NOAA Technical Report NMFS 147

November 1999

Investigation of Unusual Mortalities of Bottlenose Dolphins along the Mid-Texas Coastal Bay Ecosystem during 1992

A. A. Colbert
G. I. Scott
M. H. Fulton
E. F. Wirth
J. W. Daugomah
P. B. Key
E. D. Strozier
S. B. Galloway

U.S. Department of Commerce

NOAA Technical Report NMFS 147
A Technial Report of the *Fishery Bulletin*

Investigation of Unusual Mortalities of Bottlenose Dolphins along the Mid-Texas Coastal Bay Ecosystem during 1992

A. A. Colbert
G. I. Scott
M. H. Fulton
E. F. Wirth
J. W. Daugomah
P. B. Key
E. D. Strozier
S. B. Galloway

November 1999

U.S. Department of Commerce
Seattle, Washington

Investigation of Unusual Mortalities of Bottlenose Dolphins along the Mid-Texas Coastal Bay Ecosystem during 1992

A. A. COLBERT, G. I. SCOTT, M. H. FULTON,
E. F. WIRTH, J. W. DAUGOMAH, P. B. KEY,
E. D. STROZIER, and S. B. GALLOWAY

Center for Coastal Environmental Health
and Biomolecular Research at Charleston
National Ocean Service, NOAA
219 Fort Johnson Road
Charleston, South Carolina 29412

ABSTRACT

An investigation was conducted into the deaths of more than 220 bottlenose dolphins (*Tursiops truncatus*) that occurred within the coastal bay ecosystem of mid-Texas between January and May 1992. The high mortality rate was unusual in that it was limited to a relatively small geographical area, occurred primarily within an inshore bay system separated from the Gulf of Mexico by barrier islands, and coincided with deaths of other taxa including birds and fish. Factors examined to determine the potential causes of the dolphin mortalities included microbial pathogens, natural biotoxins, industrial pollutants, other environmental contaminants, and direct human interactions. Emphasis was placed on nonpoint source pesticide runoff from agricultural areas, which had resulted from record rainfall that occurred during the period of increased mortality.

Analytical results from sediment, water, and biota indicated that biotoxins, trace metals, and industrial chemical contamination were not likely causative factors in this mortality event. Elevated concentrations of pesticides (atrazine and aldicarb) were detected in surface water samples from bays within the region, and bay salinities were reduced to <10 ppt from December 1991 through April 1992 due to record rainfall and freshwater runoff exceeding any levels since 1939. Prolonged exposure to low salinity could have played a significant role in the unusual mortalities because low salinity exposure may cause disruption of the permeability barrier in dolphin skin. The lack of established toxicity data for marine mammals, particularly dermal absorption and bioaccumulation, precludes accurate toxicological interpretation of results beyond a simple comparison to terrestrial mammalian models. Results clearly indicated that significant periods of agricultural runoff and accompanying low salinities co-occurred with the unusual mortality event in Texas, but no definitive cause of the moralities was determined.

Introduction

Early in 1992, the mortality and stranding rate of bottlenose dolphins (*Tursiops truncatus*) increased dramatically along the mid-Texas coast[1]. In April 1992, the National Marine Fisheries Service, Southeast Fisheries Science Center (NMFS/SEFSC) was notified that the Texas Marine Mammal Stranding Network suspected anomalous dolphin mortalities were occurring along the mid-Texas coast. Causal factors of these mortalities were not known. Gender, species, length, and location data were available for most dolphins, but the overwhelming number of reported strandings prevented thorough examination of a majority of them. The volunteer stranding network was unable to respond effectively to the increased number of strandings and requested investigative assistance from the NMFS/SEFSC.

Determining cause and effect in toxicological risk assessment investigations is always tenuous. This is particularly true when dealing with taxa such as marine mammals, for which very little clinical and toxicological data exists. Most marine mammal toxicological information is chemical body burden residue data, not toxi-

[1] Worthy, Graham. 1992. Texas Marine Mammal Stranding Network, 4700 Avenue U, Bldg. 303, Galveston, TX 77551. Personal commun.

cological data per se, and scientists are forced to use comparative terrestrial mammalian toxicological data as surrogate estimates of exposure, effects, and risk. While human forensic scientists have discrete methods for ascertaining cause of death, environmental risk assessment for cause and effect are still generally based on a "weight of evidence" approach which lacks discrete evidence of toxicological effects. Instead it may be based upon exceedances of sublethal, toxicological endpoints which indicate exposure, physiological impairment, or potential reduction in health.

The Working Group on Unusual Marine Mammal Mortalities, a panel of experts convened in 1991 to advise NMFS on such situations, was consulted about the dolphin deaths in Texas and agreed that a field investigation was warranted. Data were collected during the investigation for use in evaluating this unusual mortality event. In this paper we present results of the investigation and describe potential factors which may have affected dolphin health, based on the circumstantial evidence identified. Our conclusions include recommendations for improving future scientific response to unusual marine mammal mortality events. We also review actions that have already been taken to improve future investigations of this type.

Materials and Methods

Initial Field Investigation

The study's first priority was to define the mortality event's basic characteristics in order to determine the areas of focus for sample collection and analysis. Nautical charts and maps of the Texas coastline were used to determine the extent of logistical support required to conduct field reconnaissance. These indicated that the event's suspected magnitude was probably underestimated due to the region's numerous remote habitats (Fig. 1). In these remote regions, aerial surveys were initially used to determine the magnitude and geographical extent of the dolphin mortalities. Overflights were continued biweekly from 14 April until the end of July 1992 to locate carcasses for retrieval and examination and to record presence and behavior of live dolphins in the bay ecosystem. The aerial surveys also provided the opportunity to locate areas of unusual water coloration or potential algal blooms, define the land-use characteristics of the area surrounding the bay ecosystem in terms of agricultural and industrial activities, and determine whether any fish kills or mortalities of other taxa were occurring simultaneously with the dolphins' deaths. Information from numerous local, state, federal, and academic sources regarding recent algal blooms, weather patterns, hazardous chemical spills, industrial discharges, known pollution sources, and dredging activities was collected and evaluated immediately. The available data for the stranded dolphins, including species, gender, length, and stranding location, were examined for any identifiable trends in those parameters. The reported number of dolphin deaths was compared to historical databases maintained by the Southeast U.S. Marine Mammal Stranding Network.

Local field biologists, fishermen, and other residents were informally interviewed concerning their past experiences and observations with the local dolphin population, in an effort to determine whether the number of reported strandings was different from past years. The local stranding network was also consulted about changes in member participation.

Efforts were focused on identifying potential factors responsible for the increased mortality rate, using sample collection and analysis from the dolphin carcasses and a thorough environmental/ecotoxicological evaluation. All samples collected and analyzed were treated as case evidence, using strict legal documentation and chain-of-custody procedures, in order to withstand scrutiny under potential legal action. During this mortality event, an official federal law enforcement case was opened, and enforcement agents from federal and state agencies occasionally assisted the on-site coordinator with field activities.

Examination of Dolphins

A condition code system used by the stranding network classifies dolphin carcasses into five levels of decomposition, with code 1 being a live animal and code 5 being mummified or skeletal remains. When numerous animals were reported in a single day, this system was used to prioritize their examination. Attempts were made to evaluate the parameters listed in Table 1 during thorough carcass examinations on all stranded dolphins. Whenever possible after 12 April, lung, liver, kidney, and blubber were collected, but generally the organs taken were barely identifiable due to the advanced state of decomposition. These samples were considered unsuitable for existing analyses of any type.

In order to define the age structure of the dolphins that died during this mortality event and to more accurately interpret toxicological results, teeth were collected from the stranded animals for age determination. The results were not provided for this report, but will be published separately. Similarly, skulls were archived for morphometrics, but examination for determination of inshore/offshore characteristics has not been completed.

Commercial fishermen and local biologists that spend much of their time working in the bay ecosystem were informally interviewed concerning their observations of live dolphins. They were asked if live dolphins were seen

in the study area since December, and if the animals exhibited any unusual characteristics. If unusual characteristics were observed, people were asked approximately when the signs were first noticed.

In addition to carcass examination, live dolphins were located and followed to detect unusual behavior. A marine mammal behavioral specialist was contracted to observe live dolphins in the bay ecosystem for a period of approximately two weeks.

Ecotoxicological Evaluation

Mortalities of multiple taxa, including birds and fish, were detected by aerial survey early in the investigation, resulting in an expansion of the investigation's scope to include a thorough environmental risk assessment. The fish and bird mortalities appeared to overlap spatially and temporally with dolphin strandings. During the initial aerial survey, one bay location at Sand Point, Texas was identified where dead fish, primarily hardhead catfish (*Arius felis*), black drum (*Pogonias cromis*), laughing gulls (*Larus atricilla*), and four bottlenose dolphins, were observed on a two-mile stretch of beach between Lavaca and Keller Bays. Ground observations indicated that the carcasses were scavenged by fire ants, making the laughing gulls, catfish, and black drum unsuitable for sample collection and chemical analysis. Texas Parks and Wildlife Department personnel responded to the fish kill and conducted their usual survey. No indication of trauma was apparent from the external condition, and examination by radiography showed that the birds from the Sand Point location had not been shot. Additional reports were received of dead fish and birds in other isolated locations but, due to logistical problems, those reports were not confirmed and the over-washing of the narrow beaches at high tide often prevented verification of reported mortality.

Meteorological information suggested that the area had experienced unusually heavy rainfall between December 1991 and April 1992, which drastically reduced salinity in the bay ecosystem. The excessive rainfall coincided with spring pesticide application periods in local agricultural operations. Because of the involvement of multiple taxa and altered environmental conditions, an ecotoxicological field study was designed and executed to monitor the bay area for the presence of chemical or biological stressors that could adversely affect marine life in the bay ecosystem. It is important to note that this study took place after 14 April, several weeks after the peak dolphin mortality period had occurred during March.

The ecotoxicological evaluation was focused on detecting and quantifying stressors such as biotoxins and anthropogenic chemical contaminants present in water, sediment, and biota (tissue). A total of 17 stations were selected for ecotoxicological evaluation, covering a geographical area from Corpus Christi, Texas, north toward San Luis Pass, Texas (Fig. 2; Table 2). Station locations were selected based upon:

1) The occurrence of multiple dolphin strandings along with mortalities of other species, including birds and fish. Initial field investigations and aerial overflights identified several regions where numerous bottlenose dolphin strandings had occurred during the period, including Sand Point in Lavaca Bay, the shore of the Intracoastal Waterway west of Port O'Connor, and an

Table 1
List of parameters recommended for use in examination of stranded *T. truncatus* along the Texas coast. Data records using video and photography are recommended.

	Condition code				
Sample type	1[1]	2[2]	3[3,4]	4	5
Clinical					
— respiration rate	x[5]				
— heart rate	x				
— urine sample	x				
— blood sample	x	x			
Life history					
— stomach and contents		x	x	x	x
— jawbone/teeth		x	x	x	x
— skull		x	x	x	x
— rib/spinal section		x	x	x	x
— morphometrics		x	x	x	
— reproductive organs		x	x	x	
— parasites		x	x	x	
— blubber thickness		x	x		
Parasitology	x	x	x	x	
Microbiology	x	x			
Toxicology		x	x		
Histopathology		x	x		
Necropsy		x	x		
— external observation					
— site observations		x	x		
— ecto-parasites		x	x		
— gross lesions		x	x		
— gross dissection					
— parasites		x	x		
— gross lesions		x	x		
— major organs		x			

[1] Code 1 animals are assigned to Code 2 at death.
[2] Heart blood collected shortly after death.
[3] Morphometrics should be taken on late Code 3 and Code 4 animals only for those measurements that are not altered by decomposition of the soft body tissues.
[4] Some tissues degrade quickly and will not yield samples suitable for histopathology; judgment is required.
[5] x = sample/measurement taken.

