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HEALTH AND SAFETY CONCERNS IN FISHERIES AND AQUACULTURE

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Fish are highly regarded as a nutritious, low-fat source of protein. Other animal food sources have more saturated fat; fish have a higher proportion of monounsaturated or polyunsaturated fatty acids. A diet of fish is associated with a lower occurrence of human cardiovascular disease.[20] As with any animal food source, however, there is risk of acquiring disease from fish during processing and consumption. This chapter reviews bacterial and parasitic diseases transmitted from fish, as well as diseases caused indirectly by dinoflagellates. Safety issues related to aquaculture also are covered. Recent similar reviews include Garrett et al.,[85] Jensen and Greenlees,[123] and Durborow.[62]

In the United States, fish-related illnesses usually are associated with mild gastroenteritis or localized skin infections, but are sometimes more serious, resulting in amputations or death.[169] Fish, however, is a relatively safe food: only 10% of all foodborne illnesses in the U.S. are attributed to seafood.[178] This amounts to one illness per 250,000 servings of fish in general and only one illness per 5,000,000 servings of finfish (compared to one illness per 25,000 servings of chicken).[178] More than 90% of seafood-borne illnesses and 75% of the individual cases were due to ciguatera and scombroid poisonings, and the consumption of mollusks (mostly raw oysters).[85] Diseases from consuming fish are more commonly associated with shellfish than finfish.[219]

Hazard Analysis and Critical Control Points (HACCP) guidelines are followed in aquaculture, notably in catfish[21] and trout[237] culture. Also at the national level, the Joint Subcommittee on Aquaculture in Washington, D.C. has an active

Working Group for Quality Assurance in Aquaculture Production (electronic communication from Dr. Gary Jensen, National Aquaculture Program Leader, August 13, 1998). The World Health Organization (WHO) recommends that HACCP guidelines be followed in aquaculture worldwide.[85] In one study, HACCP principles were effective in eliminating *Clonorchis sinensis* from culture ponds in Vietnam, while 45% of the control ponds were infected with the trematode.[221] In another study, the trematode *Opisthorchis viverrini* was effectively controlled in aquaculture ponds in Thailand and Laos using HACCP guidelines.[221] Mishandling fish during retailing can lead to health problems for the consumer. In order to minimize problems, the temperatures for holding fish in restaurants have changed from < 45°F and > 140°F (the same as for most nonseafoods) to < 40°F and > 145°F.[115] Wholesalers should keep fish refrigerated, on ice, or frozen.

Commercially raised fish, in general, have a lower incidence of diseases that can be transmitted to people. Static water in aquaculture ponds is purified naturally by biological processes. Denitrifying bacteria in the water and muds metabolize the nitrogenous waste products of the fish.[62] In aquaculture raceways (typical of trout production systems), water constantly flows through concrete or fiberglass tanks, removing the majority of fish wastes. And continuous exposure to open ocean water occurs in salmon net pen culture and in mussel production. Of these systems, the ones involving exposure to ocean waters carry the highest risk of possible fish contamination. The highest risk in aquaculture, however, comes from closed-loop, indoor, water-recirculating production systems. These systems have been associated with fish pathogens contagious to people, including *Mycobacterium marinum, Vibrio vulnificus,* and *Streptococcus iniae*,[62] but these recirculating systems produce a small percentage of the fish that are raised nationally and internationally.

DINOFLAGELLATE/PLANKTON BLOOMS

During the past 15 years, the number of dinoflagellates known to be toxic to fish has increased from 22 to 55.[166] There appears to be a recent overall increase of dinoflagellate blooms, perhaps due to trends in warming and stillness of coastal waters as well as an increased influence from the growing numbers of people now living in these coastal areas.[230] Pollution from rivers was blamed for large, toxic, gelatinous blooms of *Chrysochromulina polyepsis* which accounted for about $200 million in losses at 200 salmon farms in Norway and Sweden in the late 1980s.[72] Although this planktonic bloom did not lead specifically to human health problems, it does reflect the growing problem with pollution along the coasts.[102]

The U.S. Congress appropriated $7 million for the evaluation of the human health impacts of the dinoflagellate *Pfiesteria piscicida* in 1997.[220] More recently, Congress has again recognized the problem with algal blooms: House bill HR4235 introduced on July 16, 1998 cites the dinoflagellate *P. piscicida* and "other recent occurrences of harmful algal blooms, including red tides in the Gulf of Mexico and the Southeast; brown tides in New York and Texas; ciguatera fish poisoning in Hawaii, Florida, Puerto Rico, and the U.S. Virgin Islands; and shellfish poisonings in the Gulf of Maine, the Pacific Northwest, and the Gulf of Alaska."[74] Ideally, research focuses on detecting these harmful blooms before they affect people. In the United Kingdom, the Ministry of Agriculture, Fisheries, and Food checks for toxins in harvested shellfish each week from April to October and occasionally during the winter.[211] Conrad, et al.[39] have developed instructional materials to increase the number of people trained in plankton toxicity monitoring.

