Exxon Valdez Oil Spill State/Federal
Natural Resource Damage Assessment
Final Report

Pathological Studies of Sea Otters

Marine Mammal Study 6-11
Final Report

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Study History: Marine Mammal Study 6 (MM6), titled *Assessment of the Magnitude, Extent and Duration of Oil Spill Impacts on Sea Otter Populations in Alaska*, was initiated in 1989 as part of the Natural Resource Damage Assessment (NRDA). The study had a broad scope, involving more than 20 scientists over a three year period. Final results are presented in a series of 19 reports that address the various project components. Earlier versions of components of this report were included in the NRDA Draft Preliminary Status Reports for MM6 (November 1990--"Section 11 - Summary of Necropsies and Histopathological Examination"; November 1991--"Appendix A - Histopathology"). Results presented in this report have been published separately, in a book chapter (Lipscomb, T.P., R.K. Harris, A.H. Rebar, B.E. Ballachey and R.J. Haebler. 1994. Pathology of sea otters. In: T.R. Loughlin, Editor. Marine Mammals and the *Exxon Valdez*. Academic Press), and in two journal articles (Lipscomb, T.P., R.K. Harris, R.B. Moeller, J.M. Pletcher, R.J. Haebler and B.E. Ballachey. 1993. Histopathologic lesions in sea otters exposed to crude oil. Vet. Pathol. 30:1-11; Rebar, A.H., T.P. Lipscomb, R.K. Harris and B.E. Ballachey. 1995. Clinical and clinical laboratory correlates in sea otters dying unexpectedly in rehabilitation centers following the *Exxon Valdez* oil spill. Vet. Clin Path. 32:346-350).

Abstract: Following the *Exxon Valdez* oil spill, sea otters were captured and taken to rehabilitation centers. Oil exposure was assessed by visual examination on arrival at the centers. Records of 21 oiled otters that died within 10 days of arrival at the centers were reviewed to define the laboratory abnormalities and clinical syndromes associated with these deaths. The most common terminal syndrome was shock, characterized by hypothermia, lethargy and often hemorrhagic diarrhea. Accompanying laboratory abnormalities included leukopenia, lymphopenia, anemia, azotemia, hyperkalemia, hypoproteinemia/hypoalbuminemia, elevated serum transaminases, and hypoglycemia. Heavily oiled otters developed shock more rapidly and had greater numbers of laboratory abnormalities, suggesting that oil exposure was an important contributing factor. Tissues from 51 oiled and 6 unoiled sea otters that died in the centers were examined histologically. Pulmonary interstitial emphysema, gastric erosion and hemorrhage, centrilobular hepatic necrosis, and hepatic and renal lipidosis were common in oiled otters and were absent or uncommon in unoiled otters. Histologic examinations were performed on tissues from 5 oiled otters found dead shortly after the spill. Hepatic and renal lipidosis was common, and pulmonary interstitial emphysema was found. Necropsies were performed on 214 sea otters that were collected and frozen following the oil spill. Pulmonary interstitial emphysema and gastric erosion and hemorrhage were common in oiled animals, and were less frequent in unoiled animals. Tissues from 6 sea otters collected from a non-oiled area were examined, and none of these lesions were found. We conclude that pulmonary interstitial emphysema, gastric erosion and hemorrhage, centrilobular hepatic necrosis, and hepatic and renal lipidosis were associated with exposure to crude oil in sea otters.

Key Words: carcasses, *Enhydra lutris*, *Exxon Valdez*, mortality, oil spill, sea otter.

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EXECUTIVE SUMMARY

Following the *Exxon Valdez* oil spill in Prince William Sound, Alaska, sea otters (*Enhydra lutris*) that appeared oiled, were in danger of becoming oiled, or were behaving abnormally were captured and taken to rehabilitation centers. Oil exposure was assessed by visual examination on arrival at the centers. Clinical records of 21 oiled sea otters that died during the first 10 days of rehabilitation were reviewed to define the laboratory abnormalities and clinical syndromes associated with these deaths. The most common terminal syndrome was shock, characterized by hypothermia, lethargy and often hemorrhagic diarrhea. Accompanying laboratory abnormalities included leukopenia with increased number of immature neutrophils, lymphopenia, anemia, azotemia, hyperkalemia, hypoproteinemia/ hypoalbuminemia, elevations of serum transaminases, and hypoglycemia. Heavily oiled otters developed shock more rapidly and had greater numbers of laboratory abnormalities, suggesting that exposure to oil was an important contributing factor. Tissues from 51 oiled sea otters and from 6 unoiled sea otters that died in rehabilitation centers were examined histologically. Pulmonary interstitial emphysema, gastric erosion and hemorrhage, centrilobular hepatic necrosis, and hepatic and renal lipidosis were common in oil exposed otters and were absent or uncommon in unoiled otters. Histologic examinations were performed on tissues from 5 sea otters found dead with external oil present shortly after the spill. Hepatic and renal lipidosis was common, and pulmonary interstitial emphysema was found. Necropsies were performed on 214 sea otters that had been collected and frozen in the period following the oil spill. Histologic examination was not performed. Pulmonary interstitial emphysema and gastric erosion and hemorrhage were common in animals with external oil present; these lesions were found much less frequently in animals without detectable external oil. Tissues from 6 apparently normal sea otters collected from an area not affected by the oil spill were examined histologically, and none of these lesions were found. We conclude that pulmonary interstitial emphysema, gastric erosion and hemorrhage, centrilobular hepatic necrosis, and hepatic and renal lipidosis were associated with exposure to crude oil in sea otters.