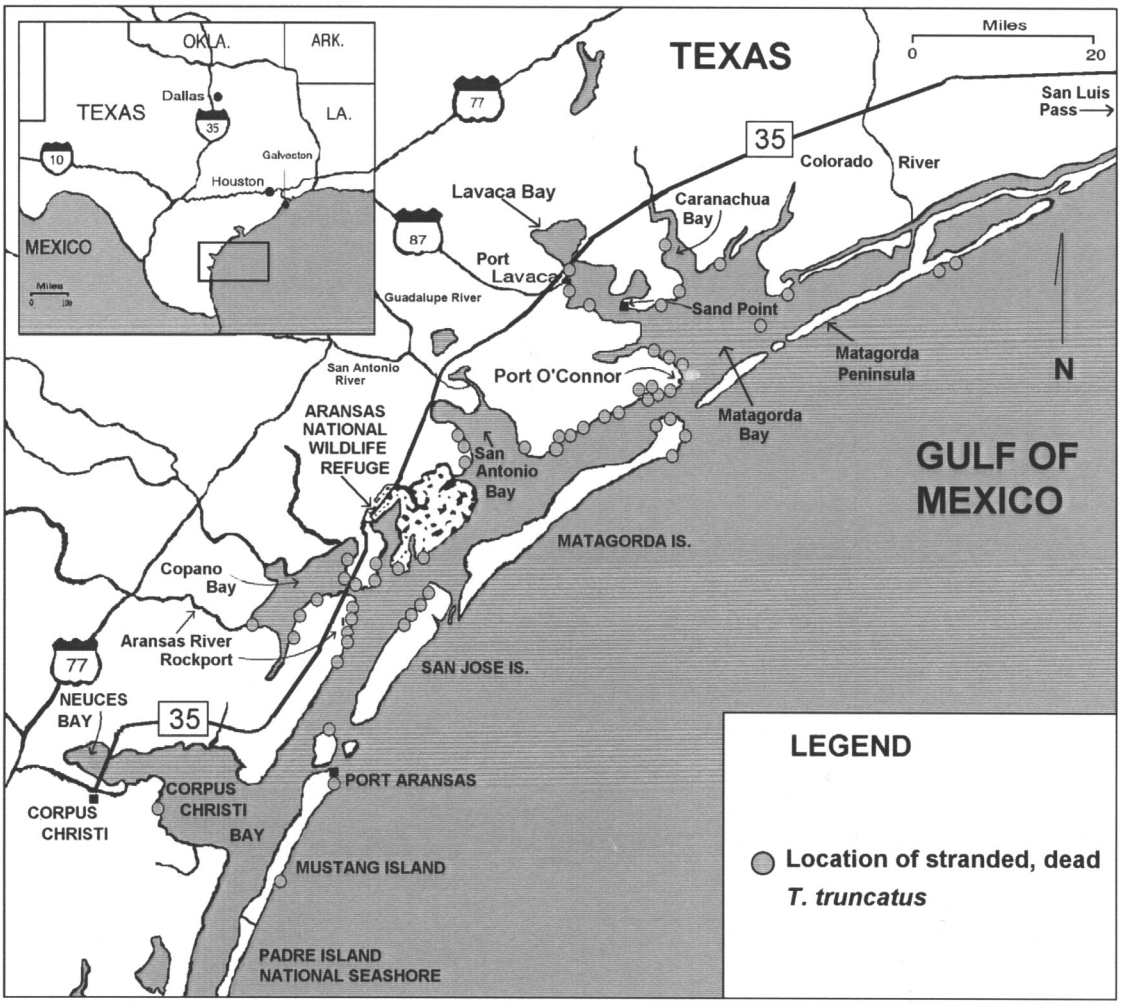

Figure 1

Representative stranding locations for a portion (57/240) of the dolphins stranded during the unusual 1992 mortality event. Note that the majority of the strandings occurred in coastal and inland bay regions rather than in offshore waters.

area in the vicinity of Rockport, Texas, near Copano Bay (Fig. 2);
2) Known point sources of pollution including industrial areas and active dredge disposal areas;
3) Critical areas adjacent to agricultural drainage basins; and
4) Known areas where domoic-acid-producing phytoplankton could be found.

The parameters measured at each station (Table 3) included:

1) Gross observations and photographs of any dead or stranded animals, including fish, birds, and marine mammals (all stations);
2) Measurement of general water quality parameters including water temperature (°C), pH, dissolved oxygen (mg/L and % saturation), and salinity (ppt) for all stations;
3) Surface water samples analyzed for priority pollutants established by the Environmental Protection Agency (EPA), including trace metals, polycyclic aromatic hydrocarbons (PAHs), and pesticides (all stations);
4) Sediment and tissue (finfish) samples analyzed for priority pollutants, including trace metals, PAHs, and pesticides (selected stations); available dolphin livers were also analyzed for mercury (Hg) due to the known Hg contamination in Lavaca Bay;
5) Water and finfish samples collected for brevetoxin analysis and icthyotoxicity tests (selected stations);
6) Oyster (*Crassostrea virginica*) samples collected and analyzed for domoic acid by J. C. Wekell (NMFS, Northwest Fisheries Science Center) using methods described by Hatfield et al. (1994) (selected stations);

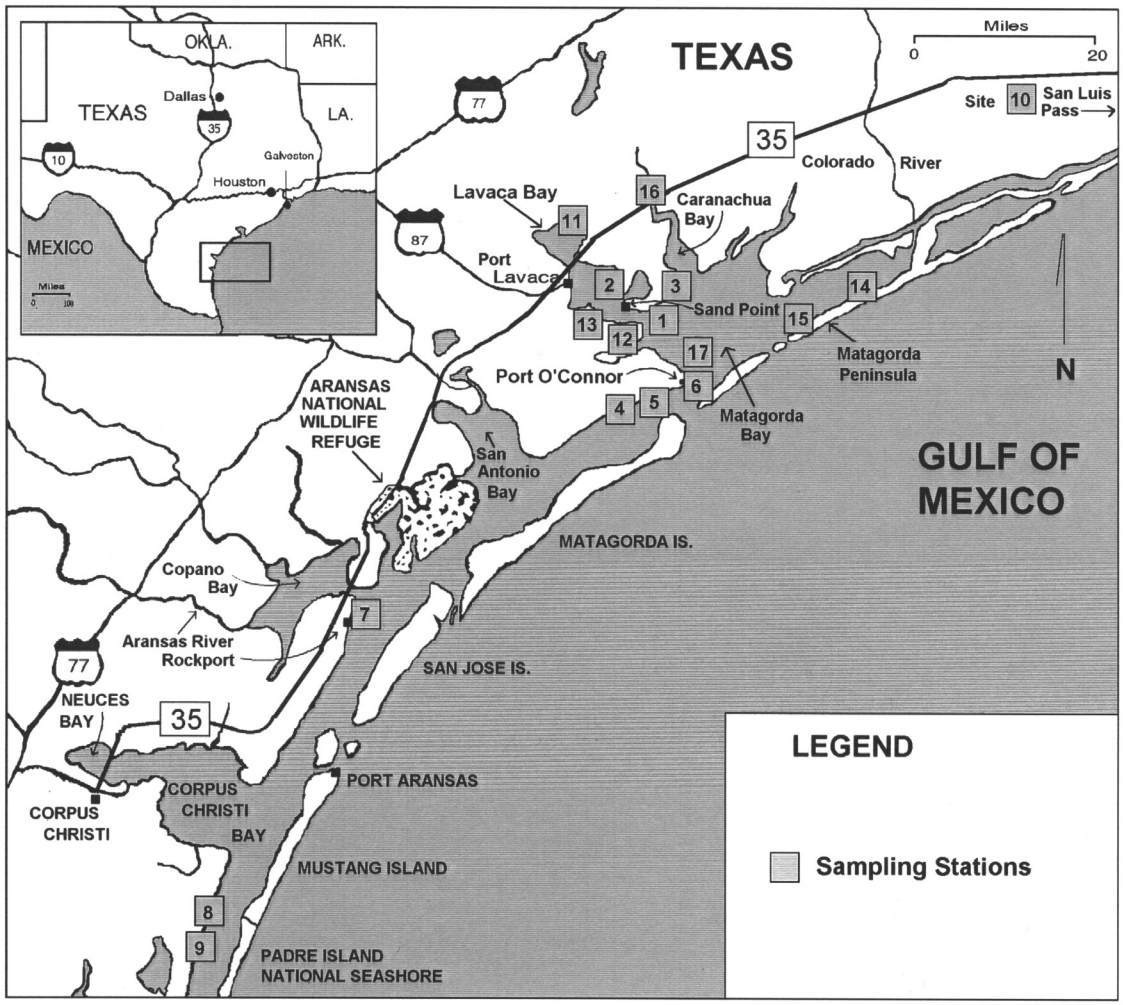

Figure 2
Station locations for sampling of water, sediments, and selected biota during the ecotoxicological assessment.

7) Plankton tows for domoic acid-producing diatoms of the genus *Pseudo-nitzchia* made by hand (50 m/site) and examined using light microscopy (selected stations);
8) Laboratory toxicity tests conducted with larval grass shrimp, *Palaemonetes pugio*, exposed for 96 h to surface water samples that tested positive for pesticides (selected stations); and
9) Morphometrics and external observations on stranded dolphins (stranding sites only).

Surface water samples (3.5 L) were initially screened for the presence of selected pesticides (polychlorinated biphenyls [PCBs], cyclodiene insecticides, triazine herbicides, and the insecticide aldicarb) using polyclonal antibody ELISA assays (Envirogard® Test Kits). Lower limits of detection (LLOD) in these assays ranged from 0.100–5 µg/L.

Surface water samples were extracted with dichloromethane, cleaned with florisil, and analyzed for priority pollutants (PAHs, PCBs, and pesticides) using gas chromatography (GC)-mass spectrometry (MS), GC-electron capture detection (ECD), and GC-nitrogen-phosphorus detection (NPD) using techniques described by Scott et al. [2] and Bush et al. (1978). Lower limits of detection ranged from 3–1,000 µg/L for pesticides and PCBs and from 2–8 µg/L for PAHs. Similar methods of analysis were used for sediment and tissue samples.

Analyses of surface waters, sediments, and tissues included priority pollutant trace metals (established by EPA) using both atomic absorption spectrometry (AAS) and intracoupled plasma spectrometry (ICPS). Dolphin

[2] Scott, G. I., M. H. Fulton, M. C. Crosby, P. B. Key, J. W. Daugomah, J. T. Waldren, E. D. Strozier, C. J. Louden, G. T. Chandler, T. F. Bidleman, K. L. Jackson, T. W. Hampton, T. Hoffman, A. Shultz, and M. Bradford. 1992. Agriculture insecticide runoff effects on estuarine organisms: correlating laboratory and field toxicity tests, ecophysiology bioassays and ecotoxicological biomonitoring. Final Report prepared for EPA, Gulf Breeze Environmental Research Laboratory, Gulf Breeze, FL 32561, 281 p.

Table 2

List of stations sampled for surface water pesticide analysis during 1992. During this sampling period locations were focused on regions where dolphin strandings occurred. For 1993 data see Pennigton (1994). (Station locations can be seen on Fig. 2.)

Station number	Station classification[1]	Location[2]
1	3	Sand Point, E end
2	3	Sand Point, W end
3	3	Sand Point, E end where road enters bay
4	3	Intracoastal Waterway (ICW), S of Port OConnor, W bank
5	3	ICW, S of Port O'Connor, E bank at dredge disposal pipes
6	3	Port O'Connor, E of Texas Parks and Wildlife Field Office
7	3	Fulton Beach
8	3	Corpus Christi, power plant intake
9	3	Corpus Christi, ponds at power plant
10	3	Galveston Bay, southern pass
11	3	Upper Lavaca Bay, park and boat landing
12	3	Lavaca Bay, dredge spoil island
13	3	Lavaca Bay, Magnolia Beach
14	3	North Matagorda Bay, live capture site
15	3	Middle Matagorda Bay, Greens Bayou live capture
16	2	Caranachua Bay
17	3	Port O'Connor jetty

[1] 1=agricultural sites; 2=stream sites downstream from agricultural areas; 3=bay sites.
[2] E=East; W=West; S=South.

liver tissues were freeze-dried and weighed into Teflon® digestion vessels with nitric acid. After the vessels were sealed, they were microwave extracted and brought to a volume of 25 ml. The samples were analyzed for heavy metals by cold vapor AAS. The lower level of detection (LLOD) for the method was 2.5 µg/g. The recovery of a standard reference material (SRM) was 83.6±8.0% (SD). QA/QC criteria were based on the SRM (Dogfish Liver Tissue [DOLT-1]) from NRC Canada Marine Analytical Chemistry Standards. For every two samples, one SRM and one blank was run. Acceptable analytical results were obtained when the SRM fell within the reported 95% CI for DOLT-1 and the blank was zero.

Results

Initial Field Investigation

Comparison of stranding data to historical databases indicated that the number and nature of the mortalities occurring in the Texas bay system were unprecedented. During 1992, a total of 240 *Tursiops truncatus* stranded along the Texas coast (Fig. 3). More than half of these strandings occurred in March (99 strandings) and April (61 strandings) (Fig. 3) in remote coastal regions of Aransas and Calhoun counties. In the southeastern U.S., 645 cetacean strandings occurred the previous year (1991), including 448 (69%) bottlenose dolphins[3]. A total of 139 cetacean strandings occurred in Texas during 1991, including 134 (96%) bottlenose dolphins; and in Aransas and Calhoun counties, a total of 5 and 8 bottlenose dolphins were reported stranded respectively that year. From 1982–91, the number of annual bottlenose dolphin strandings in Calhoun and Aransas counties ranged from 0–12/year; this indicates that the number of dolphin strandings in the counties during early 1992 was a dramatic increase.

