CHAPTER 14

DISEASES AND MORTALITIES OF FISHES AND OTHER ANIMALS IN THE GULF OF MEXICO

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14.1 INTRODUCTION

How could the environmental health of the Gulf of Mexico (Gulf or GoM) be described in regard to fish kills, diseases, and parasitic infections before the Deepwater Horizon oil spill? This might seem like a simple question, but the answer is complex and difficult to report. Nevertheless, reporting some of that complexity is the aim of this chapter.

Many people consider the GoM to be restricted to the offshore waters, but studies on diseases in offshore water deeper than 200 meters [m] (656 feet [ft]) are few, not very detailed, incomplete, and not necessarily representative of the entire Gulf. Looking at offshore fishes in the Gulf, one should also consider the nursery grounds for those species. These grounds are usually the estuaries of the Gulf. Moreover, these estuaries are independent habitats that provide important aspects of understanding mortality and diseases of animals throughout the entire Gulf. This chapter treats the entire GoM.

Several factors have been impediments to research on this topic. An important reason for not being able to fully understand disease and its importance to the GoM involves the fact that when fishes become ill or otherwise stressed, they usually get preyed upon by predators and microbial consumers. Moreover, many diseases primarily affect the larval stages of the hosts, and individuals of these stages are so small that they are usually overlooked and seldom critically examined. When someone sees infected individuals, disease conditions may not necessarily be apparent. Infections in larval fishes by one or few individuals of a parasite species can have a role in disease epidemics. Observations of such cases can be supported by experimental laboratory studies. One critical reason that diseases in the Gulf of Mexico prior to the oil spill are not well-known is because some cutting-edge molecular diagnostic tools have been developed only recently and have not been widely applied for diagnosis of marine animal diseases. Another reason why diseases of offshore fishes as well as shellfishes, other invertebrates, marine mammals, sea turtles, and birds have not been studied relates to the high expense of collecting and studying the material. For example, charges for daily rental of large research ships can be as much as $50,000; the 29.7 m (97.4 ft) USM-GCRL R/V Tommy Munro, on which research involving the continental shelf is conducted, presently costs $7,200 plus the cost of fuel. Some of our past research has avoided high collecting costs because we took advantage of various inshore and offshore studies of fishes conducted by the National Marine Fisheries Service (NMFS) by partaking in its cruises, by accompanying other biologists on their trips, by obtaining parasites from some fishes collected by others, by conducting numerous inshore and offshore collection activities from our own small vessels, by assessing diseases, and most importantly, by utilizing the vast array of literature reflecting research conducted by others.

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Information reported in this chapter covers research conducted from riverine to offshore habitats that relate to the health of marine organisms, with an emphasis on fishes.

In this chapter, parasites that can cause diseases and mortalities will be discussed, but the harmless ones also will be considered as part of the biodiversity of the GoM. Since biodiversity is widely considered to correlate with ecosystem health, the presence or abundance of parasites becomes part of that positive biodiversity. Typically, the fewer the parasites observed, the worse the environmental conditions and thus the biodiversity. Knowledge involving biodiversity of pathogens, parasites, and hosts from the GoM over the last half century has increased greatly. In fact, because about half the living fauna are symbiotic, the biodiversity of infectious agents often outweighs that of hosts and potential hosts.

Because such an enormous number of mortalities of marine life in the GoM results from eutrophication, toxins from algal blooms like the red tide, and low temperature, those conditions are treated in some detail. Moreover, stress in animals resulting from those factors plays an important role in understanding infectious diseases. Consequently, we include some of that information involving mortalities and infections in this chapter.

The purpose of this chapter involves our understanding of the environmental health of the GoM prior to April 2010. When we consider the geographic scope of the entire Gulf of Mexico, we also consider three nearby regions to be included within its borders. We do this because of the immediately adjacent identical habitat, the interchange of water across the borders recognized by Felder and Camp (2009), and the similarity of their fauna to that encountered in the Caribbean portion of the Gulf of Mexico. The fish and parasite populations in three studied locations have a strong Caribbean influence as do the birds; however, not many Caribbean-Gulf collections have actually been made. Consequently, both the checklists by Overstreet et al. (2009) and this chapter include species and diseases extending slightly outside the designated GoM borders (Felder and Camp 2009). Those include habitats north through Biscayne Bay on the Atlantic side of Florida, those located off Cancún and Cozumel (slightly south of the Gulf border of Cabo Catoche, Yucatán, Mexico), and those off Havana, Cuba. As more fish and birds from the northern Gulf of Mexico as well as elsewhere in the GoM are examined, they surely will be found to be infected with new and unreported species and diseases. Consequently, we consider it important to include all fauna indicated above to best understand the fauna of the Gulf of Mexico as reported in a compendium edited by Felder and Camp (2009).

14.2 DEFINITIONS

When reading reviews such as this, a reader must understand that different authors in the literature can either (1) use different terms for the same subject/situation or (2) use one term for different situations. The clearest way to allow a reader to understand what is being written is for the author to carefully define a term or describe the subject of investigation. A few definitions provided below will guide the reader.

- *Allelopathy* refers to a biological phenomenon by which an organism produces one or more compounds that influence the growth, reproduction, or survival of other organisms.
- *Allochthonous* in this chapter refers to an organism that obtains energy/organic matter originating from outside the system, such as point-source discharges from rivers, but also from watershed runoff and coastal tidal inlets.
- *Autochthonous* means belonging to a particular place by birth or origin. For purposes of this chapter, it refers to an organism that generates organic matter within the system produced primarily through photosynthesis, by phytoplankton productivity, or by benthic regeneration.

- *Coherence* refers to shifting baselines relative to temporal collections; one reference site is not sufficient to capture random/natural variability.
- *Disease* as a simple term refers to any alteration from the normal state of health. In medical cases, this often refers to a dose of the causative agent above a threshold value that results in harm. The term "disease" differs from the term "syndrome" in different ways by different experts; however, Dorland's Medical Dictionary (1974) defines disease as "a definite morbid process, often with a characteristic train of symptoms" and syndrome as "a combination of symptoms [signs] resulting from a single cause or so commonly occurring together as to constitute a distinct clinical entity."
- *Epizootic* as an adjective refers to a rapidly spreading disease that is temporarily prevalent and widespread in an animal population, and as a noun, refers to an outbreak of an epizootic disease. *Epidemic* has the same definition, although it is restricted by some users to cases where the animal is human; a *pandemic* is an epidemic covering a large area.
- *Infection* as defined by American parasitologists usually refers to an internal association or a combination of internal and external associations, whether that relationship results in harm or not regardless of the size of the organism. For an internal association to be called an infection by a microbiologist, the organism is usually restricted to viruses, bacteria, protists, and fungi (compare infestation). To some microbiologists, microorganisms such as bacteria that live naturally in the mouth or elsewhere in a body without causing harm are not considered infections or infectious agents by many microbiologists, but the organisms are symbionts.
- *Infestation* refers to a variety of associations, depending on the author or country of origin. These meanings are (1) an external association, the definition we prefer, (2) a metazoan symbiont (parasite), (3) a parasite's colonization, utilization, or both of the host; (4) a host being colonized, utilized, or both by parasites, (5) an environment being colonized, utilized, or both by pests, (6) a population rather than incorporated individuals, and (7) an action (as opposed to the term "infection," which would suggest a condition or a state).
- *Neoplasm/tumor* also has confusing definitions. Tumor refers to an abnormal mass of tissue. It can be benign or malignant (cancerous). Consequently, not all tumors are neoplastic; they can even be a response to inflammation or constitute a parasitic infection. However, all cancers are neoplastic. A widely used but not always accepted definition of neoplasm by the British oncologist R.A. Willis states: "A neoplasm is an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues, and persists in the same excessive manner after cessation of the stimulus which evoked the change."
- *Symbiosis* defined herein refers to an association (mutualism, commensalism, or parasitism) between organisms of different species involving a unilateral or bilateral exchange of material or energy. A symbiont is any member, usually the smallest of a pair of organisms, involved in this symbiotic relationship. *Commensalism*: a symbiotic relationship in which one of two partner species benefits and the other shows no apparent beneficial or harmful effect. *Mutualism*: a symbiotic relationship in which two or more partners gain reciprocal benefits, usually mutual ones. *Parasitism*: a symbiotic relationship in which a symbiont lives all or part of its life in or on a living host, usually benefiting while harming the host in some way and usually having a higher reproductive potential than the host. For purposes of this chapter, not all parasites harm their host or not all components of a parasite's life cycle harm its hosts. Moreover, the

symbiont can be facultative or obligate, and it can infect either a natural or accidental host.

14.3 MASS MORTALITIES, PRIMARILY INCLUDING FISH KILLS

Mass mortalities of fish are usually caused by very specific conditions or agents. The following general mass mortalities occur commonly or at least expectedly are treated first and typically involve a variety of fish species. Additional diseases and die-offs caused by more host-specific agents and conditions are usually restricted to one or few host species and will be reported below in Sections 14.4, 14.5, and 14.6.

14.3.1 Eutrophication

Most recognized animal mortalities in the GoM result as an undesired product from eutrophication, normally the production of organic matter that forms the basis of aquatic food webs. While this process of eutrophication and of dying animals constitutes a natural progression, its rate depends on a complex of many factors. However, some aspects of this process are influenced by human input, the natural environment, or disease agents that weaken the victim. Most animals that die during this process die from oxygen depletion, from toxins produced by specific harmful algal blooms (HABs), or from predators taking advantage of the weakened condition of the prey.

Harmful concentrations of dissolved oxygen (DO) fit different categories. For example, the following categories used by the National Oceanic and Atmospheric Administration (NOAA) and others are (1) anoxia (0 milligrams per liter [mg/L]), (2) hypoxia (>0 and ≤ 2 mg/L), and (3) biologically stressful conditions (>2 and ≤ 5 mg/L). All the categories typically occur in June–October in bottom waters of most estuaries in all Gulf States.[1] Note that corresponding stressful levels have been defined more stringently by others such as Livingston (2001) as >2 and ≤ 4 mg/L. In a recent comprehensive survey of the trophic status of estuaries in the continental United States, Bricker et al. (2008) concluded that 84 estuaries, representing 65 % of the total estuarine surface area, presently showed signs of moderate to high eutrophic conditions. The main constituent of the organic matter is carbon, with the rate of eutrophication usually expressed as grams of carbon per square meter per year (gram carbon/square meter/year [g carbon/m^2/year]). A eutrophic rate in an estuary is 300–500 g carbon/m^2/year, with its effect on the ecosystem dependent on export rates of flushing, microbial respiration, and denitrification as well as on recycling/regeneration rates. Ecologically, eutrophication involves scales of both time and space. Autochthonous organic matter loading involves that matter generated within the system and is produced primarily through photosynthesis by phytoplankton productivity or by benthic regeneration. The resulting phytoplankton blooms depend on the amount of light and nutrients consisting primarily of total dissolved nitrogen and phosphorous, including both inorganic and organic forms, but also silicates and other nutrients. In estuarine habitats, primary producers other than phytoplankton include mostly benthic microalgae, epiphytes, seagrasses, and other submerged aquatic vegetation (Figures 14.1 and 14.2).

Allochthonous organic matter, originating from outside the estuary, can usually be traced from rivers but also from watershed runoff and coastal tidal inlets. The nutrient sources

[1] http://www.noaa.gov/factsheets/new%20version/dead_zones.pdf.

Figure 14.1. A moderate die off of the Gulf menhaden, *Brevoortia patronus*, caused by oxygen depletion in a Mississippi bayou on June 1984.

Figure 14.2. One of thousands of floating striped mullet, *Mugil cephalus*, having died from oxygen depletion near Galveston, Texas, in July 1993, exhibiting muscular degeneration and an extensive number of dipteran maggots feeding on the decomposing flesh.

include point source discharges such as from wastewater treatment plants, industrial plants, and logging operations and nonpoint discharges from agriculture, residential lawns, and gardens. Both sources can consist of particulate matter such as plant debris, detritus, and phytoplankton and of dissolved matter including humic substances such as humic acid, mucopolysaccharides, peptides, and lipids.

Total dissolved nitrogen, phosphorous, and silicates, including both inorganic and organic forms, are basic to production of phytoplankton, which plays a central role in carbon, nutrient (primarily nitrogen and phosphorous), and oxygen cycling in estuarine and coastal waters. Phytoplankters grow rapidly, often doubling in number each day. Members of some taxonomic groups proliferate so rapidly that they form dense blooms that can affect water quality as they die, decompose, and sink, utilizing and depleting oxygen from the bottom waters. Moreover, a complex relationship exists between phytoplankton and those animals that feed on them. These animals, zooplankton and benthic filter feeders as well as some larval, juvenile, and adult fishes, graze on, depend on, and control coastal phytoplankton. When phytoplankton blooms, primarily those blooms limited by nutrient supply and light during winter and early spring when water temperatures are too low to support rapid growth of the zooplankton grazers, the excess plankton dies, resulting in oxygen depletion and fish kills. The effects of temperature on phytoplankton growth and photosynthesis are similar for most algal species, with a relatively rapid decline in production at temperatures in excess of their optimum, for example 20 (68 °F) to 25 °C (77 °F). Moreover, the photosynthesis cycle can be influenced for different variants by temperature and toxicants (Cairns et al. 1975). Also, an important aspect of photosynthesis sometimes forgotten is that while oxygen is produced during the light portion of the photosynthesis cycle, oxygen is used up during the dark or evening portion of the cycle. Consequently, a net loss of oxygen production can occur during overcast days, when the upper layer of an extensive bloom blocks the light from reaching the lower level of phytoplankton, or other conditions decreasing light to the plant community.

In contrast to the case with abundant phytoplankton in a system, zooplankton growth rates are enhanced and their biomass increases when water temperatures warm up in the spring and summer. Also, concurrent availability of nutrients for the phytoplankton can decrease because freshwater runoff, the primary source of nutrients to an estuary, often decreases, resulting in the increased grazing on and control of the phytoplankton biomass by zooplankton, benthic filter feeders, larval fishes, and some juvenile and adult fishes.

Mass mortalities of the GoM's most important natural resources in coastal and estuarine sites usually occur ephemerally, but can occur continually or seasonally. Those mortalities that occur offshore like in the "dead zone" can take place for extended periods even though boundaries of the zone can change seasonally and annually. The influence on the decline in water quality and fragile habitat health associated with nearshore events responds to factors such as rapidly growing and diversifying anthropogenic inputs associated with agriculture, aquaculture, urbanization, coastal development, and industrial expansion. Fish gills often provide an indicator of degraded water quality (Figures 14.3 and 14.4). The mass mortalities will be discussed below, but the brief background on eutrophication in general requires some attention. More detailed treatments can be found in numerous publications such as NOAA (1997), Livingston (2001), Pinckney et al. (2001), and Paerl and Justić (2011). NOAA (1997) provides specific eutrophication data for all coastal water bodies in all five Gulf of Mexico U.S. states; the book by Livingston (2001) provides continuous analysis of various rivers in the Florida coastal systems in the northeastern Gulf of Mexico from 1970 to 2000; and Pinckney et al. (2001) and Paerl and Justić (2011) describe the role of nutrient loading and eutrophication in estuarine ecology. When forecasting the future hypoxia status, the model of Justić

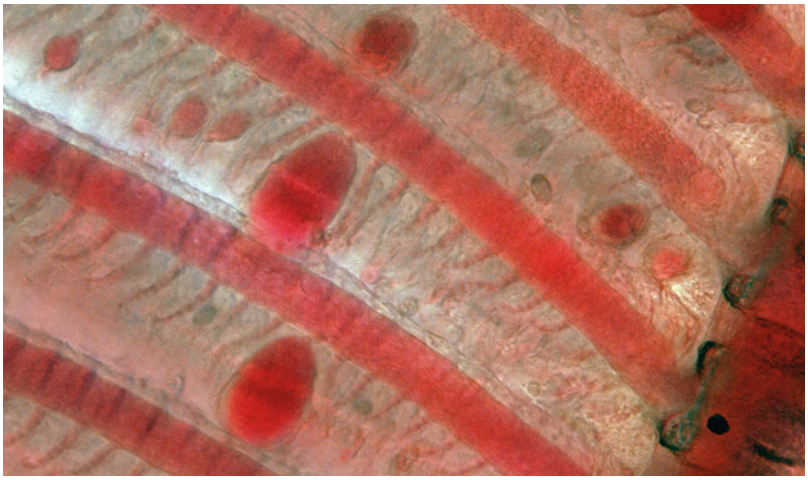

Figure 14.3. Secondary gill lamellae of the Atlantic croaker, *Micropogonias undulatus*, exhibiting progressive phases of telangiectasia, the swelling of weakened blood vessels, resulting from an early infection by the dinoflagellate *Amyloodinium ocellatum* in August 1998.

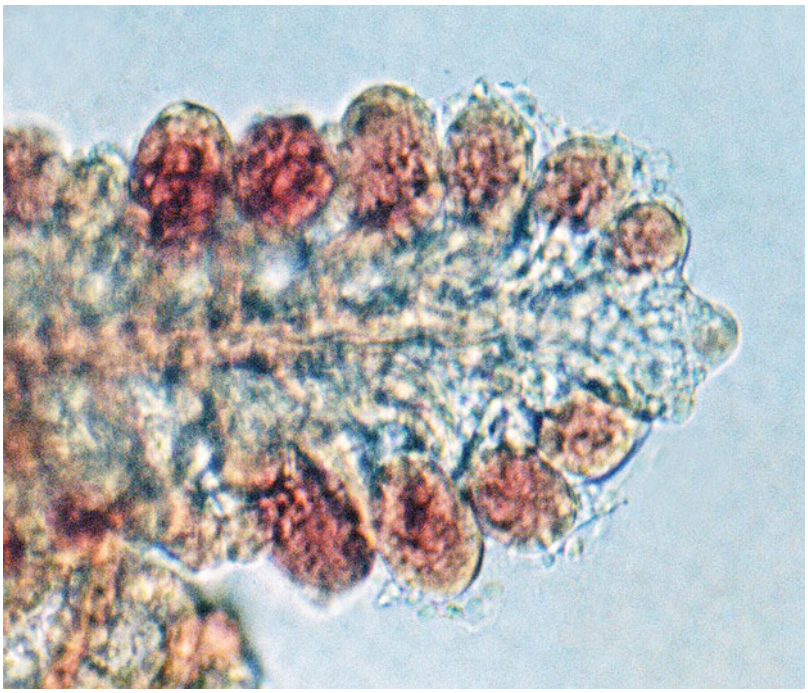

Figure 14.4. Telangiectasia, the reversible swollen blood vessels in the secondary gill lamellae of the inland silverside, *Menidia beryllina*, responding to harsh petroleum contamination.

et al. (2007) suggests that a reduction in riverine nitrogen of 40–45 % may be necessary to reach the goal of their action plan.

As a brief review, the common phenomenon of oxygen depletion usually results in mass mortalities in confined areas. For example, after a few days of overcast skies, photosynthesis with the accompanying production of oxygen during the daylight hours reduces. Without

Figure 14.5. Bluegill, *Lepomis marginatus,* with light infection of red sore disease caused by a combination of the bacterium *Aeromonas hydrophila* and the colonial peritrich ciliate *Heteropolaria colisarum* Mississippi, July 1974.

sunlight, such as during overcast or during night hours, the process of photosynthesis uses rather than produces oxygen. Consequently, if decaying matter such as dying algae, dead plant material carried from the rivers or marshes, or domestic waste from overflowing septic tanks accumulate in the confined areas, oxygen becomes depleted during both night and daylight periods. Fish will often try to avoid these conditions. Some species of fish such as menhaden, other clupeids, mullets, and catfishes are more sensitive to oxygen depletion than are other species and less likely to migrate away from areas with depleted oxygen. Some of these fish die and further reduce the amount of available oxygen, causing widespread and extensive fish kills, especially in harbors, dead-end canals, bayous, and small bays.

Fish involved in these kills are readily recognized by their pale or even whitish gills. These kills can be exacerbated by infestations of ectoparasites and bacteria. For example, centrarchid fishes such as the bluegill in Mississippi estuaries can be infested by the peritrich ciliate *Heteropolaria colisarum*. It, in turn, typically has a large concentration of the attached bacterium *Aeromonas hydrophila*. The ciliate feeds on free bacteria and organic debris, and *A. hydrophila* produces a series of proteolytic enzymes, some of which cause aesthetically displeasing lesions (Overstreet 1988) (Figure 14.5). These and other "red sore" lesions will be discussed in more detail under Section 14.4.1.1. Differentially expressed genes allow some organisms to tolerate low oxygen conditions such as in the grass shrimp *Palaemonetes pugio* exposed to cyclic hypoxia (Li and Brouwer 2013).

Mortalities caused by eutrophication occur so commonly on a seasonal and annual basis that reports seldom get published for individual cases in many areas other than in local media unless they are associated with specific bacteria, algae, toxins stresses, or other features. All harmful events do not necessarily kill the fish, and quite often the source is unknown (e.g., non-point source) (Figure 14.6). Compilations, however, are available such as for Florida (Table 14.1) and Texas and Louisiana (Zimmerman 1998; Thronson and Quigg 2008). Most mortalities seem to be caused by nuisance algae.

14.3.2 Hypoxia "The Dead Zone"

Eutrophication events discussed up to this point have dealt mostly with fish kills caused by oxygen depletion that occurred in bays and confined near-shore coastal habitats. However, other than areas in the Black Sea and Baltic Sea, a region in the northern Gulf of Mexico Continental shelf represents the largest coastal zone of hypoxia in the world. Even though this

Figure 14.6. Striped mullet, *Mugil cephalus*, caught by a commercial fisherman in December 1996 from the Pascagoula River, submitted to the National Marine Fisheries Service, and brought to us for evaluation. This case of unknown etiology affected a few of many present mullet with the pinkish-violet discoloration near a chemical plant.

Table 14.1. Dissolved Oxygen Fish Kills in Florida from 1973 to 2009 Based on Florida Fish and Wildlife Conservation Commission Fish Kill Database, http://research.myfwc.com/fishkill/[a].

Date Reported	County	City	Water Body Name	Specimen Count
11-Sep-02	Bay	Panama City	Mexico Beach, Beacon Hill area	2,000—species unknown
8-Jan-05	Bay	Panama City Beach	Crooked Creek	1,000s—mullet, shiner, bass
18-Feb-01	Charlotte	Bokeelia	Jug Creek on Pine Island	2,000—shrimp, sheepshead, species unknown
6-Nov-03	Charlotte	Placida	Placida Bay	1,000s—flounder, pinfish, pigfish, redfish
26-Oct-05	Charlotte	Port Charlotte	Vizcaya Lakes and Redwood Community	1,000s—bluegill, bass, crappie, perch, species unknown
8-Nov-05	Charlotte	Don Pedro Island	Don Pedro Island—Gulf Side	1,000s—coquinas
10-Jul-09	Charlotte	Port Charlotte	Strasburg Drive	1,000s—bass, minnow
1-Sep-09	Charlotte	Port Charlotte	Hog Island	1,000s—baitfish
23-Oct-09	Charlotte	Port Charlotte	Rossmere Road	1,000s—tilapia, bass
3-Jul-01	Citrus	Floral City	Tzalapopka Lake	1,000—bass, bluegill
16-Oct-02	Citrus	Hernando	Vanness Lake	2,000—bass, bluegill, catfish, minnows
2-Jan-04	Citrus	Crystal River	Crystal River Discharge Canal	1,000s—catfish, spotted eagle ray, sheepshead
4-Jan-04	Citrus	Citrus Springs	Barge Canal—Marker 33	1,000s—catfish, gar

(continued)

Table 14.1. (continued)

Date Reported	County	City	Water Body Name	Specimen Count
11–13-Sep-04	Citrus	Dunnellon	Withlacoohie, Withlacoochee	1,000s—bass, carp, brim, shiner, carp, catfish, shad
29-May–1-Jun-07	Citrus	Crystal River, Dunnellon	Lake Rousseau	3,000s—shad, catfish, shiner, catfish, tilapia
22-Apr-04	Collier	Naples	Lake Trafford	1,000s—catfish, bluegill, tilapia
9-Jun-04	Collier	Naples	I-75 and Pine Ridge Road	1,000s—species unknown
18-Aug-77	Escambia	Pensacola	Bayou Chico	30,000—menhaden
25-Jun-00	Escambia	Century	Unknown Water Body	2,000—shiners
27-Aug-01	Hernando	Webster	Withlacoocthee River	2,000—species unknown
26-Sep-98	Hillsborough	Tampa	Carrolwood Apts	2,000s—brim, species unknown
29-Sep-98	Hillsborough	Carrollwood	Colonial Guard Apts	2,000—brim, carp
6-May-00	Hillsborough	Riverview	Alafia—Buckhorn Springs	2,000—minnows, perch, redfish
2-Jun-00	Hillsborough	Seffner	Shangrila Subdivision	2,000—bass, crappie, tilapia
1-Aug-01	Hillsborough	Tampa	Davis Island	2,000—blue crabs, stone crabs, flounder, other species
25-Aug-01	Hillsborough	Tampa	Hillsborough Bay	2,000—All fish
17-Oct-02	Hillsborough	Lutz	Lake Hannah	2,000—bass, carp, skipjacks
10–11-Aug-03	Hillsborough	Tampa	Country Run Subdivision, Eagles Residential	1,000s—largemouth bass, shiner, bass
3-Oct-03	Hillsborough	Tampa	Bird Lake	1,000s—shiner, nile perch
2–6-May-04	Hillsborough	Apollo Beach	Masters Canal and area	1,000s—glass minnow, snook, catfish, tilapia, jack
18-Jun-04	Hillsborough	Gibsonton	Bullfrog Creek	1,000s—menhaden, sheepshead, pinfish, sand perch
13-Jul-04	Hillsborough	Tampa	Sleigh and Hillsborough River	1,000s—shad, tilapia, baitfish
1-Jun-05	Hillsborough	Apollo Beach	Masters Canal	1,000s—mullet
4–5-Sep-05	Hillsborough	Tampa	Waters Avenue, Woodlands Subdivision	1,000s—largemouth bass, bluegill, catfish, tilapia, species unknown
3-Aug-99	Lee	Fort Myers	Unknown Water Body	1,000—shiners
22-Aug-99	Lee	Fort Myers	Caloosahatchee River	1,000—baitfish
2-Apr-02	Lee	Fort Myers	Caloosahatchee River	2,000—bass, catfish, bluegill

(continued)

Table 14.1. (continued)

Date Reported	County	City	Water Body Name	Specimen Count
19–20-Apr-02	Lee	Fort Myers Beach	Estero Island	2,000s—baitfish
20-Apr-02	Lee	Fort Myers Beach	Moss Marina	2,000—baitfish
20–23-Apr-02	Lee	Sanibel	Sanibel River, Sanibel Island	2,000s—baitfish, minnows
12-Apr-04	Lee	Fort Myers	Private	1,000s—species unknown
5-May-04	Lee	Fort Myers	Lexington Country Club	1,000s—bluegill, carp
28-May-04	Lee	Sanibel	Clam Bayou	1,000s—redfish, snapper, mullet
11-Aug-05	Lee	Fort Myers	Grand Daza—Espero	1,000s—species unknown
6-Sep-05	Lee	Fort Myers	Hendry Creek	1,000s—mullet, sand perch, tilapia
27-Oct-06	Lee	Bonita Springs	Sheridan Run	1,000s—bass, carp
30-Aug-00	Manatee	Holmes Beach	Grand Canal	2,000—mullet, drum, shiners
31-May-06	Manatee	Palmetto	86 St. East	1,000s—bluegill, shiner
26-Jun-06	Manatee	Bradenton	Braden River	1,000s—bluegill, species unknown
19-Jun-09	Manatee	Bradenton	Braden River—Riverfront Drive	1,000s—snook, redfish, mullet, shiner
10-Aug-09	Manatee	Bradenton	Tidewater Preserve	1,000s—species unidentified
10-Mar-02	Miami-Dade	Miami	Venice Ave Bridge	2,000—mullet, catfish
14-Sep-02	Miami-Dade	Miami	Unknown Water Body	2,000—baitfish
22-Aug-05	Miami-Dade	Miami Springs	Miami Canal, Miami River	1,000s—species unknown
16-Nov-05	Miami-Dade	Miami	Saga Bay	1,000s—largemouth bass, peacock bass, shiner
20-Oct-09	Miami-Dade	Miami	NE 25th Court	1,000s—largemouth bass, bream, tilapia, snook
2-Oct-94	Monroe	Tavernier	Blackwater Sound	2,000—pilcards, barracuda, boxfish, puffers, spotted seatrout
20–22-Sept-03	Monroe	Key Largo	Largo Marina, Key Largo Marina	1,000s—Herring
2-Oct-06	Monroe	Lower Matecombe	Sleepy Lagoon	1,000s—glass minnow
19-Sep-09	Monroe	Marathon	Vaca Cut—109th Street, 109th St. Gulf	1,000s—sardine, pilchard, species unidentified
19-Jul-03	Okaloosa	Destin	Residential	1,000s—baitfish, softshell turtle

(continued)

Table 14.1. (continued)

Date Reported	County	City	Water Body Name	Specimen Count
11-Jul-98	Pasco	New Port Richey	Retention Pond	2,000—pinfish, sheepshead, whitebait
17-Aug-01	Pasco	Holiday	Horseshoe Lake	2,000s—species unknown
25-Aug-06	Pasco	Port Richey	Lake List to Lake Chrissy	1,000s—sunfish, brim, largemouth bass, species unknown
17-Dec-07	Pasco	Holiday	Rock Royal Dr.—Triangle Lake	1,000s—species unknown
2-Oct-09	Pasco	Odessa	Near Lake Josephine	1,000s—bream, crappie, tilapia, carp
6-Jun-94	Pinellas	St. Petersburg	Unknown Water Body	1,400—threadfin, herring
8-Nov-98	Pinellas	Clearwater	Off Exit 16	1,000—species unknown
13-Mar-00	Pinellas	St. Petersburg	Bartlett Park	2,000—snook, redfish
15-May-00	Pinellas	Clearwater	Feather Club Road	2,000—baitfish, brim
28-Jun-00	Pinellas	St. Petersburg	Lake Placid Mobile Home Park	2,000—species unknown
16-Nov-00	Pinellas	St. Petersburg	Spring Lake Apt	2,000—brim, bass
12-Jul-01	Pinellas	Oldsmar	Canal S of Lake Tarpon	1,000—shad
25-Jul-01	Pinellas	Pinellas Park	Residential Lake	1,000—bass, bluegill, shiners
19-Jun-02	Pinellas	Clearwater	Clearwater	1,000—baitfish
20-Aug-02	Pinellas	Oldsmar	Fillipi Park	2,000—catfish, trout, flounder, spade fish
8-Sep-03	Pinellas	St. Petersburg	Residential	1,000s—baitfish
14-May-04	Pinellas	St. Petersburg	County Lake	1,000s—tilapia, bluegill, crappie, golden
14-Oct-06	Pinellas	St. Petersburg	Lake Overlook	1,000s—needlefish, puffer, baitfish, greenback, pelican, blue heron
20-Aug-07	Pinellas	Seminole	Tides Golf Course	1,000s—tilapia
17–24-Aug-09	Pinellas	St. Petersburg	57th Avenue North and 112th Avenue NE	1,000s—bass, baitfish, bluegill, carp
5-Aug-78	Santa Rosa	Apalachicola	Garcon Point	10,000—alewives, croakers
2-Aug-82	Santa Rosa	Gulf Breeze	Behind Holiday Inn	15,000—alewives
5-Oct-04	Santa Rosa	Milton	Escambia—By Sandy Landing	1,000s—bass, bream, catfish
1-Nov-03	Sarasota	Venice	Residential	1,000s—species unknown
9-Jul-07	Sarasota	Sarasota	River Plantation	1,000s—bass, bluegill, species unknown
11-Jul-09	Sarasota	Osprey	Willowbend Pond	1,000—species unidentified

[a]Based on cases reported with more than 1,000 fish from a total of 1,198 cases.

zone, up to 20,700 square kilometers (km^2) (about 8,000 square miles [mi^2]) and reaching down to 30 m (about 100 ft) in depth, is called the "dead zone," it contains some life that can tolerate less than 2 mg/L oxygen. Rabalais et al. (2002) provided a good review of this seasonally and annually fluctuating zone. The zone typically occurs offshore from Louisiana between the mouth of the Mississippi River and the Texas border, but infrequently during some years it occurs off Texas, Mississippi, Alabama, and Florida. The zone receives high freshwater discharge from the nutrient-rich Mississippi and Atchafalaya rivers, and those nutrients and other organic matter help stimulate phytoplankton growth and create a stratified water column, differing in temperature, salinity, or both. The seasonally warmed surface waters establish a thermocline, with the less dense riverine fresh water further creating stratification with the saltier, cooler, denser water masses near the bottom. The phytoplankton not incorporated into the food web as well as fecal matter generated by the food web sink into bottom waters where the anaerobic bacteria decompose the matter, causing oxygen depletion. A well-defined seasonal cycle resulting from the strength and phase of river discharge, wind-mixing, regional circulation, and air–sea heat exchange processes usually generates maximum stratification during the summer and the weakest during the winter months. Because of these factors, the area comprising the zone fluctuates year to year (e.g., Fotheringham and Weissberg 1979).

In May–July 1979 after a heavy spring runoff and a diatom bloom, hypoxic bottom water developed in the upper Texas coast (Harper et al. 1981). Samples trawled from 6 m (about 20 ft) and 17 m (about 55 ft) depths consisted of only one fish species (hardhead catfish, *Ariopsis felis*), all individuals of which were dead or moribund as were many invertebrates, including the dominant polychaete population of *Paraprionospio pinnata*. Most Texas populations recovered in 1980; a few species of polychaetes that remained in low populations during the hypoxic period such as *Nereis micromma* and *Lumbrineris verrilli* increased in abundance immediately after the hypoxia abated probably because of larval recruitment; whereas others including *P. pinnata* with different life histories took much longer to reestablish.

The typical hypoxic zone, even though not as extensive as the one described above, appears from sedimentary evidence to have been present in the early 1900s and began to increase dramatically after about 1950. That is the time when the Mississippi Basin underwent a large human population increase with its increased nitrogen output through municipal wastewater systems as well as channelization and flood control of the Mississippi River along with associated deforestation, conversions of wetlands to cropland, loss of riparian zones, and expansion of agricultural discharge (e.g., Rabalais et al. 2002).

Life in the hypoxic zone differs according to the species and the oxygen concentration. Most fish are absent, some actually killed, in water with oxygen less than 2 mg/L; mantis shrimp and penaeid shrimps can tolerate 1.5 mg/L; epibenthic starfish and brittle stars die at <1.0 mg/L; and anemones, gastropods, and polychaetes die at <0.5 mg/L. At minimal levels of 0.2 mg/L, just above anoxia, sulfur-oxidation and bacteria form white mats on the sediments; at 0.0 mg/L oxygen, only black anoxic sediments exist without aerobic life. Demersal fish and invertebrates, those that live near the bottom, leave hypoxic areas and then re-occupy them by October–November. The distribution of sea turtles and cetaceans that prey upon those demersal animals seems to be somewhat dependent on the hypoxic zone (Craig et al. 2001). The oxygenated refuge habitats near the edge of the zone allow some animals to congregate (Craig 2012). The brown shrimp (*Farfantepenaeus aztecus*) and fish such as the Atlantic croaker (*Micropogonias undulatus*), spot (*Leiostomus xanthurus*), Atlantic bumper (*Chloroscombrus chrysurus*), and seatrouts collected with benthic trawls showed low DO avoidance thresholds and patterns of aggregations near these refuges. The brown shrimp, spot, and croaker showed a consistency between bottom DO avoidance thresholds and abundance in both catch per unit effort and laboratory experiments. Hazen et al. (2009) did not find that strong aggregation throughout the

entire hypoxic edge of the water column, but they did find a greater biomass in the upper 7 m (23 ft) and much less biomass below 13 m (43 ft) in their hypoxic stations compared with their non-hypoxic ones.

Specific events related to oxygen depletion such as the *jubilee phenomenon* serve as a local one of those conditions that result in edible fish for those lucky enough to take advantage of the resulting "kill." Jubilees are well known in specific areas in Alabama and Mississippi and result from specific conditions. Depending on those conditions, they can be spread out over 25 km (about 15 mi) or just a few hundred meters of beach. In Alabama, most occur in the upper Eastern shore of Mobile Bay from Great Point Clear to just north of Daphne, and in Mississippi, most occur off Bellefontaine Beach and Gulfport, although they can occur elsewhere. In Alabama, where jubilees are known from as far back as the 1860s (even though documents were searched dating back to 1821), the specific set of conditions involves early morning hours before sunrise in the summer, and overcast or cloudy previous day, a gentle wind from the east, a calm or slick bay water surface, and a rising tide. These conditions produce a stratified layer of salty Gulf water accumulating in the deepest part of the northern portion of Mobile Bay overlain by lighter, fresher river water. During the calm conditions, the salty water stagnates because of decomposing plant material washed into the bay from the upstream marshes and swamps as well as supplementation by domestic wastes and becomes low in oxygen concentration. The rising tide and gentle wind-driven surface current causes an upwelling of this stagnant bottom water, forcing some species of bottom fishes and crustaceans to move ashore (Loesch 1960; May 1973; Turner et al. 1987). In Mississippi, Charles Lyles (from Overstreet 1978; Gunter and Lyles 1979), who observed them since the late 1930s, found several conditions in common. Jubilees occurred during neap tide (tides with a small difference between high and low tide occurring after the first and last quarters of the moon) at night between late June and early September, usually with rain preceding them and water with a well-defined tea color, presumably resulting from a specific phytoplankton organism. Affected animals usually include flounder, stingrays, croaker, spot, eels, blue crabs, and shrimp plus a lot of usually inedible anchovies, needlefish, and catfish. Seldom do these fish die, but they occur in extremely dense groups gulping for air; the eels usually burrowed tail first into the moist sand with their mouths wide open. Since these occur in early morning hours, neighbors often tell other neighbors about the event so they can collect large quantities of fresh seafood in wash tubs for their freezers after being caught with nets and gigs. When the sun rises, the tide changes, or the wind direction changes, the phenomenon stops, and most of the affected fish swim away. Conditions for this phenomenon, such as the role of carbon dioxide, still require scientific attention.

Phytoplankton constitute the most abundant and widespread primary producers in GoM and world waters and therefore support the bulk of marine food webs. Several of the phytoplankton species, including members of toxic algae in addition to nuisance algae, also cause animal illness and mortality of fish and other animals.

14.3.3 Nuisance Algae

Numerous species of nuisance algae commonly produce mortality events throughout the Gulf of Mexico region. Along the West Florida Coast, primary species include *Synechococcus* spp., *Anabaena* spp., *Chlorococcus minutus, Microcystis aeruginosa*, and other cyanobacteria (previously referred to as blue-green algae) and dinoflagellate species. These events tend to occur from April to November and last weeks to months. In the Florida Panhandle, nuisance algal events are mostly episodic, with a duration of days, and occur between July and September. Species include *Anacystis* spp., *Anabaena* spp., *Cladophora* spp., *Enteromorpha* spp., *Chlamydomonas* spp., and *Aphanocapsa* spp. In the Mississippi Delta/Louisiana Coast

subregion, mortality events are mostly episodic, last from days in some estuaries to seasons in others, and generally occur between May and September in Mississippi Sound but also occur in January and February; in Barataria Bay, Louisiana, cyanobacterial blooms occur persistently throughout the year. Species in the subregion include *Exuviella* spp., *Prorocentrum minimum, Alexandrium* spp., *Anabaena circinalis, Katodinium rotundatum, Microcystis aeruginosa, Anacystis* spp., *Akashiwo sanguinea*, and others. Nuisance algal mortalities along the Texas coast occur mostly as day to month episodes between May and September except in the Upper Laguna Madre, Baffin Bay, and part of Lower Laguna Madre where *Aureoumbra lagunensis* occurs throughout the year. The latter alga produces brown tides, which occasionally block out sunlight and kill seagrasses; the blooms also occur in Florida and Mexico. During the period 1970–1995, the frequency and duration of events increased in Tampa Bay and Galveston Bay. Blooms of the dinoflagellate *Noctiluca scintillans* appear reddish orange during the day and can produce bioluminescence at night. Even though not a toxic alga, it can accumulate and emit ammonia in concentrations high enough to produce fish kills.

14.3.4 Toxic Algae: HABs, Including Red Tide

Some of the most prevalent toxic algae and associated toxins that cause animal mortalities in the Gulf of Mexico include *Alexandrium monilatum* (goniodomin A), *Karenia brevis* (brevetoxins), *Karlodinium veneficum* (karlotoxins), *Prymnesium parvum* (prymnesins), and *Akashiwo sanguinea* (surfactants). Other potential ichthyotoxic species are *Cochlodinium polykrikoides* (ichthyotoxins) and raphidophyte species such as *Chattonella marina, Heterosigma akashiwo*, and *Fibrocapsa japonica* that produce hemolysins, reactive oxygen species, polyunsaturated fatty acids, and possibly brevetoxins (Lewitus et al. 2014).

Toxic algal events in the GoM estuaries are variable in duration, lasting days to weeks in some estuaries and months to seasons in others. Impacts generally occur between June and October, except in Florida Bay and Apalachee Bay, where impacts occur between January and March. Occasionally, however, unpredictable toxic algal events may occur during any month of the year (NOAA 1997).

14.3.4.1 Red Tides, *Karenia brevis*

Most dinoflagellates are photosynthetic, possessing chlorophyll *a* and accessory pigments, and not toxic; they constitute an important and at times the dominant group of primary producers sustaining the food web. When some toxic species bloom, they cause massive fish kills. Red tide serves as the most well-known HAB in the Gulf of Mexico, with the best known species being *Karenia brevis* (previously known as *Gymnodinium breve*). A heavy bloom produces a reddish color in the water and is responsible for spectacular mass mortalities. Importantly, aerosols from a heavy bloom usually affect human respiration and occasionally cause contact dermatitis, which, in turn, provides considerable more incentive and support for research than would be received from fish kills alone. The U.S. population continued to increase between 1960 and 2010 and is projected to increase further, most significantly in coastal states, putting stress on the coasts and estuaries. Between 1965 and 1976, the number of confirmed worldwide red tide outbreaks increased sevenfold concurrent with a twofold increase in nutrient loading mainly from untreated sewage and industrial waste (Hallegreaff 1995). The threat to animals from red tide blooms is predicted by the number of dinoflagellate cells of *K. brevis*/L from a table by Lewitus et al. (2014) as (1) 1,000 cells or less (none anticipated), (2) >1,000 to 10,000 (very low, with possible human respiratory irritation, and shellfish harvesting closures when >5,000 cells/L), (3) >10,000 to 100,000 (low, human respiratory

irritation, possible fish kills, and bloom chlorophyll probably detected by satellites), (4) >100,000 to 1,000,000 (medium, human respiratory irritation and probable fish kills), and (5) >1,000,000 (high, as above plus discolored water).

Blooms of the toxic alga *K. brevis* occur almost annually in the Eastern Gulf of Mexico, most frequently in Southwest Florida waters. Consequently, blooms are commonly referred to as "Florida red tides" and, as indicated above, have attracted research dollars for several decades. In fact, the University of Miami's initial Marine Laboratory, now known as the Rosenstiel School of Marine and Atmospheric Science, was established by F.G. Walton Smith to investigate red tides (e.g., Gunter et al. 1947, 1948). Also, Sammy Ray, along with Albert Collier and William Wilson, established the Galveston Laboratory of Texas A&M to investigate red tide and culture of *K. brevis* (see Zimmerman 2010). Gunter (1947) provided a short history of the Florida red tide in which he deduced that the phenomenon had been reported since 1844. He considered the death of the fish most spectacular because the dead fish floated, a diagnostic feature for fish killed by brevetoxin. He estimated the 1946–1947 red tide killed an estimated half billion fish; he said that such catastrophic kills may cover >25,000 hectare (ha) (hundreds of square miles), and the number of fish killed may even approach 1 billion. He also considered that few, if any places, on earth can produce such vast destruction of life so quickly as the dinoflagellate blooms of the shallow sea with the possible exception of fish kills along the Peruvian coast caused by El Niño. Blooms of *K. brevis* typically occur in the Gulf of Mexico; however, they can be entrained in the loop current and transported east through the Florida Straits and then north by the Gulf Stream as far as North Carolina. Quick and Henderson (1975) investigated the pathology of fish from a 1973 to 1974 Florida kill, and their evidence suggested that dehydration, hemolysis, and interference in blood-clotting mechanisms also caused fish-death in addition to neurointoxication, the previously assumed sole cause.

Brevetoxins from *K. brevis* are indeed complicated. There are several non-proteinaceous, lipid-soluble neurotoxins as well as hemolysins. For example, Baden and Mende (1982) investigated the toxicity of two of those toxins, using Swiss white mice and the western mosquitofish as assay animals. In the mice injected with one of the toxins, hypersalivation was the most pronounced sign, although copious urination and defecation commonly occurred as well as tremors, followed by marked muscular contractions. The mice exhibited compulsory chewing motions and rhinorrhea at higher doses. When given the other toxin, a distinct compound but with related chemical structure, no hypersalivation or chewing was expressed and muscular contraction was less pronounced. Mouse bioassays were used to determine the correlation between acute intraperitoneal injections and oral toxicity of shellfish extracts, and the oral assay was not recommended. The disease in humans eating brevetoxin-contaminated mollusks that goes by the name "neurotoxic shellfish poisoning" (NSP) can be debilitating but apparently non-fatal. The first toxin tested seemed to be the predominant agent responsible for the disease; the second at the dose tested produced subacute manifestations that occur in the human disease such as labored breathing, loss of appetite, and motor incoordination. Signs of the disease generally subside in 2–3 days. These signs from both toxins are typical of muscarnic stimulants, as found in *Amanita muscaria* (a poisonous mushroom), as opposed to nicotine, another stimulant acting on acetylcholine receptors and bind to voltage-sensitive sodium channels involved in the propagation of nerve impulses. Binding opens the sodium channels at a normal resting potential and consequently inhibits sodium channel inactivation, which can result in repetitive firing in nerves. Further studies described by Baden et al. (2005) characterized additional brevetoxins, each with its own specific toxicity and based on one of two different structural features (six toxins known with one and three, thought to be more potent, with the other). More importantly, these multiple brevetoxins activate brevetoxin metabolites, which can be modulated by the different, shorter, trans-fused polyether antagonist brevenals. Brevenal,

obtained from either the environment or the dinoflagellate culture, binds receptors and inhibits brevetoxin binding and activity, counteracting the toxic effects on both mice and fish. The pulmonary receptor for both brevetoxins and brevenal seems to be distinct from the neuronal binding site. In other words, the multiple biotoxins and antagonists interact with at least neuronal, pulmonary, and enzymatic regulatory systems of animals, generating a complex combination of acute and chronic signs in animals, including humans, exposed to aerosolized bioactive substances produced by *K. brevis*.

Most data on Florida red tide fish kills acquired up to the last decade or so were anecdotal and qualitative but useful. Gannon et al. (2009) investigated the effects of the algal blooms on nearshore fish communities in five habitats in Sarasota Bay and adjacent areas. They looked at the cell density of *K. brevis* as well as data on fish density, fish species composition, water temperature and salinity, dissolved oxygen, and turbidity. The clupeid (herring-like fish) trophic guild (a guild [or ecological guild] consists of any group of species that exploit the same resources) was not affected by the cell density of the toxic algae as were all other eight fish trophic guilds. Fish density as measured by catch per unit effort (CPUE) and species richness of those other eight guilds all had a negative association with the algal cell density; 96 % of the local fish kills from 2003 to 2007 (ranging from 4 in 2007 to 72 in 2005, with more nearby) occurred during red tides. The guild consisting of the demersal invertebrate feeders was the most sensitive to the effects of the red tide, whereas the clupeids were the least sensitive, and, when excluding the clupeids, the difference between CPUE in red tide period versus non-red tide period ranged from 57 % in the mangrove habitat to 88 % in the GoM habitat. Fisheries-independent monitoring data (as opposed to fishery-dependent data, which are data collected directly from commercial and recreational fisheries sources) from the Tampa Bay area collected from 1996 through 2006, with an emphasis on the persistent red tide of 2005, analyzed by Flaherty and Landsberg (2011) showed that in the spring of 2006 there was a decline in the annual recruitment of juvenile spotted seatrout (*Cynoscion nebulosus*), sand seatrout (*Cynoscion arenarius*), and red drum (*Sciaenops ocellatus*). However, the subadult and adult abundance values for these fishes remained consistent with those of previous years. The respective recruitment periods of some of the other fishes did not correspond with the major red tide event. The importance of clupeid fishes such as Spanish sardines, thread herrings, and Atlantic shad in the understanding of fish kills has been recognized by Walsh et al. (2009).

The dinoflagellate *K. brevis* requires nutrients to form the catastrophic blooms. A nitrogen isotope budget of the coastal food web shows that diazotrophs (nitrogen fixers, primarily the filamentous cyanobacteria *Trichodesmium* spp.) form the initial nutrient source of red tides and clupeiformes (decomposing dead sardines, herrings, and bay anchovies) serve as the major recycled nutrient source for the maintenance of those blooms. In 2001, the dinoflagellate "harvested" >90 % of the clupeids along the West Florida Shelf rather than being harvested by fishermen. Fish kills typically originate when *K. brevis* cells lyse and release their toxins, which become absorbed directly across the gill membranes. Fish may also die after ingesting the dinoflagellate cells or toxins in the water, or after consuming contaminated biota (Landsberg et al. 2009).

The Center for Prediction of Red Tides (CPR) in Florida (Walsh et al. 2009) has developed models to assess and predict red tides based on nitrogen isotope ratios in portions of the food web that maintain *K. brevis*. The food web associated with *K. brevis* has shown to be extremely complicated and differs somewhat in different areas based on currents and winds. Some model components are based on features such as temperature. At summer temperatures, as much as 50 % of some Florida fish decay to inorganic forms of phosphorus and nitrogen within 1 day (Stevenson and Childers 2004; Walsh et al. 2009). Some clupeids can provide about 50 % of the nitrogen supply for red tides. Of equal concern is the nearly equal inclusion of the diatom-

based food web, including flagellates, that also feeds the herbivores (harpacticoid and calanoid copepods and certain other members of the zooplankton), in turn feeding phytoplankton-feeding fishes (clupeiformes mentioned above including the Gulf menhaden *Brevoortia patronus*, which feeds on both phytoplankton and zooplankton, plus the mugilid [striped mullet, *Mugil cephalus*] that feeds additionally on bacterial degraded phytodetritus) and the piscivorous fish like mackerel, snappers, and groupers that feed on them. Isotope data and animal kills suggest the kills in Florida and the northern Gulf in one year, like 2006, can show how the kills decreased on the West Coast of Florida and then increased on the East Coast of Florida in 2007. The tides have "downstream" consequences up to 1,000 km (621.4 mi) from the Florida Panhandle to Cape Hatteras on the Atlantic coast.

Small fish kills can also be related to dust and associated nutrients blown into the Gulf from African and occasionally Asian deserts (Garrison et al. 2003), and those kills can include related toxic dinoflagellates in addition to *K. brevis.* Actually, in the Gulf of Mexico, there are at least nine known established species in the Kareniaceae, and most produce ichthyotoxins such as brevetoxins, karlotoxins, and gymnodimines (Steidinger et al. 2008). These include five species of *Karenia* (*K. brevis*, *K. papilionacea*, *K. mikimotoi*, *K. selliformis*, *K.* cf. *longicanalis*), three of *Takayama* (*T. pulchella*, *T. helix*, and *T. tasmanica*), and *Karlodinium veneficum*, the latter confirmed as cause of fish kills in estuarine ponds. *Karenia brevis* typically occurs in high salinity waters. In 1996, a bloom occurred in inshore waters of Alabama, Mississippi, and Louisiana, contaminating oyster beds. This bloom consisted of a complex of *Karenia* species, some of which can tolerate low salinities (5–40 parts per thousand) (ppt), but *K. brevis* was the most prominent species. Maier Brown et al. (2006) examined preserved specimens from this bloom, and they also investigated salinity tolerances of three clones of *K. brevis* and compared them with a fourth. For the three clones, the experimental minimum salinity at which growth occurred ranged between 17.5 and 20.0 ppt and optimum salinity range from 20–25 to 37.5–45 ppt, depending on the clone. In the northern Gulf of Mexico bloom, the concentration of cells/milliliter (mL) for the complex was high enough to close oyster beds in salinity as low as 14 ppt. Some agents occurred in salinities less than 10 ppt in both the northern Gulf and in Florida. Brevetoxins measured in the *K. brevis* cultures were found to be higher during the stationary phase of growth and approaching senescence, regardless of salinity, suggesting that as a natural bloom ages, it could potentially become more toxic and pose an increased threat to public health. The specific 1996 bloom seemed to originate in the Florida panhandle and move westward, rather than the typical eastern movement, into Mississippi Sound because of the unusual effects of Tropical Storm Josephine (Maier Brown et al. 2006).

Fish kills resulting from *K. brevis* also occur in Texas and Mexico. Gunter et al. (1948) reported on such massive fish kills, Zimmerman (1998) edited a report covering such mortalities of a variety of animals in Texas and Louisiana in 1994, and Magaña et al. (2003) tabularized and discussed a series of referenced reports of fish kills from various locations along the Texas coast as well as Tamaulipas-Veracruz and Yucatán, Mexico, which occurred from 1935 until 2002. Because of the severe respiratory events involving irritation, stinging eyes and nose, accompanied by a dry, choking cough, resulting from inhalation of air-borne brevetoxins, historic references provide information on Mexican events occurring from 1648 to 1875 (Magaña et al. 2003) and earlier. One case in 1792 chronicled by a government official and reported by Lerdo de Tejada (1850) indicated that sales and consumption of dead fish collected from the mass mortality of fishes on Veracruz beaches resulted in violent human mortalities. Nuñez Ortega (1878) and later others suggested that the human deaths actually resulted from bacterial contamination of or ciguatera toxins in spoiled fish. Fish kills probably resulting from *K. brevis* along the Texas shelf occurred during 1529–1534 (Adorno and Pautz 2003). Cabeza de Vaca was a survivor of the Narvaez Expedition and reported that the Capoque and Han Indians

avoid fish and suspend oyster harvesting seasonally around Galveston Island; during 1534, the Avavares Indians near the Nueces River, Texas, apparently estimated seasonal changes by "the times when the fruit comes to maturity and when the fish die" (Walsh et al. 2009).

Bony fishes constitute most of the commonly killed animals, and, as indicated above, some are important sources of stored brevetoxin necessary for future blooms. They can build up to high dangerous levels in living fish tissues by being in the water with *K. brevis*, by feeding on contaminated mollusks and other invertebrates, or by feeding on contaminated fish; toxins can be abundant in the entire food web (Naar et al. 2007; Landsberg et al. 2009). Until 2000, no mass mortality of sharks or rays caused by red tide had been reported from Florida. Flewelling et al. (2010) reported the mortality of large numbers of blacktip sharks (*Carcharhinus limbatus*) and fewer Atlantic sharp nose sharks (*Rhizoprionodon terraenovae*), mostly juveniles, from the Florida Panhandle. They also examined tissues from 22 species of sharks and rays collected between 2000 and 2008 from animals both in and not associated with red tides along the West Coast of Florida and the East Coast, where some of the animals also accumulated the toxins. The amount of accumulated toxins differed among species, tissue sites, and geographical locations, and in-utero embryos also had accumulated brevetoxins. The brevetoxin concentrations in animals do not necessarily relate to being from or near blooms, and levels are not harmful for human consumption unless the liver is eaten. Large sharks seem to avoid the toxin.

Waterfowl can also be affected by red tide blooms. For example, several thousand individuals of the lesser scaup (*Aythya affinis*) and lesser numbers of other birds were found dead associated with the red tide fish kill in the Tampa Bay area. Not all birds present died. Examination for bacteria, parasites, pesticide residues, and acutely toxic material did not suggest that any was associated with the mortalities. White Peking ducklings experimentally exposed to the red tide toxins in seawater, either in addition to force-fed contaminated clams (*Mercenaria campechiensis*) or given non-contaminated clams, became lethargic, developed spastic movements of the head, and died (some individuals in the toxic seawater with non-exposed clams did not die) (Forrester et al. 1977). When Ray and Aldrich (1965) force-fed three doses of experimentally exposed oyster tissue to baby chicks, all doses produced in the chicks a loss of equilibrium, and the two higher doses produced death within 22 h. Shorebirds, including sanderlings (*Calidris alba*) and ruddy turnstones (*Arenaria interpres*), scavenged on beached individuals of the thread herring, scaled sardine, and mullets during a red tide kill. High concentrations of brevetoxin in those fish tissues corresponded with high levels in livers of shorebirds that were collected dead along the local beaches and from rehabilitation centers during the red tide event, suggesting that brevetoxin exposure serves as a risk factor for bird mortality (van Deventer et al. 2012).

