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

Histopathologic Lesions Associated with Crude Oil Exposure in Sea Otters

Marine Mammal Study 6-10
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--"Section 7(A) - Hematology and Chemistry" and "Appendix A - Histopathology"). A journal articles based on this report has been published (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).

Abstract: 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. 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.

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. 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

On March 23, 1989, the oil tanker *Exxon Valdez* ran aground on Bligh Reef in Prince William Sound, Alaska. The resulting spill of approximately 11 million gallons of North Slope crude oil was the largest in the history of the United States. In the months following the spill, over 1,000 sea otters from oil spill-affected areas are known to have died. The actual number dead was probably much greater.

OBJECTIVES

As part of an effort to determine the effects of the oil spill on sea otters, we examined tissues from otters that died in rehabilitation centers and that were found dead with and without external oil present. We also examined tissues from apparently normal sea otters from an area not contaminated by crude oil.

METHODS

Following the oil spill, sea otters that appeared oiled, were in danger of becoming oiled, or were behaving abnormally were captured and taken to one of several rehabilitation centers. Oil exposure was assessed by visual examination on arrival at the centers. Degree of oiling was graded according to the following criteria: greater than 60% body coverage - heavily oiled; 30-60% body coverage - moderately oiled; less than 30% body coverage or light sheen on fur - lightly oiled. If there was no oil visible, otters were considered unoiled. Oiled otters that died in rehabilitation centers (Group 1), unoiled otters that died in rehabilitation centers (Group 2), and otters that were found dead with external oil present (Group 3) were necropsied by various veterinarians. This fact and the lack of a standard necropsy protocol during the first few weeks after the spill resulted in great variation in the tissues collected. Documentation of necropsy findings ranged from minimal to thorough. In some cases, no necropsy report was available. Only otters with documented oil-exposure assessment were included in this study. Otters that died in rehabilitation centers were collected from oil-contaminated areas of Prince William Sound from 30 March to 17 July 1989 and died between 3 April and 4 August 1989. Animals that were found dead and necropsied were collected from contaminated areas in early April 1989. During the summer of 1989, biologists collected tissues from 6 apparently healthy sea otters (Group 4) that had been killed by gunshot in the waters surrounding the Kuril Islands, Union of Soviet Socialist Republics, as part of unrelated research. Pups were not included in the study because of the small number available.

Tissues were placed in 10% neutral buffered formalin and processed in paraffin. Tissues collected included adrenal gland, aorta, bone marrow, brain, esophagus, eye, heart, intestine, kidney, liver, lung, lymph node, mammary gland, ovary, pancreas, parathyroid, pituitary gland, skeletal muscle, skin, spinal cord, spleen, stomach, testis, thymus, thyroid, tongue, tonsil, trachea, urinary bladder, and uterus. Tissues collected from individual animals varied. Sections were cut at 5μm and stained with hematoxylin and eosin for light-microscopic examination. Selected sections were stained with oil red O.

In July and August 1990, complete necropsies were performed on 214 sea otters that had been collected from oil spill affected areas, placed in plastic bags, and frozen in the period following the spill (Group 5). Presence of external and internal oil was noted in the necropsy reports. Histologic examination was not performed because the tissue had been frozen.

RESULTS

Data on individual Group 1, 2, and 3 otters are presented in Table 1. Numbers of Group 1, 2, and 3 otters of each gender with each of the common lesions are presented in Table 2.

In Group 1 sea otters, pulmonary interstitial emphysema was the most prevalent lesion, being present in 11 of 15 (73%) heavily oiled, 5 of 11 (45%) moderately oiled, and 3 of 20 (15%) lightly oiled animals. Overall, the lesion was present in 19 of 46 (41%) Group 1 otters. It was common in heavily and moderately oiled otters that died within 8 days of arrival at the rehabilitation centers, being present in 16 of 22 (73%) animals. Histologically, the lesion appeared as expanded areas of clear space with rounded contours within the interlobular septa (Fig. 1). Occasionally, adjacent parenchyma was compressed.