Helicopter surveys of the coastline established that the extent of the event did not exceed the number of animals reported or extend beyond the area suspected to be involved. Evaluation of available data from stranded dolphins indicated that both genders and all size ranges of bottlenose dolphins (*Tursiops truncatus*) were affected, reducing the likelihood that disease factors limited to a specific gender or to certain age structure were involved. Basic data for strandings that occurred in Texas from January through April 1992 are listed in Table 4.

From the stranding location data and area nautical charts, it was apparent that a large number of dolphin

[3] Odell, Daniel K. Southeast U.S. Marine Mammal Stranding Network, Sea World Research Institute, 7007 Sea World Dr., Orlando, FL 32821. Unpubl. data.

Table 3

List of parameters measured during the field investigation of known pollution sources at sites with a heavy concentration of stranded *T. truncatus*. Matrices sampled include water (W), sediment (S), tissue (T), and phytoplankton (P).

Parameter	Matrix	LLOD (ppm= mg/L (W); mg/Kg (T or S))
Physiochemical water quality		
— Temperature	W	
— Salinity/conductivity	W	
— Dissolved oxygen	W	
— pH	W	
Chemical contaminants		
— Trace metals (Ag, As, Be, Cd, Cr, Cu, Hg, Pb, Ni, Sb, Th, Zn)	W,S,T	0.1–1.0
— Polycyclic aromatic hydrocarbons and other priority pollutants[1]	W,S,T	0.004–0.012
— Persistent pesticides[2]	W,S,T	0.00001–0.0003 (W) 0.005–0.1 (S) 0.01–1.0 (T)
— Polychlorinated biphenyls (aroclors 1016,1221, 1232, 1242,1248 and1260)	W,S,T	0.3
Biotoxins		
— Brevetoxin and domoic acid	W,T	
— Brown tide	W,P	

[1] Compounds include: Phenol; Bis (2-chloroethyl)ether; 2-chlorophenol; 1,3 dichlorophenol; 1,4 dichlorophenol; benzyl alcohol; 1,2 dichlorobenzene; 2 methylphenol; Bis (2-chloroisopropyl)ether; 4-methylphenol; N-nitro-di-n-propylamine; hexachloroethane; nitrobenzene, *isophorene*; 2-nitrophenol; benzoic acid; Bis (2-chloroethoxy)methane; 2,4 Dichlorophenol; 1,2,4 trichlorobenzene; naphthalene, 4-*chloroaniline*; haxachlorobutaniene; 4-chloro-3-methylphenol; 3-methyl naphthalene; hexachloropentadiene; 2,4,6 trichlorophenol; 2,4,5 trichlorophenol; 2 chloronaphthalene; acenaphthalene; 2,6 dinitrotoluene; 3 nitroanaline; acenaphthene; 2,4 dinitrophenol; 4-nitrophenol; dibenzofuran; 2,4 dinitrotoluene; diethylphthalate; 4-chlorophenyl-phenylether; flourene; 4-nitroaniline; 4,6-dinitro-2-methylphenol; N-nitrosodiohenylamine; 4-bromophenyl-pheny ether; hexachlorobenzene; pentachlorophenol; phenanthrene; anthracene; Di-N-butylphthalate; fluoranthene; pyrene; butylbenzylphthalate; 3,3-dichlorobenzidine; benzo (a) anthracene; chrysene; bis (2-ethylhexyl) phthalate; di-N-octylphthalate; benzo (b) fluoranthene; benzo (k) fluoranthene; benzo (a) pyrene; dibenzo (a,h) anthracene and benzo (g,h,i) perylene.

[2] Compounds include: aldrin; α-BHC; βBHC; γBHC; σBHC; chlordane; 4,4-DDE; 4,4-DDT; Diazinon; dielderin; Dursban; endosulfan (I, II and sulfate); endrin; permethrin; endrin aldehyde; EPN; heptachlor; heptachlor-epoxy; kelthane; lindane; malathion; methyl parathion; mirex; toxaphene; methoxychlor; HCB; *pentachloroanisole*; parathion; phosdrin; *Rabon*; *Trichloron*; atrazine; balin; Bravo; bromacil; dervinol; dimethoate; Dual; Eptam; Goal; hexazinone; Lasso; Ordam; oxadiazon; Paarlan; Propachlor; Propazine; Prowl; Roneet; Sencor; simazine; Sutan; Terbacil; Tillam; Tolban; Treflan and Vernam,.

deaths appeared to be concentrated along the coasts of the inner bay system from Caranachua Bay southwest to Copano Bay, but not along the outer coast of the barrier islands along the Gulf of Mexico (Fig. 1). The location of the barrier islands (Matagorda Island and Matagorda Peninsula) and the small size and number of tidal inlets greatly reduced the likelihood that such a large number of dolphins were washing into the bay system from offshore. Dolphin skulls archived from the die-off are being examined to determine if morphology was characteristic of inshore or offshore animals. Also, observations that very few dolphins had stranded on the Gulf coast of the barrier islands suggested that the dolphins probably died within the bay areas. The exact location of the animals at the time of death could not be fully established because no stranded dolphins were observed alive. Live dolphins were sighted throughout the bay system during the event, and locals indicated that live dolphins were often seen in the area year-round.

According to information collected from state, regional, and local authorities, as well as academic institutions, there were no reported oil/hazardous materials spills or other chemical discharges, no known harmful algal blooms other than a brown tide in Laguna Madre (further to the southwest), and no increase in stranding rates along other regions of the coast. This suggested that the unusual mortalities were part of an isolated incident of unknown cause limited to a small geographical region. Known pollution sources were identified in several bay areas. For example, Lavaca Bay has been contaminated with mercury since the 1960s, and portions of the bay have been closed to fishing due to high mercury levels. The Lavaca Ship Channel was dredged during early 1992; no information was available regarding the effects of dredging on the mercury levels in the water column. Isolated incidences involving chemical discharge from area industries were identified, but these were ruled out as potential causes because of the type and quantity of the chemicals.

Two common characteristics were apparent among many of the dolphin carcasses found prior to April 14. A gray, pasty substance (Plate 1) and severe skin blisters (Plate 2) were visible on several of the stranded dolphins. These conditions did not appear to have occurred post-mortem. It was necessary to try to determine when these dermal conditions first developed. Interviews with local individuals helped provide this information. Several shrimp fishermen reported observations of live dolphins with numerous blisters and sores as well as cloudy eyes. Local biologists who regularly work on the bay also reported observations of live dolphins covered with a gray, pasty substance and exhibiting blisters. Information obtained from these sources indicated that dolphins began exhibiting these characteristics as early as January 1992, immediately prior to increased mortality from February through early May 1992.

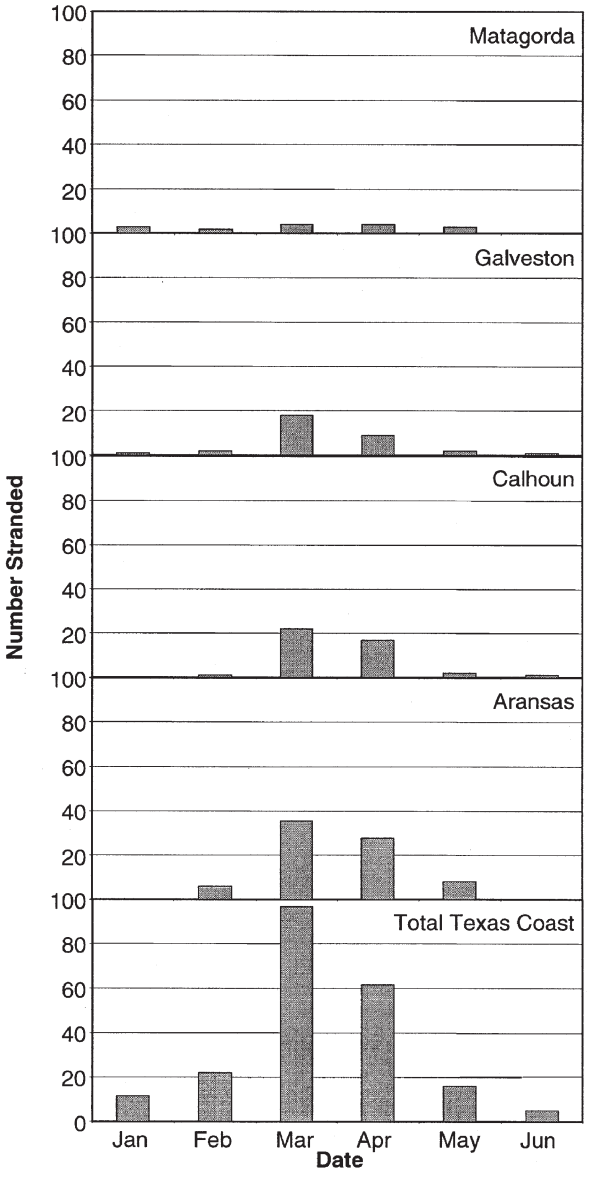

Figure 3
Stranding rates for each coastal county and the entire state of Texas for Jan.–June 1992. Note the elevated mortality rates in Aransas, Calhoun, Galveston, and Matagorda counties and the similarities in the pattern of mortality in each region.

Examination of Dolphins

The decomposed condition of dolphins precluded analysis of tissues for viruses, bacteria, organic contaminants, biotoxins, and histopathology. Several carcasses that would have been suitable for analyses were stranded prior to mid-April, but were not sampled due to the overwhelming number of strandings to which network members responded (Plates 1 and 2). Most carcasses were in a condition unsuitable for thorough sample collection (Plate 3), and only length and location data were recorded. Several attempts were made to isolate microbes from the gray substance and fluid from the blisters of dead dolphins, but in all cases, the sample quality was compromised by decomposition; culture analysis identified microorganisms primarily associated with decomposition. All stranded dolphins examined from the bay ecosystem were between late code 3 and code 5 condition. Animals were retrieved immediately after stranding reports were received. Only two dolphins in code 2 (freshly dead) condition were recovered during the period of investigation; they were stranded in the Galveston area, outside the area of concern, and not considered to be directly involved in the local event. The two dolphins had none of the external conditions previously described.

The behavioral specialist contracted during May to observe live dolphins for signs of stress identified no unusual behavior, and no live dolphins were observed with skin lesions.[4] It should be noted that the behavioral observations were made after the mortality rate had begun to decline and after surface water salinity had increased.

Because the dolphins that stranded during the field investigation period did not provide the quality of tissues needed for accurate evaluation of potential disease factors and biomarkers for certain contaminant exposures, a live capture operation was planned as quickly as possible by the NMFS/SEFSC. The intent was to capture apparently healthy dolphins in the bay ecosystem and collect relatively noninvasive samples for selected health parameters without harming the animals. The results

[4] Wursig, Berendt. Texas A&M University, College Station, TX 77843. Personal commun.

Table 4

Stranding information provided by the Texas Marine Mammals Stranding Network (G. Worthy) for the Texas coast during the mortality event reported here.

