why OC and PCB burdens in adult female otters in this study are significantly lower than those of adult males or non-adult females.

Although organochlorine residues were generally comparable in non-adult males and females, a different situation appeared to exist with sum PCBs and TEQs. In this case, residues in non-adult females were significantly higher than in the non-adult males. The reasons for this are currently obscure.

One of the most frequently quoted references in otter conservation is that of Chanin and Jefferies (1978). They pointed out that the crash in the otter population in Britain was first identified by otter hunts and that it occurred shortly after dieldrin was introduced as a pesticide in the late 1950s. There was no analytical evidence from otters to support the suggestion that the pesticide had caused the crash but it was a logical conclusion, as dieldrin had already been shown to be toxic to foxes (*Vulpes vulpes*) (Blackmore 1963) and birds of prey (Ratcliff 1980). An earlier report, which seems to have gone largely unnoticed, showed that mink exposed to dieldrin in their diet survived for about six months but then suffered almost 100 per cent mortality (Aulerich and Ringer 1970). In the UK, the use of dieldrin and aldrin (which is metabolised to dieldrin) was progressively restricted from 1962 until 1989 when they were finally banned altogether. This was followed by declines in the level of dieldrin in rivers (Zamman 1997) and in otters' livers (Simpson 1989; Simpson *et al.* 2000). As these declines occurred, the otter population started to recover and it is now widely accepted that the crash in the UK otter population was principally due to dieldrin.

However, most of the research in mainland Europe into the decline of otters has concentrated on the PCBs, much of it stimulated by studies in America that showed how toxic these compounds were to mink (Aulerich and Ringer 1977). There is little published data to suggest that dieldrin or other organochlorine pesticides have affected otters in these countries but strong evidence that they are affected by PCBs (Murk *et al.* 1998). Extensive studies during the 1980s and 90s showed that many otters in the UK had PCB levels comparable with those seen on the continent (Mason *et al.* 1986; Mason 1989; Mason and Macdonald 1994; Simpson 1998, Simpson *et al*. 2000). The present study has shown no decline in PCBs over time and, in view of the European results, it would be wrong to assume that these compounds are not affecting otters in this country.

11.14 Vitamin A

This vitamin plays an essential role in many aspects of foetal development, growth, and the integrity of epithelia, particularly of mucous membranes. It is well established from experimental studies that exposure to OCs and PCBs can induce severe vitamin A deficiency (Bank *et al*. 1989; Brunstrom et al 1991; Bröuwer *et al*. 1986).

A study on wild otters from Denmark showed low vitamin A levels to be associated with high PCB levels (Murk *et al*. 1998) and in south west England vitamin A levels in otters rose over time as hepatic concentrations of pp'DDE, dieldrin and certain PCB congeners declined (Simpson *et al.* 2000). In the present study it appeared that vitamin A levels overall had reached a plateau but in cubs, immatures and subadults there was clear evidence that levels have continued to rise. Although a causal relationship could not be proved, these changes have occurred as pollutant levels have fallen and the combination can only be beneficial to the health of otters.

Another significant finding in this study was that the highest median vitamin A levels were in adult females. Non-adult males had the next highest levels, followed by adult males. The group with the lowest levels were the non-adult females. Again, a statistical relationship was not proven but the vitamin A levels in these groups show a strikingly inverse pattern to the pollutant levels, particularly the PCBs.

The reasons for the bimodal distribution of vitamin A levels, and also the high proportion of low values in the summer months, are obscure. As the vitamin is fat soluble and stored in the liver one would not expect seasonal fluctuations in diet to have an immediate effect. The possibility that low values in summer reflect greater

degradation of vitamin A in post mortem samples due to higher temperatures was considered. However, most of the samples with the lowest values (<10 μmol/kg) occurred in the late autumn or early winter and it is likely, therefore, that a number of factors are responsible for low vitamin A levels.

There was no convincing gross pathology suggestive of vitamin A deficiency but histopathology showed that many otters had retinal dysplasia (Williams *et al*. 2004). This condition is known to occur in cases of vitamin A deficiency but had never been reported previously in otters, and possibly in any wild animal. The fact that the lesions were correlated with low vitamin A and high dieldrin levels provides further evidence of the likely impact of halogenated hydrocarbons on the health of otters.