Pfiesteria piscicida

P. piscicida is a dinoflagellate responsible for several fish kills in estuaries along the Atlantic coast; its toxin has been implicated in human health problems.[12,14,91,95,96,97,143, 149,150,152,155,156,175,224,233] The impact of this dinoflagellate has gained much attention from the general public, and there is a toll-free pfiesteria hotline (1-888-584-3110). At the time of this writing, Federal House bill HR4235 proposed appropriations of $25,500,000 to be authorized each year for 1999, 2000, and 2001 for research, education, and management activities related to the prevention, reduction, and control of harmful algal blooms including *P. piscicida*.[74]

Symptoms associated with human exposure to *P. piscicida* include eye irritation, respiratory problems, weakness, joint pain, nausea, abdominal pain, skin lesions, paresthesia, headache, myalgia, vomiting, asymmetrical excessive perspiration, decrease of speech fluency with hesitation in word choice, emotional changes, and memory loss.[25,89,155] Matuszak, et al.[155] list recommendations for managing concentration and memory difficulties. Thirteen researchers reported health problems after coming into contact with water containing the dinoflagellate or breathing the aerosol released by the organism.[166] In addition to the symptoms listed above, they experienced narcosis, kidney and liver dysfunction, and severe cognitive impairment (such as difficulty in reading, dialing telephone numbers, and doing simple arithmetic). Most symptoms reversed over time if the person avoided further contact with pfiesteria, but recurrences were reported after strenuous exercise for up to 6 years after exposure. Relapse symptoms include myalgia, asthenia, numb fingertips, sudden extreme irritability, and periods of "foggy" memory for about 12 hours after strenuous exercise.[89] Laboratory rats also experienced difficulty in learning new tasks after being injected subcutaneously with pfiesteria toxins.[141] Work with *P. piscicida* must now be performed in biohazard III containment systems in a limited-access facility. Protective measures include wearing full face respirators with organic acid filters, disposable gloves, boots, and hair covers, as well as bleaching protective clothes after use.[24] Also, aquaria containing pfiesteria must be ventilated away from laboratory workers according to OSHA guidelines.

In August 1997, at an interstate colloquium on pfiesteria, physicians and watermen gave testimonies on how they were adversely affected when exposed to bodies of water with pfiesteria outbreaks. A medical diagnostic team from Johns Hopkins University and the University of Maryland was commissioned to investigate these health issues.[155] Later that summer public health closures were ordered for Virginia and Maryland waters undergoing fish kills linked to the presence of *P. piscicida*. There are disagreements, however, on the effects on people exposed to natural pfiesteria outbreaks. For example, a study performed in May 1997 showed that crabbers working in areas affected by toxic pfiesteria blooms had no more health problems than those in pfiesteria-free water. In another case, patients' symptoms were traced to exposure to toxic hydrogen sulfide gas. Smith and Music[220] indicate that, so far, no field observation has conclusively demonstrated that ulcers are caused by *P. piscicida* or morphologically related organisms. Nevertheless, the team of physicians did find short-term memory and learning difficulties that they considered significant in 11 patients exposed to natural bodies of water containing pfiesteria. Golub, et al.[91] also reported similar effects in persons exposed to pfiesteria in its natural environment. And Bever, et al.[14] described a mild, reversible encephalopathy affecting functions associated with the frontal and temporal lobes in watermen exposed naturally to pfiesteria.

P. piscicida typically occurs during the warmest part of the year in shallow eutrophic estuaries and often precedes low oxygen conditions. Studies indicate that

excessive phosphate nutrient enrichment of these bodies of water due to human influences may increase the numbers and fish-killing activity of *P. piscicida*.[24,166] The dinoflagellate also was associated with massive fish mortalities on an aquaculture farm.[166,228] In fact, the organism was first found in fish cultured at the North Carolina School of Veterinary Medicine in 1988. Between 1991 and 1996 at least four commercial and eight research aquaculture facilities experienced fish kills from pfiesteria, resulting in losses ranging from $12,000–$90,000.[24] Hybrid striped bass and littleneck clams were the species affected.

About 24 life stages of *P. piscicida* have been described, including encysted, flagellated, and amoeboid stages. The flagellated and ameboid forms are toxic to fish challenged in the laboratory.[26,155,171] The most toxic flagellated stage is referred to as a toxic zoospore (TZ) or a toxic flagellated vegetative cell. The TZs attach to cells of the prey organism (e.g., fish) with an extension called a peduncle and then suction the prey's cellular contents.[166] *P. piscicida* cells in the dormant, encysted stage are able to detect the presence of secreta and/or excreta from a school of fish in surrounding waters. This stimulates the cells to transform to the flagellated stage, enabling them to move toward the fish while excreting a neurotoxin that narcotizes the fish ("ambush predation"). The neurotoxin causes the fish to linger in the area and can cause sores on which the pfiesteria organisms are able to feed.[152]

P. piscicida toxin is similar to the domoic acid produced by the dinoflagellate *Pseudonitzschia multiseries* in that both affect short term memory; but pfiesteria-related exposures are reversible, and seizures, myoclonus, and motor involvement are absent. Compared to ciguatoxin, produced by the dinoflagellate *Gambierdiscus toxicus,* pfiesteria toxin causes no diarrhea and requires no ingestion of seafood. Unlike domoic acid and ciguatoxin, pfiesteria toxin causes problems via inhalation *or* skin exposure. Table 1 lists symptoms of these and other dinoflagellate toxins.