Region #	Species	Sex	Length	Date reported	Lat	Long
CC108	*T. truncatus*	U		1/4/92	27°16'.6"	97°20'.85"
CC109	*T. truncatus*	U	118.75	2/2/92	27°24'.3"	97°18'.3"
CC110	*T. truncatus*	F	242	2/12/92	27°20'	97°19'.8"
CC111	*T. truncatus*	F	271.8	3/2/92	27°15'.7"	97°21'.1"
CC112	*T. truncatus*	M	257.8	3/12/92	27°28'.6"	97°16'.4"
CC113	*T. truncatus*	U	218	3/26/92	26°58'	97°22'.6"
CC114	*T. truncatus*	F	216.45	3/26/92	26°58'	97°22'.6"
GA419	Unknown	M	310	1/6/92	29°3'.52"	95°8'.3"
GA420	*T. truncatus*	M	210	1/11/92	29°32'.9"	94°23'.2"
GA421	*T. truncatus*	M	103	1/29/62	28°56'.7"	95°17'.3"
GA422	*T. truncatus*	F	98.5	2/22/92	29°17'.2"	94°47'.5"
GA423	*T. truncatus*	M	215	2/24/92	29°28'.7"	94°34'.2"
GA424	*T. truncatus*	U		2/27/92	28°52'.5"	95°22'.7"
GA425	*T. truncatus*	M	224	2/28/92	28°55'.8"	95°18'.35"
GA426	*T. truncatus*	F	174	3/3/92	28°58'.6"	95°15'.1"
GA427	*T. truncatus*	M	248	3/9/92	29°3'.7"	95°8'
GA428	*T. truncatus*	M	89	3/6/92	29°8'.7"	95°2'.8"
GA429	*T. truncatus*	F	98	3/6/92	29°11'.72"	94°57'.9"
GA430	*T. truncatus*	M	110	3/8/92	29°27'.2"	94°37'.6"
GA431	*T. truncatus*	M	162	3/8/92	29°10'.8"	94°58'.5"
GA432	*T. truncatus*	M	249	3/9/92	29°11'.1"	94°57'.9"
GA433	*T. truncatus*	M	111	3/10/92	29°9'.8"	95°1'
GA434	*T. truncatus*	M	227	3/18/92	28°55'.2"	95°20'.5"
GA435	*T. truncatus*	M	107	3/21/92	29°29'.1"	94°33'.2"
GA436	*T. truncatus*	F	255	3/21/92	29°32'.05"	94°25'.6"
GA437	*T. truncatus*	F	102	3/21/92	29°32'.05"	94°25'.6"
GA438	*T. truncatus*	F	237	3/21/92	29°32'.2"	94°24'.91"
GA439	*T. truncatus*	M	259	3/22/92	29°29'	94°55'.1"
GA440	*T. truncatus*	M	247	3/24/92	28°56'.8"	95°17'.21"
GA441	*T. truncatus*	U		3/27/92	29°15'.35"	94°50'.75"
GA442	*T. truncatus*	M	101	3/28/92	28°7'	94°34'.2"
GA443	*T. truncatus*	M	118	3/28/92	29°13'.9"	94°53'.1"
GA444	*T. truncatus*	U	106	3/29/92	29°21'.9"	94°45'.3"
GA445	*T. truncatus*	M	249	3/29/92	29°30'.3"	94°29'.2"
GA446	*T. truncatus*	M	255	3/30/92	29°40'.41"	94°49'.4"
GA447	*T. truncatus*	M	109	3/30/92	29°15'	94°51'.3"
GA448	*T. truncatus*	M	260	3/31/92	29°13'	94°54'.6"
GA449	*T. truncatus*	M	105	3/31/92	29°9'.2"	94°9'
GA450	*T. truncatus*	F	130	4/2/92	29°13'.7"	94°53'.6"
GA451	*T. truncatus*	F	111	4/6/92	29°18'.33"	94°46'.1"
GA452	*T. truncatus*	F	105	4/9/92	29°17'.28"	94°47'.5"
GA453	*T. truncatus*	M	105	4/11/92	29°16'.6"	95°48'.5"
GA454	*T. truncatus*	U		4/13/92	29°30'.9"	94°29'.1"
GA455	*T. truncatus*	M	256	4/15/92	29°10'.71"	94°58'.1"
GA456	*T. truncatus*	M	248	4/19/92	29°2'	95°3'.7"
GA457	*T. truncatus*	F	218	4/17/92	29°16'.82"	94°48'.1"
GA458	*T. truncatus*	F	241	4/19/92	29°28'.8"	94°34'
GA459	*T. truncatus*	U	142	4/22/92	29°32'.45"	94°24'.45"
PA233	*T. truncatus*	U	204	1/6/92	27°43'.3"	97°20'.5"
PA234	*T. truncatus*	F	284	1/8/92	27°40'.3"	97°10'.2"
PA235	*T. truncatus*	M	256	1/27/92	28°3'	97°1'.8"
PA236	*T. truncatus*	F	230	2/5/92	27°37'.1"	97°12'.5"
PA237	*T. truncatus*	M	170	2/5/92	28°3'.3"	97°2'
PA238	*T. truncatus*	F	234	2/7/92	27°49'.8"	97°7'.3"
PA239	*T. truncatus*	U	281	1/29/92	27°90'.3"	97°10'
PA240	*T. truncatus*	F	239	2/10/92	28°2'	97°1'.5"

continued

Table 4 (continued)

Region #	Species	Sex	Length	Date reported	Lat	Long
PA241	*T. truncatus*	F	200	2/8/92	27°51'	97°2'.6"
PA242	*T. truncatus*	F	88.5	2/13/92	27°45'.765"	97°6'.48"
PA243	*T. truncatus*	M	106	2/13/92	27°36'.02"	97°12'.5"
PA244	*T. truncatus*	F	248	2/18/92	27°52'.7"	97°1'.6"
PA245	*T. truncatus*	M	260	2/19/92	27°55'	97°4'.7"
PA246	*T. truncatus*	F	206	3/5/92	28°17'.4"	96°48'.5"
PA247	*T. truncatus*	F	175	3/4/92	27°40'	97°10'.5"
PA248	*T. truncatus*	M	270	3/5/92	28°18'	96°48'
PA249	*T. truncatus*	F	228	3/5/92	28°18'	96°48'
PA250	*T. truncatus*	M	258	3/9/92	28°4'	97°2'
PA251	*T. truncatus*	M	270	3/9/92	28°6'.1"	97°1'.4"
PA252	*T. truncatus*	U		2/28/92	28°8'.5"	96°53'
PA253	*T. truncatus*	U		3/2/92	28°7'	96°54'
PA254	*T. truncatus*	U		3/2/92	28°3'.5"	96°57'.5"
PA255	*T. truncatus*	U		3/2/92	28°3'.5"	96°57'.5"
PA256	*T. truncatus*	U		3/2/92	28°3'.5"	96°57'.5"
PA257	*T. truncatus*	M	217	3/4/92	28°3'.8"	97°1'.9"
PA258	*T. truncatus*	F	224	3/16/92	28°5'	97°2'
PA259	*T. truncatus*	F		3/17/92	28°5'.7"	97°1'.9"
PA260	*T. truncatus*	M	101	3/17/92	28°7'	96°59'
PA261	*T. truncatus*	M	120	3/17/92	28°0'	97°3'
PA262	*T. truncatus*	F	252	3/18/92	28°8'	97°0'
PA263	*T. truncatus*	F	236	3/19/92	28°10'.2"	47°1'
PA264	*T. truncatus*	M	272	3/19/92	28°8'	97°0'
PA265	*T. truncatus*	M	199	3/19/92	28°5'.6"	97°3'.2"
PA266	*T. truncatus*	M	250	3/20/92	28°2'.8"	97°1'.6"
PA267	*T. truncatus*	F	269	3/20/92	28°5'.5"	97°2'
PA268	*T. truncatus*	M	225	3/24/92	28°53'.4"	97°2.8"
PA269	*T. truncatus*	F	245	3/23/92	28°7'.5"	96°58'.8"
PA270	*T. truncatus*	F	212	3/24/92	28°5'.1"	97°2'
PA271	*T. truncatus*	M	222	3/24/92	28°5'.1"	97°2'
PA272	*T. truncatus*	M	229	3/24/92	28°6'.4"	97°1'.3"
PA273	*T. truncatus*	F	208	3/14/92	28°5'.1"	97°4'.5"
PA274	*T. truncatus*	F	267	3/24/92	28°5'.2"	97°13'
PA275	*T. truncatus*	U	154	3/25/92	28°9'	97°1'.8"
PA276	*T. truncatus*	M	285	3/30/92	28°6'.6"	97°1'.3"
PA277	*T. truncatus*	M	261	3/31/92	28°0'.4"	97°3'.4"
PA278	*T. truncatus*	M	261	3/31/92	28°3'.1"	97°1'.8"
PA279	*T. truncatus*	F	264	3/30/92	28°14'.9"	96°47'.2"
PA280	*T. truncatus*	M	210	3/19/92	28°17'.5"	96°48'.5"
PA281	*T. truncatus*	U	269	4/1/92	28°1'.3"	97°2'.8"
PA282	*T. truncatus*	M	123	4/2/92	28°2'.5"	97°1'.58"
PA283	*T. truncatus*	M	96	4/4/92	27°46'.9"	97°23'.3"
PA285	*T. truncatus*	M	190	4/6/92	27°50'.5"	97°2'.7"
PA286	*T. truncatus*	U		4/3/92	27°59'.4"	97°4'
PA287	*T. truncatus*	F	238	4/7/92	28°3'.9"	97°2'
PA288	*T. truncatus*	U		4/7/92	27°54'.4"	97°3'.5"
PA289	*T. truncatus*	F	224	4/9/92	28°3'.1"	97°1'.9"
PA290	*T. truncatus*	F	247	4/9/92	28°1'.5"	97°2'.8"
PA291	*T. truncatus*	M	179	4/9/92	28°4'.8"	97°5'.5"
PA292	*T. truncatus*	M	263	4/11/92	28°39'.4"	97°10'.8"
PA293	*T. truncatus*	U	238	4/10/92	28°18'	96°48'
PA294	*T. truncatus*	M	98	4/11/92	28°2'.5"	97°1'.5"
PA295	*T. truncatus*	U		4/13/92		
PA296	*T. truncatus*	U	241	4/14/92	28°3'.7"	97°7'.7"
PA297	*T. truncatus*	U	237	4/15/92	28°9'	97°1'.5"
PA298	*T. truncatus*	F	115	4/17/92	28°0'.8"	97°3'.2"

continued

Table 4 (continued)

Region #	Species	Sex	Length	Date reported	Lat	Long
PA299	*T. truncatus*	F	239	4/20/92	28°5'.2"	97°3'.9"
PA300	*T. truncatus*			4/28/92		
PA301	Unknown	M	230.7	4/29/92	28°7'.35"	96°59'.1"
PA302	*T. truncatus*	U		4/27/92	28°1'.9"	97°1'.5"
PA 303	*T. truncatus*	U		3/24/92	28°8'	96°52'.5"
PA 304	*T. truncatus*	U		3/24/92		
PA 305	*T. truncatus*	U		4/28/92	27°49'.38"	97°8'.92"
PA 306	*T. truncatus*	M	223	4/30/92	27°52'.38"	97°5'.87"
PA 307	*T. truncatus*	F	180.4	4/26/92	28°11'.83"	97°1'.3"
PO176	*T. truncatus*	M	252	1/22/92	28°45.8'	95°37.2'
PO177	*T. truncatus*	M	254	1/22/92	28°37.5'	95°54.3'
PO178	*T. truncatus*	M	189	1/24/92	28°35.7'	95°58.8'
PO179	Unknown cetacean			2/9/92	28°27.8'	96°16'
PO180	*T. truncatus*	F	236	2/25/92	28°40.2'	95°48.1'
PO181	*T. truncatus*	F	210	2/25/92	28°36.05'	95°58'
PO182	*T. truncatus*	U	211	2/20/92	28°24.4'	96°28.7'
PO183	*T. truncatus*	U		3/10/92	28°22.6'	96°42'
PO184	*T. truncatus*	U	250	3/10/92	28°22.2'	96°28.2'
PO185	*T. truncatus*	U	245	3/13/92	28°26.2'	96°24.8'
PO186	*T. truncatus*	U	295	3/13/92	28°26.2'	96°24.7'
PO187	K. breviceps	U	240	3/18/92	28°7.3'	96°46.2'
PO188	*T. truncatus*	M	212	3/20/92	28°44.2'	96°23.9'
PO189	*T. truncatus*	U	154	3/20/92	28°33.7'	96°32.3'
PO190	*T. truncatus*	U	205	3/22/92	28°33.7'	96°32.3'
PO191	*T. truncatus*	U		3/19/92	28°20.7'	96°47.6'
PO192	Unknown	U		3/19/92	28°20.7'	96°47.6'
PO193	*T. truncatus*	M	215	3/24/92	28°18.8'	96°37.6'
PO194	*T. truncatus*	U	280	3/20/92	28°20.7'	96°35.5'
PO195	*T. truncatus*	U	255	3/20/92	28°21.3'	96°34.7'
PO196	*T. truncatus*	M	205	3/24/92	28°23.4'	96°31.5'
PO197	*T. truncatus*	U	245	3/24/92	28°23.8'	96°30.7'
PO198	*T. truncatus*	M	228	3/24/92	28°24.4'	96°29.6'
PO199	*T. truncatus*	U	240	3/24/92	28°26'	96°25.7'
PO201	*T. truncatus*	F		3/27/92	28°36.8'	96°56.3'
PO202	*T. truncatus*	U		3/27/92	28°36.8'	96°56.3'
PO203	Unknown dolphin			3/26/92	28°22.5'	96°23.5'
PO204	*T. truncatus*			3/26/92	28°26'	96°25.3'
PO205	*T. truncatus*	U	238	3/27/92	28°41.6'	96°39.8'
PO206	*T. truncatus*	M	214	3/15/92	28°19'	96°37.4'
PO207	*T. truncatus*	M	270	3/20/92	28°19.7'	96°36.5'
PO208	*T. truncatus*	M	275	3/20/92	28°20'	96°36.2'
PO209	*T. truncatus*	U		3/20/92	28°21.1'	96°35'
PO210	*T. truncatus*	F	113		28°19.3'	96°37'
PO211	*T. truncatus*	U		3/15/92	28°18.9'	96°37.5'
PO212	*T. truncatus*	M	211	3/29/92	28°26.6'	96°24.2'
PO213	*T. truncatus*	U		3/19/92	28°35.8'	96°10.7'
PO214	*T. truncatus*		180		28°41.6'	96°25'
PO215	*T. truncatus*	U		3/27/92	28°40.9'	96°17.4'
PO216	Unknown	U		4/5/92	28°13.7'	96°37.4'
PO217	Unknown	U		4/5/92	28°11.7'	96°40.4'
PO218	*T. truncatus*	U		4/6/92	28°26.2'	96°25.3'
PO219	*T. truncatus*	U		4/5/92	28°20'	96°24.7'
PO220	*T. truncatus*	F	202	4/8/92	28°26.2'	96°24.8'
PO221	*T. truncatus*	M	250	4/8/92	28°24.2'	96°25.2'
PO222	*T. truncatus*			4/8/92	28°24'	96°24.5'
PO223v	*T. truncatus*	F	111	4/12/92	28°33.7'	96°32.3'
PO224	*T. truncatus*	F	230	4/10/92	28°39.2'	96°35.8'

continued

Table 4 (continued)