11.15 Endocrine disruption and other factors affecting adrenal, thyroid and baculum size

Enlarged adrenals have been reported previously in otters with severe bite wounds, which are in late pregnancy or lactating, or which have renal calculi (Simpson 1997, 1998; Bradshaw and Slater 2002). In addition, a strong positive correlation between adrenal size and hepatic concentrations of PCB congeners 138, 153 and 180 was seen in otters from south west England (Simpson 1998). A similar relationship to PCB levels was anticipated in the present study but was not apparent. However, adrenal size was significantly greater in otters dying of disease compared with healthy ones killed in accidents.

The histological appearance of many of these enlarged adrenals showed extreme hyperplasia of the outer layer of the cortex, the zona glomerulosa. This layer is predominantly responsible for the production of mineralocorticoids but it is the middle layer, the zona fasciculata and, to a lesser extent the inner layer, the zona reticulosa, which produce glucocorticoids in response to stress. However, the cells of the zona fasciculata often appeared shrunken and had an irregular architecture. Neither they nor those of the zona reticulosa were obviously hyperplastic. These anomalous results indicate the need for an in-depth histopathological study of otter adrenals.

Adrenal and thyroid weights were, as expected, positively correlated to body length and age class. However, whereas otters dying from disease had enlarged adrenals, their thyroids were regressed. Freezing had a negative effect on the weights of both organs. These factors need to be taken in to account when investigating the possible influence of pollutants on organ size.

In the earlier report on otters from south west England there was an indication of a possible positive association between thyroid weight and DDE levels (Simpson 1998). The results of the present study confirm this and, in addition, show a temporal relationship, with thyroid weights decreasing and organochlorine levels declining over the same time period. To the best of the author's knowledge, this is the first time a relationship has been recorded between DDT and a physiological response in otters. It was disappointing, therefore, that the small study to investigate whether thyroid morphology was affected by pollutants proved inconclusive (Millins 2005). The study was compromised by the small number of thyroids that were suitable for histopathological examination and for which pollutant analytical results were available. In the light of the latest results, and as more livers have now been analysed, further investigations of thyroid morphology in relation to pollutant levels should be carried out.

In human medicine increased levels of hypospadias (congenital defect of the urethra), cryptorchidism (failure of testes to descend), testicular cancer and poor semen quality have been attributed to environmental pollutants such as OC pesticides, PCBs and related compounds (Toppari *et al*. 1996; Turner 1999; Toppari and Skakkebaek 2000). Similarly, studies on a range of animal species including fish, reptiles, and mammals have concluded that environmental pollutants can alter the development and expression of male sexual characteristics (Toft *et al*. 2003; Guillette *et al*.1996; Gunderson *et al*. 2004; Oshako *et al*. 2001). Penis development in female polar bears (*Ursus maritimus*) is believed to be due to endocrine disruption cased by environmental pollutants (Wiig *et al.* 1998).

A study in North America compared baculum size of Canadian river otters in a polluted river system with those from relatively unpolluted reference sites (Henny *et al.* 1996). Juveniles from the polluted system had significantly smaller baculi than those from the reference sites. There was also a strong negative correlation between baculum size and hepatic levels of OCs, PCBs, dioxins and furans. However, in subadults or adults there was no significant difference in baculum size between the two populations. The authors suggest that the effects of pollutants on penis development are temporary, with high levels of exposure having an effect early in development, followed by a period of normal growth as levels stabilise.

The preliminary investigation in the present study into the possible effects of pollutants on baculum size showed a strong negative correlation between baculum length and hepatic concentrations of dieldrin and the metabolites of DDT (Zinkann 2004). However, unlike the American study, no samples from a relatively unpolluted population were available for comparison and small baculi and high pollutant concentrations in young cubs could simply reflect their stage of growth and a highly contaminated milk diet prior to weaning. At the time of that study the results of pollutant analysis were only available for 58 of the 139 otters where baculi had been collected. Since then, more baculi and liver samples have been collected, including samples from otters from the west coast of Scotland where OC levels may be lower. In future, it may be possible to use these data to investigate further the relationship between pollutant levels and penis development in Eurasian otters.

12. Conclusions

This study indicates that the health status of the otter population in south west and southern England is generally good. Most of the otters examined were in good nutritional condition and there was no evidence of significant infectious disease. Over time there was a marked increase in the proportion of otters suffering from bite wounds and in 2003 more than half the animals examined had been bitten. Although some wounds were due to attacks by domestic dogs or conflict with mink, most were due to intraspecific aggression. It is likely that the increase in intraspecific aggression over time was largely due to competition for resources as the otter population expanded.