INTRODUCTION

In the months following the *Exxon Valdez* oil spill, over 900 sea otters (*Enhydra lutris*) from oil spill-affected areas are known to have died. Carcasses collected from these areas and otters that died in rehabilitation centers are included in this figure (Doroff et al. 1993). The actual number that died was certainly much greater.

Within days of the spill, Exxon funded an effort to rehabilitate oil-contaminated sea otters (Davis 1990). Initially, clinical veterinarians working in the rehabilitation effort performed partial necropsies on some of the otters that died. Soon, veterinary pathologists from the University of Alaska and the Environmental Protection Agency provided assistance. Later, rehabilitation centers were constructed and other veterinarians with special training in pathology were hired by Exxon to provide diagnostic support.

In late April 1989, veterinary pathologists from the U.S. Fish and Wildlife Service (Service) assumed responsibility for pathologic evaluation of oil spill-affected sea otters. The Service requested assistance from veterinary pathologists of the Armed Forces Institute of Pathology (AFIP) in June 1989. Eventually, as part of the Natural Resources Damage Assessment, AFIP veterinary pathologists were asked to carry out histopathological studies of the tissue specimens collected by all parties and to perform necropsies on carcasses that had been collected and frozen. A veterinary clinical pathologist was requested to assess hematology and clinical chemistry findings in otters that had been held in the rehabilitation centers.

In spite of the best efforts of many dedicated people working under extremely difficult conditions, there are significant limitations in the pathological studies. The absence of a detailed necropsy protocol and of full documentation of necropsy findings during the first several weeks after the spill caused important data to be lost. Often, samples of all major organs were not collected. In some cases, no necropsy report was available. Specimens for toxicologic analysis for petroleum hydrocarbons were not consistently collected. The absence of a detailed toxicology protocol suggests that toxicologic samples may not have been collected properly. The lack of a consistent numbering system for identification of specimens caused major problems; some samples were useless because they could not be identified. Because of inclement weather and the remote locations of the rehabilitation centers, many blood samples could not be transported to laboratories quickly enough to prevent significant deterioration of the samples. Thus, in a number of cases, data could not be used because significant deterioration of the specimens was considered likely to have occurred. Since more than one laboratory was used to analyze blood samples, problems with comparability of results were encountered. The laboratory tests were performed to aid the clinical veterinarians in diagnosis and treatment of individual animals, not as part of a consistent protocol; thus, there is variation in the amount and type of laboratory data available for each otter. These problems illustrate the need for development of contingency plans that include detailed protocols before disasters occur.

Studies of Sea Otters that Died in Rehabilitation Centers

Following the oil spill, sea otters that appeared oil contaminated, were considered to be in danger of becoming oil contaminated, or that behaved abnormally were captured and

transported to rehabilitation centers (Bayha and Hill 1990). On arrival, oil exposure was assessed by visual examination. Degree of oil contamination was graded according to the following criteria: oil covering greater than 60% of the body - heavily contaminated; oil covering 30-60% of the body - moderately contaminated; oil covering less than 30% of the body or a light sheen on the fur - lightly contaminated; if no oil was visible, otters were considered uncontaminated.

Clinical, Hematologic, and Blood Chemistry Studies

Clinical records and laboratory data of 21 oil-contaminated sea otters that died within 10 days of arrival at the rehabilitation centers were examined. Seven were heavily contaminated, 5 moderately contaminated, and 9 lightly contaminated. Selection of these otters was based on completeness of clinical records, availability of laboratory data from acceptable samples, and availability of results of histopathologic examination.

Clinically, shock was the most common terminal syndrome and was characterized by hypothermia, lethargy and often hemorrhagic diarrhea. This syndrome was rarely observed on arrival at the centers but generally developed within 48 hours in heavily and moderately oil-contaminated otters. Lightly contaminated otters generally developed shock during the second week after arrival. A high proportion of otters in all 3 grades of oil contamination had seizures at or near the time of death. Anorexia was also relatively common.

Blood values were compared to reference ranges established for normal sea otters from southeastern Alaska. Abnormalities were interpreted according to conventions used for the interpretation of laboratory data in dogs and other carnivores/omnivores (Duncan and Prasse 1986).