Gastric erosions were seen in 2 of 14 (14%) heavily oiled, 7 of 9 (78%) moderately oiled, and 4 of 17 (24%) lightly oiled Group 1 sea otters. Among total Group 1 otters, 13 of 40 (32%) had the lesion. Histologically, there were focal areas of coagulative necrosis, measuring 1 to 3 mm, affecting superficial to mid-level gastric mucosa (Fig. 2). Variable amounts of hemorrhage and blood pigments were present in the necrotic areas. Small numbers of neutrophils were sometimes scattered along the margins of the erosions.

Hepatic lipidosis was present in 8 of 16 (50%) heavily oiled, 5 of 12 (42%) moderately oiled, and 1 of 19 (5%) lightly oiled Group 1 otters. Among total Group 1 otters, 14 of 47 (30%) had the lesion. The prevalence of renal lipidosis was somewhat less than that of hepatic lipidosis. Overall, 10 of 42 (24%) Group 1 otters had renal lipidosis. All Group 1 animals with hepatic or renal lipidosis were female. All animals with renal lipidosis also had hepatic lipidosis. Several of the affected animals were pregnant or lactating, but several others were not. Both lesions were common in heavily and moderately oiled animals that died within 8 days of arrival at the centers, 13 of 22 (59%) animals with hepatic lipidosis and 10 of 22 (45%) animals with renal lipidosis, and did not occur in animals that died later. The liver lesion was characterized by the presence of usually multiple but occasionally single, round, unstained intracytoplasmic vacuoles in periportal to midzonal hepatocytes (Fig. 3). Affected proximal convoluted tubular epithelium of the kidney contained similar intracytoplasmic vacuoles that were usually single (Fig. 4). In oil red O-stained sections of liver and kidney, the intracytoplasmic vacuoles stained red, indicating the presence of lipid.

Centrilobular hepatic necrosis occurred in 4 of 16 (25%) heavily oiled, 3 of 12 (25%) moderately oiled, and 4 of 19 (21%) lightly oiled Group 1 otters. Among all Group 1 otters, 11 of 47 (23%) were affected. In affected livers, centrilobular hepatocytes had undergone coagulative necrosis (Fig. 5). Among all Group 1 otters, multifocal hepatic necrosis was present in 6 of 47 (13%) and focally extensive hepatic necrosis suggestive of infarction was present in 4 of 47 (8%). Multifocal hepatic necrosis occurred in an animal that died on the

first day in captivity and in animals that died after 3, 4, 5, 26, and 27 days. Focally extensive hepatic necrosis was found in animals that died after 4, 6, 8, and 27 days.

Of the 6 unoiled otters that died in rehabilitation centers (Group 2), one (17%) had gastric erosions, 1 (17%) had hepatic lipidosis and multifocal hepatic necrosis, and 1 (17%) had focally extensive hepatic necrosis.

Of the 5 sea otters found dead with external oil present (Group 3), 1 had pulmonary interstitial emphysema and hepatic and renal lipidosis, and 2 others had hepatic and renal lipidosis. One of the otters with hepatic and renal lipidosis was male and the other 2 were female.

Of the 6 apparently previously healthy sea otters collected from an area that had not been affected by an oil spill (Group 4), none had pulmonary interstitial emphysema, gastric erosions, hepatic or renal lipidosis, or hepatic necrosis. Four were male and 2 were nonpregnant, nonlactating females.