Since red tide blooms have been known in the Gulf of Mexico, they have been associated with mortality of numerous animals at higher trophic levels, such as marine birds, sea turtles, and marine mammals (Gunter et al. 1948; Quick and Henderson 1974; Forrester et al. 1977; and others). Because of the ability for fishes and invertebrates (see list of maximum brevetoxin concentrations in bivalves listed by Landsberg et al. (2009)) to bioaccumulate the toxins, blooms do not necessarily have to be present to kill animals. Landsberg et al. (2009) listed hundreds of manatees (*Trichechus manatus*) and bottlenose dolphins (*Tursiops truncatus*) killed in both reported and unpublished mass mortalities and not necessarily concurrent with blooms. Even though presently impossible to determine specific lethal concentrations of the toxins and their metabolites, the presence of high levels in the animals was either solely responsible for the deaths or in combination with other harmful factors. Twiner et al. (2012) critically investigated bottlenose dolphin mortalities from the Florida Panhandle and found high levels as they also did for the clupeid *Brevoortia* sp., which was found abundant as a dietary prey in their stomach. When dead manatees from the 1996 red tide bloom were necropsied, Bossart et al. (1998)

observed severe nasopharyngeal, pulmonary, hepatic, renal, and cerebral congestion in all cases. Some exhibited pulmonary edema and hemorrhage. Immunohistochemical staining using a polyclonal primary antibody to brevetoxin exhibited intense positive staining of lymphocytes and macrophages in the lung, liver, secondary lymphoid tissues, nasal mucosa, and meninges. These data suggest that manatee mortality may occur after chronic inhalation and ingestion rather than responding in an acute event. Local rehabilitation centers have successfully recovered several species of birds, turtles, and manatees that would otherwise probably have died from the red tide. The reason humans do not die or become severely ill from inhaling aerosols or ingesting brevetoxin accumulated in fish or bivalves probably relates to their ability to avoid lethal doses. This contrasts to ciguatera toxin, which is a similar compound acting in the same manner; however, its toxin from the epibenthic dinoflagellate *Gambierdiscus toxicus* can be bioaccumulated in fishes to a much more harmful concentration without causing mortality of the fish (Naar et al. 2007).

14.3.4.2 Fish Kills From Algal Agents Other than *K. brevis*

Additional investigations on pathology of fish will show other related agents being responsible for fish mortalities. When fish kills occurred in estuarine aquaculture facilities in Maryland, they were determined to be caused by at least two isolated karlotoxins from the dinoflagellate *Karlodinium veneficum* (as *K. micrum*) by Deeds et al. (2006). *Karlodinium veneficum* has been reported from Florida in the Gulf of Mexico, has caused fish kills in Maryland and South Carolina, and is considered a cosmopolitan species. Fish from kills near Perth, Western Australia, examined by the senior author had diagnostic epithelial necrosis and shortening or loss of the secondary lamellae of the gills, the primary signs observed in the sheepshead minnow (*Cyprinodon variegatus*), a common fish in the northern Gulf of Mexico. Concentrations of toxins in filtered water from fish kills rapidly killed the experimental fish.

Also, the dinoflagellate *Pfiesteria piscicida* can produce lesions, and at one time was considered the cause of ulcerated mycosis of Atlantic menhaden, resulting in fish kills along the Atlantic coast to the GoM (Dykstra and Kane 2000) (Figure 14.7). Considerable research has gone into the cause of these lesions, and now Blazer et al. (1999) and Vandersea et al (2006) have determined that the primary agent is the pathogenic oomycete *Aphanomyces invadans*. *Pfiesteria piscicida* and later *Pseudopfiesteria shumwayae* (see Litaker et al. 2005) were originally thought to secrete potent exotoxins that caused the lesions, acute fish kills, and human disease in the mid-Atlantic estuaries. However, bioassays with *P. shumwayae* and larval fish revealed no toxin was emitted and mortality occurred only in treatments where fish and

Figure 14.7. Atlantic menhaden (*Brevoortia tyrannus*) from St. Johns River, Florida, in June 1985, exhibiting typical lesions now recognized as caused by the oomycete fungus *Aphanomyces invadans*. Fish collected and photographed by Harry Grier of the Florida Department of Natural Resources. Permission to reprint granted by H. Grier to R.M. Overstreet.

dinospores demonstrated physical contact. Dinospores swarmed toward and attached to the skin, actively feeding on and denuding fish of their epidermis and killing them by micropredation (Vogelbein et al. 2002).

Some dinoflagellates produce a toxin harmful and even deadly to humans and marine mammals, but not recognized as causing fish kills. One of these toxins is saxitoxin (STX) puffer fish poisoning, which can also on occasion include paralytic shellfish poisoning (PSP). The signs of eating toxins accumulated in puffers progress from tingling and numbness of the mouth, lips, tongue, face, and fingers; to paralysis of extremities, nausea, vomiting, and ataxia; to decreasing breathing and possibly to death by asphyxiation. The toxin occurs in the Gulf of Mexico as determined by Landsberg et al. (2006) and Deeds et al. (2008). The toxins can be produced by *Pyrodinium* by means of the shellfish, *Alexandrium cohorticula, A. minutum, A. ostenfeldii, Gymnodinium catenatum*, and some freshwater cyanobacteria, all of which occur in the Gulf of Mexico, but verified cases caused by the toxins in the GoM come from the bioluminescent *Pyrodinium bahamense.* Within all puffer species, they are stored in the skin, muscle, and viscera with an emphasis on ovary, making those structures a risk for human consumption. The toxin in the southern puffer (*Sphoeroides nephelus*) from the Gulf side of Florida is much less in quantity than in fish from the Atlantic side, where it can remain not depurated for over a year. However, one should realize that toxin produced by one strain of a species often does not represent that production for the species. For example, the toxin for PSP produced by 17 strains of the dinoflagellate *Alexandrium tamarense* had a wide range in the amount based on mouse bioassays. Furthermore, 15 sub-strains taken from one of those strains also had a considerable range in the amount, and that toxin from two different strains differed in the derivatives produced (see Thessen et al. 2009).

The golden alga *Prymnesium parvum* occurs worldwide, but it is best known from inland waters of Texas and estuaries of the Gulf of Mexico. Under certain environmental stresses, it produces massive fish kills, including kills of mussels and clams. Even though the alga has been identified from many locations, it often does not cause mortalities. Allelopathy has been shown to be one reason. That is a biological phenomenon by which an organism, in this case a concurrent cyanobacteria (a prokaryotic phytoplankton that has bacteria-like cellular features such as lacking a well-defined nucleus and membrane-bound organelles), produces one or more substances that influence the growth, survival, or reproduction of another organism. James et al. (2011) showed that one substance, the cyanotoxin microcystin-LR, inhibited growth of *P. parvum*, but the necessary concentration could also kill a number of other aquatic organisms.

Another non-dinoflagellate alga that attracts attention is a complex of diatoms responsible for "amnesic shellfish poisoning" (ASP). Most diatoms constitute highly proactive phytoplankton in estuaries, supporting both planktonic and benthic food webs, but the colonial *Pseudo-nitzschia* spp. produce a domoic acid toxin (DA) that causes ASP. The toxin accumulates in bivalves, but ASP is most common along the Pacific Coast in upwelling systems where seabirds and marine mammals die from it, and, consequently, marine resource management agencies both along the Pacific Coast and the GoM close shellfish beds when DA levels are high because ASP causes loss of memory in humans.

Diatoms in the genus *Pseudo-nitzschia* occur frequently in the northern Gulf in offshore and estuarine plankton, on sediments, and in both shellfish tissues and seawater in Mississippi Sound as well as in Alabama, Louisiana, and Texas (Dortch et al. 1997; Macintyre et al. 2011). Although not all species of this genus are toxic, and no case of human ASP has been reported from the GoM, when counts of the diatom and concentrations of DA in oyster tissue exceed federal guidelines, oyster reefs are temporarily closed. However, because DA occurs in GoM shellfishes, because it is produced by several species in the genus, and because it imposes a major human threat, it is presently being investigated in some detail. Even though the disease is

considered a problem in high salinity waters, various species occur over a salinity range of 1 to >35 ppt in Louisiana, where oysters are typically harvested in 10–20 ppt (Thessen et al. 2005). These authors identified seven species in low salinity waters, and some are toxigenic. Much has been learned about these diatoms from laboratory work as well as from species around the world. Experimental studies have shown that the problem is extremely complex. Different strains of one species isolated from the same water sample exhibited broad differences in growth rate and toxin content when cultures contained different nitrogen sources, ammonia, nitrite, and urea (Thessen et al. 2009). Two clones of one species produce toxins; however, they preferentially utilized different nitrogen sources. Two of nine isolates of another species and two of five of still another produced DA, but the content varied by orders of magnitude. If that does not exemplify the complexity of the problem, then it should be noted that DA, in addition to being accumulated in bivalves, also occurs in tissues of zooplankton, crustaceans, echinoderms, echiurans, tunicates, and fishes; it also occurs in tissues of marine mammals, birds, and humans, all of which could be killed by it, as well as occurring in sediments, demonstrating stable transfer through the marine food web and abiotically to the benthos (Trainer et al. 2012). The latter review included considerably more information on the cosmopolitan nature and complexity in taxonomy, toxin production, toxin storage/release, bloom initiation/retention, and nutrient requirements for some of the 14 recognized species, and also mentioned that preliminary work suggested the necessity for the presence of an epibiont bacterium before sexual reproduction could occur in some clones of one species grown in axenic culture.

To reiterate the aspect of human illnesses from HABs, those that occur worldwide result from harmful algal toxins and their derivatives including saxitoxins (STX, including some paralytic shellfish poisoning [PSP]), okadaic acid (diarrheic shellfish poisoning), brevetoxins amnesic shellfish poisoning [ASP] (neurotoxic shellfish poisoning (NSP)), ciguatoxins (ciguatera fish poisoning), domoic acid (am/domoic acid poisoning), azaspiracid toxins (azaspiracid poisoning), and hepatoxins and microcystins. Dinoflagellates produce all these toxins except for domoic acid, which as discussed above is produced primarily by diatom species of the genus *Pseudo-nitzschia*, and hepatoxins and microcystins produced by cyanobacteria such as species of *Anabaena* and *Microcystis*. In addition to being produced by dinoflagellates, saxitoxin can be produced by several species of cyanobacteria, and brevetoxin can be produced by some species of raphidophytes. Deadly phycotoxins include domoic acid, saxitoxins, and ciguatoxins. Perhaps the deadliest of the phycotoxins are the STXs because of the rate of human mortality associated with exposure and the broad geographic range of distribution of STX-producing organisms. Saxitoxins produced by multiple dinoflagellate species as well as several species of cyanobacteria and can cause PSP. Moreover, the toxins can be transferred and bioaccumulate throughout aquatic food webs and therefore be vectored to terrestrial biota, including humans (Deeds et al. 2008). Ciguatera is more common in Mexican and eastern Caribbean reefs than in the northern Gulf (e.g., Okolodkov et al. 2007).

At least 15 species of *Prorocentrum, Dinophysis*, and *Phalacroma* are known to produce okadaic acid (OA) or its derivatives in the world's oceans, and those species occur in the GoM. However, only isolates of *Dinophysis* cf. *ovum, Prorocentrum texanum, P. hoffmannianum*, and *P. lima* have been demonstrated to produce OA in the Gulf. The toxin accumulates in bivalves, and the human disease associated with eating such bivalves is termed "diarrhetic shellfish poisoning"; conclusive evidence pointing to OA by itself causing fish disease has not been established.

14.3.5 Cold Kill

Cold kills appear conspicuous after a period of low temperature. They, however, are restricted to shallow waters and not as common as one might believe. Under normal conditions,

when a cold front passes through an area, most fishes and invertebrates bury or migrate to more tolerable areas and do not die. Typically, it is the rate at which the temperature drops rather than the temperature per se that kills fish. Fish kills are more prevalent in the typically warmer southern waters of Texas and Florida than in the more temperate northern Gulf of Mexico where the rate change during freezing conditions is not as great and the fishes are more able to acclimate. A good example of this situation occurred in Mississippi in January 1973 and was studied in some detail by Overstreet (1974). During the evenings of January 13–14, 1973, a thin sheet of ice covered the surface of Paige and Cooper bayous in Jackson County, Mississippi. On the 15th, these bayous, approximately 1 to 5 m (about 3 to 16 ft) deep and completely fresh during this time in the year, became covered by a layer of the striped mullet, *Mugil cephalus*, which had surfaced and died. By January 16, the 0.6 m (2 ft) tide washed out the majority of fish, but a minimal estimation of a few hundred thousand carcasses still remained. A large number of shellcrackers, bream, bass, and catfish actively fed when local residents, who frequently fed them, placed food in the water, suggesting that no toxin occurred in the water and no low concentration of oxygen existed. In the morning of the 16th, several coastal habitats were inspected for dead and living fish, and corresponding values were obtained for salinity, chlorosity, and calcium in the water. A few other bayous also contained dead striped mullet such as the Ocean Springs Small Craft Harbor, which contained additional dead species of the striped mullet such as white mullet (*Mugil curema*), Atlantic tarpon (*Megalops atlanticus*), and fat sleeper (*Dormitator maculatus*).

Fishermen caught striped mullet during and after January 13 and 14 in nearby Graveline Bay, Bayou Porteaux, and other areas where no dead fish was observed. The unusual thing about the areas from which the mullet survived was that the water had a salinity greater than 6 ppt. At least the dying fish from Paige Bayou also exhibited starvation, had distended gallbladders with associated leaking bile (Figure 14.8), and demonstrated high levels of dichlorodiphenyltrichloroethane (DDT) metabolites and endrin pesticide residues unlike mullet samples from where no fish had died. An average-sized dead mullet was 230 millimeters

Figure 14.8. Striped mullet, *Mugil cephalus*, exhibiting viscera showing enlarged leaking gallbladder and intestine devoid of food and representative of moribund mullet during a cold kill on November 16, 1973.

(mm) (9 inches [in]) standard length, with a weight of 255 grams (g) (9 ounces [oz]), and large fish such as these are more susceptible to a variety of stresses. Foci of hepatic necrosis and an abundance of lipid material but not glycogen were demonstrated in hepatocytes of the fish livers from the mass mortalities relative to control samples. Far fewer ciliate protozoan parasites and no monogenoid or copepod infested the gills of dying mullet, and those parasites were also common in Davis Bayou where there was no mortality.

Experimental studies (e.g., Cummings 1955; McFarland 1965) have shown that when the striped mullet is gradually transferred from seawater to freshwater, it can regulate serum ion concentration, muscle ion concentration, and osmolarity and surface permeability may be reduced by prolactin in relationship with temperature. At least those dying mullet in water less than 6 ppt salinity with 4.5 g chloride/L and 94 parts per million (ppm) calcium probably had a failing ion-osmoregulatory mechanism and were unable to acclimate to the rapidly dropping temperature.

On January 25 along Cooper and Paige bayous and on January 22 in a canal off Mary Walker Bayou, each location had a few thousand bloated and decomposing floating dead fish with attached filamentous algae as long as 4 cm (1.6 in). At the same time, healthy mullet without any indication of attached algae were present (see later comment on pseudo-fish kills). In his lengthy discussion about all aspects of the mortalities, Overstreet (1974) discounted with adequate evidence several hypotheses for the mortalities, presented by interviews with longtime residents of the area.

The most severe cold fronts appear to affect the coastal biota of Florida, Texas, and occasionally in between. Severe cold fronts, presumably with air temperature less than −12 °C (10 °F), recorded for coastal Mississippi include at least January 1899, February 1914, January 1985, and December 1989 (Bergeron 2015). Cold fronts passing over the shallow waters of the Gulf in western Florida occasionally result in chilled and helpless or dead fish with massive numbers washed ashore. In waters of Cedar Keys and north, most fish in a 3-day 1917 cold wave left the coastal waters for protection from the rapid temperature drop. Dead fish were usually small, 5–8 cm (2–3 in), accompanied by crabs and small shrimp. Near Tampa, mullet, grunts, and jacks died, and further south toward Key West, "tons" of fish became numb, washed ashore, and had to be buried to avoid the stench (Finch 1917). Finch (1917) even quoted a Federal fisheries biologist as saying that a benefit of that cold spell to oysters was the near eradication of a parasite that had previously been killing the oysters near Cedar Key and Port Inglis.

Willcox (1887) reported that thousands of smelly fish killed in bays and rivers from Cedar Keys to the mouth of the Caloosahatchee River at Punta Rassa by an 1886 freeze. The numbers and species of dead fish, including oysters, differed by location, but few actually occurred along the shore of the Gulf, and those that did occurred near inlets and probably resulted from tidewater carrying them out from the bays. Nine freezing episodes at Sanibel Island, Florida, from 1886 through 1936 were reported by Storey and Gudger (1936), who listed the 1886 one as the worst for both fishes and vegetation. The air temperature in Fort Myers was −4.4 °C (24 °F) and that near the salt water was −2.2 °C (28 °F) and lasted for a day; water temperature never reached 0 °C (32 °F). About 1.3 cm (0.5 in) of ice formed in the cisterns and rainbarrels; the weather turned warm and it rained after the freeze. Generally, the local common fish species often died, but in some cases the larger fishes became lethargic and recovered before they washed ashore. Only the hardiest of fish at Sanibel Island can tolerate a water temperature rapidly dropping below 15.6 °C (60 °F). Lethargic fish have often been gathered and eaten in Florida as well as Mississippi. Those in Florida, especially those already putrefying, are often gathered and used as fertilizer. Apparently, fishing typically recovers within 2 to 3 weeks after a freeze. Another major fish kill in southern Florida occurred during January 27 through 29, 1940, when minimum air temperatures ranged from −0.6 °C (31 °F) in Miami to 10 °C (50 °F) in Key

West. Most of the killed fish included bonefish, moon fish, several different snappers, grunts, porgies, mullet, and jacks. Lesser numbers of several fishes also died or became lethargic (Miller 1940). An estimate of nearly 450,000 kilograms (kg) (1,000,000 pounds [lb]) of stunned but good edible specimens were gathered and sold by fishermen from Key Largo to Key West. Digital thermal infrared data acquired by a NOAA-5 meteorological satellite followed three consecutive cold fronts which crossed South Florida and northern Bahamas in January 1977 (Roberts et al. 1982). The third and most severe frontal system crossed the shallow, carbonate Florida Bay and depressed water temperature for 7–8 days below 16 °C (61 °F), a thermal threshold for most reef corals. Water temperature in Florida Bay decreased to at least 13 °C (55 °F). Coral and fish kills occurred along the Florida Reef Tract, with mortality at Dry Tortugas estimated at 91 %. Low water temperature was suggested as the major factor-inducing stress in this reef system. Roberts et al. (1982) discussed works by others indicating extensive drowned and killed Holocene coral reefs in the southeastern Florida shelf margin during the first stages of shallow, widespread flooding of the shelf during the sea level rise occurring approximately 7000 years Before Present. They considered the topography of the southeastern Florida shelf and other high latitude reef areas as probably being dramatically affected by the combination of reef growth and severe cold water stress.

Texas is probably the most vulnerable area on earth to cold kills. It occupies approximately 900,000 ha (3,400 mi^2) of bay waters with offshore depths being only 1.8–2.4 m (6–8 ft) deep. Polar fronts push south to the southern part of the state and occasionally are strong enough to cross the Gulf of Mexico and over the Isthmus of Tehuantepec down to Nicaragua on the Pacific coast. Fish kills extend south into Mexico (Gunter 1947). The shallow bays of Texas are connected to the GoM by typically narrow passes; consequently, the rapid drop in temperature often traps the fishes within the bays. Gunter and Hildebrand (1951) described animal kills occurring in 1951 in and around Aransas Pass. The storm with winds up to 64 km/h (40 mi/h) dropped temperatures below freezing on January 29 and remained there for 5 days, with air temperatures as low as −8 °C (18 °F). Gunter (1941) also described that the animals killed from a front passing through the same general area during January 18–22, 1940. In both cases, there were several million fish and other animals killed by the cold and numerous others numbed. Those two papers considered the freezes somewhat equivalent catastrophes to those of 1924, 1899, and 1886, but certainly not as severe as those in 1941 and 1949, although considerable mortality occurred in the 1947 freeze. Gunter (1947) considered a catastrophic cold kill to occur on the average of every 14 years from 1856 to 1940 with less damaging ones occurring at shorter intervals. Biologists of the Game, Fish, and Oyster Commission estimated that the amount of fish killed in 1951 ranged from 27 to 41 million kg (30,000–45,000 tons). The dead species differed in different areas, but most included the hardhead catfish, spotted seatrout, red drum, black drum (*Pogonias cromis*), mullets, silver perch, spot, Atlantic croaker, bay anchovy (*Anchoa mitchilli*), striped anchovy (*Anchoa hepsetus*), Atlantic cutlassfish (*Trichiurus lepturus*), toadfish, and other fishes as well as the brown shrimp, a variety of crabs, bivalves including oysters, and the occasional brown pelican, lesser scaup, white egret, and other birds plus loggerhead turtle. Based on photos of windrows roughly 0.4 km (approximately 1,500 ft) long of mass mortalities in Laguna Madre, it was concluded that southern area also incurred heavy fish kills extending for some 50 km (30 mi) along the upper Laguna shore, with lesser damage in the lower Laguna. Many of the fish as well as clams, gastropods, and starfish that died along the shore of the Gulf of Mexico became lethargic and rolled up on the beach by the heavy surf caused by the norther. In the 1980s, Texas coasts experienced three winter mass mortalities with 14 million fish killed in December 1983, 11 million in February 1989, and another 6 million in December 1989 (McEachron et al. 1994). McEachron et al. (1994) used a stepwise, standardized approach to sampling, which they admitted caused an underestimated mortality

count, especially for small (<200 mm [8 in]) animals as well as the illegal activities of fishermen removing dead and dying fish prior to the census. The composition of the fish species accounting for over 50 % in each freeze were striped mullet (*Mugil cephalus*), pinfish (*Lagodon rhomboides*), Gulf menhaden (*Brevoortia patronus*), and bay anchovy. They noted that the size classes of a species affected varied between some of the freezes, but not all species, e.g., pinfish. This observation they felt led to an "instantaneous picture" of the species population structure at the time of the kill. Hence, the recommendation to fisheries managers was to respond to mass mortalities by imposing regulations to reduce fishing efforts immediately following the event to allow recruitment and compensatory mechanisms to take place.

Commercial fishing after the cold kills, at least the 1940 episode, showed a dramatic decline (Gunter 1941). While there was some difference in the decline among commercial catches from the regions of Galveston, Matagorda, Aransas, and Laguna Madre, the red drum, spotted seatrout, and black drum all declined by 78 % while that of the southern flounder declined by 95 %. However, dead flounder do not float and because of their shape they are not easily trawled or dredged, so some mortalities could have easily escaped notice. He also tabulated data for catches after the 1940 freeze from both the year of the freeze and of the prior year and noted that there was no difference in decline from catches in the Gulf of Mexico. But there was in the bays, where the water was much shallower. It took about 3 years for the commercial catch to recover. Texas fishermen seem to agree that fish will be scarce for a few months after severe cold spells; whereas those from Florida and Mississippi estimate a 2- to 3-week period. As suggested above, this difference can be explained primarily by many of the fish in Florida, Mississippi, and offshore Texas migrating to more tolerable water or recovering after a water temperature rise after being affected but not killed.

The nice thing about cold kills is that residents as well as numerous animals such as piscivorous birds and raccoons make a healthy feast of the freshly dead or numbed fish! Such is not the case for fish killed by most other causes.

14.3.6 Pseudo-Fish Kills

"Pseudo-fish kills" is our term for fish that had died a week or two earlier and submerged, only to undergo bacterial degeneration during the warm period following the cold weather. Cases of pseudo-fish kills also can occur from fish kills other than those caused by low temperatures. Metabolic byproducts or gases consisting of methane, hydrogen sulfide, and carbon dioxide are produced, becoming trapped in the body cavity. Once enough gas accumulates, the body becomes lighter and the dead fish floats to the surface. Many local citizens are sure they see fish in the state of dying and report a fish kill to state agencies, research facilities, and newspapers. Our examination of such fish, always of decayed and smelly fish usually with attached green algae, indicated that the actual mortality had taken place during the prior cold snap or other mortality event.

14.3.7 Heat Kills

Because the rapid rate change from normal to low temperatures is usually what kills fish during a freezing period, one might expect a rapid rate change from normal to high temperature to be the cause of fish kills. Theoretically that could happen, except that even when the temperature of water starts increasing at a relatively rapid rate, most fish readily escape to relatively cooler habitats. There are situations, usually those resulting from anthropogenic changes involving heated effluents, which can kill aquatic organisms. Also, there are cases where fish get inadvertently washed up into a vulnerable position and are inescapably trapped in

a body of water that rapidly increases in temperature before reduction of acceptable oxygen levels, killing fish. That situation is rare. Usually when the water temperature is increased, it takes a long enough period that eutrophication takes place and fish actually die from oxygen depletion rather than from a high temperature. Probably most important, this increased temperature reduces resistance to diseases and allows the agents to become established and to become more pathogenic. In such cases, the weakened fish become readily devored as prey items before they die from disease; they are not witnessed as dead bodies. Complicated changes in normal parasite life cycles can occur such that a parasite such as a trematode or nematode will produce an infection at a different time of year than it is typically found in the normal environment. Because of the seasonal biology of the fish, infections might take place in an unpredictable situation during an atypical season, resulting in fish death or morbidity.

We know of no marine example in the Gulf of Mexico involving parasites relative to thermal pollution; however, Khan and Hooper (2007) evaluated the effects of thermal discharge on the parasites of the winter flounder (*Pseudopleuronectes americanus*) near the coastal fossil fuel generating plant at Holyrood, Newfoundland, Canada. The water discharged into Conception Bay, but the temperature change extended up to only 1 m (3 ft) below the surface, which was 3–4 °C (37–39 °F) in May and 7–8 °C (45–46 °F) in June compared with benthic water at 0 °C (32 °F). Only summer samples were taken, although sampling occurred at a few reference sites. Biological features such as condition factor and organ indices sampled below the plume revealed no significant difference with reference samples except that the male somatic index was significantly greater than that of the other two sampled groups. The parasites, however, were another matter. Metacercariae of the heterophyid *Cryptocotyle lingua* had a greater prevalence and mean intensity of infection than reference samples, whereas the mobile peritrich ciliate *Trichodina jadranica* and the monogenoid *Gyrodactylus pleuronecti* occurred significantly less on the gills of fish samples beneath the plume when compared with those from the reference sites. Additionally, four internal parasites, one myxosporidian and three trematodes, were significantly more abundant in the reference samples, suggesting an environmental change-affected transmission of the parasites when exposed to the thermal effluent. The salmonid brown trout (*Salmo trutta*) showed an attraction to the hot water effluent from the Forsmark nuclear power plant located in the low salinity coast of the Bothnian Sea associated with the Baltic Sea, Sweden. Thulin (1987) reported a few of 401 Swedish fish with skeletal abnormalities, 22 % with the leech *Piscicola geometra*, and 9 % with the copepod *Caligus lacustris*, all apparently unexpected findings. Khan and Hooper (2007) provided several freshwater examples involving parasite indicators of thermal effluents, but we only mention one (Camp et al. 1982) in which the prevalence and abundance of metacercaria of the trematode *Ornithodiplostomum ptchocheilus* in the western mosquitofish (*Gambusia affinis*) over a 53-month study expressed a fluctuating difference in infections in thermal effluent and ambient temperatures during 31 of those months in a thermal reservoir in South Carolina. The thermal effluent initiated the trematode life cycle a few months earlier than that occurred in the ambient water, with both shedding of the cercariae and recruitment of the metacercariae being affected; but so were the nesting and foraging activities of the waterfowl definitive hosts that tend to prefer the warmer water in winter and cooler water in the summer. Of course the biology of the fish and snail were also affected.

To take complications and thermal interactions one step further, the effect of viruses on cyanobacteria should be considered. For example, *Microcystis aeruginosa* is a common species responsible for blooms and for producing HAB toxins. This situation is usually considered as part of the relatively simple eutrophication process. However, there are several undescribed viruses that infect the "blue-green algae" including *M. aeruginosa*, and which probably influence mortality. They are likely a primary factor in determining plankton crashes;

moreover, it is temperature that seems to control the crashes (Honjo et al. 2006). Paerl and Huisman (2008) stress the point that rising temperatures, often above 25 °C (77 °F), favor cyanobacteria over diatoms, green algae, and other phytoplankton species. The resulting stratification earlier in spring and destratification later in autumn increased residence times and reduced vertical mixing. More intense precipitation with associated increased nutrient discharge in conjunction with the viral infections all ultimately promoted blooms and crashes with their associated fish kills.

Hot water pouring into a cooling canal from the Florida Power & Light Company fossil fuel generating plant at Turkey Point in 1969 killed thousands of fish. The company was operating under a special permit to discharge water into Biscayne Bay at temperatures higher than allowed by pollution laws. These mortalities occurred before the company finished construction of two nuclear generating plants. In addition to the dead fish, there was a variety of dead crustaceans, mollusks, corals, and algae occurring up to 1.4 km (1 mi) from the outfall during early summer in 1969. A virtually complete kill of aquatic organisms occurred over an area of about 8.4×10^5 m^2 (200 acres) (Laws 2000). Because of those expected harmful effects, Roessler and Tabb (1974) conducted an extensive survey and determined that average temperature elevations above ambient summer water temperatures caused the depletion in the biota. The area which was elevated above 4 °C (39 °F) was approximately 30 ha (75 acres), the area between 3 (37 °F) and 4 °C (39 °F) was approximately 40 ha (100 acres), and the area between 2 (36 °F) and 3 °C (37 °F) was approximately 50 ha (125 acres). A total area of about 120 ha (300 acres) showed a decline in abundance of biota that was statistically measurable for at least part of the year. A relatively rapid recolonization took place during the winter in a portion of this area. The inner barren zone of about 20 ha (50 acres) recovered slowly because of the death of the rhizomes of the turtle grass and changes in the sediment. The optimal temperature for maximum biodiversity was between 26 (79 °F) and 28 °C (82 °F); about 50 % of the animals were excluded when the temperature was between 30 (86 °F) and 34 °C (94 °F). About 75 % were excluded when the temperature was between 35 (95 °F) and 39 °C (102 °F), over the thermal tolerance range (TTR). Most of the animals occurred where the red algae complex of species of *Laurencia* and *Digenea* was abundant; whereas few animals occurred where no algae or seagrasses occurred. These two authors predicted that an increased temperature with increased water flow in conjunction with the nuclear generators would harm an increased area without implementation of alternate methods of cooling. No indication of pollution as measured by standard chemical indicators resulted from secondary treated sewage from a local sewage treatment plant, suggesting that the kill in 1969 was caused entirely by elevated temperatures resulting from the power plant and not eutrophication. Another kill of about 2,000 fish occurred in June 1971 when the discharge water from the fossil fuel plant again reached 40 °C (104 °F) (Associated Press 1971).

The above case uses both reports of counted or estimated dead fish and reports documenting lack of or less abundant catches of fish relative to other reference collections. More often when dealing with temperature, nothing exists but circumstantial or theoretical data. This is especially true with temperature because numerous factors influence other factors, and conditions are seldom repeated so they can be adequately compared. For example, Cairns et al. (1975) provides a good article on the effects of temperature upon toxicity of chemicals to aquatic animals. The amount of literature is extensive but mostly based on laboratory studies and not adequate to make "scientifically justifiable generalizations." The number of variables is extensive, especially when including the interaction of temperature with what type or degree of toxicity; what chemical, state of compound, or mixture of compounds; what organism, in what life stage, and under what physiological condition; and environmental influences such as salinity, pH, and alkalinity. Temperature can be both a lethal factor and a controlling factor

but without consistent thresholds. Temperatures outside the zone of tolerance fall in the zone of resistance, and the length of time before death is useful for trying to determine the cause of thermal death. For example, because tissue anoxia occurs at high temperatures, the toxicants act differently: copper increases metabolic demand and zinc blocks oxygen uptake by the gills, and either may be rendered more active physiologically by an increase in temperature. The body temperature of most fishes and other animals, except marine mammals and birds, corresponds almost exactly with the water temperature, taking about 3 min/1 °C for heat exchange; and the rate of metabolism undergoes an approximately twofold increase with every 10 °C (50 °F) rise in temperature, commonly referred to as Q_{10}. Cairns et al. (1975) provide and discuss the effects of temperature on a wide variety of toxicants; however, few are examples from the Gulf of Mexico and most deal with experimental studies. DeLorenzo et al. (2009) showed that temperature, salinity, and life stages of grass shrimp all affect the degree of toxicity of common pesticides to this shrimp. Lloyd (1987) provided more detail on interactions and modification of the response caused by variation of physiochemical conditions. An experimental study (Bao et al. 2008) also demonstrated that the effects of temperature above the TTR significantly increased the toxicity and hence the ecological risks of two common anti-fouling biocides (chlorothalonil and copper pyrithione) to a copepod (*Tigriopus japonicus*) and a dinoflagellate (*Pyrocystis lunula*). Male copepods were more sensitive to both compounds than the females, and the toxicity of the two biocides differed.

14.3.8 Hypersalinity (Over-Salinity)

In areas of the GoM where levels of salinity can become great enough to kill fish, the levels seldom actually reach those high concentrations. The best known is Laguna Madre of the Texas coast; this lagoon, approximately 210 km (131 mi) long by 6.5 km (4 mi) wide, is separated from the Gulf of Mexico by a narrow barrier known as Padre Island. Under normal conditions, commercial fisheries production is greater in this lagoon that in any other region of Texas (Gunter 1947). During dry years, the salinity may reach three times that of normal seawater (about 32 ppt), killing vast numbers of fish (Gunter, on our reprint, crossed out the words "specific gravity" and replaced it with "salinity"). Even though not well documented, large kills occur approximately every 10 years, but minor kills occur every year. The number of kills probably increases as the lagoon fills. We are not aware of the development of any recent artificial pass leading to the Gulf of Mexico.

Every few years in Mississippi (because of the prevailing winds), the salinity in the bays and bayous will vary between 0 and $\geq$40 ppt. During the periods of high salinity, we have not seen fish kills, but we note that the components of the biota differed dramatically from that expected. For example, we have occasionally seen fishes such as the Spanish mackerel (*Scomberomorus maculatus*), lookdown (*Selene vomer*), and Atlantic moonfish (*Selene setapinnis*) as well as the bottlenose dolphin abundant in the Back Bay of Biloxi.

14.3.9 Sulfate Reduction and Anaerobic Methane Oxidation

A different environment occurs in deepwater bathyal areas of the Gulf of Mexico associated with hydrate-bearing sediments where crude oil and methane advect through fault conduits to the seafloor. These areas can be considered toxic to the surrounding aerobic fauna that occupies most of the seafloor, but the occupants come in contact with different chemical compositions. The oil and gas seeps located at 590–630 m (1,900–2,100 ft) at 6–9 °C (43–48 °F) are typically overlain by chemo-synthetic communities consisting of thiotrophic bacterial (e.g., *Beggiatoa* spp.) mats, methane atrophic mussels (*Bathymodiolus* spp.), and other symbiotic

associations. These well-established geologic areas are dynamic, resulting in fluctuating faunas within and surrounding them. For example, bottom waters contain about 29 mM/L of dissolved sulfate, but pore fluids from oil and gas seeps are depleted down to 0.3 mM/L sulfate. That ambient bottom water contains less than 1 μM/L sulfide, but the sediments contain 1 mM/L and all pore fluids from seeps contain up to 20 mM/L, with those concentrations in gas seeps generally higher than those in oil seeps at the same depth. This inverse relationship between sulfate and sulfide results from bacterial consumption of sulfate and concomitant production of hydrogen-sulfide during anaerobic sulfate reduction. Bacterial mats of *Beggiatoa* spp. at the seawater–sediment interface obtain their energy by oxidizing hydrogen-sulfides and producing molecular sulfur. In contrast, the *Bathymodiolus* spp. mussels from the hydrocarbon seeps, but not hydrothermal vents, contain mostly methanotropic bacterial symbionts (e.g., Aharon and Fu 2000). Gas and crude oil escape by venting and seepage is ongoing, but major expulsion events are estimated to take place at a frequency of every 300 or even less than a hundred years (Roberts 2011). Gas hydrate, an ice-like substance comprised of a gas molecule like methane surrounded by a crystalline cage of water molecules as ice, concentrates vast amounts of methane in water at depths greater than 300–500 m (980–1640 ft) and contributes to seafloor hazards. In the GoM, the gas hydrates undergo repeated near-surface formation and dissociation varying seasonally and with warm-water loop eddies. However, the largest natural geohazard associated with hydrates and methane release involves periodic landslides (Hutchinson et al. 2011). Anaerobic oxidation of methane is a microbial process taking place in anoxic marine sediments where oxidization occurs with different terminal electron acceptors such as sulfate, nitrate, nitrite, and metals.

14.3.10 Sediments and Drilling Fluids

Sedimentation resulting from storms and river flow can have a major effect on mortality of fish and covers invertebrates. Examination of gills of morbid or dead fish allows differentiation among fish killed by cold or toxins (usually reddish unless acid, nitrate in freshwater fishes), oxygen depletion (pink or white), or sediments (mud, sand, or eroded tissue).

Drilling fluids (muds) are used by offshore petroleum-drilling operations. These aqueous suspensions consist of a variety of components that are pumped down the center of the drill bit and have a composition that varies with the needs of the drilling operation. Examples are lubrication, cooling, prevention of intrusion of seawater into the borehole, antibacterial action, suspension of drill cuttings, and capture of hydrogen sulfide. The fluid may be partially or entirely discharged into the surrounding water during and after the procedures. Because of the toxic nature of some of the fluids, the U.S. Environmental Protection Agency (USEPA) conducted some laboratory tests to determine whether some substances interfered with fertilization and normal development of fish and invertebrates (Crawford 1983). Different fluids had different toxic effects. As an example, a concentration of 10 ppt of some fluids caused the diminution of heartbeat rate of the model killifish; concentrations of 1 ppt had an effect on hatching and coordination of swimming of the fry of that fish. Some fluids had no effect on the fish, and no one fluid could be considered typical. Consequently, it is hard to evaluate the effect of drilling fluids on wild populations, but animal communities surrounding drilling operations can be compared with those on nearby reference sites not undergoing such activities.

14.4 FISHES

The following sections treat mortality, health, and indicators of specific groups of animals, starting with fishes and separate from the above sections that treated general mass mortalities.

14.4.1 Infectious Parasites and Diseases

The status of fishes and the fish communities in the GoM needs to be understood relative to natural and contaminated conditions so that microbial and parasitic diseases and fish mortalities can be assessed critically. Helpful background information on many of the important model fishes such as Atlantic croaker, bay anchovy, hardhead catfish, Gulf menhaden, spot, and pinfish has been provided by Lewis et al. (2007). Comparative data are also available for the effects of hurricanes on these fish assemblages (Lewis et al. 2011).

Because the majority of the fish that die in the GoM usually are not seen or sampled since they become part of the food web, the impact of parasite-induced mortality on a population is difficult to determine (Scott and Dobson 1989; McCallum and Dobson 1995; Rousset et al. 1996). Consequently, statistical approaches have been established to estimate mortality. Lester (1984) presented six methods to estimate mortality caused by parasites in wild fish populations. Shaw et al. (1998) reviewed 49 published wildlife host-macroparasite systems and determined that in 90 % of the data sets, the negative binomial distribution provided the statistically satisfactory fit. This statistical analysis has been used by many to account for estimates of the mortality in populations due to parasite infections (May and Anderson 1978; Dietz 1982; Kennedy 1984; Scott 1987; Shaw and Dobson 1995).

Statistical evidence of parasites controlling the abundance of a population has been demonstrated by Hudson and colleagues (Hudson 1986; Hudson and Dobson 1990, 1997a, b; Hudson et al. 1985, 1992, 1998; Dobson and Hudson 1992) using the interaction of the nematode *Trichostrongylus tenuis* on the red grouse, *Lagopus lagopus scoticus*. Bruning et al. (1992) developed a population-dynamic model for phytoplankton and the impact of fungal parasites on their populations. They calculated that four parameters are needed to determine the loss-rate: prevalence of infection, developmental time for the parasite, specific growth rate of the uninfected host, and the difference between the infected and uninfected host mortality due to factors other than parasitism. Knudsen et al. (2002) used a long-term study with indirect methods to indicate that the nematode *Cystidicola farionis* increased the mortality rate in its final host (Arctic charr, *Salvelinus alpinus*). They indicated parasite-induced host mortality in hosts older than 10 years. The convex age-abundance curve indicates that heavily infected fish disappear from the population. This loss of fish has been demonstrated in other studies (Pennycuick 1971; Kennedy 1984; Esch 1994). Krkošek et al. (2007) used mathematical and empirical data to relate the influence of the copepod *Lepeophtherius salmonis* on farmed salmon, which get repeatedly infected. Not only does the parasite cause mortality of these farmed salmon, it also causes over 80 % mortality of wild juvenile salmon (*Oncorhynchus gorbuscha*) in the waters adjacent to the farms. Those authors postulated that local extinction of wild pink salmon could occur if parasite-outbreaks abate the ecosystem's ability to support the wild population.

14.4.1.1 Bacterial

During certain periods throughout most years in most areas of the GoM, lesions occur commonly on specific fishes. These sores or ulcers usually occur in fins, tail, mouth, or near

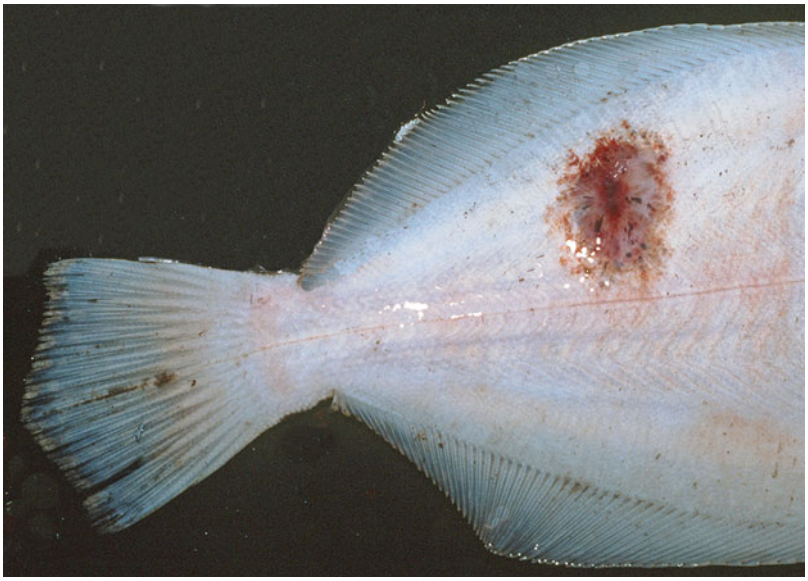

Figure 14.9. Southern flounder, *Paralichthys lethostigma*, exhibiting relatively common bacterial lesion on blind side of specimen from Pascagoula estuary, Mississippi, 1987.

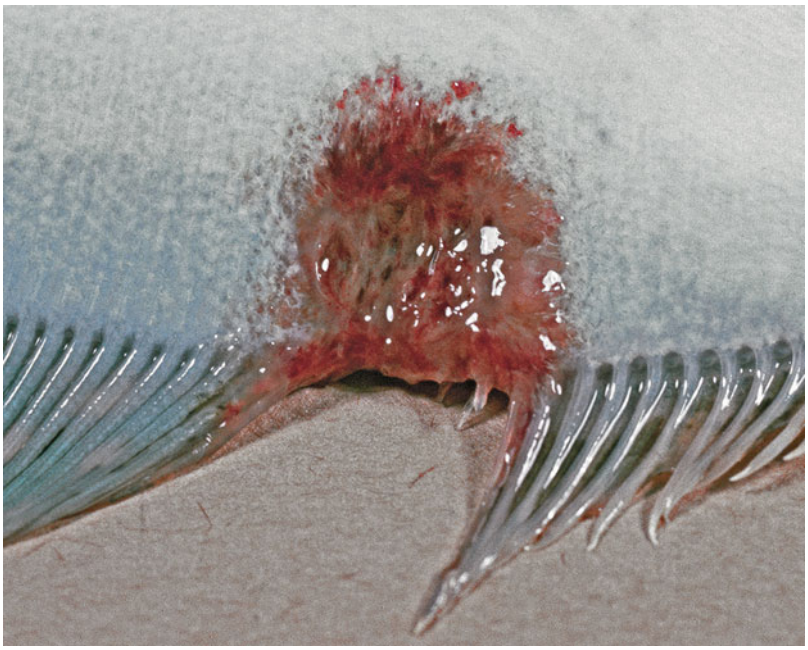

Figure 14.10. Southern flounder, *Paralichthys lethostigma*, exhibiting relatively common bacterial lesion on fin and blind side from specimen in Pascagoula estuary, Mississippi, May 1987.

anus, and regulatory agencies typically refer to those lesions or to the infected host fish as "red sore," "tail rot," "fin rot," or "mouth rot" (Figures 14.9, 14.10, and 14.11). Diagnoses usually include positive results for *Vibrio, Pseudomonas*, and other bacteria, and, if the salinity is low, counterpart infections involve *Aeromonas, Flexibacter*, and other bacteria. Causes can include

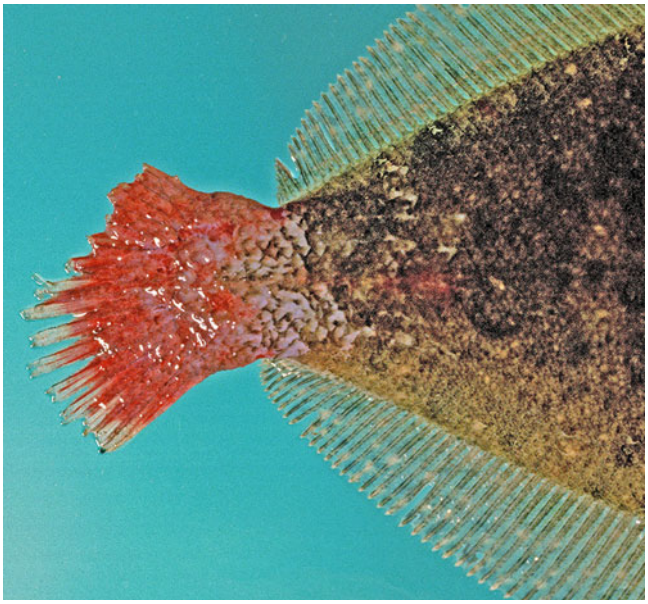

Figure 14.11. Southern flounder, *Paralichthys lethostigma*, exhibiting severe bacterial tail rot of specimen from Pascagoula estuary, Mississippi, in 1997.

injury from predator wounds, trawling or other fishing activities, harsh water quality conditions, internal or external parasites, plasmid or viral infections, co-infection with other bacteria, or even primary bacterial infections. Most often, aggravation by any one of those stressors impairs the fish skin or alimentary tract, enhancing bacterial invasion and growth. To take this process further, sometimes a wound with associated necrotic host tissue supports establishment and growth of barnacles and other fouling agents. Couch and Nimmo (1974) observed over a 10-year period in Escambia Bay, FL, a high prevalence of fin rot syndrome associated with mortalities in Atlantic croaker and spot during warm weather and oxygen depletion. They were able to demonstrate experimentally the induction of fin rot syndrome in 90 % of the exposed spot to a polychlorinated biphenyl (PCB) (3–5 µg/L of Aroclor 1254) with an associated 80 % mortality, but no attempt was made to isolate the bacteria. In most cases in the northern Gulf, the lesions undergo repair in the absence of stressors, and mortalities are not conspicuous.

The most common lesions in marine and estuarine GoM fishes involve *Vibrio* and related bacteria and some form of stress playing a role in the disease process. *Vibrio alginolyticus, Vibrio anguillarum, Vibrio carchariae, Vibrio cholerae, Vibrio damselae, Vibrio ordalii, Vibrio parahaemolyticus,* and *Vibrio vulnificus* all have been reported to cause disease in marine fish and require salt to grow (Colwell and Grimes 1984) (Figures 14.9, 14.10, 14.11, 14.12, and 14.13). As methods for identification become more sophisticated, strains and new species are recognized. For an example in wild fish, a bacterial mortality event was restricted to menhaden and striped mullet in the Galveston Bay (TX) area in November 1968. The cause was attributed to *Photobacterium damselae piscicida,* formerly known as *Pasteurella piscicida* (Lewis et al. 1970; Panek 2005). This bacterium occurs ubiquitously in the gut of marine fishes. Thune et al. (2003) reported that in Louisiana from 1990 to 1995, heavy mortalities (32 cases) were reported in coastal hybrid striped–bass farms, with four farms closing as a direct result of *Photobacterium damselae piscicida*. This pathogen has also become a pathogen of significance in cultured cobia (*Rachycentron canadum*), with 80 % mortality at some sites

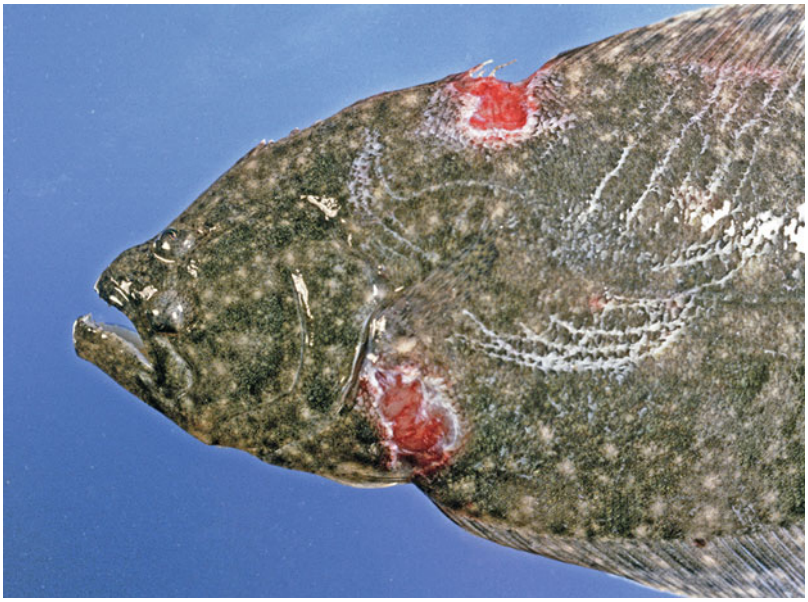

Figure 14.12. Southern flounder exhibiting ulcers and rake marks thought to be produced by an Atlantic bottlenose dolphin "playing with fish" or after escape from an unsuccessful attempt to capture the prey in Mississippi Sound in 1997.

Figure 14.13. Atlantic croaker, *Micropogonias undulatus*, exhibiting relatively common bacterial fin lesions and tail rot from shrimp trawling grounds in Mississippi Sound during October 1996.

(McLean et al. 2008). In fact, isolates from cage-cultured cobia have included *V. alginolyticus, V. parahaemolyticus, V. vulnificus,* and *V. harveyi* (McLean et al. 2008). All stages of cobia can succumb to vibriosis, and this disease can account for 45 % mortality in cage-stocked juveniles.

Vibrios not associated with overt disease in marine organisms are ubiquitous in the northern Gulf of Mexico, where *Vibrio vulnificus* has been isolated from the intestines of sheepshead (*Archosargus probatocephalus*), red snapper (*Lutjanus campechanus*), little tuna

Figure 14.14. Red snapper, *Lutjanus campechanus*, exhibiting bacterial exophthalmos.

(*Euthynnus alletteratus*), Atlantic croaker, Atlantic spadefish (*Chaetodipterus faber*), Atlantic stingray (*Dasyatis sabina*), black drum, crevalle jack (*Caranx hippos*), gafftopsail catfish (*Bagre marinus*), Gulf menhaden (*Brevoortia patronus*), Gulf toadfish (*Opsanus beta*), pigfish (*Orthopristis chrysoptera*), pinfish (*Lagodon rhombodies*), scaled sardine (*Harengula jaguana*), hardhead sea catfish (*Ariopsis felis*), southern kingfish (*Menticirrhus littoralis*), Spanish mackerel (*Scomberomerus maculatus*), and white mullet (*Mugil curema*) (DePaola et al. 1994). DePaola et al. (1994) noted that the prevalence of *V. vulnificus* collected offshore in the Gulf (32–35 ppt) was 11.8 % compared with 13 % in Galveston open Gulf beaches (18.9 ppt) and 68 % in the Galveston Bay estuary (11.3 ppt). Tao et al. (2012) found that a statistically significant ($p < 0.0001$) inverse correlation between *V. vulnificus*-positive fish and salinity existed as did a positive correlation ($p < 0.03$) between water temperature and *V. vulnificus*-positive fish in Gulf locations of Ocean Springs, Mississippi, Gulf Shores and Dauphin Island, Alabama. In addition to the fish listed above by DePaola et al. (1994), Tao et al. (2012) also reported ladyfish (*Elops saurus*), striped mullet, silver perch (*Bairdiella chrysoura*), sand weakfish (*Cynoscion arenarius*), spotted weakfish (*Cynoscion nebulosus*), and red drum (*Sciaenops ocellatus*) to have *V. vulnificus* on the body surface. Buck (1990) isolated *V. alginolyticus, V. damselae*, and *V. parahaemolyticus* from fish from the GoM and adjacent Sarasota Bay, Florida. These isolates came from the gills, intestinal tract, mouth, surface skin, spines, and teeth.

Exophthalmia, a condition known as "bugeye," commonly affects fishes in the GoM. The most common cause is bacterial as shown in the red snapper maintained in culture (Figure 14.14). Figure 14.15 shows a case in the sheepshead minnow that also was probably bacterial in nature. The condition, however, can result from a variety of causes. When a trematode metacercaria infects the eye, especially in a semi-enclosed locality, a large percentage of the fish intermediate host population can be infected. Several species of diplostomoids produce this effect, and some

Figure 14.15. Sheepshead minnow, *Cyprinodon variegatus*, exhibiting exophthalmos, or bugeye, from Mississippi bayou in 1996, probably caused by bacterial infection, but similar condition occurs in eyes of several different fishes as a result of bacterial, viral, diplostomoid trematode metacercariae, or cestode metacestode as well as in cultured fish as nutritional deficiency or gas-bubble disease.

species can number 30 or more large encysted individuals in the vitreous humor or several hundred other small ones in the lens of a single eye. Presumably, heavily infected fish in the Gulf, the same as demonstrated elsewhere or in experimental studies, are vulnerable to predation by the appropriate bird or mammal final host. Metacestodes occur less commonly, but we have seen them in the eyes of Florida pompano (*Trachinotus carolinus*) and puffers. Lymphocystis, a viral disease reported later, can usually be recognized in living fish. Nutritional deficiency and gas-bubble disease usually affect fish in culture under poor husbandry conditions.

What is termed "red sore" disease in Mississippi and elsewhere in the northern Gulf is an ulcerative condition common in euryhaline fish and can occur at epizootic levels greater than 50 % prevalence, based on fishermen's comments to us, in sheepshead (*Archosargus probatocephalus*) (Figure 14.16), black drum (*Pogonias cromis*), and centrarchids. Most cases in low salinity water are associated with *Aeromonas hydrophila*; but some lesions, especially those from fish in 15–20 ppt, had *Pseudomonas* spp. and *Vibrio* spp. Overstreet (1988) considered most of the cases he investigated as resulting from contamination, but they were associated with some secondary infections resulting from natural factors and mechanical damage from fishing or other activities. He updated prior reports from Mississippi Sound (Overstreet and Howse 1977) as approximating red sore lesions occurring in 10 % of spot and southern flounder in summer months and a lower percentage during the rest of the year. The Atlantic croaker and southern kingfish (*Menticirrhus littoralis*) also exhibit lesions, often associated with fishing activities. Overstreet also cited literature reporting 35–40 % of striped mullet in Punta Gorda, Florida, in late summer and several species in West Florida with *Vibrio damselae*. *Aeromonas hydrophila* and *Vibrio anguillarum* are also known to cause a bacterial hemorrhagic septicemia

Figure 14.16. Sheepshead, *Archosargus probatocephalus*, showing ossified lesion with secondary bacterial infection; this condition can be common in low salinity conditions; Back Bay of Biloxi, Mississippi, April 1977.

and fin rot in the Florida pompano (*Trachinotus carolinus*) and striped bass (*Morone saxatilis*) in brackish water of Alabama (Hawke 1976).

The influence of environmental stressors such as low salinities or pollution with high organic content may initiate and exacerbate this disease (Overstreet 1978, 1988). *Aeromonas hydrophila* and the colonial peritrich ciliate *Heteropolaria colisarum* interact to produce ulcerating lesions in centrarchid fishes occurring in freshwater and low salinity habitats (Figure 14.5). The bacterium associates with the ciliate, and, when the organic load in the habitat increases in amount, the ciliate increases in number. Different proteolytic enzymes produced by the motile bacterium (Barrett et al. 2012) cause erosion of the epithelium, lysis of the skeletal musculature, hemolysis, and hemorrhagic septicemia (Overstreet and Howse 1977; Cipriano et al. 1984). Ultrastructural study of the ciliate by Hazen et al. (1978) assumed that since the point of the stalk attachment as shown by scanning electron microscopical images to the fish surface was not the site of pathologic changes, the relationship with the ciliate was benign. In our (Overstreet and Howse 1977) ultrastructural transmission electron microscopical images, the point of attachment was a dense granular layer overlying the spreading fibrillar attachment. Sinuous spiral fibers running longitudinally down the stalk attach directly to the collagenous lamellae of the fish scale. No cellular membrane separated host tissue from the attachments of fibrillar of granular layers. We suggested the ciliate could invade the collagenous layer. Whether this invasion could occur without the interaction of bacterial enzymes is unknown, but bacteria alone, with accompanying ciliates, could readily cause red sore lesions in Hazen's material as well as ours. In fish with severe septicemia, the liver and kidneys served as foci for toxic products produced by the bacteria. The structural integrity of both organs was destroyed, leaving minor pathologic changes in the spleen and heart (Huizinga et al. 1979).

Interesting questions concerning aspects of these red sores involve the seriousness, longevity, and cause of red sore lesions. We think the infections in sheepshead from Mississippi (Figure 14.16) provide examples of lesions associated with low salinity. During occasional periods of low salinity, sheepshead with lesions occurred and were caught by hook and line

commonly from under the U.S. 90 Highway Biloxi-Ocean Springs Bridge. Because of the lesions, some calcified and some with attached barnacles, fishermen were concerned about keeping and eating them and threw them back into the water. We examined several critically, and some individuals with special marks (tattoos) were captured over and over. Most other species migrated a short distance to higher salinity waters. The lesions in some fish covered more than a third of the body surface and exhibited large areas of skeletal muscles along the flank. Because some fishermen recognized the individuals, they reported to us that the fish remained alive with lesions as long as the salinity concentration remained low, sometimes a few months. The presence of low salinity acorn barnacle supports this longevity in low salinity. Once the salinity concentration increased, fish did not exhibit lesions. We examined some and could detect scale and epithelial regeneration, suggesting recovery from the infections; regenerating tissues remained detectable for a couple of months.