The most common hematologic abnormality in otters of all grades of oil contamination was leukopenia (decreased white blood cell count) characterized by decreased mature neutrophils with increased numbers of immature neutrophils (degenerative left shift) and decreased lymphocytes. Degenerative left shifts indicate severe inflammation. Diarrhea with bowel stasis is a possible cause of the inflammation in these otters. The lymphopenia reflects either systemic stress or the influence of glucocorticoids administered by clinical veterinarians. Severe stress results in release of large amounts of glucocorticoids from the adrenal cortices which cause sequestration and possible destruction of circulating lymphocytes. Anemia was also relatively common but could not be further characterized from the available data.

The main clinical chemistry abnormalities were azotemia, hyperkalemia, hypoproteinemia/hypoalbuminemia, increased serum transaminases indicative of hepatocellular leakage, and hypoglycemia.

Azotemia (the accumulation of nitrogenous waste products in the blood) was the most common clinical chemistry abnormality, and its prevalence was similar in otters of all 3 grades of oil contamination. Azotemia indicates inadequate kidney function, which may result from a primary kidney problem (renal azotemia), insufficient blood and oxygen supply to the kidney (prerenal azotemia), or obstruction of the lower urinary tract (postrenal azotemia). Unfortunately, urine specific gravities were not available to aid in differentiation of prerenal from renal azotemia. Clinical findings did not support postrenal azotemia. The clinical histories suggest that shock and hemorrhagic diarrhea may have caused poor renal blood supply resulting in prerenal azotemia. In the few animals that developed true renal

azotemia (serum urea nitrogen values greater than 200 mg/dl), long-term reduced renal blood supply probably led to primary terminal renal injury.

Hypoproteinemia/hypoalbuminemia (abnormally low blood protein and albumin) and hyperkalemia (abnormally high blood potassium) were found less commonly than azotemia but were probably also related to diarrhea and shock. Possible causes of hyperkalemia in these otters include release of potassium from dying cells and acidosis. Acidosis is a condition that causes decreased blood pH. Severe diarrhea is a frequent cause of acidosis. Protein loss due to diarrhea is the most likely cause of the hypoproteinemia/ hypoalbuminemia.

Increased hepatocellular leakage-associated serum transaminases were only slightly less common than azotemia. Hepatocellular leakage might have been caused by primary hepatotoxicity, but may also have been caused by anorexia. In fasting associated with anorexia, tissue stores of fat are mobilized and transported to the liver resulting in increased cell membrane permeability of hepatocytes with leakage of transaminases into the blood. Anorexia is also the most likely cause of the hypoglycemia which probably caused the seizures.

Histopathologic Studies

Histopathologic examinations were performed on 51 oil-contaminated and on 6 uncontaminated sea otters that died in rehabilitation centers. Pups were excluded because of the small number available. Samples from 6 apparently healthy adult sea otters that were killed by gunshot in an area not affected by an oil spill as part of unrelated research were used as normal controls. Of the 51 oil-contaminated otters that died in rehabilitation centers, 16 were heavily contaminated, 13 were moderately contaminated, and 22 were lightly contaminated. Complete sets of tissues were not available from all otters. A more detailed description of this study is reported elsewhere (Lipscomb et al. 1993).

Among oil-contaminated otters that died in the centers, interstitial pulmonary emphysema was the most common lesion. It was found in 11/15 (73%) heavily contaminated, 5/11 (45%) moderately contaminated, and 3/20 (15%) lightly contaminated otters. Histologically, the lesion appeared as expanded areas of clear space within the interlobular septa of the lung (Fig. 1).

Gastric erosions were found in 2/14 (14%) heavily contaminated, 7/9 (78%) moderately contaminated, and 4/17 (24%) lightly contaminated otters. Microscopically, the erosions consisted of discrete, 1-3 mm diameter areas of coagulative necrosis that affected superficial to mid-level gastric mucosa (Fig. 2).

Hepatic lipidosis occurred in 8/16 (50%) heavily contaminated, 5/12 (42%) moderately contaminated, and 1/19 (5%) lightly contaminated otters. The lesion was characterized by variably sized, single to multiple, round, sharply delineated, unstained intracytoplasmic vacuoles in hepatocytes (Fig. 3). The distribution of the lesion was predominantly periportal, but in severe cases, it was diffuse. Renal lipidosis was found only in otters that also had hepatic lipidosis. It was present in 10/42 (24%) oil-contaminated otters that died in rehabilitation centers. The microscopic appearance was characterized by single or multiple, variably sized, round, discrete, unstained intracytoplasmic vacuoles within proximal and distal renal tubular epithelium (Fig. 4). The intracytoplasmic vacuoles in both liver and kidney stained red with oil red O indicating the presence of lipid.

Centrilobular hepatic necrosis was seen in 4/16 (25%) heavily contaminated, 3/12 (25%) moderately contaminated, and 4/19 (21%) lightly contaminated otters. In affected livers, centrilobular hepatocytes exhibited nuclear pyknosis, karyorrhexis, karyolysis, and increased eosinophilia of cytoplasm with preservation of basic cell shape (Fig. 5).

Among the 6 uncontaminated otters that died in rehabilitation centers, one had gastric erosions, one had hepatic lipidosis and multifocal hepatic necrosis, and one had focally extensive hepatic necrosis. The remaining otters included one with peritonitis caused by small intestinal perforation. Another had mild acute enteritis and mild subacute hepatitis of undetermined cause. The remaining otter in this group had no significant histologic lesions and no necropsy report was available.

