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Pathologic Findings in Florida Manatees (*Trichechus manatus latirostris)*

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Abstract

This report describes pathologic findings associated with mortality in Florida manatees (*Trichechus manatus latirostris*) (n = 68) between January 1996 and January 2004. The most frequent causes of death among these Florida manatees were trauma (47%), cold stress syndrome (CSS) (18%), inflammatory/infectious disease (12%), and suspected brevetoxicosis (9%). There were few perinatal deaths (7%). Probably all deaths due to trauma, as well as some, and perhaps many, cases of CSS, may be regarded as anthropogenic, reinforcing the need for conservation and management strategies to mitigate these impacts on this endangered species. Cause of death was determinable in a high proportion (94%) of sample cases, demonstrating the importance of performing timely gross and microscopic necropsy examinations on marine mammals.

Key Words: Florida manatee, *Trichechus manatus latirostris*, pathology, Florida, cold stress syndrome, mortality

Introduction

The Florida manatee (*Trichechus manatus latirostris*) presently is categorized as an endangered species (U.S. Marine Mammal Commission, 2001). Long-term survival of the Florida manatee may be jeopardized by human-related traumatic injuries, perinatal mortality, and the degradation and destruction of natural habitat caused by widespread development in Florida (U.S. Fish and Wildlife Service, 2001). Based on clinical findings, Florida manatees appear to be remarkably resistant to natural disease and the sublethal effects of traumatic human-related injury (Bossart et al., 2002; Buergelt & Bonde, 1983; Buergelt et al., 1984). These disease-resistant traits may partially result from a remarkably efficient and responsive immune system (Bossart, 1999).

Notable exceptions to natural disease resistance in the Florida manatee are found with the morbidity and mortality associated with prolonged cold water exposure, and the inhalation and/or ingestion of Florida red tide brevetoxins (Bossart, 2001; Bossart et al., 1998, 2003a; O'Shea, 1988; O'Shea et al., 1985); however, the general pathologic aspects of disease associated with mortality in manatees have not been well-characterized. Pathologic findings in Florida manatees have not been described since 1984 (Buergelt et al., 1984).

In this report, we document the pathologic findings associated with mortality in 68 Florida manatees that died between January 1996 and January 2004. The cause of death could be determined in most cases, which demonstrates the importance of performing gross and microscopic necropsy examinations on stranded marine mammals. Understanding the pathologic features associated with the mortality of Florida manatees is important for the future management of this endangered population and may provide insights into the health of the ecosystem in which these animals inhabit.

Materials and Methods

Necropsies were conducted on Florida manatees (n = 68) between January 1996 and January 2004 (Appendix A). Manatees were necropsied by the staff of the Marine Mammal Pathobiology Laboratory (MMPL) of the Florida Fish and Wildlife Conservation Commission in St. Petersburg, Florida (Florida Marine Research Institute, 2004). Only carcasses with minimum postmortem decomposition, comprising approximately 17% of the total number of manatees necropsied, were examined for this study.

A complete necropsy, which followed a recommended procedure (Bonde et al., 1983), was performed on each carcass. Morphometrics were collected and age categories were estimated by body length. Age/size categories consisted of perinates (≤ 150 cm), subadults (151-275 cm), and adults (≥ 276 cm) (Ackerman et al., 1995). Tissue sections from the brain, lung, heart, liver, spleen, superficial and deep lymph nodes, thymus (if present), gall bladder, stomach, cardiac gland, pancreas, duodenal diverticulum, small intestine, cecum, cecal horn, colon, mesenteric fat, kidney, adrenal gland, thyroid gland, skeletal muscle, blubber, and skin were collected for histologic examination from all manatees. Tissues were placed in 10% neutral buffered formalin, routinely processed, embedded in paraffin, sectioned at 5 µm, and stained with hematoxylin and eosin. Special stains used were Gomori's methenamine silver, Brown and Brenn, and periodic acid-Schiff.