Ciguatera Poisoning

Ciguatoxin, which causes ciguatera poisoning in people, originates from gambiertoxins produced by the dinoflagellate *Gambierdiscus toxicus*. The dinoflagellates cover and mix with foods that are eaten by herbivorous fish; these fish accumulate and concentrate the gambiertoxins. When the herbivorous fish are eaten by larger carnivorous fish, there is an oxidative biotransformation of the gambiertoxin to the more potent ciguatoxin.[142] The carnivorous fish include grouper, snapper, amberjack (and other large jack species), king mackerel, and barracuda found in South Florida, the Bahamas, the Caribbean, and the Pacific Ocean.[62,73] These fish are not cultured on farms and are not likely aquaculture candidates.

A farm-raised salmon, possibly imported from Chile, was suspected of causing ciguatera poisoning in 1992.[66] The affected woman became seriously ill 1½ hours after eating the fish and experienced hyperventilation, abdominal pain, nausea, diarrhea, tooth pain, a metallic taste in her mouth, increased heart rate, numbness in her extremities, a freezing sensation, and the inability to distinguish hot from cold temperatures. She was not able to eat solid food for over a month, and she continued to experience severe headaches, numbness in her arms, and a heavy sensation on her chest for more than a year. The condition usually resolves within 1–2 days,[81] but intensive medical supervision is needed in severe cases.[179] The severity and duration of ciguatera can be significantly reduced with intravenous mannitol.[142] The current estimate for the number of ciguatera cases worldwide is 20,000.[145]

The ciguatoxin CTX-1 is the most potent sodium channel toxin known. Work is underway to improve techniques for detecting CTX-1 in potentially harmful fish.

TABLE 1. Dinoflagellate-Caused Diseases—Toxins and Symptoms

Condition	Toxin	Organism	Symptoms
Pfiesteria-related	Unknown	*Pfiesteria piscicida*	Eye irritation, respiratory problems, joint pain, weakness, nausea, abdominal pain, skin lesions, paresthesia, headache, myalgia, vomiting, excessive perspiration, emotional changes, memory loss, narcosis, kidney dysfunction, liver dysfunction, severe cognitive impairment
Ciguatera fish poisoning	Ciguatoxin	*Gambierdiscus toxicus*	Nausea, diarrhea, vomiting, abdominal pain, lower extremity pain and weakness, paresthesia, temperature reversal, aching teeth, shock, coma (rare), brachycardia (rare), cerebral edema, death (rare)
Paralytic shellfish poisoning	Saxitoxin	*Protogonyaulax brevis* *Alexandrium* spp.	Paresthesia of mouth and extremities, diarrhea, vomiting, abdominal pain, ataxia (rare), respiratory arrest (rare), death (rare)
Neurologic shellfish poisoning	Brevetoxins and several toxins that can be aerosolized	*Ptychodiscus brevis* *Gymnodinium breve*	Paresthesia, temperature reversal, diarrhea, vomiting, ataxia
Amnesic shellfish poisoning	Domoic acid	*Pseudonitzschia multiseries* (*Nitzschia pungens*)	Gastroenteritis, memory deficits, confusion, short-term memory loss, death
Diarrhetic shellfish poisoning	Okadaic acid Dinophysis toxins (DTXs 1–4)	*Dinophysis* *Prorocentrum*	Nausea, vomiting, diarrhea, abdominal pain, feverish feeling > 8 hr duration

Adapted from Matuszak DL, Sanders M, Taylor JL, et al: Toxic *Pfiesteria* and human health. Md Med J 46(10): 515–520, 1997.

These techniques include antibody-based assays,[142] binding and cytotoxicity assays,[51] and a high-performance liquid chromatography/mass spectometry (HPLC/MS) ionization technique called atmospheric pressure ionization.[165]

Neurologic Shellfish Poisoning

Brevetoxins from the dinoflagellate *Ptychodiscus brevis (Gymnodinium breve*, red tide organism) cause neurologic or neurotoxic shellfish poisoning (NSP). Brevetoxins differ from most other dinoflagellate toxins in that they can become aerosolized and cause respiratory irritation.[220] Although not reported in aquaculture settings, NSP has caused massive fish kills in the Gulf of Mexico.[214,219] A microplate receptor-binding assay was successfully developed to test for brevetoxins in fish field samples.[138] The successful synthesis of brevetoxin A in 1998 by Nicolaou[170] opens the door for future design and synthesis of analogues which can be studied further biochemically.

Paralytic Shellfish Poisoning

Paralytic shellfish poisoning (PSP) is caused by saxitoxins (STXs) produced by dinoflagellates *Protogonyaulax (Gonyaulax) tamarensis* and *Alexandrium* spp.[244]

Fertile, planktonic-rich waters on the northwestern Atlantic coast of Spain are ideal for growing mussels, and incidents of paralytic shellfish poisoning have been attributed to cultured mussels from this region since 1976.[84,137,205] Mussels produced by aquaculture were a source of PSP in France and Norway.[230] Those problems were attributed to a species of *Alexandrium*. No cases of PSP have been reported from the United Kingdom since 1968.[211] Another source of PSP is *Pyrodinium bahamense* var. *compressa* in tropical countries. In 1992, cultured mussels in the Philippines caused PSP in many people.[92,230]

A saxitoxin receptor-binding assay and HPLC were both successful in detecting STX in serum and urine samples of PSP patients.[54] The test results corresponded well with the amount of tainted shellfish consumed and the severity of illness. PSP toxins were no longer detected in serum samples after 20 hours (although they were at high levels in the urine), indicating a fairly rapid elimination of the toxins. STX in clinical serum and urine samples also was detected using a saxiphilin-based assay.[13] Saxiphilin is a protein from certain amphibians and reptiles that binds strongly with STX.