The 6 apparently healthy otters collected from an area that had not been affected by an oil spill had no significant histologic lesions. Various incidental lesions were found in otters in each of the 3 groups.

Possible Mechanisms of Lesion Development and Clinicopathologic Correlations

The most common serious complication of petroleum hydrocarbon ingestion in human beings and animals is aspiration pneumonia (Eade 1974, Richardson and Pratt-Thomas 1951, Rowe et al. 1951). Aspiration pneumonia occurs when large amounts of foreign material enter the lungs through the airways. Oil-contaminated sea otters attempt to remove the oil by grooming with the mouth (Siniff et al. 1982), which would seem to provide ample opportunity for aspiration. However, no evidence of aspiration pneumonia was found in oil-contaminated sea otters.

Interstitial pulmonary emphysema, which is the accumulation of bubbles of air within the supportive connective tissues of the lungs, was remarkably common in oil-contaminated sea otters. Dyspnea was prevalent in oil-contaminated sea otters presented to rehabilitation centers. Many of these otters also had subcutaneous emphysema (Williams et al. 1990), which forms by extension of pulmonary emphysema through the mediastinum into muscle fascia and subcutaneous tissue. Rupture of alveolar walls is the usual mechanism by which air enters the pulmonary interstitium. Alveoli may rupture when there is a combination of forced expiration or coughing and bronchiolar obstruction that produces greatly increased pressures within alveoli (Cotran et al. 1989). Sea otters have well-developed pulmonary interlobular septa and thus may be predisposed to the development of interstitial emphysema (Dungworth 1985). Interstitial emphysema has been reported in sea otters with pneumonia (Cornell et al. 1979, Mattison and Hubbard 1969). Pneumonia was not found in the oil-contaminated sea otters. Although not previously reported, inhalation of volatile components of crude oil such as benzene might have damaged alveolar septa and caused the lesion, but other lesions likely to result from inhalation of an irritant vapor, such as interstitial pneumonia, were not found. Another possibility is that small amounts of oil were aspirated and caused coughing and dyspnea that ruptured alveolar walls and forced air into the interstitium, yet the volume of oil that entered the lungs was small enough to be removed without development of pneumonia. Pressure changes that occur during diving may also have been involved. The pathogenesis of interstitial pulmonary emphysema in oil-contaminated sea otters is undetermined.

Gastric erosions were common in oil-contaminated sea otters that died in the centers and were also found in 1/6 uncontaminated sea otters that died in the centers. The most

likely mechanisms by which oil-contaminated sea otters might develop gastric erosions are either because of severe stress or because of a direct effect of the oil on the gastric mucosa. Gastrointestinal erosion/ulceration has been reported in sea otters that died in captivity and in the wild and has been attributed to stress (Mattison and Hubbard 1969, Stullken and Kirkpatrick 1955). Erosions caused by ingestion of corrosive liquids are extensive, but the erosions in these sea otters were small, discrete, and largely confined to the stomach. Thus, the erosions were probably caused by stress. It is unclear whether gastric erosions in oil-contaminated sea otters that died in captivity developed because of stress of capture and captivity or because of stress associated with oil exposure. It is likely that all sources of stress contributed to development of gastric erosions.

Hepatic lipidosis (the accumulation of lipids within hepatocytes) was present frequently in oil-contaminated sea otters that died in the centers and also was found in 1/6 uncontaminated sea otters that died in the centers. Renal lipidosis (the accumulation of lipid within the tubular epithelial cells of the kidney) was somewhat less common and was found only in otters that also had hepatic lipidosis. Potential causes of hepatic and renal lipidosis include toxicity, mobilization of stored fat due to inadequate food consumption, and hypoxia. Hepatic lipidosis caused by hypoxia is primarily centrilobular (Kelly 1985), but the lipidosis in these sea otters was predominantly periportal, indicating that hypoxia is unlikely to be the cause. Experimentally oil-contaminated sea otters had marked increases in activity and metabolic rate with unchanged or decreased time devoted to feeding (Costa and Kooyman 1982, Siniff et al. 1982). Thus, mobilization of stored fat is likely to occur in oil-contaminated sea otters. Anorexia and elevated hepatocellular leakage-associated serum transaminases were common in oil-contaminated sea otters that died in the centers. Some otters with elevated transaminases had hepatic lipidosis. The hepatocellular leakage may have resulted from accumulation of lipid in hepatocytes because of fat mobilization. A direct or metabolite-associated toxic effect may also have caused the hepatorenal lipidosis. Hepatic lipidosis has been reported in rats (Bogo et al. 1982), mice (Gaworski et al. 1982), cattle (Winkler and Gibbons 1973), sheep (Adler et al. 1992), and a ringed seal (Smith and Geraci 1975) exposed to petroleum hydrocarbons, but mechanisms were not determined. Lipidosis of renal tubular epithelium of undetermined cause has been reported in hydrocarbon-exposed rats (Bogo et al. 1982). In the sea otters, no renal lesions were found that would be expected to cause azotemia, and there was no evidence of urinary tract obstruction. Therefore, the azotemia is considered prerenal.