We often captured, using 5-min long trawls, specimens of the Atlantic croaker that had been trawled and tossed overboard, often multiple times, by shrimp fishermen. Many of these had red sores, fin-erosion, abrasion, tail-rot, and other lesions. Presumably, some became prey of seabirds, fishes, and bottlenose dolphin, but after shrimp season we could see a time series of regenerating lesions in many specimens, suggesting recovery in a significant proportion of released fish. Few scientific studies have been conducted to understand the multiple causes of bacterial lesions.

Fin erosion can result from anthropogenic factors. These lesions can be chemical contamination, fishing techniques as indicated above, or a variety of other activities. For example, Sherwood and Mearns (1977) provided an example using strong observational and experimental evidence linking chlorinated hydrocarbon pollutants (e.g., DDT) with fin-erosion in a southern California flatfish (Dover sole, *Microstomus pacificus*) exposed to discharged municipal wastewater. In addition to lesions, the ratio of liver to body weight of laboratory-exposed fish was higher than in controls and similar to that recorded from fish from a heavily contaminated study-location.

The striped mullet, *Mugil cephalus*, in Figure 14.17 should not be confused with a fish exposed to a toxicant. It became trapped in a nearly fresh water pond at Ingalls West Bank Overpass near Pascagoula, Mississippi, and could not escape unlike the sheephead above that could have left the low salinity habitat.

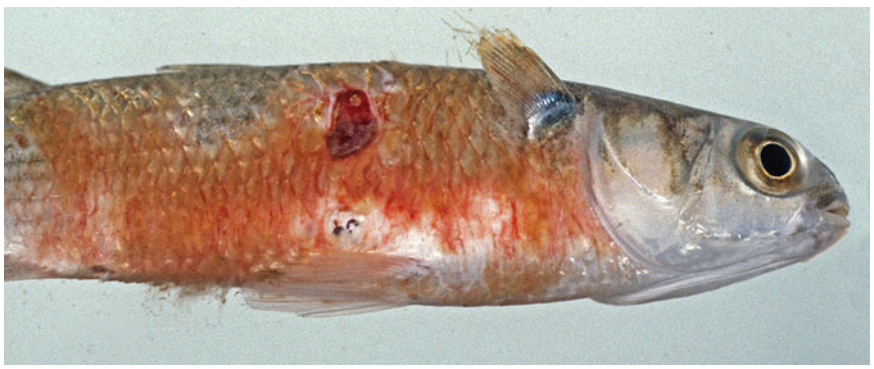

Figure 14.17. Striped mullet, *Mugil cephalus*, exhibiting bacterial (*Aeromonas hydrophila*) ulcerated lesions, abraded fins, hemorrhaging, and concurrent *Saprolegnia*-like fungal infection. A series of similarly affected mullet became trapped in nearly fresh water pond at Ingalls West Bank Overpass, Pascagoula, Mississippi, March 1992.

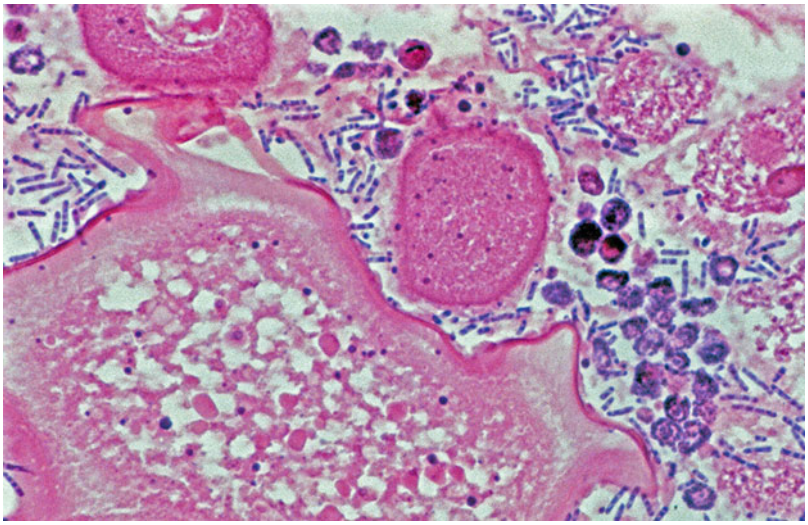

Figure 14.18. Red drum, *Sciaenops ocellatus*, with histological section of ovary showing *Streptococcus* infection in September 1991.

This was one of many such fish exhibiting a bacterial (*Aeromonas hydrophila* and *Shewanella putrefaciens* [numerous colonies on live fish, not a contaminant]) ulcerated lesion, abraded fins, hemorrhaging, and concurrent *Saprolegnia*-like fungal infection. No septicemia occurred, but the spleen exhibited a large number of macrophage aggregates, indicating the tissue damage (Overstreet 1997).

Large-scale mortality events involving bacteria as a primary causative or suspected agent have occurred, even though rarely, in the GoM. One such documented event involving *Streptococcus* sp. non-hemolytic Group B, type I_b occurred during August–September 1972 along the Alabama and northwestern Florida coastlines (Plumb et al. 1974; Wilkinson et al. 1973). Tens of thousands of fish died, mostly Gulf menhaden (*Brevoortia patronus*), but also hardhead catfish, striped mullet, Atlantic croaker, pinfish (*Lagodon rhomboides*), spot, stingray (*Dasyatis* sp.), and sand seatrout (*Cynoscion nothus*); no abnormal environmental condition was apparent during the time of the event (e.g., Figure 14.18). Experimental studies conducted with isolates obtained from two separate sites in Alabama, Soldier Creek and Bon Secour, caused 70 and 90 % mortality, respectively (Plumb et al. 1974). An isolate obtained from a "fish" from Mobile Bay, AL in September 1972 was also identified as *Streptococcus* sp. non-hemolytic Group B, type I_b (Wilkinson et al. 1973). Cook and Lofton (1975) conducted pathogenicity studies with an isolate obtained from the kidney of the menhaden from Soldier's Creek referred to as *Streptococcus* 922. Using intraperitoneal injection of the isolate, they found a mortality rate of 40–90 % in Atlantic croaker, 33–40 % in Gulf menhaden, 100 % in striped mullet, and 57–100 % in spot. Rasheed and Plumb (1984) also performed an experimental study with an isolate that was serologically identical to those above obtained from the Gulf killifish (*Fundulus grandis*) and concluded that in the killifish, disease occurred only when the portal of entry was an injured area of the body. Panek (2005) attributed a massive 1999 fish kill in the northeast Caribbean to the β-hemolytic *Streptococcus iniae*.

Fish kills due to meningitis caused by the gram-positive anaerobic bacteria *Eubacterium* sp. have also been observed in the coastal areas of the northern Gulf. This anaerobic bacterium has been cultured from striped mullet and red drum involved in an extensive mortality event near Port Aransas, Galveston, and Orange, Texas, in 1973 (Henley and Lewis 1976). Fish kills in

the Biscayne Bay, Florida, region have occurred repeatedly, and Udey et al. (1976) isolated *Eubacterium* sp. from the brain tissue of all the dead and moribund fish sampled. The fish involved in the large Biscayne Bay kills included striped mullet, snook (*Centropomus undecimalis*), Gulf flounder (*Paralicthys albigutta*), and striped mojarra (*Diapterus plumieri*). Udey et al. (1976) also reported that experimental studies conducted on an isolate did not produce a toxin, nor was the agent pathogenic for mammals. It did produce mortality in channel catfish after 14 days when injected intraperitoneally. They found that this bacterium was present in every mullet tested, and they also isolated it from six other species of fishes from the Biscayne and Florida Bay areas. This bacterium can be present in a species without any display of disease. Udey et al. (1977) classified these bacteria as *Eubacterium tarantellas*, and they noted that marine fishes not entering the bays did not have *E. tarantellas*. The organism would not grow at salt concentrations above 2 %. Lewis and Udey (1978) indicated that several species of estuarine and marine fishes act as reservoir hosts. *Eubacterium tarantellas* has also been identified from the ovaries of black drum and red drum (Nieland and Wilson 1995) (also compare Figure 14.18).

The same or related bacteria from infections in fish also infect other animals in the same waters as the fishes. For examples, Cook and Lofton (1973) found *Beneckea* sp. type I to be an opportunistic infection when the crab shell has been damaged causing shell disease. The chitinoclastic bacteria in the genera *Beneckea, Pseudomonas*, and *Vibrio* have been isolated from the lesions of blue crab and penaeid shrimp. Shields and Overstreet (2007) listed bacteria isolated from the shell and hemolymph of the blue crab and indicated there was no relationship between black spot lesions and bacteria in the hemolymph. Lightner and Lewis (1975) observed a septicemic bacteria disease with *Vibrio alginolyticus* in wild brown, white, and pink shrimps obtained from commercial bait dealers in Galveston, Texas, in 1972 and 1973. These moralities ranged from "a few a day" to nearly 100 %. One bait camp had both *Vibrio alginolyticus* and *V. anguillarum* isolated from white shrimp that underwent 50 % mortality. Witham (1973) described a *Bacteroides* sp. infection in 140 loggerhead turtle (*Caretta caretta*) hatchlings (1–3 months old) that appeared in September of 1970 at a mariculture tank at Hutchinson Island,

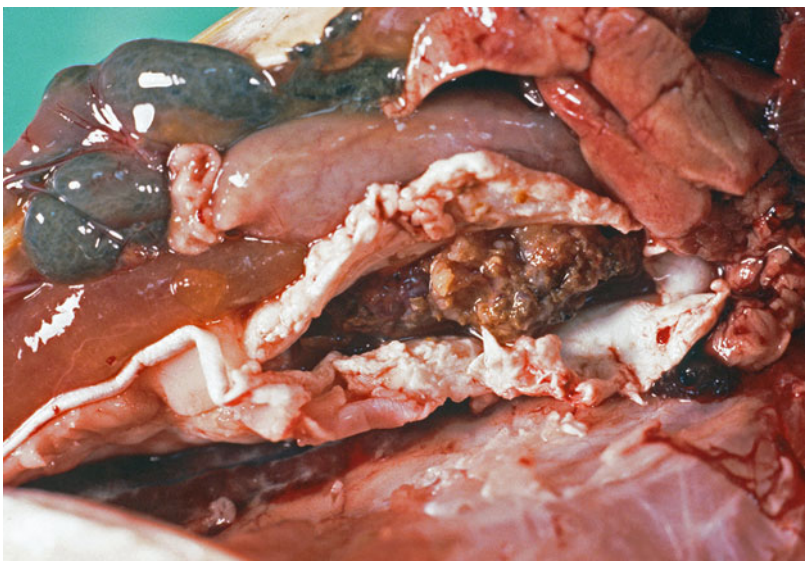

Figure 14.19. Atlantic croaker, *Micropogonias undulatus*, from Pascagoula estuary exhibiting systemic granuloma in September 1980 primarily involving swim bladder and probably caused by nocardiosis or mycobacteriosis; condition can result in fish cultured with a deficiency in Vitamin B or C.

Florida. The disease presented with necrotic, spreading skin lesions causing death within 3–7 days over a period of 3 months with a cumulative 98 % mortality. *Bacteroides* sp. was considered the primary pathogen, although *Pseudomonas aeruginosa* and *Staphylococcus epidermis* were also present.

Many other bacteria infect fishes, some hard to detect or determine without culturing blood or tissues. On the other hand, some infections produce obvious disease. Figure 14.19 exhibits systemic granuloma in a wild fish. Similar cases caused by bacteria and dietary deficiencies occur in cultured fishes.

14.4.1.2 Viral

The disease lymphocystis constitutes an interesting virus infection for a variety of reasons. It does not occur in an abundance of individuals, but various strains do infect a variety of host fishes in the GoM (Figures 14.20, 14.21, 14.22, 14.23, 14.24, and 14.25). It typically infects connective tissue cells in the skin of the body and fins, and each infected cell hypertrophies

Figure 14.20. Atlantic croaker, *Micropogonias undulatus*, with lymphocystis mass in and above eye in fish caught in June 1992 off Marsh Point, Ocean Springs, Mississippi.

Figure 14.21. Atlantic croaker with extensive lymphocystis infection, Mississippi, 1996.

Figure 14.22. Atlantic croaker showing moderate infection of lymphocystis, Mississippi, April 1985.

Figure 14.23. Spot, *Leiostomus xanthurus*, with heavy infection of different strain of lymphocystis, Mississippi, October 1978 (similar infection in October 1987).

Figure 14.24. Atlantic spadefish, *Chaetodipterus faber*, exhibiting still another strain of lymphocystis, Mississippi, November 1979.

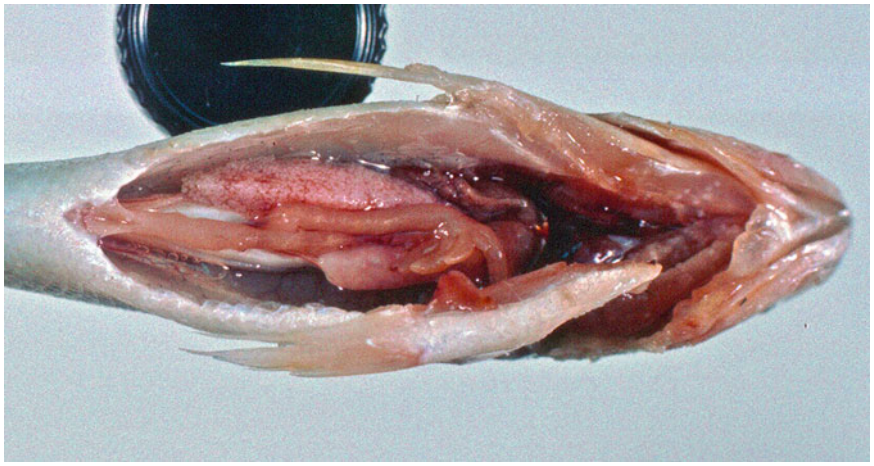

Figure 14.25. Silver perch, *Bairdiella chrysoura*, with internal infection of lymphocystis obvious in spleen; spleen located parallel to intestine, large and pinkish because of hypertrophied lymphocystis-infected cells rather than being relatively small and dark brownish.

and can be observed as a nodule with careful examination; accumulation of these cells provides a raspberry-like tumorous growth, a cluster which is readily observable and defined as a "pseudo" tumor by Anders (1989) that is unnecessary here because we consider non-cancerous lesions as tumors. Because this viral disease does not cause mass mortalities, an epizootic can be readily followed. Lymphocystis was first reported from the GoM in a few individuals by Christmas and Howse (1970) in the Atlantic croaker and sand seatrout, *Cynoscion arenarius*, from relatively polluted locations during winter months. Individual cells can be recognized because they become greatly enlarged and possess an alcianophilic hyaline capsule that stains positive for PAS (periodic acid-Schiff). Icosahedral-shaped viral particles remain confined to the cytoplasm of the host cell, which for some strains can increase in size up to 2 mm (0.1 in) in diameter with a volume about one million-fold that of the normal host fibroblast (Anders 1989). The double-stranded DNA virions have been placed in the genus *Cystivirus* (Iridoviridae). Infections in Mississippi are most common in the Atlantic croaker and silver perch, *Bairdiella chrysoura*, and we will discuss later the unique details of this latter infection, which can occur in internal organs.

Overstreet and Howse (1977) and Overstreet (1988) reported on the history of epizootics in the Mississippi area that reached the peak of as much as 50 % of the croaker population in the mid-1970s. Between 1966 and 1969, a total of 12 of 32,688 croaker and sand seatrout exhibited infections. During the following 18 months, Overstreet and Howse (1977) observed 15 of 2,500 croaker infected. By the mid-1970s, croaker examined by them and reported by commercial shrimp fishermen had increased to hundreds of cases, with as many as half of the croaker in a trawled sample observed to be infected on several occasions. The shrimpers who had been catching croaker in Mississippi coastal waters for years recalled seeing infected individuals only during that period. Specific cases in February 1971 and summer of 1973 were mentioned (Edwards and Overstreet 1976). In June 1984 when thousands of various-sized boats trawled for shrimp, each discarded several thousand young-of-the-year croaker from their by-catch. Many declared to R. Overstreet that about 20 % of their juvenile croaker catches exhibited lymphocystis, especially in Biloxi Bay, and he verified several of those observations. Earlier on June 28, 1976, 11 out of 80 croaker in Mississippi Sound immediately north of Dauphin Island, Alabama, exhibited infections restricted to less than 2 % of their body surface. In contrast, 2 of

174 croaker samples from the higher salinity Gulf water south of the island exhibited 60 and 80 % of their surface area covered, but they could have previously acquired the infection in Mississippi Sound. What is apparently the same strain ranges from at least Texas to Sapelo Island, Georgia (Smith 1970).

We have conducted experimental studies with a few of the strains occurring in Mississippi waters. Studies by us and by Cook (1973) have shown that the enlarged cells became apparent within 5–7 days after inoculation when maintained at 25 °C, but within 9–11 days at 20 °C, with salinities at least between 10 and 35 ppt not having an effect. The disease cell clusters typically sloughed off between 20 and 30 days after inoculation. Infections could not be produced in about 2 % of fish tested from the wild. Cook (1973) reported the strain from croaker could be used to infect croaker, sand seatrout, and black drum, *Pogonias cromis*, but not spot, spotted seatrout, or bluegill. We also showed that the strain from the silver perch would not infect Atlantic croaker or spot, and the strain from spot would not infect Atlantic croaker.

The atypical strain from the silver perch produced internal infections in the heart, behind the eye, in the kidney, mesentery, spleen (Figure 14.25), liver, and ovary as well as external in the skin, fin, and gills. The presence of the cymothoid isopod *Lyroneca ovalis* causing lesions to the gill or the presence of damaged gills suggesting a prior isopod infestation indicated that such lesions might allow the virus to enter the bloodstream and infect specific cells in internal organs (Howse et al. 1977). Ultrastructural investigation of infections in the heart of the silver perch demonstrated infections similar to those in the skin in the epicardium, trabecular spaces, and sub-endocardium, but not in the adjacent myocardial cells. Wharton et al. (1977) established a fibroblast-like cell line from the swim bladder of the silver perch in which growth of the lymphocystis virus was supported but not of other viruses from fishes and mammals. They did not observe formation of the hyaline capsule in vitro, although frozen virus from their cell lines did produce tumor cells in vivo that contained this structure. They suggested that the L-15 growth medium that they used did not have sufficient muco-polysaccharide to produce the capsule. About 10,000 early stage, 200 mg (0.007 oz) juvenile red drum were imported from Texas to Israel, where they were to be reared. When the fish reached 20 g (0.7 oz), some cutaneous lymphocystis lesions appeared, and within 2 months, several hundred displayed severe infections. Some of these fish contained internal lesions, most prominently in the spleen. The origin of the virus was not indicated (Colorni and Diamant 1995).

Internal infections with the lymphocystis-virus seemed to have resulted from experimental infections until Dukes and Lawler (1975) reported on naturally occurring ocular lesions in the silver perch from Mississippi and the sand seatrout from Texas. The cells occurred in or behind the eye as well as on the cornea or adjacent skin surfaces. Figure 14.20 shows a previously unreported case of the Atlantic croaker with a lymphocystis mass occurring in and above the eye.

Most lymphocystis-infected fishes in the Gulf are sciaenids, and several strains exist, as discussed above; but members in other families have also been observed from the Gulf with lymphocystis in low prevalence. The common snook, *Centropomus undecimalis*, from Campeche, Mexico, has an infection with the hyaline capsule thicker than those reported for the species from sciaenids (Howse 1972). An infection in a 27-cm (11 in) standard length specimen of the Spanish mackerel from Venice, Florida, collected on July 11, 1986, was described by Overstreet (1988). The Atlantic spadefish, *Chaetodipterus faber*, from Mississippi in November 1979 exhibits (Figure 14.24) another new record of what is probably another strain of lymphocystis. The bluegill in freshwater habitats as well as low salinity estuaries of Mississippi occasionally exhibits what appears to be a different strain and probably what was reported by Weissenberg (1945). Our unpublished experimental infections showed that it did not become established in local sciaenid species.

Because of the spotty distribution and other factors, the expression of lymphocystis appears to have a relationship with specific toxicants or conditions. Christmas and Howse (1970) found the few affected fish occurring in industrially contaminated areas of Mississippi. Overstreet (1988) hypothesized that one or few specific toxicants, rather than general stress, may have enhanced infections by lowering host resistance.

Other circumstantial evidence occurs for induction of infections outside the Gulf of Mexico. Perkins et al. (1972) suggested that PCBs may have been responsible for lymphocystis in adults of two species of flatfishes, common hosts for lymphocystis in areas more temperate than the Gulf of Mexico, in the Irish Sea at the same time that young individuals in an uncontaminated area did not develop the disease; however, Shelton and Wilson (1973) considered hydrographic conditions such as low salinity to be a better explanation. Wolthaus (1984) reported infections in the dab, *Limanda limanda*, a flatfish in the southern North Sea, to be associated with acid iron waste from titanium dioxide production, regardless of season, but others (Möller 1985) questioned the validity of that cause. Mellergaard and Nielsen (1995) also studied the dab from 1984 to 1993. Because a severe oxygen depletion occurred in the late summer of 1986 and 1988, they were able to observe peak prevalences of lymphocystis and epidermal papilloma, another viral disease, of 14.7 and 3.3 % in 1989, respectively. They suggested that the stress caused by the oxygen depletion triggered outbreaks of both viral diseases. We know of no good experimental work relating lymphocystis infections to specific toxicants or stresses.

A massive fish kill assumed to be caused by "hardhead catfish virus" involved millions of hardhead catfish (*Ariopsis felis*), occurred in 1996, and spread from Texas to southwest Florida as followed by the Gulf of Mexico Aquatic Mortality Response Network (GMNET) sponsored by the USEPA and Gulf state agencies. Most beaches, bays, and river mouths contained thousands of dead and dying hardhead catfish and an occasional related gafftopsail catfish

Figure 14.26. Hardhead catfish, *Ariopsis felis*, from Back Bay of Biloxi, Mississippi, with hemorrhaging lesion on fin, viral die-off of May 28, 1996.

(*Bagre marinus*). For example in Mississippi, we observed or collected and examined many specimens inshore from Mississippi Sound, Back Bay of Biloxi, Davis Bayou, Biloxi Channel, and the Pascagoula River as well as specimens floating offshore from Horn and Ship islands, starting with a major kill with tons of dead and dying fish on May 28–31, 1996 and followed by collections of moribund fish from smaller kills at least on June 24, July 17, and August 6, 1996, in water with salinity ranging from 7 to 32 ppt. Initially, fish measured 25–35 cm (10–14 in) in total length, but later, some smaller fish also died. What appeared to be a recurrence of the kill occurred on November 3, 1998, with about 1,000 fingerlings dying in the mouth of the Pascagoula River among many already dead catfish. Most fish grossly exhibited hemorrhaging lesions in the gills and pectoral fins (Figure 14.26), and some had lesions of the mouth, lip, pelvic fins, and anus. Sections showed extensive hyperplasia in the gills, and the adjacent, non-hyperplastic, pale-appearing areas demonstrated an abundance of mobile peritrich trichodinid ciliates. Sections of visceral organs demonstrated an abundance of melanin-macrophage centers and appeared abnormal. A light red area in the liver in a few fish was shown to be infected with *Vibrio fluvialis*, an infection that did not occur in the corresponding kidneys, spleen, or systemically in the blood. This bacterium identified for us by Dawn Rebarchik at GCRL is known to be pathogenic to humans and crustaceans (Eyisi et al. 2013).

From moribund specimens from Biloxi Bay and Mississippi Sound, we (R. Overstreet and Eugene Foor) collected tissues from anterior and posterior kidney, spleen, liver, and brain and prepared them for electron microscopic observation. Intranuclear paracrystalline arrays of viral particles occurred abundantly in all preparations. The center-to-center spacing of the individual particles measured 35–50 nanometers (nm) ($1.4–2 \times 10^{-6}$ in). In all tissues, the particles appeared to be a DNA icosahedral virus, and it showed little selectivity in the cell type parasitized, indicating the virus had a wide host cell range. Whether or not the virus was the lethal agent, acted synergistically with other causative agents, or otherwise became expressed in dying cells only remains to be determined. However, based on the host specificity, high density of virions in dying cells, and the fact that Jan Landsberg also found the virus in moribund catfish in Florida, the virus appears to be the primary causative agent. As indicated above, an infection, not necessarily lethal, typically transforms into a disease when interacting with a stress. Since the disease is highly host-specific, the stress probably is one specifically associated with the catfish, such as one dealing with reproduction. Jan Landsberg and R. Overstreet planned to produce an extensive joint report on the mortality and the agent. We had saved considerable material at −70 °C (−94 °F) for later analyses and experiments, but all thawed during Hurricane Katrina, and we consequently had to destroy it.

We conducted a gillnet survey prior to the mortalities, and the hardhead catfish was the most abundantly captured fish. Using other methods, we would probably have found that the bay anchovy and Gulf menhaden were just as abundant, but, regardless, the catfish constituted much of the local biomass. The catfish population decreased considerably after the period of mortalities, and specimens were rare for the next few years. Additional mortalities of thousands of catfish occurred later in Mobile Bay, Alabama, and Mississippi Sound in May 2009, and these fish probably also had the viral infection. Perhaps these catfish represented specimens without an acquired immune response. Thousands of hardhead catfish also washed up dead in a lagoon in Brevard County in the East Coast of Florida in September 2005, but the cause of those mortalities is apparently unknown.

What may be the same disease as we encountered in the northern portion of the Gulf of Mexico produced 50,000 dead Mayan sea catfish, *Arius assimilus*, in Chetumal Bay in southern

Mexico from June to mid-October 1996. The catfish were also large, of 10–35 cm (4–14 in) total length (Suárez-Morales et al. 1998). The purpose of the report was to report a species of *Argulus*, but the cause of the catfish mass mortality was unknown. Additionally, about 50 tonnes (110,000 lb) of dead marine ariid catfish also related to the hardhead catfish (large individuals of *Netuma barba, Cathorops spixii, Genidens genidens*, and *Sciadeichthys luniscutis*), spreading along 1,800 km (11,184 mi) of beaches, estuaries, and lagoons of Uruguay and southern Brazil in 1994 (Costa 1994). A series of episodes occurred over 16 months during 1994 and 1995. Some of the dead fish exhibited hemorrhaging on the ventral surface and necrosis in the liver and kidney. Virus-like particles in the kidney measuring 32–42 nm in diameter were suggested to be a herpesvirus and associated with spawning stress. Other similar mortalities occurred along the coast of Sierra Leone, Western Africa, in 1980–1981 and more intensely in 1990–1993 (Ndomahina 1994). Another mass mortality of the marine catfish *Arias maculatus* (listed as the junior synonym *Tachysurus maculatus*) was reported from a 120 ha (0.5 mi^2) area near Therespuram, Tuticorin, India, in 1–2 weeks of January 1980 (Natarajan et al. 1982). Young individuals became entrapped when the water retreated after the monsoon. Whether a virus was present was not indicated, but the catfish was the only species mentioned; the dead and dying fish were stressed from a combination of high salinity, low dissolved oxygen concentration, and the presence of hydrogen sulfide.

As questioned in discussing the Gulf mortalities, whether the virus directly killed the catfish or was induced by some stress or other condition has not been established for the catfish infections from any locality, but the virus definitely caused necrosis of the infected cells. Few such massive marine fish kills have been associated with a host-specific virus, and the catfish virus and a herpesvirus infecting clupeids are great examples. Jones et al. (1997) reviewed the Australasian pilchard mortalities of 1995, which started in South Australia and spread to Geraldton, Western Australia; Noosa, Queensland; and New Zealand. A rapid spread of about 25–30 km/day (15–18 mi/day) was suggested to be caused by seabirds or other animals eating dead or dying fish and then defecating. This pandemic occurred in the Australasian pilchard, *Sardinops sagax*, and individuals died within a few minutes after clinical signs of respiratory distress occurred. Acute to subacute inflammation of the gills followed by epithelial hypertrophy and hyperplasia, and the herpesvirus in the gills was not observed in unaffected pilchards, and no correlation existed with oceanographic conditions or the presence of plankton. Another review of the same and the later 1998/1999 pilchard epidemic suggests that the origin may have resulted from importing large quantities of the pilchard into Australia to feed cage-cultured tuna (*Thunnus maccoyii*) (Gaughan 2002). Other large-scale mortalities of clupeids have also occurred such as 1,000 tonnes (2.2 million lb) of Pacific herring (*Clupea harengus pallasi*) in British Columbia in 1949; Meyers et al. (1986, 1994) described viral hemorrhagic septicemia virus associated with epizootic hemorrhages of the skin of Pacific herring in Alaska, which may have been the same virus infecting prior epidemics in British Columbia as well as in Australia.

Probably an unrelated disease in the hardhead catfish is represented by X-cell epidermal lesions not involving any visceral organs and described by Diamant et al. (1994). The description was based on a single specimen captured from Lake Pontchartrain, Louisiana. What may be the same disease was found in three of 434 sampled specimens of a related bagrid catfish (*Chrysichthys nigrodigitatus*) in the Cross River Estuary of Nigeria in 1984 and 1985 (Obiekezie et al. 1988). The cases were identified as epidermal papillomas, and transmission electron microscopical studies gave no indication of the virus or other microorganism.

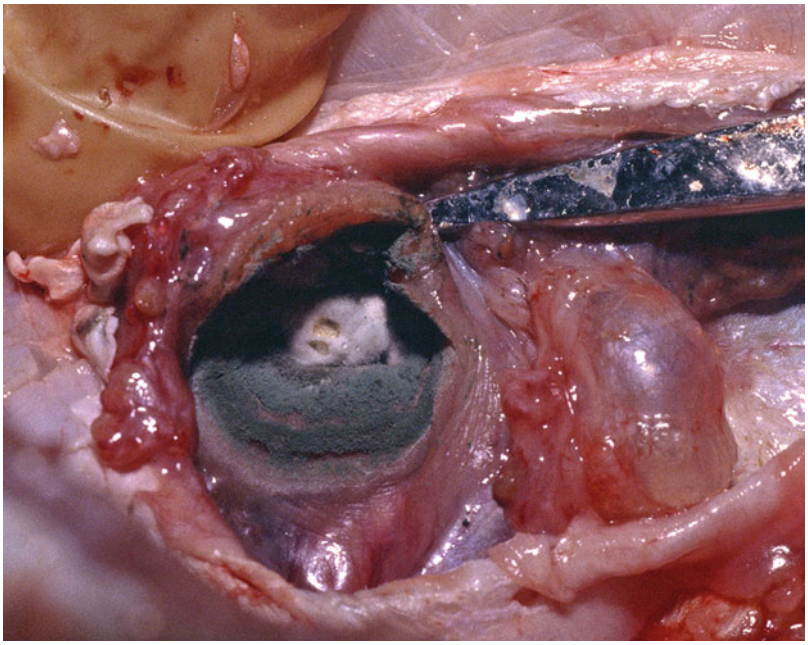

Figure 14.27. Red snapper, *Lutjanus campechanus*, exhibiting dual deuteromycete fungal infections with *Penicillium corylophilum* (*white cottony* appearance of mycelium) and *Cladosporium spaerospermum* in the dissected swim bladder. The infection was presumably acquired when the swim bladder was deflated with hypodermic needle after the fish was rapidly raised up from relatively deep water.

14.4.1.3 Fungal

Fungal infections can be seen more readily in freshwater fishes because the hyphae extend externally. What is probably freshwater *Saprolegnia* occurred in a marine fish trapped in freshwater (Figure 14.17). The related fungus *Aphanomyces invadans* occurs as an ulcerative skin lesion in the Atlantic menhaden (*Brevoortia tyrannus*) (Figure 14.7) along the Atlantic coast; the lesion and associated mortalities had been previously attributed to the dinoflagellate *Pfiesteria piscida* until the critical investigations by Blazer et al. (1999) and Vogelbein et al. (2001). Similar ulcerative lesions have been reported from the striped mullet, silver mullet, black drum, sheepshead, and silver perch in the Gulf from Florida, so Sosa et al. (2007) conducted an experimental study with *Aphanomyces invadans* and other oomycete fungi in the striped mullet and determined that only it caused the lesion. Other fungi such as *Lacazia loboi* causes infections (lobomycosis) in offshore bottlenose dolphin extending into the Gulf (Rotstein et al. 2009).

Little has been reported on harmful fungal infections in fish from Gulf high salinity waters. Figure 14.27 shows a dissected swim bladder of a red snapper (*Lutjanus campechanus*) that had been captured and held in a large tank for many months. It, but not 13 other cohabiting individuals, demonstrated erratic swimming behavior and was therefore necropsied. Both the swim bladder and posterior kidney of that fish exhibited dual deuteromycete fungal infections with *Penicillium corylophilum* and *Cladosporium spaerospermum*. The infection, which was not systemic, was presumably acquired when the inflated swim bladder was deflated with a hypodermic needle after the fish was rapidly raised up from relatively deep water. When cultured fungi were injected into the non-related Gulf killifish, no infection was apparent after 1 month (Blaylock et al. 2001).

14.4.1.4 Protozoan Diseases

Fish dying from a protozoan disease are difficult to obtain because (1) fish seldom die in mass mortalities but rather a few fish at a time, (2) infected fish are usually stressed and consequently become prey for predators before they can be collected, (3) unless morbid fish can be obtained, the agents, unless cyst-formers or those that produce specific gross pathologic alterations, typically deteriorate and become difficult or impossible to identify, and (4) infected or infested fish are actually responding to a toxin or other stress, which additionally allows predilection to a protozoan infection. Helpful general and specific books that treat protozoans and myxosporans of fish (Lom and Dyková 1992; Dyková and Lom 2007) provide a starting place for understanding those groups.

The holophryid *Cryptocaryon irritans* could be an important pathogenic ciliate in the Gulf, but it seldom has been known to cause mortality in wild fish. It has been reported from red drum cultured in ponds in Palacios, Texas (Overstreet 1983b), and caused problems in some marine aquaria. It is the counterpart of the well-known freshwater *Ichthyophthirius multifiliis*, and both have a similar cycle involving feeding trophonts that inhabit the basal layer of the epithelial cells on the skin and gills, a free-living tomont, and an encysted tomont that produces tomonts, which in turn develop into infective theronts that bore through the gelatinous cyst wall and infect a variety of fishes. The histophagus trophonts feed on the epidermis. Dickerson (2006) discusses both ciliates. A few different strains of *C. irritans* have been differentiated. Diggles and Adlard (1997) reported sequence differences among isolates from Moreton Bay and Heron Island, Queensland, Australia; Israel; and the United States. The strain from wild fish in Moreton Bay remains unchanged with that maintained in the laboratory for over 10 years. In Queensland, Australia, when fish are brought into the laboratory aquaria, infections often build up and fish die. The ciliate was considered rare in nature until Diggles and Lester (1996b) showed with critical, sensitive examination for encysted tomonts that 13 of 14 fish species exhibited infections with no seasonality in prevalence or intensity of infection in water temperature between 15 (59 °F) and 27 °C (80 °F). They (Diggles and Lester 1996a), however, showed in experimental temperatures of 20 (68 °F) and 25 °C (77 °F) that trophonts stayed on fish longer and tomonts took longer to excyst, producing larger theronts at 20 °C (68 °F) than at 25 °C (77 °F). The host of origin played a role in tomont incubation period and tomont size. These data suggest that *C. irritans* might play a more important role in the health of Gulf fish, if it was not for the abundance of the equally pathogenic dinoflagellate *Amyloodinium ocellatum* discussed below.

Trichodinid mobiline peritrich ciliates occur on the gills of marine and estuarine fishes as well as on the skin of freshwater fishes in the entire Gulf. When hosts are under stress, they also infect renal tubules. These are disc-shaped or hemispherical ciliates with a cytostome for feeding on bacteria and organic detritus located on the aspect facing away from the host. They usually do not occur in high enough numbers to cause mortality except in aquaculture and habitats with an excess of organic matter, or on stressed fish such as the hardhead catfish infected with the virus mentioned earlier. Infections on juvenile red drum from Mississippi are figured by Overstreet (1983b). They have a simple lifecycle and reproduce when the host is stressed or food is abundant; consequently, their presence has been and can be used as a biological indicator.

The dysteriid ciliate *Brooklynella hostilis* (Figure 14.28) infects several reef fish and the lethargic individuals, with the histophagic ciliate infecting their gills and skin, conspicuously sloughing the infected skin. Consequently, the infection described by Lom and Nigrelli (1970) from a public aquarium in Brooklyn, New York, is sometimes termed “slime-blotch disease” or “clownfish disease” because it often infects clownfishes in marine aquaria. It used to be a

Figure 14.28. *Brooklynella hostilis*, relatively common ciliate in Mississippi waters and elsewhere in the Gulf, Atlantic coast, and Caribbean where it has been associated with fish kills. Staining and photograph by Hongwei Ma (permission to reprint granted by H. Ma to R.M. Overstreet).

problem for aquaculture or public display facilities only. In past years, R. Overstreet could seldom place a yellow-headed jawfish, *Opisthognathus aurifrons*, from the Florida Keys into an aquarium without the disease becoming conspicuous. However, within the last 30 years, infections have been detected on wild dying fish and have been associated with a Caribbean-wide mass mortality event in 1980, as well as repeated mass mortalities in South Florida and the eastern Caribbean (Williams and Bunkley-Williams 2000). But Landsberg (1995) hypothesized a more reasonable hypothesis that infections and mortalities resulted from a synergistic relationship of *B. hostilis*, *Uronema marinum*, amebae, and pathogenic bacteria with biotoxins. For example, a change in abiotic and biotic factors such as Hurricane Andrew or flooding in the Mississippi Delta may have led to changes in the successional colonization or cover of microalgae on the coral reefs. The macroalgae *Caulerpa* spp. temporarily replaced turtlegrass and other sea grasses on which many fish and invertebrates fed, and they produce the toxin caulerpenyne. That toxin as well as indirect consumption of toxic epiphytic dinoflagellates (such as *Gambierdiscus toxicus*, *Prorocentrum* spp., or *Ostreopsis*) by herbivorous fish suppresses their resistance to the protozoan diseases, causing chronic toxicity and disease as well as bioaccumulation, chronic toxicity, and mortality of those predators that fed on the herbivores. *Brooklynella hostilis*, which in many respects resembles its counterpart in fresh water *Chilodonella*, undergoes rapid multiplication by simple binary fission and then weakens and kills the host.

The scuticociliate ciliate *Uronema marinum* occurs as a free-living component of many marine systems, but it can also become a facultative parasite and infect the gills, skin, viscera, and body muscle in Pacific and Atlantic marine fishes, including those in the Gulf (Figure 14.29).

Figure 14.29. *Uronema marinum*, a cosmopolitan ciliate found in Mississippi estuaries and in blue crab hemolymph and capable of causing disease in cultured and wild fishes and invertebrates. Staining and photograph by Hongwei Ma (permission to reprint granted by H. Ma to R.M. Overstreet).

Occasionally, the species will enter a wild, wounded, or otherwise stressed fish and replicate rapidly, but it is more likely to cause problems in pen-reared or other cultured fish. What has been identified as *Uronema marinum* has been well studied in aquacultured olive flounder, *Paralichthys olivaceus*, in Korea, but Song et al. (2009) have shown that probably more than one species was involved in the several studies. By obtaining isolates of different scuticociliates from Korea and Japan, cloning and identifying them, and conducting experimental infections with the isolates in flounder, they determined that *Uronema marinum* did not invade the gills, skin, or brain but did produce mortality in up to 30 % of the flounder. Some strains of *Miamiensis avidus* (syn. *Philasterides dicentrarchi*), however, readily invaded the tissues and produced about 100 % mortality and others produced 70 % mortality when those fish were immersed with the ciliate. *Pseudocohnilembus persalinus* and *Pseudocohnilembus hargisi* did not produce mortalities. There are a variety of scuticociliates that can invade tissues of marine and estuarine fishes and invertebrates.

Gulf fundulids, or killifishes, primarily the Gulf killifish, *Fundulus grandis*, commonly exhibited heavy infections of the coccidian *Calyptospora funduli*. The liver (Figure 14.30) and associated pancreatic nodules serve as the primary sites, but infections also occur in the mesentery, gonads, and other tissues. The killifish acquires infections by feeding on grass shrimp in which development to the infective stage occurs (Solangi and Overstreet 1980; Hawkins et al. 1984; Fournie et al. 2000). When a fish that is not a good, susceptible host feeds on the infected grass shrimp, it may acquire the parasite but without normal development or with a strong host inflammatory response; seldom was the accidental atheriniform host killed (Fournie and Overstreet 1993). In *Fundulus grandis*, the parasite occupied up to 95 % of the

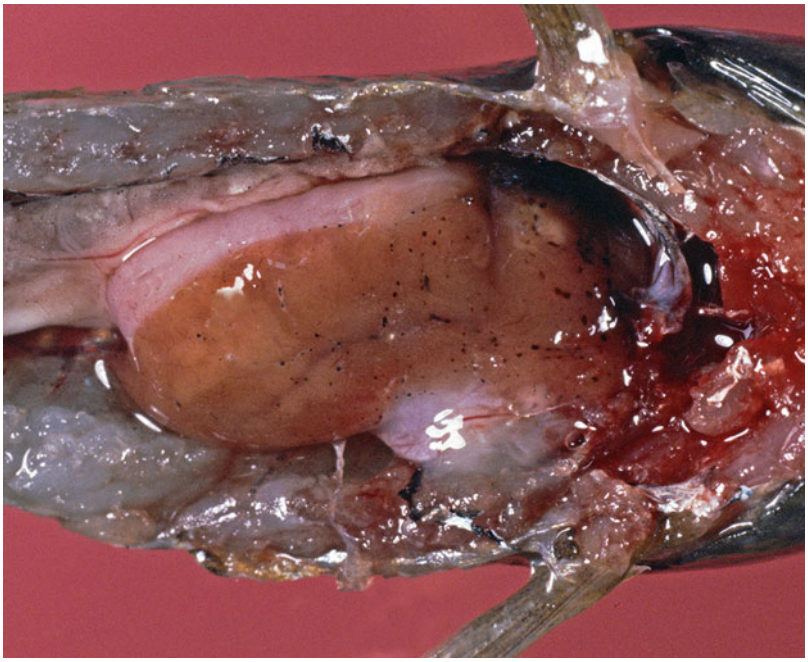

Figure 14.30. Gulf killifish, *Fundulus grandis*, with exposed liver exhibiting chalky appearing area infected by the apicomplexan *Calyptospora funduli,* typically highly prevalent, May 1980.

liver tissue without apparent harm. However, the liver stores glycogen as well as other nutrients, enzymes, and minerals for use by the killifish host when under stress. Consequently, in freezing conditions and presumably in other stressful conditions that require the stored products that, under normal conditions, do not kill uninfected killifish, heavily infected fish die readily (Solangi et al. 1982). Studies on infections in contaminated and pristine habitats in Mississippi are underway.

Most members of the genus *Eimeria*, morphologically similar to *Calyptospora*, have a direct lifecycle in which the infective stage is transmitted directly to the definitive host without developing in an intermediate host. Various species are highly pathogenic and cause mortalities in domestic and zoo animals. Whether this is true for *Eimeria southwilli* that infects the cownose ray has not been determined. However, when wild rays (*Rhinoptera bonasus*) were sampled from Pamlico Sound, North Carolina, 34/37 exhibited oocysts of this coccidian in the coelomic fluid of the seemingly healthy rays (Stamper et al. 1998). When cownose rays were placed in captivity for public display, all those except for the few specimens treated for coccidial infections died from or were associated with a highly pathogenic infection of *E. southwelli*, which was not present in other sympatric species of rays.

The parasitic dinoflagellate *Amyloodinium ocellatum* probably represents the most harmful parasite in aquaculture and display aquaria associated with the Gulf of Mexico. It has a simple lifecycle, with the feeding trophont attached to the gills (Figures 14.31 and 14.32). Once it reaches a certain size or undergoes stress, it retracts its rhizoid from the host epithelium, drops off the host, produces a thin cyst wall, and undergoes a series of synchronous divisions by binary fission. Tomites resulting from this division sporulate to form up to 256 free-living, infective, "*Gymnodinium*-like" dinospores. Under normal conditions, a host in nature would have few, if any, trophonts. However, if restricted to a confined area like an aquarium, pond, or raceway, the number of feeding trophonts on the gills increases logarithmically, and the fish

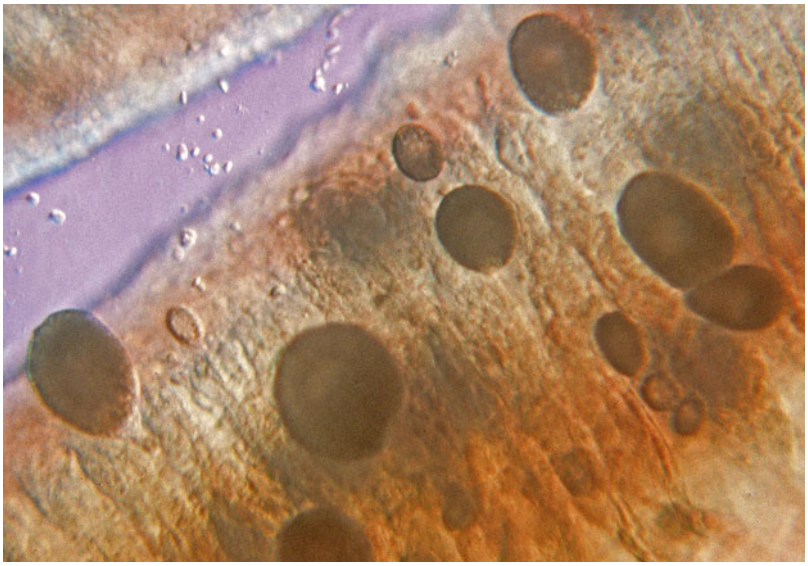

Figure 14.31. A moderate infection of the parasitic dinoflagellate *Amyloodinium ocellatum*, typically found in low mean intensity of infection in gills of the Atlantic croaker, July 1981.

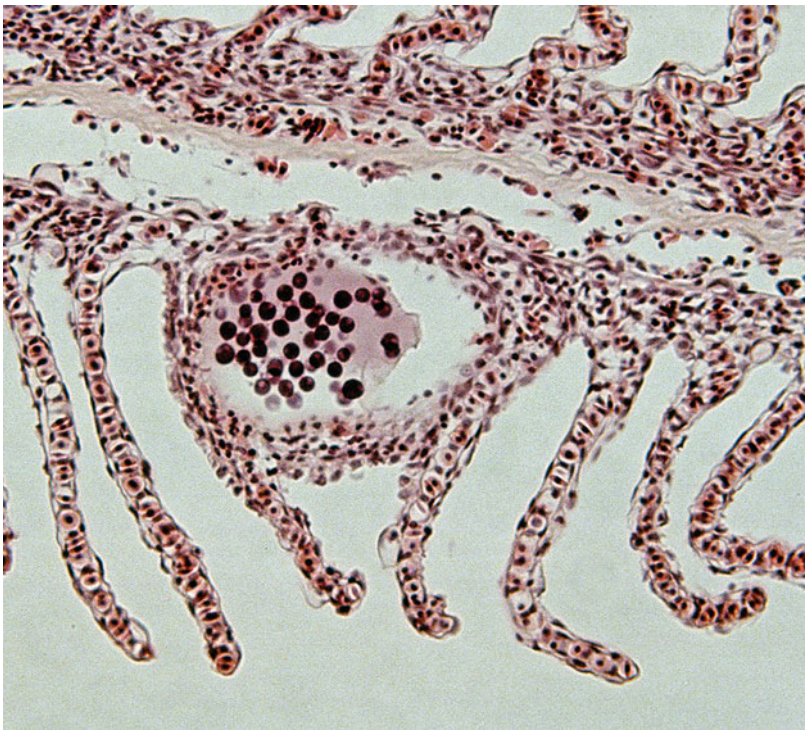

Figure 14.32. Histological section of gill of Atlantic croaker through parasitic dinoflagellate *Amyloodinium ocellatum* and associated minimal hyperplastic host response.

host becomes weakened and dies. In fact, in extremely heavy infections, trophonts may cover much of the body. Since this opportunity to encounter numerous dinospores does not usually occur in nature, mass mortalities as seen in culture are improbable. Overstreet (1993), however, reported a mass mortality on October 31 and November 1, 1984, of fish in a marina and adjoining canal in Alabama; a histological section of the spot gill illustrated the heavy infection with both large and small feeding trophonts. Of the nearly 47,000 dead fish, nearly all consisted of 15–20 cm (6–8 in) long spot.

Not all fishes are susceptible to infection with *A. ocellatum*. Lawler (1980) surveyed fish in Mississippi Sound for natural infections of parasitic dinoflagellates on fishes and found four species which R. Oversrreet confirmed. Lawler recorded 16 of 43 fish species from 28 families with *A. ocellatum*. A raceway was continually stocked with an abundant number of encysted trophonts and dying infected individuals, and specimens of 79 fishes were introduced to the heavy concentration of dinospores. Of those, 71 died and had trophonts covering the body and body-openings as well as in the intestine, allowing for protection during periods of treatment.

Figure 14.33. Sheepshead minnow, *Cyprinodon variegatus*, exhibiting tumorous-like growths caused by the invading myxosporidian *Myxobolus lintoni*, Mississippi, June 1976.

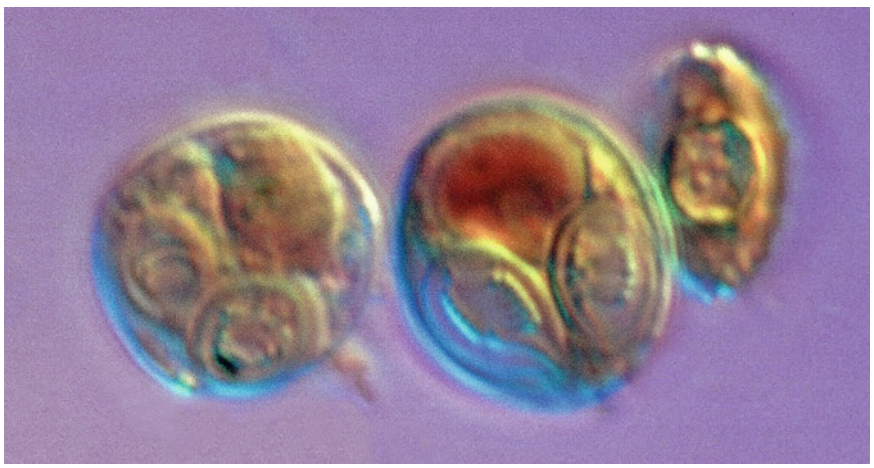

Figure 14.34. Spores of myxosporidian *Myxobolus* stained with Lugol's solution showing positive response for iodinophilous vacuole.

Resistant fishes included *Anguilla rostrata*, *Opsanus beta*, three species of *Fundulus, Cyprinodon variegatus*, *Poecilia latipinna, Menidia beryllina*, two species of *Dormitator*, and *Gobionellus hastatus*; some acquired a few trophonts if placed separately with the dinospores, but none showed erratic behavior or died and most produced an abundance of mucus.

The Myxozoa has historically been considered a protozoan taxon, but evaluation of the development of members clearly shows the group to be multicellular. Sequence data still requires additional data from select taxa as indicated by Evans et al. (2010), but the phylogenetic placement tends to relate the highly divergent group more with Cnidaria than with Bilateria. The histozoic myxosporidian *Myxobolus lintoni* invades tissues in the sheepshead minnow, *Cyprinodon variegatus*, and produces protruding neoplastic-like growths (Figures 14.33 and 14.34). The infection was described from the Gulf in Mississippi and Louisiana by Overstreet and Howse (1977), who considered infections to indicate polluted habitats. Unidentified infections of what is surely the same species had been earlier reported from polluted areas of Galveston Bay by Rigdon and Hendricks (1955). Additional cases were noted from Mississippi and Louisiana by Overstreet (1988), even though no suspect toxicant was suggested. We attempted to conduct experimental studies suggested by Overstreet (1993) to determine conditions necessary to induce invasion, but no infected fish were available when we had the presumed tubificid oligochaete hosts in culture. The infection probably can have a detrimental effect on the fish.

Many species of *Myxobolus* and *Kudoa* exist in the marine, estuarine, and riverine system associated with Mississippi Sound. For example, species of *Myxobolus* infect the bulbus arteriosus and gills of centrarchid fishes and tissues (Cone and Overstreet 1997, 1998) and those of *Kudoa* infect muscle tissue of several fishes (Dyková et al. 1994). These are all rather host-specific, but, under the proper conditions, they could cause a weakened condition in the hosts. *Kudoa hypoepicardialis* infects the space between the epicardium and compact myocardium of several marine fishes (Blaylock et al. 2004), and, unlike most species, it is associated with an inflammatory response and shows close affinity to species that cause myoliquefaction of muscle tissue.

Figure 14.35. Striped mullet, *Mugil cephalus*, infected with *Myxobolus* cf. *episquamalis* under the epithelium of scales. Fish was captured in Mississippi waters on June 3, 1996, but a species similar or identical to that infecting mullet was restricted to petroleum contaminated waters of Akko, Israel; Overstreet and Howse (1977) mentioned that such an infection at that time had yet to be seen in Mississippi.

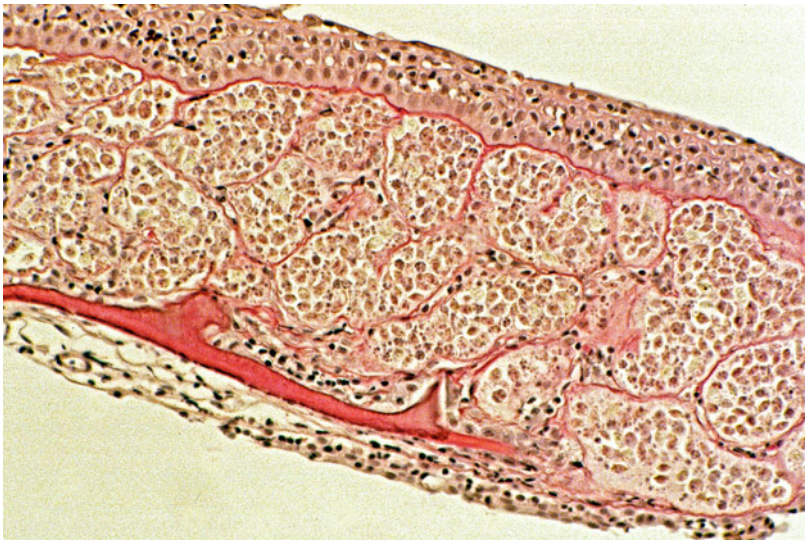

Figure 14.36. Histological section showing Myxobolus cf. episquamalis under epithelium of scales of fish shown in Figure 14.35.

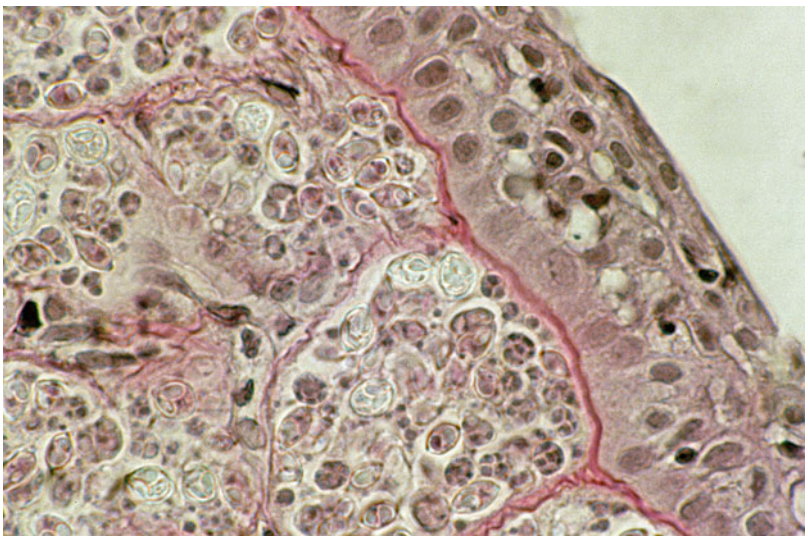

Figure 14.37. Histological section showing close up of *Myxobolus* cf. *episquamalis*.

Overstreet and Howse (1977) reported infected *Mugil cephalus* from the Mediterranean Sea with *Myxobolus* sp. on the scales. Mullet fishermen from along the Israeli coast indicated that the relatively common infection was restricted to the petroleum-contaminated waters of Akko, Israel. Consequently, they blamed infections on the oil. When we reported the infection, we indicated that we had not seen it in the Gulf, but infections would appear to be a good indicator of petroleum contamination. Overstreet (1997) reported the species in that same case as *Myxobolus* cf. *episquamalis*. In 1996, we collected what appears to be the same or similar species in a few striped mullet in Mississippi and call it *Myxobolus* cf. *episquamalis* until the

myxosporidian from the Mediterranean, the Gulf, and elsewhere can be sequenced and compared (Figures 14.35, 14.36, and 14.37). Figures 14.36 and 14.37 show histological sections of the infection and the spores from the Mississippi infection.

