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POST MORTEM PROTOCOL FOR OTTERS

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1 INTRODUCTION

Otters found dead in the field, or which die in captivity, are of great research value. However, in order to obtain maximum information it is important that carcases are handled correctly, and that post mortem examinations are carried out in a consistent, detailed and methodical way. This sub-chapter describes a post mortem protocol designed to give optimal results, and also to ensure that meaningful comparisons can be made when carcases are examined in different laboratories.
Ideally, carcases should be examined immediately after being found. This is often not possible in which case they should be placed in a refrigerator (4-6°C), but for no more than three days. Where it is apparent that a carcases cannot be examined within this timescale they should immediately be placed in a deep freeze (-18° to -20°C). Freezing however, produces gross and histopathological alterations, thus reducing the value of the post mortem examination. All post mortem observations, laboratory results and details of tissues stored in deep freeze or in preservative should be recorded in a consistent way on standard forms.

Appendix I is an example of the form used by the author to record organ weights, etc.
Provided the data is recorded the precise layout is not important and individual investigators will have their own preferences.

Post Mortem Protocol

1. Give the carcase a unique laboratory reference number. This number must be used to identify all samples taken from the carcase.

2. Record the date when the otter was found, location (with National Grid Reference if possible), name and address of finder, and apparent cause of death.

3. Record the date of the post mortem examination and the names of participating veterinarians.

4. Weigh the carcase (dry) to the nearest 100g for adults and to the nearest 10g for cubs.

5. With the animal on its back, and using a rigid rule, measure the length from nose to anus and from anus to tail tip. In the case of road traffic accident specimens check that the spine is not fractured, as this can give erroneous results.

6. If radiographic facilities are available consider making x-ray plates, especially if shooting or unexplained trauma is suspected. X-rays are also very helpful in diagnosing cases of hydrocephalus. Scan the carcase for the presence of a microchip.

7. Examine all external features, checking for scars and bite wounds, especially to the head, feet and perineum. Check teeth for wear, calculus formation, abscesses and fractures. Note skin condition and condition of foot pads and claws. Examine eyes and ears. Record sex, noting position/development of testicles and appearance of *os penis*. Place any external parasites in 70% ethanol.

8. Make a mid-line incision from the point of jaw to the anus. Reflect skin and observe fat deposits over flanks. Record extent of fat on a scale of 1 to 3. Remove fat samples (10-20g), wrap in aluminium foil and place in deep freeze. In the case of females observe mammary gland development and check for presence of milk/colostrum.

9. Remove lower rib cage and abdominal wall. Observe organs *in situ*. If any appear abnormal consider taking samples for bacteriological examination. Photograph any abnormalities, remembering to include the case reference number in the photograph.

10. Remove and weigh all organs, including thyroid glands, thymus, adrenal glands and pancreas. Weigh small organs to two decimal places.

11. Where resources permit, place a section of all tissues in buffered formal saline (BFS)* for histological examination. Samples should not exceed 5mm in thickness, except lung which may be up to 10mm in thickness. Small organs, such as thyroid glands, may be fixed whole. Any tissues showing pathological lesions should always be placed in fixative. If there is a history of the animal having shown neurological signs place at least one side of the brain in BFS.

12. If organs show lesions suggestive of a viral infection, e.g., canine distemper or Aleutian disease place small, uncontaminated, samples in sterile containers and hold at 4°C prior to contacting a virology reference laboratory for their advice on storage and handling. If this is not possible place samples directly in deep freeze.

13. In order to minimise contamination, delay opening the alimentary tract until all other organs have been examined. Examine, and if possible identify, any stomach contents before placing them in deep freeze. Always freeze stomach contents in cases of suspected poisoning.

14. Cut both kidneys longitudinally, using at least two parallel cuts, and check for renal calculi. If present, place calculi in a clean bijou bottle (do not place in 10% BFS).

15. Place duplicate samples (approximately 20g) of liver, kidney, skeletal muscle and brain in aluminium foil and transfer to deep freeze for future toxicological examination.

16. If the otter is freshly dead, place approximately 20g of liver in deep freeze for vitamin A analysis. This should be carried out within one month.

17. For genetic analysis collect a piece of kidney or tongue (approximately 5g). Store either in 95% ethanol or, without preservative, in deep freeze. Take care to use clean instruments on uncontaminated surfaces.