Centrilobular hepatic necrosis, which is death of hepatocytes that surround central veins of the liver, was relatively common in oil-contaminated sea otters that died in rehabilitation centers and was not found in uncontaminated sea otters that died in the centers. Potential causes of centrilobular hepatic necrosis include toxins and conditions that cause hepatic ischemia such as anemia, heart failure, and shock. Other lesions that would support heart failure were not found. Shock was a common syndrome in oil-contaminated otters that died in rehabilitation centers. Anemia was also common. Crude oil ingestion (Leighton 1986) and gastric erosion with hemorrhage are possible causes of anemia, but gastric erosions and centrilobular hepatic necrosis rarely occurred in the same otters, so anemia due to gastric erosion was not a common cause of centrilobular necrosis. It is likely that shock, and in some cases anemia, contributed to centrilobular hepatic necrosis. Toxin-induced hepatic necrosis is also possible. Centrilobular hepatic necrosis of undetermined cause was found by researchers who gave crude oil orally to birds (Leighton 1986). Hepatic necrosis

probably contributed to the increases in hepatocellular leakage transaminases observed in some otters in the rehabilitation centers.

The histopathologic studies failed to identify the cause of the inflammatory stimulus responsible for the degenerative left shift identified in the hematology results.

Studies of Sea Otters that Died in the Wild

Histopathologic Studies

Tissues from 5 oil-contaminated sea otters that were found dead in oil spill-affected areas were examined histologically. One had interstitial pulmonary emphysema and hepatorenal lipidosis. Two others had hepatorenal lipidosis. The remaining two otters had no significant histologic lesions and no necropsy reports were available. The presence of interstitial pulmonary emphysema and hepatorenal lipidosis in non-captive oil-contaminated sea otters and the absence of these lesions in normal controls suggest that these lesions were caused by oil exposure rather than captivity.

Necropsy Studies

Following the oil spill, sea otters carcasses were collected from oil spill-affected areas, placed in plastic bags, and frozen. In the summer of 1990, carcasses were thawed and complete necropsies were performed. Specimens were collected for toxicologic analysis in accordance with an established protocol. Results of toxicologic testing are reported elsewhere in this volume. Histologic examinations were not performed because of the artifacts produced by freezing and thawing of tissues.

A total of 214 of the carcasses examined were judged adequately preserved. Pups were not included because of the small number available. Of these 214 carcasses, 152 were externally oil-contaminated and 62 had no detectable external oil. Among oil-contaminated otters, 100/152 (66%) had interstitial pulmonary emphysema; 83/152 (55%) had gastric erosion and hemorrhage; 64/152 (42%) had both of these lesions. In the uncontaminated group, 13/62 (21%) had interstitial pulmonary emphysema and 4/62 (6.5%) had gastric erosion and hemorrhage. All 4 that had gastric erosion and hemorrhage also had interstitial pulmonary emphysema. Among uncontaminated sea otters, 2 had lesions that contributed to their deaths, one had vegetative valvular endocarditis and the other had a gunshot wound in the thorax. Lungs of both of these otters had interstitial emphysema. A variety of incidental lesions were found in many of the otters.

Emphysematous lungs were characterized by generally diffuse expansion of interlobular septa by clear, round to oblong, gas-filled bubbles that ranged from a few millimeters to 5 cm in diameter (Fig. 6). Pulmonary parenchyma adjacent to affected septa was compressed. Occasionally, the emphysema extended into mediastinum, muscle fascia, and subcutaneous tissues of the neck and back.

Stomachs with erosions contained small to abundant amounts of dark red to black blood (Fig. 7). Numbers of erosions varied from a few to about 50. Pylorus was affected most frequently, but in many cases all regions of the gastric mucosa were affected. The amount of hemorrhage in the lumen generally correlated with the number of erosions.

Rarely, 1 or 2 erosions were present in the duodenal mucosa. The erosions were punctate, round to oval mucosal defects with bright to dark red bases. Diameter ranged from 2 mm to 1 cm and was usually 2 to 4 mm. Other lesions found microscopically in oil-contaminated sea otters such as hepatic and renal lipidosis and centrilobular hepatic necrosis could not be conclusively identified by gross examination alone.

This study confirms the association of interstitial pulmonary emphysema with exposure to crude oil in sea otters. The incidence of emphysema was 3 fold higher in oil-contaminated versus uncontaminated otters. There are several possible explanations for the presence of emphysema in some of the uncontaminated otters. These otters may have been lightly oil-contaminated but were able to remove the oil prior to death. They may have breathed volatile components of crude oil while not coming in contact with liquid crude oil. Their interstitial pulmonary emphysema may have had an unrecognized cause other than crude oil exposure.