Two hundred fourteen sea otter carcasses that were collected, placed in plastic bags, and frozen in the period following the spill (Group 5) were thawed and necropsied. One hundred fifty-two (71%) had detectable external oil present, and 62 (21%) had no detectable oil present. Pulmonary interstitial emphysema was present in 100 of 152 (66%) otters with external oil present and in 13 of 62 (21%) otters with no detectable external oil. Interlobular septa of affected lungs were expanded by bubbles of trapped air that ranged from 1 mm to 3 cm in diameter. The emphysema was frequently diffuse and severe. In such cases, large amounts of adjacent pulmonary parenchyma were compressed. Extension of the air into the mediastinum was common, and involvement of the pericardium and the subcutis of the neck and thorax were occasionally found. Gastric erosions were present in 83 of 152 (55%) otters with external oil present and in 4 of 62 (6%) otters with no detectable external oil. The erosions generally were 1 to 3 mm punctate mucosal defects with dark red bases. Occasionally, erosions were linear and up to 1 cm in length. In some cases only a few erosions were present in the pylorus, but often erosions were numerous and were scattered throughout the gastric mucosa. Occasionally, similar erosions were present in the proximal duodenum. Accompanying hemorrhage was always present and varied from scant in animals with few erosions to abundant in animals with numerous erosions. Internal oil was found in 32 of 152 (21%) otters with external oil present and in 1 of 62 (2%) otters with no detectable external oil. The oil appeared as multiple, small, usually less than 3 mm diameter, black or brown flecks and was found on the tracheal, bronchial, esophageal, and gastric mucosae. Exposure of the oil to ultraviolet light in a darkened room caused the oil to glow and appear yellow to green. Blood did not glow or change color under these conditions. Specific liver and kidney lesions could not be confidently identified by gross examination.

Various incidental lesions were found infrequently in Groups 1, 2, 3, and 5. Thyroid follicular ectasia was common in all groups examined histologically.

DISCUSSION

Pulmonary interstitial emphysema was prevalent in oiled sea otters that died in rehabilitation centers and in sea otters with external oil present that were found dead, frozen, and later thawed and necropsied (Group 5). The incidence of the lesion correlated with degree of oiling in Group 1 otters. It was also present in 1 of 5 otters found dead with

external oil present. Emphysema was not seen in unoiled otters that died in rehabilitation centers nor in apparently normal otters; however, it was present in several Group 5 otters that did not have detected external oil. Interstitial emphysema was diagnosed by others in many oiled sea otters presented to rehabilitation centers (Williams et al. 1990). Although not recognized prior to the *Exxon Valdez* oil spill, it is clear that exposure to crude oil causes sea otters to develop emphysema. The pathogenesis of the lesion in this setting is unclear. Alveolar tears are the usual route by which air enters the pulmonary interstitium. Alveolar tears can occur when there is a combination of forced expiration or coughing and bronchiolar obstruction that produces sharply increased pressures within alveoli (Cotran et al. 1989c, Dungworth 1985). In anatomically predisposed species such as cattle the lesion may occur agonally, presumably due to forced expiration combined with bronchiolar collapse (Dungworth 1985). Pulmonary interstitial emphysema has been reported in sea otters with pneumonia (Cornell et al. 1979, Mattison and Hubbard 1969) and has been seen rarely as a mild focal lesion in sea otters that died without evidence of respiratory disease or oil exposure (Lipscomb, personal observation). Sea otters may have an anatomical predisposition to development of interstitial emphysema, but exposure to crude oil resulted in a remarkably high incidence of the lesion. During the early days of the spill, inhalation of volatile components of crude oil such as benzene might have damaged alveolar septa and caused the lesion, but neither interstitial pneumonia nor other lesions that might result from inhalation of an irritant vapor were found in affected sea otters. Aspiration of oil may have caused powerful forced expirations that could result in interstitial emphysema. Oiled sea otters attempt to remove oil by grooming (Siniff et al. 1982), which involves use of the mouth; this process provides ample opportunity for aspiration. However, aspiration pneumonia was not found. We speculate that sea otters may have a highly developed cough reflex that effectively prevents aspiration of oil into the lungs but promotes the development of interstitial emphysema. The Group 5 otters with emphysema and no detectable oil present may have successfully removed detectable oil prior to death, or the emphysema may have been caused by a different mechanism. Postmortem examination failed to indicate the cause of the emphysema in these otters.