Amnesic Shellfish Poisoning

Amnesic shellfish poisoning (ASP) occurs in people who consume seafood contaminated with domoic acid, a neurotoxin produced by the dinoflagellate *Pseudonitzschia multiseries (Nitzschia pungens)*.[219] (Some species of the red alga genus *Chondria* also produce domoic acid). The toxin accumulates in the digestive gland of mussels, clams, and crabs,[11,16,22,62,86,217,229,249] and also concentrates in finfish such as anchovies and herring.[250,251] Domoic acid mimics glutamic acid and affects the human brain and nervous system.[219]

Hardy, et al. found that trout feed made from tainted anchovies or herring contained domoic acid, but the toxin did not accumulate in tissues of the trout consuming the contaminated feed.[105] Cultured mussels, on the other hand, can acquire domoic acid from their environment and can cause human health problems. In one case, contaminated mussels cultured in eastern Canada caused three deaths, six permanent hospitalizations, and over a hundred cases of permanent short-term memory loss.[188,192,227,230] Most cases of ASP occur in North America and Canada; no cases have ever been reported in the United Kingdom.[211] Domoic acid is easy to detect in the laboratory with HPLC and ultraviolet tests.[68]

Diarrhetic Shellfish Poisoning

Recent reports describe diarrhetic shellfish poisoning (DSP) from cultured Spanish mussels.[84,205] The shellfish concentrate toxins produced by dinoflagellates of the genera *Dinophysis* and *Prorocentrum*. DSP also has been reported from mussels raised in France, where 10,000 and 2000 people were affected in 1984 and 1985.[219,230] The first occurrence of DSP in the United Kingdom was in 1997 when 49 patients presented with symptoms (Table 1) 30 minutes after consuming mussels in two London restaurants.[211] Before this incident, the most recent occurrence of any dinoflagellate-related shellfish toxicity in the UK was a case of paralytic shellfish poisoning in 1968.[211] DSP was first reported in Europe in 1961 and has persisted as a significant problem.[219]

Much recent research has focused on detection of DSP toxin(s). HPLC-fluorimetric determination of diarrhetic shellfish toxins has been successful, and sample clean-up for this process has been improved.[93] Another method of detecting these toxins is microliquid chromatography coupled with tandem mass spectrometry

(micro-LC-MS-MS).[57] Okadaic acid and dinophysis toxins (DTXs 1–4) are the primary toxins associated with diarrhetic shellfish poisoning.[211] A new toxin (an analogue of okadaic acid) responsible for DSP was isolated and characterized from *Dinophysis acuta*.[58]

SCOMBROID POISONING

Scombroid poisoning is the most common illness associated with seafood.[145] Scombroid poisoning occurs when people consume fish with high concentrations of **histamine**, producing an allergic-type reaction in the patient.[62] The disease often is misdiagnosed as an allergy of fish. The patient typically experiences itching, hives, skin rash, burning sensation in the mouth, flushing, headache, palpitations, nausea, vomiting, diarrhea,[184,206,226] tachycardia (120–150 bpm), and, in severe cases, bronchospasm, hypotension,[127] cardiovascular shock, subendocardial myocardial infarction, and acute pulmonary edema with myocardial ischemia.[6] Vision loss was a unique symptom reported for the first time in 1996.[157] A bitter, peppery (sometimes metallic) taste often is noticed when the affected fish are ingested.[127] The condition usually lasts about 1 day.

Fish in the family Scombroidae, including tuna, mackerel, bonito, and skipjack, frequently are associated with scombroid poisoning.[226] Sardines, mahi-mahi, bluefish, and herring also may cause the disease. A wide range of product forms, such as fresh, canned, dried, and salted fish, can be involved.[151] Histamine concentrates in the meat of affected fish (usually dark-fleshed fish) from the bacterial metabolism of the amino acid histidine found in the flesh. Bacteria naturally found on the fish (not from contamination), including *Proteus margonii, Klebsiella* sp., *Vibrio* sp., *Clostridium* sp.,[135,180] *Escherichia coli*, and *Salmonella* sp.,[127] are responsible for the histamine production. Other active histamine formers include *Morganella morganii, Plesiomonas shigelloides, Enterobacter intermedium,* and three *Serratia* species.[146] The build-up of histamine can happen in 3–4 hours if the fish are not refrigerated.

Another theory explaining scombroid poisoning is that the affected fish contain a potentiator that elicits histamine production in the patient.[127,226] This theory contends that the histamine in the fish is not what causes the allergy-like symptoms, because histamine breaks down when ingested directly by people.