In this study, traumatic injuries were determined based on historic criteria established by the MMPL. The traumatic injuries consisted of sharp penetrating trauma from boat propellers and blunt trauma from boat hull or skeg impacts. Death by trauma was further subdivided based on gross and microscopic findings into acute (i.e., injury considered severe enough to have caused death immediately or within a few hours with no pathologic evidence of preexisting disease) or chronic categories (i.e., injury that was not immediately fatal, but resulted in secondary inflammatory/infectious disease and death).

The criteria for death by cold stress syndrome (CSS) were based on the temporal correlation with cold weather prior to death and the combined presence of advanced emaciation and the characteristic gross and microscopic skin, fat, lymphoid, myocardial, and/or intestinal lesions recently characterized by Bossart et al. (2003a). Similarly, criteria for suspected brevetoxicosis were based on the temporal correlation of a bloom of red tide (*Karenia brevis*), high tissue levels of brevetoxins determined by an ELISA technique, the presence of characteristic gross and microscopic nasal and/or pulmonary lesions (Bossart et al., 1998), and the absence of traumatic injury or other indications of preexisting disease conditions.

Results

The age/size and sex distribution of the manatees in the study, as well as the pathologic findings, are summarized in Figure 1, Table 1, and in greater detail by case number in Appendix A.

The most common cause of death (47%, Cases 1-32, Appendix A) among these Florida manatees was trauma secondary to human-inflicted injury,

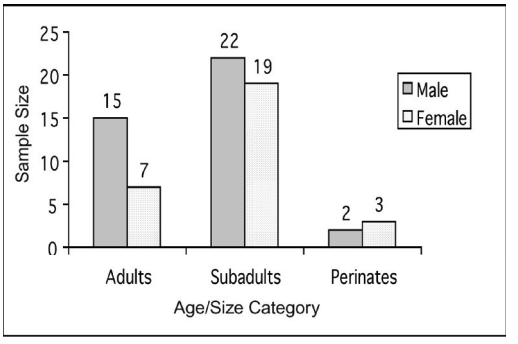

Figure 1. Age and sex distribution of 68 Florida manatee mortality cases between 1996 and 2004

including sharp penetrating trauma from boat propellers and blunt trauma from nonpropeller boat hull or skeg impacts. Traumatic death was found primarily in subadults (59%) with no sex predilection (Table 1).

Death from trauma in these Florida manatees involved, in decreasing order of frequency, thoracic injury (66%), combined thoracic and abdominal injury (13%), central nervous system (CNS) injury (13%), and abdominal injury (9%) (Appendix A). Among the thoracic injuries, 14 cases (67%) were considered chronic with death generally resulting from chronic-active pleuropneumonia and often sepsis secondary to open boat propeller wounds of the thorax. Similarly, three abdominal injury cases also were categorized as chronic with death resulting from a disseminated serositis or multiorgan inflammatory disease secondary to open boat propeller wounds to the abdomen. The remaining seven, thoracic trauma (33%), CNS, and combined thoracic/abdominal trauma cases were considered acute in nature with death resulting from acute pulmonary, CNS, or combined pulmonary and abdominal hemorrhage, respectively, and shock.

Cases 1-14 were categorized as chronic thoracic trauma. The most common gross and microscopic chronic trauma lesions were pleuritis/pleuropneumonia (n = 12), multiple rib fractures (n = 12), multiple dorsolateral penetrating propeller wounds of the thoracic cavity (n = 10), blunt hull or skeg injury (n = 5), multiple lung lacerations (n = 3), pneumothorax (n = 3), and pneumonia with multisystemic disease (n = 2). Case 5 also had a testicular interstitial cell tumor.

Cases 15-17 involved abdominal trauma cases only and were considered chronic based on the presence of multiple open penetrating wounds of the abdominal cavity (Cases 15 and 17) or multiple healed abdominal propeller injuries (Case 16) and associated concurrent disease states. Case 15 had a severe splenitis and enteritis, Case 16 had a severe abdominal serositis with intralesional

Table 1. Cause of death for 68 Florida manatees by age/size and sex in decreasing order of frequency, January 1996–January 2004

Cause of death	N	Adult		Subadult		Perinatal	
		Male	Female	Male	Female	Male	Female
Trauma	32	7	6	12	7		
Cold stress syndrome	12	1		4	7		
Inflammatory/infectious disease	8	2		3	2		1
Suspected brevetoxicosis	6	2	1	2	1		
Cachexia	4					2	2
Unknown	4	2		1	1		
Intestinal foreign body	1				1		
Cardiomyopathy	1	1					
Total	68						

bacilli, and Case 17 had a severe necrotizing cellulitis associated with propeller injuries.