14.4.1.5 Helminth (Worm) Parasites

In most cases, metazoan symbionts that are acquired by their fish definitive hosts serve as part of a complicated life history and do not harm their fish hosts. For example, adult trematodes, all considered as true parasites in textbooks, typically do not accumulate in large enough numbers to harm their hosts. A few trematodes such as blood flukes are more likely to be exceptions than other taxa. These typically mature in blood vessels without competition with other parasites. A few species that occur in kidneys, mesenteric vessels, and heart tissues can hypothetically accumulate in high enough numbers to harm their hosts. On the other hand, seldom is the abundance of the infected molluscan hosts present in large enough numbers in the habitat to overwhelm the definitive host. Exceptions occur when the mollusk concentrates below or near a net-pen or other aquaculture facility. The blood fluke lifecycle does not incorporate a second intermediate host; the cercaria penetrates the definitive fish host, and the metacercaria, or schistosomula, develops within the fish, usually not in the circulatory system. Bullard and Overstreet (2002) discuss details about how heavy infections of adults in the heart and in vessels associated with mesentery, kidney, and other organs could cause mortalities in cultured fish from the Gulf and are already known to cause mortalities in freshwater culture systems. In Southeast Asia, where marine aquaculture occurs more frequently, the blood-fluke *Cruoricola lates* causes mortality in the centropomid sea-bass *Lates calcarifer* (see Herbert et al. 1994). Numerous described and undescribed blood flukes occur in potential culturable fish. While most blood flukes occur in the lumen of the vessels, *Psettarium anthicum* and *Littorellicola billhawkinsi* thread themselves within the myocardial lacunae of the ventricle and atrium of the heart of cobia and Florida pompano (*Trachinotus carolinus*) and could directly harm the heart tissues if present in high intensity (Bullard and Overstreet 2006; Bullard 2010). Both hosts are popular recreational fishes, and heavy infections would influence the ability of fishermen to reel-in these fishes from relatively deep water; the cobia has unique, ubiquitous, perivenous, smooth muscle cords in viscera which may allow it to counter the effects of the trematode infection (Howse et al. 1992). The adults of other blood-fluke species could also harm their hosts, and, if in high enough intensity, the miracidia hatching from their eggs lodged in the gill filaments could destroy enough gill tissue so that the hosts would bleed to death. Some fish of importance in the sushi and sashimi market as well as cage culture such as the northern bluefin tuna, *Thunnus thynnus*, have blood flukes in the heart such as *Cardicola forsteri* that require investigation (Bullard et al. 2004).

In cases involving numerous life cycles of other non-blood fluke trematodes, the fish can serve as the intermediate host and can accumulate large numbers of or in some cases few metacercariae (larvae or juveniles). These stages develop from the infective, usually free-living cercariae, agents produced by asexual development and shed from the first intermediate molluscan host. The metacercariae either can cause physical damage or influence the behavior of the fish host so that it is more likely to be eaten by the predatory definitive host. A small number of metacercariae usually do not harm the fish, but when they do, infections can result in predation or mortality. An example involves *Bolbophorus damnificus* and its ability to cause multimillion dollar economic losses to the catfish industry. Overstreet and Curran (2004) report that seldom does a catfish fingerling harbor more than 48 encysted metacercariae, even though adults can harbor more than that (Figure 14.38), and metacercariae of other diplostomoids can occur in nervous tissue infections surpassing 2,000 metacercariae. Presumably, more than

Figure 14.38. Adult channel catfish (*Ictalurus punctatus*) with metacercaria of the diplostomoid *Bolbophorus damnificus*; in the tail; infections of these in fingerling fish in aquaculture kills millions of dollars' worth of fish yearly because the American white pelican, the definitive host, feeds on the fish and transmits the infection, October 1997.

48 kills the fish because we experimentally killed fish exposed to low numbers of cercariae. We also observed kidney damage in pond-reared catfish with over 40–80 metacercariae. Furthermore, Labrie et al. (2004) determined experimentally that fish with approximately four metacercariae died when also exposed to the bacterium *Edwardsiella ictaluri* as long as the exposure was during the first 28 days of exposure, the time it takes for the protective cyst wall to form around the metacercariae. No fish died if exposed to either the trematode or bacterium when not in combination. Other trematodes maturing in either the American white pelican, *Pelecanus erythrorhynchos*, the only known final host of *B. damnificus,* or double-crested cormorant, *Phalacrocorax auritus*, can also kill the catfish in pond-culture and other fishes in the pond, if present in large enough numbers (Overstreet and Curran 2004, 2005; Overstreet et al. 2002).

As indicated elsewhere, infections by juvenile trematodes and other helminths and other parasites may debilitate or otherwise alter the behavior of a fish host so that it is more readily eaten by the definitive host. Usually, only under exceptionally stressful conditions do these parasites cause mass mortalities. An example of a trematode that both debilitates and alters host behavior is the heterophyid *Ascocotyle pachycystis* in the lumen of the bulbus arteriosus of the sheepshead minnow in the northern Gulf. The prevalence and intensity of infections of this trematode are specific to both habitat and season. In low-salinity pools, the fish is unparasitized; along the shorelines open to bays and large estuaries, the intensity may be high but in very few individuals; in isolated or semi-enclosed estuarine sloughs, the infected snail host (*Littorodinops monroensis*) and the fish remain in close proximity during the period of cercarial release, resulting in both high prevalence and intensity of infection. As many as 6,800 of these spherical, thick-cysted, approximately 250 μm (0.01 in) in diameter metacercaria can increase the size of the bulbus arteriosus by mechanical blockage to several times that of its uninfected size. Recruitment of the metacercaria is highest during the first year, with an accumulation of 100–300 parasites per month throughout the year. The rates were only 50–200 metacercaria per month during peak months and less during their third and final year. Larger fish accumulated parasites at higher rates than smaller ones and were more heavily parasitized (Coleman and Travis 1998). Those field studies plus experimental swimming performance studies (Coleman

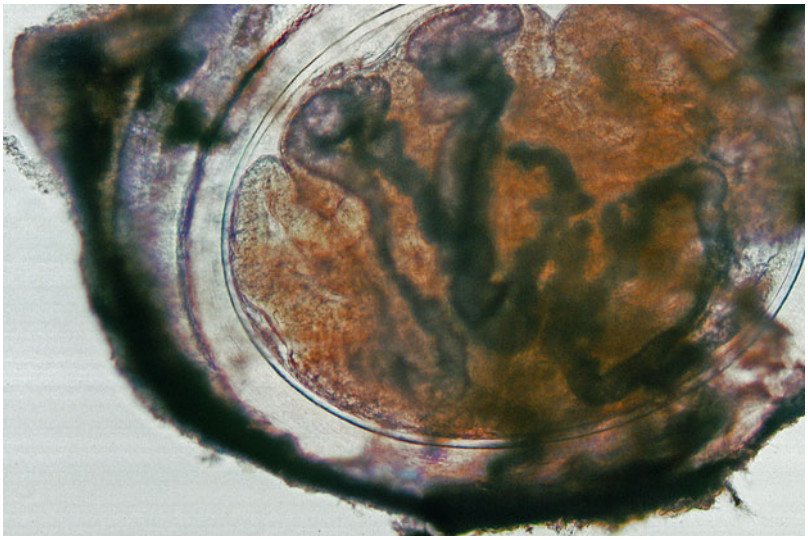

Figure 14.39. Metacercarial cyst of the heterophyid trematode *Scaphanocephalus* cf. *expansus* acquired from skin of the gray snapper, *Lutjanus griseus*, in Florida Keys (March 11, 1997); these cysts, referred to as black spots, help attract osprey or eagle definitive hosts.

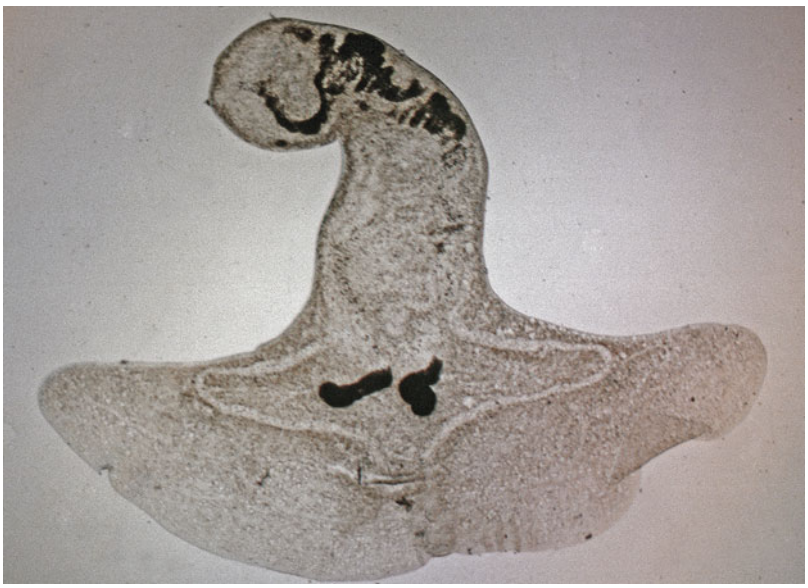

Figure 14.40. Metacercaria of *Scaphanocephalus* cf. *expansus* released from cyst figured as 14.39.

1993) showed that swimming was most detrimentally affected at low temperatures, with low dissolved oxygen levels reducing survivorship of the fish most during the winter when fed on by raccoons, other mammals, and wading birds in which the trematode matured.

The metacercaria of *Scaphanocephalus* cf. *expansus* with melanization surrounding the cyst (Figures 14.39 and 14.40) represents a mechanism allowing their osprey and eagle definitive hosts to more easily prey on infected individuals. Such responses are called "black spots" or

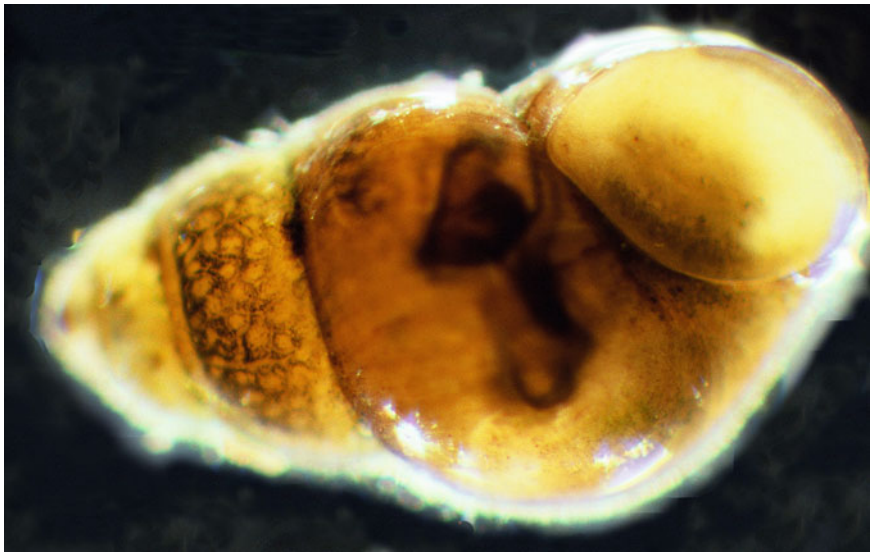

Figure 14.41. Hydrobiid snail (henscomb hydrobe, *Littoridinops tenuipes*) exhibiting microphallid trematode metacercaria of *Atriophallus minutus* in upper whorls infective to ducks (being studied with Richard Heard), Mississippi, December 1997.

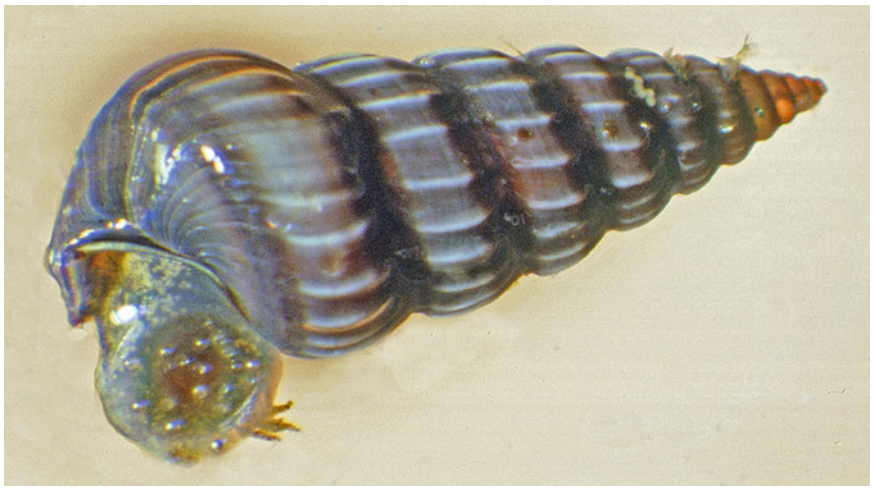

Figure 14.42. The potamidid *Cerithidea scalariformis* (ladder hornsnail) showing notocotylid metacercaria attached to operculum. Florida, being studied with Richard Heard.

other names, and trematodes in several families use this strategy to be preyed on and perpetuate the species, usually in bird definitive hosts.

Whereas most metacercariae infect tissues of fishes and crustaceans, a few occur in or on snails. Various examples are there in Figures 14.41 and 14.42. The large chapter by Bullard and Overstreet (2008) treats all aspects of digeneans and even discusses aspects of Gulf species not available elsewhere.

Cestodes also can influence the behavior of intermediate hosts so that their final host more readily feeds on them. Also, they can be overburdened with infections or the cestode can injure

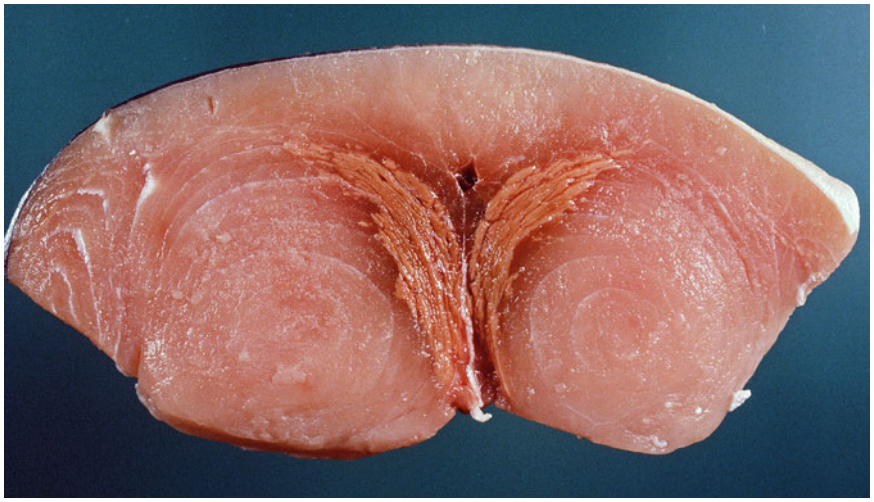

Figure 14.43. Trypanorhyncha cestode in flesh of swordfish, *Xiphias gladius;* the harm to the fish caused by such an infection is unknown.

Figure 14.44. Western mosquitofish, *Gambusia affinis*, exhibiting the invasive Asian fish tapeworm (*Bothriocephalus acheilognathi*), which has spread further across coastal Mississippi after Hurricane Katrina; specimen from Escatawpa River, Mississippi, May 1997.

visceral organs. Small cestode species are abundant and many are reported by Jensen and Bullard (2010). Relatively large species in the flesh gain the attention of fishermen, seafood consumers, and others. Several of those Gulf species have been reported (Overstreet 1977, 1978; Palm 2004; Palm and Overstreet 2000b). Even though *Otobothrium cysticum* is small, it can heavily infect butterfishes and economically affect the seafood fishery (Palm and Overstreet 2000a). Trypanorhychs in swordfish flesh (Figure 14.43) influence consumers and markets, if they realize this object is a parasite and not part of the fish. In any event, it does not harm consumers, even if eaten raw. Others such as *Poecilansystrum caryophullum* in sciaenid fishes

Figure 14.45. Red snapper, *Lutjanus campechanus*, after being fixed showing infestation of monogenoid *Neobenedenia mellini* that was causing mortalities in snapper specimens after being captured, January 2000; these worms are clear and difficult to see on live fish.

also have an unsatisfactory response by many seatrout fishermen and consumers (see questionnaire given by Overstreet 1983a). That cestode species seems to live for 2–3 years and establish an immune response by the fish against further infections. Because only about one-half of a population becomes infected, most spotted seatrout acquire a mean intensity of infection of only 1.5–4.4 worms per fish, depending on location, salinity, and presence of its shark final host (Overstreet 1977). Individual seatrout <14 cm (5.5 in) are seldom seen infected, and, if worms occur, they often associate with vulnerable organs rather than the flesh, where they occur in larger individuals. Consequently, they may kill a few hosts, but not affect the population.

When an introduced species infects a host, it can harm the host in some cases. The invasive Asian fish tapeworm (*Bothriocephalus acheilognathi*) has spread throughout much of the United States, including Gulf habitats with low-salinity or fresh water, but does not seem to have caused noticeable mortalities in the mosquitofish (Figure 14.44) or several other hosts.

Monogenoids consist of helminths that readily harm their hosts, but, as in most infections with trematodes, those usually need to occur in confined habitats like ponds or display aquaria. This group differs from the trematodes because they have a direct life cycle that does not incorporate intermediate hosts. Some species produce two types of eggs, one that will hatch soon and another that can over-winter or otherwise undergo a long period before hatching. The hatched oncomiracidium larva can infest the same individual or another of the same species or, in a few cases, other species. The capsalid *Neobenedenia mellini* provides a good example of a species that infests and can kill a wide range of non-related fishes over a wide geographic area. Figure 14.45 shows specimens of it on a red snapper after the fish was captured in the wild and transferred to an aquarium, where it killed many snapper. Bullard et al. (2000b) list many wild and captive hosts for this parasite from the Gulf and Caribbean. The species probably causes mortality of wild stressed fishes, primarily reef species. It occurs on the gills in lightly infested individuals and can cover most of the body including under the eyelids in heavy infestations. Paperna and Overstreet (1981) and Paperna et al. (1984) reported on a related species in the Gulfs of Elat and Suez, where it infested and caused lesions in a wide range of mullets. Initially

when transferred into the laboratory, fish periodically died for up to 2 months, then infested individuals did not die during the next month, and those surviving finally were free of infestation after 3 months, apparently having undergone a "self-cure." On the eastern shore of the Gulf of Suez in El Bilaim Lagoon, large numbers of heavily infested, emaciated individuals of the keeled mullet, *Liza carinata*, died in April 1974 and again in February 1975. Living individuals became so lethargic that they could be captured by hand.

Lesions caused by relatively large Gulf monogenoids in sharks have been investigated in some detail. For example, the affect of the hexabothriid *Erpocotyle tiburonis* on the gills of the bonnethead shark, *Sphyrna tiburo*, produced intense hyperplastic lesions in the epithelium and resulted in the death of sharks, when reared in public aquaria. The same species of worm occurred in lower intensity and produced relatively minor lesions in wild shark individuals investigated (Bullard et al. 2001). The same authors (Bullard et al. 2000a) also studied skin lesions caused by the microbothriid *Dermophthirius penneri* on the wild blacktip shark, *Carcharhinus limbatus*. Lesions appeared as multifocal, well-demarcated, light gray patches on the skin, but they were chronic conditions not associated with secondary bacterial infections or any debilitating disease.

Three small species of *Rhabdosynochus* infesting gill lamellae of three species of snook (common snook, *Centropomus undecimalis*; swordspine snook, *Centropomus ensiferus*; and fat snook, *Centropomus parallelus*) create problems for managing aquaculture in South Florida, especially for the common snook (Kritsky et al. 2010). *Rhabdosynochus rhabdosynochus* restricts itself to hosts in fresh and low salinity brackish water, but *Rhabdosynochus hargisi* and *Rhabdosynochus hudsoni* tolerate wider salinity concentrations, and, therefore, seem to survive typical migrations of the hosts between marine and riverine systems. Consequently, some species of *Rhabdosynochus* are always present, and treatment with freshwater dips ineffectively controlled infestations in culture facilities.

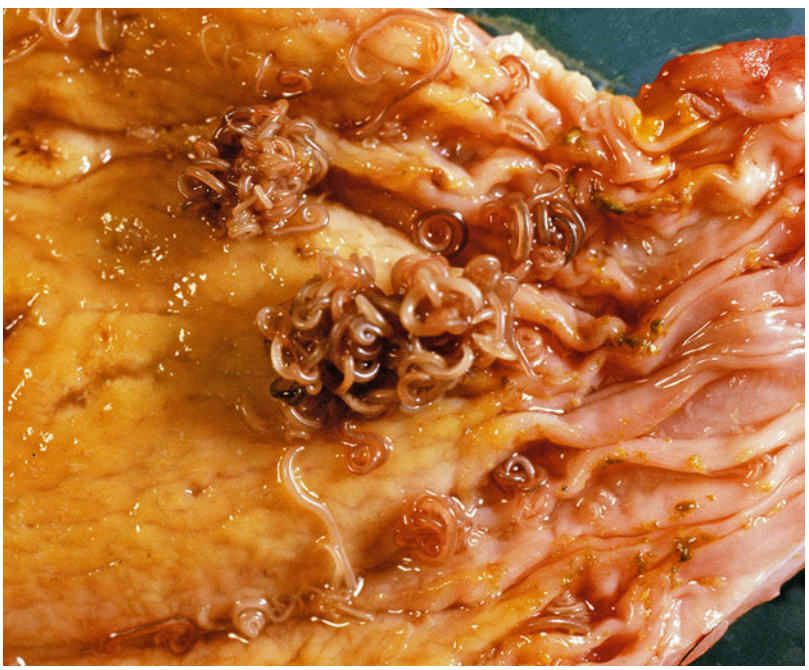

Figure 14.46. Stomach of one of several water birds that exhibit ulcers containing *Contracaecum* spp., the juveniles of which can harm fish intermediate hosts, if in abundance, December 1997.

Nematodes play an important role in the parasite community. Adults have separate males and females as opposed to being hermaphroditic like the flatworms discussed above. In some cases, the adults can cause mortality, but these usually involve situations where the habitat is confined. The Gulf contains numerous species of *Contracaecum*, and the adults occur in piscivorous birds. The American white pelican hosts five species (Overstreet and Curran 2005), and in some cases, juvenile specimens embedded in the proventriculus may cause disease and even mortality. But adults, typically embedded in encapsulated ulcers, can number over 1000 per bird and rather than harming the bird, they often leave the protective ulcer, embed in the prey fish, and help break down the fish tissue. Presumably, they are helpful rather than harmful. Pelicans, herons, egrets, and many other water birds serve as definitive hosts (Figure 14.46). Fish and crustaceans are the typical second intermediate host after a copepod or other crustacean first intermediate host. When in high enough numbers, the third stage infective juvenile can weaken or cause mortality, but infections seldom cause mass mortalities. If the juvenile infects larval or postlarval fish, it can kill the fish that probably have little effect on the fish population. The effect that juveniles embedded in the liver, kidney, spleen, or other organs may have on the reproductive ability of some fish hosts should be investigated.

The nematode *Eustrongylides ignotus* obtains considerable attention because the red worm as long as a human finger can cause human peritonitis when people eat raw second intermediate fish hosts (Overstreet 2003, 2013). Small fish such as the western mosquitofish and small Gulf killifish obtain infections by feeding on the oligochaete first intermediate host. Larger fish that feed on infected mosquitofish and young killifish serve as paratenic hosts, in which the worm grows considerably but remains the same stage infective to the heron or egret bird final host. The striped bass, *Morone saxatilis*, is known to die from an association with the infection in harsh conditions (Mitchell et al. 2009).

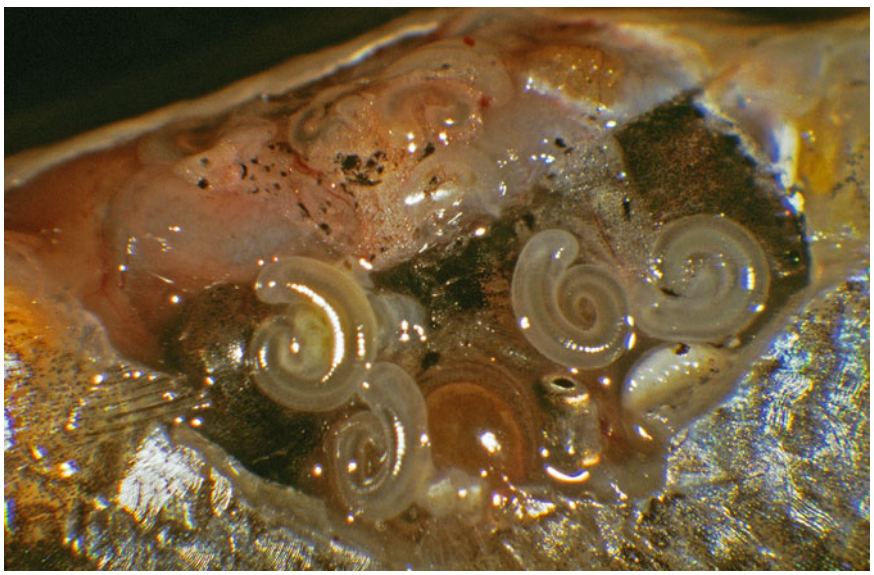

Figure 14.47. Western mosquitofish, *Gambusia affinis*, exhibiting nymphs of the pentastomid *Sebekia mississippiensis*, in the body cavity; these nymphs mature in the lungs of the American alligator, Mississippi Bayou, May 1997.

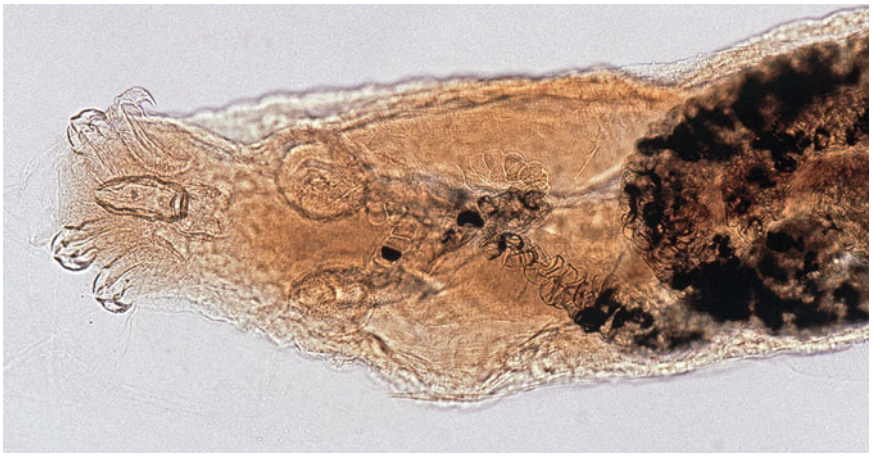

Figure 14.48. Adult female of *Sebekia mississippiensis* from the lungs of the American alligator.

Figure 14.49. Hardhead catfish, *Ariopsis felis*, with the cymothoid isopod *Nerocila acuminata*, which can occasionally be found infesting its dorsal fin in Mississippi estuaries.

14.4.1.6 Other Metazoan Parasites

Western mosquitofish that occur in bodies of water with the American alligator often contain nymphs of the pentastomid *Sebekia mississippiensis* in their body cavity (Overstreet et al. 1985) (Figure 14.47). Fish such as the Atlantic croaker, Gulf killifish, and bluegill that feed on the mosquitofish can become infected, probably debilitated, and serve as indicators of habitats containing the alligator (Figure 14.48) as well as potentially producing human infections (Overstreet 2013).

Figure 14.50. The cymothoid isopod, *Anilocra acuta,* feeding in lesion of ill-spotted gar, *Lepisosteus oculatus*, in small boat basin, Ocean Springs, Mississippi, December 1976; not sure if isopod is responsible for weakened condition of gar or if already weakened condition attracts isopods and argulids to attach and feed.

Figure 14.51. Lane snapper, *Lutjanus synagris*, from off Horn Island, Mississippi, showing exposed cymothoid isopod (in the *Livoneca redmanii-ovalis* complex), June 1997.

Isopods probably have a major influence on fish health and mortality in the Gulf, but they do not seem to cause mass fish mortalities. Members of the Cymothoidae are conspicuous because of their relatively large size. This is apparent for *Nerocila acuminata*, which occasionally infests the dorsal fin of the hardhead catfish in Mississippi estuaries and elsewhere throughout the Gulf (Figure 14.49). A species that seems more pathogenic is *Anilocra acuta* shown feeding in a lesion of an ill spotted gar (*Lepisosteus oculatus*) in the small boat basin at GCRL in Ocean Springs, Mississippi (Figure 14.50). We do not know if the isopod in several

such cases caused a weakened condition in the gar or if an already weakened condition attracted isopods and argulids to attach and feed. In any event, we could bend over and cradle the gars, successfully lifting them out of the water. There are cases where crustaceans can transmit viruses and other pathogenic agents to their hosts. One such case discussed earlier (Section 14.4.1.2) suggests internal lymphocystis virus in the silver perch being transmitted from a skin infection through gill lesions produced by the cymothoid *Livoneca ovalis*. The taxonomy of the *Livoneca redmanii-ovalis* complex needs attention, but the Gulf species is common in the Gulf and infests a wide range of hosts. Figure 14.51 shows a lane snapper, *Lutjanus synagris*, from off Horn Island, Mississippi, with a typically exposed specimen. Usually a single specimen occurs on the gills, and the effect that individual has on the host or the population is difficult to establish. Blaylock and Overstreet (2002), however, provided photographs (Figures 12.38 and 12.39 from Blaylock and Overstreet (2002)) showing six individuals causing a flaring of the operculae and heavily eroded gill filaments of an anemic year-old juvenile spotted seatrout in Mississippi. *Livoneca ovalis* (most likely a separate species of the *Livoneca redmanii-ovalis* complex) was noted by both Pearson (1929) for seatrout from Texas and Overstreet (1983a) for seatrout from Mississippi to occur on runted fish during their first 2 years and was more than likely to cause a gradual mortality of those fish. The infested spotted seatrout in Mississippi ranged between 10 and 17 cm (4–7 in) in length from November through May and often hosted two specimens of the isopod. The same species also caused extensive erosion of the gills of juvenile red drum in Mississippi marshes (Overstreet 1983b). Cymothoids are also known to cause mass mortalities in cultured fish, but the majority of cases involve fishes infested by cymothoids that are not on their natural hosts (Smit et al. 2014).

Careful field and laboratory studies in the Great Barrier Reef on the cymothoid *Anilocra pomacentri* attached laterally on the pomacentrid reef fish *Chromis nitida* demonstrated an association often thought to be relatively harmless because the isopod stays attached for a long period. However, observations on a single cohort of fish showed the significantly depressed growth, reproduction, and survivorship of the infested fish. A parasitized female fish produced only 12 % of the number of eggs produced by a non-parasitized counterpart of the same size. Mortality of juvenile fish was estimated to be 88 % relative to 66 % during the first 70 days after recruitment. Within 48 h of attachment, the isopod penetrates through the skin into the muscle (Adlard and Lester 1995). In laboratory trials, fish mortality from infestation ranged from 78 % for small fish compared with 28 % for larger fish within 4 days of experimental infestation (Adlard and Lester 1994).

Most cymothoid life cycles have been based on speculation; two have been described with considerable detail: *Glossobius hemiramphi* from the ballyhoo, *Hemiramphus brasiliensis*, from South Florida (Bakenhaster et al. 2006) and *A. pomacentri* mentioned above from Australia (Adlard and Lester 1995). The final marsupial stage on the female known as a manca immediately swims to the appropriate final host and develops into a male. Then in turn, this protandric hermaphrodite develops into a female, which feeds heavily on host blood, apparently only during periods related to onset of vitellogenesis. The adult female occupies the buccal cavity of the ballyhoo or externally dorsal and posterior to one of the eyes of the chromis. Some other species probably differ because juvenile stages have been collected from hosts other than the recognized final host.

Another of the several Gulf cymothoids that attracts attention of biologists is *Olencira praegustator*, a species that infests the tongue of the Gulf menhaden. Guthrie and Kroger (1974) point out that their data, which also covers infestations of the Atlantic menhaden along the Atlantic coast, suggest that injured or infested adult menhaden school with juvenile menhaden in estuarine nursery areas rather than with their own year-class of fish in offshore waters. When enough infested individuals were present, they formed schools independent of

the uninfested juveniles and remained longer in the estuary. Because male and female *Cymothoa excisa* attached to the tongue of three Caribbean snappers from seagrass beds and occupied so much space in the mouth cavity, Weinstein and Heck (1977) thought the isopod would affect the condition factor of the fish; but they found no such effect.

Other species of isopods play important roles in fish health, especially if in any way restrained. For example, most cirolanids such as *Cirolana parva* are actually micropredators that will devour restrained fishes in a short period; in fact, it and other cirolanids have been used by the shark cartilage industry for cleaning the shark carcasses of flesh prior to processing (Poore and Bruce 2012). Poore and Bruce (2012) also cited how the Florida shark industry over one summer collapsed when cirolanids (*Natalolana* spp.) swarmed and ate their way into living sharks, killing them by destroying their vital organs. Others such as *Rocinela signata* have temporary fish hosts to obtain blood meals. Excorallanids such as *Excorallana* spp. in their early stages parasitize bony fishes as well as sharks and rays and then may retire to sponges to molt and reproduce between blood meals. However, *Excorallana delaneyi* in sponges from the Gulf was not seen on fishes (Stone and Heard 1989). Many species of three genera (*Excorallana, Alcirona*, and *Lanocira*) in the Gulf occur temporarily on fishes and then associate in cryptic habitats with sponges, ascidians, tube-molluscs, corals, and mangroves (Delaney 1989). The tridentellid *Tridentella ornata* infests the nasal cavity of several grouper species and the red porgy, *Pagrus pagrus*, in the Gulf off Florida where they are thought to "pounce" on a fish just long enough to get a blood meal (Kensley and Heard 1997). Schotte et al. (2009) list just two named gnathid isopods from the Gulf, but we have seen adults and larvae of a few unidentified species, so more species exist. The late larvae (pranizae) of species that use elasmobranchs remain attached in the oral or buccal cavity until ready to mature, but species obtaining bloodmeals from bony fish drop off and molt after each blood meal. Except for a few species that permanently attach to fish, one rarely observes larvae of the teleost feeders. Paperna and Overstreet (1981) report on one such Red Sea species that provides a good example. Mullet were placed in floating cages located in water 1–2 m (3–7 ft) and 6–8 m (20–26 ft) deep in the Gulf of Elat. Larvae from the benthos attacked fish at night and fed on their blood. Those fish in cages closest to the bottom, especially when not accustomed to their cages, became anemic from heavy infestations. The larva fed three separate times and after each, it molted and increased in size before reaching maturity. The larvae molted into male or female adults 6–8 days after their last blood meal at 24 °C (75 °F). After 22–24 additional days, the eggs developed, the young free-living larvae searched out fish for a blood meal, and the full-sized, free-living female, after producing about 90 larvae, died. Many of the mullet in the shallow cages died, but few larvae became attached and fed on mullet from the cages in deeper water.

Caligid copepods, often referred to as sea lice, commonly occur in large numbers on Gulf fishes such as flounders, hardhead catfish, drums, and seatrouts, but the effect of adult copepods on these and other hosts has not been established. Frasca et al. (2004) described in good detail the operculum lesion in wild black drum infested by *Sciaenophilus tenuis*. This and other caligids, however, would certainly have a detrimental role in cage culture, if it became more prominent in the Gulf. Ho (2000) discussed nine species reported to have caused mortality in Asian non-salmonid fishes. Salmonid aquaculture in the North and South Pacific, Scandinavian countries, and elsewhere are plagued by *Lepcophtheirus salmonis, Caligus elongatus,* and related species. Hewitt (1971) reported that the presumed introduced *Caligus epidemicus* killed several wild fishes in southern Australia. One reason caligids cause mortalities in aquaculture is because there is no intermediate host. Different species have different number of larval stages, but the non-feeding naupliar stages produce a copepodid that attaches to the fish by a frontal filament as chalimus stages. We have observed unidentified chalimus stages attached to fry and

Figure 14.52. Bay anchovy, *Anchoa mitchilli*, from Mississippi Sound with copepod *Lernaeenicus radiatus*, attached *within* blood vessels; infections probably weaken adult fish that more than likely will attract predators; when in postlarval fish, the host would probably be much more stressed and vulnerable, June 1981.

young juveniles from planktonic and other collections. We can only speculate that one such attached copepod can either make that fish vulnerable to predation or produce mortality.

Felley et al. (1987) examined over 27,000 fish from 33 taxa from the ichthyoplankton of Calcasieu Estuary, Louisiana, and reported caligid chalimus stage copepods attached to the dorsum of what was probably the bay anchovy (*Anchoa mitchilli*) and attached to the ventrum of the Gulf menhaden. Only 3.6 % of the postlarval anchovies and 0.2 % of the postlarval menhaden were infested. Additionally, 4.4 % of unidentified gobies had infestations of pre-adult copepods. Infestations were not observed on larval fish, and the length of the copepods on the postlarvae averaged about 15 % and reached almost 40 % of the host length. The authors concluded that the substantial hydrodynamic drag probably produced considerable stress. Overstreet (1978, 1983b) provided figures of a chalimus stage attached to the fin of the Florida pompano and postlarval red drum from off Mississippi.

The pennellid copepod *Lernaeenicus radiatus* probably has a major impact on fishes in the Gulf. Both the Gulf menhaden and bay anchovy as well as other anchovies are susceptible to heavy infestations during some years. Also the Atlantic croaker, seatrouts, Gulf killifish, and gobies often contain one or two of this embedded copepod. The lifecycle in Mississippi (Overstreet 1978) involves chalimus stages both attached to and free on the gills of the intermediate host, rock sea bass (*Centropristis philadelphica*), primarily in high salinity waters. Then the male breaks its frontal filament and transfers a spermatophore to the female, leaves the sea bass within 3–5 days, infects another fish, matures, and produces eggs within a week. Its anterior extends into the fish flesh so that its head, which forms antler-like appendages, clings around a vertebra or some other structure adjacent to a rich blood supply that serves as its food source. When vital organs of the fish are disturbed or, if too many individuals infect a host, the host can die. We used the Gulf killifish as an experimental host, and death usually occurred

Figure 14.53. Yellow fishlouse, *Argulus flavescens*, a potentially harmful branchiuran.

when more than one or two individuals infected it. The abundance of the parasite typically depended on the abundance of the infested sea bass. Figure 14.52 shows the copepod embedded in a bay anchovy a short distance posterior to its eye.

Skin erosion and ulcerations caused by species of *Argulus* (Branchiura, a separate crustacean group superficially similar to caligid copepods) occur commonly in aquaculture and in fish on public display. Species from the Gulf (for example, Figure 14.53 showing a species from the southern flounder, but that argulid species also occurs on Atlantic croaker, *Mugil cephalus*, dasyatid rays, and other fishes, including freshwater ones) are reported by Overstreet (1978) and Overstreet et al. (1992). A species also infested the Mayan sea catfish, *Arius assimilis* in Chetumal Bay, Mexico (Suárez-Morales et al. 1998). We have seen few cases where harm has resulted in fish from the wild. Two are caused by *Argulus lepidostei* infesting ill spotted gar in conjunction with the isopod *Anilocra acuta* mentioned above and another species causing deep excavations in the skin of the gizzard shad in a Louisiana estuary. Kolipinski (1969) also reported on an infestation on the Florida spotted gar in a pond in the Everglades National Park of what was later described as *Argulus meehani* and may be the same species as occurred in Mississippi. He reported and figured the mass mortality of more than 2,000 gar. This and other aggressive argulids species such as *Argulus catostomi* can kill a fish overnight. He thought the reason that the gar died was because of a prior oxygen depletion that affected animals other than the gar, resulting in the loss of some animals that normally feed on the argulids. An experimental study showed that the flagfish, *Jordanella floridae*, and golden topminnow, *Fundulus chrysotus*, as well as a water scorpion can eat as many as 30 individuals in 28 h. Overstreet et al. (1992) also provided the microscopic anatomy of this interesting group.

Symbiotic barnacles, like those infesting the blue crab, other crustaceans, sea turtles, and marine mammals, do not infest fishes. However, some festering and marine barnacles that attach to a wide variety of substrata do embed in the skin of a few fishes, especially drums, sheepshead, and a few other species such as the hardhead catfish (Figure 14.54). Overstreet (1983b) figured a rare infestation of *Balanus improvisus* in the skin of the red drum from

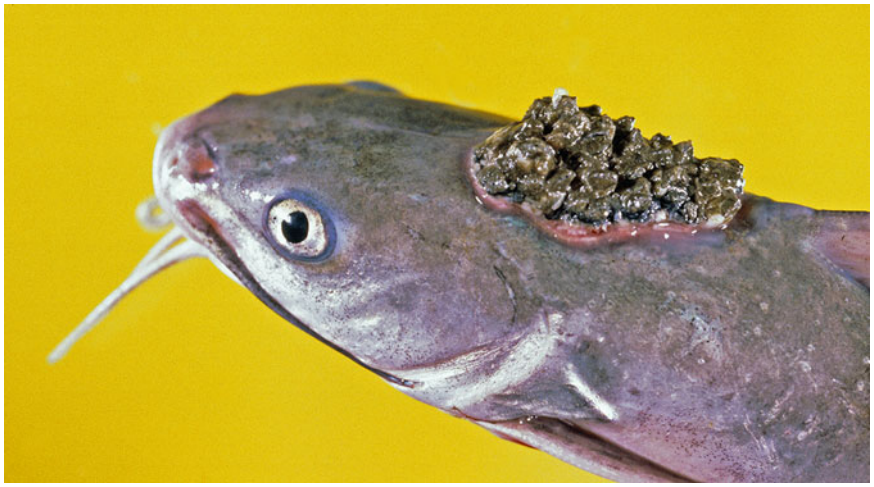

Figure 14.54. Hardhead catfish, *Ariopsis felis* (SL 207 mm), with abnormal growth of acorn barnacles and secondary bacterial infection, Graveline Bayou, Mississippi, May 11, 1988.

Mississippi Sound, but he had seen it much more commonly up to 2010 in the red drum, black drum, and sheepshead.

14.4.2 Use of Parasites to Evaluate the Effects of Catastrophic Events

Many articles exist using the parasites in or on fishes as indicators to solve problems. Some parasites or group of parasites indicate general heath of the fish model, general ecosystem health, migration of the fish, stock of the fish, trophic level of the fish, feeding behavior of the fish, stress on the fish, and other features. As stressed in the article by Overstreet (1997), the ability to achieve a good indicator/answer depends on the fish species and the parasites chosen. These will differ according to the question asked, and most certainly every fish species and every parasite species will not answer a specific question. The more that is known about parasite species, the better the indications. In other words, the longevity of the larval or adult stages of the worm and the seasonality of the worm may be critical when determining when and where the infection took place. In some cases, the habitat of the first or second intermediate host will indicate in what general or specific habitat the infection was required. For example, the Atlantic croaker typically provides a good model to answer many questions. It uses the estuary to develop, and even though the juveniles move through the passes into high salinity Gulf waters sometime between late spring and early autumn depending on location, temperature changes, and other factors, some individuals remain in the estuary throughout the year. On the other hand, when the salinity of an estuary increases, mature fish from the Gulf move into the estuary and this migration can be detected by the species of parasites present. Species that occur in the croaker inshore often differ from those occurring offshore. The number of species present, or species richness, especially when the origin of the infection is known provides an indicator of the diet of the fish and general health of the environment. A healthy environment includes a large number of infected intermediate hosts, and the adult worms in the croaker indicate a healthy fish rather than one being harmed by the parasites. Moreover, the presence of long-lived species in croaker from the Gulf may indicate from which estuary it was derived. Unlike a croaker, which has a fairly large feeding habitat in the estuary, the Gulf killifish has a very restricted home range. Consequently, the species present in the killifish from a specific habitat

indicate the health of that specific habitat. For example, after a catastrophic event, many of the parasites are not available to infect the killifish because their hosts no longer inhabit the area. Both the croaker and the killifish harbor each about 70 different species in the Mississippi estuary, with many in the croaker being adult stages and many in the killifish being larval stages. Many of those larval stages may occur encysted within tissues for over a year. Therefore, when monitoring juvenile killifish that were born after the catastrophic event, the absence of the specific parasites will indicate the absence of either the corresponding intermediate or definitive hosts. When monthly or seasonally infections of the specific parasites become established in the killifish, the sampled habitat with all of its hosts is becoming healthier. The western mosquitofish, which also has a restricted home range, has many more larval stages than adult stages of parasites, and it also has 70 or so different parasites in Mississippi; it provides a good indicator of environmental health in both freshwater and low salinity habitats. Overstreet (1997) illustrates the large number of invertebrates and vertebrates such as birds, snakes, turtles, alligator, raccoon, and others that have to inhabit the location sampled at least for a period long enough to transmit the infection. Many of the occupants of a habitat, either the individuals or their offspring, become reestablished after a catastrophe. However, it takes much longer for the occupant hosts to become infected and transmit infective stages to the three example model fish. That is why the biodiversity of parasites in those models indicates the rate of restoration of a healthy environment.

Not all parasites in the model fish are harmless or absent after a catastrophic event. Many of these have a direct lifecycle in which reproduction takes place in or on the model fish rather than an indirect cycle that requires feeding on or being in the general area of infective stages. A polluted environment may be conducive to replication of these parasites with the direct cycle. Furthermore, toxic components in the environment may stress the model fish so that it is more susceptible to extensive reproduction of the parasite, and it becomes harmed. Consequently, examination of parasites provides a variety of indications of both host and environmental health.

Sometimes the use of all the parasites or helminths from all the hosts in a community or restricted habitat provides the best indication of harm or restoration. The example below on the use of parasites to assess hurricanes serves as a good example.

Hurricanes have the potential to cause considerable mass mortalities of fish and other animals, and this damage depends on the date/temperature, the amount of and direction of wind, the length of time the winds hover over the habitat, the amount of tidal amplitude, the local geography, the habitat, and other features. In many cases during and after severe storms, researchers are not available to survey the conditions because the power is out, generators and boats are destroyed, roads are blocked keeping away researchers and passage to surrounding locations, time or ability to attend to destroyed or damaged laboratory and personal facilities is curtailed, and of course the relative importance of human fatalities outweighs those of fish. In a few cases, animal mortalities are reported or described, but the most useful information regarding the effects of storms relates to indicators, which will be discussed below.

Storms are responsible for many fish kills, as well as kills of other organisms. Mortalities and strandings associated with a few storms in southern Florida have been surveyed. Several days or weeks of onshore winds in regions where such winds are not common, such as quiet waters over broad shoals and behind sandbars, can produce heavy seas with a turbulence resulting in harmful sands and other sediments that accumulate and clog gills of the fish as well as erode the gill filaments and cover otherwise healthy habitat. Robins (1957) noted that hurricanes and lesser storms produced accumulations of dead and dying fish along Marco Beach and Sanibel Island, Florida, in the Gulf of Mexico. During most storms, fishes common in the area probably escaped to safer waters; however, such storms provided ichthyologist

Robins the opportunity to collect rare or previously unknown fishes that washed up on the beaches. Under the proper conditions, massive fish kills of common species can occur. Hurricane Donna in September 1960 provided the opportunity to assess loss of biota from the Cape Sable region of northern Florida Bay including specific portions of the Everglades National Park by Tabb and Jones (1962) because they were very familiar with the biota of the area. Winds up to 241 km/h (150 mph) produced tides ranging from 0.46 m (1.5 ft) below to 3.7 m (12.1 ft) above mean water and destroyed mangrove trees, created a massive drift line of turtle grass (*Thalassia testudinum*), and churned up the calcium carbonate marl with associated hydrogen sulfide, all in different regions. Direct action of the storm with its turbulence stressed and suffocated many fish, resulting in massive fish kills in the shallow waters. The dead fish and decomposing vegetation resulted in oxygen depletion, which in turn resulted in post-hurricane fish kills and absence of several common fishes. Some fish species were scarce after the storm, but recreational fishes were abundant in deep water off Cape Sable soon after the storm; the effects in the shallow estuary were also temporary. Landings of the pink shrimp by the Tortugas fishery were about six times greater than prior landings during the same period, suggesting that the storm caused this shrimp to move from its nursery to the fishing grounds earlier than usual and at a smaller size. The healthy blades of turtle grass continually fragment, especially with extremes of temperature. The amount of turtle grass washed ashore in Biscayne Bay, just north of the area with massive fish kills, was massive; however damage to the grass beds and associated fauna due to freshwater runoff in nearshore areas could have been more severe than that caused by the physical storm damage (Thomas et al. 1961).

Hurricane Andrew (August 16–28, 1992, Category 5, with winds up to 282 km/h (175 mph)) (Tilmant et al. 1994) was a brief but extremely strong storm, with unidirectional currents and onshore tidal surges. Seagrass beds remained remarkably untouched unlike during Hurricane Donna when they were heavily destroyed. Andrew produced massive fish kills in the mangrove zone of Everglades National Park, in which the perturbed bottoms left a hydrogen sulfide smell but no sign of fish kills after 4 weeks. Also, upland forest communities were destroyed (Smith et al. 1994), but little damage occurred underwater with the exception of some submerged hardbottom communities of Biscayne Bay that encountered a loss of sponges, corals, and algae. Coral damage on upper reef surface of a few reefs (Elkhorn Reef) displaced some fish that used reefs as a protective habitat. There were few other mortalities; about half of the sea turtle eggs had already hatched, no dead manatee or crocodile occurred, and their habitats were not destroyed.

Strong storms like Hurricanes Katrina and Rita resulted in some locally restricted fish kills such as reported from the Pascagoula River floodplain lakes (Alford et al. 2008, 2009), but, for the most part, no data support the overall short-term mortality events. A few newspaper articles and photos exhibited exceptional fish kills, but most common species left the area prior to the harmful waves and returned soon after the storms. The following years typically produced good catches by recreational fishermen.

We consider a powerful method to define losses of biodiversity and faunal abundance to be through parasitological indicators. This tool is useful for damage and recovery caused by hurricanes as well as other destructive events. The perfect example is the activity caused by the Category 5 Hurricane Katrina, which occurred on August 29, 2005, with the final landfall in Mississippi near the Louisiana state line; it blew as a 280 km/h (174 mi/h) storm with gusts as high as 433 km/h (269 mi/h), with surges over 9 m (29 ft) high. The surges penetrated 10 km (6 mi) inland in Mississippi and 20 km (12 mi) along the bays and rivers. The storm devastated an area equal to the size of Great Britain. It washed away much of Southwest Pass, Louisiana, where the Mississippi River meets the Gulf of Mexico. It also washed away 25 % of the footprint of the barrier islands off Mississippi and Louisiana. It scoured 1 m (3.3 ft) of

sediments in water 25 m (82 ft) to 30 m (98 ft) deep, redistributing sediments and depositing 30 cm (1 ft) of sediment at a depth of 50 m (164 ft) and killing most of the infauna but without bodies visible to the human eye. A 6-month drought following the hurricane also influenced biodiversity, mostly the terrestrial fauna and flora that was visible to the local human population. The way that parasites were used to assess the damage was reported by Overstreet (2007) and updated in this chapter.

The presence or absence of parasites in local fishes, especially resident fishes, provides an indication of the loss and recovery of the overall aquatic biota after a hurricane. Based on the species of parasite, with emphasis on its lifecycle, the length of time it takes to become reestablished, the longevity of the parasite, and the effect of the parasite on the host, one can evaluate the effects of perturbation, surge, and presence of toxicants. Most helminths have complicated lifecycles that include two to four hosts, involving a variety of invertebrates and vertebrates that may be specific, closely related, or general. Consequently, by understanding the lifecycle of a specific parasite, one can conclude that all host members of that specific cycle occur in the environment in order for the parasite to be present in the fish being investigated. It is also important that the fish being monitored fits some of the established criteria such as having a restricted home range, a relatively short lifespan, or other features necessary to indicate the specific problem being investigated (Overstreet 1997). When trying to determine how long it takes for reestablishing specific members of the infauna that were lost to perturbation, one has to examine for parasites that have cycles that require those specific hosts as members of the infauna. This is done by noting the date that individuals of the parasite show up in the specific model fish host species, preferably juvenile fish individuals born after the storm.

In the case of perturbations, some trematodes include bivalves, part of the infauna, as first or second intermediate hosts. As an example for an early reestablishment of both the parasite and the bivalve, *Diplomonorchis leiostomi* (Monorchiidae) occurred as immature specimens temporarily in spot (a sciaenid fish) from a few locations in estuaries in Mississippi in February 2006, 6 months after the storm. That trematode reoccurred in spot from a few habitats in January 2007. By March 2007, it occurred in the Atlantic croaker, the common host for that adult parasite. It occurred in low mean intensity throughout specific habitats in patchy distributions for 2.5 years post-Katrina after which time it occurred commonly in higher numbers in more individual fish in more habitats. Another trematode example from Mississippi was the monorchiid *Lasiotocus* cf. *minutus*, which matures in the Gulf killifish (*Fundulus grandis*) and is acquired from the Florida marshclam, *Cyrenoida floridana*, in marsh habitats. Even though the killifish, similar to the spot, returned to its normal habitat within days to weeks after the storm, the trematode did not show up in fish born after the storm until 19 months and then was common after 2 years. The nematode *Eustrongylides ignotus* infects the killifish as a second intermediate host after the fish feeds on the benthic oligochaete intermediate host or on a mosquitofish that had previously fed on the oligochaete, and in that case making the killifish a paratenic host. The juvenile nematode was absent from the killifish for 1.5 years and remained relatively uncommon and patchy until 2010. This conspicuous red nematode takes a long time to develop in the oligochaete and ultimately matures in the proventriculus of a few herons. In contrast with most of the parasites that occurred in infauna, some acquired from copepods were relatively common in fish hosts by 7 months. At that time, the Atlantic croaker was infected with juvenile nematodes of *Hysterothylacium reliquens*, adult nematodes of *Spirocamallanus cricotus,* and adult specimens of the hemiurid trematode *Lecithaster confusus*, which requires a snail before infecting the copepod prey.

In the freshwater area perturbed by the storm, there was a loss of the sphaeriid clam infauna, which hosted members of the gorgoderid trematodes *Phyllodistomum* spp. It took

20 months for the first species to show up in the fish, and it was very rare; by 28 months, multiple species were relatively common in the urinary bladder of catfishes, sunfishes, and fundulids.

The storm surge from Hurricane Katrina gradually encroached the rivers, bayous, marshlands, and uplands of Mississippi as well as lesser surges along Louisiana and Alabama coasts, overflowing the banks and flooding the entire area for at least 20 km (12 mi) along the bays and rivers. In an area with approximately a half meter diurnal tides, over 8 m (26 ft) of surge devastated most of this area. Much of this devastation resulted from the much greater retreat of the water relative to the ebb. From the point of view of the parasites, low salinity and freshwater was rapidly replaced with 32 ppt water, which remained in some ponds, low lands, and other areas. Consequently, snail hosts for haploporid and other trematodes had difficulty reestablishing and were sometimes outcompeted by related species. Two of these haploporid trematodes, *Culuwiya beauforti* (usually common in winter months) and *Dicrogaster fastigata* (now known as *Xiha fastigata*, usually common in summer months), are transmitted by hydrobiid snails as their first and only intermediate host, occurred but were rare 11 months later, and then disappeared from their striped mullet final host. They reappeared 8 months later but disappeared again, finally becoming common in June 2007. Their mean intensities were low in March 2008, but finally became common again in 2009. The striped mullet continually migrates from the shallower Gulf of Mexico and Mississippi Sound into the lower salinity estuarine areas, and the snails had disjunct stocks. Several species of heterophyid trematodes also utilize hydrobiid snails, but they use specific sites in their second intermediate host fishes and mature in a variety of birds and mammals. One initially showed up as a metacercaria (larval stage) in its fish host in June 2006, but others were more delayed, one metacercaria showing up in February 2008 and those of several others by 2009. In freshwater rivers in coastal Mississippi counties, numerous collections of centrarchids, catfishes, catostomids, fundulids, and other fishes yielded numerous trematode species pre-Katrina, but not until August 2006 did any fish exhibit any trematode. The first case of *Megalogonia ictaluri*, an allocreadiid presumably hosted by a sphaeriid (fingernail clam) or unionid clam to an insect or crustacean, appeared in a channel catfish in August 2006 and more were present in May 2007. *Plagiocirrus loboides,* an opecoelid hosted by a snail to a crustacean, did not show up in its fundulid final hosts in spite of numerous attempts by Steve Curran to collect it until early 2011, except for a single infection in April 2007. A few other freshwater parasites appeared by mid-2007 and many more by March 2008.

The Atlantic croaker is also one of our model fish species for parasite infections, and it usually has an abundance of parasites. One of these is the adult cryptogonimid *Metadena spectanda,* which has a typical snail-fish-fish trematode cycle to be published soon and which was first seen after the storm in July 2006 but uncommon until reestablished in 2009. Another helminth is the adult acanthocephalan *Dollfusentis chandleri*, which was not reestablished until early 2009.

In regard to parasites in the Gulf killifish and Atlantic croaker, those that do not have an indirect complicated lifecycle like monogeneans showed up shortly after the storm. The coccidian protozoan *Calyptospora funduli* in the liver and other visceral tissues of the Gulf killifish was extremely common pre-Katrina, but it did not show up until August 2006 and then occurred only patchily along the western coast of Mississippi where all properties were destroyed; it also occurred in a patchy distribution along the eastern Mississippi coastline but not until 2010. This difficult reestablishment seems strange to us because the intermediate host is the daggerblade grass shrimp (*Palaemonetes pugio*), which is extremely abundant along the entire coastline.

In contrast with the above cases, the haploporid *Intromugil mugilicolus* from the striped mullet had not been seen for decades before Hurricane Katrina occurred; it appeared

commonly in March 2007 and March 2008. That worm is typically found in the mullet during winter months and absent by May or June. We do not know the snail intermediate host, but it is probably a hydrobiid that was rare in our Mississippi estuaries pre-Katrina.

Parasites in migratory fish are another matter. Adults of the bucephalid *Prosorhynchoides ovatus* in the Atlantic tripletail (*Lobotes surinamensis*) occurred abundantly inshore and offshore both before and after the storm as did other bucephalids from other offshore migratory fishes such as scombrids. The bucephalid lifecycle involves a bivalve to a fish to the final fish host.