Scombroid poisoning is probably the most prevalent disease contracted from finfish and typically results from wild, captured fish not being refrigerated promptly on the fishing vessel.[222] Prevention is best achieved by keeping fish refrigerated during all stages, from harvest to consumption.[9] This disorder has not occurred, and is unlikely to occur, from ingestion of cultured fish for two reasons: First, fish from the Scombroidae family are not cultured, and there is more of a tendency to raise fish with a light-colored flesh as opposed to the darker flesh more commonly associated with scombroid toxicity. Second, aquacultured fish enter a processing plant alive and reach the final stage on ice or frozen within about 20 minutes.[62]

Scombroid poisoning is commonly treated as an allergy with antihistamines. Hydrocortisone, diphenhydramine (Benadryl), cimetidine, chlorpheniramine, and fluids have been prescribed.[127,157] If the patient presents shortly after eating the affected fish (maybe within 4 hours or less), ipecac may be indicated to induce vomiting and rid the patient of the source of the histamine.[56] Downs has noticed that the bright red rash and headache disappear as the patient is vomiting.[56] Use of histamine-1 (H_1)[157] or an H_2 blocker[56] such as cimetidine (Tagamet)[222] have been recommended if severe headache persists for several days after ingestion. The patient does *not* need to avoid eating fish.

BACTERIAL INFECTION

Bacterial infection in humans can occur from contact with fish infected by *Mycobacterium marinum, Streptococcus iniae, Vibrio, Aeromonas, Erysipelothrix rhusiopathiae,* and *Burkholderia pseudomallei.* Some bacteria in the genera *Vibrio* and *Aeromonas* also may cause infections.

Mycobacterium marinum

M. marinum was first isolated and named by Aronson in 1926; it was recovered from sick and dying saltwater fish in the Philadelphia aquarium.[4] The first discovery, however, appears to have been 100 years earlier by Laennec.[90] Literature from the 1960s and before may refer to this same bacterium as *M. platypoecilus* or *M. balnei.* Tuberculoid infections in people using public swimming pools were reported in 1939 from Sweden and in 1951 from the United States.[90,172] Linell and Norden[144] identified the causative organism in 1954 after 80 persons (who had used the same public swimming pool) were diagnosed with granulomatous skin lesions.[90,231] A *M. marinum* outbreak at a Colorado swimming pool in 1961 also affected a large number of swimmers.[90] These early findings led to the disease's once-common name of "swimming pool granuloma." Today, however, due to sanitary chlorination practices, these kinds of outbreaks are rarely seen. Instead, the names "fish tank granuloma" and "fish handler's disease" are sometimes used because of the association with home aquariums and water-related activities such as swimming, fishing, and boating.[118] *M. marinum* infections also are associated with skin lesions of butchers.[77]

The bacteria will not grow at 37°C, so it usually is confined to the extremities such as the arm and hand (and sometimes the knee, foot, or mouth). The dominant arm/hand typically becomes infected.[53] Except in cases involving immunocompromised patients, the lesions usually are circumscribed and either single nodular or multiple sporotrichoid.[118] Two cases of septic arthritis have been attributed to *M. marinum.*[107,182] In addition to tropical fish in home tanks, *M. marinum* has been found in fathead minnows held in aquaria as bioassay test animals[61] and in commercial aquaculture recirculating systems.[62]

Almost all cases of *M. marinum* infections in current medical literature attribute the source of infection to a **home aquarium.**[10,19,53,62,67,75,103,118,119,181,182,189,202,213,242] In one case, an immunocompromised infant contracted a serious *M. marinum* infection after he was bathed in the same bathtub where the family's aquarium had just been cleaned.[181] The infection caused disseminated osteomyelitis with erythematous pustules resembling chicken pox and eventual death. Osteomyelitis caused by *M. marinum* usually occurs only in immunocompromised patients, including those with AIDS. In another case, a 48-year-old woman undergoing immunosuppressive therapy developed osteomyelitis of her right hindfoot, causing painful swelling of her ankle, septic arthritis of a joint in her finger, and tenosynovitis of the left palm.[10] Her condition was caused by *M. marinum* contracted from a fish tank. In one atypical case, an immunocompetent patient developed osteomyelitis in the finger, leading to amputation.[239]

Although less common than *M. marinum*, two other mycobacteria (*M. fortuitum* and *M. chelonei*) also are capable of infecting both fish and humans.[62] *M. fortuitum* grows at both 30° and 37°C, while *M. marinum* and *M. chelonei* grow at 30°C but not at 37°C. The latter two may be missed in hospital microbiology labs, because incubators are usually set only at 37°C for growing typical human pathogens. Physicians with patients having granulomatous lesions and a history of working around aquaria should routinely have lesion samples cultured at 30°C or at room

temperature. An interesting growth characteristic of *M. marinum* is the production of yellow pigment from colonies exposed to sunlight when grown on Ogawa medium[119] or supplemented Middlebrook 7H9 agar.[199]

Another factor making laboratory identification of *M. marinum* difficult is its slow growth rate; this acid-fast bacteria's presence can be detected slightly after 5 days of growth on trypticase-soy agar blood plates at 25°C when alpha hemolysis occurs, producing a slight greening on the blood plate.[62] The slow identification process can be expedited by automated sequencing of amplified 16S-rDNA, which has been shown to be feasible for identifying *M. marinum*.[75] And information on DNA-DNA hybridization and 16S-rRNA gene sequence analysis may contribute to the development of nucleic acid-based tests for more rapid detection of mycobacteria.[232] Lack of early detection and treatment of some deep infections may result in permanent limitation of range of motion or a need for excision or amputation.[213] *M. marinum* infections can be confused with tuberculosis, tularemia, nocardia bacterial infections, coccidiomycosis, histoplasmosis, blastomycosis, syphilis, leishmaniasis, yaws, sporotrichosis, skin tumors, and chicken pox.[67,181]