Region #	Species	Sex	Length	Date reported	Lat	Long
PO225	*T. truncatus*	M	177	4/14/92	28°36'	96°25.2'
PO226	*T. truncatus*	U	253	4/14/92	28°35.1'	96°26.8'
PO229	*T. truncatus*	M	267	4/17/92		
PO230	*T. truncatus*	M	115	4/18/92	28°27.7'	96°25'
PO231	*T. truncatus*	M	253	4/18/92	28°35.1'	96°35.1'
PO232	*T. truncatus*	M	234		28°24'	96°29'
PO233	*T. truncatus*	U			28°24.1'	96°28.9'
PO234	*T. truncatus*	M	248	4/18/92	28°26.3'	96°19.8'
PO235	*T. truncatus*	U		4/16/92	28°35'	96°34.6'
PO236	*T. truncatus*	M	235	4/19/92	28°19.5'	96°30.8'

Plate 1
Example of gray, pasty substance on the skin of a stranded dolphin. Photographed by Tom Wagner of Texas Parks and Wildlife Department.

of this capture study are published separately.[5] Though the current serological tests for dolphin morbillivirus were not available at the time of the live capture, tests for canine distemper were negative. A subsequent study using new technology has shown that 24% of the dolphins captured and examined during July 1992 tested positive for cetacean morbillivirus (Duignan et al., 1996). It is not known if the animals captured in July were present in the bay ecosystem during the peak mortality period.

To determine whether the live dolphins in the bay area were part of a resident or migratory population, satellite tagging and tracking of dolphins were conducted in the area where the mortality event occurred. Lynn (1995) reported that nearly all of the 35 dolphins branded during live capture appeared to be resident to the Matagorda-Espiritu Santo Bay area, and overall mark/recapture population size estimates from the photo-identification study demonstrated that 218±71.4 (95% CI) dolphins used an area of 312 km^2 in Matagorda and Espiritu Santo Bays.

[5] Sweeney, J. C. 1992. Veterinary assessment report, *Tursiops truncatus*, Matagorda Bay, Texas, July, 1992. Contr. Rep. NOAA, NMFS, Southeast Fisheries Science Center, Miami Laboratory Contrib. MIA-92/93-41, 10 p. + 5 append.

Plate 2
Example of blisters and circular lesions on the skin of a stranded dolphin. Photographed by Tom Wagner of Texas Parks and Wildlife Department.

Results from the metals analysis on sampled dolphin livers are shown in Table 5. Ranges for the 5 metals (arsenic, cadmium, lead, mercury, and selenium) are 0.0059–1.0885 µg/g for As, 0.0007–0.1980 for Cd, 0.0039–0.3237 for Pb, 0.5980–932.1566 for Hg, and 0.5313–363.639 µg/g for Se. For each metal, the highest concentration was quantified from one stranded dolphin, PO226. For the remainder of the dolphins, trace metal concentrations were generally comparable among individuals. Linear regression analysis of individual metals concentration and total length of dolphins showed two clusters of R^2 values. R^2 values <0.2 were found for As (0.18), Hg (0.10), and Se (0.11). Those metals with R^2 values >0.2 were Cd (0.47) and Pb (0.45). These results indicate that as total length increases, generally so does metals concentration within the liver. However, it is important to note that length and age in marine mammals have not been well correlated. Additionally, some metals concentrations within the livers exceeded the Food and Drug Administration (FDA) action levels and other international limits for metals in fisheries products. For Hg, the U.S. has a limit of 1.0 ppm in fillets, while the most restrictive limit found is 0.1 ppm in fillets (Venezuela) (EPA, 1989).

Table 5
Metals data quantified from livers of stranded dolphins during the unusual mortality event along the Texas coast during 1992.

	Metals concentrations (µg/g dry weight)				
Sample #	Arsenic	Cadmium	Lead	Mercury	Selenium
PA259	0.1223	0.0086	0.0521	1.9197	1.2177
PA260	0.1385	0.0031	0.0039	0.8112	1.3533
PA294	0.0922	0.0016	0.0055	0.5980	0.5313
PA298	0.1844	0.0007	0.0129	0.6224	3.7370
PA301	0.1994	0.0424	0.1067	10.3978	5.1535
PO199	0.4644	0.1372	0.1126	33.6303	23.2716
PO210	0.0703	0.0027	0.0122	0.8686	1.9325
PO212	0.1956	0.0649	0.0539	13.3652	8.5036
PO221	0.1638	0.0538	0.1002	47.9008	23.4912
PO223	0.0059	0.0012	0.0443	1.7375	0.9801
PO224	0.4559	0.0423	0.0548	17.1141	9.3483
PO225	0.3127	0.0074	0.0496	2.7288	2.0019
PO226	1.0885	0.1980	0.3237	932.1566	363.6390
PO231	0.4128	0.1211	0.2453	172.2030	80.1658
PO232	0.1378	0.0708	0.1560	34.6706	22.1409
PO234	0.0298	0.0786	0.0623	27.2880	14.8838
PO236	0.2655	0.0896	0.1034	26.9704	16.0083

Plate 3
Typical condition of stranded dolphins during the 1992 unusual mortality event in Texas. Photographed by Ann Colbert, on-site coordinator of the investigation.

Ecotoxicological Evaluation

General Water Quality Measurements of surface water temperatures at each station indicated temperatures ranging from 21.8–28.8°C, averaging 25.4°C (Table 6). Dissolved oxygen ranged from 5.8–15.8 mg/L (82–200% saturation), averaging 7.95 mg/L (103% saturation). The pH ranged from 7.9–9.3, averaging 8.43. High pH (9.3) was observed at Station #9 (Corpus Christi power plant) which exceeded EPA water quality criteria for pH. Salinities throughout the area were extremely low, ranging from 0–24 ppt, averaging 12 ppt. Evaluation of rainfall (Fig. 4) and riverflow records (Fig. 5) indicated that excessive rainfall caused the extremely low salinities and the high river flow measured during January–April 1992.

Chemical and Toxicological Analysis of Surface Water Samples Screening of surface water samples for pesticides and PCBs resulted in the detection of triazine herbicides (i.e. atrazine) (0.1–1.0 µg/L) at all sites assayed. The insecticide aldicarb (≥2–<5 µg/L) was detected at four sites during April 1992 (Table 7). These results indicated that significant pesticide runoff from agricultural fields into shallow bays had occurred following the periods of excessive rainfall during January–April 1992. No PCBs or cyclodiene pesticides were detected (Table 7). Subsequent sampling during July 1992 also detected triazine herbicides (0.1–1.0 µg/L) at all sites assayed, and aldicarb (>2–<5 µg/L) at four sites (Table 8). These findings suggested that pesticide inputs continued well into the summer, after peak application and rainfall periods.

Subsequent GC-ECD analysis of surface water samples for PCBs, PAHs, and pesticides failed to detect the presence of any of these chemical contaminants. Aldicarb was not detected by GC-NPD analysis at levels above the LLOD (3 µg/L). An identifiable peak was observed in one sample, but it was below the LLOD (<3 µg/L). These data suggest that any aldicarb present was at concentration <3 µg/L. This agrees in part with the ELISA screening bioassay results which indicated levels >2–<5 µg/L. Subsequent sampling and analysis during 1993 quantified the presence of aldicarb in surface water by GC-NPD at concentrations of 2–3 µg/L (Pennington et al., 1994). During this follow up study in 1993, rainfall

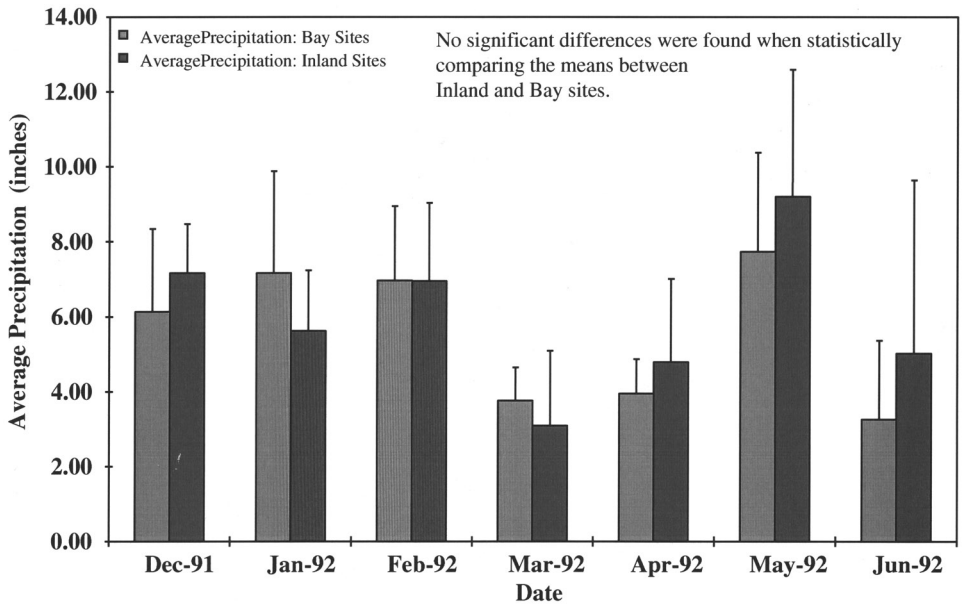

Figure 4
Comparison of the average precipitation at bay and inland sites along the Texas coast, Dec. 1991–June 1992.

levels were at or below normal for the region for the February to October study period.

GC-NPD analysis also indicated that the triazine herbicide detected by ELISA at concentrations >0.1–1.0 µg/L was atrazine. Subsequent sampling and analysis in a follow-up study in the area during 1993 indicated atrazine concentrations ranging from 0.1–>1.0 µg/L and that 98% of >130 samples collected were positive for atrazine (Pennington, 1996). Other GC-MS, GC-ECD, GC-NPD, AA, and ICP results did not detect the presence of any other priority pollutants in surface waters. Toxicological analysis of selected surface water samples using *P. pugio* larvae indicated no toxicity in any of the samples analyzed (e.g. those containing aldicarb and atrazine).

Chemical Analysis of Sediments Chemical analysis of sediment samples collected at selected stations indicated the presence of several trace metals including arsenic (As) (0.26–7.64 mg/kg), mercury (Hg) (<0.020–0.050 mg/kg), selenium (Se) (<0.033–0.063 mg/kg), barium (Ba) (9.0–56.0 mg/kg), chromium (Cr) (<1.0–3.00 mg/kg), copper (Cu) (<1.00–1.00 mg/kg), lead (Pb) (<2.00–5.00 mg/kg), and nickel (Ni) (<1.00–1.00 mg/kg). None of the concentrations of trace metals detected exceeded the current Sediment Quality Guidelines (Effects Range Low [ERLs] or Effect Range Median [ERMs]) as described by

Table 6
Results of general water quality parameters at bay stations sampled during the Texas marine mammal stranding study.