Cases 18-24 were categorized as acute thoracic trauma. The common gross and microscopic lesions of this category were acute pulmonary hemorrhage ($n = 6$), multiple rib fractures ($n = 5$), multiple lung lacerations ($n = 4$), multiple penetrating wounds of the thoracic cavity ($n = 3$), and hemothorax ($n = 3$).

Cases 25-28 were categorized as combined thoracic and abdominal injury and all were considered acute cases. The common gross and microscopic lesions of this category were multiple deep penetrating propeller wounds of the thoracic and abdominal cavities ($n = 4$), multiple rib fractures ($n = 3$), renal laceration with hemorrhage ($n = 2$), and abdominal hemorrhage ($n = 2$).

Cases 29-32 were categorized as acute CNS injury. Brain or spinal cord hemorrhage ($n = 4$), multiple skull fractures ($n = 2$), and multiple vertebral fractures ($n = 1$) were the observed lesions in these cases.

Cold stress syndrome accounted for 18% of the Florida manatee deaths in this study (Cases 33-44). Subadults (92%) were the most common age group for CSS deaths. There were no perinates found or sex predilection observed in this category. The most common lesions observed were marked depletion of internal fat stores ($n = 11$), widespread lymphoid depletion ($n = 6$), multifocally extensive dermatitis ($n = 6$), severe serous fat atrophy ($n = 4$), colitis or enterocolitis ($n = 4$), myocardial degeneration ($n = 3$), and pneumonia ($n = 2$).

Deaths caused by cachexia (6%, Cases 45-48) included all perinates with no sex predilection. The universal gross and microscopic lesions consisted of emaciation, widespread serous fat atrophy, acinar atrophy of the pancreas, and hepatocellular atrophy. There was no evidence of inflammatory or infectious disease.

Suspected brevetoxicosis was the cause of death for six individuals of our study sample (9%, Cases 49-54) and included only adults and subadults with no sex predilection. Gross and microscopic lesions were limited to rhinitis, widespread pulmonary edema, and multiorgan congestion and hemorrhage. No evidence of infectious disease was present.

Primary inflammatory or infectious disease accounted for 12% of our sample (Cases 55-62). Case 63 died from infectious disease secondary to a perforating intestinal foreign body consisting of a fishhook and monofilament line. Inflammation of the intestinal tract (38%) and pneumonia (38%) were the most common disease types in this category. Microscopic lesions in this category included enteritis or enterocolitis ($n = 4$, including Case 63 with intralesional bacteria), bronchopneumonia ($n = 2$, with intralesional bacteria), bronchointerstitial pneumonia ($n = 1$), systemic lymphadenitis ($n = 3$), peritonitis ($n = 2$), and hepatitis ($n = 1$). Based on histologic appearance, the inflammatory lesions were all considered infectious in etiology; however, only three of these cases had intralesional gram-negative coccobacilli that were considered causal.

Case 64 died of a cardiomyopathy and exhibited changes consistent with right-sided congestive heart failure, the first report of this condition in manatees. The right ventricle and atrium were dilated and thin walled and the liver had a nutmeg appearance with marked congestion. Microscopically, the heart had severe right ventricular cardiomyocyte degeneration, diffuse fibrosis, interstitial edema, wavy attenuated myofibers, loss of cross-striations, and anisokaryosis with karyomegaly. The liver had severe centrilobular fibrosis, hepatocellullar atrophy, congestion, and moderate hemosiderosis. The etiology could not be determined, but the heart changes did not appear to be associated with recent infectious or inflammatory disease.

Finally, the cause of death could not be determined for Cases 65-68. Mild inflammatory, degenerative, or nonspecific changes were noted among these cases (Appendix A); however,

they were not considered severe enough to have resulted in death.