Gastric erosions were common in oil-exposed sea otters that died in rehabilitation centers and in oil-exposed sea otters found dead and examined grossly but not histologically. They were also found in an unoiled otter that died in a center and in a few Group 5 otters with no detectable oil. An explanation for the relatively low frequency in heavily oiled Group 1 otters (Table 1) is not readily apparent. Only rarely was oil found in stomachs of otters with gastric erosions. Rapidly developing gastric erosions that appear following severe stress occur in humans and animals (Barker and Van Dreumel 1985, Cotran et al. 1989b). Gastrointestinal erosion/ulceration and hemorrhage have been reported in sea otters that died in captivity and in the wild and have been attributed to stress (Mattison and Hubbard 1969, Stullken and Kirkpatrick 1955). All of the gastric erosions seen in this study were acute; none showed signs of healing. Those present in otters that died shortly after arrival at the rehabilitation centers might have developed prior to capture because of stress associated with oil exposure, as a direct effect of oil on the gastric mucosa, or because of stress associated with capture and captivity. Erosions caused by ingestion of corrosive liquids are extensive (Fenoglio-Preiser et al. 1989), but the erosions we encountered were small and relatively uniform. Those seen in otters that died several days or more after arrival at the centers clearly developed in captivity.

Hepatic lipidosis was common in oiled otters that died in rehabilitation centers and in oiled otters that were found dead. The incidence of the lesion correlated with the degree of oiling. It was also seen in an unoiled otter that died in a rehabilitation center. Renal lipidosis was somewhat less common and occurred only in otters that also had hepatic lipidosis. All animals with hepatorenal lipidosis were female except for 1 oiled male that was found dead. When the oil spill occurred, many females were in late gestation or had recently pupped and were lactating. Hepatic lipidosis is known to have various causes including toxins, mobilization of stored fats due to inadequate food intake, hepatocellular hypoxia, certain metabolic disorders such as diabetes mellitus, and obesity (Kelly 1985). Causes of renal lipidosis include toxins, hypoxia, and decreased food intake (Jones and Hunt 1983a, 1983b). Studies of experimentally oiled otters report marked increases in activity and metabolic rate with unchanged or decreased time devoted to feeding (Costa and Kooyman 1982, Siniff et al. 1982). Animals with high energy demands like those that occur during peak lactation or late gestation are predisposed to hepatic lipidosis (Mac Lachlan and Cullen 1988). Thus, hepatic and renal lipidosis may have been caused by an oil exposure-associated increase in energy demand with constant or decreased food intake resulting in mobilization of stored fat. Many of the affected otters were further predisposed because of high energy demands due to lactation or pregnancy. Hepatic lipidosis in pregnant and lactating females may have been "physiologic," as occurs in ruminants, although this phenomenon has not been reported in sea otters. A direct toxic effect is possible, but accompanying hepatic and renal necrosis was not regularly present, and it is unusual for toxins to preferentially affect one gender. The fatty liver of hepatocellular hypoxia primarily affects centrilobular hepatocytes (Kelly 1985), but lipid accumulation in these otters was periportal to midzonal. The high incidence of lipidosis in otters that died during the first few days of captivity, its absence in otters that died after captivity day 8, its presence in 3 of 5 oiled otters that were found dead in the wild, and the absence of reports of lipidosis in otters that died in captivity suggest that captivity was not the cause of the lesion in our otters.

Centrilobular hepatic necrosis was also relatively common in oiled otters that died in rehabilitation centers and was not found in unoiled otters that died in the centers. Causes of centrilobular hepatic necrosis include toxins and conditions that cause hepatic ischemia, such as anemia, shock, and heart failure (Kelly 1985). Some oiled otters became anemic while at rehabilitation centers (Wilson et al. 1990). Crude oil ingestion (Leighton 1986) and gastric erosion with hemorrhage are possible causes of anemia. However, gastric erosions and centrilobular hepatic necrosis were found in the same otter infrequently, so anemia due to gastric hemorrhage was not a common cause of centrilobular hepatic necrosis. It is likely that many otters experienced shock (Wilson et al. 1990). Shock might result from oil exposure or captivity, but centrilobular hepatic necrosis has not been reported previously in otters that died in captivity. Multifocal hepatic necrosis and focally extensive hepatic necrosis suggestive of infarcts occurred at low frequency in both oiled and unoiled sea otters that died in rehabilitation centers. The causes of these lesions were not found.