Station #	Temp (°C)	Salinity (ppt)	pH	DO_2 (mg/L)	% Saturation
1A	23.7	10.0	8.4	8.1	109
1B	27.8	11.0	8.3	6.7	91
2	25.8	11.0	8.3	7.8	104
3	26.8	11.0	8.3	6.9	92
4	27.4	3.0	8.7	7.2	95
5	25.3	8.0	8.5	7.3	NM[1]
6	24.5	22.0	NM	7.4	99
7	25.5	3.0	8.4	7.8	98
8	28.8	23.0	8.3	5.8	95
9A	26.7	24.0	9.3	9.1	122
9B	26.7	24.0	9.3	15.8	>200
10	25.0	24.0	8.3	6.6	91
11	21.8	0.0	8.3	7.6	87
12	22.6	3.0	7.9	7.0	82
13	22.4	3.0	8.2	8.2	92
Average	25.40	12.0	8.4	7.9	103.4
SE±	0.54	2.33	0.10	0.60	8.00
Range	21.8–28.8	0.0–24.0	7.9–9.3	5.8–15.8	82–>200

[1] NM = not measured.

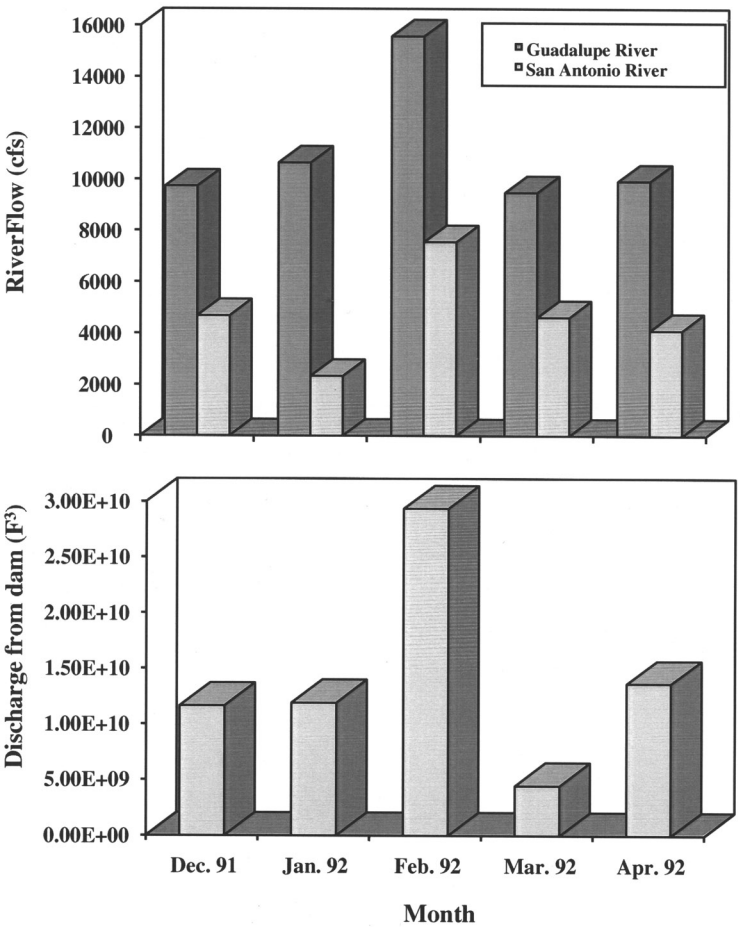

Figure 5
River flow and water discharge data from dams in the Quadelupe and San Antonio Rivers, Dec. 1991–April 1992. Note the elevated river flow and water discharges during February 1992.

Long et al. (1995). Also, no detectable levels of persistent pesticides, PAHs, or PCBs were found in sediment samples. These results indicate that none of the sediments sampled within the study area contained contaminants at concentrations known to be toxic to living marine resources.

Chemical Analysis of Animal Tissues Chemical analysis of whole fish (prey species) samples collected by cast-net from selected stations within the bay system indicated the presence of DDD (<2–10 µg/Kg), DDE (<1–40 µg/Kg), As (0.14–0.42 mg/Kg), Se (0.19–0.49 mg/Kg), Hg (<0.020–0.042 mg/Kg), Ba (<1.00–11.00 mg/Kg), Cu (<1.00–7.00 mg/Kg), and Ni (<1.00–5.00 mg/Kg). None of the detectable contaminants exceeded FDA action levels/tolerances for edible fishery products. These results indicated that fish (mullet, menhaden, catfish, and anchovy) within the area of concern did not contain elevated concentrations of those chemical contaminants. Rather, fish appeared to contain only degradation products from DDT (i.e. DDD and DDE) and trace metals at rather low concentrations, suggesting that dolphin mortality was probably not related to bioaccumulation through the food chain of any of the contaminants listed above.

Analysis of Surface Waters and Finfish for Biotoxins Analysis of surface waters and finfish for brevetoxin were negative (<LLOD). Icthyotoxicity tests exposing sheepshead minnow (*Cyprinodon variegatus)* to extracts of surface water and finfish were negative, indicating that brevetoxin was not present in harmful concentrations in any of the samples analyzed. Similarly, analyses of surface water and adult oysters (*Crassostrea virginica*) for domoic acid, using methods described by Hatfield et al. (1994), were negative. Light microscope examination of plankton tows col-

Table 7
Results of April 1992 water samples using the Enviroguard® Test Kits as a screening tool.

Site	Concentration			
	Cyclodiene (ppb)	PCBs (ppm)	Triazines (ppb)	Aldicarb (ppb)
Cherry Point, SC (reference)	—[1]	—	>0.1, <1.0	< llod [2]
Sand Point, East Bay	< llod	< llod	> 1.0	>2, <5
Sand Point, West Bay	< llod	< llod	> 1.0	>2, <5
Port O'Connor, ICW	—	—	> 1.0	< llod
Port O'Connor, ICW dredge spoil	—	—	> 1.0	< llod
Fulton Beach, Rockport	—	—	> 1.0	< llod
Corpus Christi, aquaculture ponds	< llod	< llod	> 1.0	< llod
Corpus Christi, power plant intakes	—	—	>0.1, <1.0	≥2
San Luis Pass	—	—	>0.1, <1.0	≥2

[1] — = Not screened.
[2] <llod = Lower limits of detection are 0.5 ppb for cyclodienes; 0.1 ppm for PCBs and 2.0 ppm for aldicarb.

Table 8
Results of July 1992 water samples using the Envirogard® Test Kits as a screening tool.

Site	Concentration (ppb)		
	Station	Triazines	Aldicarb
Upper Lavaca Bay; park and boat landing	11	> 0.1, < 1.0	< LLOD [1]
Corpus Christi; ponds at power plant	9	> 0.1, < 1.0	< LLOD
Port O'Connor Jetty	17	< LLOD	< LLOD
Lavaca Bay; Magnolia Beach	13	> 0.1, < 1.0	< LLOD
Lavaca Bay; Dredge Spoil Island	12	> 0.1, < 1.0	< LLOD
Sand Point, west end of spit	2	> 0.1, < 1.0	< LLOD
Port O'Connor ICW at dredge disposal pipe	5	> 0.1, < 1.0	> 2.0, < 5.0
Port O'Connor ICW at Dewberry Island	4	> 0.1, < 1.0	< LLOD
Fulton Beach	7	> 0.1, < 1.0	< LLOD
North Matagorda Bay; live capture site	14	> 0.1, < 1.0	> 2.0, < 5.0
Middle Matagorda Bay; Green's Bayou	15	> 0.1, < 1.0	> 2.0, < 5.0
ICW; S Port O'Connor; Channel Marker #19	*	< LLOD	> 2.0, < 5.0

[1] LLOD = lower limits of detection: for triazines = 0.1 ppb and aldicarb = 2.0 ppb.
* This sample was taken only once at the end of the study, it is located within the bay, south of stations 4 and 5.

lected in the area during the investigation (after 12 April) revealed no representatives of the domoic acid-producing diatom genus *Pseudo-nitzschia* nor other harmful phytoplankton species. These results suggest that known biotoxin-producing organisms were not present in the water column at effective concentrations during the investigation period after peak mortality had passed.

Discussion

The deaths of more than 220 dolphins during a 4-month period within the Texas inner bay estuarine ecosystems were unprecedented and clearly unusual. Evaluation of information collected during the investigation and environmental contaminant data indicated no specific causes of this unusual mortality event. The stranded animals' advanced stage of decomposition prevented the thorough

histopathology and virology examinations that might have been crucial to the investigation, as well as forensic contaminant analyses for non-persistent pesticides. Reijnders (1988) noted the potential problem of establishing cause and effect relationships during periods of marine mammal population declines or strandings. He concluded that future research should focus on baseline chemical contaminant surveys which establish uptake kinetics, and identification of marine mammals' physiological responses to chemical contaminants. Without information such as condition of the animals' lymph nodes, blubber, lungs, livers, and other organs, it is impossible to draw definitive conclusions about their health or the potential causes of their deaths. Circumstances of this unusual mortality investigation further emphasize the importance of assessing dermal toxicity of chemical contaminants in marine mammals and understanding the combination of dermal, oral, and food chain exposures in formulating environmental risk assessments.

Results of environmental studies indicated that significant rainfall and freshwater runoff from agriculture sources entered the bay ecosystem from December 1991 through March 1992. The effects of prolonged low salinity exposure on dolphins have not been rigorously studied. Early research (Simpson and Gardner, 1972; Greenwood et al., 1974; Harrison and Thurley, 1974) suggested that low salinity can cause increased sloughing of the skin; epidermal-dermal separation resulting in blistering; and formation of a gray, pasty substance on the skin, as was observed on many of the stranded dolphins and live animals during the 1992 Texas event. These altered skin conditions were observed on live dolphins from mid-January through March 1992, but the skin conditions apparently began improving during early to mid-April as salinities in the area returned to normal. Animals with the pasty, grayish skin coloration were observed after mid-April, but none with blisters and open wounds. No animals in this condition were observed during the live-capture operation in July. This trend coincides with a gradual increase in salinity due to the reduction of the record rainfall and the accompanying freshwater runoff that was observed in this area from December 1991 through March 1992. The possibility exists that members of a resident dolphin population might not leave their area of residency if unfavorable environmental conditions such as low salinity develop (Wells).[6] In addition, Shane (1977) noted that the offshore population of bottlenose dolphins off the Texas coast rarely interacted with the bay population. These findings may be significant if long-term low salinity exposure and/or dermal absorption of environmental contaminants played roles in the observed dolphin mortalities.

Field observations confirmed that dolphins were present in the Texas bay ecosystem during the period of investigation when extremely low salinity conditions had developed from the unusually high rainfall observed during early 1992 and when pesticides were detected in the inner bay areas. For some species such as bottlenose dolphins, resident inshore populations spend extensive periods in close proximity to both point and nonpoint sources of contamination, where surface water, sediment, and prey species contaminant concentrations are likely to be much higher than in offshore areas.

Comparisons of total Hg levels in liver tissue from stranded dolphins examined during this unusual mortality event with previous unusual mortality events (Texas, 1990[7]; east coast of U.S., 1987–88[8]; South Carolina coasts[9]) indicated that Hg levels were lower or comparable in dolphins from Texas during 1992. Even though different laboratories and analytical techniques were used to obtain and compare the mercury values between these studies, these findings generally suggest that liver Hg concentrations in dolphins from the 1992 unusual mortality event in Texas were not unusually high levels compared to previous dolphin strandings along the mid-Atlantic and Gulf coasts. This finding is significant because the Lavaca Bay area had been historically contaminated with mercury, and there was concern that mercury poisoning might have played a role in the mortality event. We recognize that age data and mercury speciation would have been important in evaluating the mercury data, and it is unfortunate that these data were not available for interpretation of results.

Exposure to pollution point sources would generally be confined to the immediate proximity of the discharge and buffer zone, since contaminant concentrations would be diluted within the immediate proximity of the input (Marcus and Scott, 1990). The primary concerns for point source pollution inputs are environmentally persistent pollutants, which may pose a potential chronic toxicity to estuarine living marine resources, including marine mammals. Environmental monitoring studies of marine mammals have clearly indicated the presence of environmentally persistent pesticides and PCBs in cetacean tissues[8] (Borrell and Aguilar, 1990; O'Shea and Brownell, 1994).

[6] Wells, Randall. Mote Marine Laboratory, 1600 Thompson Parkway, Sarasota, FL 34236. Personal commun.

[7] Hansen, L. J. (ed.). 1992. Report on investigation of 1990 Gulf of Mexico bottlenose dolphin strandings. NMFS, Southeast Fisheries Science Center, Contrib. MIA-92/93-21, 219 p.