Discussion

This is the first published study of the general pathologic findings in Florida manatees since 1984 (Buergelt et al., 1984). It demonstrates notable mortality trends, which may have future management implications for this endangered marine mammal.

The proportion of cases in the current study due to trauma (47%) was higher than the 24% of trauma deaths reported from a larger sample (n = 895) obtained from the database examined by the MMPL during the same time period. This larger database, which included both fresh and more decomposed carcasses, included more manatees killed by watercraft-related trauma than were available for histopathologic analysis (Lightsey et al., in prep.). Thus, the greater number of manatees from the larger MMPL sample which were in an advanced stage of decomposition probably inflated the proportion of deaths categorized as having an "undetermined" cause of death, which, in turn, probably masked some cases of death due to trauma. Additionally, the higher proportion of deaths due to trauma may be related to the smaller proportion of perinates (7%) in the current sample vs 23% in the MMPL database.

It is unclear why subadults predominated among the individuals in our sample killed by trauma; however, the pattern is disturbing because of the potential negative impact on the future breeding population. In some cases of traumatic death, the event was probably of sufficient severity to cause almost immediate death; however, pathologic observations on a high proportion of the remaining trauma deaths suggest that these manatees lived some time (probably weeks) after the initial traumatic event. One manatee in this group (Case 5) had a benign interstitial cell tumor which, to our knowledge, is the first testicular neoplasm reported in this species. This was considered an interesting but incidental finding.

Death from CSS is due to a series of cascading events starting with prolonged exposure to cold water and followed by nutritional, metabolic, and immunologic disturbances culminating in multisystemic, life-threatening opportunistic infectious disease (Bossart et al., 2003a). The predominance of subadults observed in this category may indicate a relationship of low body mass, the ability to maintain thermal homeostasis, and susceptibility to the effects of cold temperature leading to CSS. Additionally, the absence of perinates in this category may reflect a dependant perinate's reliance on maternal experience for guidance to warm water sources and a constant available source of high calorie nutrition through nursing.

The pattern of enteric and pulmonary inflammatory disease observed among study subjects, primarily adults and subadults, is similar to that seen in captive manatees, especially those who were orphaned as perinates (Bossart, 2001). Case 63, which involved bacterial enteritis secondary to a perforating foreign body (fish hook and monofilament line), is a previously undescribed lesion; however, monofilament line leading to intestinal intussusceptions without perforation has previously been reported in manatees (Buergelt et al., 1984; Forrester et al., 1975).

Brevetoxins have neurotoxic and hemolytic properties and are produced by the dinoflagellate (*Karenia brevis*) found in Florida red tide blooms. Florida manatees seem particularly prone to the chronic effects of inhalational and ingestional brevetoxicosis, with the primary pathologic lesions consisting of a catarrhal rhinitis, multiorgan congestion and hemorrhage, and, less commonly, hemosiderosis and nonsuppurative meningitis (Bossart et al., 1998). The precise pathogenic mechanism of this intoxication in manatees is unknown, however, and the exclusionary diagnosis of brevetoxicosis is based on the absence of preexisting disease; the typical but nonspecific pathologic lesions; and a temporal correlation of high levels of brevetoxins in tissues by an ELISA technique and the presence of red tide.

The condition of the four perinatal manatees that died from cachexia was consistent with prolonged protein-caloric deficiency, and these manatees likely were orphaned. The clinicopathologic features were similar to those often seen in orphaned manatees presented to rehabilitation facilities for medical care (Bossart, 2001).

Considering the endangered status of this species, the continued high level of human-related mortality in Florida manatees is worrisome. The unfortunate trend of high human-inflicted mortality in Florida manatees is likely multifactorial, involving complex geopolitical and socioeconomic factors unique to the United States. Additionally, in the United States, it has been suggested that the Florida manatee may not be as charismatic as other endangered wildlife species and, therefore, such high human-related mortality may be more publicly (i.e., animal welfare groups) and politically tolerated (Bossart, 1999). Based on these data, the growing human population of Florida will likely result in increased human-related manatee mortalities. Ultimately, the conservation of the Florida manatee depends on the understanding of the unique problems that man creates for this endangered species and the adoption of appropriate actions based on this knowledge.