Fauna of the sandy barrier islands off Mississippi include a variety of crustaceans, clams, snails, polychaetes, acorn worms, brittle stars, and other invertebrates. Even though those beaches were lost or reshaped, most of the nearshore beach fauna was reestablished, according to Richard Heard of USM, within 6–12 months. Some of the species on the Gulf side took longer, up to 2 years, especially those species without planktonic larvae such as pericaridean crustaceans (like amphipods and isopods). Reestablishment of these animals was easier to determine than that of those from the muddy benthos, but what is important to remember in regard to parasites is that once the invertebrate has reestablished, worms from an infected final host have to deposit eggs and the resulting larvae have to infect those invertebrate hosts. As indicated above, this infection of a parasite may take several years, and the final monitoring results provide information on the presence of all the hosts for a particular parasite in the ecosystem and the overall environmental health of that ecosystem.

Parasites are also good indicators of toxicants in the ecosystem. The myxosporidian *Henneguya gambusi* probably infects *Gambusia affinis*, the western mosquitofish, in extremely low intensity and is difficult to detect. However, when in a stream along coastal Mississippi that was contaminated with the heavy metals chromium, copper, and arsenate, the mosquitofish exhibited heavy infections that involved the intestine, gonads, kidney, and even brain tissue (Overstreet and Monson 2002). Infections presumably kill the mosquitofish in nature since when we transferred samples of fish from this location into the laboratory and maintained them in aquaria, a little more than half the fish died within 6 months when compared with none dying nor expressing an infection in samples collected from non-contaminated locations. No infected fish survived 12 months, but non-infected ones had a high survival. After the hurricane, infections were absent; however, they occurred again in August 2007. The surge apparently flushed out the contaminated water and the infected oligochaete intermediate hosts. The mosquitofish from this location before the hurricane contained no adult tapeworm infection. After the storm, the invasive Asian fish tapeworm (*Bothriocephalus acheilognathi*) showed up in the mosquitofish (Figure 14.44); this adult cestode had previously been collected from mosquitofish in locations numerous kilometers from that contaminated location.

Parasites have been shown to be good indicators of pollution, especially polycyclic aromatic hydrocarbons (PAHs) and PCBs, but usually as non-point sources. Some studies treat the Gulf (e.g., Overstreet and Howse 1977; Skinner 1982; Overstreet 1988; Landsberg et al. 1998; Vidal-Martínez et al. 2014), but most studies treat other areas (e.g., Khan and Thulin 1991; MacKenzie et al. 1995; Austin 1999; Broeg et al. 1999; Dzikowski et al. 2003). Most of the studies have shown that the parasites of fish are more sensitive biomarkers to environmental stressors than the fish by themselves. Landsberg et al. (1998) related specific natural and chemical stressors to specific parasites in the silver perch in Florida. Skinner (1982) showed that the gills of the yellow fin mojarra (*Gerres cinereus*), grey snapper, and timucu (*Strongylura timucu*) from a polluted but not a non-polluted area in Biscayne Bay, Florida, expressed excessive mucus production, epithelial hyperplasia, fused lamellae, and telangiectasia. Three species of monogenoids on the gills of fish from the polluted area occurred in significantly greater mean intensity, presumably because of gill pathology and altered host resistance to the parasites. Pech et al. (2009)

examined the effects PAHs and other chemicals on the parasites of the checkered puffer (*Spheroides testudineus*) in Yucatán lagoons. Vidal-Martínez et al. (2003) restricted one Mexican study to metazoan parasites of the Mayan catfish in Chetumal Bay; their most significant finding was that DDT concentration affected the presence of the trematode *Mesostephanus appendiculatoides* more than the PAHs. Another study showed that parasites from the pink shrimp (*Farfantepenaeus duorarum*) also responded to the chemical contamination in Campeche Sound, Mexico (Vidal-Martínez et al. 2003). Khan (1990), who studied the effects of the Exxon Valdez oil spill, determined the oil affected the presence and intensity of infections of internal parasites. More parasites occurred in the sparid *Boops boops* in Spain after the Pestige oil spill than before, indicating that different intermediate hosts became established after the spill (Pérez-del Olmo et al. 2007). Sures (2004) investigated the sensitivity of various parasites in accumulating heavy metals, and many accumulated more than the fish host. Experimental work with exposures of known PAHs to investigate the induced lesions and effects on parasites has revealed good parasite indicators. When fishes were exposed to oil for a lengthy period and then depurated in oil-free water, the fishes, with gills expressing hyperplasia, demonstrated an increase in the prevalence and intensity of both trichodinids and monogenoids (e.g., Khan and Kiceniuk 1984, 1988; Khan 1990). Water-soluble fractions of crude oil seemed to have a more toxic effect on internal helminths than oil-contaminated sediments; the prevalence and intensity of infections in both types of exposure were less than in reference controls (Khan and Kiceniuk 1983). Sediments contaminated with PAHs and PCBs were also exposed to fish and determined to have an effect on their parasites (Marcogliese et al. 1998; Moles and Wade 2001). Data collected from wild fish, especially when compared with laboratory studies, become even more significant when additional data on bioaccumulation and biomarkers such as molecular, immunological, endocrine, histological, anatomical, and others can be used in conjunction with parasite data (e.g., Van der Oost et al. 2003; Monserrat et al. 2007).

Histopathological information also provides good biomarkers for contamination because both specific and general lesions reflect specific contamination. Meyers and Hendricks (1982) summarize the literature and lesions in experimentally exposed aquatic animals caused by PAHs, PCBs, heavy metals, and numerous other compounds. Solangi and Overstreet (1982) describe lesions resulting from exposure of whole crude oil and water-soluble fractions to the tidewater silverside and hogchoker; they also showed recovery from the lesions when the oil exposure was removed. Misdiagnoses are common in the literature, and future studies can benefit from studying the review by Wolf et al. (2015).

14.4.3 Biodiversity

The role of biodiversity is not a category of disease, and biodiversity can be considered a tool to evaluate catastrophic events treated above in Section 14.4.2. We treat it separately because of its relationship with health. Since parasites comprise about half the Earth's biota, they form a critical component of biodiversity of the Gulf. An abundance of parasites, with an emphasis on helminths, indicates a healthy ecosystem or a healthy host species. This method provides an especially powerful marker because most helminths have three, plus or minus two hosts, in a specific cycle. Consequently, the presence of that specific helminth in a habitat indicates that all members of the cycle are or had recently been present in the habitat.

Because of this cycle, the absence of helminths provides indicators of disruption. The reason for the indication is that adverse impacts on the corresponding intermediate host (or final host) for the species or the population result in fewer species or smaller parasite populations. Fewer parasite species or parasite populations show there was a disruption, even if it was not otherwise apparent.

Table 14.2. Numbers of Helminth Species and Their Hosts Reported from the Gulf of Mexico Based On Felder and Camp (2009).

Vertebrate Host Helminth Group	No. Potential Host Species	No. of Reported Hosts	No. of Reported Species in 2007	No. of Reported Species in 1954	No. Listed as Endemic, 2007	No. Listed as Endemic, 1954
Chondrichthyes (Elasmobranchs)	128					
Trematodes (adult)		4	3	0	3	–
Cestodes (adult and metacestodes)		27	65	37	–	
Acanthocephalan		3	1			
Actinopterygii (bony fishes)	1,409				–	
Trematodes		347	371	198	130	164
Cestodes (adult and metacestodes)		128	41	13	–	
Acanthocephalans (adult)		67	16	7	5	
Reptiles	9					
Trematodes		8	40	15	6	7
Aves	395				–	
Trematodes		57	122	3	38	2
Acanthocephalans			16		3	
Marine mammals	31					
Trematodes		6	9	0	2	–
Cestodes (adult and metacestodes)		4	4	0	–	
Acanthocephalans		1	1		0	
Totals	1,972	652	689	273	187	173

Some protozoans (actually, Protozoa constitutes several independent phyla) have complicated life cycles with multiple hosts and can also provide indications of ecosystem or host health. Unlike the adult helminths, except for the Monogenoidea that can reproduce on their hosts, individual protozoans can produce offspring in or on the fish host. The protozoans such as coccidians, myxosporidians, microsporidians, ciliates, and others as well as monogenoids can replicate in or on the host. Consequently, a disruption in the system can stress the host or otherwise make it more susceptible to excessive replication resulting in a prolific increase in parasite numbers, a harmed host, and an indication of a disrupted and unhealthy ecosystem.

We will use biodiversity of parasites both as individuals and as communities to provide exemplary information on healthy and harmed model systems. The associated biota also goes through seasonal and long-term alterations, and these alterations can also be modified in detectable ways by both anthropogenic and natural environmental events.

Understanding biodiversity of parasites and their hosts in the Gulf of Mexico is accompanied by numerous problems. Table 14.2 helps us start understanding some of those problems regarding helminths. A large volume on Gulf biodiversity by Felder and Camp (2009) provides

checklists of all named species of most animal taxa reported by the various authorities of the different taxa. The interesting aspect of the volume is that it updates the checklists occurring in an earlier bulletin listing species known at that time, also by experts in their fields (Galtsoff 1954). Not all parasite taxa were listed in either volume, but the table lists data on three helminth groups, including described adult trematodes (flukes) by Overstreet et al. (2009), adult and some undescribed metacestodes (tapeworms) by Jensen (2009), and acanthocephalans (spiny headed worms) by Salgado-Maldonado and Amin (2009). Nevertheless, the baseline of known parasitic biota is increasing, but assessments can be difficult in some cases because some major groups were not included, not all species in those three groups are known or described, and not all specific or general life cycles for listed species have been determined.

Defining the parasite fauna from the Gulf of Mexico may be difficult because some fish definitive or intermediate host species swim into and out of freshwater, and bird hosts fly to and from coastal or marine areas from adjacent freshwater or from localities other than the Gulf of Mexico. Probably, the best way to define a trematode as a Gulf species would be to determine if its molluscan host was a Gulf resident; however, even that restriction creates confusion because seasonal and yearly dynamics of the infection involve salinity and because details of the life history are usually lacking (the molluscan host(s) of many trematodes have not been discovered) (Overstreet et al. 2009). Also, some mollusks tolerate or thrive in low salinity water with as little as 1–2 ppt, yet the fish or bird host might spend most of its life in high salinity waters.

Many of the fish and parasite populations have a strong Caribbean influence as do the birds, but not many Caribbean Gulf collections have actually been made. Consequently, the checklists of Overstreet et al. (2009) include species extending slightly outside the designated Gulf (Felder and Camp 2009) up through Biscayne Bay on the Atlantic side of Florida and those located off Cancún and Cozumel (slightly south of the Gulf border of Cabo Catoche, Yucatán, Mexico, as will those off Havana, Cuba) as indicated in the introduction. As more fish and birds from the northern Gulf of Mexico as well as elsewhere in the Gulf are examined, they surely will be found to be infected with new and unreported species. Consequently, it is important to include all fauna indicated above to best understand the fauna of the Gulf of Mexico.

Assessing information from Table 14.2, we see that there were totals of 1,541 fishes and 395 birds presently described and reported from the Gulf of Mexico. Of those hosts reported to have trematodes, and not all did, there were 351 (23 %) of the fishes and 57 (14 %) of the birds. The interesting thing about this is that in 1954 there were only 198 fishes and 3 birds that were reported to be infected with adult trematodes.

If one looks at the adult trematodes listed from fishes as being endemic, there were 164 species (83 %) reported in 1954 and 133 (36 %) in 2007. The reason for this decrease reflects the increase in the number of fishes and localities examined, the improvement in identifications, and recent recognition of many species being widespread. The values for all listed adult trematodes from all definitive hosts provide similar data, 186/577 is 32 % in 2007 compared with 173/216 = 80 % as indicated by Manter (1954). Since 1954, numerous new records of fish trematode species have been reported, including those from deepwater fishes and other hosts. However, since only 23 % of possible fish hosts have been examined, and those were infected with 1.1 trematode species per fish, we predict there may be an additional thousand adult trematodes to be discovered.

Reasons for the relatively few fish species examined include the very high cost to collect fish from offshore waters, the difficulty to obtain good quality fresh specimens from the fish when aboard vessels or even on land, and the paucity of taxonomists to identify and describe the parasites. Up to now, the fish that have been examined for trematodes and other parasites have been examined from few geographical areas during a single season and in small numbers

by authorities that live and work near the collection sites, primarily in Texas, Mississippi, and Florida.

In addition to trematodes from hosts not yet examined, there are many trematodes (and cestodes) that look superficially like other species, making them hard to identify. These cryptic species are now easier to identify using molecular methods. An example is a complex of species that are reported in the older literature as or close to *Homalometron pallidum* (Trematoda: Apocreadiidae) from a wide range in North America and infecting several hosts. As it turns out, numerous species exist in the complex. In the northern Gulf, the common species is *Homalometron palmeri*, which infects at least four sciaenid fishes, two fundulids, and a gerreid. It is sympatric with *Homalometron manteri* in another sciaenid and appears similar to *Homalometron pseudopallidum* in Argentina (Curran et al. 2013a). On the other hand, the actual *H. pallidum* occurs in fundulids in New England (Curran et al. 2013a). There also occur a few freshwater species from fundulids not inhabiting the southeast, but they are being described and they appear similar to *H. pallidum*. In freshwater in Mississippi and Louisiana, there are two cryptic species similar to *Homalometron armatum*, which occur northward to Lake Erie and Ontario, Canada, as well as in Mississippi, and can be separated by molecular means but with unreliable morphological differentiation (Curran et al. 2013b). A similar Gulf and Caribbean species, *Homalometron elongatum*, infecting a gerreid and appearing more elongated than *H. pallidum* and also possessing three pairs of relatively large opposing oral papillae projecting from near the mouth, makes it distinguishable from *H. pallidum* and another species by both molecular and minor morphological differences. That species, *Homalometron lesliorum*, also infects a gerreid but occurs in Costa Rica and Nicaragua on the Pacific Ocean side (Parker et al. 2010).

Similar appearing complexes also occur for nematodes (Fagerholm et al. 1996; and others) and for cestodes (Jensen 2009; Caira and Healy 2004; Caira et al. 2001; and others). Right now the published library of gene sequences involving trematodes and cestodes is relatively small, but it is gradually expanding and will be extremely helpful in identifying species in the future, thereby creating a more realistic and usable baseline. Once molecular means are used more routinely to compare similar or identical specimens from the Gulf of Mexico, southeastern coasts of the United States, Western Caribbean Sea, and the Pacific Ocean adjacent to the Panama Canal, several of the identifications will be found to be wrong, and several that are suspected to be incorrect will be found to be correct. All in all, we expect that the number of actual species will be considerably more than the presently reported number.

On the other hand, many of the trematodes and cestodes found in birds will be found to be species acquired in the northern or southern ranges of their migratory patterns and do not truly represent Gulf of Mexico species. To make the matter more confusing, the migratory pattern of many seabirds is inadequate, incorrect, or not known at all. Moreover, few cestodes have been reported from true Gulf seabirds (Hoberg 1996; Hoberg and Klassen 2002) and Jensen (2009) did not include birds in her review of Gulf cestodes. Moreover, the lack of knowledge about all seabird parasites in the Gulf makes understanding the history, ecology, and biogeography in marine systems difficult.

In the case of marine mammals and marine turtles from the central Gulf of Mexico, few parasites have been reported. Many of the animals that have been examined are those that get sick and migrate to shore or nearshore habitats to recuperate or die, and the records of their parasites actually reflect records of transient species rather than true Gulf residents with parasites originating in the Gulf. On the other hand, ill fishes and birds usually get eaten before reaching coastlines. To examine stranded or dead marine mammals and marine turtles for parasites and diseases, a researcher requires a Federal permit, which historically has been difficult to obtain. Moreover, most obscure species are unavailable for examination until

several days after death of the host. Also, because of the migratory behaviors of many of the mammals and turtles, few of the parasites and diseases originate in or are endemic to the Gulf of Mexico. Also, every few years, ocean currents shift for a short period of time allowing pelagic fishes and other animals to locate near areas they seldom occupy and where few animals get periodically examined for parasites or diseases. Once again, this can provide misleading data on Gulf of Mexico parasites and diseases.

Some nematodes can survive a few days in a dead marine mammal or at least be identified, whereas trematodes and cestodes are much more difficult to find and usually rapidly degenerate after the death of their host. Consequently, recent molecular tools are more likely to detect more nematodes. Moreover, more nematodes, trematodes, cestodes, and other parasites that have complex life cycles will be listed when identified based on sequence data (when a library is or becomes available) based on larval and juvenile stages.

The Atlantic croaker (*Micropogonias undulatus*) from coastal Mississippi has been periodically examined for trophically transmitted parasites over the last 40 years. These parasites may serve as bioindicators of biodiversity, food web structure, prey utilization by hosts, and environmental health. In this chapter, we restrict data to those of the camallanid nematode

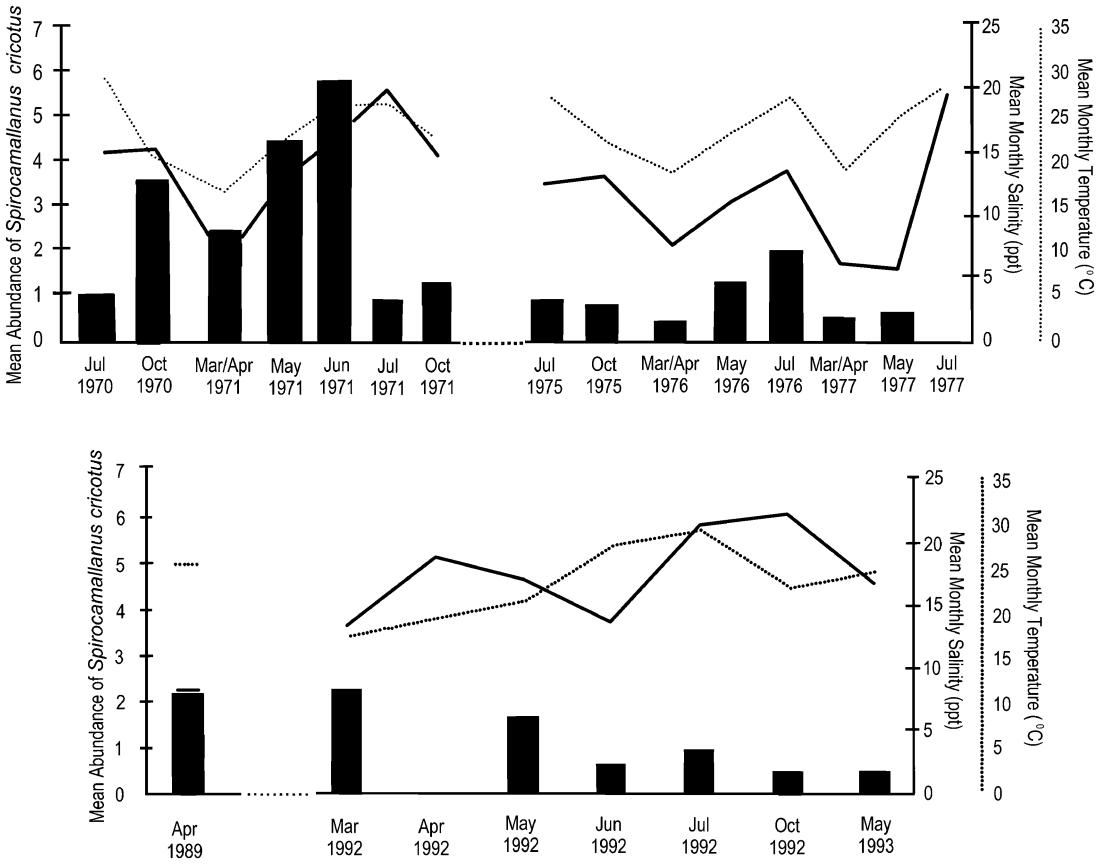

Figure 14.55. Mean abundance (number of individual nematodes recorded/number of individual fish examined) of the nematode *Spirocamallanus cricotus* from the Atlantic croaker, *Micropogonias undulatus,* from Ocean Springs, Mississippi, over several time periods of varying salinity and water temperature. The *solid line* represents salinity (ppt), and the *dotted line* represents temperature (°C). Graphs constructed by Andrew T. Claxton.

Spirocamallanus cricotus from relatively pristine Ocean Springs, Mississippi, to show historic patterns of annual and seasonal variation in abundance. This nematode is acquired by the croaker as well as numerous other fishes (Fusco and Overstreet 1978) when it feeds on copepods such as harpacticoids, the white shrimp (*Litopenaeus setiferus*) (see Fusco 1980), Atlantic brief squid (*Lolliguncula brevis*), and probably other hosts. Patterns of mean abundance, the number of the nematodes in all the croaker examined, whether infected or not, over time suggest the populations of this parasite exhibit extreme seasonal and annual variability, with some time periods exhibiting heavily infected croaker, whereas other periods exhibit few, if any, specimens of *S. cricotus* (Figure 14.55). Over the course of decades, both prevalence (% infected) and mean intensity of this parasite infecting croaker varied. In total, 1,307 croaker examined for this chapter had 2,193 individual *S. cricotus* from the 1970s and 1990s. In the early 1970s, prevalence of a sample reached as high as 74.6 %. In contrast, prevalence of *S. cricotus* in Atlantic croaker from the later 1970s was as low as 1.7 %. Low prevalence was also generally encountered during the 1990s when some collections, such as that in April of 1992, demonstrated no *S. cricotus*.

Fluctuations of *S. cricotus* over time do not appear to be strongly driven by either water temperature or salinity. The period of highest mean abundances occurred during the early 1970s (Figure 14.55). In subsequent years, this parasite was less abundant, even when environmental conditions were similar to the period when abundances were high. The lack of *S. cricotus* in later time periods suggests a decrease in either the presence or consumption of infected intermediate hosts by Atlantic croaker, different susceptibilities to infection, differences in host density, or different climatic conditions after the early 1970s. The potential mechanisms for this difference could be numerous; however, this lack of coherence, a reliable estimate of mean abundance under normal conditions, over time would hinder before-and-after comparisons of parasite population structure in relation to environmental disruptions (see review by Underwood 1994).

While the mechanism driving patterns of *S. cricotus* mean abundance in croaker remains unknown, the presence of a decline following the early 1970s was still apparent in the 1990s (Figure 14.55). In addition, abundance of *S. cricotus* often peaked within a time period during the early spring and summer months during the two periods in the 1970s and 1990s. The changes in abundance that occur on a seasonal basis would represent a further impediment to before–and–after comparisons of environmental disruption since this may necessarily involve comparing different seasons within a year. The decrease in *S. cricotus* over time is most clearly illustrated when comparing late spring and early summer months from the three time periods with one another and using that comparison to control for possible confounding seasonal effects. Even in cases where similar salinities occurred during the same months such as the spring and early summer months of the early 1970s and 1990s, mean abundance of *S. cricotus* was different, and that would suggest mechanisms besides those directly or indirectly related to either salinity or seasonality. In addition to abundance, prevalence and mean intensity also varied among time periods. Between March and June of 1971, prevalence ranged from 71 to 75 % with mean intensity varying from 3.3 to 7.2 worms/infected host. In the same months in 1976, prevalence was from 10 to 50 %, with mean intensity ranging from 2.6 to 3.9. In the 1990s, prevalence during the spring and early summer ranged between 0 and 44 %, with infected croaker harboring 3.2–3.7 worms/host. Thus, the shift in *S. cricotus* that is the result of changes in prevalence, intensity, and abundance suggests either changes in food web structure among and within time periods or physiological alterations in host immune response, but the pathway remains unresolved.

Currently, a study is in progress that will include more data that will allow for comparisons between polluted and non-polluted areas of coastal Mississippi, including the relatively

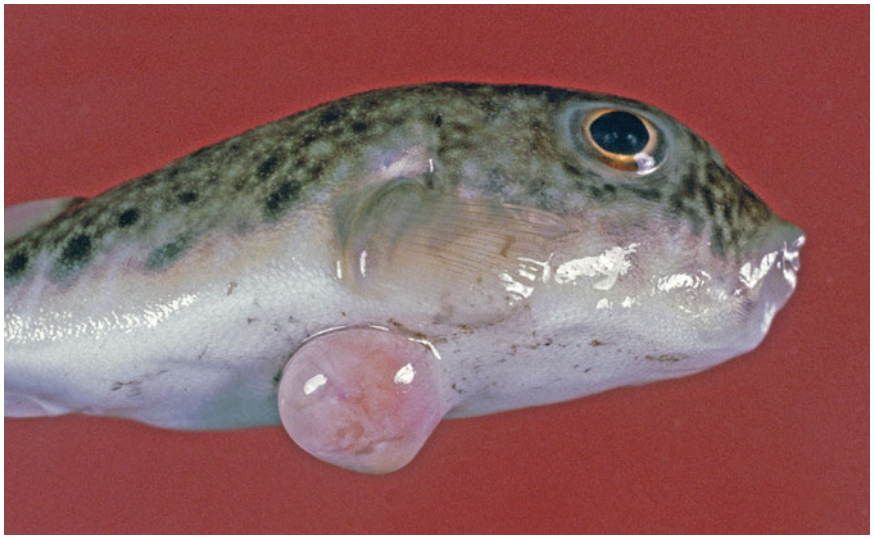

Figure 14.56. Least puffer, *Sphoeroides parvus*, exhibiting teratoma comprised of liver tissue, July 1981.

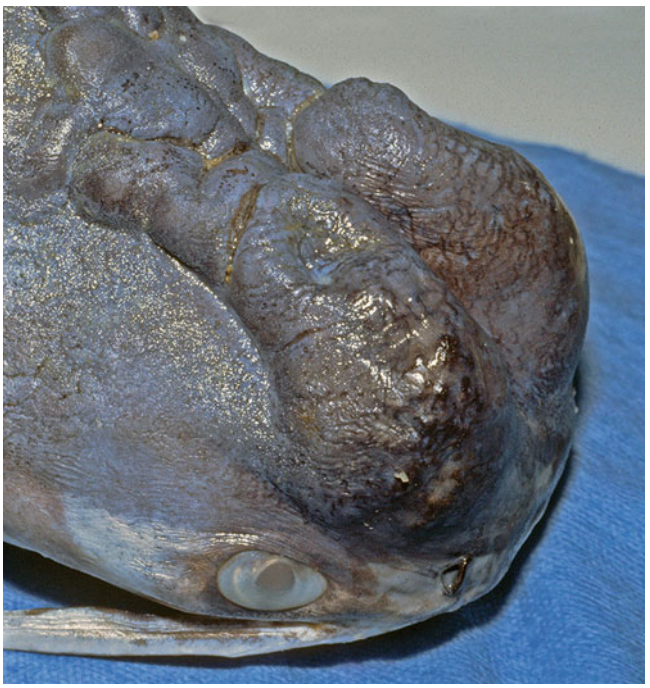

Figure 14.57. Gafftopsail catfish, *Bagre marinus,* head exhibiting disfiguring ossification, West Pascagoula River, Mississippi, June 1992.

non-polluted Ocean Springs locality. Those comparisons will examine both the effects of pollution on food web structure and whether or not the decrease in mean abundance over time is a local phenomenon.

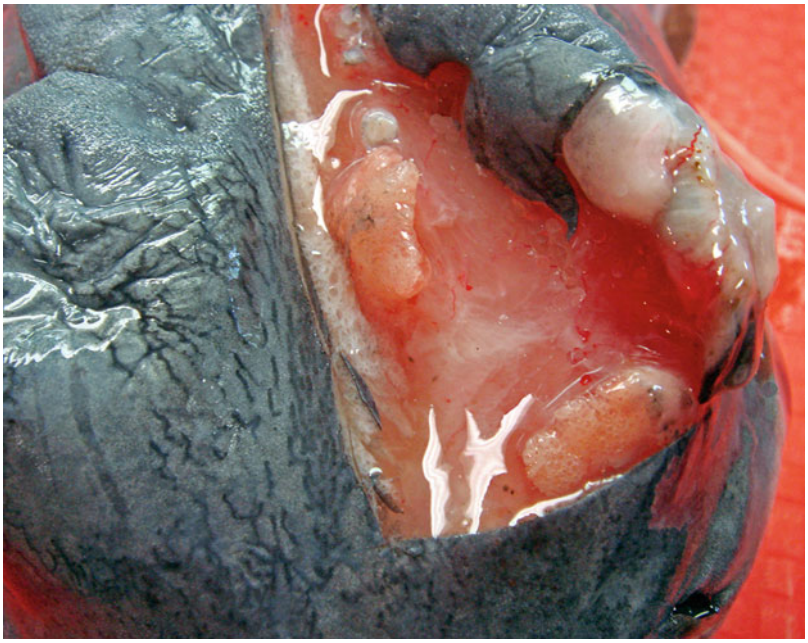

Figure 14.58. Gafftopsail catfish, *Bagre marinus,* head of different specimen exhibiting same disfiguring ossification, with head dissected to show *whitish* and *pinkish* "vacuolated" bony projections, off Horn Island, Mississippi.

14.4.4 Noninfectious Diseases and Conditions

14.4.4.1 Neoplasms

Terminology relating to cancer or cancerous conditions can be confusing because terms often overlap in meaning. For this report, we will use the term "neoplasm" which is defined as an abnormal growth of tissue that is not controlled by the surrounding tissue and continues to grow even after the stimulus that initiated it is removed (e.g., Groff 2004). A neoplasm can be a discrete structure such as a hepatocellular carcinoma or epidermal papilloma, or a neoplasm can be disseminated within tissues of the body as with a lymphoma, for example. The term "tumor" generally refers to a swelling (Figures 14.56, 14.57, and 14.58) and can be synonymous with a neoplasm but not necessarily. The term "cancer" implies malignancy, is a clinical term, and is probably best restricted to use when referring to higher animals and humans. The debate as to the role that environmental factors versus spontaneous genetic mutations or other factors such as viruses play in the initiation of neoplasms has not been fully settled, but it is well-known that environmental conditions, including life style choices such as smoking, poor diet, excessive sunlight, and certain workplace exposures might account for as many as two thirds of all cancers (Anonymous 2003); but it is clear that certain genetic traits make individuals more susceptible to exposure to environmental carcinogens (Perera 1997). These factors hold as well for fishes exposed to cancer-causing agents in the wild or the laboratory.

Perhaps no biologic condition in wildlife evokes concern as does the occurrence of clusters of neoplastic lesions particularly if the neoplasms turn out to be caused by environmental conditions or exposure to chemical carcinogens. In the last few decades, fish have been shown to be susceptible to developing neoplastic lesions from both environmental and genetic stimuli. Groff (2004) has reviewed neoplasia in fishes in general. Overviews by Harshbarger and Clark

(1990) and Harshbarger et al. (1993) concluded that epizootics (clusters) of neoplasms have occurred in fish from over 40 locations in North America. The neoplasms arose from a variety of cells and tissues including nervous, connective, reproductive, and digestive tissues as well as blood. With the exception of neoplasms involving the liver (hepatocellular and biliary adenomas and carcinomas) and skin (mainly epidermal papillomas), most of the neoplasms were unrelated to exposure to environmental conditions. Skin and liver neoplasms that occurred in 14 mostly bottom-dwelling fish species were strongly associated with exposure to PAH contaminants in sediments. Prominent among reports of contaminant-induced neoplasia in fishes from North American waters are liver neoplasms in English sole (*Parophrys vetulus*) from the Puget Sound (Myers et al. 1990), brown bullhead (*Ictalurus nebulosus*) from tributaries of Lake Erie (Baumann et al. 1990), and winter flounder (*Pseudopleuronectes americanus*) from Boston Harbor, Massachusetts (Murchelano and Wolke 1991). Both liver and epidermal neoplasms in white suckers (*Catostomus commersoni*) and brown bullhead from western waters of Lake Ontario were associated with chemical contaminants (Hayes et al. 1990). The reports above list bottom-dwelling species from cold water sites. Vogelbein et al. (1990) reported a high prevalence of liver neoplasm in mummichog (*Fundulus heteroclitus*) from a creosote-contaminated site in the Elizabeth River, Virginia, demonstrating that contaminant-induced liver neoplasia in fishes is not limited to bottom dwelling species from higher latitudes. Nevertheless, epizootics of hepatic neoplasia have not been reported from tropical or subtropical locations. Experimental studies have confirmed that environmental carcinogens such as the PAHs can cause hepatic neoplasia in laboratory-reared fish species (Hawkins et al. 1988, 1990; Fabacher et al. 1991). Furthermore, mechanisms by which chemicals cause carcinogenesis in fish relate closely, if not mirror, similar mechanisms in mammals at the organismic, tissue, cellular, and molecular levels (Ostrander and Rotchell 2005; Bailey et al. 1987; Ostrander et al. 2007).

Although clusters or epizootics of piscine neoplasms are rare in the Gulf and related systems, nerve sheath neoplasms in the bicolor damselfish (*Pomacentrus partitus*) from the Florida Keys are the best studied. They affect 5–10 % of the individuals in populations throughout the Caribbean (Schmale et al. 1983, 1986). The lesion has been considered as a

Figure 14.59. Striped mullet, *Mugil cephalus*, exhibiting a fibrosarcoma, Mississippi Sound, September 1979.

model for neurofibromatosis (von Recklinghausen's disease) in humans. Subsequently, it was found that the disease was transmissible from tumor-bearing to non-tumor-bearing specimens through tissue homogenates (Schmale and Hensley 1988) and that a virus-like agent, probably a retrovirus (Schmale et al. 1996), was responsible for the disease in damselfish (Schmale et al. 2002). Lucke (1942) reported similar nerve sheath neoplasms at a prevalence of 0.5–1.0 % in three snapper species from the Dry Tortugas near Key West. No etiologic factor for these tumors has been identified.

Otherwise, reports of neoplastic lesions in fishes from the Gulf of Mexico are of single or low numbers of cases from a variety of species and none have been clearly linked to exposure to any kind of carcinogen. Single case reports or those involving a small number of cases include squamous cell carcinoma in gulf menhaden (*Brevoortia patronus*) by Fournie et al. (1987), capillary hemangiomas in a scamp (*Mycteroperca phenax*) (Fournie et al. 1985), a hepatocellular neoplasm in a wild-caught sheepshead minnow (*Cyprinodon variegatus*) (Oliveira et al. 1994), several cases of subcutaneous fibrosarcomas (fibromatoses or fibromas) in striped mullet (*Mugil cephalus*) (Edwards and Overstreet 1976; Overstreet 1988) (note more severe neoplasms in September 1979; Figure 14.59), and subcutaneous fibromas in southern flounder (*Paralichthys lethostigma*) and the sea catfish (*Arias felis*) (Overstreet and Edwards 1976). Overstreet (1983b) illustrated an epidermal papilloma in a red drum (*Sciaenops ocellatus*). McCain et al. (1996) found hepatic neoplasms (adenomas) and preneoplastic hepatic lesions (basophilic, eosinophilic, and clear cell foci) in several specimens of hardhead catfish from chemically contaminated areas of Tampa Bay.

Few broadly based sampling programs have been conducted in the GoM using biomarkers of fish health as an indicator of the condition of the environment from which the fish were collected. The most robust and comprehensive sampling program was the USEPA's Environmental Monitoring and Assessment Program (EMAP) that examined tens of thousands of fish

Table 14.3. RTLA Tumor Specimens in Fishes Affiliated with the Gulf of Mexico.

Diagnosis	Scientific and (Common) Name	RTLA No.
Adenocarcinoma stomach	*Ocyurus chysurus* (yellowtail snapper)	7673, 7674
	Unknown serranid	7670
Adenocarcinoma stomach; Carcinoma in situ stomach	*Ocyurus chysurus* (yellowtail snapper)	7677
Angiosarcoma	**Kryptolebias marmoratus* (mangrove rivulus)	6136-7, 6139-6146, 6148-56
Carcinoma in situ stomach	*Epinephelus morio* (red grouper)	7671
	Ocyurus chysurus (yellowtail snapper)	7672, 7676
Cholangiocarcinoma	*Seriola* sp. (type of amberjack)	3820
Chondrofibroma	*Ariopsis felis* (hardhead catfish)	1113
Chondroma	*Squalus acanthias* (spiny dogfish)	3144
Dermal fibrosarcoma	*Brevoortia gunteri* (finescale menhaden)	1848
Epidermal papilloma	*Lepisosteus platostomus* (shortnose gar)	3913
	Mugil cephalus (striped mullet)	5469
	Sciaenops ocellatus (red drum)	1904
Esthesioneuroblastoma of the lateral line	*Cyprinodon variegatus* (sheepshead minnow)	3102

(continued)

Table 14.3. (continued)

Diagnosis	Scientific and (Common) Name	RTLA No.
Fibrolipoma	*Pogonias cromis* (black drum)	1662
Fibroma	*Eugerres plumieri* (striped mojarra)	678
	Lagodon rhomboides (pinfish)	3626
	Mugil cephalus (striped mullet)	807, 821
	Paralichthys lethostigma (southern flounder)	1112
	Seriola sp. (type of amberjack)	3628
Follicular cell carcinoma thyroid	*Ocyurus chysurus* (yellowtail snapper)	7664
Ganglioneuroblastoma	*Pomatomus saltatrix* (bluefish)	5241
Hemangioendothelioma	*Mycteroperca phenax* (scamp)	3179
Hemangiopericytoma	*Cyprinodon variegatus* (sheepshead minnow)	3808, 3809
Hepatocellular carcinoma	**Kryptolebias marmoratus* (mangrove rivulus)	2348, 2430-1, 2434-8, 3390, 5446-7
Hepatocytic adenoma	*Bagre marinus* (gafftopsail catfish)	5528
Iridophoroma; neurilimmoma	*Lutjanus apodus* (schoolmaster)	2289
Leiomyoma	*Mugil cephalus* (striped mullet)	5470
Lipoma	*Amia calva* (bowfin)	6397, 6399
	Brevoortia gunteri (finescale menhaden)	1664
	Eugerres (Diapterus) plumieri (striped mojarra)	596
	Paralichthys dentatus (summer flounder)	710
	Seriola sp. (type of amberjack)	4885
Mixed germ cell-sex cord stromal tumor	*Rachycentron canadum* (cobia)	7754
Myxoma	*Mugil cephalus* (striped mullet)	3131
Neurilemmal sarcoma	*Lutjanus griseus* (gray snapper)	1481
Neurilemmoma	Snapper (unidentified)	1378
Neurofibroma	*Lutjanus griseus* (gray snapper)	3892
	Stegastes partitus (bicolor damselfish)	3177
Neurofibrosarcoma	*Stegastes partitus* (bicolor damselfish)	3176
Ocular chondrosarcoma	**Kryptolebias marmoratus* (mangrove rivulus)	3973-4
Ossifying fibroma	*Caranx hippos* (crevalle jack)	1233
Rectal adenocarcinoma	*Balistes vetula* (queen triggerfish)	6435
Reticulum cell sarcoma (spleen)	*Carcharhinus plumbeus* (sandbar shark)	523
Schwannoma	*Mugil cephalus* (striped mullet)	3963
Squamous cell carcinoma	*Brevoortia patronus* (Gulf menhaden)	3618
Thyroid (?) carcinoma	*Abudefduf saxatilis* (sergeant major)	5918

* indicates that the *Kryptolebias marmoratus* (mangrove rivulus) possibly is from experimental laboratory studies. Some of these data were provided by Jeffrey C. Wolf, DVM, DACVP, Chief Scientific Officer, Manager of Virginia Pathology.

from estuarine locations along the coasts of the Gulf (see Summers 1999). The EMAP program examined over 64,000 fish specimens from the Louisianan Province from 1991 to 1994 for gross abnormalities, including tumors and lesions on the skin, malformations of the eye, gill abnormalities, and parasites. Total gross pathologies were seen in 408 specimens for an overall incidence of 0.6 %. Parasites accounted for 61 % of all gross pathologies. Nevertheless, there was a positive correlation between the occurrence of gross pathologies and sediment-contaminant concentrations (Fournie et al. 1996).

Although not a sampling program per se, the Registry for Tumors in Lower Animals (RTLA) served the environmental and comparative pathology community well for many years (Harshbarger 1977). Supported by the National Cancer Institute and housed in the Smithsonian Institution in Washington, DC, the registry for the most part depended on independent contributions from scientists and lay persons all over the world and provided diagnostic services on the accessions. Perusal of nearly 8,000 records of specimen accessions by the RTLA (Harshbarger 1965–1981 and Jeff Wolf personal communication) yielded around 75 cases of tumors in fishes from the Gulf and nearby waters or in species known to inhabit Gulf waters (Table 14.3).

Overall, neoplastic lesions have rarely been reported from fishes from the Gulf. Furthermore, to our knowledge, no epizootics of chemically induced tumors comparable to those reported above have occurred in wild fishes from the Gulf. Overstreet (1988) reviewed the occurrence of neoplasms and related histopathological conditions in fishes from the coasts of the southeastern United States, particularly the Gulf of Mexico, and found scattered examples of neoplastic lesions in individual fish species but no epizootic of neoplasia. The paucity of reports of neoplasms in Gulf fishes might be related to several factors. These include the fact that the Gulf is a rather large body of water to study, especially when compared with water bodies in other regions, and except for a few locations, it is relatively free of industrial pollution. Probably most importantly, however, the Gulf has not been studied as intensively as some other North American aquatic systems. Not all fish species are equally susceptible to chemically induced neoplasms (Hawkins et al. 1985) and susceptibility depends on habitat preferences with bottom dwelling species more susceptible than pelagic species. Most importantly, in the case of PAH exposure, the ability of the fish to convert the compounds to carcinogenic intermediates determines in large part their susceptibility to develop neoplasia (Ostrander et al. 2007). Clearly there are fish species in the Gulf that are susceptible to chemically induced carcinogenesis. For example, Atlantic croaker has been shown to be capable of metabolizing polynuclear aromatic hydrocarbons to their carcinogenic intermediates (Willett et al. 2009). Also, indigenous Gulf species including sheepshead minnow (*Cyprinodon variegatus*), Gulf killifish (*Fundulus grandis*), inland silverside (*Menidia beryllina*), and mangrove rivulus (*Kryptolebias marmoratus*) all developed liver tumors following exposure to the direct acting carcinogen methylazoxymethanol acetate (Hawkins et al. 1985). To date, the only chemically induced epizootic of neoplasia in a fish from a warm water system remains the case of mummichog (*Fundulus heteroclitus*) exposed to creosote residues in Virginia (Vogelbein et al. 1990). Nevertheless, it is likely that geographically broadly based, multi-species, sampling programs focused on examining specimens from contaminated sites will yield neoplasm prevalences in line with other well-studied systems.

14.4.4.2 Developmental Abnormalities

Abnormalities can be the result of genetics, environmental conditions, biological conditions, anthropogenic activities, and other causes. Manipulation of fish regarding reproductive activities to produce offspring utilized in aquaculture, research, and display provides good

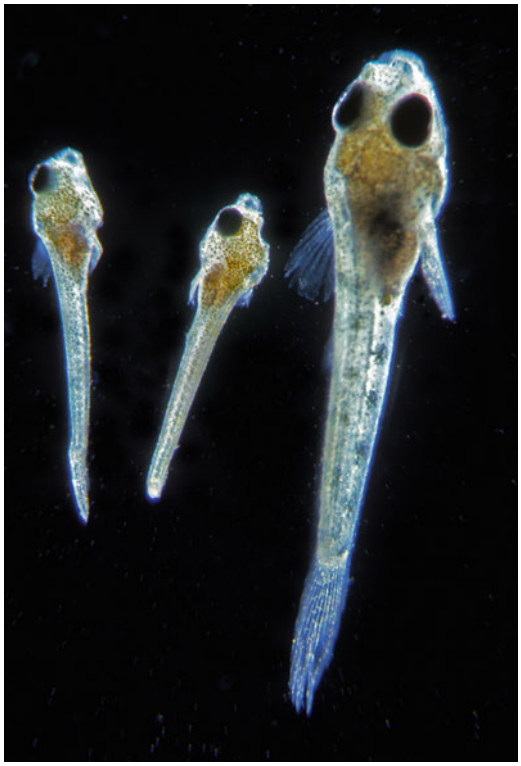

Figure 14.60. Sheepshead minnow, *Cyprinodon variegatus*, examples of abnormal 6-day-old fish resulting from experimental stripped mating; offspring from natural mating under experimental conditions seldom produce abnormal fish.

Figure 14.61. Striped mullet, *Mugil cephalus*, with atretic and fibrotic ovaries that had undergone hydration but not spawning and consequently underwent atresia after cold snap, December 1988.

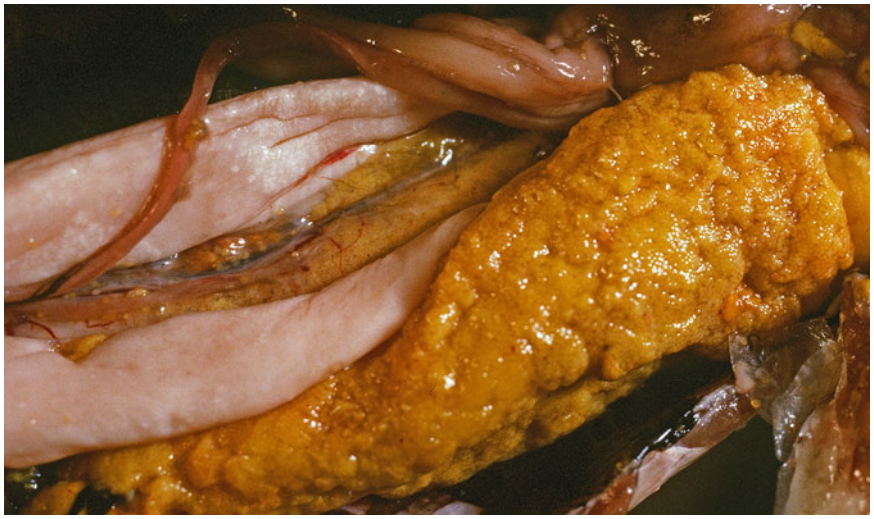

Figure 14.62. Striped mullet, *Mugil cephalus*, abnormal hermaphroditic specimen from Mississippi Sound showing pinkish testes and orangish ovaries, December 1997.

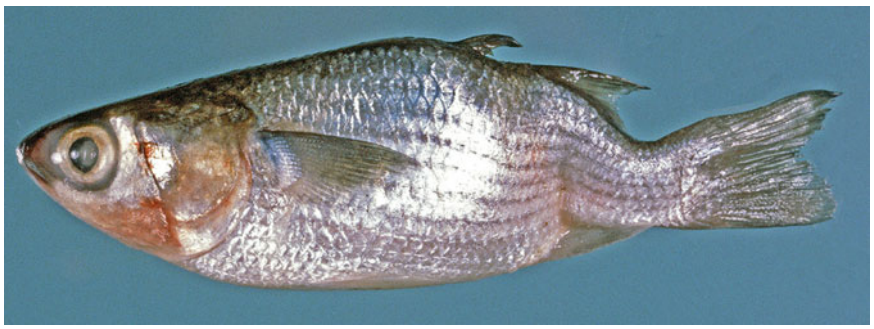

Figure 14.63. Striped mullet, *Mugil cephalus*, exhibiting scoliosis and lordosis, Mississippi Sound, June 1981.

examples of abnormalities. Figure 14.60 demonstrates different-sized fish 6 days after eggs and sperm have been artificially brought together; similar abnormalities can result from suboptimal temperatures and salinities. Under normal conditions involving light-dark cycles, nutrition, and temperature, ova become hydrated, deposited, and then fertilized. Figure 14.61 shows the two ovaries containing an abundance of ova that became hydrated but not deposited, presumably because temperature or other conditions inhibited that process. After a short period, the hydrated ova hardened and started to undergo atresia, the process of their degeneration and that of the ovarian follicle. Rather than being soft and pliable, each ovary was hard and crunchy. Figure 14.62 demonstrates the gonads of a hermaphroditic striped mullet. The presence of both ovary and testes can occur naturally in some species of fish simultaneously or one at a time. This is not the case with the striped mullet, and such a condition can be a genetic abnormality or induced by a specific contaminant. When an affected individual occurs, the cause is most likely genetic. When a significant proportion of the population exhibits hermaphroditism, the cause may result from a group of chemicals in the habitat known as endocrine-disrupting compounds, such as steroids, hormones like estrogen, and some detergents and pesticides.

Figure 14.64. Spanish mackerel, *Scomberomorus maculatus*, from off barrier islands in Mississippi exhibiting a rare case of scoliosis and lordosis, November 1981.

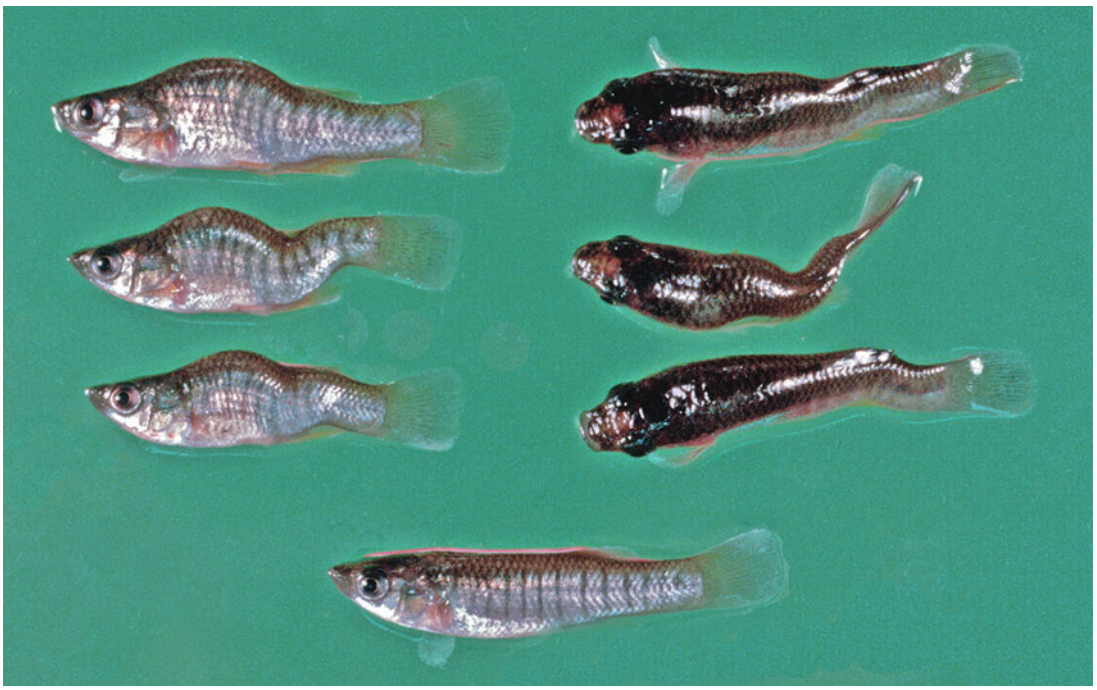

Figure 14.65. Series of longnose killifish, *Fundulus similis*, from Mississippi Sound, showing scoliosis and lordosis, July 1981.

Three main types of spinal column abnormalities occur in fish: scoliosis (lateral deformity, zig-zag shape), lordosis (dorsal deformity, V-shape, or loss of normal curvature of the lower or posterior spine), and kyphosis (ventral deformity, inverse V-shape, front to back deformity, or hunched back); consequently, some authors prefer to join lordosis and kyphosis together as lordosis, referring to dorso-ventral deformity or as scoliosis when combining all three abnormalities.

Figures 14.63 and 14.64 show a combination of scoliosis and at least lordosis in the striped mullet and Spanish mackerel. The mackerel abnormality represents a unique case, but we have

Figure 14.66. Horse-eye jack, *Caranx latus*, with abnormal partial double lateral line, off Mississippi barrier islands, November 1971.

Figure 14.67. Southern flounder, *Paralichthys lethostigma*, exhibiting partial albinism on eyed dorsal side, Bayou Caddy, Bay St. Louis, Mississippi, August 16, 1993.

seen several such distorted mullet over previous years (e.g., Overstreet 1978). Figure 14.65 exhibits a variety of skeletal abnormalities in the longnose killifish. Afonso et al. (2000) discuss causes of skeletal abnormalities involving genetics, development, and environmental situations. Such cases are relatively common in aquaculture and when rearing fish for research. As an example, when we (Overstreet et al. 2000) reared small fish for carcinogenicity studies, scoliosis and other abnormalities ultimately became obvious in several older specimens unless the fry were fed an adequate diet of algae, ciliates, or immature nematodes during their initial three or so days of culture.

Abnormalities of the lateral line system in fish seem to be rather unusual. A previously unreported case of a partial double lateral line in the horse-eye jack was seen once only (Figure 14.66).

Figure 14.68. Hogchoker, *Trinectes maculatus*, from Mississippi Sound showing ambicolorate pigment pattern, blind side.

Figure 14.69. Hogchoker, *Trinectes maculatus*, from Mississippi Sound, also showing abnormal pigmentation pattern on the eyed side of same fish as in Figure 14.68.

Flatfishes in coastal and estuarine waters less than 5 m (16 ft) in depth represent good models for developmental (typically reversal) and pigmentation (typically albinism and ambicoloration) abnormalities (Figure 14.67), and the foundations for discussing them were established by Norman (1934) and Gudger (1934). During development of the normal flatfish fry, one eye rotates to the opposite side so that both eyes end up on a predetermined side. Moreover, pigmentation that responds to light and background colors develops on the upper, eyed side, leaving the blindside pale. This behavior and the ability to burrow in the substrate allow flatfishes to avoid most predators from both above and below them. Abnormal cues involving light and temperature (Gartner 1986) during early development result in skeletal (primarily head and fin) and pigmentation abnormalities. Dawson (1962, 1967, 1969), Moore and Posey (1974), and Gartner (1986) described and reviewed many abnormalities in the hogchoker and other

Figure 14.70. Atlantic croaker, *Micropogonias undulatus*, exhibiting pugnose abnormality, Mississippi Sound, November 1979.

Figure 14.71. Atlantic croaker, *Micropogonias undulatus*, with top specimen exhibiting abnormal micro-eye condition, Mississippi Sound, September 1979.

Figure 14.72. Hardhead catfish, Ariopsis felis, conjoined twins from mouth of male brooding fish, Mississippi Sound.

flatfishes, and these hogchoker abnormalities occur more commonly (Figures 14.68 and 14.69) than similar abnormalities in most other flatfishes in the Gulf. Overstreet (1978) figured and discussed a rare case of reversal of a fringed flounder (*Etropus crossotus*). Partial albinism of the southern flounder is shown in Figure 14.67, and ambicoloration, or pigmentation on the blindside of the hogchoker shown in Figure 14.68, with abnormal pigmentation also on the eyed-side of the same individual (Figure 14.69).

Dawson (1964, 1966, 1971) and Dawson and Heal (1976) provide a series of very useful bibliographies of anomalies of fishes; each includes an index for fishes and a separate index for anomalies, allowing a reader to find nearly all abnormalities reported before 1976. Most of those from the Gulf are rare. Some examples of abnormalities include the pugnose condition in the Atlantic croaker (Figure 14.70) that also occurs in other sciaenid fishes such as the spotted seatrout in the Gulf (Overstreet 1983a). Another involving the Atlantic croaker is the lack of an eye or a micro-eye (Figure 14.71), a condition also reported for the red drum in Texas (Overstreet 1983b). The red drum from Texas is also known to exhibit scale disorientation (Gunter 1948), a common abnormality in pinfish in Biscayne Bay, Florida. Figure 14.72 shows conjoined twins still containing a yolk sac when taken from the mouth of a wild male brooding hardhead catfish. Both of these twins and those of a Japanese medaka obtained in culture (Overstreet et al. 2000) were maintained alive for a few weeks in a culture dish. More than likely, such twins would become easy prey if not carefully protected.

Rubber and plastic trash can also encircle or otherwise harm fish as well as birds, marine mammals, and seabirds. An Atlantic croaker apparently swam through a rubber band and ultimately grew around it (Overstreet 1978; Overstreet and Lyles 1974), similar to a situation where mackerel became ringed with condoms occurring near sewage effluents. Sharks also become encircled in plastic packing straps (e.g., Overstreet 1978). Because of the serious problem with trash in the past, there has been a recent attempt not to contaminate the seas.

Abnormalities also constitute good indicators of polluted environments. The best investigated area for this chapter consists of sites within Biscayne Bay, Florida. Skinner and

Kandrashoff (1988) observed over 10,000 fishes within 45 species caught by gill nets throughout the Bay over a 10-year period from 1970 to 1982. They found the most heavily-affected species were the Western Atlantic seabream (*Archosargus rhomboidalis*) with skin hemorrhaging and scale disorientation; yellow mojarra (*Gerres cinereus*) with fin erosion and eye abnormalities; Florida pompano (*Trachinotus carolinus*) with emaciation; and pinfish (*Lagodon rhomboides*) with scale disorientation. The striped mullet and Atlantic croaker were caught in significant enough numbers in 1973 and 1974 to compare prevalence of fin and skin hemorrhaging. Such bacterial infections affected all individuals in 26 of 43 collections of the mullet and more than half of the individuals in the remaining 17 collections; all individuals of the croaker in 24 of 30 collections were affected, with more than half of the fish in the remaining six collections affected. From the same general area in 1991 and 1992, Gassman et al. (1994) caught 3,650 fish of over 60 species by hook and line, but with 70 % of those belonging to one of four target species, the seabream, blue striped grunt (*Haemulon sciurus*), pinfish, and gray snapper (*Lutjanus griseus*). Missing or deformed dorsal fin rays were the most common abnormalities in the snapper (4.6 %), scale disorientation in pinfish (7.3 %), and both in the seabream (3.0 % and 3.8 %, respectively). The prevalence of these abnormal maladies was correlated with the concentration of total and aromatic hydrocarbons in sediment samples from locations within 2 km (1.2 mi) of the survey sites, but not with sediment concentrations of aliphatic hydrocarbons, polychlorinated biphenyls, or heavy metals. The grunt had a low-frequency of a variety of abnormalities, but they appear to be associated with sediment copper levels. Most of the abnormalities occurred in two locations in the more contaminated northern part of the Bay. Skin and fin hemorrhaging and eye abnormalities were seldom observed in this study as they were in that by Skinner and Kandrashoff (1988), but this difference may result from method of collection or an increase in water quality. A similar study to that by Gassman et al. (1994) conducted from November 1989 to June 1990 by Browder et al. (1993) emphasized a depression in the dorsal profile, known as "saddleback," and accounted for 76 % of all the abnormalities that they observed. A study by Corrales et al. (2000) focused on scale disorientation in pinfish from the same area. This abnormality consisting of discrete patches of scales rotated dorsally or ventrally away from the normal scale position was also reported by Overstreet (1988). Corrales et al. (2000) also found the abnormality, affecting as much as 34 % of the body surface, most prevalent in the northern part of the Bay in the pinfish, on which they also conducted experimental studies. Acute and chronic exposure to physical traumas was insufficient to induce formation of the disorientation; however, the condition could appear spontaneously in some normal juvenile and adult specimens maintained in the laboratory for 5.5 months. Their observations suggest that development occurs rapidly and is most likely the result of a sudden change in growth characteristics of cells in the affected area.