[8] Geraci, J. A. 1989. Clinical investigation of the 1987-88 mass mortality of bottlenose dolphins along the central and south Atlantic coast. Final Report to the National Marine Fisheries Service, U.S. Navy Office of Naval Research, and Marine Mammal Commission, 63 p.

[9] Beck, Kevin. SEFSC Charleston Laboratory, NMFS, 219 Fort Johnson Rd., Charleston, SC 29412. Personal commun.

Exposure to nonpoint source pollution poses a somewhat different problem. Nonpoint source pollution generally describes a large volume of pollution input relative to the watershed size, emanating from a wide geographical area, generally containing low concentrations of environmental pollutants such as nutrients, bacteria, and environmental contaminants including trace metals, PAHs, and pesticides[10, 11] (Hoffman et al., 1984). Nonpoint source pollution exposures frequently occur concomitant with low salinity[10, 11]. In a study of nonpoint source agricultural pesticide runoff, insecticide (azinphosmethyl, endosulfan, and fenvalerate) concentrations were measured in estuarine tidal creeks that exceeded acute toxicity levels found in laboratory toxicity tests on other marine species[10, 11]. This runoff resulted in local fish kills. Acute in situ toxicity tests measured significant inhibition of the enzyme acetylcholinesterase (AChE) in finfish, altered bioenergetic metabolism (respiration, nitrogen excretion, condition index, and gonadal index) in finfish and mollusks, and inhibited reproduction in the fish, crustacean, molluscan, and benthic copepod fauna examined. These studies were confined to the headwaters of tidal creeks. Bottlenose dolphins had been observed feeding within a 0.5 km range of the study sites used by Scott and coworkers and would have potentially been exposed to contaminants in surface waters and finfish. However, no significant dolphin strandings were observed in this region of South Carolina. Assessment of environmental monitoring data collected in this study did not indicate the presence of priority pollutants at toxicologically significant concentrations (> water quality criteria) in the surface water and sediment samples of the area.

Surface water analysis indicated the presence of pesticides (e.g. atrazine and aldicarb), but analysis of selected prey fish tissues collected after the peak of dolphin mortality did not indicate the presence of these pesticides or other toxicologically significant contaminants. This is not surprising given the short persistence and low bioaccumulation potential for many of these nonpersistent compounds, as well as the fact that sampling occurred weeks after peak dolphin mortality had occurred. Another notable factor is that detectable levels of pesticides were observed using randomly collected temporal samples rather than biased rain event samples. Higher levels of pesticides would be detected immediately following runoff events[10, 11]. The detection of these pesticides in random water samples suggests that pesticide runoff had occurred and that peak pesticide concentrations in runoff were probably not sampled during 1992. Pennington et al. (1994), in a subsequent study of pesticide runoff in this area of Texas in 1993, only found aldicarb in ditches adjacent to agricultural fields at similar concentrations measured in Matagorda Bay in 1992 (2–3 µg/L). It is very important that normal rainfall was observed during 1993, whereas record rainfall levels were measured during 1992. These findings clearly indicate the extent of the record rainfall during 1992, because pesticide levels in bays were similar to pesticide levels in ditches adjacent to agricultural fields during normal periods of rainfall. Because dolphins were observed in this area during the period, and tagging studies suggested a resident population, this information demonstrates the likely increased exposure of dolphins to pesticides and concomitant low salinity conditions during early 1992.

In addition to aldicarb, atrazine was also measured in surface water samples. Atrazine usage accounts for 16.7% of the applied herbicides along the Gulf of Mexico. Adjacent to Matagorda Bay, Texas, atrazine usage ranks this watershed with the third highest application rate in the region [12]. As with aldicarb, there have been no studies of toxicity for atrazine in bottlenose dolphins, although atrazine generally has much lower mammalian toxicity.

The fresh-water outflow into the Texas bays was higher for the period from December 1991 through May 1992 than had been recorded in that area since 1939 [13]. The most critical implication of significant low salinity exposure was supported by water flow records and the pesticide measurements in mid and outer bay sampling stations. In much of the area, agricultural fields extend up to the bay beaches. Because the event coincided with agricultural planting and pesticide application periods, it was likely that the amount of contaminants in agricultural runoff would be higher than normal during this period. Pait et al.[12] similarly reported peak pesticide usage in Texas during this same time period. Additionally, Pait et al. reported that the top four estuarine drainage areas in the U.S. were located in Texas.[14] These

[10] Scott, G. I., M. H. Fulton, D. W. Moore, G. T. Chandler, P. B. Key, T. W. Hampton, J. M. Marcus, K. L. Jackson, D. S. Baughman, A. H. Trim, L. Williams, C. J. Lauden, and E. R. Patterson. 1988. Agricultural insecticide runoff effects on estuarine organisms: correlating laboratory and field toxicity testing with ecotoxicological biomonitoring. Report to U.S. EPA, Gulf Breeze Environmental Research Laboratory, Gulf Breeze, FL 32561, 688 p.

[11] Scott, G. I., M. H. Fulton, D. W. Moore, G. T. Chandler, P. B. Key, J. M. Marcus, K.L . Jackson, D. S. Baughman, A. H. Trim, L. Williams, C. J. Louden, and E. R. Patterson. 1990. Agricultural insecticide runoff effects on estuarine organisms: correlating laboratory and field toxicity testing with ecotoxicological biomonitoring. Report to U.S. EPA, Gulf Breeze Environmental Research Laboratory, Gulf Breeze, FL 32561, 232 p.

[12] Pait, A. S., A. E. DeSouza, and D.R. Farrow. 1992. Agricultural pesticide use in coastal areas: a national summary. NOS, NOAA, Rockville, MD, 112 p.

[13] Texas Water Commission. 1992. Texas Water Commission District 12, 4410 Dillon Ave., Suite 47, Corpus Christie, TX 78415-5326. Unpubl. data.

[14] Pait, A. S., D. R. Farrow, J. A. Lowe, and P. A. Pacheco. 1989. Agricultural pesticide use in estuarine drainage areas: a preliminary summary for selected pesticides. Ocean Assessments Division, NOS, NOAA, Rockville, MD, 134 p.

estuarine areas contained 47%–75% agricultural drainage. Subsequent monitoring from February–October 1993 indicated that peak pesticide levels occurred in the February–March sampling period (Pennington et al., 1994). These findings suggest that the peak pesticide concentrations were probably not sampled during 1992 as sampling did not begin until April 1992.

The water samples collected at Sand Point, Texas, where the dead fish, birds, and dolphins were observed, showed presence of the carbamate insecticide aldicarb, as well as triazine herbicides. These pesticides were also detected at other sites in the bay ecosystem. Aldicarb and triazine herbicides were detected during mid-April 1992 and again during July 1992, as far out into Matagorda Bay as the inland coast of Matagorda Peninsula (Tables 7 and 8). Both of these sampling periods took place long after (1–4 months) the major rain events had caused depressed salinity, and after the dolphin mortality rate had begun to decrease.

While aldicarb usage on agriculture in Texas (7.8% of total U.S. usage)[15] is relatively low compared to other pesticides, given its high water solubility and high mammalian toxicity potential, measurement of levels approaching 2–5 µg/L in seawater may be toxicologically important. The oral LD_{50} for aldicarb in rats is 0.9–1.0 mg/kg and the acute dermal LD_{50} in rats is 2.5–3.0 mg/kg. Wildlife toxicity values ranging from 1.0–2.0 mg/kg have been reported in bobwhite quail and mallard ducks. The mode of action for aldicarb toxicity is inhibition of the enzyme acetylcholinesterase (AChE). Clinical effects of AChE poisoning have been reported at aldicarb doses as low as 0.002 mg/kg/day in humans. The major metabolite, aldicarb sulfoxide, has 76 times greater anti-AChE activity than the parent compound.[16] Increasing salinity was found to increase aldicarb toxicity to a freshwater fish (Japanese Medaka) by enhancing the formation of aldicarb sulfoxide through cytochrome P450 and flavin monooxygenase enzyme systems (El-Alfy and Schlenk, 1997). While enhanced toxicity was observed with increasing salinities in this freshwater species, these findings clearly indicate alterations in aldicarb toxicity are possible. The extreme conditions found during this study do not rule out the potential for metabolically altered aldicarb toxicity in exposed organisms. Unfortunately, the dose-response relationship and toxicity of aldicarb in marine mammals is unknown. Because oral and dermal LD_{50} values have not been established for bottlenose dolphins and other marine mammals undergoing continuous aqueous exposure when pesticides are present in their environment, the toxicological significance of the aldicarb and atrazine concentrations measured in Texas bay waters is not clear, even though the concentrations were well below oral and dermal LD_{50} values for terrestrial mammals.

Another factor may be that marine mammals are more sensitive to neurotoxic chemicals, such as pesticides, than other mammalian species. Anderson (1994) reported that whales appeared to be much more sensitive to neurotoxic biotoxins, such as saxitoxin, than humans. Anderson (1994) suggests that increased neurotoxic sensitivity may have resulted from three possible factors. First, whales may receive a continuous dose of a toxin, whereas human estimates are based on single exposures (oral or dermal). Second, during swimming, the mammalian diving reflex channels more blood and oxygen to the heart and brain, bypassing the kidney and liver which are major organs of detoxification. This may result in greater toxicity potential in marine mammals. Third, whales may die indirectly from other causes following sublethal exposure to neurotoxins. Due to the lack of information from animal tissues and water before and during the peak mortality period, it is unclear whether or not similar phenomena may have caused enhanced susceptibility to potentially neurotoxic pesticides in bottlenose dolphins swimming and feeding in the shallow Texas bays. In future unusual mortality events, increased neurotoxic sensitivity of marine mammals should be considered when evaluating mammalian toxicity data, particularly if Class I mammalian neurotoxic agents are present in potential exposure pathways (water, sediment, or biota).

Aldicarb and atrazine are potential endocrine disrupting chemicals that may affect endocrine function and immune response in mammals, fish, and other species (Porter et al., 1985; Olson et al., 1987; Boyd et al., 1990; Gill et al., 1991; Colborn and Clements, 1992). Soto et al. (1994) have further shown that the effect of endocrine disrupting chemicals can be cumulative, as subclinical pesticide doses were as effective in altering endocrine function as a single large dose of an individual pesticide in a human breast cell line. Olson et al. (1987) reported that exposure of mice to aldicarb in drinking water significantly affected immune function. There were significant reductions in splenic plaque-forming cell response in low dose (1 ppb) aldicarb exposure. An inverse dose-response effect on immune function was observed, which was measured only in low concentrations and not at higher aldicarb doses. Also, exposure of rats to aldicarb in combination with other insecticides (methomyl) and herbicides (metribuzin) significantly affected thyroid function (e.g. increased thy-

[15] Gianessi, L. P., and C. A. Puffer. 1990. The use of selected pesticide active ingredients included in the national pesticide survey, p. 128. Report to the Environmental Protection Agency. Quality of the Environment Division, Resources for the Future, Inc., 1616 P Street, NW, Washington, DC 20036.

[16] DHEW/NCI. 1979. Toxicology and carcinogenesis studies of aldicarb. National Institute of Health. Report #136, NIH Pub. #79-1391, p. 2.

roxin levels) and caused neurological changes, learning impairment, and altered immune and endocrine function when combined with triazine herbicides (Porter et al., 1993).

Significant ($p \leq 0.05$) correlations have been reported between increased persistent pesticide concentrations (DDE, total PCB) and declining health status (e.g. suppressed immunity) among dolphins studied during the live capture in Texas bays during July 1992. Additionally, persistent pesticide concentrations in blubber samples were elevated among dolphins infected with the cetacean morbillivirus.[17] Exposure to low salinity conditions and nonpersistent pesticides could have provided additional stress to resident populations.

Two critical facets of the investigation of organic contaminants as causative agents in a mortality event are seriously limited by the lack of marine mammal scientific information. One of those aspects is the mode of toxicity. For example, if water analysis creates cause for concern regarding a particular contaminant, and that contaminant is known not to bioaccumulate in tissues but instead is toxic by inhibiting enzyme activity, then distinct problems arise in establishing a cause-effect relationship. First, the routinely conducted contaminant analysis of blubber tissue would not be an effective investigative tool for detection of that specific contaminant. Secondly, if fresh blood and brain tissue are not available to test for enzyme activity levels, and a database large enough to have established normal and abnormal values for the parameters is also not available, then there is not currently an effective forensic tool to evaluate exposure to the particular contaminant. The other critical aspect is the fact that simple detection of an environmental contaminant is not usually enough to establish a causal relationship in terms of adverse effects on many marine species. Evidence needs to be obtained from animal tissues or from controlled studies demonstrating effects in models for the species involved, in order for any causal hypotheses to be verified. Scientific data needs to be available that suggests the particular type of animal involved is in some way adversely affected by exposure to a particular contaminant's detected concentration.