Bite wounds are a significant cause of mortality and, as animals dying from wounds and secondary infections are less likely to be collected for examination than those killed on roads, the true level of mortality from fighting is almost certainly higher than shown in this study. Sampling bias may also explain the high proportion of otters suffering from bite wounds, as it is believed that otters engaged in aggressive interactions are more likely to be killed in road accidents. There was unequivocal evidence of sampling bias during the summer months when adult males outnumbered females by almost four to one.

As the prevalence of bite wounds increased, so did the proportion of otters suffering from dental lesions and renal calculi. It is suspected that both these conditions are a consequence of otters fighting, although in the case of the calculi the mechanism is unclear. If the otter population continues to increase, it is likely that the proportion affected by bite wounds will also increase, and there will be a corresponding increase in mortality.

The study provides strong evidence of aggressive interactions between otters and mink. In view of this, it is likely that further expansion of the otter population may influence the distribution and size of the mink population. As mink are known to be a serious threat to water voles (*Arvicola terrestris*), an increase in otter numbers may, in turn, influence the status of water voles in the regions.

DDT and dieldrin were banned more than 15 years ago. Although this study has shown that levels are declining, they were still detected in the tissues of almost every otter. However, tissue levels on their own are of limited value, and an important achievement of this study was being able to demonstrate a link between the organochlorine levels and pathological conditions, including retinal dysplasia, hypovitaminosis A and enlarged thyroid glands.

PCB levels in otters in south west and southern England declined between 1988 and1996 but there is no evidence that levels continued to fall between 1996 and 2003. There is still potential for these and related compounds to adversely affect otters and other aquatic species. In addition, the continued presence of these compounds in fresh water systems has implications for human health.

13. Recommendations

Although wildlife conservation is not the prime function of the Environment Agency (Harman 2000), it is the lead organisation in the UK Biodiversity Action Plan for some 39 species, including otters. Key components of the Environment Agency's input include coordinating the collection of otter corpses for post mortem examination, recording basic data and analysing tissue samples to monitor pollutant levels. The Environment Agency is committed to continuing with this work in the foreseeable future and this can be achieved with basic post mortem examinations, irrespective of whether the otters are submitted freshly dead or deep frozen.

If in addition, the Environment Agency intends to continue to monitor the otter population for evidence of disease, it will be necessary for post mortem examinations to be carried out in greater depth. Freshly dead specimens are required for detailed pathology, including histopathology and microbiology. Frozen carcases are of limited value. The fresh carcases need to be examined by an experienced veterinary pathologist who, in turn, needs to be able to collaborate with specialists in other disciplines to pursue novel lines of research. As the density of the otter population increases, so does the risk of disease outbreaks. In order to monitor the health status of otters, the Environment Agency should continue to submit freshly dead specimens for veterinary pathological examination.

Whereas the collection, storage and transport of frozen otters is relatively straightforward, this is not the case for fresh carcases. Ideally, they should be received at the laboratory, chilled and in an insulated container, within 24 hours of death. At present correct delivery fails in a significant number of cases. If meaningful monitoring of the health of otters is to be achieved, the system of collection and transport of fresh carcases needs to be improved.

Many of the conclusions in this report are based on consistent, uninterrupted, long-term submission of specimens. Unfortunately, there are now parts of south west and southern England where, for a variety of reasons, dead otters are no longer collected in a systematic way. As a result, the value of the whole data set is diminished. It is recommended that renewed efforts are made to maintain consistency in collection of corpses. Without this, there is a risk that invalid conclusions may be made in the future regarding otter numbers, distribution, age or sex ratios, disease conditions and levels of pollutants in a particular region.

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Glossary of veterinary terms

Adrenal glands A pair of endocrine glands located near the cranial pole of the kidneys

Aetiology The cause of a disease condition

Amorphous Having no definite shape or form

Anaerobic Growth in the absence of oxygen

Atrophy Decrease in size, or wasting of, a normally developed tissue.