The high incidence of gastric erosion and hemorrhage in oil-contaminated sea otters is particularly interesting. This lesion was commonly identified histologically in oil-contaminated sea otters that died in rehabilitation centers, but it was unclear whether stress from oil exposure or from capture and captivity was the cause. This study indicates that gastric erosion and hemorrhage are associated with exposure to crude oil in the absence of capture and captivity.

Data from the necropsies were studied in an attempt to determine cause of death. Sea otters that were oil-contaminated, had one or both lesions associated with crude oil exposure (interstitial pulmonary emphysema and/or gastric erosion and hemorrhage), and that did not have lesions indicative of another possible cause of death, were considered to have strong evidence of death caused by oil exposure. Sea otters that fit these criteria included 123/214 (57%). Those otters that were oil-contaminated and had neither lesions associated with crude oil exposure nor lesions indicative of another possible cause of death were considered to have evidence of death caused by oil exposure. Sea otters that conformed to these criteria comprised 29/214 (14%). Carcasses that did not have detectable external oil, did not have lesions associated with oil exposure, and did not have lesions indicative of another possible cause of death were considered to have an undetermined cause of death. This group consisted of 49/214 (23%). Carcasses that had neither detectable external oil nor lesions indicative of another possible cause of death, but that did have emphysema and/or gastric erosions included 11/214 (5%) and were also considered to have an undetermined cause of death. The uncontaminated sea otter with emphysema and a gunshot wound in its thorax and the uncontaminated sea otter with emphysema and vegetative valvular endocarditis comprised 2/214 (1%) and were considered to have died primarily because of conditions unrelated to oil exposure.

CONCLUSIONS

In spite of significant limitations, these studies represent the largest and most detailed investigation of the pathological effects of an oil spill on a marine mammal species. The findings support some long-held assumptions and bring to light much new information.

Because they lack a thick layer of subcutaneous fat similar to the blubber of pinnipeds and cetaceans, sea otters rely on their pelage for protection from the cold water they inhabit.

Thus, it was suspected that sea otters would be highly vulnerable to oil spills because contact with crude oil would dramatically decrease the insulating properties of their fur, resulting in hypothermia and death (Williams et al. 1988). Indeed, the oil spill had a devastating effect on the sea otters of Prince William Sound, and hypothermia was a major problem in sea otters presented to rehabilitation centers (Williams et al. 1990). Death caused by hypothermia can occur without distinctive gross or histologic lesions.

Clinical, hematologic/blood chemistry, and postmortem findings, combined with previous research, suggest the following scenario: Oil-contaminated sea otters rapidly become hypothermic. They devote themselves to a life or death struggle to remove the oil by grooming. Feeding is drastically curtailed, and energy stores are rapidly depleted. Grooming is marginally effective at best and results in ingestion of crude oil. By unknown mechanisms, exposure to the oil causes interstitial pulmonary emphysema which compromises respiration. Their desperate situation causes a powerful stress reaction. Gastric erosions form as the physiologic effects of stress reach a critical level. Hemorrhage into the gut begins. The combined effects of these factors overwhelm the otters; shock ensues, followed by death.

Some sea otters succumb to hypothermia rapidly, and no lesions form. Others live long enough to develop some or all of the morphological markers that characterize this syndrome: interstitial pulmonary emphysema, gastric erosion and hemorrhage, hepatic and renal lipidosis, and centrilobular hepatic necrosis. Otters that are captured and taken to rehabilitation centers are subjected to additional stressors but are given medical and supportive care.

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Figure 1. Lung; Interstitial emphysema. Interlobular septum is expanded by gas bubbles. Adjacent alveoli are atelectatic.