Sea otters are largely dependent on the insulating properties of their pelage for protection from the cold waters they inhabit. It had been suspected that hypothermia would be a major problem in oiled sea otters because oil markedly increases the thermal conductance of their coats (Williams et al. 1988). Hypothermia was a common problem in oiled sea otters presented to rehabilitation centers (Williams et al. 1990). Death caused by hypothermia can occur without distinctive gross or microscopic lesions (Cotran et al. 1989a).

It is likely that stress and shock were significant medical problems (Wilson et al. 1990). Both oil exposure and captivity are stressful to sea otters (Siniff et al. 1982, Stullken and Kirkpatrick 1955). We believe that hypothermia, stress, shock, respiratory compromise associated with interstitial emphysema, hemorrhage from gastric erosions, and hepatic necrosis contributed to the deaths of oiled sea otters.

CONCLUSIONS

In summary, pulmonary interstitial emphysema, gastric erosion and hemorrhage, hepatic and renal lipidosis, and centrilobular hepatic necrosis were common in oiled sea otters that died in rehabilitation centers and were absent or uncommon in the small group of unoiled sea otters that died in rehabilitation centers. Pulmonary interstitial emphysema and gastric erosion and hemorrhage were prevalent in oiled sea otters that were examined grossly but not histologically; these lesions were found much less commonly in sea otters without detectable oil that were examined grossly but not histologically. None of these lesions were seen in apparently normal, unoiled sea otters and, with the exception of gastric erosion and hemorrhage, have not been previously reported in association with death in captivity. Additionally, pulmonary interstitial emphysema and hepatic and renal lipidosis were present in a small group of oiled sea otters that were found dead in the wild. Pathologic examination of larger numbers of both oiled and unoiled sea otters not held in captivity would be useful in separating lesions resulting from exposure to crude oil and those resulting from effects of captivity.

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Table 1. Sex, arrival date, death date, number of days at rehibilitation center, and lesions found in oiled and unoiled sea otters that died in rehabilitation centers and in oiled otters that were found dead.

Otter #	Sex	Arrival date	Death date	Days in center	EMP	GE	HL	RL	CLHN
GROUP 1 (oiled, died in center)									
Heavily Oiled									
1	F	7 Apr	7 Apr	<1	X	X	X		
2	F	4 Apr	5 Apr	1		X			
3	F	9 Apr	10 Apr	1	X		X	X	
4	F	6 Apr	7 Apr	1	X		X		
5	F	6 Apr	7 Apr	1					X
6	F	5 Apr	8 Apr	3	X		X	X	
7	F	31 Mar	3 Apr	3	X		X		
8	F	4 Apr	7 Apr	3	X				
9	F	19 Apr	23 Apr	4			X	X	
10	F	3 Apr	7 Apr	4	X				
11	F	5 Apr	10 Apr	5	X		X	X	
12	M	30 Mar	5 Apr	6	X				
13	M	2 Apr	9 Apr	7	X				X
14	F	1 Apr	9 Apr	8	X		X	X	X
15	M	1 Apr	10 Apr	9					
16	M	2 Apr	28 Jul	117					X
Moderately Oiled									
17	F	9 Apr	10 Apr	1			X	X	
18	F	4 Apr	5 Apr	1	X		X	X	X
19	F	8 Apr	9 Apr	1	X	X	X	X	
20	F	7 Apr	8 Apr	1			X	X	
21	F	6 Apr	8 Apr	2	X		X	X	X
22	F	3 Apr	6 Apr	3	X	X			
23	M	9 Apr	13 Apr	4		X			

Otter #	Sex	Arrival date	Death date	Days in center	EMP	GE	HL	RL	CLHN
24	F	4 Apr	9 Apr	5		X			
25	M	18 Apr	29 Apr	11					
26	F	5 Apr	18 Apr	13	X				
27	F	11 May	24 May	13		X			
28	M	5 Apr	5 May	30		X			
29	F	11 May	24 Jul	74		X			
				Lightly Oiled					
30	F	20 Apr	20 Apr	<1					X
31	F	5 Jun	5 Jun	<1					
32	F	6 Apr	7 Apr	1					
33	F	5 Jun	6 Jun	1					
34	M	13 Jun	14 Jun	1		X			
35	F	9 Apr	11 Apr	2			X		
36	F	1 Apr	4 Apr	3					
37	F	4 Apr	7 Apr	3	X	X			
38	M	6 Apr	12 Apr	6	X				
39	F	10 May	17 May	7					
40	M	19 Apr	27 May	8					X
41	F	25 May	4 Jun	10		X			
42	M	20 May	31 May	11					
43	F	13 Jun	27 Jun	14		X			X
44	F	8 Apr	28 Apr	20					
45	M	8 Apr	29 Apr	21					
46	M	8 Apr	1 May	23					
47	M	6 Apr	29 Apr	23					
48	F	6 Apr	30 Apr	24	X				
49	F	10 Apr	6 May	26					X
50	F	11 May	7 Jun	27					