Autolysis The disintegration of cells or tissues by endogenous enzymes after death

Basophilic Readily stains with basic dyes

Baculum (or ***os penis***) The bone present in the penis of many species of carnivores

Calculus, calculi Abnormal concretion of mineral salts, for example dental tartar, kidney stones

Callus Unorganised network of bone around a fracture

Calyx, calices Cup-shaped recess in the kidney pelvis which encloses a medullary pyramid

Cardiac Pertaining to the heart

Cerebellum Caudal division of the brain which moderates functions such as gait

Colloid Thyroid: thick secretion in follicles

Cortex Adrenal: outer, firm, layer comprising the larger part of the adrenal gland. Renal: the smooth textured outer layer of the kidney

Cortical Pertaining to, or emulating from, the cortex

Emphysematous Distension of a tissue, especially lung, by air or gas

Endocardial Pertaining to the lining of the cavities of the heart

Epidermis The outermost, non-vascular layer of the skin

Epithelium The cellular covering of the internal and external surfaces of the body

Focus The chief centre of a morbid process (a small, defined, lesion)

Follicle Spleen: small clusters of dividing lymphocytes. Thyroid: cyst-like units filled with a colloid substance

Foramen magnum Large opening at the base of the skull, connecting the vertebral canal to the cranial cavity

Glomerulus Encapsulated tuft of capillaries in kidney involved in blood filtration

Gonad Testis or ovary

Granuloma, granulomata Tumour-like mass of granulation tissue due to chronic inflammatory process

Haematogenous Disseminated through the blood stream

Hepatic Pertaining to the liver

Hepatocytes Predominant cells of liver

Histology The study of the minute anatomical structure and function of tissues

Hyperplasia Increase in size of organ or tissue caused by increased number of normal cells

Hypertrophy Increase in size of organ or tissue caused by increase in size of existing cells

Hypoplasia Incomplete or under-development of organ or tissue

Hypospadias Congenital defect in development of urethra, often leading to premature ventral opening.

Inspissated Being thickened, dried

Lenticular Pertaining to the lens

Lesion Any pathological or traumatic discontinuity of tissue or loss of function

Leukoencephalopathy Pathology of the white matter of the brain

Medulla Central or inner portion of a gland, for example, adrenal or kidney

Metaplasia Change in the type of adult cells in a tissue to an abnormal form

Microaerophilic Requiring a lower level of oxygen than is present in the atmosphere

Mucosa Mucous membrane lining, such as in the alimentary tract

Necrosis Morphological changes indicative of cell death

Ocular Pertaining to the eye

Oedema Abnormal accumulation of fluid in intercellular spaces

Parenchyma The essential or functional elements of an organ, as opposed to its supporting framework

Pathogen Any disease producing agent

Pathology The structural and functional manifestations of a disease

Pericardium The thin sac-like structure enclosing the heart

Plantar surface Pertaining to the sole of the foot or digit

Perineum The region between and around the anus and genital organs

Purulent Containing or forming pus

Renal Pertaining to the kidney

Retinal dysplasia A developmental abnormality of the eye, resulting in folds and rosettes in the retina

Saprophytic Capable of living on dead or decaying organic matter

Splenic Pertaining to the spleen

Squamous Scaly or plate-like

Subcapsular Beneath a capsule

Sulcus, **sulci** A linear depression, especially one separating the convolutions of the brain surface

Thyroid glands A pair of endocrine glands located caudal to the larynx

Notes and abbreviations

The following abbreviations are used:

AI	Adrenal index
CI	Condition index
CEH	Centre for Ecology and Hydrology (formerly Institute of Terrestrial Ecology)
ELISA	Enzyme-linked immunosorbent assay. A technique that uses labelled antibodies to measure the quantity of a protein
GLM	General linear model
HPA	Health Protection Agency
IUPAC	International Union for Pure and Applied Chemistry
OCs	Organochlorine pesticides
PCR	Polymerase chain reaction, a method of amplifying particular sequences of DNA
PCBs	Polychlorinated biphenyls
PCB congener	One of a set of closely related chemical compounds known as polychlorinated biphenyls
PAS	Periodic acid Schiff
RSPCA	Royal Society for the Prevention of Cruelty to Animals
RTA	Road traffic accidents
sp., spp.	An organism defined to genus level but not a particular species
TEF	Toxic equivalent factor
TEQ	Toxic equivalent
TI	Thyroid index
VLA	Veterinary Laboratories Agency
VWT	Vincent Wildlife Trust

Chemical names

The organochlorine pesticides, their derivates and related compounds referred to in this report are shown by their abbreviated or common name followed by their full chemical name.