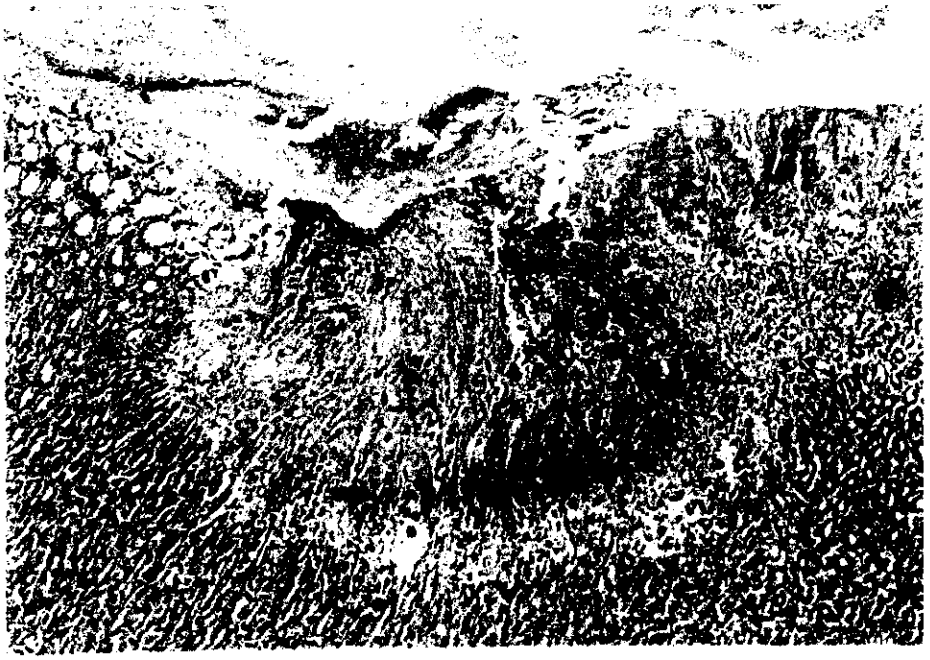

Figure 2. Gastric mucosa; Erosion. Focal area of coagulative necrosis. (Reprinted by permission of Veterinary Pathology from Lipscomb, T. P., Harris, R. K., Moeller, R. B., Pletcher, J. M., Haebler, R. J., and Ballachey, B. E. 1993. Histopathologic lesions in sea otters exposed to crude oil. Veterinary Pathology 30, 6).

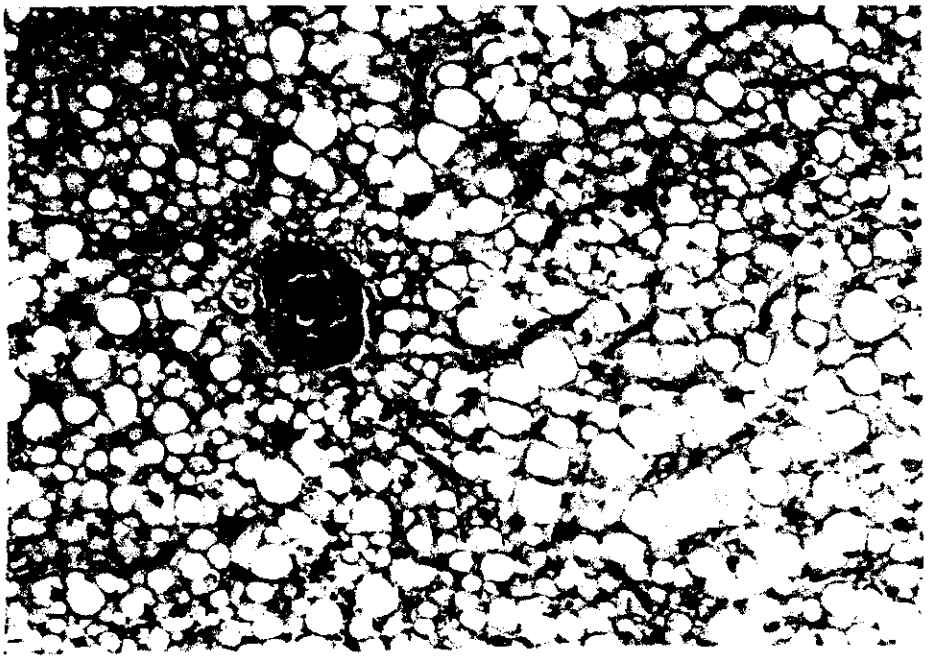

Figure 3. Liver; Lipidosis. Diffuse vacuolation of hepatocytes caused by lipid accumulation. Portal area is at left.

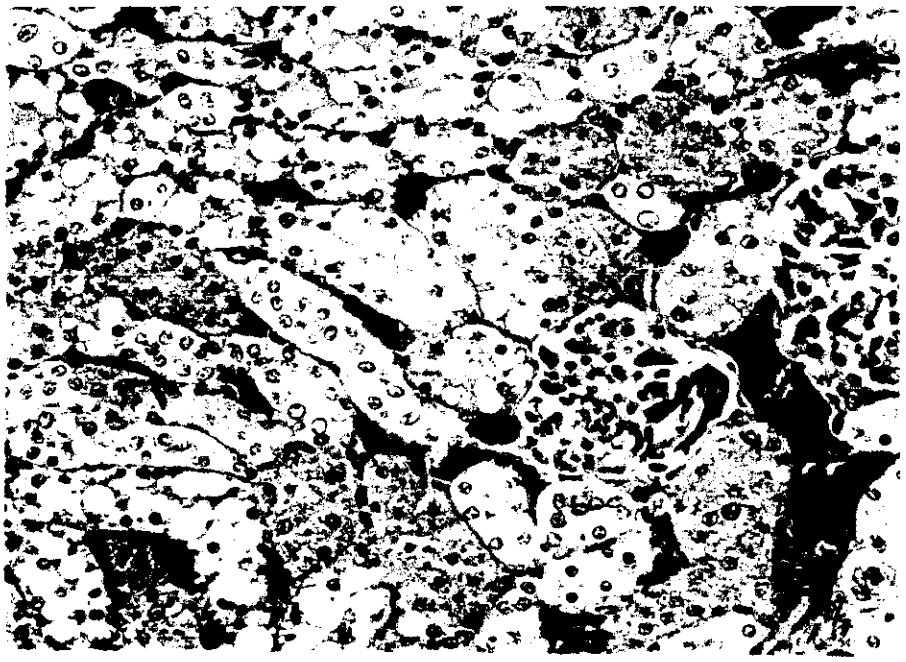

Figure 4. Kidney; Lipidosis. Vacuolation of tubular epithelium caused by lipid accumulation.