18. Remove at least one upper incisor tooth for age determination. This may be stored frozen or in BFS.

19. If possible collect a serum sample for future antibody studies. Pericardial fluid, if available, is a good alternative. Hold in deep freeze.

20. If the animal is pregnant, or if neonates/foetuses are submitted, the weight, sex and crown – rump length should be recorded. It is particularly important to look for evidence of developmental defects, e.g. anophthalmia, hydrocephalus, cleft palate.

Any abnormal looking organs, including placenta, should be cultured for evidence of bacterial infection. Samples should also be placed in BFS. If possible examine liver for vitamin A status.

*BFS : This is usually 10% but concentrations between 5 and 10% are satisfactory.

2 DISEASES

2.1 Non-infectious Diseases

Traumatic injuries, principally caused by road traffic accidents and intra –specific aggression are common (SIMPSON, 1997). Bite wounds are mostly to the face, feet and anus/genitals (SIMPSON and COXON, 2000). In some cases bites may result in fracture of the baculum (os penis) (STEPHENS, 1957). In *L. canadensis*, defective development of the baculum, as well as small or missing testes, has been linked to polychlorinated hydrocarbon pollutants (HENNY, GROVE and HEDSTROM, 1996). A cryptorchid otter in Cornwall had a nil detectable hepatic Vitamin A level (SIMPSON *et al.,* 2000). Hydrocephalus has been recorded in cubs, but the cause is obscure (GREEN, 1998). Five out of thirteen otters that died following an oil spill in Shetland were shown to be suffering from haemorrhagic gastroenteritis, believed to be due to ingestion of oil (BAKER *et al.*, 1981).

Urolithiasis is common, especially in captive otters (STEPHENS, 1957; KEYMER, LEWIS and DON, 1981). Salivary calculi, or sialolyths, have been reported in a number of otters in South West England (SIMPSON, 1998) and in a single case from Shetland (BAKER *et al.*, 1981). As with urolithiasis, the aetiology is unknown. Gall stones, or choleliths, have been noted by a number of investigators but their significance is obscure (MADSEN *et al.*, 1999; WELLS, KEYMER and BARNETT, 1989). Cases of cystic or/and convoluted uteri have been recorded in Norway, England and Denmark and although they appear pathological this is not proven (HEGGBERGET, 1988; SIMPSON, 1997; ELMEROS and MADSEN, 1999).

Blindness was reported to be common in otters in England between 1957 and 1980. One or both eyes were affected, appearing white, but they were not examined by a pathologist (WILLIAMS, 1989). A similar case has been reported recently in Denmark (MADSEN *et al.*, 1999). The precise nature of the lesion in both countries remains uncertain. However, lenticular cataracts were seen in a single case in Norfolk, England (WELLS *et al.*, 1989). Recent investigations in South West England showed clear evidence of retinal dysplasia in approximately 12% of cases and suspected lesions in a further 25% (WILLIAMS, FLINDALL and SIMPSON, 1998).

Otters have been observed showing signs of inco-ordination/disorientation in Ireland and England (MASON and O'SULLIVAN, 1992; WELLS *et al.*, 1989) but neurohistological examinations were either not carried out or no lesions were seen.

Adrenal hyperplasia was reported in a single case in Norfolk (KEYMER *et al.*, 1988) and in a number of otters in South West England. In the latter cases it appeared that males dying of bite wounds and females in late pregnancy/lactating were most likely to be affected (SIMPSON, 1997). However, although stress may be implicated there was a positive correlation between adrenal size and hepatic concentration of some PCB congeners (SIMPSON, 1998). Adrenal aplasia, together with renal aplasia, has been reported in *L. canadensis* in the USA and appears to be linked to levels of polyhalogenated hydrocarbons in the environment (HENNY *et al.*, 1996).

Chronic mercury poisoning has been suspected in otters in Shetland (KRUUK and CONROY, 1991) and high tissue levels have also been recorded in England (MASON, LAST and MACDONALD, 1986). The highest levels in these cases were similar to those seen in experimental poisoning in *L. canadensis* (O'CONNOR and NIELSEN, 1981). Unfortunately, brains were not examined histologically.