Abreviation	Name
Cyclodiene pesticides	
Aldrin	1,2,3,4,10,10-hexachloro-1,4,4*a,*5,8,8a-hexahydro-exo-1,4-endo-5,8-dimethanonaphthalene;
Dieldrin (HEOD)	1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo,exo-5,8-dimethanonaphthalene;
Endrin	1,2,3,4,10,10-hexchloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-endo,endo-1,4:5,8-dimethanonapthalene
Isodrin	1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4:5,8-dimethanonaphthalene
DDT and its derivatives[1]	
op'-DDT	ortho,para'-dichlorodiphenyltrichloroethane
pp'-DDT	para,para'-dichlorodiphenyltrichloroethane
op'-DDE	ortho,para'-dichlorodiphenyldichloroethane
pp'-DDE	para,para'-dichlorodiphenyldichloroethane
op-TDE	ortho,para'-dichloro-bis(p-chlororphenyl)ethane
pp'TDE	para,para'-dichloro-bis(p-chlororphenyl)ethane
HCB	Hexachlorobenzene
HCH[2]	Hexachlorocyclohexane
alpha HCH	alpha-hexachlorocyclohexane
beta HCH	beta-hexachlorocyclohexane
delta HCH	delta-hexachlorocyclohexane
gamma HCH (Lindane)	gamma-hexachlorocyclohexane
TCDD	Tetrachlorodibenzodioxin

[1] The different isomers of DDT and its derivatives, are distinguished by the positioning of the chlorine (Cl) atoms on the two phenyl rings; either in the ortho (*o*) or para (*p*) positions.

[2] HCH has different chiral isomers, which are distinguished by the orientation of the chlorine atoms on the cyclohexane ring. Gamma-HCH is the main constituent of the pesticide lindane.

Appendix 1. List of otters supplied with origin

Reference	Grid Reference	Origin
M153/4/96	ST 180282	Somerset
M161/4/96	SS 672135	Devon
M191/4/96	ST 427255	Somerset
M113/9/96	ST 010420	Somerset
M20/10/96	SS 620199	Devon
M24/10/96	SS 538424	Devon
M108/10/96	SW 794485	Cornwall
M161/10/96	ST 330290	Somerset
M27/11/96	SX 416880	Devon
M104/11/96	SS 255155	Cornwall
M154/11/96	SW 770480	Cornwall
M158/11/96	SX 021629	Cornwall
M217/11/96	SW 517372	Cornwall
M9/1/97	SW 374273	Cornwall
M41/1/97	SY 718909	Dorset
M42/1/97	SS 526348	Devon
M140/1/97	SW 632405	Cornwall
M222/1/97	SW 692230	Cornwall
M48/3/97	ST 044143	Devon
M61/3/97	SW 940480	Cornwall
M113/3/97	SX 525996	Devon
M125/3/97	ST 427257	Somerset
M77/4/97	SX 890976	Devon
M78/4/97	SS 567003	Devon
M80/4/97	SX 916768	Devon
M81/4/97	SS 665152	Devon
M86/4/97	ST 322324	Somerset
M131/4/97	SW 536354	Cornwall
M164/4/97	SX 0049	Cornwall
M53/5/97	ST 484402	Somerset
M7/6/97	SX 115711	Cornwall
M86/6/97	SX 196870	Cornwall
M43/8/97	SX 404859	Devon
M12/9/97	ST 029087	Devon
M111/9/97	ST 461145	Somerset
M193/10/97	ST 376285	Somerset
M194/10/97	SS 741028	Devon
M208/10/97	SX 389549	Cornwall
M115/12/97	ST 494398	Somerset
M120/12/97	SX 965814	Devon
M126/12/97	SX 965815	Devon
M144/1/98	SX 335573	Cornwall
M181/1/98	SX 751933	Devon
M15/2/98	SX 038636	Cornwall
M49/3/98	SS 539438	Devon
M131/3/98	SW 547364	Cornwall