Abnormalities in fish from Biscayne Bay, Florida, even in areas of high input of sewage and urban runoff, occurred considerably less than those in fish caused by hydrocarbon contamination in the Hudson River estuary (Smith et al. 1979) and Puget Sound, Washington (Malins et al. 1984).

Clearly, recording lesions and abnormalities in fish provides good indicators of environmental health, but relating specific abnormalities to specific contaminants is usually difficult. We (Sun et al. 2009) examined hybrid tilapia from six stations from four rivers in southern Taiwan during spring and autumn from 1994 through 1996. All stations were contaminated from different non-point sources; it is important to point out that the areas were so extensively polluted that few fish other than tilapia inhabited the rivers, and consequently those locations were quite different from those with an abundance of species occurring in the less-polluted Biscayne Bay, Florida. Nevertheless, tilapia-complex provided a useful sentinel. Therefore, examples of deformities provide this dramatic difference. Contamination was derived from

agriculture, industry, and domestic wastes, and specific contaminants were recorded although specific ones could not be related to specific deformities of which we noted 20 different categories. In the Kao-Ping River, scale disorientation occurred in 18 versus 2 % of the fish in the autumn of 1994 and 1995, respectively, compared with 8 and 10 % of the fish affected with disoriented scales in the spring periods. This could be compared with 33 % of the fish in the Tongkong River in the autumn of 1995 when 23 % of the fish had a bent jaw; none of the fish at that time in the Kao-Ping River had such a deformity. The percentage of fish with an opaque cornea declined from 55 to 0 % between autumn 1995 and spring 1996, and 12 % of the fish in autumn had exophthalmia. But none was blind as were a few fish in the other rivers. Never did skeletal deformities determined with radiographs occur in more than 7 % of the samples from any river during any season. The percentage of fish with frayed fins in autumn (57 %) contrasted with 27 % of those in the spring from the Kao-Ping River compared with 37 versus 1 % in the Tongkong River. Autumn was the rainy season with increased river flow and suspended sediments.

Another example from freshwater is provided because of the large number of fish examined and the abundance of abnormalities (Slooff 1982). It treats the bream (*Abramis brama*) in the Rhine River and its branches running into the North Sea. Nearly 7,000 fish were divided into males and females, examined for skeletal anomalies, and example prevalence values for specific locations were 22.7 % with deformed fins, 3.0 % with pugheadedness, 1.2 % with lack of fins or girdle, 0.7 % with spinal curvature, 0.9 % with asymmetric cranium, 1.5 % with shortened operculae, and 5.9 % with fusion of vertebrae. The prevalence of deformed fins and pugheadedness in both males and females increased in the 12 years of fish life. As with other studies, specific abnormalities could not be attributed to specific contaminants.

Figure 14.73. Green turtle (*Chelonia mydas*) from Florida Keys exhibiting fibropapillomatosis, especially in tissue around neck, near anus, and in anterior flippers; note especially on soft tissue ventral to pelvic girdle and on flippers the abundance of attached turtle barnacle, *Chelonibia testudinaria*, which has a morphological form specific to marine turtles but has recently been shown to be molecularly the same as *Chelonibia patula* on carapace of the blue crab and many other hosts (Cheang et al. 2013).

14.5 OTHER VERTEBRATE REPRESENTATIVES

14.5.1 Sea Turtles

Sea turtles have numerous parasites and diseases. Overstreet et al. (2009) reported the trematodes from the Gulf, and Herbst (1999) provided a review of all the infectious diseases. For purposes of this chapter, we will restrict ourselves to the single example of tumorous growths.

Fibropapillomatosis (FP) (Figure 14.73), tumorous growth, of marine turtles affects primarily the green turtle (*Chelonia mydas*), but has also been reported from other turtles such as loggerhead (*Caretta caretta*), olive ridley (*Lepidochelys olivacea*), hawksbill (*Eretmochelys imbricata*), leatherback (*Dermochelys coriacea*), and flatback (*Natator depressus*) (Huerta et al. 2002). Foley et al. (2005) examined data collected by the U.S. Sea Turtle Stranding and Salvage Network based on 4,328 dead or debilitated green turtles in the eastern half of the United States from Massachusetts to Texas from 1980 to 1998 and found that 22.6 % (682/3,016) of the turtles in the southern half of Florida had tumors. During that period, the percentage of turtles in southern Florida with tumors progressively increased from about 8 to over 30 %. Most of these were in the GoM, with 39 % in inshore areas and 15 % in offshore areas. Most cases were found in coastal waters characterized by habitat degradation and pollution, a large extent of shallow water area, and low wave energy during fall and winter months, and the occurrence of tumors occurred mostly in the intermediate-sized (48–70 cm [19–27 in] curved carapace length) animals. Many were emaciated or tangled in fishing line, but they showed about an equal percentage of attack by sharks as those without tumors. Historical data reported by Smith and Coates (1938) showed that in the early 1930s, less than 2 % of green turtles captured in southern Florida exhibited tumors compared with later prevalences as high as 92 % (Herbst 1994). Fibropapillomatosis spread to elsewhere in the Gulf of Mexico, Caribbean Sea, and the western Atlantic by the mid-1980s, occurring in 10 % of the stranded green turtles in the early 1980s and increasing to 30 % in the late 1990s for those found below the 29°N latitude. The presence of FP in stranded green turtles found in Florida increased at the rate of 1.2 % per year from 1980 to 1998 (Foley et al. 2005).

The size, location, and number of tumors contribute to progressive debilitation and eventual death. Tumors, ranging from 0.1 to greater than 30 cm (12 in), are typically observed externally in the inguinal and axillary regions, at the base of the tail, around the neck, in the mouth, and on the conjunctiva of the eye (Smith and Coates 1938). Gross lesions as large as 20 cm (8 in) in diameter occur internally in the lungs, kidney, heart, gastrointestinal tract, and liver (Herbst 1994).

With an increase in the number of studies on FP, a causative agent including at least the chelonid fibropapilloma-associated herpesvirus (CFPHV) (Family *Herpesviridae*, Subfamily *Alphaherpesvirinae*, proposed genus *Chelonivirus*) was determined (Stacy et al. 2008; Davidson 2010; Bicknese et al. 2010). It has also been classified as Chelonid herpesvirus 5, also restricted to marine turtles (Lu et al. 2003) in which fibropapillomas, fibromas, lung-eye-trachea disease, grey patch disease, and loggerhead genital-respiratory and orocutaneous diseases have been reported. Development of these herpesvirus infections can be acute, latent and quiescent, or appear as a disease causing highly pathogenic and life-threatening conditions to occur (Aguirre et al. 1998, 2002; Herbst 1994; Quackenbush et al. 1998, 2001; Stacy et al. 2008; Ariel 2011; Alfaro-Núñez et al. 2014). In Florida, four distinct viral variants of CFPHV have been described (A–D) (Ene et al. 2005). In the Gulf of Mexico, *C. mydas*, *C. caretta*, and *L. kempii* all have the C variant, with the A variant, the most commonly

detected one, being present in both the green and loggerhead turtle populations. The D variant was found only in the loggerhead.

Green turtle (*Chelonia mydas*) fibropapillomatosis with or without mortality has been associated with herpesvirus, retrovirus, natural tumor-promoter okadaic acid, arginine, external parasites, trematode egg interaction, and environmental factors demonstrating that these combinations of conditions can be pathogenic and life-threatening (Aguirre et al. 1998; Herbst 1994; Casey et al. 1997; Landsberg et al. 1999; Dailey and Morris 1995; Alfaro-Núñez et al. 2014; Foley et al. 2005; Ene et al. 2005; Work et al. 2004; Van Houtan et al. 2010). There does not seem to be any single factor inducing infections or causing mortality. Work et al. (2001) concluded that turtles with severe FP were immunosuppressed, but immunosuppression was not a prerequisite for development of FP and neither were trace metals nor organic contaminants (Aguirre et al. 1994). Induction of FP by herpesviruses seems to be promoted by a metabolic influx of the amino acid arginine; lysine inhibits the virus and proline aids the viral infection. Moreover, eutrophication spurs nuisance algal blooms where arginine would be elevated, consequently promoting the FP tumors in *C. mydas*. Okadaic acid also promotes tumors and is produced by toxic benthic dinoflagellates in the genus *Prorocentrum*, which are fed on heavily by *C. mydas* exhibiting FP in Hawaii (Landsberg et al. 1999).

There appears to be an interesting relationship of FP with blood flukes (in the Gulf, at least two different described species of Spirorchiidae infect *C. mydas*, and three infect *C. caretta* (e.g., Overstreet et al. 2009)). Adult flukes and their eggs were initially assumed to be related to FP in relatively large specimens of *C. mydas*, but later considered not to be the immediate cause of the disease. In the mid 1960s, R. Overstreet removed tumors from many caged 20 cm (8 in) and larger specimens of *C. mydas* maintained by R.E. Schroeder in Marathon, Florida Keys, Florida, and saw some spirorchiid eggs in larger turtles, but all the tumors contained considerable cyanobacteria, diatoms, and other algae appearing to cause some cellular response. After one or more viruses had been implicated in the etiology of FP, Aguirre et al. (1998) studied the relationship between blood fluke infections and fibropapillomatosis in *C. mydas* in Hawaii. A generalized thickening and hardening of major vessels (aortic, pulmonary, mesenteric, and hepatic) and thrombosis with complete or partial occlusion occurred in turtles containing both FP and spirorchiidiasis and were considered primary causes of mortality. Similar pathogenesis has been reported from both wild and cultured sea turtles worldwide (e.g., Aguirre et al. 1998). Chen et al. (2012) provided several references on fluke infections in *C. mydas* associated with mortality, but they considered the mortality in the stranded juvenile turtles, apparently without FP, in Taiwan probably from fishery by-catch rather than the fluke-associated pathogenic alterations. When a large number of young *C. mydas* exhibiting a robust nutritional condition became moribund or died from a single hypothermic event in Florida, Stacy et al. (2010) took advantage of them to assess the pathological responses to spirorchiid infections. They determined that the responses differed relative to trematode species, species of turtle (*C. mydas* and *C. caretta*), and size of turtle host. Even though some turtles exhibited severe pathological alterations, only one specimen of *C. mydas* died from a worm infection even though infections probably contributed to poor health in others. In a report of Australian strandings in which size of turtles was not given, blood flukes caused death in 10 of 96 and contributed to death in 29 more of the 96 (Gordon et al. 1998). In those, many were chronically ill, whereas in Florida, examined turtles died from a variety of acute insults. An apparent undescribed quite pathogenic species of *Neospirorchis* sp. was most common as adult worms and associated egg masses in large *C. mydas* and *C. caretta* and infected the leptomenenges, thymic gland, and thyroid gland rather than heart and major arteries like *Neospirorchis pricei* and the other blood flukes. In general, adults of most species produce proliferative endarteritis, with parasitic granulomas and thrombosis inhibiting blood flow; eggs typically become trapped in capillaries,

including those of the highly vascularized fibropapillomas, and sometimes associated with inflammation. The important point is that spirorchiids seldom cause acute infections with death, but they are often associated with mortalities when in combination with other diseases like FP. Stacy et al. (2010) also reported *C. mydas* (7 of 15) with anemia to have a severe leech infestation including egg cases involved with FP, presumably *Ozobranchus margoi*, which is discussed by Sawyer et al. (1975) as a leech occurring externally on sea turtles and harboring up to 900 individuals with lesions associated with their attachment sites. Three of those 15 turtles had the talitroidean amphipod *Hyachelia tortugae* in the skin and fibropapillomas.

The protection and population recovery of sea turtles on a global scale has had increasing attention during the past 35 years (Raustiala 1997; Wright and Mohanty 2006; Campbell 2007; Hamann et al. 2010). One of the global research priorities for marine turtles is still the etiology and epidemiology of the pandemic FP and the management of this disease (Hamann et al. 2010). Long-term studies have already shown that the disease has peaked in some regions, remained constant in others, and increased elsewhere (Van Houtan et al. 2010).

14.5.2 Birds

Birds need to adopt a strategy optimizing the use of energy for activities like reproduction and host defense. This strategy requires a "trade off" of physiological choices for both the host and pathogenic agents to maintain genetic fitness. For example, during one season or one year, a bird or group of birds may be in poor nutritional health and therefore have to direct all resources to staying alive with little or no ability to mount a defense against parasites or to grow or to reproduce. During another time of year, this same bird may have enough resources to effectively resist parasites, grow, and reproduce (Wobeser 2008). An example of a heavy infection of helminths in a group of 45 lesser scaup ducks comprised almost one million individuals, including 52 different species (Bush and Holmes 1986). Each helminth species has a life cycle with multiple hosts, making infections problematic to fully understand without knowing the complete cycle. Additionally, chicks may become infected with numerous large adult nematodes from regurgitation by parent birds. Fagerholm et al. (1996) suggest this based on observations of patent specimens of *Contracaecum magnipapillatum* in adults, chicks, and dead chicks in the breeding habitat of the black noddy (*Anous minutus*). Interactions among two or more species might be additive, synergistic, or antagonistic, resulting in host mortalities, and little is known about these or even effects of high numbers of single species in wild birds. Even though studies have focused on a few dead birds killed by parasites (see Atkinson et al. 2008), rarely do parasitic infections result in "piles of dead birds" because highly pathogenic ones tend not to impact a host population, since rapid mortalities would limit transmission to others. Populations are more detrimentally affected by the sublethal effects of chronic infections mediating reduced fecundity (Hudson and Dobson 1997b). One should also expect true seabirds, those that derive all food from the sea, defecate in the sea, and die at sea, to suffer less than coastal birds from microbial, protozoan, and probably helminth agents (Lauckner 1985).

Table 14.4 lists avian mortalities in the Gulf from 1999 to 2010, and most cases result from bacterial, fungal, and viral infections as well as toxicants. The book edited by Thomas et al. (2007) explains the agents in detail. Tropical Storm Arlene moved through the Gulf into Breton National Wildlife Refuge in June 2005 at a time when birds were vulnerable as reflected in the table. Vargo et al. (2006) summarized beached bird surveys in Pinellas County, Florida, mostly reporting mortality resulting from brevetoxin. The harmful effects of red tides on birds were discussed earlier, including an informative report by Forrester et al. (1977).

Table 14.4. Epizootics for Birds in the Northern Gulf of Mexico from 1999 to 2010 as Reported by the National Wildlife Health Center, Quarterly Mortality Reports[a].

State	Locality	Dates	Species	Number of Mortalities (e = estimate)	Cause of Death
FL	Peace River, Charlotte Harbor	01/08/99–01/14/99	Lesser scaup	50(e)	Hepatitis
TX	Colorado, Frio, Matagorda, Waller Co.	11/26/99–02/01/00	Snow goose, white-fronted goose	3,189	Avian cholera
TX	Waller Co.	01/09/00–01/31/00	Wood duck, American coot, mottled duck, green-winged teal, gadwall	291	Avian cholera
TX	Laguna Atascosa NWR	01/10/00–02/10/00	Snow goose, green-winged teal, American avocet, sandhill crane	200(e)	Open
AL	Baldwin Co., Gulf Shores	07/01/00–12/30/00	Unidentified pelican, common loon, double-crested cormorant, unidentified gull, northern gannet	100(e)	Open
TX	San Bernard NWR	11/20/00–12/01/00	Snow goose	75(e)	Open
FL	Monroe Co., Florida Keys	12/31/00–05/17/01	Brown pelican, common loon, great blue heron	250(e)	Open
TX	Nueces Co., Gulf Beach	01/28/01–03/15/01	Double-crested cormorant	100(e)	Salmonellosis
FL	Lee Co., Gasparilla Is	10/30/01–12/30/01	American white pelican	20(e)	Toxicosis: brevetoxin
LA	Offshore Louisiana	01/05/02–01/10/02	Brown pelican	50(e)	Exposure: Hypothermia
AL	Gulf Shores State Park	02/25/02–03/10/02	Unidentified loon, brown pelican, herring gull, mallard, northern gannet	20(e)	Aspergillosis
TX	Willacy Co.	07/23/02–08/01/02	Black-bellied whistling duck, eared grebe	80(e)	Toxicosis: salt
TX	Cameron Co., Harlingen	08/19/02–08/22/02	Laughing gull	32	Salmonellosis
FL	Okaloosa Co., Destin Harbor	02/01/02–07/10/02	Brown pelican, common loon, osprey, American white pelican, wood duck	60(e)	Open

(continued)

Table 14.4. (continued)

State	Locality	Dates	Species	Number of Mortalities (e = estimate)	Cause of Death
TX	Aransas NWR	12/01/03–12/05/03	Snow goose	80(e)	Open
FL	Volusia, Orange, Brevard, Martin, Palm Beach, and Broward Counties	03/08/03–04/15/03	Northern gannet, unidentified cormorant	2,500(e)	Emaciation
LA	Jefferson Parish	01/07/04–01/15/04	Eastern brown pelican	50(e)	Open
FL	Manatee Co.	07/01/04–07/31/04	Wood stork; white ibis; great blue heron; roseate spoonbill; unidentified pelican	24(e)	Open; toxicosis suspect
FL	Pinellas Co.	05/11/04–06/12/04	Mallard; muscovy; American coot; unidentified cormorant	70(e)	Botulism suspect
LA	Breton NWR	06/12/05–06/23/05	laughing gull, brown pelican, ring-billed gull, little blue heron	7,200(e)	Trauma: storm toxicosis: Oil
FL	Panama City	09/26/05–11/15/05	American coot	30	Toxicosis: suspect
FL	North Miami Beach	09/04/06–10/01/06	Muscovy, unidentified egret, NOS heron, white ibis, tricolored heron	48	Botulism suspect
FL	Key West	02/04/07–02/15/07	Unidentified seabird, brown pelican	40(e)	Toxicosis: domoic acid (red tide) suspect
TX	Galveston County Beaches, Aransas, and Nueces Counties	06/07/07–06/30/07	Northern gannet	100(e)	Emaciation: Starvation suspect
FL	St. Marks NWR	04/04/08–04/11/08	Common loon, red-breasted merganser	100(e)	Undetermined

[a]http://www.nwhc.usgs.gov/publications/quarterly_reports/index.jsp, accessed February 10, 2015.

Figure 14.74 shows a few specimens of the chewing louse *Piagetiella peralis* in the gular pouch of the American white pelican associated with a combination of louse excrement and blood located next to petechial hemorrhaging. Heavy infestations commonly cause severe ulcerating lesions covering much of the naked body of pelicans <1 week old before the lice enter the throat of older juveniles. Because of these heavy infestations and associated

Figure 14.74. A few of the many pouch lice, *Piagetiella peralis,* infesting the gular pouch of an American white pelican.

Figure 14.75. Chick of nesting least tern, *Sternula antillarum*, died of hyperthermia (heat stroke) along beach in Gulfport, Mississippi, July 1980.

Figure 14.76. The brown pelican, *Pelecanus occidentalis*, with a malformed bill; the bird had an excessive infestation of the chewing lice *Colpocephalum unciferum and Pectinopygus tordoffi* on the feathers because of its lack of ability to successfully preen, September 1993.

secondary bacterial infections, Samuel et al. (1982) suggested that the louse can have a significant effect on juvenile pelican populations.

Based on histopathological evidence in June 1980, nearly all of several hundred nesting least terns (*Sternula antillarum*) died of hyperthermia (Figure 14.75) along a narrow beach in Gulfport, Mississippi (Overstreet and Rehak 1982). The unusual heat-stroke apparently occurred because of the temperature-humidity complex arising from delayed hatching because of 39 cm (15.4 in) of rain in mid-May and because the nearby waters had low salinity, resulting in extended time for parents having to leave their nests while foraging for the bay anchovy. This fish species comprises a major dietary item of the tern and its near-nest population was small compared with most years.

Abnormalities have been seen in birds, and the best example is that of the brown pelican that had its bill so malformed that it depended on viscera from fish tossed to it by fishermen cleaning their fish in an Ocean Springs, Mississippi, harbor (Figure 14.76). Because the bird could not preen, its feathers contained an enormous number of lice. When a seagull on the end of the GCRL pier was ill, one could slowly approach it and pick it up, only to have many lice migrate up his or her arms.

14.5.3 Marine Mammals

Every few years, mortalities of marine mammals have occurred in the Gulf of Mexico. To examine dead or dying stranding animals, one required a permit or special permission and such was seldom obtained before the animal decomposed too much to be evaluated. A few cases of single stranded animals appeared to result from an ectopic parasite. Trematodes and nematodes or their eggs have been found in the brains of these animals along the Pacific coast, and, in the Gulf during the 1980s, we observed in stranded bottlenose dolphins lung infections of metastrongyle nematodes *Halocercus lagenorhynchi* and *Skrjabinalius cryptocephalus* associated with pneumonia. Those findings compared favorably with those reported by Fauquier et al. (2009). Dailey et al. (1991) and the latter article additionally found *H. lagenorhynchi* lungworms in neonates, suggesting this nematode crosses the placenta of pregnant females. Because of the lack of pinnipeds in the Gulf, we do not see fish with larval nematodes of

Figure 14.77. Flipper of stranded common bottlenose dolphin, *Tursiops truncatus*, exhibiting at least two tooth rake marks acquired during aggressive interactions between dolphins, 1997.

Figure 14.78. Stranded oceanic spotted dolphin, *Stenella frontalis*, March 15, 1993, not commonly seen stranded on Mississippi beaches; most strandings on beaches in the northern Gulf are the bottlenose dolphin.

species that mature in pinnipeds, but we do see larvae in offshore fish that mature in dolphins and other cetaceans. Because of the ability to examine the bottlenose dolphin (*Tursiops truncatus*), we use it as an example of marine mammal in the Gulf. Most dolphin exhibit external lesions. Individual bottlenose dolphin can be identified by wounds, tooth rake marks,

secondary infections, and even symbiotic barnacles as figured by Overstreet (1978) and Figure 14.77. Other species of dolphin strand along Gulf beaches, but they are rare as exemplified by the stranded oceanic spotted dolphin, *Stenella frontalis,* identified from a Mississippi beach by the number of teeth (Figure 14.78).

To assess microorganisms of the bottlenose dolphin as a component of biodiversity as well as potential dolphin health and public health risks, Buck et al. (2006) reported aerobic microorganisms associated with free-ranging bottlenose dolphins in coastal waters off Florida, Texas, and North Carolina during 1990–2002. We examined blowhole and fecal samples of some of those and other dolphins for similar purposes. We also examined microorganisms from captive dolphins in Mississippi before and after Hurricane Katrina and discovered many of the same organisms reported by Buck et al. (2006) and by Williams and Barker (2001).

Evaluations of mass mortalities of bottlenose dolphin since 1990 have tentatively established that brevetoxin and morbillivirus have been at least partially responsible (Waring et al. 2007). From January through May 1990, a total of 367 bottlenose dolphin stranded in the northern Gulf of Mexico, but the cause was not established. In March and April 1992, 111 stranded in Texas, and some of these animals tested positive for previous exposure to cetacean morbillivirus. The NOAA Fisheries Working Group on Unusual Marine Mortality Events was formalized in 1992 and has evaluated several mortality events. Morbillivirus was diagnosed on the basis of histopathologic lesions, immunohistochemical chemical demonstrations of the morbilliviral antigen, and detection of morbillivirus RNA by RT-PCR in 35 of 67 stranded dolphins that occurred in the Florida Panhandle and spread west to Alabama and Mississippi, with most of the dolphins dying in Texas between 1993 and 1994 (Lipscomb et al. 1996); 29 additional dolphins exhibited advance postmortem autolysis and diagnosis was equivocal. That was a follow-up study of one (Lipscomb et al. 1994) reporting morbillivirus from a 1987–1988 epizootic from stranded Atlantic Coast bottlenose dolphins that was the first such report outside Europe. Mortalities from other types of events included dolphins in Mississippi in 1996, 120 in the Florida Panhandle between August 1999 and February 2000, and 107 from the same location in March and April 2004; all these dolphin died concurrent with red tide blooms and red tide fish kills and were assumed to be killed by brevetoxin. The West Indian manatee (*Trichechus manatus*) also succumbs to brevetoxin as indicated earlier in the section on red tides.

Figure 14.79. White shrimp, *Litopenaeus setiferus,* with the microsporidian *Agmasoma penaei* in the cephalothorax and along the dorsum, superficially appearing like developing gonads.

Figure 14.80. The microsporidian *Perezia nelsoni* in the skeletal musculature of the tail (abdomen) of the lower shrimp (brown shrimp, *Farfantepenaeus aztecus*) causing cotton shrimp compared with an uninfected white shrimp on top, February 1977.

Figure 14.81. A microsporidian infection (cotton shrimp) in top shrimp tail; about four species of microsporidians cause this condition in the Gulf, with *Perezia nelson* and *Tuzetia weidneri* being the most common; both tails come from the white shrimp, *Litopenaeus setiferus,* July 1977.

14.6 INVERTEBRATES

We treat invertebrates with essentially the same approach as for fishes, but we use representative-selected examples from penaeid shrimps, the blue crab, the eastern oyster, and corals. Each group is presented separately.

14.6.1 Shrimps

Like most invertebrates, shrimps, notably penaeid shrimps, succumb to a variety of diseases and other agents. Because penaeids are cultured commercially worldwide, considerable data on shrimp health have been published (e.g., Overstreet 1973, 1983c, 1987; Lightner 1996; Sindermann and Lightner 1988; Lotz and Overstreet 1990; Overstreet and Lotz 2016). For purposes of this chapter, we will discuss select ciliates, microsporidians, and viruses as examples of pathogenic organisms.

About five or six microsporidian species infect penaeids in the Gulf, and individuals with infections in the abdominal (tail) muscle are colloquially called cotton shrimp or milk shrimp (Canning et al. 2002), sometimes including *Agmasoma penaei* and sometimes not (Figures 14.79, 14.80, and 14.81). Microsporidia is a phylum of unicellular spore-forming parasites now recognized as related to fungi. *Agmasoma penaei*, primarily restricted to the white shrimp, *Litopenaeus setiferus,* does not infect striated muscles but rather the muscles lining blood vessels, foregut, hindgut, and germinal tissue of the gonads. It can occur in multiple infections in a single host (Overstreet 1973). Until recently, it has been difficult to critically separate species (Canning et al. 2002; Sokolova et al. 2015). In any event, historical data sometimes separate them and sometimes combine all species, and, in some cases, microsporidians infect a large portion of the shrimp populations. The same species occur in the South Carolina estuaries, where Miglarese and Shealy (1974) examined a total of 67,658 white shrimp on a monthly basis and found an increase in prevalence from about 15.0 % in July up to a peak of 89.5 % in November 1973. In 1919, about 90 % of the white shrimp along the Louisiana coast had their gonads destroyed by *A. penaei*; however, the largest known white shrimp crops during that general period were produced in 1920 and 1921 (Viosca 1945). Viosca (1945) stressed that evidence showed that with a prolific species like the white shrimp, the food supply and other ecological factors represented more important factors for production than the actual number of eggs laid. The most severe epizootic of this microsporidian in the Gulf of Mexico was recorded in 1929, resulting in the prevalence of 90 %, mass mortality of this shrimp, loss of 99 % of the shrimp egg production, and, in contrast with the 1919 event, an unprofitable white shrimp fishery for several following years (Gunter 1967; Muncy 1984). Lightner (1996) indicated

Figure 14.82. Penaeid shrimp showing bacterial infection in abdominal musculature.

Figure 14.83. White shrimp, *Litopenaeus setiferus,* exhibiting heavy infestation of fouling peritrich ciliate and detritus; this condition appears superficially similar to black gill disease caused by two apostomes ciliates and occasionally seen in an abundance of shrimp from Louisiana and Texas, September 1979.

the prevalence in wild populations normally did not exceed 1 %, and whereas we do not disagree, we have observed prevalence values as high as 25 % in inshore white shrimp from Mississippi and Louisiana on about three occasions during the last 45 years. Perhaps, lactic acid buildup in infected shrimp keep them from migrating offshore, similar to what was suggested for the blue crab infected with *Ameson michaelis* (see Shields and Overstreet 2007). Unlike the lack of a known lifecycle for other shrimp microsporidians from the Gulf, the cycle for *A. penaei* has been achieved experimentally. Iverson and Kelley (1976) fed pink shrimp (*Farfantepenaeus duorarum*) infected with *A. penaei* to spotted seatrout and then fed the seatrout feces to uninfected hatchery-reared postlarvae (about 1 cm [0.39 in] long) pink shrimp for 3–5 weeks. When examined grossly, no sign of infection was apparent, but histological sections demonstrated spores or other signs of infection.

Penaeid shrimps worldwide exhibit infections with more than 25 species of viruses, and most of these infections are in cultured shrimp. Lightner (2011) provided a mini-review of all the viruses in the Americas, and Lightner et al. (2012) reviewed the history of all shrimp pathogens in the Americas (Figure 14.82). The first known virus, *Baculovirus penaei*, occurs in the natural environment (Overstreet 1994; Overstreet and Lotz 2016) and provides a good contrast to the introduced viruses. Overstreet and Lotz (2016) indicated how both *B. penaei* and the viruses introduced into the Gulf can revert from being relatively harmless to the shrimp population to becoming highly pathogenic.

Black gill disease causes concern in the shrimp fishery in the Gulf and along the Atlantic coast. Actually, the disease is a syndrome because blackish or brownish gills can be caused by a variety of agents and conditions. These include several chemical irritants such as cadmium, copper, crude oil, and ammonia; microbial agents such as the virus IHHNV, bacteria *Vibrio* spp., fungi *Fusarium* sp.; ciliates such as peritrichs, apostomes, and scuticociliates; ascorbic acid deficiency; and other causes. We have seen several epidemics of apostomes, and infected shrimp with melanistic responses from Texas, Louisiana, and Mississippi were so conspicuous that the commercial product was not acceptable for the market. The conspicuous shrimp gills contained *Hyalophysa chattoni* and *Gymnodinioides inkystans*, even though other species occurred. *Hyalophysa chattoni* was the most common but least pathogenic. Moreover, it also infected grass shrimp in which no melanistic response occurred. Figure 14.83 shows a brownish version of black spot, and that shrimp was infested with attached colonial peritrichs. The effects of this disease on the population have not been established but probably depend on the stress allowing the ciliates to invade and establish on the gills. Stocks of heavily infested shrimp

Figure 14.84. "Golden shrimp" an abnormally discolored white shrimp on top with normal specimen of white shrimp underneath, Mississippi Sound, May 1985.

Figure 14.85. The palaemonid river shrimp *Macrobrachium ohione* comes down the river to encounter salt water for its larvae. Specimens can take on a variety of colors, so this "golden river shrimp" from the Pascagoula River may not be portraying a genetic phenotype as we attribute to the golden penaeid.

in aquaculture died from oxygen-depletion probably because the ciliates competed with the shrimp for oxygen (Overstreet 1973).

Other "protozoans" not normally infecting penaeids have the ability to influence the seafood market. For example, an aseptate gregarine infected *Litopenaeus vannamei* in the commercial "seed-production" facility in Texas and caused considerable economic loss until a presumed lifecycle of the coccidian could be established (Jones et al. 1994).

As with most animals, abnormalities in shrimps become apparent as more individuals are examined. Overstreet and Van Devender (1978) observed a hamartoma (non-neoplastic growth) primarily in postlarval brown and white shrimps that occurred near a harbor in Ocean Springs, Mississippi, but not elsewhere (except 2 of 33 that were near). This growth, with a 100 %

Figure 14.86. "Violet shrimp" a white shrimp on top with reference white shrimp on bottom; we saw this case once only and it involved several shrimp in the Pascagoula River, Mississippi, December 1981.

prevalence in some samples, was probably induced by a heavy metal contaminant. Also, shrimp on occasion from multiple locations have been observed with abnormal discoloration. Figure 14.84 shows a golden shrimp, perhaps a genetic anomaly. On the other hand, a palaemonid river shrimp that migrates to the estuary to spawn can acquire a variety of colors, including a golden color (Figure 14.85). A violet discoloration (Figure 14.86) more than likely is a response to a contaminant.

14.6.2 Crabs

Not all animals respond to disease agents similarly, and the susceptibility to specific agents has an influence on population structure. The blue crab provides an example model that we think responds strongly to disease as well as to predation and annual variation in salinity, temperature, and winds. The blue crab differs in its life history from many other crab species. When females mate, they seldom molt again, and they then migrate from the estuary into higher salinity Gulf water to spawn and die, while the males continue to molt, grow, and thrive in the estuary. In the warm Gulf, females can spawn multiple times, producing millions of eggs. From the disease point of view, we think that different specific disease agents can have a detrimental effect on specific Gulf crab populations, and these agents differ annually and interact strongly with predation of weakened, infected crabs. Crab stocks in the Gulf differ in recruitment from those stocks along the East U.S. coast. The megalopae settling from the plankton appeared from collections using similar sampling methods to be 10–100 times more abundant from Alabama to Texas than from Delaware to South Carolina (Heck and Coen 1995). In contrast, these authors and Perry et al. (1998) reported the abundances of juveniles as similar on both coasts and explained the difference in the extra loss of young crabs reaching carapace width of 30 mm (1.2 in) in the Gulf to be caused by predation. Such may be true, but even then, that loss could also result from disease killing a significant portion of the crabs or weakening them and allowing for additional predation.

We do not believe the disease agents necessary to control the crab population to be the same every year or years. Shields and Overstreet (2007) reviewed numerous agents that have the ability to kill or weaken the crabs. We will mention a few, and the first will exemplify one that occurs irregularly. The barnacle *Loxothylacus texanus* has been abundant for a few years during a few periods only during the last 45 years. This parasite is internal but has an externa (the female, protruding, brood chamber) located under the abdominal flap and does not look like a barnacle. Shields and Overstreet (2007) described the complicated life history, which will be briefly stated here because of its importance in understanding the prevalence of infections. There are separate male and female dispersal naupliar larvae that are attracted to light and to high salinity; they develop into relatively small female and larger male cypris larvae. The female cyprid metamorphoses into a kentrogon, which penetrates through the thin membrane between appendage joints of postmolt crabs when less than 18 mm (0.7 in) wide. A wormlike vermigon is released from the kentrogon and wraps around the crab midgut, producing the interna. The interna forms a complex of root-like branches that drain nutrition from the host, and, under appropriate environmental conditions following the host's final molt, it extrudes a virgin externa under the crab abdomen. Numerous young crabs with an equal number of males and females can be infected simultaneously under appropriate temperature and salinity conditions. Male cyprids have a weaker phototactic response, and therefore fertilize the virgin externae in crabs when in the benthos. The cyprid larvae are not viable below 12 ppt, and a mortality of 10 % still occurs at 15 ppt. Infected crabs cannot tolerate low salinity water and survive best in 25–30 ppt. Seldom does the water temperature and salinity in this estuarine environment accommodate the combination of production of barnacle larvae, fertilization, and availability of young crabs simultaneously. When these conditions are appropriate at the same time that infected crabs are present and producing barnacle naupli, over half the young crab population can become infected. Not only does the parasite kill or weaken the infected crabs when the salinity is not high enough, but infected crabs, stunted and castrated from the infection, compete with non-infected individuals for space, food, and sexual partners.

Figure 14.87. Blue Crab, *Callinectes sapidus*, covered with relatively large specimens of the Florida rocksnail, *Stramonita haemastoma floridana*; we have seen crabs commonly feeding heavily on younger specimens of the drill, October 1980.

Figure 14.88. Blue Crab, *Callinectes sapidus*, showing lesions extending through the carapace; we see such cases irregularly, May 1999.

Figure 14.89. The portunid speckled swimming crab, *Arenaeus cribrarius*, exhibiting abnormal right chelipeds, July 1981; similar cases also occur with the blue crab.

Loxothylacus texanus is just one of several parasites that can control the population of juvenile blue crabs in Mississippi and elsewhere in the Gulf in addition to predation by other blue crabs and fishes. Shields and Overstreet (2007) review these in detail. Three other that can infect young crabs include the microsporidian *Ameson michaelis*, the parasitic dinoflagellate *Hematodinium perezi*, and the introduced white spot syndrome virus, or WSSV, assigned by the International Committee on Taxonomy of Viruses as the only member of the genus

Whispovirus within the family Nimaviridae. We experimentally killed crabs with the virus, but penaeid shrimps seem much more susceptible. We (Juan Carrillo, Janet Wright, and R. Overstreet) are presently investigating the effect of each of these three agents on the health and mortality of the blue crab and should be able to determine if the blue crab population is continually being controlled by at least one in a series of several disease agents.

We have seen an abundance of relatively small specimens of the Florida rocksnail, *Stramonita haemastoma floridana*, in the stomach of feeding crabs. We have seen large crabs commonly with an abundance of larger rocksnail drills on the crab carapace (Figure 14.87). This drill feeds heavily on young oysters. Blue crabs, especially those collected from contaminated locations or trapped in crab cages for several days, often exhibit shell disease. This disease results when chitonoclastic bacteria or fungi gain entrance to the shell. Such crabs appear orangish, brownish, or blackish in small to large lesions. Crabs with extensive shell disease indicate stress, keeping the individuals from molting. The lesions, caused by any of several bacteria or fungi, do not exhibit a distinct relationship with the abundance of any of the several bacteria infecting the crab hemolymph (Shields and Overstreet 2007). In some cases, lesions in the carapace will extend into the body cavity (Figure 14.88). Moreover, the blue crab and related portunid crabs occasionally exhibit abnormal chelipeds (Figure 14.89) or other structures.

14.6.3 Oyster

The Eastern oyster, *Crassostrea virginica*, exhibits high susceptibility to mortality resulting from the interaction of salinity, temperature, diseases, and predation. For purposes of this chapter, we will first provide information on *Perkinsus marinus*, previously known as *Dermocystidium marinum* or short as "dermo." This agent, initially described in 1949, probably kills more oysters in the Gulf, including Mexico and Venezuela, than any other agent; it is typically classified in the protozoan Phylum Apicomplexa, but genetic sequencing places it closer to the dinoflagellates (Ford 2011). Moreover, polymerase chain reaction (PCR) analyses can identify the species. Consequently, some infections reported from oysters in the Caribbean, Cuba, Brazil, and elsewhere using Ray's Fluid Thioglycollate Medium may involve related infectious species. Acquisition of infections usually occurs during the warm months of the year, during which the agent proliferates at water temperatures above 18 °C (64 °F) and salinities greater than 15 ppt; experimental infections have been achieved at 10 °C (50 °F) and 3 ppt and proliferation is most rapid at about 25–30 °C (77–86 °F) (Ford 2011). Mortalities between 5 and 30 % typically occur during the first year of an epizootic, reaching 60–80 % by the end of the second year, with mortalities commonly averaging 20–30 % in enzootic waters. While light infections typically influence the host little, advanced stages result in reduced feeding, growth, and reproduction, leaving oysters weak and emaciated before they die from the infection, some other infection, or predation because the infection weakened the host making it vulnerable to predators.

Wilson et al. (1990) examined oysters for prevalence and intensity of infection from 48 locations ranging from Laguna Madre in southern Texas to the Everglades in Florida as part of NOAA's Status and Trends Mussel Watch Program and found the prevalence exceeded 75 % at 25 locations. The intensity of infection did not vary with either sex or reproductive stage of the oyster; however, the distribution of infections was affected by latitude, total PAH content, and industrial and agricultural land use. PAH and pesticide concentrations were dependent on point sources, with the highest concentration values being in St. Andrews Bay, Florida; Vermillion Bay, Louisiana; and Galveston Bay, Texas. Soniat (1996) summarized data from Tabasco, Mexico, to the Everglades and found infections responded to similar factors,

suggesting a combination of temperature and salinity was important but did not explain much of the variation in levels of infection. He also discussed the possibility of increased susceptibility caused by pollutants. Gold-Bouchot et al. (1995, 1997) investigated PAH fractions in oyster tissues from Tabasco and concluded the hydrocarbon concentrations were not responsible for oyster mortality, and histopathological lesions responded more to cadmium and salinity, and mortalities were confounded by the presence of *P. marinus*. A more extensive study by Noreña-Barroso et al. (1999) from Campeche, Mexico, found and was concerned by higher levels of PAHs than previously reported. MacKenzie and Wakida-Kusunoki (1997) summarized the oyster industry of eastern Mexico from Texas to Campeche.

Recent studies have shown that considerable variation exists among strains from 86 clonal cultures derived from 76 parental cultures originating from the Atlantic coast to the Gulf coast (Reece et al. 2001). They determined that 12 different composite genotypes existed, but only one was unique to Gulf Coast isolates. A single oyster can be infected with multiple strains, and virulence differs with genotypic differences. Based on earlier data from fewer isolates, Bushek and Allen (1996) found that two isolates from the mid-Atlantic region produced heavier infections in a shorter period of time than did two from the Gulf. Moreover, isolates differ in the production and activity of some extracellular proteases (La Peyre et al. 1998), and protease inhibitors differ between species of oysters (Faisal et al. 1998). These and other papers help explain why the host defense mechanisms differ in response to infections.

Two diseases in addition to *P. marinus* that cause devastating epizootics in oysters from the Northeast United States are *Haplosporidium nelsoni* (MSX) and *Haplosporidium costale* (SSO). These and other diseases and the defense mechanisms against them are detailed by Ford and Tripp (1996). Most interesting is the finding by Ulrich et al. (2007) that PCR amplification of the ribosomal rRNA detected MSX in 30 of 41 oysters sampled from Florida to the Gulf of Mexico south to Venezuela, even though an epizootic had never been reported.

A histopathological survey for infectious and noninfectious diseases in oysters as well as fishes at Pascagoula, Mississippi; Mobile, Alabama; and Pensacola, Florida; was conducted by Couch (1985); and, in most cases, the three Pascagoula locations indicated a higher prevalence of diseases. However, *P. marinus* was only apparent in 4 % from that location, even though we have seen much heavier infections in the past. He considered epithelial atrophy of the digestive gland to be the best indicator of environmental health. The normal digestive diverticula exhibited deep, thick epithelium forming triradiate and quadriradiate lumina. A total of 35.4 % of the oysters in the Pascagoula harbor demonstrated atrophy compared with 12.5 % in Mobile and 10.2 % in Pensacola. The values from the Pascagoula were heavily influenced by nearly 100 % of the oysters exhibiting failing diverticular epithelia in January and May of 1980. Twenty of the 4,496 oysters from the Pascagoula exhibited proliferative hemocyte (blood cell) disorders. These and other rare neoplastic disorders in oysters have not been evaluated using modern methods. Like Couch (1985), we have recognized thigmotrich and other ciliate protozoans as common symbionts in oysters. He mentioned they were usually nonpathogenic; however, they occasionally occluded water tubules in the gills and digestive tubules. We believe ciliates may be good indicators of water quality and environmental health. Many parasites and disease agents not involved with mass mortalities were reported by Couch (1985), Ford and Tripp (1996), and others and will not be discussed here.

Deserving of some attention are the symbionts, pests, and fouling agents that occur on or in the shell of oysters, often making them brittle and susceptible to predation. Overstreet (1978) provided illustrations and White and Wilson (1996) gathered together considerable literature on the organisms. As examples, the oyster drill (*Stramonita haemastoma*) killed more than 80 % of young oysters in 9 months. In Mobile Bay (Figure 14.87), this gastropod becomes active when the temperature reaches about 12 °C (54 °F) in the water when salinity is about 15–20 ppt (see

Garton and Stickle 1980). The blue crab feeds abundantly on young oysters, and polyclad turbellarian flatworms (*Stylochus ellipticus* and *Stylochus frontalis*) become a major threat in water with salinity above 15 ppt. Seven species of sponges are in the family Clionidae; the primary ones are *Pinone truitti*, which thrives at a salinity of 10–15 ppt and forms small holes in the shell, and *Cliona celata*, which prefers higher salinities and serves as the only species to form large coarse holes in the shell. The clam *Diplothyra curti* bores into and weakens the shell. A polychaete, *Polydora websteri*, uses a chemical agent to penetrate all layers of the shell and resides in mud-filled blisters. Another species, *Polydora cornuta*, also lives subtidally until it enters the oyster and resides in tubes consisting of mud particles held together by the oyster's mucus. In addition to weakening the shells, the symbionts produce dark lesions that can be esthetically displeasing to one eating oysters on the half shell. These and other symbionts can weaken the shell such that the black drum (*Pogonias cromis*) and cownose ray (*Rhinoptera bonasus*) can easily crush the shell of an oyster 8 cm (3.2 in) long; smaller oysters with weakened shells are consumed by a variety of other fishes.

Vibrio bacteria have commonly caused rapid epizootic mortalities of larval oysters in hatcheries, but because the density of the larvae in natural waters is much lower, mortalities are probably not common. Nevertheless, vibrio infections in adult oysters demand attention because they are known to cause human disease in those that eat raw oysters. For example, a survey of 575 laboratory-confirmed cases of vibrio gastroenteritis from Florida to Texas from 1988 to 1997 produced patients with illness that lasted a median of 7 days, produced fever in half of them, and produced bloody stools in 25 % of them. A total of 53 % of the 445 patients for whom data were available had eaten raw oysters in the week before disease-onset (Altekruse et al. 2000). A total of 31 % of the cases involved *Vibrio parahaemolyticus*, 24 % involved *Vibrio cholerae* (non-O1, non-O139), 12 % involved *Vibrio mimicus,* and the others involved six other species of Vibrio. The presence and density of *Vibrio parahaemolyticus* depends

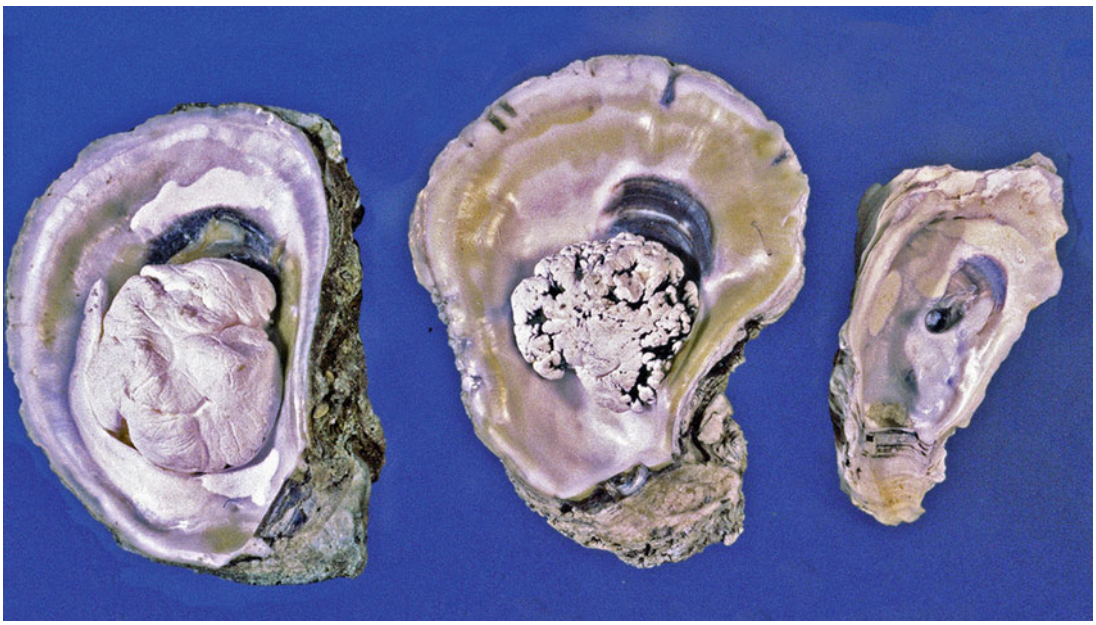

Figure 14.90. Valves of eastern oyster, *Crassostrea virginica*, with two on left exhibiting unusual abnormal nacreal bodies on internal valve surface; perhaps these resulted from repetitive covering of an abscess or foreign body. The valve on right shows myostracum similar to that historically reported in Maryland oysters as *maladie du pied* attributable to an infestation of the fungus *Ostracoblabe implexa*, October 1978.

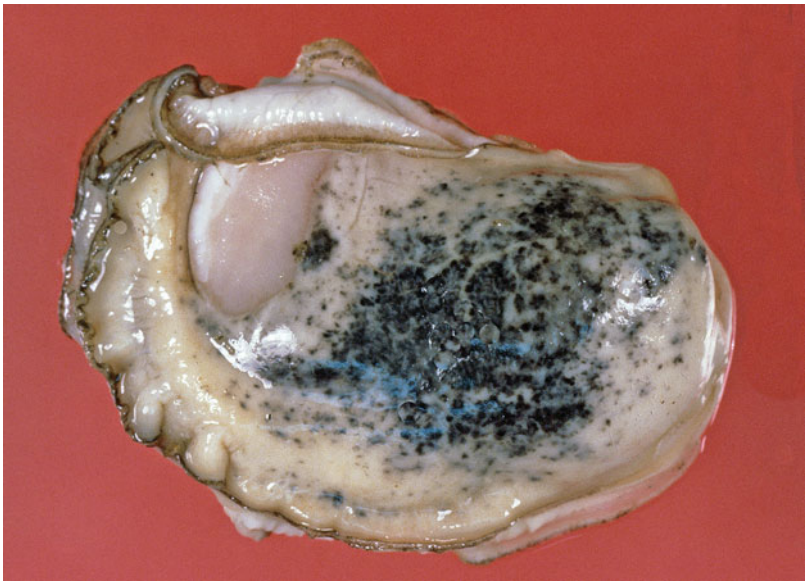

Figure 14.91. Nacreal mantle of eastern oyster, *Crassostrea virginica*, containing unusual punctate melan-like pigmentation response, January 1981.

primarily on temperature, but the bacterium requires salt to survive. It is most common during the summer, and, as an example, 44 % of the oyster samples and 30 % of the water samples in Mississippi from April to August contained the bacterium; while the total densities of the bacterium may be informative, the authors do not recommend densities as a good means to predict risk of human infection (Zimmerman et al. 2007). The presence of *Vibrio vulnificus* also requires attention because in certain high-risk individuals, those with a history of liver disease, alcoholism, and immune deficiencies, infection by means of consumption of raw shellfish or through exposed wounds may result in primary septicemia, meningitis, pneumonia, and death. The bacterium occurs regularly year-round in tropic and subtropical areas such as Charlotte Harbor, Florida, where the temperature remains moderate throughout the year, and salinity strongly controls the seasonal distribution of this bacterium between the sediment and water column. The bacterium occurred most commonly in summer months when the salinity was about 15 ppt (Lipp et al. 2001).

All aspects of the anatomy, biology, and fisheries of the eastern oyster occur in a lengthy book edited by Kennedy et al. (1996), which includes chapters on diseases (Ford and Tripp 1996) and pests (White and Wilson 1996) as indicated above. Overstreet (1978) provided a booklet including symbionts of the oyster. It figured a number of them, possibly including the valve on the right in Figure 14.90 with what appears to be a fungal infection (*Ostracoblabe implexa*) that is rare in Mississippi but more common on the Atlantic coast in cooler waters. Chris Dugan of the Maryland Department of Natural Resources (personal communication) estimated infections of perhaps 1 % in Maryland. Neither Chris or Dorothy Howard of the Oxford NOAA Laboratory (personal communication) have seen the other cases, but other melanistic conditions in the mantle of oysters along the Atlantic coast exist that have a more amorphous appearance rather than punctate (Figure 14.91). The cases figured here are rare in contrast with the more common conditions (Overstreet 1978).

Figure 14.92. Diseased maze coral, *Meandrina meandrites*. Photograph by Stephen Spotte (permission to reprint granted by S. Spotte to R.M. Overstreet).

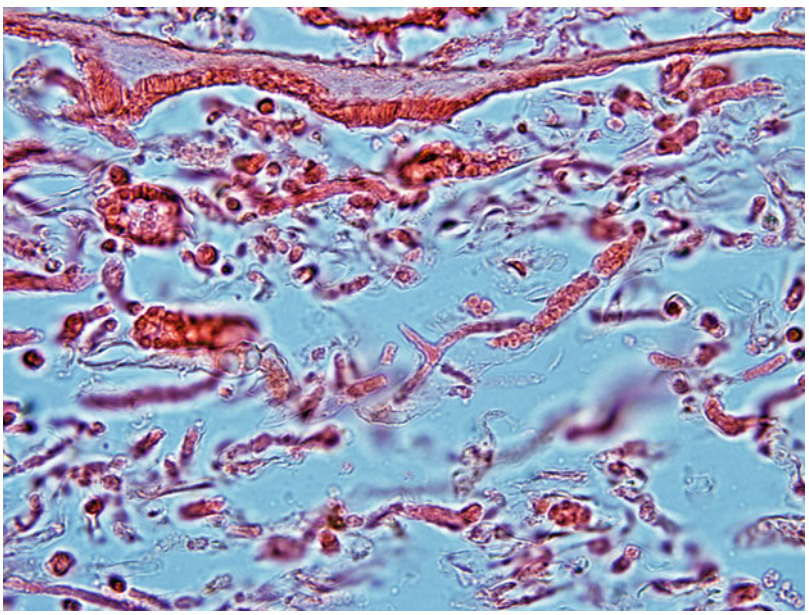

Figure 14.93. Fungal infection from diseased *Meandrina meandrites*. Photograph by Juan Carrillo (permission to reprint granted by J. Carrillo to R.M. Overstreet).

14.6.4 Corals

Corals in the tropical and subtropical areas of the Gulf of Mexico form a significant habitat for a large number of fishes. Consequently, loss of these corals by bleaching, disease, or other causes such as hurricanes can have a major impact on these fishes and other animals, and even change the habitat from a coral-dominated state to an algae-dominated state (Hughes 1994). Bleaching refers to the loss or degradation of photosynthetic symbiotic agents from the

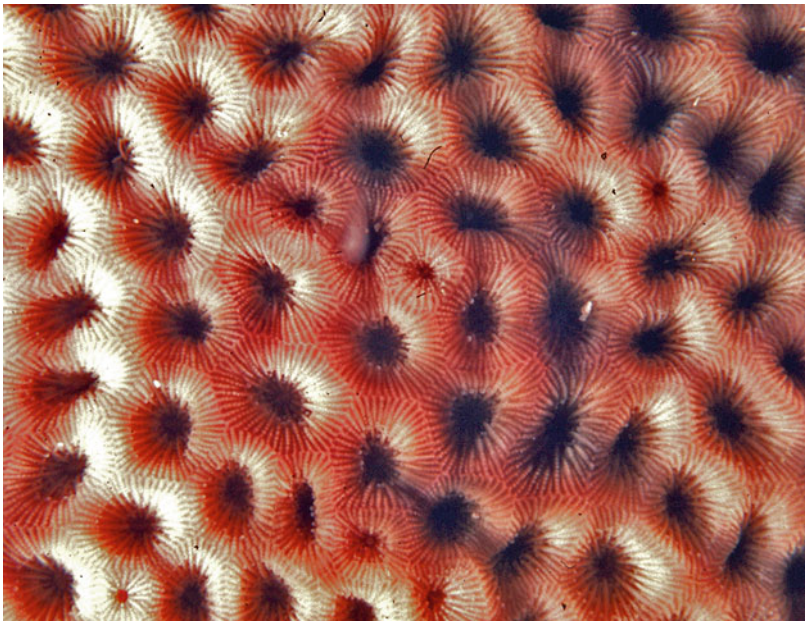

Figure 14.94. Diseased starlet coral *Siderastrea* sp. Photograph by Stephen Spotte (permission to reprint granted by S. Spotte to R.M. Overstreet).

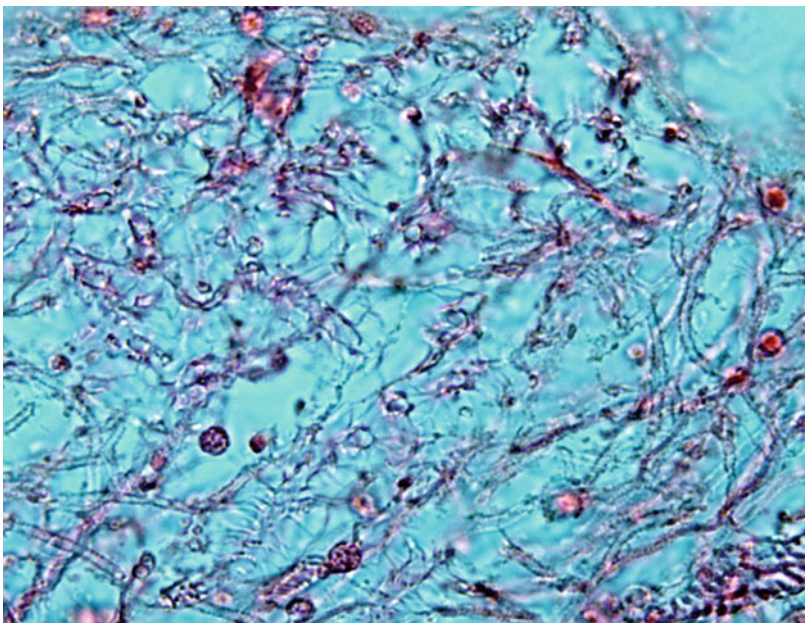

Figure 14.95. Fungal infection from diseased *Siderastrea* sp. Photograph by Juan Carrillo (permission to reprint granted by J. Carrillo to R.M. Overstreet).

endodermis of their hosts. These hosts include hard corals (stony corals), soft corals (gorgonians), and hydrozoans such as fire corals as well as other associated animals. The symbionts comprise dinoflagellates, red and green algae, and cyanobacteria or the pigments from these symbionts. Bleaching constitutes a clear sign of an unhealthy coral environment. Corals can survive being bleached for several months. In the case of the boulder star coral (*Montastraea*

annularis), colonies remained bleached for 7 months after the 1987 bleaching event; they could recover and reestablish their symbionts, but those without their symbionts survived but failed to undergo gametogenesis (Szmant and Gassman 1990).