Otter #	Sex	Arrival date	Death date	Days in center	EMP	GE	HL	RL	CLHN
51	F	20 May	19 Jun	30					
			GROUP 2 (unoiled, died in center)						
52	F	29 May	29 May	<1					
53	M	13 Apr	14 Apr	1					
54	F	5 Jul	6 Jul	1			X		
55	M	25 Jun	27 Jun	2					
56	F	19 Jun	3 Jul	14					
57	M	17 Jul	4 Aug	18			X		
			GROUP 3 (found dead oiled)						
58	M								
59	M						X	X	
60	M								
61	F						X	X	
62	F				X		X	X	

EMP = emphysema
GE = gastric erosion
HL = hepatic lipidosis
RL = renal lipidosis
CLHN = centrilobular hepatic necrosis
F = female
M = male

Table 2. Oiling status, sex, and lesions found in oiled and unoiled sea otters that died in rehabilitation centers, and in oiled sea otters found dead.

			Emphysema		Gastric erosion		Hepatic lipidosis		Renal lipidosis		Centrilobular necrosis	
Degree of oiling	Sex	*n*	# Affected /Total Examined*	%	# Affected /Total Examined*	%	# Affected /Total Examined*	%	# Affected /Total Examined*	%	# Affected /Total Examined*	%
					GROUP 1 (oiled, died in center)							
HO	F	12	9/11	82	2/10	20	8/12	67	5/11	45	2/12	17
HO	M	4	2/4	50	0/4	0	0/4	0	0/2	0	2/4	50
HO	T	16	11/15	73	2/14	14	8/16	50	5/13	38	4/16	25
MO	F	10	5/8	62	5/6	83	5/9	56	5/9	56	3/9	33
MO	M	3	0/3	0	2/3	67	0/3	0	0/3	0	0/3	0
MO	T	13	5/11	45	7/9	78	5/12	42	5/12	42	3/12	25
LO	F	15	2/14	14	3/13	23	1/12	8	0/12	0	3/12	25
LO	M	7	1/6	17	1/4	25	0/7	0	0/5	0	1/7	14
LO	T	22	3/20	15	4/17	24	1/19	5	0/17	0	4/19	21
					GROUP 1 Totals							
	F	37	16/33	48	10/29	34	14/33	42	10/32	31	8/33	24
	M	14	3/13	23	3/11	27	0/14	0	0/10	0	3/14	21
	T	51	19/46	41	13/40	32	14/47	30	10/42	24	11/47	23
					GROUP 2 / unoiled, died in center							
U	F	3	0/3	0	0/3	0	0/3	33	0/3	0	0/3	0
U	M	3	0/3	0	1/3	33	0/3	0	0/3	0	0/3	0
U	T	6	0/6	0	1/6	17	1/6	17	0/6	0	0/6	0
					GROUP 3 / found dead oiled Totals							
	F	2	1/2	50	0/2	0	2/2	100	2/2	100	0/2	0
	M	3	0/3	0	0/3	0	1/3	33	1/3	33	0/3	0
	T	5	1/5	20	0/5	0	3/5	60	3/5	60	0/5	0

HO = heavily oiled
MO = moderately oiled
LO = lightly oiled
U = unoiled
F = female
M = male
T = female and male totaled
* = # available for comparison; all relevant tissues were not available from all otters.