Reference	Grid Reference	Origin
M202/3/98	SX 085840	Cornwall
M17/4/98	SX 113713	Cornwall
M50/4/98	SS 955201	Devon
M138/4/98	SU 477275	Hampshire
M45/5/98	ST 337292	Somerset
M46/5/98	SS 464285	Devon
M47/5/98	SS 467374	Devon
M48/5/98	SX 947881	Devon
M49/5/98	SS 969138	Devon
M50/5/98	SW 893636	Cornwall
M70/5/98	SS 923229	Devon
M98/5/98	ST 031103	Devon
M123/6/98	*	*
M124/6/98	*	*
M132/6/98	SX 569931	Devon
M30/8/98	SX 961860	Devon
M60/8/98	SX 362556	Cornwall
M52/9/98	SS 921353	Somerset
M134/9/98	SY 727957	Dorset
M135/9/98	SX 806416	Devon
M10/11/98	SS 991460	Somerset
M28/11/98	SW 934676	Cornwall
M63/11/98	SX 190770	Cornwall
M55/12/98	SW 760590	Cornwall
M18/1/99	ST 146214	Somerset
M48/1/99	SY 028956	Devon
M49/1/99	SX 080852	Cornwall
M50/1/99	ST 435256	Somerset
M62/1/99	ST 265181	Somerset
M63/1/99	ST 030381	Somerset
M64/1/99	SS 935351	Somerset
M89/1/99	SX 024630	Cornwall
M103/1/99	ST 140261	Somerset
M17/2/99	ST 067408	Somerset
M21/2/99	ST 862113	Dorset
M37/2/99	ST 726128	Dorset
M38/2/99	SS 626248	Devon
M39/2/99	SS 532313	Devon
M40/2/99	SX 859786	Devon
M159/2/99	ST 824111	Dorset
M10/3/99	SX 754653	Devon
M43/3/99	SS 595387	Devon
M26/4/99	SX 141653	Cornwall
M27/4/99	ST 083237	Somerset
M28/4/99	SX 567931	Devon
M94/5/99	SX 977855	Devon
M106/5/99	SW 725180	Cornwall
M116/5/99	SX 519608	Devon
M123/5/99	SS 955125	Devon
M132/5/99	SX 408932	Devon
M6/6/99	SX 113710	Cornwall
M10/6/99	SX 953847	Devon

Reference	Grid Reference	Origin
M34/6/99	SS 655250	Devon
M35/6/99	ST 863133	Dorset
M61/6/99	SW 721221	Cornwall
M109/6/99	SX 323597	Cornwall
M7/7/99	SX 521586	Devon
M101/7/99	SS 568058	Devon
M36/8/99	N/A	N/A
M37/8/99	N/A	N/A
M38/8/99	N/A	N/A
M56/8/99	SX 273 658	Cornwall
M69/8/99	SX 509846	Devon
M93/8/99	SW 895635	Cornwall
M7/9/99	ST 275129	Somerset
M53/10/99	SW 809516	Cornwall
M79/10/99	SX 088528	Cornwall
M89/10/99	ST 198003	Devon
M128/10/99	SS 993142	Devon
M129/10/99	ST 494306	Somerset
M130/10/99	SS 438168	Devon
M32/11/99	SX 214801	Cornwall
M39/11/99	ST 485375	Somerset
M77/11/99	SY 924867	Dorset
M117/11/99	SS 783077	Devon
M11/12/99	SS 727055	Devon
M46/12/99	SS 748054	Devon
M65/12/99	ST 342479	Somerset
M105/12/99	ST 480616	Somerset
M5/1/00	SX 344657	Cornwall
M7/1/00	SX 168746	Cornwall
M19/1/00	ST 417072	Somerset
M27/1/00	SX 153873	Cornwall
M28/1/00	SX 153873	Cornwall
M38/1/00	ST 161309	Somerset
M46/1/00	SY 291921	Devon
M47/1/00	SX 106884	Cornwall
M48/1/00	SX 216851	Cornwall
M55/1/00	SX 362962	Devon
M59/1/00	SS 984432	Somerset
M60/1/00	ST 600414	Somerset
M127/1/00	SX 796615	Devon
M146/1/00	SW 683378	Cornwall
M42/2/00	SX 954846	Devon
M43/2/00	ST 390153	Somerset
M55/2/00	SY 674915	Dorset
M56/2/00	SZ 034996	Dorset
M78/2/00	ST 398432	Somerset
M146/2/00	SS 683464	Devon
M36/3/00	SS 538061	Devon
M67/3/00	SX 602572	Devon
M99/3/00	SX 032739	Cornwall
M9/4/00	SS 583348	Devon
M10/4/00	SU 485288	Hampshire