Many antibiotics are effective in treating *M. marinum* infections (Tables 2 and 3). Treatment should continue for several weeks after apparent lesions disappear; regimens typically last from 3–6 months,[90] but may extend to 12 months or longer in some patients. The long treatment time required for *M. marinum* is attributable, in part, to its ability to survive inside the human host's macrophages (a behavior similar to that of *M. tuberculosis*).[8]

A medical team in Taiwan[213] treated a patient with clarithromycin and ethambutol for a total of 18 months, even though the wound healed completely after 7 months—perhaps because the wound had previously progressed to synovitis and osteomyelitis after another doctor had given the patient local steroid injections. Huminer, et al.[118] also noted a worsening of a *M. marinum* hand lesions after another practitioner injected the site with hydrocortisone. Clarithromycin[247] was found to be effective after only 1 month, with no relapse, in a patient examined 6 months later. Levofloxacin (300 mg/d for 11 weeks) was considered by Iijima and Saito[119] to have an advantage in treating *M. marinum* skin infections, because it is distributed in a greater amount in the skin than in the serum.

A thorough drug susceptibility study recently was performed on 60 clinical strains of *M. marinum* from 10 geographical sites in the United States.[77] The order of effectiveness was trimethoprim-sulfamethoxazole = ethambutol > clarithromycin > minocycline = doxycycline > amikacin. Rifampin was only marginally effective in inhibiting growth of M. marinum, and ciprofloxacin was not active.[77] Many of the unsuccessful antibiotics listed in Tables 2 and 3 were prescribed for the patient before an accurate diagnosis of *M. marinum* was made. Minimum inhibitory concentration Etests demonstrated by Flynn, et al.[77] provided accurate susceptibility results within a reasonable length of time.

Age of the patient also is a consideration in choosing an antibiotic; Brady, et al.[19] successfully used clarithromycin and rifampin to treat a *M. marinum* infection around a 2-year-old boy's mouth, but did not consider using doxycycline and minocycline because they are not typically used on children younger than 8 years. Ethambutol also was excluded due to the boy's young age, but trimethoprim-sulfamethoxazole was considered as an option.[19]

In sensitivity tests run before treating fish infected with *M. marinum*, bacterial isolates from the fish were resistant to ethambutol, pyrazinamide, and ethionamide, but were susceptible to rifampin and streptomycin.[37] However, the surviving fish

TABLE 2. Antibiotics in Treatment of *Mycobacterium marinum* Infection

Author(s), Date	Effective	Ineffective*
Feddersen, et al (1996)	Clarithromycin + rifampin + protionamid	Imipenen + amikacin
Gluckman (1995)	Ethambutol + rifampin, clarithromycin, azithromycin, clarithromycin + ethambutol, ciprofloxacin, doxycycline, minocycline, trimethoprim-sulfamethoxazole	Isoniazid
Iijima, et al (1997)	Levofloxacin	Clarithromycin, minocycline, cycloserine, rifampin, trimethoprim, kanamycin, enviomycin, capreomycin, streptomycin
Weinstein, et al (1997)	Clarithromycin, minocycline, doxycycline, imipenem, rifampin, ciprofloxacin, ethambutol, clofazamine, amikacin	Penicillin, ampicillin, erythromycin, cefoxitin, streptomycin
Ramakrishnan, 1997	Rifampin + ethambutol	
Phillips, et al (1995)	Rifampicin + isoniazid	Flucloxacillin + topical mupiricin, pyrazinamide
Hanau, et al (1994)	Isoniazid + rifampin + ethambutol + amikacin	Cephalexin, dicloxacillin
Shih, et al (1997)	Clarithromycin + ethambutol + rifampin + doxycycline	Isonizaid + ethambutol + rifampicin
Dorronsoro, et al (1997)	Cotrimoxazol, rifampin + ethambutol	
Ries, et al (1990)	Isoniazid + rifampin + ethambutol	
Edelstein (1994)	Rifampin + ethambutol, minocycline (10/14 cases)	Minocycline (4/14 cases)
Parent, et al (1995)		Rifampin, isoniazid
Flynn, et al (1997)	Trimethoprim-sulfamethoxazole, ethambutol, clarithromycin, minocycline, doxycycline, amikacin	Rifampin, ciprofloxacin
Barton, et al (1997)	Doxycycline	
Brady, et al (1997)	Clarithromycin + rifampin	
Kuhn, et al (1995)	Clarithromycin	
Huminer, et al (1986)	Sulfamethoxazole + trimethoprim, ethambutol	Clotrimazole topical cream + 0.025% fluocinolone acetonide cream, oral erythromycin sterate, ampicillin sodium + cloxacillin sodium, rifampin + minocycline hydrochloride, isoniazid, streptomycin sulfate

* *M. marinum* is resistant to these antibiotics.