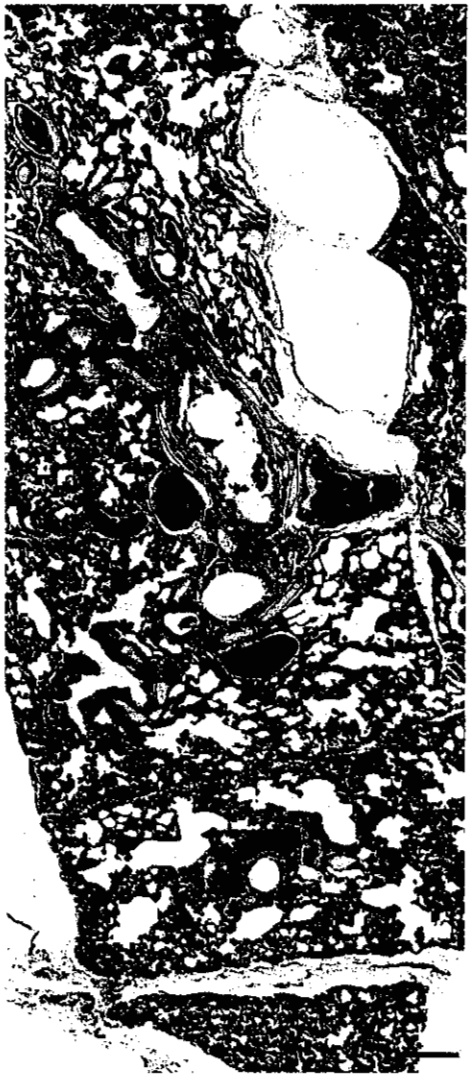

Figure 1. Lung: otter No. 11, interstitial emphysema.