Reference	Grid Reference	Origin
M21/4/00	ST 085435	Somerset
M76/4/00	SW 895635	Cornwall
M77/4/00	SW 895635	Cornwall
M88/4/00	SS 968140	Devon
M89/4/00	ST 168265	Somerset
M15/5/00	SZ 320960	Hampshire
M19/6/00	SS 373042	Devon
M20/6/00	SY 290974	Devon
M21/6/00	SS 663405	Devon
No number	SX 441587	Devon
M108/6/00	ST 333328	Somerset
M109/6/00	SX 933967	Devon
M12/7/00	SU 552325	Hampshire
M83/8/00	ST 150261	Somerset
M5/9/00	SY 250950	Devon
M93/9/00	SW 426316	Cornwall
M3/10/00	SX 947878	Devon
M35/10/00	SY 923868	Dorset
M61/10/00	ST 829247	Dorset
M62/10/00	SU 150072	Hampshire
M75/10/00	SS 670136	Devon
M92/10/00	SX 120715	Cornwall
M93/10/00	SU 503325	Hampshire
M42/11/00	ST 330399	Somerset
M95/11/00	SS 646098	Devon
M101/11/00	SW 588374	Cornwall
M23/12/00	SZ 163932	Dorset
M52/12/00	SU 546329	Hampshire
M53/12/00	SU 435149	Hampshire
M65/12/00	SW 945605	Cornwall
M100/12/00	SW 940610	Cornwall
M5/310801	SX 168982	Cornwall
M22/161101	SX 037626	Cornwall
M28/061201	SS 923 335	Somerset
M29/061201	SX 357718	Cornwall
M30/061201	SX 869695	Devon
M31/061201	SY 042883	Devon
M34/101201	SU 527127	Hampshire
M43/131201	SU 5590683	Hampshire
M44/171201	SS 575 465	Devon
M45/171201	ST 371178	Somerset
M46/201201	SX 882526	Devon
M47/201201	SX 563932	Devon
M54/040102	SW 756304	Cornwall
M55/040102	SS 685345	Devon
M60/160102	SX 247843	Cornwall
M61/160102	SS 215037	Cornwall
M65/240102	SS 971428	Somerset
M66/240102	ST 024144	Devon
M67/240102	ST 655266	Somerset
M68/240102	ST 388275	Somerset
M73/100202	SW 822473	Cornwall

Reference	Grid Reference	Origin
M76/130202	TQ 055013	Sussex
M78/140202	N/A	N/A
M93/190302	TR 225682	Kent
M102/280302	SX 544732	Devon
M108/170402	SX 218916	Cornwall
M111/240402	SW 737538	Cornwall
M141/170602	SX 185587	Cornwall
M142/170602	SX 155945	Cornwall
M144/210602	SW 653454	Cornwall
M148/240602	SX 368803	Cornwall
M149/240602	SW 476313	Cornwall
M153/240602	SX 973911	Devon
M156/290602	ST 181235	Somerset
M157/290602	SX 357718	Cornwall
M159/290602	ST 947173	Wiltshire
M160/290602	SU 076109	Dorset
M167/120702	SS 262098	Cornwall
M168/120702	SX 312919	Cornwall
M170/120702	ST 181235	Somerset
M174/170702	SX 665944	Devon
M175/170702	SS 444007	Devon
M176/170702	SX 845771	Devon
M196/250702	ST 310400	Somerset
M197/250702	ST 453165	Somerset
M198/250702	SS 942412	Somerset
M200/050802	SS 783077	Devon
M201/080802	SW 700314	Cornwall
M208/150802	SW 732398	Cornwall
M212/190802	ST 693059	Dorset
M213/190802	ST 589346	Somerset
M214/190802	SY 986987	Dorset
M215/190802	SY 926994	Dorset
M216/190802	SS 599240	Devon
M244/030902	SY 969988	Dorset
M249/110902	SX 918666	Devon
M252/090902	ST 171200	Somerset
M253/090902	SX 829752	Devon
M258/090902	ST 165214	Somerset
M259/090902	SS 956123	Devon
M261/230902	ST 483174	Somerset
M262/230902	ST 158409	Somerset
M263/230902	ST 377283	Somerset
M264/230902	ST 161206	Somerset
M267/270902	ST 529745	Somerset
M268/270902	ST 533674	Somerset
M287/031002	SU 487211	Hampshire
M288/031002	SX 058784	Cornwall
M290/051002	SW 547372	Cornwall
M292/141002	ST 261256	Somerset
M293/141002	ST 261257	Somerset
M294/231002	SW 942690	Cornwall
M299/011102	SW 842615	Cornwall