A variety of specific diseases infects corals and cause mortality in the Gulf and associated waters. The cyanobacteria *Phormidium corallyticum* causes black-band disease (BBD), expressed by a narrow dark band of its filaments encircling the coral and capable of destroying an entire colony. White band disease (WBD) has an unconfirmed etiology, but is possibly caused by adverse environmental conditions associated with a primary bacterial agent, either with or without secondary bacterial infections. Other diseases exist that can be recognized by their gross appearance. Corals are known to be infected by a variety of other internal microscopic symbionts, and we have observed a coccidium, various fungi (Figures 14.92, 14.93, 14.94, and 14.95), and bacteria in sections of both bleached and unbleached tissues. Ultrastructural investigation by Renegar et al. (2008) provided more detail of fungal infections in *Siderastrea siderea*, and it showed the affected tissue had less integrity with more degranulation and vacuolization than could be determined with regular bright-field histopathology. They also determined that identification of the fungal species was difficult. Williams and Bunkley-Williams (1990) provided a review of worldwide coral mortalities occurring from 1969 through the 1987–1988 event, including the periodic 1979–1980, 1981–1983, and 1986–1988 events. Coral disease in the Florida Keys spread rapidly from 1996 to 1998, and the different diseases exhibited different patterns of spread (Porter et al. 2001).

The event in 2005 was especially catastrophic, so we will emphasize it. Brandt and McManus (2009) provided important information on the relationship between coral bleaching and disease in the reef-building corals in the Florida Keys during that 2005 event, which also included infections in the Caribbean Sea. Both features had a positive correlation with high temperatures and with each other, but specific interactions between the two differed. White plague infections developed in the mountainous star coral (*Montastraea faveolata*), following heavy bleaching on those colonies. On the other hand, colonies of the massive starlet coral (*Siderastrea siderea*) with dark spots disease (DSD) bleached more extensively than the assumed healthy colonies. Co-occurrence of bleaching and BBD on the boulder brain coral (*Colpophyllia natans*) was apparent throughout the entire bleaching event. Bleaching, white plague (WP), and BBD each can alter structure of the coral populations by means of death of the living tissue, and DSD seems to be the most important indicator of overall reef health. Yellow band syndrome (YBS) as well as WP and BBD do not always cause mass mortalities, and environmental deterioration is considered to be responsible for the morbidity and accompanying spread of diseases (e.g., Porter et al. 2001). White pox causes great epidemic losses of the elkhorn and staghorn corals (*Acropora palmata* and *Acropora cervicornis*) in Florida and the Caribbean Sea. Lesions occurring in 1998 and spreading an average of 2.5 cm^2/day (0.4 in^2/day) resulted from the common human fecal enterobacterium *Serratia marcescens* (see Patterson et al. 2002).

The lack of a clear understanding of what harmful conditions actually occur on the reef seems to be partly associated with the poor characterization of diseases, syndromes, and the different stages of each as well as the fact that some reports are restricted to a single reef site and single coral species (Jordán-Dahlgren and Rodríguez-Martínez 2004). These authors studied two reefs on the western edge of the Campeche Bank in the southeastern Gulf of Mexico off the Yucatán Peninsula and not part of the primary surface circulation patterns of the Gulf. They found that of 24 coral species, only 10 included some affected colonies. Over 97 % of those affected in both reefs belong to only six coral species, *Montastraea annularis*-complex, great star coral (*Montastraea cavernosa*), knobby brain coral (*Pseudodiploria clivosa*), symmetrical brain coral (*Pseudodiploria strigosa*), mustard hill coral (*Porites*

Figure 14.96. Specimens of the leech *Calliobdella vivida*, a micropredator that feeds on blood from several fish hosts and transmits specific blood parasites (trypanosomes and hemogregarines) to the striped mullet in Mississippi.

astreoides), and *Siderastrea siderea*. Therefore, only 5.6 % of the examined colonies exhibited disease conditions, and only 3 % of those corresponded to characterized diseases and syndromes. They found only the diseases WP, WBD, and BBD and the syndromes YBS and a new one termed "thin dark-line syndrome" (TDLS). They concluded that the sensitivity of specific coral species was not the most important factor influencing disease in the shallow reef habitat nor were the type or prevalence of the conditions nor the site or density of the colonies. When these authors (Jordán-Dahlgren et al. 2005) tested the relationship of local industrial pollutants and local urban pollution with the same disease conditions in colonies of *Montastraea annularis*-complex on from one to three reefs in 1996, 1998, and 2001, they found no direct relationship. Rather than finding that the presence of disease related to environmental quality, they suggested that the reasons for disease, predominated by TDLS and YBS, resulted from their relationship with the Caribbean Sea and the warming surface water. When fragmentation of corals occurred during passage of a hurricane, WP occurred most commonly on the unattached colony fragments, especially those in contact with the sediment (Brandt et al. 2013). Coral mortalities caused by infectious disease and temperature stress both respond to cellular responses, emphasizing granular acidophilic amebocytes. Mydlarz et al. (2008) studied this cellular response in the mesoglea (connective tissue) of the common sea fan (*Gorgonia ventalina*) in the Florida Keys to the fungus *Aspergillus sydowii* and to temperature stress and concluded that this inflammatory response may allow survival of the sea fan and other corals during stressful climatic events.

Disease and corals from the East and West Flower Garden Banks in the northwestern Gulf of Mexico, an area created by uplift of underlying salt domes of Jurassic origin that rose from 100 m (330 ft) to within 17 m (55 ft) of the water's surface, were studied by Hickerson et al. (2008). Historically, the prevalence of disease in those banks was low until February 2005, when the banks experienced widespread coral disease. The plague-like disease (WP) continued to be surveyed after 2005 and was found to be most prominent during the winter months rather than in the warmer months as it occurs in the Caribbean Sea. These authors noted no WBD, which was common elsewhere in the tropical Western Atlantic.

14.6.5 Micropredators

There is only a semantic difference between a micropredator and a parasite in some cases. Some adult trematodes in the intestine of a fish may engulf host tissues without causing disease but are always considered a parasite. Leeches, isopods, argulids, and other animals obtain a blood meal from fishes and often are not considered a symbiont. Not only do they depend on the host, but they often transmit one or more blood parasites to the host. For the leech *Calliobdella vivida* on flatfishes, it transmits *Typanoplasma bullocki* to the summer flounder in the Chesapeake Bay and the hogchoker in Mississippi. When the proper alignment of low temperature, fish with a corresponding reduced immune response, and optimal salinity of 15–22 ppt for an abundance of the leech occur, the flatfishes get infected, develop splenomegaly, and often die (Overstreet 1982; Burreson and Zwerner 1982, 1984; Burreson and Frizzell 1986). The leech also infests the striped mullet (Figure 14.96), which also has blood parasites, but the effect on the host population has not been determined.

14.7 WHAT IS NECESSARY FOR A GOOD BASELINE FOR THE FUTURE?

Clearly, the pathobiological effects of oil spills in the Gulf of Mexico could be evaluated better if current baseline data on parasites and diseases of Gulf organisms were available. First, we lack a good and current baseline for data on parasites and diseases of Gulf organisms. This is underscored by the obvious research gaps on species and organisms as well as the large number of references from decades old studies that form the basis for much of this contribution. Second, there needs to be increased acuity in recognizing and diagnosing biologically relevant pathological lesions and distinguishing them from the range of normal changes in tissue architecture. This acuity is necessary before accurately ascribing biologic effects to natural or manmade causes. Third, we need prospective knowledge of potential manmade and natural impacts to organisms in the Gulf ecosystem. Below we expand on these three points and offer some possible solutions.

In a non-intuitive way, parasites in marine organisms can reflect the health or completeness of the ecosystem in which they are found. On the surface, it might appear that parasites infect host organisms that are weakened or injured, but because many parasites depend on multiple hosts, the absence of one of those hosts can indicate a level of ecological damage. Long-term studies focused on identifying and quantifying parasite burden as well as range-extensions and identification of new parasites or new hosts. Understanding species in these host-parasite relationships at different trophic levels would be invaluable in assessing large scale impacts on the Gulf. However, two elements to achieve this are missing. First is the commitment of marine management and regulatory agencies to fund long-term, broadly-based studies to establish a robust baseline data set. Second, the scientific workforce needed to accomplish those studies is dwindling. Few traditional marine parasitology programs or programs that more broadly deal with marine pathobiology remain. The Gulf oil spill brings to the forefront the need for scientists who are competent in general marine biology and ecology, parasitology, pathology, and bacteriology as well as in the associated molecular tools that accompany those disciplines.

The public concern around the reported occurrence of pathological lesions and malformations in marine organisms often outweighs the real biological significance of those findings. As with parasites, there is a thin database for lesions and malformations and a general lack of trained scientists to interpret those changes. Accurate diagnosis of lesions and malformations is key to determining their etiology. At the histologic level, a lesion represents a point in time of a dynamic process and diagnosis is often subjective. To achieve the *best* diagnoses from

histological samples, the National Toxicology Program of the Department of Health and Human Services instituted the "Pathology Working Group" process wherein a panel of trained and knowledgeable pathologists evaluates contributed histopathological cases from environmental or laboratory studies and develops consensus diagnoses for those cases. A similar process needs to be applied to marine samples and conducted under the auspices of a relevant federal management or regulatory agency. This approach, along with discouraging scientists from releasing findings before they are vetted by peer review, will help maintain the integrity of the science and the confidence the public has in the scientific process.

Finally, we could better evaluate the toxicological impacts of oil spills or similar events in the Gulf if high quality baseline data were available to make before and after comparisons. This, again, is partly due to a poor baseline of data from which to make comparisons, but more broadly, it is due to relevant agencies focusing on long-term environmental events from a point-source perspective rather than concentrating on ecosystem-level effects. Yet every toxicological event begins at the lowest biochemical or molecular level of organization before it proceeds to higher level effects. A case in point is the information needed to evaluate how the oil dispersant Corexit would behave when applied in large quantities over a long period of time. All we basically knew was the acute toxicity of the compound to a small number of species. Long-term laboratory toxicological studies conducted at near "real world" toxicant concentrations are difficult and expensive to carry out but could lead to a valuable understanding of the fate and effects of potentially harmful agents in the marine environment.

14.8 CONCLUSIONS

Our knowledge of the state of health of the Gulf of Mexico fauna prior to 2010 remains based on diverse sources of academic and gray literature as well as our own unpublished investigations in which anecdotal or single-case incidents play a large role. A few long-term datasets exist, but much of those data is spotty and uneven. Long-term studies into the future will really be necessary to interpret the frequency, periodicity, intensity, and causes of disease and mortality events. We think the loss of many coral habitats from uncertain causes and the loss of estuaries because of increased populations near coastlines have a detrimental effect on the Gulf. On the other hand, an increased interest in the environment can have a positive effect on animal health.

Before 2010, episodes of fish kills, infections, and abnormalities had been documented. Acute, mass mortalities attracted attention, but when such an event occurred, attempts were made to ascribe single causes for them. Elevated mortalities are usually due to a convergence of factors, with interacting hosts, agents, and environmental conditions producing a "perfect storm." Such a balance is constantly present to some degree. At least some microbial agents, parasite infections, and environmental conditions occur in large cycles of several decades; whether this results from some underlying periodicity or from random co-occurrence of contributing factors is not certain.

Physical and chemical factors most frequently trigger large-scale mortalities. Eutrophication occurs throughout the Gulf where high nutrient input occurs. Low oxygen levels from eutrophication produce a major stress leading to fish mortality and also lead to disease and parasite-caused mortality. Red tides have a major influence on the health of fishes and other animals from the West Coast of Florida and occasionally elsewhere in the Gulf. Cold kills, which occur primarily inshore where it is hard for some animals to escape, are more disastrous in South Texas and South Florida because species there are not as well acclimated to tolerate rapid temperature changes as they are in higher latitudes of the Gulf. Heat kills, hypersalinity, sulfate reduction, sediments, and drilling fluids all have been implicated in

mortality events, but they produce more localized effects. Hurricanes can occur anywhere in the Gulf, but resulting fish kills depend on the geography of the areas that the hurricanes pass through and on damage to the environment. As with most catastrophic events, the presence and absence of specific parasites can provide a good indication of environmental health and its restoration.

Few diseases cause mass mortality. When investigated, the cause usually involves one or more stresses, with an interaction between host, disease agent, and the environment. Most diseases involving infectious agents are usually shown to be highly restricted to certain geographic areas or to certain species. The most obvious infectious disease-caused mass mortality came from a catfish die-off occurring in 1996 and more cases later from Florida to Texas caused directly or indirectly by the virus. We do not know if that virus becomes intermittently introduced or if it always occurs in the habitat in low numbers until some threshold is surpassed, triggering a pandemic. Some event such as reproductive activity of the catfish may have served as the stressor, but no catastrophic event coincided with the mortality. What seems to be the same agent infects fishes in the southern Gulf of Mexico, South America, Africa, and India.

Parasites often cause disease conditions and mortalities in hosts, usually intermediate hosts, as a part of the parasitic strategy to complete the parasite life history. However, these effects tend to be ongoing at a low level without harm to the ecosystem. In cases where mass mortality occurs, changes in anthropogenic or natural environmental conditions are involved. Major stress can affect resistance of hosts to disease organisms, especially bacterial or protozoal agents. Diseases caused by a few species seem to serve as a means of host population control. Parasites, even when not harming their hosts, can be extremely useful as bioindicators in providing information about stock assessment, biological activities of hosts such as migration, feeding, and restoration of habitats as well as habitat and ecosystem health.

Neoplasms, some virally induced, have seldom been observed or reported in Gulf of Mexico fishes, although their occurrence has likely been underestimated; elsewhere, neoplasms have served as good indicators of various contaminants, particularly sediment-bound polynuclear aromatic hydrocarbons. Consequently, more attention to documenting them is warranted. Developmental abnormalities and histopathological alterations, which have been seen in many Gulf species, can indicate levels of stress from a variety of environmental factors. More quantitative data would allow researchers to tease out what factors may be involved.

Data on disease conditions in non-fish vertebrates are uneven. The best known condition in sea turtles is fibropapillomatosis, and it appears to have multiple causes. Bird mortality events are sometimes ascribed to bacterial, fungal, and viral infections, but the effects of these agents can be exacerbated by environmental conditions that sap energy and deplete needed resources. Brevetoxins and morbillivirus have been implicated in periodic marine mammal mortalities, but the cause of others is unclear, and most data are based on skewed samples from strandings. Diseases of penaeid shrimps and the blue crab have been well documented, but the effect of these diseases on host populations in the Gulf remains unclear. In the eastern oyster, the protozoan disease known as “dermo” has received a great deal of research attention. We know that its impact on oyster populations varies widely according to salinity, temperature, genotype of the infectious agent, and perhaps interaction with specific contaminants, but its variation in severity from location to location in the Gulf has not been adequately explained. Other agents and fouling agents affect oysters, but their impacts and interactions are less well studied. Loss of corals by bleaching and disease has had a major influence on tropical and subtropical Gulf communities because with their loss has come the loss of the associated fishes and invertebrates. Definitions of symbionts, parasites, and micropredators

differ according to different fields and different schools of thought. Nevertheless, described associations have allowed the accumulated information to be helpful in understanding disease in the Gulf. For example, some leeches are not considered symbionts because they obtain blood meals from their hosts. However, when obtaining these meals, some species transmit protozoan parasites, which cause debilitating disease and mortality. Moreover, optimal environmental conditions promote heavy infestations of the leeches, having a significant influence on the host population.

To better understand diseases and mortalities in the Gulf, there is a need for monitoring both diseases and mortalities; for conducting more long-term, broad-scaled field work; for acquiring more expertise; and for developing more critical tools for evaluating health of the animals and health of the ecosystem.

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All images are original to the Gulf Coast Research Laboratory, The University of Southern Mississippi, unless otherwise indicated.

REFERENCES

Adlard RD, Lester RJG (1994) Dynamics of the interaction between the parasitic isopod, *Anilocra pomacentri*, and the coral reef fish, *Chromis nitida*. Parasitology 109:311–324

Adlard RD, Lester RJG (1995) The life-cycle and biology of *Anilocra pomacentri* (Isopoda, Cymothoidae), an ectoparasitic isopod of the coral reef fish, *Chromis nitida* (Perciformes, Pomacentridae). Aust J Zool 43:271–281

Adorno R, Pautz PC (eds) (2003) The Narrative of Cabeza de Vaca: Álvar Núñez-Cabeza de Vaca. University of Nebraska Press, Lincoln, NE, USA. 204 p

Afonso JM, Montero D, Robaina L, Astorga N, Izquierdo MS, Ginés R (2000) Association of a lordosis-scoliosis-kyphosis deformity in gilthead seabream (*Sparus aurata*) with family structure. Fish Physiol Biochem 22:159–163

Aguirre AA, Balazs GH, Zimmerman B, Galey FD (1994) Organic contaminants and trace metals in the tissues of green turtles (*Chelonia mydas*) afflicted with fibropapillomas in the Hawaiian Islands. Mar Pollut Bull 28:109–114

Aguirre AA, Spraker TR, Balazs GH, Zimmerman B (1998) Spirorchidiasis and fibropapillomatosis in green turtles from the Hawaiian Islands. J Wildl Dis 34:91–98

Aguirre AA, Balazs GH, Spraker TR, Murakawa SKK, Zimmerman B (2002) Pathology of oropharyngeal fibropapillomatosis in green turtles *Chelonia mydas*. J Aquat Anim Health 14:298–304

Aharon P, Fu B (2000) Microbial sulfate reduction rates and sulfur and oxygen isotope fractionations at oil and gas seeps in deepwater Gulf of Mexico. Geochim Cosmochim Acta 64:233–246

Alfaro-Núñez A, Bertelsen MF, Bojesen AM, Rasmussen I, Zepeda-Mendoza L, Olsen MT, Gilbert MTP (2014) Global distribution of chelonid fibropapilloma-associated herpesvirus among clinically healthy sea turtles. BMC Evol Biol 14:206

Alford JB, O'Keefe DM, Jackson DC (2008) Developing a framework for fisheries restoration in the Pascagoula River Basin following Hurricane Katrina. In: American Fisheries Society Symposium 64:201–218

Alford JB, O'Keefe DM, Jackson DC (2009) Effects of stocking adult largemouth bass to enhance fisheries recovery in Pascagoula River floodplain lakes impacted by Hurricane Katrina. In: Proc Annu Conf Southeast Assoc Fish and Wildl Agencies 63:104–110

Altekruse SF, Bishop RD, Baldy LM, Thompson SG, Wilson SA, Ray BJ, Griffin PM (2000) *Vibrio* gastroenteritis in the U.S. Gulf of Mexico region: The role of raw oysters. Epidemiol Infect 124:489–495

Anders K (1989) Lymphocystis disease of fishes. In: Ahne W, Kurstak E (eds) Viruses of lower vertebrates. Springer, Berlin Heidelberg, Germany, pp 141–160

Anonymous (2003) Cancer and the environment: What you need to know. National Cancer Institute, Rockville, MD, USA. 42 p

Ariel E (2011) Viruses in reptiles. Vet Res 42:100

Associated Press (1971) Hot water blamed for fish kill at Turkey Point. Daytona Beach Morning J, June 24, pp 42

Atkinson CT, Thomas NJ, Hunter DB (eds) (2008) Parasitic diseases of wild birds. Wiley-Blackwell, New York, NY, USA. 279 p

Austin B (1999) The effects of pollution on fish health. J Appl Microbiol Symp Suppl 85:234S–242S

Baden D, Mende TJ (1982) Toxicity of two toxins from the Florida red tide marine dinoflagellate, *Ptychodiscus brevis*. Toxicon 20:457–461

Baden DG, Bourdelais AJ, Jacocks H, Michelliza S, Naar J (2005) Natural and derivative brevetoxins: Historical background, multiplicity, and effects. Environ Health Perspect 113:621–625

Bailey G, Selivonchick D, Hendricks J (1987) Initiation, promotion, and inhibition of carcinogenesis in rainbow trout. Environ Health Perspect 71:147–153

Bakenhaster MD, McBride RS, Price WW (2006) Life history of *Glossobius hemiramphi* (Isopoda: Cymothoidae): Development, reproduction, and symbiosis with its host *Hemiramphus brasiliensis* (Pisces: Hemiramphidae). J Crustacean Biol 26:283–294

Bao VWW, Koutsaftis A, Leung KMY (2008) Temperature-dependent toxicities of chlorothalonil and copper pyrithione to the marine copepod *Tigriopus japonicus* and dinoflagellate *Pyrocystis lunula*. Australas J Ecotoxicol 14:45–54

Barrett AJ, Rawlings ND, Woessner JF (2012) Handbook of proteolytic enzymes, 3rd edn. Elsevier, Academic Press. 4094 p

Baumann PC, Harshbarger JC, Hartman KJ (1990) Relationship between liver tumors and age in brown bullhead populations from two Lake Erie tributaries. Sci Total Environ 94:71–87

Bergeron K (2015) Coast's 'coldest-ever' day is one for history books. Biloxi Sun Herald, January 18, pp 3F

Bicknese EJ, Childress AL, Wellehan JFX (2010) A novel herpesvirus of the proposed genus *Chelonivirus* from an asymptomatic bowsprit tortoise (*Chersina angulata*). J Zoo Wildl Med 41:353–358

Blaylock RB, Overstreet RM (2002) Diseases and parasites of the spotted seatrout. In: Bartone SA (ed) Biology of the spotted seatrout, Marine biology series. CRC Press, Boca Raton, FL, USA, pp 197–225

Blaylock RB, Overstreet RM, Klich MA (2001) Mycoses in red snapper (*Lutjanus campechanus*) caused by two deuteromycete fungi (*Penicillium corylophilum* and *Cladosporium sphaerospermum*). Hydrobiologia 460:221–228

Blaylock RB, Bullard SA, Whipps CM (2004) *Kudoa hypoepicardials* n. sp. (Mhxozoa: Kudoidae) and associated lesions from the heart of seven perciform fishes in the Northern Gulf of Mexico. J Parasitol 90:584–593

Blazer VS, Vogelbein WK, Densmore CL, May EB, Lilley JH, Zwerner DE (1999) *Aphanomyces* as a cause of ulcerative skin lesions of menhaden from Chesapeake Bay tributaries. J Aquat Anim Health 11:340–349

Bossart GD, Baden DG, Ewing RY, Roberts B, Wright SD (1998) Brevetoxicosis in manatees (*Trichechus manatus latirostris*) from the 1996 epizootic: Gross, histologic, and immunohistochemical features. Toxicol Pathol 26:276–282

Brandt ME, McManus JW (2009) Disease incidence is related to bleaching extent in reef-building corals. Ecology 90:2859–2867

Brandt ME, Smith TB, Correa AMS, Vega-Thurber R (2013) Disturbance driven colony fragmentation as a driver of a coral disease outbreak. PLoS One 8(2):e57164

Bricker SB, Longstaff B, Dennison W, Jones A, Boicourt K, Wicks C, Woerner J (2008) Effects of nutrient enrichment in the nation's estuaries: A decade of change. Harmful Algae 8:21–32

Broeg K, Zander S, Diamant A, Körting W, Krüner G, Paperna I, Westernhagen HV (1999) The use of fish metabolic, pathological and parasitological indices in pollution monitoring. Helgol Mar Res 53:171–194

Browder JA, McClellan DB, Harper DE, Kandrashoff MG, Kandrashoff W (1993) A major developmental defect observed in several Biscayne Bay, Florida, fish species. Environ Biol Fishes 37:181–188

Bruning K, Lingeman R, Ringelberg J (1992) Estimating the impact of fungal parasites on phytoplankton populations. Limnol Oceanogr 37:252–260

Buck JD (1990) Potentially pathogenic marine *Vibrio* species in seawater and marine animals in the Sarasota, Florida, area. J Coast Res 6:943–948

Buck JD, Wells RS, Rhinehart HL, Hansen LJ (2006) Aerobic microorganisms associated with free-ranging bottlenose dolphins in coastal Gulf of Mexico and Atlantic Ocean waters. J Wildl Dis 42:536–544

Bullard SA (2010) *Littorellicola billhawkinsi* n. gen., n. sp. (Digenea: Aporocotylidae) from the myocardial lacunae of Florida pompano, *Trachinotus carolinus* (Carangidae) in the Gulf of Mexico; with a comment on the interrelationships and functional morphology of intertrabecular aporocotylids. Parasitol Int 59:587–598

Bullard SA, Overstreet RM (2002) Potential pathological effects of blood flukes (Digenea: Sanguinicolidae) on pen-reared marine fishes. Proc Gulf Caribb Fish Inst 53:10–25

Bullard SA, Overstreet RM (2006) *Psettarium anthicum* sp. n. (Digenea: Sanguinicolidae) from the heart of cobia *Rachycentron canadum* (Rachycentridae) in the northern Gulf of Mexico. Folia Parasitol 53:117–124

Bullard SA, Overstreet RM (2008) Digeneans as enemies of fishes (Chapter 14). In: Eiras JC, Segner H, Wahli T, Kapoor BG (eds) Fish diseases, Vol 2. Science Publishers, Enfield, NH, USA, pp 817–976

Bullard SA, Frasca S Jr, Benz GW (2000a) Skin lesions caused by *Dermophthirius penneri* (Monogenea: Microbothriidae) on wild-caught blacktip sharks (*Carcharhinus limbatus*). J Parasitol 86:618–622

Bullard SA, Benz GW, Overstreet RM, Williams EH Jr, Hemdal J (2000b) Six new host records and an updated list of wild hosts for *Neobenedenia melleni* (MacCallum) (Monogenea: Capsalidae). Comp Parasitol 67:190–196

Bullard SA, Frasca S Jr, Benz GW (2001) Gill lesions associated with *Erpocotyle tiburonis* (Monogenea: Hexabothriidae) on wild and aquarium-held bonnethead sharks (*Sphyrna tiburo*). J Parasitol 87:972–977

Bullard SA, Goldstein RJ, Goodwin RH III, Overstreet RM (2004) *Cardicola forsteri* (Digenea: Sanguinicolidae) from the heart of a northern bluefin tuna, *Thunnus thynnus* (Scombridae), in the northwest Atlantic Ocean. Comp Parasitol 71:245–246

Burreson EM, Frizzell LJ (1986) The seasonal antibody response in juvenile summer flounder (*Paralichthys dentatus*) to the hemoflagellate *Trypanoplasma bullocki*. Vet Immunol Immunopathol 12:395–402

Burreson EM, Zwerner DE (1982) The role of host biology, vector biology, and temperature in the distribution of *Trypanoplasma bullocki* infections in the lower Chesapeake Bay. J Parasitol 68:306–313

Burreson EM, Zwerner DE (1984) Juvenile summer flounder, *Paralichthys dentatus*, mortalities in the western Atlantic Ocean caused by the hemoflagellate *Trypanoplasma bullocki*: Evidence from field and experimental studies. Helgol Meeresunters 37:343–352

Bush AO, Holmes JC (1986) Intestinal helminths of lesser scaup ducks: An interactive community. Can J Zool 64:142–152

Bushek D, Allen SK Jr (1996) Host-parasite interactions among broadly distributed populations of the eastern oyster *Crassostrea virginica* and the protozoan *Perkinsus marinus*. Mar Ecol Prog Ser 139:127–141

Caira JN, Healy CJ (2004) Elasmobranchs as hosts of metazoan parasites. In: Carrier JC, Musick JA, Heithaus MR (eds) Biology of sharks and their relatives. CRC Press, Boca Raton, FL, USA, pp 523–551

Caira JN, Jensen K, Healy CJ (2001) Interrelationships among tetraphyllidean and lecanicephalidean cestodes. In: Littlewood DTJ, Bray RA (eds) Interrelationships of the Platyhelminthes. Taylor & Francis, London, UK, pp 135–158

Cairns J, Heath AG, Parker BC (1975) The effects of temperature upon the toxicity of chemicals to aquatic organisms. Hydrobiologia 47:135–171

Camp JW, Aho JM, Esch GW (1982) A long-term study on various aspects of the population biology of *Ornithodiplostomum ptychocheilus* in a South Carolina cooling reservoir. J Parasitol 68:709–718

Campbell LM (2007) Local conservation practice and global discourse: A political ecology of sea turtle conservation. Ann Assoc Am Geogr 97:313–334

Canning EU, Curry A, Overstreet RM (2002) Ultrastructure of *Tuzetia weidneri* sp. n. (Microsporidia: Tuzetiidae) in skeletal muscle of *Litopenaeus setiferus* and *Farfantepenaeus aztecus* (Crustacea, Decapoda) and new data on *Perezia nelsoni* (Microsporidia, Pereziidae) in *L. setiferus*. Acta Protozool 41:63–77

Casey RN, Quackenbush SL, Work TM, Balazs GH, Bowser PR, Casey JW (1997) Evidence for retrovirus infections in green turtles *Chelonia mydas* from the Hawaiian Islands. Dis Aquat Organ 31:1–7

Cheang CC, Tsang LM, Chu KH, Cheng I-J, Chan BKK (2013) Host-specific phenotypic plasticity of the turtle barnacle *Chelonibia testudinaria*: A widespread generalist rather than a specialist. PLoS One 8(3):e57592

Chen H, Kuo RJ, Chang TC, Hus CK, Bray RA, Chen IJ (2012) Fluke (Spirorchiidae) infections in sea turtles stranded on Taiwan: Prevalence and pathology. J Parasitol 98:437–439

Christmas JY, Howse HD (1970) The occurrence of lymphocystis in *Micropogon undulatus* and *Cynoscion arenarius* from Mississippi estuaries. Gulf Res Rep 3:131–154

Cipriano RC, Bullock GL, Pyle SW (1984) *Aeromonas hydrophilia* and motile aeromonad septicemias of fish. U.S. Fish & Wildlife Publications, Paper 137. http://digitalcommons.unl.edu/usfwpubs/134.

Coleman FC (1993) Morphological and physiological consequences of parasites encysted in the bulbus arteriosus of an estuarine fish, the sheepshead minnow, *Cyprinodon variegatus*. J Parasitol 79:247–254

Coleman FC, Travis J (1998) Phenology of recruitment and infection patterns of *Ascocotyle pachycystis*, a digenean parasite in the sheepshead minnow, *Cyprinodon variegatus*. Environ Biol Fish 51:87–96

Colorni A, Diamant A (1995) Splenic and cardiac lymphocystis in the red drum, *Sciaenops ocellatus* (L.). J Fish Dis 18:467–471

Colwell RR, Grimes DJ (1984) *Vibrio* diseases of marine fish populations. Helgol Meeresunters 37:265–287

Cone DK, Overstreet R (1997) *Myxobolus mississippiensis* n. sp. (Myxosporea) from gills of *Lepomis macrochirus* in Mississippi. J Parasitol 83:122–124

Cone DK, Overstreet RM (1998) Species of *Myxobolus* (Myxozoa) from the bulbus arteriosus of centrarchid fishes in North America, with a description of two new species. J Parasitol 84:371–374

Cook DW (1973) Experimental infection studies with lymphocystis virus from Atlantic croaker. In: Avault JW (ed) Proceedings of the 3rd Annual Workshop of the World Mariculture Society. Louisiana State University, Baton Rouge, LA, USA, pp 329–335

Cook DW, Lofton SR (1973) Chitinoclastic bacteria associated with shell disease in *Penaeus* shrimp and the blue crab (*Callinectes sapidus*). J Wildl Dis 9:154–159

Cook DW, Lofton SR (1975) Pathogenicity studies with a *Streptococcus* sp. isolated from fishes in an Alabama-Florida fish kill. Trans Am Fish Soc 104:286–288

Corrales J, Nye LB, Baribeau S, Gassman NJ, Schmale MC (2000) Characterization of scale abnormalities in pinfish, *Lagodon rhomboides*, from Biscayne Bay, Florida. Environ Biol Fishes 57:205–220

Costa MP (1994) Parecer tecnico n. 007/NAHP: Mortandade de bagres - Baia de Babitonga (SC) a Barra do Una (SP). CETESB, Sao Paulo, Brazil. 52 p

Couch JA (1985) Prospective study of infectious and noninfectious diseases in oysters and fishes in three Gulf of Mexico estuaries. Dis Aquat Organ 1:59–82

Couch JA, Nimmo DR (1974) Detection of interactions between natural pathogens and pollutants in aquatic animals. In: Amborski RL, Hood MA, Miller RR (eds) Proceedings of the Gulf Coast Regional Symposium on Diseases of Aquatic Animals. Wetland Res Publ No LSA-SG-74-05. Louisiana State University, Baton Rouge, LA, USA, pp 261–268

Craig JK (2012) Aggregation on the edge: Effects of hypoxia avoidance on the spatial distribution of brown shrimp and demersal fishes in the Northern Gulf of Mexico. Mar Ecol Prog Ser 445:75–95

Craig JK, Crowder LB, Gray CD, McDaniel CJ, Henwood TA, Hanifen JG (2001) Ecological effects of hypoxia on fish, sea turtles, and marine mammals in the northwestern Gulf of

Mexico. In: Robalais NN, Turner RE (eds) Coastal hypoxia: Consequences for living resources and ecosystems. Coastal and Estuarine Studies, Washington, DC, USA, pp 269–292

Crawford RB (1983) Effects of drilling fluids on embryo development. EPA Project Summary EPA-600/S3-83-021. U.S. Environmental Protection Agency, Environmental Research Laboratory, Gulf Breeze, FL, USA

Cummings EG (1955) The relation of the mullet (*Mugil cephalus*) to the water and salts of its environment: Structural and physiological aspects. PhD Dissertation, North Carolina State College, Raleigh, NC, USA. 103 p

Curran SS, Tkach VV, Overstreet RM (2013a) A new species of *Homalometron* (Digenea: Apocreadiidae) from fishes in the northern Gulf of Mexico. J Parasitol 99:93–101

Curran SS, Tkach VV, Overstreet RM (2013b) Molecular evidence for two cryptic species of *Homalometron* (Digenea: Apocreadiidae) in freshwater fishes of the southeastern United States. Comp Parasitol 80:186–195

Dailey MD, Morris R (1995) Relationship of parasites (Trematoda: Spirorchidae) and their eggs to the occurrence of fibropapillomas in the green turtle (*Chelonia mydas*). Can J Fish Aquat Sci 52(suppl 1):84–89

Dailey M, Walsh M, Odell D, Campbell T (1991) Evidence of prenatal infection in the bottlenose dolphin (*Tursiops truncatus*) with the lungworm *Halocercus lagenorhynchi* (Nematoda: Pseudaliidae). J Wildl Dis 27:164–165

Davidson AJ (2010) Herpesvirus systematics. Vet Microbiol 143:52–69

Dawson CE (1962) Notes on anomalous American Heterosomata with descriptions of five new records. Copeia 1962:138–146

Dawson CE (1964) A bibliography of anomalies of fishes. Gulf Res Rep 1:308–399

Dawson CE (1966) A bibliography of anomalies of fishes. Gulf Res Rep 2(Suppl 1):169–176

Dawson CE (1967) Three new records of partial albinism in American Heterosomata. Trans Am Fish Soc 96:400–404

Dawson CE (1969) Three unusual cases of abnormal coloration in northern Gulf of Mexico flatfishes. Trans Am Fish Soc 98:106–108

Dawson CE (1971) A bibliography of anomalies of fishes. Gulf Res Rep 3(Suppl 2):215–239

Dawson CE, Heal E (1976) A bibliography of anomalies of fishes. Gulf Res Rep 5(Suppl 3):35–41

Deeds JR, Reimschuessel R, Place AR (2006) Histopathological effects in fish exposed to the toxins from *Karlodinium micrum*. J Aquat Anim Health 18:136–148

Deeds JR, Landsberg JH, Etheridge SM, Pitcher GC, Longan SW (2008) Non-traditional vectors for paralytic shellfish poisoning. Mar Drugs 6:308–348

Delaney PM (1989) Phylogeny and biogeography of the marine isopod family Corallanidae (Crustacea, Isopoda, Flabellifera). Contributions in Science No. 409, Natural History Museum of Los Angeles County, Allen Press, Lawrence, KS, USA. 75 p

DeLorenzo ME, Wallace SC, Danese LE, Baird TD (2009) Temperature and salinity effects on the toxicity of common pesticides to the grass shrimp, *Palaemonetes pugio*. J Environ Sci Health B 44:455–460

DePaola A, Capers GM, Alexander D (1994) Densities of *Vibrio vulnificus* in the intestines of fish from the US Gulf Coast. Appl Environ Microbiol 60:984–988

Diamant A, Fournie JW, Courtney LA (1994) X-cell pseudotumors in a hardhead catfish *Arius felis* (Ariidae) from Lake Pontchartrain, Louisiana, USA. Dis Aquat Organ 18:181–185

Dickerson HW (2006) *Ichthyophthirius multifiliis* and *Cryptocaryon irritans* (Phylum Ciliophora). In: Woo PTK (ed) Fish diseases and disorders, Vol 1, CAB International, Wallingford, Oxfordshire, UK, pp 116–153

Dietz K (1982) Overall population patterns in the transmission cycle of infectious disease agents. In: Anderson RM, May RM (eds) Population biology of infectious diseases. Springer, Berlin, Germany, pp 87–102

Diggles BK, Adlard RD (1997) Intraspecific variation in *Cryptocaryon irritans*. J Eukaryot Microbiol 44:25–32

Diggles BK, Lester RJG (1996a) Influence of temperature and host species on the development of *Cryptocaryon irritans*. J Parasitol 82:45–51

Diggles BK, Lester RJG (1996b) Infections of *Cryptocaryon irritans* on wild fish from southeast Queensland, Australia. Dis Aquat Organ 25:159–167

Dobson AP, Hudson PJ (1992) Regulation and stability of a free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models. J Anim Ecol 61:487–498

Dorland (1974) Dorland's medical dictionary, 25th edn. WB Saunders, Philadelphia, PA, USA. 1748 p

Dortch Q, Robichaux R, Pool S, Milsted D, Mire G, Rabalais NN, Soniat TM, Fryxell GA, Turner RE, Parsons ML (1997) Abundance and vertical flux of *Pseudo-nitzschia* in the northern Gulf of Mexico. Mar Ecol Prog Ser 146:249–264

Dukes TW, Lawler AR (1975) The ocular lesions of naturally occurring lymphocystis in fish. Can J Comp Med 39:406–410

Dyková I, Lom J (2007) Histopathology of protistan and myxozoan infections in fishes: An atlas. Academia, Praha, Czech Republic. 219 p

Dyková I, Lom J, Overstreet RM (1994) Myxosporean parasites of the genus *Kudoa* Meglitsch, 1947 from some Gulf of Mexico fishes: Description of two new species and notes on their ultrastructure. Eur J Protistol 30:316–323

Dykstra MJ, Kane AS (2000) *Pfiesteria piscicida* and ulcerative mycosis of Atlantic menhaden—current status of understanding. J Aquat Anim Health 12:18–25

Dzikowski R, Paperna I, Diamant A (2003) Use of fish parasite species richness indices in analyzing anthropogenically impacted coastal marine ecosystems. Helgol Mar Res 57:220–227

Edwards RH, Overstreet RM (1976) Mesenchymal tumors of some estuarine fishes of the northern Gulf of Mexico. I. Subcutaneous tumors, probably fibrosarcomas, in the striped mullet, *Mugil cephalus*. Bull Mar Sci 26:33–40

Ene A, Su M, Lemaire S, Rose C, Schaff S, Moretti R, Lenz J, Herbst LH (2005) Distribution of chelonid fibropapillomatosis-associated herpesvirus variants in Florida: Molecular genetic evidence for infection of turtles following recruitment to neritic developmental habitats. J Wildl Dis 41:489–497

Esch GW (1994) Population biology of the diplostomatid trematode *Uvilifer ambloplitis*. In: Scott ME, Smith G (eds) Parasitic and infectious diseases: Epidemiology and ecology. Academic Press, San Diego, CA, USA, pp 321–336

Evans NM, Holder MT, Barbeitos MS, Okamura B, Cartwright P (2010) The phylogenetic position of Myxozoa: Exploring conflicting signals in phylogenomic and ribosomal data sets. Mol Biol Evol 27:2733–2746

Eyisi OAL, Nwodo UU, Iroegbu CU (2013) Distribution of Vibrio species in shellfish and water samples collected from the Atlantic coastline of South-East Nigeria. J Health Popul Nutr 31:314

Fabacher DL, Besser JM, Schmitt CJ, Harshbarger JC, Peterman PH, Lebo JA (1991) Contaminated sediments from tributaries of the Great Lakes: Chemical characterization and carcinogenic effects in medaka (*Oryzias latipes*). Arch Environ Contam Toxicol 21:17–34

Fagerholm HP, Overstreet RM, Humphrey-Smith I (1996) *Contracaecum magnipapillatum* (Nematoda, Ascaridoidea): Resurrection and pathogenic effect of a common parasite

from the proventriculus of *Anous minutus* from the Great Barrier Reef, with a note on *C. variegatum*. Helminthologia 33:195–207

Faisal M, MacIntyre EA, Adham KG, Tall BD, Kothary MH, La Peyre JF (1998) Evidence for the presence of protease inhibitors in eastern (*Crassostrea virginica*) and Pacific (*Crassostrea gigas*) oysters. Comp Biochem Physiol B Biochem Mol Biol 121:161–168

Fauquier DA, Kinsel MJ, Dailey MD, Sutton GE, Stolen MK, Wells RS, Gulland FMD (2009) Prevalence and pathology of lungworm infection in bottlenose dolphins *Tursiops truncatus* from southwest Florida. Dis Aquat Organ 88:85–90

Felder DL, Camp DK (eds) (2009) Gulf of Mexico origin, waters, and biota: Biodiversity (Vol. 1). Texas A&M University Press, College Station, TX, USA. 1393 p

Felley SM, Vecchione M, Hare SGF (1987) Incidence of ectoparasitic copepods on ichthyoplankton. Copeia 1987:778–782

Finch RH (1917) Fish killed by the cold wave of February 2-4, 1917, in Florida. Mon Weather Rev 1917:171–172

Flaherty KE, Landsberg JH (2011) Effects of a persistent red tide (*Karenia brevis*) bloom on community structure and species-specific relative abundance of nekton in a Gulf of Mexico estuary. Estuaries and Coasts 34:417–439

Flewelling LJ, Adams DH, Naar JP, Atwood KE, Granholm AA, O'Dea SN, Landsberg JH (2010) Brevetoxins in sharks and rays (Chondrichthyes, Elasmobranchii) from Florida coastal waters. Mar Biol 157:1937–1953

Foley AM, Schroeder BA, Redlow AE, Fick-Child KJ, Teas WG (2005) Fibropapillomatosis in stranded green turtles (Chelonia mydas) from the eastern United States (1980-98): Trends and associations with environmental factors. J Wildl Dis 41:29–41

Ford SE (2011) Dermo disease of oysters caused by *Perkinsus marinus*. ICES Identification Leaflets for Diseases and Parasites of Fish and Shellfish, Leaflet 30. 5p

Ford SE, Tripp MR (1996) Diseases and defense mechanisms. In: Kennedy VS, Newell RIE, Eble AF (eds) The eastern oyster *Crassostrea virginica*. Maryland Sea Grant College, College Park, MD, USA, pp 581–660

Forrester DJ, Gaskin JM, White FH, Thompson NP, Quick JA, Henderson GE, Woodard JC, Robertson WD (1977) An epizootic of waterfowl associated with a red tide episode in Florida. J Wildl Dis 13:160–167

Fotheringham N, Weissberg GH (1979) Some causes, consequences and potential environmental impacts of oxygen depletion in the northern Gulf of Mexico. Offshore Tech Conf Proc OTC 3611:2205–2207

Fournie JW, Overstreet RM (1993) Host specificity of *Calyptospora funduli* (Apicomplexa: Calyptosporidae) in atheriniform fishes. J Parasitol 79:720–727

Fournie JW, Overstreet RM, Bullock LH (1985) Multiple capillary haemangiomas in the scamp, *Mycteroperca phenax* Jordan and Swain. J Fish Dis 8:551–555

Fournie JW, Vogelbein WK, Overstreet RM (1987) Squamous cell carcinoma in the Gulf menhaden, *Brevoortia patronus* Goode. J Fish Dis 10:133–136

Fournie JW, Summers JK, Weisburg SB (1996) Prevalence of gross pathological abnormalities in estuarine fishes. Trans Am Fish Soc 125:581–590

Fournie JW, Vogelbein WK, Overstreet RM, Hawkins WE (2000) Life cycle of *Calyptospora funduli* (Apicomplexa: Calyptosporidae). J Parasitol 86:501–505

Frasca S Jr, Kirsipuu VL, Russell S, Bullard SA, Benz GW (2004) Opercular lesion in wild black drum, *Pogonias cromis* (Linnaeus, 1766), associated with attachment of the sea louse *Sciaenophilus tenuis* (Copepoda: Siphonostomatoida: Caligidae). Acta Ichthyol Piscat 34:115–127

Fusco AC (1980) Larval development of *Spirocamallanus cricotus* (Nematoda: Camallanidae). Proc Helminthol Soc Wash 47:63–71

Fusco AC, Overstreet RM (1978) *Spirocamallanus cricotus* sp. n. and *S. halitrophus* sp. n. (Nematoda: Camallanidea) from fishes in the northern Gulf of Mexico. J Parasitol 64:239–244

Galtsoff PS (1954) Gulf of Mexico, its origin, and marine life. U.S. Fish and Wildlife Service. Fishery Bulletin 55(89):xiv, 604 pp

Gannon DP, McCabe EJB, Camilleri SA, Gannon JG, Brueggen MK, Barleycorn AA, Palubok VI, Kirkpatrick GJ, Wells RS (2009) Effects of *Karenia brevis* harmful algal blooms on nearshore fish communities in southwest Florida. Mar Ecol Prog Ser 378:171–186

Garrison VH, Shinn EA, Foreman WT, Griffin DW, Holmes CW, Kellogg CA, Majewski MS, Richardson LL, Ritchie KB, Smith GW (2003) African and Asian dust: From desert soils to coral reefs. BioScience 53:469–480

Gartner JV Jr (1986) Observations on anomalous conditions in some flatfishes (Pisces: Pleuronectiformes), with a new record of partial albinism. Environ Biol Fishes 17:141–152

Garton D, Stickle WB (1980) Effects of salinity and temperature on the predation rate of *Thais haemastoma* on *Crassostrea virginica* spat. Biol Bull 158:49–57

Gassman NJ, Nye LB, Schmale MC (1994) Distribution of abnormal biota and sediment contaminants in Biscayne Bay, Florida. Bull Mar Sci 54:929–943

Gaughan DJ (2002) Disease-translocation across geographic boundaries must be recognized as a risk even in the absence of disease identification: The case with Australian *Sardinops*. Rev Fish Biol Fish 11:113–123

Gold-Bouchot G, Simá-Alvarez R, Zapata-Peréz O, Güemez-Ricalde J (1995) Histopathological effects of petroleum hydrocarbons and heavy metals on the American oyster (*Crassostrea virginica*) from Tabasco, Mexico. Mar Pollut Bull 31:439–445

Gold-Bouchot G, Zavala-Coral M, Zapata-Pérez O, Ceja-Moreno V (1997) Hydrocarbon concentrations in oysters (*Crassostrea virginica*) and recent sediments from three coastal lagoons in Tabasco, Mexico. Bull Environ Contam Toxicol 59:430–437

Gordon AN, Kelly WR, Cribb TH (1998) Lesions caused by cardiovascular flukes (Digenea: Spirorchidae) in stranded green turtles (*Chelonia mydas*). Vet Pathol 35:21–30

Groff JM (2004) Neoplasia in fishes. Vet Clin Exot Anim 7:705–756

Gudger EW (1934) Ambicoloration in the winter flounder, *Pseudopleuronectes americanus*: Incomplete ambicoloration without other deformity; Complete ambicoloration with a hooked dorsal fin and with the rotating eye just over the dorsal ridge. Am Mus Novit 717:1–8

Gunter G (1941) Death of fishes due to cold on the Texas coast, January, 1940. Ecology 22:203–208

Gunter G (1947) Catastrophism in the sea and its paleontological significance, with special reference to the Gulf of Mexico. Am J Sci 245:669–676

Gunter G (1948) A discussion of abnormal scale patterns in fishes, with notice of another specimen with reversed scales. Copeia 1948:280–285

Gunter G (1967) Some relationships of estuaries to the fisheries of the Gulf of Mexico. In: Lauff GA (ed) Esturaries. Am Assoc Adv Sci, Washington, DC, USA, pp 621–638

Gunter G, Hildebrand HH (1951) Destruction of fishes and other organisms on the south Texas coast by the cold wave of January 28-February 3, 1951. Ecology 32:731–736

Gunter G, Lyles CH (1979) Localized plankton blooms and jubilees on the Gulf Coast. Gulf Res Rep 3:297–299

Gunter G, Smith FW, Williams RH (1947) Mass mortality of marine animals on the lower west coast of Florida, November 1946-January 1947. Science 105:256–257

Gunter G, Williams RH, Davis CC, Smith FW (1948) Catastrophic mass mortality of marine animals and coincident phytoplankton bloom on the west coast of Florida, November 1946 to August 1947. Ecol Monogr 18:309–324

Guthrie JF, Kroger RL (1974) Schooling habits of injured and parasitized menhaden. Ecology 55:208–210

Hallegreaff GM (1995) Harmful algal blooms: A global overview. In Hallegreaff GM, Anderson DM, Cembella AD, eds, Manual on harmful marine microalgae. IOC Manuals and Guides 33. UNESCO, Paris, France, pp 1–22

Hamann M, Godfrey MH, Seminoff JA, Arthur K, Barata PCR, Bjorndal KA, Bolten AB, Broderick AC, Campbell LM, Carreras C, Casale P, Chaloupka M, Chan SKF, Coyne MS, Crowder LB, Diez CE, Dutton PH, Epperly SP, FitzSimmons NN, Formia A, Girondot M, Hays GC, Cheng IJ, Kaska Y, Lewiston R, Mortimer JA, Nichols WJ, Reina RD, Shanker K, Spotila JR, Tomás J, Wallace BP, Work TM, Zbinden J, Godley BJ (2010) Global research priorities for sea turtles: Informing management and conservation in the 21st century. Endang Species Res 11:245–269

Harper DE Jr, McKinney LD, Salzer RR, Case RJ (1981) The occurrence of hypoxic bottom water off the upper Texas coast and its effects on the benthic biota. Contrib Mar Sci 24:53–79

Harshbarger JC (1965–1981) Activities reports. Registry of tumors in lower animals. Smithsonian Institution, Washington, DC, USA

Harshbarger JC (1977) Role of the registry of tumors in lower animals in the study of environmental carcinogenesis in aquatic animals. Ann New York Acad Sci 298:280–282

Harshbarger JC, Clark JB (1990) Epizootiology of neoplasm in bony fish of North America. Sci Total Environ 94:1–167

Harshbarger JC, Spero PM, Wolcott NM (1993) Neoplasms in wild fish from the marine ecosystem emphasizing environmental interactions. In: Couch JA, Fournie JW (eds) Pathobiology of marine and estuarine organisms. CRC Press, Boca Raton, FL, USA, pp 157–176

Hawke JP (1976) A survey of the diseases of striped bass, Morone saxatilis and pompano, Trachinotus carolinus cultured in earthen ponds. In Proceedings of the Annual Meeting-World Mariculture Society Vol 7, No. 1–4, Blackwell Publishing Ltd, Oxford, UK, pp 495–509

Hawkins WE, Fournie JW, Overstreet RM (1984) Intrahepatic stages of *Eimeria funduli* (Protista: Apicomplexa) in the longnose killifish, *Fundulus similis*. Trans Am Microsc Soc 103:185–194

Hawkins WE, Overstreet RM, Fournie JW, Walker WW (1985) Small aquarium fishes as models for environmental carcinogenesis: Tumor induction in seven fish species. J Appl Toxicol 5:261–264

Hawkins WE, Walker WW, Overstreet RM, Lytle TF, Lytle JS (1988) Dose-related carcinogenic effects of water-borne benzo[a]pyrene on livers of two small fish species. Ecotoxicol Environ Saf 16:219–231

Hawkins WE, Walker WW, Overstreet RM, Lytle JS, Lytle TF (1990) Carcinogenic effects of some polycyclic aromatic hydrocarbons on the Japanese medaka and guppy in waterborne exposures. Sci Total Environ 94:155–167

Hayes MA, Smith IR, Rushmore TH, Crane TL, Thorn C, Kocal TE, Ferguson HW (1990) Pathogenesis of skin and liver neoplasm in white suckers from industrially polluted areas in Lake Ontario. Sci Total Environ 94:105–123

Hazen EL, Craig JK, Good CP, Crowder LB (2009) Vertical distribution of fish biomass in hypoxic waters on the Gulf of Mexico shelf. Mar Ecol Prog Ser 375:195–207

Hazen TC, Raker ML, Esch GW, Fliermans CB (1978) Ultrastructure of red-sore lesions on largemouth bass (*Micropterus salmoides*): Association of the ciliate *Epistylis* sp. and the bacterium *Aeromonas hydrophila*. J Protozool 25:351–355

Heck KL, Coen LD (1995) Predation and the abundance of juvenile blue crabs: A comparison of selected East and Gulf Coast (USA) studies. Bull Mar Sci 57:877–883

Henley MW, Lewis DH (1976) Anaerobic bacteria associated with epizootics in grey mullet (*Mugil cephalus*) and redfish (*Sciaenops ocellata*) along the Texas Gulf Coast. J Wildl Dis 12:448–453

Herbert BW, Shaharom-Harrison FM, Overstreet RM (1994) Description of a new blood-fluke, *Cruoricola lates* n.g., n.sp. (Digenea: Sanguinicolidae), from sea-bass *Lates calcarifer* (Bloch, 1790) (Centropomidae). Syst Parasitol 29:51–60

Herbst LH (1994) Fibropapillomatosis of marine turtles. Annu Rev Fish Dis 4:389–425

Herbst LH (1999) Infectious diseases of marine turtles. In: Eckert KL, Bjorndal KA, Abreu-Grobois FA, Donnelly M (eds) Research and Management Techniques for the Conservation of Sea Turtles. IUCN/SSC Marine Turtle Specialist Group, Washington, DC, USA, pp 208–213

Hewitt GC (1971) Two species of *Caligus* (Copepoda, Caligidae) from Australian waters, with a description of some developmental stages. Pac Sci 25:145–164

Hickerson EL, Schmahl GP, Robbart M, Precht WF, Caldow C (2008) State of coral reef ecosystems of the Flower Garden Banks, Stetson Bank, and other Banks in the northwestern Gulf of Mexico. In: Waddell JE, Clarke AM (eds) The state of coral reef ecosystems of the United States and Pacific Freely Associated States: Memorandum NOS NCCOS 73. NOAA/NCCOS Center for Coastal Monitoring and Assessment's Biogeography Team. Silver Spring, MD, USA, pp 189–217

Ho J-S (2000) The major problem of cage aquaculture in Asia relating to sea lice (Southeast Asia chapter). In: Liao I, Lin C (eds) Cage aquaculture in Asia, Proceedings of the First International Symposium on Cage Aquaculture in Asia. Asian Fisheries Society/Manila and World Aquaculture Society, Bangkok, Thailand, pp 13–19

Hoberg EP (1996) Faunal diversity among avian parasite assemblages: The interaction of history, ecology, and biogeography in marine systems. Bull Scand Soc Parasitol 6:65–89

Hoberg EP, Klassen GJ (2002) Revealing the faunal tapestry: Co-evolution and historical biogeography of hosts and parasites in marine systems. Parasitology 124:3–22

Honjo M, Matsui K, Ueki M, Nakamura R, Fuhrman JA, Kawabara Z (2006) Diversity of virus-like agents killing *Microcystis aeruginosa* in a hyper-eutrophic pond. J Plankton Res 28:407–412

Howse HD (1972) Snook (Centropomus: Centropomidae): New host for lymphocystis, including observations on the ultrastructure of the virus. Am Midl Nat 88:476–479

Howse HD, Lawler AR, Hawkins WE, Foster CA (1977) Ultrastructure of lymphocystis in the heart of the silver perch, *Bairdiella chrysura* (Lacepede), including observations on normal heart structure. Gulf Caribb Res 6:39–57

Howse HD, Overstreet RM, Hawkins WE, Franks JS (1992) Ubiquitous perivenous smooth muscle cords in viscera of the teleost *Rachycentron canadum*, with special emphasis on liver. J Morphol 212:175–189

Hudson PJ (1986) The effect of a parasitic nematode on the breeding production of red grouse. J Anim Ecol 55:85–92

Hudson PJ, Dobson AP. (1990). Red grouse population cycles and the population dynamics of the caecal nematode *Trichostrongylus tenuis*. In: Lance AN, Lawton JH (eds) Red grouse population processes BES/RSPB Red Grouse Workshop. BES/RSPB Publications, pp 5-19

Hudson PJ, Dobson AP (1997a) Transmission dynamics and host-parasite interactions of *Trichostrongylus tenuis* in red grouse (*Lagopus lagopus scoticus*). J Parasitol 83:194–202