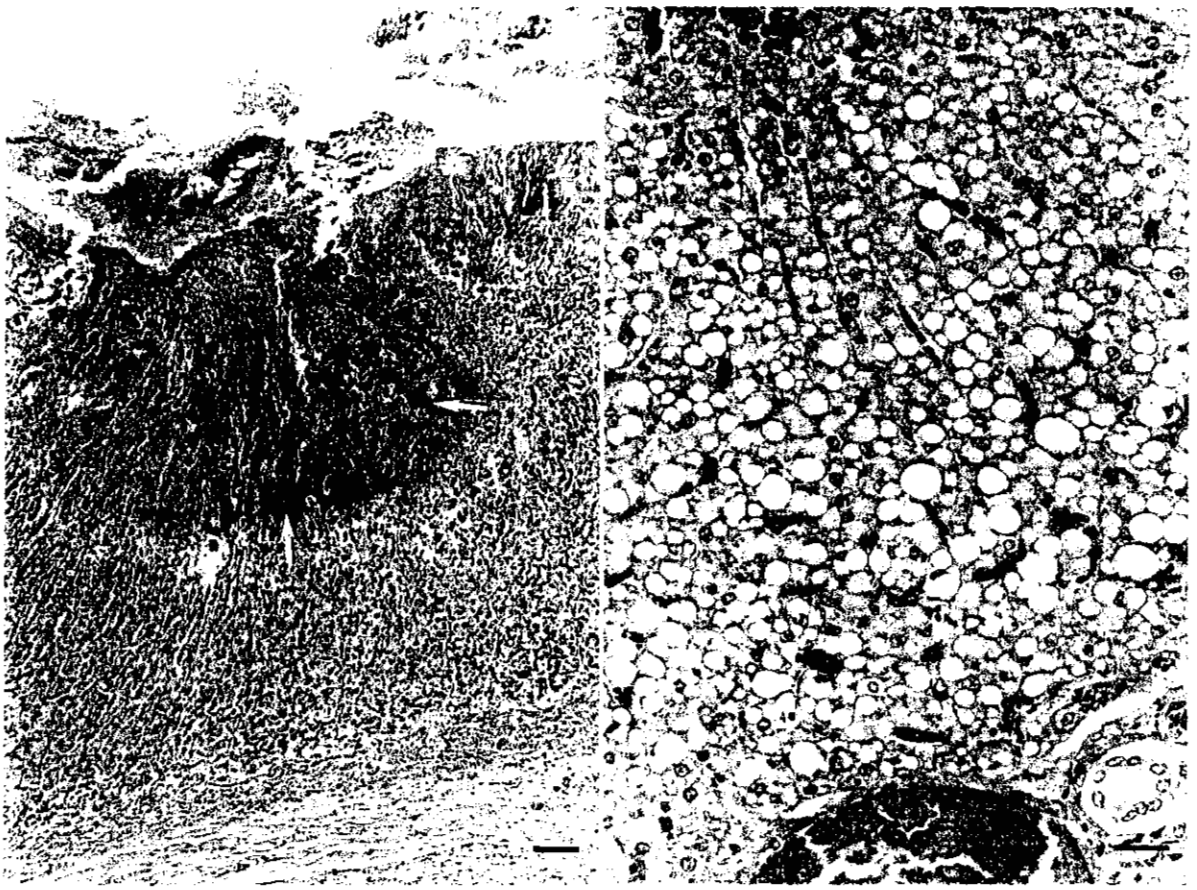

Figure 2. Gastric mucosa: otter No. 22, focal area of coagulative necrosis.

Figure 3. Liver: otter No. 20, diffuse lipidosis that is more severe in periportal hepatocytes.

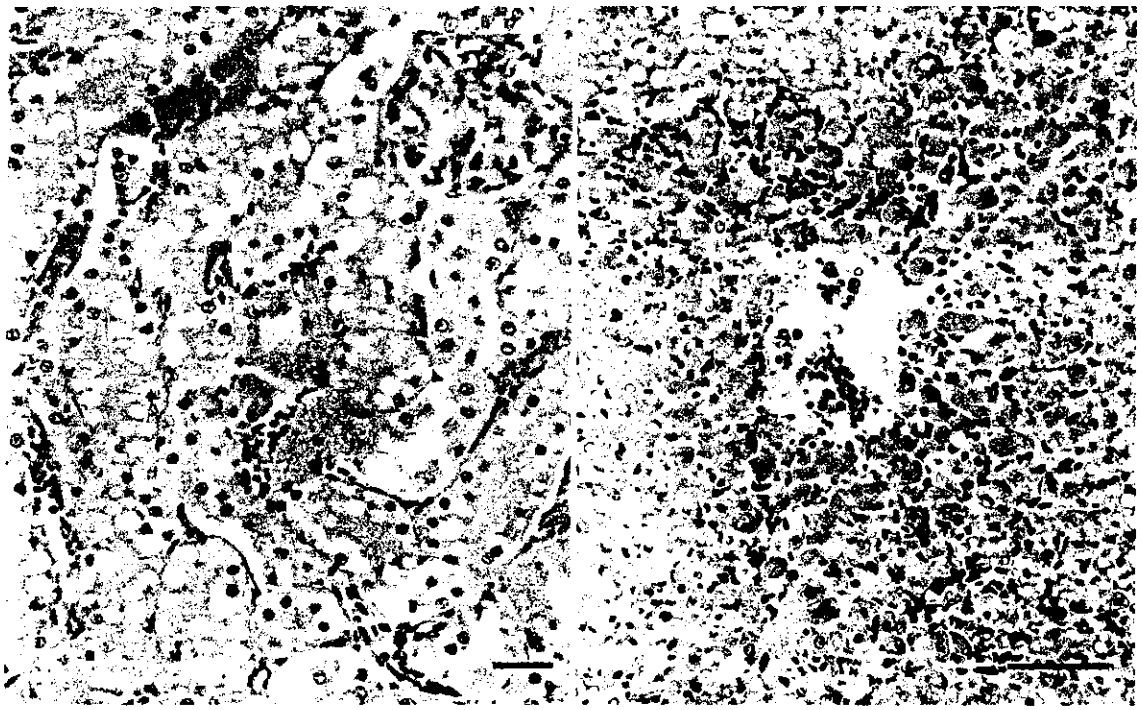

Figure 4. Kidney: otter No. 11, lipidosis of the proximal and distal tubular ephithelium.

Figure 5. Liver: otter No. 18, centrilobular necrosis and midzonal lipidosis.