Reference	Grid Reference	Origin
M303/151102	SW 805357	Cornwall
M315/201102	SX 253551	Cornwall
M316/211102	SY 372938	Dorset
M318/251102	SW 721221	Cornwall
M319/011202	SW 552366	Cornwall
M320/021202	SW 640289	Cornwall
M321/021202	SW 537366	Cornwall
M322/031202	ST 232347	Somerset
M329/030103	SX 841015	Devon
M330/060103	SW 577389	Cornwall
M332/100103	SX 331581	Cornwall
M346/130103	SX 728376	Devon
M347/130103	SS 265215	Devon
M353/211102	SX 631518	Devon
M354/211102	SS 865386	Somerset
M355/230103	SS 453250	Devon
M356/230103	SX 857786	Devon
M357/230103	SS 950095	Devon
M358/280103	SS 996440	Somerset
M359/280103	ST 221283	Somerset
M360/280103	ST 515228	Somerset
M361/280103	ST 575200	Somerset
M367/310103	SS 964130	Devon
M368/310103	SX 318833	Cornwall
M377/130203	SU 352238	Hampshire
M384/190203	SS 935074	Devon
M385/190203	SU 545328	Hampshire
M386/190203	SU 459473	Hampshire
M387/200203	SW 685203	Cornwall
M388/200203	ST 253343	Somerset
M391/200203	SS 979457	Somerset
M392/200203	ST 325072	Somerset
M397/260203	SS 996439	Somerset
M398/260203	SX 596969	Devon
M399/260203	SS 503133	Devon
M400/260203	SS 444007	Devon
M402/050303	SX 194967	Cornwall
M407/140303	SW 982734	Cornwall
M414/240303	ST 238258	Somerset
M423/280303	SX 907903	Devon
M424/280303	SS 986447	Somerset
M430/040403	SX 678413	Devon
M450/220503	SS 561466	Devon
M452/290503	ST 005113	Devon
M453/290503	ST 259926	Devon
M454/290503	ST 416426	Somerset
M460/050603	SO 135270	Powys [w]
M470/090603	SW 906597	Cornwall
M495/200703	SW 748472	Cornwall
M500/290703	SU 069369	Wiltshire
M501/290703	SX 655560	Devon
M504/310703	SX 432720	Cornwall

Reference	Grid Reference	Origin
M513/130803	SU 499209	Hampshire
M517/190803	ST 310398	Somerset
M518/190803	SS 996439	Somerset
M524/060903	SU 570323	Hampshire
M525/080903	SW 862655	Cornwall
M549/240903	SS 920220	Devon
M550/240903	ST 554685	Somerset
M551/240903	ST 041103	Devon
M557/021003	SX 109536	Cornwall
M561/071003	SY 917864	Dorset
M562/071003	SZ 130998	Dorset
M573/151003	SX 067768	Cornwall
M575/221003	ST 929009	Dorset
M580/301003	SX 832452	Devon
M582/301003	SX 848764	Devon
M587/041103	SW 855456	Cornwall
M592/111103	SX 252551	Cornwall
M596/121103	SX 188606	Cornwall
M600/201102	ST 392315	Somerset
M601/201103	SS 567374	Devon
M602/201103	SX136736	Cornwall
M625/171203	ST 132204	Somerset
M624/161203	SW 773374	Cornwall
M608/271103	SU 153026	Hampshire
M609/011203	SW 670300	Cornwall
M613/031203	SX 095697	Cornwall
M614/031203	SY 281980	Devon

KEY

N/A = Data not available.
* = Submitted by Vincent Wildlife Trust. One cub originated from Devon and the other from Scotland.
Powys ^{W} = Specimen via RSPCA but from Wales.

We are The Environment Agency. It's our job to look after your environment and make it **a better place** – for you, and for future generations.

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The Environment Agency. Out there, making your environment a better place.

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