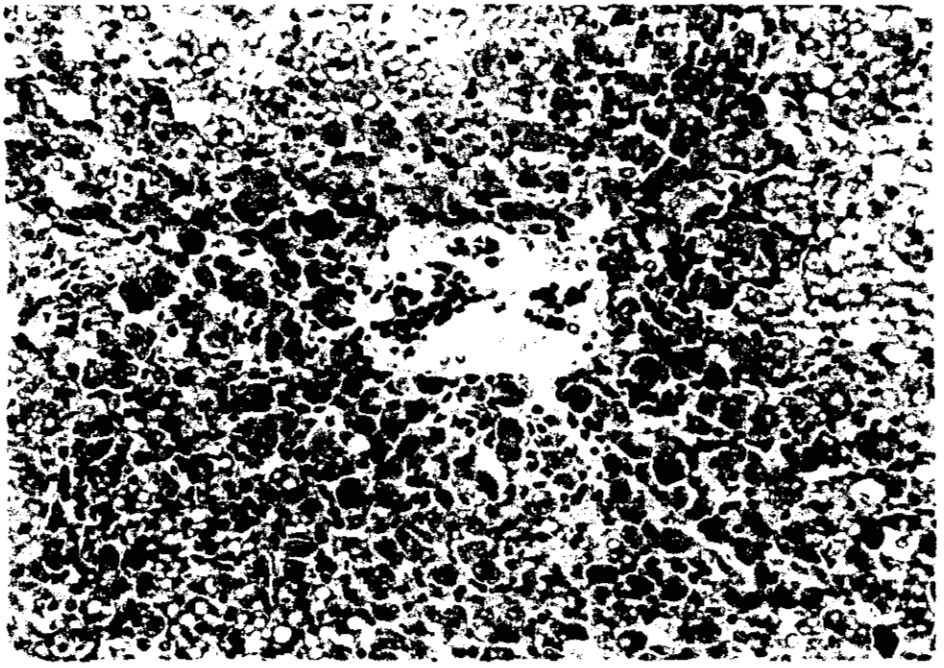

Figure 5. Liver; Centrilobular necrosis and midzonal lipidosis. Central vein is at center. (Reprinted by permission of Veterinary Pathology from Lipscomb, T. P., Harris, R. K., Moeller, R. B., Pletcher, J. M., Haebler, R. J., and Ballachey, B. E. 1993. Histopathologic lesions in sea otters exposed to crude oil. Veterinary Pathology 30, 7).

Figure 6. Lung; Interstitial emphysema. Interlobular septa are expanded by trapped air (arrows). Heart = H and liver = L. (Photograph courtesy of Dr. Terrie M. Williams.)

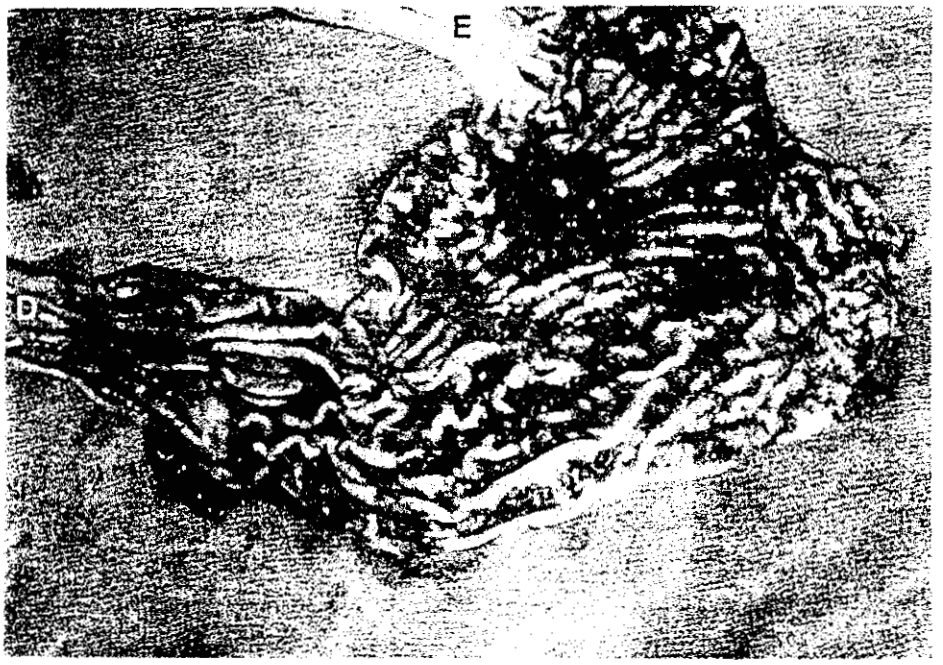

Figure 7. Opened stomach; Gastric hemorrhage. Mucosal surface is covered by dark, partially digested blood. Esophagus = E and duodenum = D.