2.2 Infectious Disease

There is little evidence of significant infectious disease in wild otters. STEPHENS (1957) referred to a case of tuberculosis in Cornwall, England but the organism was not typed. More recently *Mycobacterium avium* ssp. *avium* was shown to be the cause of massive lesions involving the mesenteric lymph nodes in an otter in Scotland (A. PATTERSON, *pers. comm.*). Small greyish granulomata, which may resemble those of tuberculosis, are sometimes seen in the lungs. These are due to inhaled spores of the fungus *Emmonsia* sp. The condition is referred to adiaspiromycosis and is common in otters in England (SIMPSON and GAVIER-WIDEN, 2000) and in Finland (RUDBACK and STJERNBERG, 1998). Other bacterial infections occasionally recorded are pseudotuberculosis, caused by *Yersinia pseudotuberculosis* (KEYMER, 1992) and salmonellosis. *Salmonella binza* was isolated from the gut of an otter in Norfolk and could possibly have been derived from poultry. *S. enteritidis*, phage type 6, caused fatal gastroenteritis in a captive Asian small clawed otter (*Aonyx cinerea*) which had been fed on day old chicks (V. R. SIMPSON, *unpublished data*) and *S. enteritidis* was also isolated from a wild otter in Russia (BENKOVSKII, GOLOVINA and SCHERBINA, 1973).

An otter which had apparently died after eating toads had multiple haemorrhages in the lungs and *Aeromonas hydrophila* was isolated on culture (SIMPSON and RULE, *unpublished data*). The same organism was isolated from the heart and lungs of an otter which died from severe adiaspiromycosis (SIMPSON and GAVIER-WIDEN, 2000).

Leptospirosis has been suggested as a possible cause of jaundice in otters (KEYMER, 1992). However, there is, as yet, no supporting evidence for this condition in otters, and histological examination of a large numbers of livers and kidneys from South West England showed no lesions suggestive of leptospirosis (SIMPSON, 1998).

As yet unnamed *Brucella* sp. has been isolated from otters, as well as various pinnipeds and cetaceans, in Scotland (FOSTER *et al.*, 1996). The significance of this isolate is as yet uncertain. *Plesiomonas shigelloides* was implicated as a probable cause of abortion in an otter foetus in Scotland (WEBER and ROBERTS, 1989).

Viral infections of otters appeared to be very uncommon. Although there are records of canine distemper affecting captive otters in Germany (GEISEL, 1979), and distemper virus inclusion bodies have been seen in otherwise healthy wild otters in Denmark, there do not appear to be reports of it causing clinical disease in wild otters. There is a single record of rabies in a wild otter, also in Germany (WILHELM and VOGT, 1981). A tentative diagnosis of Aleutian disease was made histologically on an otter from Norfolk, England (WELLS *et al.*, 1989). Feline infectious peritonitis has been suspected in a captive *A. cinerea* (VAN de GRIFT, 1976).

Although various parasites have been recorded in otters there is little evidence that they cause disease. Infection of *L .canadensis* with the kidney worm *Dioctophyme renale* is not uncommon in North America and the parasite has been recorded in *L. lutra* in the UK (CORBET and HARRIS, 1991). An unidentified strongyle larva was seen histologically in the renal pelvis of an otter from South West England (SIMPSON, 1998). Another animal in the same study had *Sarcocystis* sp. in the external eye muscles. In a study in Denmark *Angiostrongylus vasorum* larvae were identified in the lungs of a single otter (MADSEN *et al.,* 1999).

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APPENDIX 1

Otter *Post Mortem* Data – sample record sheet

Name of laboratory:

Case Reference No: **Pathologist**

Total Length:	cm		Date Submitted:
Nose to anus:	cm		Date of PM:
Anus to Tail:	cm		Sex:
Body Weight:	kg		Fresh or Frozen:

Organ	Weight (gm)	Histol.	Freeze	Notes/special instructions
Heart				
Liver				
Spleen				
Right Kidney				
Left Kidney				
Right Thyroid				
Left Thyroid				
Right Adrenal				
Left Adrenal				
Lung				
Cardiac Thymus				
Pancreas				
Right testis				
Left testis				
Foot Pad	———		———	
Eye/s	———		———	
Fat	———	———		
Muscle	———			
Uterus/Gonads	———		———	
Brain/spinal cord	———			
Salivary Gland	———		———	
Bladder	———		———	
Stomach Contents		———		
Rib/bone	———	———		
Incisor Tooth	———		———	
Blood/serum	———	———		
Liver: Vitamin A	———	———		
Urine	———	———		