CSS and brevetoxicosis appear to represent unique mortality factors for the Florida manatee and should be included in management plans, especially considering the disappearance of manmade sources of warm water effluent to which manatees have habituated in cold weather, and the suspected increased incidence of red tide blooms (Bossart et al., 1998; U.S. Marine Mammal Commission, 2001). In the case of CSS, serious concern exists about the inevitable shutdown and/or deregulation of aging power plants that produce the warm water effluent that manatees have become dependent on in cold weather (U.S. Fish and Wildlife Service, 2001). Because manatees are unable to adapt quickly to a diminished availability of warm water, the elimination of these refuges could have profound effects on the mortality and distribution of this compromised species.

Finally, the cause of death was determinable in a high proportion of cases (94%). A similar trend was recently reported in stranded dolphins, which demonstrates the importance of performing timely gross and microscopic necropsy examinations on manatees, and marine mammals in general (Bossart et al., 2003b).

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Appendix A. Pathologic findings in Florida manatees from January 1996 to January 2004

Case number	Age/Size	Sex	Cause of death	Primary lesions
1	Adult	M	Trauma (thoracic)	Pleuritis, Bronchioltis, Enteritis
2	Adult	F	Trauma (thoracic)	Pleuritis, Pneumonia, Lymphadentis
3	Subadult	F	Trauma (thoracic)	Pleuritis, Pneumonia, Serous Fat Atrophy
4	Subadult	F	Trauma (thoracic)	Pleuritis, Pneumonia, Cellulitis
5	Adult	M	Trauma (thoracic)	Pneumonia, Myocarditis, Interstitial Cell Tumor
6	Subadult	M	Trauma (thoracic)	Pneumonia, Splenitis, Cholangiohepatitis
7	Adult	F	Trauma (thoracic)	Pleuritis, Pneumonia, Colitis
8	Adult	M	Trauma (thoracic)	Pleuritis, Serositis, Myositis
9	Subadult	F	Trauma (thoracic)	Pleuritis, Serous Fat Atrophy
10	Subadult	M	Trauma (thoracic)	Pleuropneumonia, Serositis
11	Subadult	F	Trauma (thoracic)	Pleuropneumonia, Serositis
12	Subadult	F	Trauma (thoracic)	Pleuropneumonia, Cellulitis, Pyelonephritis
13	Adult	M	Trauma (thoracic)	Pleuropneumonia, Heart Degeneration
14	Subadult	F	Trauma (thoracic)	Pleuropneumonia, Nephritis
15	Subadult	M	Trauma (abdominal)	Splentis, Nephrosis, Enteritis
16	Adult	M	Trauma (abdominal)	Serositis
17	Subadult	M	Trauma (abdominal)	Cellulitis, Lymphoid Depletion
18	Subadult	M	Trauma (thoracic)	Acute Pulmonary Hemorrhage, Rib Luxations
19	Adult	F	Trauma (thoracic)	Acute Pulmonary Hemorrhage and Edema, Rib Fractures
20	Subadult	M	Trauma (thoracic)	Rib Fractures
21	Subadult	M	Trauma (thoracic)	Acute Pulmonary Hemorrhage, Rib Fractures
22	Subadult	F	Trauma (thoracic)	Acute Pulmonary Hemorrhage, Rib Fractures
23	Subadult	M	Trauma (thoracic)	Acute Pulmonary Hemorrhage and Edema, Rib Fractures
24	Subadult	M	Trauma (thoracic)	Acute Pulmonary Hemorrhage and Edema, Rib Fractures
25	Subadult	M	Trauma (combined[1])	Acute Pulmonary Hemorrhage, Rib Fractures
26	Adult	F	Trauma (combined[1])	Rib Fractures, Renal Laceration/Hemorrhage
27	Adult	M	Trauma (combined[1])	Rib Fractures, Renal Laceration/Hemorrhage
28	Adult	M	Trauma (combined[1])	Renal Laceration, Vertebral Fractures
29	Adult	F	Trauma (CNS[2])	Vertebral Fractures, Spinal Cord Hemorrhage
30	Subadult	M	Trauma (CNS[2])	Cranial Fractures, Subdural Hemorrhage
31	Subadult	M	Trauma (CNS[2])	Cranial Fractures, Cerebral Hemorrhage
32	Adult	F	Trauma (CNS[2])	Cranial Contusion, Meningeal Hemorrhage
33	Subadult	M	Cold Stress Syndrome	Serous Fat Atrophy, Dermatitis, Lymphoid Depletion
34	Subadult	M	Cold Stress Syndrome	Pancreatic Atrophy, Serous Fat Atrophy, Colitis
35	Subadult	F	Cold Stress Syndrome	Pneumonia, Enterocolitis, Dermatitis
36	Adult	M	Cold Stress Syndrome	Cellulitis, Heart Degeneration, Lymphoid Depletion
37	Subadult	M	Cold Stress Syndrome	Dermatitis, Serous Fat Atrophy, Heart Degeneration
38	Subadult	F	Cold Stress Syndrome	Dermatitis, Heart Degeneration
39	Subadult	F	Cold Stress Syndrome	Serous Fat Atrophy, Enterocolitis
40	Subadult	F	Cold Stress Syndrome	Lymphoid Depletion, Thymic Atrophy
41	Subadult	M	Cold Stress Syndrome	Lymphoid Depletion, Heart Degeneration
42	Subadult	F	Cold Stress Syndrome	Lymphoid Depletion, Enterocolitis, Dermatitis
43	Subadult	F	Cold Stress Syndrome	Lymphoid Depletion, Thymic Atrophy
44	Subadult	F	Cold Stress Syndrome	Dermatitis, Pneumonia, Hepatitis
45	Perinatal	M	Cachexia	Hepatic/Pancreatic/Serous Fat Atrophy
46	Perinatal	F	Cachexia	Hepatic/Pancreatic/Serous Fat Atrophy
47	Perinatal	M	Cachexia	Hepatic/Pancreatic/Serous Fat Atrophy
48	Perinatal	F	Cachexia	Hepatic/Pancreatic/Serous Fat Atrophy
49	Subadult	M	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage
50	Subadult	F	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage
51	Adult	F	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage
52	Adult	M	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage

53	Subadult	M	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage
54	Adult	M	Suspected brevetoxicosis	Rhinitis, Multiorgan Congestion and Hemorrhage
55	Subadult	F	Inflammatory/Infectious Disease	Hepatitis, Lymphadentis, Heart Degeneration
56	Subadult	M	Inflammatory/Infectious Disease	Enterocolitis, Peritonitis, Sepsis
57	Adult	M	Inflammatory/Infectious Disease	Enterocolitis, Peritonitis, Sepsis, Lymphadenitis
58	Adult	M	Inflammatory/Infectious Disease	Pneumonia, Nodular Goiter
59	Perinatal	F	Inflammatory/Infectious Disease	Lymphadentis, Sepsis
60	Subadult	M	Inflammatory/Infectious Disease	Pneumonia (Bacterial), Hepatic Necrosis, Sepsis
61	Subadult	F	Inflammatory/Infectious Disease	Enteritis, Lymphoid Depletion
62	Subadult	M	Inflammatory/Infectious Disease	Pneumonia (Bacterial), Myocardial Necrosis
63	Subadult	F	Intestinal Foreign Body	Enteritis
64	Adult	M	Cardiomyopathy	Heart Degeneration, Hepatic Fibrosis/Congestion
65	Subadult	F	Unknown	Pancreatic Atrophy (Mild), Emphysema (Moderate)
66	Adult	M	Unknown	Lymphoid Depletion, Pulmonary Edema
67	Subadult	M	Unknown	Heart Degeneration (Moderate)
68	Adult	M	Unknown	Enterocolitis (Mild), Rhinitis (Mild)

[1]Combined thoracic and abdominal trauma
[2]Central nervous system