(that were treated with antibiotics) were found to still be carrying the *M. marinum* bacteria at the end of the study, indicating that eradication was not achieved.[37] This may indicate that people working with fish supposedly cured from *M. marinum*

TABLE 3. Specific Antibiotics Against *Mycobacterium marinum:* Quantification of Reports*

Antibiotic(s)	Number of Reports of Effectiveness	Number of Reports of Ineffectiveness
Rifampin + ethambutol	8	1
Rifampin only	1	3
Clarithromycin only	4	1
Clarithromycin + other drugs	3	0
Doxycycline	5	0
Trimethoprim-sulfamethoxazole	3	0
Ethambutol only	3	0
Levofloxacin	1	0
Ciprofloxacin	2	1
Minocycline	13	5
Isoniazid**	3	4
Streptomycin	0	3

* Reports are derived from current medical literature (see Table 2).
** Always in combination with one or more of: rifampin, ethambutol, amikacin.

could remain at risk of infection. Destruction of the infected fish and disinfection of the facilities with chlorine bleach often are recommended.[76]

Recommended preventive measures include wearing gloves when cleaning aquaria, processing (and diagnosing) fish, or performing other activities around fish and water sources suspected of harboring *M. marinum.*[76] Immunocompromised patients, including those who are HIV-positive, should avoid cleaning aquaria; *M. marinum* infections can occur more easily in an immunocompromised individual and are more serious and harder to control.[103,181,202] Adequate chlorination of swimming pools also is imperative for preventing infection.

Fish infected with *M. marinum* often have an enlarged spleen (10 to 20 times larger than a normal spleen), which is granular with white or gray lesions throughout the organ.[62] The kidney and liver also may be granular.[76] The liver may have a characteristic white color covered with petechial hemorrhaging, and the gills may be characteristically white.[62] Fish from closed, recirculating aquaculture systems and aquaria should be suspected of carrying *M. marinum.*

Streptococcus iniae

S. iniae bacteria was first reported from Amazon freshwater dolphins in 1976 and again in 1978.[190,191] In 1986, the bacteria was discovered to cause meningoencephalitis in tilapia (St. Peter's fish)[185] and trout.[30] It was not until 1991 in Texas and 1994 in Ottawa Canada[30] that *S. iniae* was found to cause infections in people.[70,116,248]

In late 1995 and early 1996, four patients of Chinese descent became infected systemically with *S. iniae* while cleaning and preparing fresh, whole, farm-raised tilapia.[29] Three of the cases involved cellulitis, and the fourth involved endocarditis, meningitis, and probable septic arthritis. The three patients with cellulitis all had hand injuries while cleaning the fish and experienced symptoms within 2 days. Their conditions were successfully treated with beta-lactam antibiotics or clindamycin. The fourth patient was a 77-year-old man who experienced knee pain, intermittent sweats, fever, dyspnea, and confusion after several days. His illness was diagnosed as endocarditis and meningitis (along with arthritis in the knee), and he recovered after treatment

with beta-lactam antibiotics and erythromycin. Antibiotic sensitivity results showed that three of the four *S. iniae* strains recovered were resistant to bacitracin (the fourth was sensitive).[30] All were sensitive to beta-lactams, macrolides, trimethoprim-sulfamethoxazole, tetracycline,[30] and fluoroquinolones.[29] In other trials, *S. iniae* was susceptible to penicillin, cefazolin, ceftriaxone, erythromycin, clindamycin, trimethoprim-sulfamethoxazole, and ciprofloxacin, but resistant to gentamicin.[246]

Weinstein and eleven other researchers[246] from Canada and the United States surveyed *S. iniae* cases from late 1995 through most of 1996. Of nine patients with invasive *S. iniae* infections, eight had cellulitis of the hand and one had endocarditis. All were of Asian descent and had processed live or freshly killed fish. Eight had percutaneous injuries. Six of the nine species of fish involved were tilapia. Using pulsed-field gel electrophoresis (PFGE), the research team found that *S. iniae* isolates from humans and fish were highly related and, in some cases, indistinguishable. Not all *S. iniae* bacteria pose a dangerous health risk. Only clones A and A' of *S. iniae* were shown to be pathogenic to humans and tilapia; the clones differed from each other by only one band in the PFGE tests.[246] Using HindIII and EcoRI ribotyping of *S. iniae*, Eldar, et al.[71] found that strains of the pathogen occurring in Israel differed from those in the U.S. Such tests should be valuable in tracking occurrences of the disease across international borders.

Although several cases of *S. iniae* infection have occurred in people purchasing certain whole, live aquacultured fish, no incidences have been reported among fish farm workers.[215] Infections usually are relatively mild, from handling fish, and no infections have occurred from eating fish. Shoemaker and Klesius[215] state that it is probably too early to label *S. iniae* infection as an emerging human health problem.

S. iniae infections occur mostly in closed, recirculating aquaculture systems[62,197] but also have been reported from ponds.[69] Optimal disease-producing conditions for this bacteria are 20°C (compared to 15°, 25°, 30°, or 35°C) and a pH of 9 (compared to more acidic water).[186] Over 20 different species of fish around the world have been reported with *S. iniae* infections, but tilapia, striped bass, striped bass x white bass hybrids, and yellowtail are the most severely affected.[197] The pathogen has been reported from freshwater and marine environments and from both cultured and wild habitats.[197] Clinical signs of fish infected with *S. iniae* include characteristic erratic swimming, darkened color, hemorrhage on the operculum and at the base of fins, exophthalmia with cloudiness of the eye, and a swollen abdomen.[130,196] Treatment of infected hybrid striped bass with the antibiotic enrofloxacin (a fluoroquinolone) reduced mortalities from 56% to 11% in one trial and from 40% to 17% in another trial.[225] The most effective dose appeared to be 5 mg of enrofloxacin per kg of fish body weight; this provided control of the disease while still being palatable. In addition, there appears to be some potential for development of a vaccine against this pathogen.[71]