Hudson PJ, Dobson AP (1997b) Host-parasite processes and demographic consequences. In: Clayton DH, Moore J (eds) Host-parasite evolution: General principles and avian models. Oxford University Press, Oxford, UK, pp 128–154

Hudson PJ, Dobson AP, Newborn D (1985) Cyclic and non-cyclic populations of red grouse: A role for parasitism? In: Rollinson D, Anderson RM (eds) Ecology and genetics of host-parasite interactions. Academic Press, London, UK, pp 77–89

Hudson PJ, Newborn D, Dobson AP (1992) Regulation and stability of a free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments. J Anim Ecol 61:477–486

Hudson PJ, Dobson AP, Newborn D (1998) Prevention of population cycles by parasite removal. Science 282:2256–2258

Huerta P, Pineda H, Aguirre A, Spraker T, Sarti L, Barragán A (2002) First confirmed case of fibropapilloma in a leatherback turtle (*Dermochelys coriacea*). In: Mosier A, Foley A, Brost B (eds) Proceedings of the 20th Annual Symposium on Sea Turtle Biology and Conservation. U.S. Department of Commerce, Washington, DC, National Oceanic and Atmospheric Administration Technical Memorandum NMFS-SEFSC-477. 193 p

Hughes TP (1994) Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. Science 265:1547–1551

Huizinga HW, Esch GW, Hazen TC (1979) Histopathology of red-sore disease (*Aeromonas hydrophila*) in naturally and experimentally infected largemouth bass *Micropterus salmoides* (Lacepede). J Fish Dis 2:263–277

Hutchinson DR, Ruppel CD, Roberts HH, Carney RS, Smith MA (2011) Gas hydrates in the Gulf of Mexico. In: Buster NA, Holmes CW (eds) Gulf of Mexico origin, waters, and biota, Vol 3: Geology. Texas A&M University Press, College Station, TX, USA, pp 247–275

Iverson ES, Kelley JF (1976) Microsporidiosis successfully transmitted experimentally in pink shrimp. J Invertebr Pathol 27:407–408

James SV, Valenti TW, Roelke DL, Grover JP, Brooks BW (2011) Probabilistic ecological hazard assessment of microcystin-LR allelopathy to *Prymnesium parvum*. J Plankton Res 33:319–332

Jensen K (2009) Cestoda (Platyheminthes) of the Gulf of Mexico. In: Felder DL, Camp DK (eds) Gulf of Mexico Origin, Waters, and Biota, Vol 1: Biodiversity. Texas A&M University Press, College Station, TX, USA, pp 487–499

Jensen K, Bullard SA (2010) Characterization of a diversity of tetraphyllidean and rhinebothriidean cestode larval types, with comments on host associations and life-cycles. Int J Parasitol 40:889–910

Jones JB, Hyatt AD, Hine PM, Whittington RJ, Griffin DA, Bax NJ (1997) Australasian pilchard mortalities. World J Microbiol Biotechnol 13:383–392

Jones TC, Overstreet RM, Lotz JM, Frelier PF (1994) *Paraophioidina scolecoides* n. sp., a new aseptate gregarine from the cultured Pacific white shrimp, *Penaeus vannamei*. Dis Aquat Organ 19:67–75

Jordán-Dahlgren E, Rodríguez-Martínez RE (2004) Coral Diseases in Gulf of Mexico Reefs. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin Heidelberg, Germany, pp 105–118

Jordán-Dahlgren E, Maldonado MA, Rodríguez-Martínez RE (2005) Diseases and partial mortality in *Montastraea annularis* species complex in reefs with differing environmental conditions (NW Caribbean and Gulf of México). Dis Aquat Organ 63:3–12

Justić D, Bierman VJ, Scavia D, Hetland RD (2007) Forecasting Gulf's hypoxia: The next 50 years? Estuaries Coasts 30:791–801

Kennedy CR (1984) The use of frequency distributions in an attempt to detect host mortality induced by infections of diplostomatid metacercariae. Parasitology 89:209–220

Kennedy VS, Newell RIE, Eble AF (1996) The eastern oyster *Crassostrea virginica*. Maryland Sea Grant College, College Park, MD, USA. 734 p

Kensley B, Heard RW (1997) *Tridentella ornata* (Richardson 1911), new combination: Records of hosts and localities (Crustacea: Isopoda: Tridentellidae). Proc Biol Soc Wash 110:422–425

Khan RA (1990) Parasitism in marine fish after chronic exposure to petroleum hydrocarbons in the laboratory and to the Exxon Valdez oil spill. Bull Environ Contam Toxicol 44:759–763

Khan RA, Hooper RG (2007) Influence of a thermal discharge on parasites of a cold-water flatfish, *Pleuronectes americanus*, as a bioindicator of subtle environmental change. J Parasitol 93:1227–1230

Khan RA, Kiceniuk J (1983) Effects of crude oils on the gastrointestinal parasites of two species of marine fish. J Wildl Dis 19:253–258

Khan RA, Kiceniuk J (1984) Histopathological effects of crude oil on Atlantic cod following chronic exposure. Can J Zoo 62:2038–2043

Khan RA, Kiceniuk JW (1988) Effect of petroleum aromatic hydrocarbons on mongeneids parasitizing Atlantic cod, *Gadus morhua* L. Bull Environ Contam Toxicol 41:94–100

Khan RA, Thulin J (1991) Influence of pollution on parasites of aquatic animals. Adv Parasitol 30:201–238

Knudsen R, Amundsen P-A, Klemetsen A (2002) Parasite-induced host mortality: Indirect evidence from a long-term study. Environ Biol Fishes 64:257–265

Kolipinski MC (1969) Gar infested by *Argulus* in the Everglades. QJ Florida Acad Sci 32:39–49

Kritsky DC, Bakenhaster MD, Fajer-Avila EJ, Bullard SA (2010) *Rhabdosynochus* spp. (Monogenoidea: Diplectanidae) infecting the gill lamellae of snooks, *Centropomus* spp. (Perciformes: Centropomidae), in Florida, and redescription of the type species, *R. rhabdosynochus*. J Parasitol 96:879–886

Krkošek M, Ford JS, Morton A, Lele S, Myers RA, Lewis MA (2007) Declining wild salmon populations in relation to parasites from farm salmon. Science 318:1772–1775

La Peyre J, Kristensen H, McDonough K, Cooper R (1998) Effects of protease inhibitors on the oyster pathogen *Perkinsus marinus* and oyster cells in vitro. In: Kane AS, Poynton SL (eds) Proc 3rd Int Symp Aquatic Animal Health. Aquatic Pathology Center (APC) Press, Baltimore, MD, USA. 151 p

Labrie L, Komar C, Terhune J, Camus A, Wise D (2004) Effect of sublethal exposure to the trematode *Bolbophorus* spp. on the severity of enteric septicemia of catfish in channel catfish fingerlings. J Aquat Anim Health 16:231–237

Landsberg JH (1995) Tropical reef-fish disease outbreaks and mass mortalities in Florida, USA: What is the role of dietary biological toxins? Dis Aquat Organ 22:83–100

Landsberg JH, Blakesley BA, Reese RO, McRae G, Forstchen PR (1998) Parasites of fish as indicators of environmental stress. Environ Monit Assess 51:211–232

Landsberg JH, Balazs GH, Steidinger KA, Baden DG, Work TM, Russell DJ (1999) The potential role of natural tumor promoters in marine turtle fibropapillomatosis. J Aquat Anim Health 11:199–210

Landsberg JH, Hall S, Johannessen JN, White KD, Conrad SM, Abbott JP, Flewelling LJ, Richardson RW, Dickey RW, Jester ELE, Etheridge SM, Deeds JR, Van Dolah FM, Leighfield TA, Zou Y, Beaudry CG, Benner RA, Rogers PL, Scott PS, Kawabata K, Wolny JL, Steidinger KA (2006) Saxitoxin puffer fish poisoning in the United States, with the first report of *Pyrodinium bahamense* as the putative toxin source. Environ Health Perspect 144:1502–1507

Landsberg JH, Flewelling LJ, Naar J (2009) *Karenia brevis* red tides, brevetoxins in the food web, and impacts on natural resources: Decadal advancements. Harmful Algae 8:598–607

Lauckner G (1985) Diseases of Aves (marine birds). In: Kinne O (ed) Diseases of marine animals, Vol 4, Part 2, Introduction: Reptilia, Aves, Mammalia. Biologische Anstalt Helgoland, Hamburg, Germany, pp 627–643

Lawler AR (1980) Studies on *Amyloodinium ocellatum* (Dinoflagellata) in Mississippi Sound: Natural and experimental hosts. Gulf Caribb Res 6:403–413

Laws EA (2000) Aquatic pollution: An introductory text. John Wiley & Sons, New York, NY, USA. 672 p

Lerdo de Tejada MM (1850) De una noticia de los descubrimientos hechos en las islas y en el continente Americano, y de las providencias dictadas por los Reyes de España para el gobierno de sus nuevas posesiones, desde el primer viage de Don Cristobal Colon, hasta que se emprendió la conquista de Mexico. Apuntes históricos de la heróica ciudad de Vera-Cruz, Imp. de I. Cumplido, México

Lester RJG (1984) A review of methods for estimating mortality due to parasites in wild fish populations. Helgol Meeresunters 37:53–64

Lewis DH, Udey LR (1978) Meningitis in fish caused by an asporogenous anaerobic bacterium. Fish Disease Leaflet 56. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC, USA. 5 p

Lewis DH, Grumbles LC, McConnell S, Flowers AI (1970) *Pasteurella*-like bacteria from an epizootic in menhaden and mullet in Galveston Bay. J Wildl Dis 6:160–162

Lewis M, Jordan S, Chancy C, Harwell L, Goodman L, Quarles R (2007) Summer fish community of the coastal northern Gulf of Mexico: Characterization of a large-scale trawl survey. Trans Am Fish Soc 136:829–845

Lewis MA, Goodman LR, Chancy CA, Jordan SJ (2011) Fish assemblages in three Northwest Florida urbanized bayous before and after two hurricanes. J Coastal Res 27:35–45

Lewitus A, Bargu S, Byrd M, Dorsey C, Flewellin L, Flowers A, Heil C, Kovach C, Lovko V, Steidinger K (2014) Resource Guide for Harmful Algal Bloom Toxin Sampling and Analysis. Alliance, Gulf of Mexico, http://gulfofmexicoalliance.org/documents/pits/wq/goma_hab_toxin_resource_guide.pdf. Accessed on January 15, 2015

Li T, Brouwer M (2013) Gene expression profile of hepatopancreas from grass shrimp *Palaemonetes pugio* exposed to cyclic hypoxia. Comp Biochem Physiol Part D: Genomics Proteomics 8:1–10

Lightner DV (1996) A handbook of shrimp pathology and diagnostic procedures for disease of cultured penaeid shrimp. World Aquaculture Society, Baton Rouge, LA, USA

Lightner DV (2011) Virus diseases of farmed shrimp in the Western Hemisphere (the Americas): A review. J Invertebr Pathol 106:110–130

Lightner DV, Lewis DH (1975) A septicemic bacterial disease syndrome of penaeid shrimp. Mar Fish Rev 37:25–28

Lightner DV, Redman RM, Pantoja CR, Tang KFJ, Noble BL, Schofield P, Mohney LL, Navarro SA (2012) Historic emergence, impact and current status of shrimp pathogens in the Americas. J Invertebr Pathol 110:174–183

Lipp EK, Rodriguez-Palacios C, Rose JB (2001) Occurrence and distribution of the human pathogen *Vibrio vulnificus* in a subtropical Gulf of Mexico estuary. Hydrobiologia 460:165–173

Lipscomb TP, Schulman FY, Moffett D, Kennedy S (1994) Morbilliviral disease in Atlantic bottlenose dolphins (*Tursiops truncatus*) from the 1987-1988 epizootic. J Wildl Dis 30:567–571

Lipscomb TP, Kennedy S, Moffett D, Krafft A, Klaunberg BA, Lichy JH, Regan GT, Worthy GAJ, Taubenberger JK (1996) Morbilliviral epizootic in bottlenose dolphins of the Gulf of Mexico. J Vet Diagn Invest 8:283–290

Litaker W, Steidinger KA, Mason PL, Landsberg JH, Shields JD, Reece KS, Haas LW, Vogelbein WK, Vandersea MW, Kibler SR, Tester PA (2005) The reclassification of *Pfiesteria shumwayae* (Dinophyceae): *Pseudopfiesteria*, gen. nov. J Phycol 41:643–651

Livingston RJ (2001) Eutrophication processes in coastal systems: Origin and succession of plankton blooms and effects on secondary production in Gulf Coast estuaries. CRC Press, Boca Raton, FL, USA. 327 p

Lloyd R (1987) Special tests in aquatic toxicity for chemical mixtures: Interactions and modification of response by variation of physicochemical conditions. In: Vouk VB, Butler GC, Upton AC, Parke DV, Asher SC (eds) Methods for Assessing the Effects of Mixtures of Chemicals. John Wiley & Sons, New York, NY, USA, pp 491–507

Loesch H (1960) Sporadic mass shoreward migrations of demersal fish and crustaceans in Mobile Bay, Alabama. Ecology 41:292–298

Lom J, Dyková I (1992) Protozoan parasites of fishes. Elsevier Science Publishers, Amsterdam, The Netherlands. 315 p

Lom J, Nigrelli RF (1970) *Brooklynella hostilis* n.g., n.sp., a pathogenic cyrtophorine ciliate in marine fishes. J Protozool 17:224–232

Lotz JM, Overstreet RM (1990) Parasites and predators. In: Chàvez JC, Sosa NO (eds) The aquaculture of shrimp, prawn and crawfish in the world: Basics and technologies. Midori Shobo Co, Ltd, Ikebukuro, Toshima-ku Tokyo, Japan, pp 96–121 [In Japanese]

Lu YA, Wang Y, Aguirre AA, Zhao ZS, Liu CY, Nerurkar VR, Yanagihara R (2003) RT-PCR detection of the expression of the polymerase gene of a novel reptilian herpesvirus in tumor tissues of green turtles with fibropapilloma. Arch Virol 148:1155–1163

Lucke B (1942) Tumors of the nerve sheath in fish of the snapper family (Lutianidae). Arch Pathol 34:133–150

Macintyre HL, Stutes AL, Smith WL, Dorsey CP, Abraham A, Dickey RW (2011) Environmental correlates of community composition and toxicity during a bloom of *Pseudo-nitzschia* spp. in the northern Gulf of Mexico. J Plankton Res 33:273–295

MacKenzie CL Jr, Wakida-Kusunoki AI (1997) The oyster industry of eastern Mexico. Mar Fish Rev 59:1–13

MacKenzie K, Williams HH, Williams B, McVicar AH, Siddall R (1995) Parasites as indicators of water quality and the potential use of helminth transmission in marine pollution studies. Adv Parasitol 35:85–144

Magaña HA, Contreras C, Villareal TA (2003) A historical assessment of *Karenia brevis* in the Western Gulf of Mexico. Harmful Algae 2:163–171

Maier Brown AF, Dortch Q, Van Dolah FM, Leighfield TA, Morrison W, Thessen AE, Steidinger K, Richardson B, Moncreiff CA, Pennock JR (2006) Effect of salinity on the distribution, growth, and toxicity of *Karenia* spp. Harmful Algae 5:199–212

Malins DC, McCain BB, Brown DW, Chan SL, Myers MS, Landahl JT, Prohaska PG, Friedman AJ, Rhodes LD, Burrows DG, Gronlund WD, Hodgins HO (1984) Chemical pollutants in sediments and diseases of bottom-dwelling fish in Puget Sound, Washington. Environ Sci Technol 18:705–713

Manter HW (1954) Trematoda of the Gulf of Mexico. Fish Bull 55:335–350

Marcogliese DJ, Nagler JJ, Cyr DG (1998) Effects of exposure to contaminated sediments on the parasite fauna of American Plaice (*Hippoglossoides platessoides*). Bull Environ Contam Toxicol 61:88–95

May EB (1973) Extensive oxygen depletion in Mobile Bay, Alabama. Limnol Oceanogr 18:353–366

May RM, Anderson RM (1978) Regulation and stability of host-parasite population interactions: II. Destabilizing processes. J Anim Ecol 47:249–267

McCain BB, Brown DW, Hom T, Myers MS, Pierce SM, Collier TK, Stein JE, Chan S-L, Varanasi U (1996) Chemical contaminant exposure and effects in four fish species from Tampa Bay, Florida. Estuaries 19:86–104

McCallum H, Dobson A (1995) Detecting disease and parasite threats to endangered species and ecosystems. Trends Ecol Evol 10:190–194

McEachron LW, Matlock GC, Bryan CE, Unger P, Cody TJ, Martin JH (1994) Winter mass mortality of animals in Texas bays. Northeast Gulf Sci 13:121–138

McFarland WN (1965) The effect of hypersalinity on serum and muscle ion concentrations in the striped mullet, *Mugil cephalus* L. Publ Inst Mar Sci Univ Texas 10:179–186

McLean E, Salze G, Craig SR (2008) Parasites, diseases and deformities of cobia. Ribarstvo 66:1–16

Mellergaard S, Nielsen E (1995) Impact of oxygen deficiency on the disease status of common dab *Limanda limanda*. Dis Aquat Organ 22:101–114

Meyers TR, Hendricks JD (1982) A summary of tissue lesions in aquatic animals induced by controlled exposures to environmental contaminants, chemotherapeutic agents, and potential carcinogens [Toxicity to fishes]. Mar Fish Rev 44:1–17

Meyers TR, Hauck AK, Blankenbeckler WD, Minicucci T (1986) First report of viral erythrocytic necrosis in Alaska, USA, associated with epizootic mortality in Pacific herring, *Clupea harengus pallasi* (Valenciennes). J Fish Dis 9:479–491

Meyers TR, Short S, Lipson K, Batts WN, Winton JR, Wilcock J, Brown E (1994) Association of viral hemorrhagic septicemia virus with epizootic hemorrhages of the skin in Pacific herring *Clupea harengus pallasi* from Prince William Sound and Kodiak Island, Alaska, USA. Dis Aquat Organ 19:27–37

Miglarese JV, Shealy MH (1974) Incidence of microsporidean and trypanorhynch cestodes in white shrimp, *Penaeus setiferus* Linnaeus, in South Carolina estuaries. SC Acad Sci Bull 36:93

Miller EM (1940) Mortality of fishes due to cold on the southeast Florida coast, 1940. Ecology 21:420–421

Mitchell AJ, Overstreet RM, Goodwin AE (2009) *Eustrongylides ignotus* infecting commercial bass (*Morone chrysops* female × *Morone saxatilis* male) and other fishes in the southeastern USA. J Fish Dis 32:795–799

Moles A, Wade TL (2001) Parasitism and phagocytic function among sand lance *Ammodytes hexapterus* Pallas exposed to crude oil-laden sediments. Bull Environ Contam Toxicol 66:528–535

Möller H (1985) A critical review on the role of pollution as a cause of fish diseases. In: Ellis AE (ed) Fish and shellfish pathology. Academic Press, London, UK, pp 169–182

Monserrat JM, Martínez PE, Geracitano LA, Amado LL, Martins CMG, Pinho GLL, Chaves IS, Ferreira-Cravo M, Ventura-Lima J, Bianchini A (2007) Pollution biomarkers in estuarine animals: Critical review and new perspectives. Comp Biochem Physiol C Toxicol Pharmacol 146:221–234

Moore CJ, Posey CR Sr (1974) Pigmentation and morphological abnormalities in the hogchoker, *Trinectes maculatus* (Pisces, Soleidae). Copeia 1974:660–670

Muncy RJ (1984) Species profiles: Life histories and environmental requirements of coastal fishes and invertebrates (South Atlantic). White shrimp [*Penaeus setiferus*]. U.S. Fish and Wildlife Service FWS/OBS-82/11.27. U.S. Army Corps of Engineers TR EL-82-4, pp 1–19

Murchelano RA, Wolke RE (1991) Neoplasms and nonneoplastic liver lesions in winter flounder, *Pseudopleuronectes americanus*, from Boston Harbor, Massachusetts. Environ Health Perspect 90:17–26

Mydlarz LD, Holthouse SF, Peters EC, Harvell CD (2008) Cellular responses in sea fan corals: Granular amoebocytes react to pathogen and climate stressors. PLoS One 3(3):e1811

Myers MS, Landahl JT, Krahn MM, Johnson LL, McCain BB (1990) Overview of studies on liver carcinogenesis in English sole from Puget Sound: Evidence for a xenobiotic chemical etiology. 1: Pathology and epizootiology. Sci Total Environ 94:33–50

Naar JP, Flewelling LJ, Lenzi A, Abbott JP, Granholm A, Jacocks HM, Gannon D, Henry M, Pierce R, Baden DG, Wolny J, Landsberg JH (2007) Brevetoxins, like ciguatoxins, are potent ichthyotoxic neurotoxins that accumulate in fish. Toxicon 50:707–723

Natarajan P, Ramadhas V, Ramanathan N (1982) A case report of mass mortality of marine catfish. Sci Cult 48:182–183

Ndomahina ET (1994) An investigation into reported mass mortalities of catfishes of the genus *Arius* in the coastal waters of Sierra Leone. Preliminary Report, Institute of Marine Biology and Oceanography, Fourah Bay College, Freetown, Sierra Leone

Nieland DL, Wilson CA (1995) Histological abnormalities and bacterial proliferation in red drum and black drum ovaries. Trans Am Fish Soc 124:139–143

NOAA (National Oceanic and Atmospheric Administration) (1997) NOAA's estuarine eutrophication survey, Vol 4: Gulf of Mexico region. Office of Ocean Resources Conservation and Assessment, Silver Spring, MD, USA. 77 p

Noreña-Barroso E, Gold-Bouchot G, Zapata-Perez O, Sericano JL (1999) Polynuclear aromatic hydrocarbons in American oysters *Crassostrea virginica* from the Terminos Lagoon, Campeche, Mexico. Mar Pollut Bull 38:637–645

Norman JR (1934) A systematic monograph of the flatfishes (Heterosomata), Vol 1: Psettosisae, Bothidae, Pleuronectidae. British Museum (Natural History), Adlard and Son, Ltd, London, UK. 459 p

Nuñez Ortega A (1878) Ensayo de una explicación del orígen de las grandes mortandades de peces en el Golfo de México. La Naturaleza 4:188–197

Obiekezie AI, Möller H, Anders K (1988) Diseases of the African estuarine catfish *Chrysichthys nigrodigitatus* (Lacépède) from the Cross River estuary, Nigeria. J Fish Biol 32:207–221

Okolodkov YB, Campos-Bautista G, Garate-Lizarraga I, Gonzalez-Gonzalez JAG, Hoppenrath M, Arenas V (2007) Seasonal changes of benthic and epiphytic dinoflagellates in the Veracruz reef zone, Gulf of Mexico. Aquat Microb Ecol 47:223–237

Oliveira MFT, Hawkins WE, Overstreet RM, Walker WW (1994) Hepatocellular neoplasm in a wild-caught sheepshead minnow (*Cyprinodon variegatus*) from the northern Gulf of Mexico. Gulf Res Rep 9:65–67

Ostrander GK, Rotchell JM (2005) Fish models of carcinogenesis (Chapter 9). In: Mommsen TP, Moon TW (eds) Biochemistry and Molecular Biology of Fishes, Vol 6: Environmental Toxicology. Elsevier BV, Amsterdam, The Netherlands, pp 255–288

Ostrander GK, Di Giulio RT, Hinton DE, Miller MR, Rotchell JR (2007) Chemical carcinogensis. In: DiGiulio RT, Hinton DE (eds) The toxicology of fishes. CRC Press, Boca Raton, FL, USA, pp 531–597

Overstreet RM (1973) Parasites of some penaeid shrimps with emphasis on reared hosts. Aquaculture 2:105–140

Overstreet RM (1974) An estuarine low-temperature fish-kill in Mississippi, with remarks on restricted necropsies. Gulf Res Rep 4:328–350

Overstreet RM (1977) *Poecilancistrium caryophyllum* and other trypanorhynch cestode plerocercoids from the musculature of *Cynoscion nebulosus* and other sciaenid fishes in the Gulf of Mexico. J Parasitol 63:780–789

Overstreet RM (1978) Marine maladies? Worms, germs, and other symbionts from the northern Gulf of Mexico. Mississippi-Alabama Sea Grant Consortium, MASGP-78-021. 140 p

Overstreet RM (1982) Abiotic factors affecting marine parasitism. In: Mettrick DF, Desser SS (eds) Parasites—Their world and ours. Fifth International Congress of Parasitology Proceedings and Abstracts, Vol 2, August 7–14, Toronto, Canada. Elsevier Biomedical Press, New York, NY, USA, pp 36–39

Overstreet RM (1983a) Aspects of the biology of the spotted seatrout, *Cynoscion nebulosus*, in Mississippi. Gulf Res Rep Suppl 1:1–43

Overstreet RM (1983b) Aspects of the biology of the red drum, *Sciaenops ocellatus*, in Mississippi. Gulf Res Rep Suppl 1:45–68

Overstreet RM (1983c) Metazoan symbionts of crustaceans (Chapter 4). In: Provenzano AJ (ed) The biology of Crustacea: Pathobiology, Vol 6. Academic Press, New York, NY, USA, pp 155–250

Overstreet RM (1987) Solving parasite-related problems in cultured Crustacea. Int J Parasitol 17:309–318

Overstreet RM (1988) Aquatic pollution problems, southeastern U.S. coasts: Histopathological indicators. Aquat Toxicol 11:213–239

Overstreet RM (1993) Parasitic diseases of fishes and their relationship with toxicants and other environmental factors (Chapter 5). In: Couch JA, Fournie JW (eds) Pathobiology of marine and estuarine organisms. CRC Press, Boca Raton, FL, USA, pp 111–156

Overstreet RM (1994) BP (*Baculovirus penaei*) in penaeid shrimps. USMSFP 10th Anniversary Review GCRL Special Publication 1:97–106

Overstreet RM (1997) Parasitological data as monitors of environmental health. Parassitologia 39:169–175

Overstreet RM (2003) Presidential address: Flavor buds and other delights. J Parasitol 89:1093–1107

Overstreet RM (2007) Effects of a hurricane on fish parasites. Parassitologia 49:161–168

Overstreet RM (2013) Waterborne parasitic diseases in ocean. In: Kanki P, Grimes DJ (eds) Infectious diseases: Selected entries from the encyclopedia of sustainability science and technology. SpringerScience+Business Media, New York, NY, USA, pp 431–496

Overstreet RM, Curran SS (2004) Defeating diplostomoid dangers in USA catfish aquaculture. Folia Parasitol 51:153–165

Overstreet RM, Curran SS (2005) Parasites of the American white pelican. Gulf Caribb Res 17:31–48

Overstreet RM, Edwards RH (1976) Mesenchymal tumors of some estuarine fishes of the northern Gulf of Mexico. II. Subcutaneous fibromas in the southern flounder, *Paralichthys lethostigma*, and the sea catfish, *Arius felis*. Bull of Mar Sci 26:41–48

Overstreet RM, Howse HD (1977) Some parasites and diseases of estuarine fishes in polluted habitats of Mississippi. Ann New York Acad Sci 298:427–462

Overstreet RM, Lotz JM (2016) Host-symbiont relationships: Understanding the change from guest to pest (Chapter 2). In: Hurst CJ (ed) The Rasputin effect: When commensals and symbionts become parasitic, Vol 3: Advances in Environmental Microbiology. Springer International Publishing Switzerland, pp 27–64

Overstreet RM, Lyles CH (1974) A rubber band around an Atlantic croaker. Gulf Res Rep 4:476–478

Overstreet RM, Monson P (2002) Myxosporan infection expressed by exposure to heavy metals. In: Abstracts of The Tenth International Congress of Parasitology in Conjunction with the 77th Annual Meeting of the American Society of Parasitologists, Vancouver, BC, Canada, August 4–10

Overstreet RM, Rehak E (1982) Case report—heat-stroke in nesting least tern chicks from Gulfport, Mississippi, during June 1980. Avian Dis 26:918–923

Overstreet RM, Van Devender T (1978) Implication of an environmentally induced hamartoma in commercial shrimps. J Invertebr Pathol 31:234–238

Overstreet RM, Self JT, Vilet KA (1985) The pentastomid *Sebekia mississippiens* sp. n. in the American alligator and other hosts. Prac Helminthol Soc Wash 52:266–277

Overstreet RM, Dyková I, Hawkins WE (1992) Branchiura. In: Harrison FW, Humes AG (eds) Microscopic anatomy of invertebrates, Vol 9: Crustacea (Chapter 8). Wiley-Liss, New York, NY, USA, pp 385–413

Overstreet RM, Barnes SS, Manning CS, Hawkins WE (2000) Facilities and husbandry (small fish models) (Chapter 2). In: Ostrander G (ed) Handbook of experimental animals: The laboratory fish. Academic Press Limited, London, UK, pp 41–63

Overstreet RM, Curran SS, Pote LM, King DT, Blend CK, Grater WD (2002) *Bolbophorus damnificus* n. sp. (Digenea: Bolbophoridae) from the channel catfish *Ictalurus punctatus* and American white pelican *Pelecanus erythrorhynchos* in the USA based on life-cycle and molecular data. Syst Parasitol 52:81–96

Overstreet RM, Cook JO, Heard RW (2009) Trematoda (Platyhelminthes) of the Gulf of Mexico. In: Felder DL, Camp DK (eds) Gulf of Mexico—Origin, Waters and Biota, Vol 1: Biodiversity. Texas A&M University Press, College Station, TX, USA, pp 419–488

Paerl HW, Huisman J (2008) Blooms like it hot. Science 320(5872):57–58. doi:10.1126/science.1155398

Paerl HW, Justić D (2011) Primary producers: Phytoplankton ecology and trophic dynamics in coastal waters. In: Wolanski E, McLusky DS (eds) Treatise on Estuarine and Coastal Science, Vol 6, Elsevier Academic Press, Waltham, MA, USA, pp 23–42

Palm HW (2004) The Trypanorhyncha Diesing, 1863. PKSPL-IPB Press, Bogor, Indonesia. 710 p

Palm HW, Overstreet R (2000a) *Otobothrium cysticum* (Cestoda: Trypanorhyncha) from the muscle of butterfishes (Stromateidae). Parasitol Res 86:41–53

Palm HW, Overstreet R (2000b) New records of trypanorhynch cestodes from the Gulf of Mexico, including *Kotorella pronosoma* (Stossich, 1901) and *Heteronybelinia palliata* (Linton, 1924) comb. n. Folia Parasitol 47:293–302

Panek FM (2005) Epizootics and disease of coral reef fish in the Tropical Western Atlantic and Gulf of Mexico. Rev Fish Sci 13:1–21

Paperna I, Overstreet RM (1981) Parasites and diseases of mullets (Mugilidae). In: Oren OH (ed) Aquaculture of grey mullets. Cambridge University Press, Great Britain, UK pp 411–493

Paperna I, Diamant A, Overstreet RM (1984) Monogenean infestations and mortality in wild and cultured Red Sea fishes. Helgol Meeresunters 37:445–462

Parker JH, Curran SS, Overstreet RM, Tkach VV (2010) Examination of *Homalometron elongatum* Manter, 1947 and description of a new congener from *Eucinostomus currani* Zahuranec, 1980 in the Pacific Ocean off Costa Rica. Comp Parasitol 77:154–163

Patterson KL, Porter JW, Ritchie KB, Polson SW, Mueller E, Peters EC, Santavy DL, Smith GW (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci 99:8725–8730

Pearson JC (1929) Natural history and conservation of the redfish and other commercial sciaenids on the Texas coast. Bull Bur Fish 44:129–214

Pech D, Vidal-Martínez VM, Aguirre-Macedo ML, Gold-Bouchot G, Herrera-Silveira J, Zapata-Pérez O, Marcogliese DJ (2009) The checkered puffer (*Spheroides testudineus*) and its helminths as bioindicators of chemical pollution in Yucatan coastal lagoons. Sci Total Environ 407:2315–2324

Pennycuick L (1971) Seasonal variations in the parasite infections in a population of three-spined sticklebacks, *Gasterosteus aculeatus* L. Parasitology 63:373–388

Perera FP (1997) Environment and cancer: Who are susceptible? Science 278:068–1073
Pérez-del Olmo A, Raga JA, Kostadinova A, Fernández M (2007) Parasite communities in *Boops boops* (L.) (Sparidae) after the Prestige oil-spill: Detectable alterations. Mar Pollut Bull 54:266–276
Perkins EJ, Gilchrist JRS, Abbott OJ (1972) Incidence of epidermal lesions in fish of the north-east Irish Sea area, 1971. Nature 238:101–103
Perry HM, Warren J, Trigg C, Devender TV (1998) The blue crab fishery of Mississippi. J Shellfish Res 17:425–434
Pinckney JL, Paerl HW, Tester P, Richardson TL (2001) The role of nutrient loading and eutrophication in estuarine ecology. Environ Health Perspect 109:699–706
Plumb JA, Schachte JH, Gaines JL, Peltier W, Carroll B (1974) *Streptococcus* sp. from marine fishes along the Alabama and northwest Florida coast of the Gulf of Mexico. Trans Am Fish Soc 103:358–361
Poore GC, Bruce NL (2012) Global diversity of marine isopods (except Asellota and crustacean symbionts). PLoS One 7(8):e43529
Porter JW, Dustan P, Jaap WC, Patterson KL, Kosmynin V, Meier OW, Patterson ME, Parsons M (2001) Patterns of spread of coral disease in the Florida Keys. In: Porter JW (ed) The ecology and etiology of newly emerging marine diseases. Kluwer Academic Publishers, Dordrecht, The Netherlands, pp 1–24
Quackenbush SL, Work TM, Balazs GH, Casey RN, Rovnak J, Chaves A, duToit L, Baines JD, Parrish CR, Bowser PR, Casey JW (1998) Three closely related herpesviruses are associated with fibropapillomatosis in marine turtles. Virology 246:392–399
Quackenbush SL, Casey RN, Murcek RJ, Paul TA, Work TM, Limpus CJ, Chaves A, duToit L, Perez JV, Aguirre AA, Spraker TR, Horrocks JA, Vermeer LA, Balazs GH, Casey JW (2001) Quantitative analysis of herpesvirus sequences from normal tissue and fibropapillomas of marine turtles with real-time PCR. Virology 287:105–111
Quick JA, Henderson GE (1974) Effects of *Gymnodinium breve* red tide on fishes and birds: A preliminary report on behavior, anatomy, hematology, and histopathology. In Amborski RL, Hood MA, Miller RR (eds), Proceedings of Gulf Coast Regional Symposium on Diseases of Aquatic Animals. Pub # LSU-SG-74-05. Center for Wetland Resources, Louisiana State University, Baton Rouge, LA, USA, pp 85–114
Quick JA, Henderson GE (1975) Evidences of new ichthyointoxicative phenomena in *Gymnodinium breve* red tides. In: LoCicero VR (ed) Proceedings of the First International Conference on Toxic Dinoflagellate Blooms, November 1974, Boston, MA. MA Science and Technology Foundation, Wakefield, MA, USA, pp 413–422
Rabalais NN, Turner RE, Wiseman WJ Jr (2002) Gulf of Mexico hypoxia, AKA "The dead zone". Annu Rev Ecol Syst 33:235–263
Rasheed V, Plumb JA (1984) Pathogenicity of a non-haemolytic group B *Streptococcus* sp. in Gulf killifish (*Fundulus grandis* Baird and Girard). Aquaculture 37:97–105
Raustiala K (1997) States, NGOs, and international environmental institutions. Int Studies Q 41:719–740
Ray SM, Aldrich DV (1965) *Gymnodinium breve*: Induction of shellfish poisoning in chicks. Science 148:1748–1749
Reece KS, Bushek D, Hudson KL, Graves JE (2001) Geographic distribution of *Perkinsus marinus* genetic strains along the Atlantic and Gulf coasts of the USA. Mar Biol 139:1047–1055
Renegar DA, Blackwelder PL, Miller JD, Gochfeld DJ, Moulding AL (2008) Ultrastructural and histological analysis of Dark Spot Syndrome in *Siderastrea siderea* and *Agaricia*

agaricites. In: Proceedings of the 11th International Coral Reef Symposium, Ft. Lauderdale, FL, USA, July 7–11, pp 185–189
Rigdon RH, Hendricks JW (1955) Myxosporidia in fish in waters emptying into Gulf of Mexico. J Parasitol 41:511–518
Roberts HH (2011) Surficial geology of the northern Gulf of Mexico continental slope. In: Buster NA, Holmes CW (eds) Gulf of Mexico Origin, Waters, and Biota, Vol 3: Geology. Texas A&M University Press, College Station, TX, USA, pp 209–228
Roberts HH, Rouse LJ, Walker ND, Hudson JH (1982) Cold-water stress in Florida Bay and northern Bahamas: A product of winter cold-air outbreaks. J Sediment Petrol 52:145–155
Robins RC (1957) Effects of storms on the shallow-water fish fauna of southern Florida with new records of fishes from Florida. Bull Mar Sci 7:266–275
Roessler MA, Tabb DC (1974) Studies of effects of thermal pollution in Biscayne Bay, Florida. EPA-660 / 3-74-014. U.S. Environmental Protection Agency, Washington, DC, USA
Rotstein DS, Burdett LG, McLellan W, Schwacke L, Rowles T, Terio KA, Schultz S, Pabst A (2009) Lobomycosis in offshore bottlenose dolphins (*Tursiops truncatus*), North Carolina. Emerg Infect Dis 15:588–590
Rousset F, Thomas F, De Meeûs T, Renaud F (1996) Inference of parasite-induced host mortality from distributions of parasite loads. Ecology 77:2203–2211
Salgado-Maldonado G, Amin OM (2009) Acanthocephala of the Gulf of Mexico. In: Felder DL, Camp DK (eds) Gulf of Mexico Origin, Waters, and Biota, Vol 1: Biodiversity. Texas A&M University Press, College Station, TX, USA, pp 539–552
Samuel WM, Williams ES, Rippin AB (1982) Infestations of *Piagetiella peralis* (Mallophaga: Menoponidae) on juvenile white pelicans. Can J Zool 60:951–953
Sawyer RT, Lawler AR, Overstreet RM (1975) Marine leeches of the eastern United States and the Gulf of Mexico with a key to the species. J Nat Hist 9:633–667
Schmale MC, Hensley T (1988) Transmissibility of a neurofibromatosis-like disease in bicolor damselfish. Cancer Res 48:3828–3833
Schmale MC, Hensley G, Udey LR (1983) Multiple schwannomas in the bicolor damselfish, *Pomacentrus partitus* (Pisces, Pomacentridae). Am J Path 112:238–241
Schmale MC, Hensley GT, Udey LR (1986) Neurofibromatosis in the bicolor damselfish (*Pomacentrus partitus*) as a model of von Recklinghausen neurofibromatosis. Ann New York Acad Sci 486:386–402
Schmale MC, Aman MR, Gill KA (1996) A retrovirus isolated from cell lines derived from neurofibromas in bicolor damselfish (*Pomacentrus partitus*). J Gen Virol 77:1181–1187
Schmale MC, Gibbs PD, Campbell CE (2002) A virus-like agent associated with neurofibromatosis in damselfish. Dis Aquat Organ 49:107–115
Schotte M, Markham JC, Wilson GDF (2009) Isopoda (Crustacea) of the Gulf of Mexico. In: Felder DL, Camp DK (eds) Gulf of Mexico origin, waters, and biota, Vol 1: Biodiversity. Texas A&M University Press, College Station, TX, USA, pp 973–986
Scott ME (1987) Temporal changes in aggregation: A laboratory study. Parasitology 94:583–595
Scott ME, Dobson A (1989) The role of parasites in regulating host abundance. Parasitol Today 5:176–183
Shaw DJ, Dobson AP (1995) Patterns of macroparasite abundance and aggregation in wildlife populations: A quantitative review. Parasitology 111:S111–S133
Shaw DJ, Grenfell BT, Dobson AP (1998) Patterns of macroparasite aggregation in wildlife host populations. Parasitology 117:597–610

Shelton RGJ, Wilson KW (1973) On the occurrence of lymphocystis, with notes on other pathological conditions, in the flatfish stocks of the north-east Irish Sea. Aquaculture 2:395–410

Sherwood MJ, Mearns AJ (1977) Environmental significance of fin erosion in Southern California demersal fishes. Ann New York Acad Sci 298:177–189

Shields JD, Overstreet RM (2007) Diseases, parasites, and other symbionts. In: Kennedy VS, Cronin LE (eds) The blue crab, *Callinectes sapidus*. Maryland Sea Grant, College Park, MD, USA, pp 223–339

Sindermann CJ, Lightner DV (1988) Disease diagnosis and control in North American marine aquaculture. Elsevier, New York, NY, USA. 431 p

Skinner RH (1982) The interrelation of water quality, gill parasites, and gill pathology of some fishes from South Biscayne Bay, Florida. Fish Bull 80:269–280

Skinner RH, Kandrashoff W (1988) Abnormalities and diseases observed in commercial fish catches from Biscayne Bay, Florida. Water Resour Bull 24:961–966

Slooff W (1982) Skeletal anomalies in fish from polluted surface waters. Aquat Toxicol 2:157–173

Smit NJ, Bruce NL, Hadfield KA (2014) Global diversity of fish parasitic isopod crustaceans of the family Cymothoidae. Int J Parasitol Parasites Wild 3:188–197

Smith CE, Peck TH, Klauda RJ, McLaren JB (1979) Hepatomas in Atlantic tomcod *Microgadus tomcod* (Walbaum) collected in the Hudson River estuary in New York. J Fish Dis 2:313–319

Smith FG (1970) A preliminary report on the incidence of lymphocystis disease in the fish of the Sapelo Island, Georgia, area. J Wildl Dis 6:469–471

Smith GM, Coates CW (1938) Fibro-epithelial growths on the skin in large marine turtles, *Chelonia mydas* (Linnaeus). Zoologica 23:93–98

Smith TJ, Robblee MB, Wanless HR, Doyle TW (1994) Mangroves, hurricanes, and lightning strikes. BioScience 44:256–262

Sokolova Y, Pelin A, Hawke J, Corradi N (2015) Morphology and phylogeny of *Agmasoma penaei* (Microsporidia) from the type host, *Litopenaeus setiferus*, and the type locality, Louisiana, USA. Int J Parasitol 45:1–16

Solangi MA, Overstreet RM (1980) Biology and pathogenesis of the coccidium *Eimeria funduli* infecting killifishes. J Parasitol 66:513–526

Solangi MA, Overstreet RM (1982) Histopathological changes in two estuarine fishes, *Menidia beryllina* (Cope) and *Trinectes maculatus* (Bloch and Schneider), exposed to crude oil and its water-soluble fractions. J Fish Dis 5:13–35

Solangi MA, Overstreet RM, Fournie JW (1982) Effect of low temperature on development of the coccidium *Eimeria funduli* in the Gulf killifish. Parasitology 84:31–39

Song JY, Kitamura S, Oh MJ, Kang HS, Lee JH, Tanaka SJ, Jung SJ (2009) Pathogenicity of *Miamiensis avidus* (syn. *Philasterides dicentrarchi*), *Pseudocohnilembus persalinus*, *Pseudocohnilembus hargisi* and *Uronema marinum* (Ciliophora, Scuticociliatida). Dis Aquat Organ 83:133–143

Soniat TM (1996) Epizootiology of *Perkinsus marinus* disease of eastern oysters in the Gulf of Mexico. J Shellfish Res 15:35–43

Sosa ER, Landsberg JH, Kiryu Y, Stephenson CM, Cody TT, Dukeman AK, Wolfe HP, Vandersea MW, Litaker RW (2007) Pathogenicity studies with the Fungi *Aphanomyces invadans*, *Achlya bisexualis*, and *Phialemonium dimorphosporum*: Induction of skin ulcers in striped mullet. J Aquat Anim Health 19:41–48

Stacy BA, Wellehan JFX, Foley AM, Coberley SS, Herbst LH, Manire CA, Garner MM, Brookins MD, Childress AL, Jacobson ER (2008) Two herpesviruses associated with disease in wild Atlantic loggerhead sea turtles (*Caretta caretta*). Vet Microbiol 126:63–73

Stacy BA, Foley AM, Greiner E, Herbst LH, Bolten A, Klein P, Manire CA, Jacobson ER (2010) Spirorchiidiasis in stranded loggerhead *Caretta caretta* and green turtles *Chelonia mydas* in Florida (USA): Host pathology and significance. Dis Aquat Organ 89:237–259

Stamper MA, Lewbart GA, Barrington PR, Harms CA, Geoly F, Stoskopf MK (1998) *Eimeria southwelli* infection associated with high mortality of cownose rays. J Aquat Anim Health 10:264–270

Steidinger KA, Wolny JL, Haywood AJ (2008) Identification of Kareniaceae (Dinophyceae) in the Gulf of Mexico. Nova Hedwigia Beih 133:269–284

Stevenson C, Childers DL (2004) Hydroperiod and seasonal effects on fish decomposition in an oligotrophic everglades marsh. Wetlands 24:529–537

Stone I, Heard RW (1989) *Excorallana delaneyi*, n. sp. (Crustacea: Isopoda: Excorallanidae) from the northeastern Gulf of Mexico, with observations on adult characters and sexual dimorphism in related species of *Excorallana* Stebbing, 1904. Gulf Caribb Res 8:199–211

Storey M, Gudger EW (1936) Mortality of fishes due to cold at Sanibel Island, Florida, 1886-1936. Ecology 17:640–648

Suárez-Morales E, Kim IH, Castellanos I (1998) A new geographic and host record for *Argulus flavescens* Wilson, 1916 (Crustacea, Arguloida), from Southeastern Mexico. Bull Mar Sci 62:293–296

Summers JK (1999) The ecological condition of estuaries in the Gulf of Mexico. U.S. Environmental Protection Agency, EPA 620-R-98-004. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC, USA. 71 p

Sun PL, Hawkins WE, Overstreet RM, Brown-Peterson NJ (2009) Morphological deformities as biomarkers in fish from contaminated rivers in Taiwan. Int J Environ Res Public Health 6:2307–2331

Sures B (2004) Environmental parasitology: Relevancy of parasites in monitoring environmental pollution. Trends Parasitol 20:170–177

Szmant AM, Gassman NJ (1990) The effects of prolonged "bleaching" on the tissue biomass and reproduction of the reef coral *Montastrea annularis*. Coral Reefs 8:217–224

Tabb DC, Jones AC (1962) Effect of Hurricane Donna on the aquatic fauna of North Florida Bay. Trans Am Fish Soc 91:375–378

Tao Z, Larsen AM, Bullard SA, Wright AC, Arias CR (2012) Prevalence and population structure of *Vibrio vulnificus* on fishes from the northern Gulf of Mexico. Appl Environ Microbiol 78:7611–7618

Thessen AE, Dortch Q, Parsons ML, Morrison W (2005) Effect of salinity on *Pseudo-nitzschia* species (Bacillariophyceae) growth and distribution. J Phycol 41:21–29

Thessen AE, Bowers HA, Stoecker DK (2009) Intra-and interspecies differences in growth and toxicity of *Pseudo-nitzschia* while using different nitrogen sources. Harmful Algae 8:792–810

Thomas LP, Moore DR, Work RC (1961) Effects of Hurricane Donna on the turtle grass beds of Biscayne Bay, Florida. Bull Mar Sci 11:191–197

Thomas NJ, Hunter DB, Atkinson CT (eds) (2007) Infectious diseases of wild birds. Blackwell Publishing, Ames, IA, USA. 484 p

Thronson A, Quigg A (2008) Fifty-five years of fish kills in coastal Texas. Esturaries Coasts 31:802–813

Thulin J (1987) Some diseases and parasites of trout (*Salmo trutta*) attracted to a hot water effluent. In: Stenmark A, Malmberg G (eds) Parasites and diseases in natural waters and aquaculture in Nordic Countries. Zoo-Tax Naturhistoriska riksmuseet, Stockholm, Sweden. 72 p

Thune RL, Fernandez DH, Hawke JP, Miller R (2003) Construction of a safe, stable, efficacious vaccine against *Photobacterium damselae* ssp. *piscicida*. Dis Aquat Organ 57:51–58

Tilmant JT, Curry RW, Jones R, Szmant A, Zieman JC, Flora M, Robblee MB, Smith D, Snow RW, Wanless H (1994) Hurricane Andrew's effects on marine resources. BioScience 44:230–237

Trainer VL, Bates SS, Lundholm N, Thessen AE, Cochlan WP, Adams NG, Trick CG (2012) *Pseudo-nitzschia* physiological ecology, phylogeny, toxicity, monitoring and impacts on ecosystem health. Harmful Algae 14:271–300

Turner RE, Schroeder WW, Wiseman WJ (1987) The role of stratification in the deoxygenation of Mobile Bay and adjacent shelf bottom waters. Estuaries 10:13–19

Twiner MJ, Flewelling LJ, Fire SE, Bowen-Stevens SR, Gaydos JK, Johnson CK, Landsberg JH, Leighfield TA, Mase-Guthrie B, Schwacke L, Van Dolah FM, Wang Z, Rowles TK (2012) Comparative analysis of three brevetoxin-associated bottlenose dolphin (*Tursiops truncatus*) mortality events in the Florida panhandle region (USA). PLoS One 7(8):e42974

Udey LR, Young E, Sallman B (1976) *Eubacterium* sp. ATCC 29255: An anaerobic bacterial pathogen of marine fish. Fish Health News 5:3–4

Udey LR, Young E, Sallman B (1977) Isolation and characterization of an anaerobic bacterium, *Eubacterium tarantellus* sp. nov., associated with striped mullet (*Mugil cephalus*) mortality in Biscayne Bay, Florida. J Fish Board Can 34:402–409

Ulrich PN, Colton CM, Hoover CA, Gaffney PM, Marsh AG (2007) *Haplosporidium nelsoni* (MSX) rDNA detected on oysters from the Gulf of Mexico and the Caribbean Sea. J Shellfish Res 26:195–199

Underwood AJ (1994) On beyond BACI: Sampling designs that might reliably detect environmental disturbances. Ecol Appl 4:3–15

Van der Oost R, Beyer J, Vermeulen NP (2003) Fish bioaccumulation and biomarkers in environmental risk assessment: A review. Environ Toxicol Pharmacol 13:57–149

Van Deventer M, Atwood K, Vargo GA, Flewelling LJ, Landsberg JH, Naar JP, Stanek D (2012) *Karenia brevis* red tides and brevetoxin-contaminated fish: A high risk factor for Florida's scavenging shorebirds? Bot Mar 22:31–37

Van Houtan KS, Hargrove SK, Balazs GH (2010) Land use, macroalgae, and a tumor-forming disease in marine turtles. PLoS One 5(9):e12900

Vandersea MW, Litaker RW, Yonnish B, Sosa E, Landsberg JH, Pullinger C, Moon-Butzin P, Green J, Morris JA, Kator H, Noga EJ, Tester PA (2006) Molecular assays for detecting *Aphanomyces invadans* in ulcerative mycotic fish lesions. Appl Environ Microbiol 72:1551–1557

Vargo GA, Atwood K, Van Deventer M, Harris R (2006) Beached bird surveys on Shell Key, Pinellas County, Florida. Fla Field Nat 34:21–22

Vidal-Martínez VM, Aguirre-Macedo ML, Noreña-Barroso E, Gold-Bouchot G, Caballero-Pinzón PI (2003) Potential interactions between metazoan parasites of the Mayan catfish *Ariopsis assimilis* and chemical pollution in Chetumal Bay, Mexico. J Helminthol 77:173–1847

Vidal-Martínez VM, Centeno-Chalé OA, Torres-Irineo E, Sánchez-Ávila J, Gold-Bouchot G, Aguirre-Macedo ML (2014) The metazoan parasite communities of the shoal flounder (Syacium gunteri) as bioindicators of chemical contamination in the southern Gulf of Mexico. Parasites Vectors 7:541

Viosca P Jr (1945) A critical analysis of practices in the management of warm-water fish with a view to greater food production. Trans Am Fish Soc 73:274–283

Vogelbein WK, Fournie JW, Van Veld PA, Huggett RJ (1990) Hepatic neoplasms in the mummichog *Fundulus heteroclitus* from a creosote-contaminated site. Cancer Res 50:5978–5986

Vogelbein WK, Shields JD, Haas LW, Reece KS, Zwerner DE (2001) Skin ulcers in estuarine fishes: A comparative pathological evaluation of wild and laboratory-exposed fish. Environ Health Perspect 109:687–693

Vogelbein WK, Lovko VJ, Shields JD, Reece KS, Mason PL, Haas LW, Walker CC (2002) *Pfiesteria shumwayae* kills fish by micropredation not exotoxin secretion. Nature 418:967–970

Walsh JJ, Weisberg RH, Lenes JM, Chen FR, Dieterle DA, Zheng L, Carder KL, Vargo GA, Havens JA, Peebles E, Hollander DJ, He R, Heil CA, Mahmoudi B, Landsberg JH (2009) Isotopic evidence for dead fish maintenance of Florida red tides, with implications for coastal fisheries over both source regions of the West Florida shelf and within downstream waters of the South Atlantic Bight. Prog Oceanogr 80:51–73

Waring GT, Josephson E, Fairfield CP, Maze-Foley K (2007) U.S. Atlantic and Gulf of Mexico Marine Mammal Stock Assessments-2006. NOAA Technical Memorandum NMFS-NE-201, April. U.S. Department of Commerce, NMFS Southeast Fisheries Science Center, Miami, FL, USA.

Weinstein MP, Heck KL (1977) Biology and host-parasite relationships of *Cymothoa excisa* (Isopoda, Cymothoidae) with three species of snappers (Lutjanidae) on Caribbean coast of Panama. Fish Bull 75:875–877

Weissenberg R (1945) Studies on virus diseases of fish IV: Lymphocystis disease in Centrarchidae. Zoologica (NY) 30:169–184

Wharton JH, Ellender RD, Middlebrooks BL, Stocks PK, Lawler AR, Howse D (1977) Fish cell culture: Characteristics of a cell line from the silver perch, *Bairdiella chrysura*. In Vitro 13:389–397

White ME, Wilson EA (1996) Predators, pests, and competitors. In: Kennedy VS, Newell RIE, Eble AF (eds) The eastern oyster *Crassostrea virginica*. Maryland Sea Grant College, College Park, MD, USA, pp 559–579

Wilkinson HW, Thacker LG, Facklam RR (1973) Nonhemolytic group B streptococci of human, bovine, and ichthyic origin. Infect Immun 7:496–498

Willcox J (1887) Fish killed by cold along the Gulf of Mexico and coast of Florida. Bull U S Fish Commission 6:123

Willett KL, McDonald SJ, Steinberg MA, Beatty KB, Kennicutt MC, Safe SH (2009) Biomarker sensitivity for polynuclear aromatic hydrocarbon contamination in two marine fish species collected in Galveston Bay, Texas. Environ Toxicol Chem 16:1472–1479

Williams EH, Bunkley-Williams L (1990) The world-wide coral reef bleaching cycle and related sources of coral mortality. Atoll Res Bull, No. 335. NMNH Smithsonian Institution, Washington, DC, USA. 71 p

Williams EH, Bunkley-Williams L (2000) Marine major ecological disturbances of the Caribbean. Infect Dis Rev 2:110–127

Williams ES, Barker IK (eds) (2001) Infectious diseases of wild mammals, 3rd edn. Iowa State University Press, Ames, IA, USA. 558 p

Wilson EA, Powell EN, Craig MA, Wade TL, Brooks JM (1990) The distribution of *Perkinsus marinus* in Gulf Coast oysters: Its relationship with temperature, reproduction, and pollutant body burden. Int Revue Ges Hydrobiol 75:533–550

Witham R (1973) A bacterial disease of hatchling loggerhead sea turtles. Fla Sci 36:226–228

Wobeser GA (2008) Parasitism: Cost and effects. In: Atkinson CT, Thomas NJ, Hunter DB (eds) Parasitic diseases of wild birds. Wiley-Blackwell, Ames, IA, USA, pp 3–9

Wolf JC, Baumgartner WA, Blazer VS, Camus AC, Engelhardt JA, Fournie JW, Frasca S, Groman DB, Kent ML, Khoo LH, Law JM, Lombardini ED, Ruehl-Fehlert C, Segner HE,

Smith SA, Spitsbergen JM, Weber K, Wolfe MJ (2015) Nonlesions, misdiagnoses, missed diagnoses, and other interpretive challenges in fish histopathology studies a guide for investigators, authors, reviewers, and readers. Toxicol Pathol 43:297–325

Wolthaus BG (1984) Seasonal changes in frequency of diseases in dab, *Limanda limanda*, from the southern North Sea. Helgol Meeresunters 37:375–387

Work TM, Rameyer RA, Balazs GH, Cray C, Chang SP (2001) Immune status of free-ranging green turtles with fibropapillomatosis from Hawaii. J Wildl Dis 37:574–581

Work TM, Balazs GH, Rameyer RA, Morris RA (2004) Retrospective pathology survey of green turtles *Chelonia mydas* with fibropapillomatosis in the Hawaiian Islands, 1993-2003. Dis Aquat Organ 62:163–176

Wright B, Mohanty B (2006) Operation Kachhapa: An NGO initiative for sea turtle conservation in Orissa. In: Shanker K, Choudhury B (eds) Marine turtles of the Indian subcontinent. University Press, Hyderabad, India, pp 290–302

Zimmerman AM, DePaola A, Bowers JC, Krantz JA, Nordstrom JL, Johnson CN, Grimes DJ (2007) Variability of total and pathogenic *Vibrio parahaemolyticus* densities in northern Gulf of Mexico water and oysters. Appl Environ Microbiol 73:7589–7596

Zimmerman R (1998) Characteristics and causes of Texas marine strandings. NOAA Tech Rep NMFS 143. U.S. Department of Commerce, Washington, DC, USA. 85 p

Zimmerman RJ (2010) A history of the NMFS laboratory at Galveston. Gulf Mex Sci 1–2:82–96