The possibility exists that exposure to some additional acute environmental factor, which can compromise immune systems and allow for various declining health conditions to develop in the animals, could be additive and cumulative. Thus, the primary cause of death for each individual animal may not necessarily be consistent in the population as the primary cause of the entire event. This is particularly true when dealing with potential endocrine disrupting chemicals which may affect young and old, as well as males and females, differently. Likewise, a disease condition in the animals could weaken the defense mechanisms against environmental contaminants, facilitating various conditions leading to death. Because no serum from dolphins was available for morbillivirus tests during the unusual mortality event, and the current PCR-based tests were not available, we cannot rule out the possibility that this virus may have played a role in the event. Results from analyses conducted following the live capture study clearly indicated 24% of the dolphins captured had been exposed to morbillivirus (Duignan et al., 1996) and that these dolphins had a greater environmental contaminant load[16]. A number of questions remain unanswered due to the decomposition stage of stranded animals. Were the dead stranded dolphins ever exposed to morbillivirus and did they have even higher levels of environmental contaminants? Additionally, what does a 24% prevalence rate for morbillivirus represent among dolphins in this area?

The scope of future investigations can be better planned as more data becomes available to support conclusions drawn on analytical results. Subsequent research on dolphin skin morphological characteristics likely to affect dermal absorption has suggested that use of terrestrial animal models for dermal toxicology in dolphins is inappropriate (Colbert, 1996). In addition, research is underway to establish baseline levels for AChE (the target enzyme for carbamate insecticides such as aldicarb) activity in bottlenose dolphins. All of the information collected during past investigations is important in developing parameters for better assessment of future mortality events. Research is currently underway regarding the effects of certain contaminants on marine mammal species, and health assessment studies of wild dolphin populations are being conducted when possible.

Since this investigation, several actions have been taken to alleviate some of these methodological problems. The Working Group on Unusual Marine Mammal Mortalities was formally established under the Marine Mammal Health and Stranding Response Act of 1992. This group annually reviews relative information on marine mammal mortalities and makes recommendations to the federal agencies involved. A Small Cetacean Necropsy Guide[18] has been drafted to help guide and standardize the collection of tissues for specific analyses. Training is being provided to Marine Mammal Stranding Network personnel in marine mammal evidence collection and associated investigation protocols.[19] Also, a contingency fund for response has been established under the Act, but appropriation of funds will vary over

[17] Reif, J. S., L. Hansen, S. Galloway, and G. Mitchum. 1996. The relationship between chlorinated hydrocarbon contaminants and selected health parameters in bottlenose dolphins (*Tursiops truncatus*) from Matagorda Bay, Texas, 1992. Manuscr. in prep.

[18] Galloway, S. B., and A. Colbert (eds.). 1997. NMFS Charleston Laboratory, 219 Fort Johnson Rd., Charleston, SC 29412. Manuscr. in prep.

[19] Rowles, T. 1998 Ongoing training workshop plans. Office of Protected Resources, NMFS, Silver Spring, MD 20910.

time and with each event.

In conclusion, results of field studies and investigations conducted in the unusual mortalities of bottlenose dolphins along the mid-Texas coast during 1992 indicated no specific cause of the observed deaths. Rather, four factors were identified which may have contributed to an overall cumulative effect on environmental conditions that had the potential to play a role in the 1992 unusual mortality event. These included evidence that dolphins were exposed to prolonged low salinity conditions which could have altered the permeability barrier of the skin; the co-occurrence of nonpersistent but toxic pesticides which are potential endocrine disrupting chemicals that may chronically affect immune function and health; the presence of elevated levels of persistent pesticides in surviving dolphins of poor health status; and the correlation of elevated persistent pesticide concentrations in surviving dolphins testing positive for morbillivirus. While no statistical correlation was found with the specific timing of runoff events and stranding rates, the mortality event coincided with a period of extensive record rainfall, low salinity conditions within the bay, and increased agricultural pesticide runoff only in inland bays where the strandings occurred and not in offshore waters. Aldicarb and atrazine concentrations detected in July after dolphin mortality had declined did not co-occur with extensive low salinity conditions and accompanying acute, pulsed pesticide exposure at higher concentrations, suggesting pesticide exposure per se was not the cause of this event. Rather, observed dolphin mortalities may have resulted from a combination of prolonged rainfall, low salinity conditions, and the increased discharge of pesticides and other chemical contaminants into Texas bays.

Acknowledgments

The 1992 investigation was conducted with the support of many agencies and institutions, researchers, and other individuals. We hesitate to list any individuals for fear of leaving someone out, but we sincerely thank all of the participants from the agencies and institutions listed below for their information, intense efforts, and dedication. An investigation such as the one described here requires time and physical efforts far beyond the call of duty from many of the people involved. Many researchers donated their time, expertise, and other resources to assist in this investigation. The people of Texas were gracious to our field team during our extended visit along their coast, and we thank them for making the experience as pleasant as possible under the circumstances.

Thanks to Texas A&M University, Texas Department of Health, Texas Marine Mammal Stranding Network, Texas Parks and Wildlife Department, Texas Veterinary Medical Diagnostic Laboratories, Texas Water Commission, University of Texas, U.S. Coast Guard, U.S. Fish and Wildlife Service, The Working Group on Unusual Marine Mammal Mortalities; and to National Marine Fisheries Service Southeast Fisheries Science Center Laboratories in Galveston, Texas, Miami, Florida, Beaufort, North Carolina, and Charleston, South Carolina; National Marine Fisheries Service Office of Protected Resources, Silver Spring, Maryland; and National Marine Fisheries Service Office of Protected Species, St. Petersburg, Florida.

Literature Cited

Anderson, D. M.
1994. Red tides. Sci. Amer. 271(2):52–58.

Borrell, A., and A. Aguilar.
1990. Loss of organochlorine compounds in the tissues of a decomposing stranded dolphin. Bull. Environ. Contam. Toxicol. 45:46–53.

Boyd, C. A., M. H. Weiler, and W. P. Porter.
1990. Behavioral and neurochemical changes associated with chronic exposure to low-level concentrations of pesticide mixtures. J. Toxicol. Envir. Health 30:209–221.

Bush, P., M.Tanner, J. Kiker, R. Page, N. Booth, and O. Fletcher.
1978. Tissue residue studies on toxaphene in broiler chickens. J. Agric. Food. Chem. 26(1):126–130.

Colbert, A.
1996. Morphological investigation of bottlenose dolphin (*Tursiops truncatus*) skin for use in cutaneous absorption studies. MS thesis, North Carolina State University, College of Veterinary Medical Sciences, Raleigh, NC, 92 p.

Colborn, T., and C. Clements (eds.).
1992. Advances in modern environmental toxicology (M. A. Mehlman, series ed.). Volume XXI - Chemically-induced alterations in sexual and functional development: The Wildlife/Human Connection. Princeton Scientific Publishing Co., Inc., NJ.

Duignan, P. J., C. House, D. K. Odell, R. S. Wells, L. T. Hansen, M. T. Walsh, D. J. St. Aubin, B. K. Rima, and J. R. Geraci.
1996. Morbillivirus infection in bottlenose dolphins: evidence for recurrent epizootics in the western Atlantic and Gulf of Mexico. Mar. Mamm. Sci. 12:499–515.

El-Alfy, A., and D. Schlenk.
1997. Effect of salinity on aldicarb toxicity to Japanese medaka (*Oryzas latipes*). *In* Society of Environmental Toxicology and Chemistry Abstract Book, SETAC 18th Annual Meeting, Bridging the Global Environment: Technology, Communication, and Education. Pensacola, FL, p 321.

Environmental Protection Agency.
1989. Assessing human health risks from chemically contaminated fish and shellfish: a guidance manual. Office of Marine and Estuarine Protection and Office of Water Regulation and Standards, Washington, DC. Report EPA-503/8-89-002, 91 pages + appendices.

Gill, T. S., J. Pande, and H. Tewari.
1991. Hemopathological changes associated with experimental aldicarb poisoning in fish (*Puntius conchonius* Hamilton). Bull. Environ. Contam. Toxicol. 47:628–633.

Greenwood, A. G., R. J. Harrison, and H. W. Whitting.
1974. Functional and pathological aspects of the skin of marine mammals. *In* R.J. Harrison (ed.), Functional anatomy of marine mammals, Vol. 2, p. 73–110. Acad. Press, NY.

Harrison, R. J., and K. W. Thurley.
1974. Structure of the epidermis in *Tursiops, Delphinus, and Pho-*

coena. *In* R.J. Harrison (ed.), Functional anatomy of marine mammals, Vol. 2, p. 45–71. Acad. Press, NY.

Hatfield, C. L., J. C. Wekell, E. J. Gauglitz Jr., and H. J. Barnett.
1994. Salt clean-up procedure for the determination of domoic acid by HPLC. Natural Toxins 2:206–211.

Hoffman, E. J., G. L. Mills, J. S. Latimer, and J. G. Quinn.
1984. Urban runoff as a source of polycyclic aromatic hydrocarbons to coastal waters. Envir. Sci. Technol. 18:580–587.

Long, E. R., D. D. MacDonald, S. L. Smith, and F. D. Calder.
1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Envir. Manag. 19(1):81–97.

Lynn, S.
1995. Movements, site fidelity, and surfacing patterns of bottlenose dolphins on the central Texas coast. MS thesis, Texas A&M University, College Station, TX, 92 p.

Marcus, J. M., and G. I. Scott.
1990. The use of sediment heavy metal bioavailability as a method of environmental hazard assessment. *In* W.G. Landis and W.H. van der Schalie (eds.), Aquatic toxicology and risk assessment: thirteenth volume, ASTM STP 1096, American Society for Testing and Materials, Philadelphia, p. 110–122.

Olson, L. J., B. J. Erickson, R. D. Hinsdill, J. A. Wyman, W. P. Perter, L. Binning, R. Bidgood, and E. Norheim.
1987. Aldicarb immunomodulation in mice: an inverse dose-response to parts per billion levels in drinking water. Arch. Environ. Contam. Toxicol. 16:433–439.

O'Shea, T. J., and R. L Brownell Jr.
1994. Organochlorines and metal contaminants in baleen whales: a review and evaluation of conservation implications. Sci Total Envir. 154:179–200.

Pennington, P. L.
1996. The toxicity of the herbicides atrazine and alachlor on the estuarine phytoplankter with an emphasis on acute toxicity testing of individual herbicides, herbicide mixtures, and multi-generational chronic bioassays. MS thesis, University of Charleston, Charleston, SC, 142 p.

Pennington, P. L., G. I. Scott, M. H. Fulton, J. W. Daugomah, E. D. Strozier, P. B. Key, and B. C. Thompson.
1994. Analysis of pesticide runoff from mid-Texas estuaries and risk assessment implications for marine phytoplankton. Poster presentation at the 15th Annual Society of Environmental Toxicology and Chemistry Meeting, Denver, CO, 22 p.

Porter, W. P., S. M. Green, N. L. Debbink, and I. Carlson.
1993. Groundwater pesticides: interactive effects of low concentrations of carbamates aldicarb and methomyl and the triazine metribuzin on thyroxine and somatotropin levels in white rats. J. Toxicol. Envir. Health 40:15–34.

Porter, W. P., R. Hindsdill, A. Fairbrother, L. J. Olson, J. Jaeger, T. Yuill, S. Bisgaard, W. G. Hunter, and K. Nolan.
1985. Toxicant-disease-environment interactions associated with suppression of immune system, growth, reproduction. Science 224:1014–1017.

Reijnders, P. J. H.
1988. Ecotoxicological perspectives in marine mammalogy: Research principles and goals for a conservation policy. Mar. Mamm. Sci. 4:91–102.

Shane, S. H.
1977. Population biology of *Tursiops truncatus* in Texas. *In* Proceedings of the Second Conference on the Biology of Marine Mammals, San Diego, CA; 12–15 December, 1977. p.57.

Simpson, J. G., and M. B. Gardner.
1972. Comparative microscopic anatomy of selected marine mammals. *In* S. Ridgway (ed.), Mammals of the Sea, p. 298–419. Thomas, Springfield, IL.

Soto, A. M., K. L. Chung, and C. Sonnenschein.
1994. The pesticides endosulfan, toxaphene, and dieldrin have estrogenic effects on human estrogen-sensitive cells. Envir. Health Persp. 102:380–383.