Vibrio

Several bacterial species of the genus *Vibrio* cause diseases in fish and are occasionally spread to people.[167] Perhaps the most notorious is *V. vulnificus*, which causes wound infections leading to mortality in 25–50% of patients, despite prompt treatment.[18] Wounds in humans have been reported from finfish[62,114,124,132] and, in one case, from a crab bite[168] (which proved to be fatal). One case occurred in Louisville, Kentucky in 1991 where workers at a closed, recirculating aquaculture system became infected through contact with infected tilapia.[62] Live tilapia also were responsible for causing vibrio infections in Israel in 1996.[85]

Symptoms of *V. vulnificus* wound infections include blisters and ulcers on the extremities, cellulitis, edema, purpura, hemorrhagic bullae, and necrotic eschar at the bullous lesion sites.[114] Systemic infections (which usually originate from eating raw oysters) may involve chills, fever, blister formation, ecchymotic hemorrhages, pain in the lower extremities, changes in mental status, vomiting and diarrhea, and a 50% mortality rate.[161] Veenstra, et al.[240] and Amaro and Biosca[2] reported *V. vulnificus* biotype 2 isolated from eels as being pathogenic to humans. This biotype is indole negative, differentiating it from the more typical *V. vulnificus* biotype 1. Amaro, et al. recommend raising eels in freshwater instead of sea or brackish water to prevent the growth of *V. vulnificus*.[1] The current author, however, reported finding this bacteria in tilapia grown in fresh water.[62]

Photobacterium (Vibrio) damsela is commonly found in fish and can infect humans.[60,79] It is capable of causing rapid fatalities in people, even in those with no predisposing condition.[36,80,258] Pedersen, et al.[183] recently isolated this bacterium from aquacultured rainbow trout for the first time. A fatality resulted from a person receiving a wound from a catfish,[36] and another infection involving *Vibrio* sp. occurred from a catfish spine puncture wound.[160] Reducing the occurrence of *P. damsela* infections in fish should result in lower risk for those working with fish. Santarem, et al. demonstrated some protection of turbot (*Scophthalmus maximus*) against *P. damsela* when the turbot were administered beta-glucans and/or the bacteria's O-antigen.[207]

Symptoms in the *P. damsela* case reported by Midani and Rathore, involving a puncture wound of a patient's great toe by a saltwater catfish spine, included erythema, swelling with minimal discharge, and inability to bear weight on the toe without pain.[160] The patient had a 3-cm laceration, a yellowish discharge, decreased range of motion, and a fever of 38.5°C 48 hours after the injury. Wound treatment recommendations for this and similar cases include hot water emersion of the stung area for pain relief, thorough irrigation and debridement, culture of the wound (notifying the microbiology laboratory to look for marine bacteria), radiography of the wound to check for foreign objects, and tetanus prophylaxis in some cases.[160] More than one antimicrobial must be used for empiric treatment of saltwater catfish spine injuries. Midani and Rathore suggest that therapy include parenteral treatment with doxycycline, a third-generation cephalosporin, and an aminoglycoside. For milder infections, they suggest enteral treatment with tetracycline or one of its analogues, or trimethoprim-sulfamethoxazole. The authors note that poor results have been obtained in animals with penicillin and cefotaxime.

Probably the most commonly diagnosed *Vibrio* species in freshwater fish is *V. parahemolyticus*. In humans, it often causes mild enteritis, but may cause abdominal pain, vomiting, watery diarrhea, fever, chills and headache.[241]

Ingestion of raw or undercooked fish (typically **shellfish**) is usually the cause of human infections caused by *V. alginolyticus*,[18,112] *V. cholerae* (O group 1 and non-O1),[38,64,148,162] and *V. hollisae*.[147] *V. alginolyticus* causes skin infections, stump ulcer, enteritis, and conjunctivitis. *V. cholerae* O group 1 causes severe vomiting and diarrhea. *V. alginolyticus, V. cholerae, V. vulnificus,* and *V. parahemolyticus* all have been reported in farm-raised tiger shrimp from India,[15] and the less virulent *V. cholerae* non-O1 was found in a small number of aquacultured, warm-water shrimp imported into Denmark.[43] The *Vibrio* species most commonly found from fish in a marine environment is *V. anguillarum*, but it does not infect humans.[82]

Several techniques have been tried to reduce or eliminate *Vibrio* species from shellfish. Moving shellfish to clean waters for depuration purposes was not effective

in purging vibrio,[101] but the use of short-term heat treatment was effective in reducing *V. vulnificus* to undetectable levels.[40] Although success also has been achieved using irradiation to reduce *Vibrio* to undetectable levels, the process has not been approved in the U.S.[123]

Crayfish handlers' disease is caused by various species of *Vibrio*.[76] Typical symptoms are itching and burning at the injury site, followed by swelling and stiff, painful joints. Treatment is application of a local antibiotic cream or powder four times a day.[76] The antibiotics penicillin, tetracycline, erythromycin, chloramphenicol, and novobiocin are reported effective.[76] Prevention is attained by avoiding cuts or puncture wounds inflicted around water environments and protecting any existing skin wounds.

Midani and Rathore point out the importance of quickly and accurately identifying vibrio when it is involved in puncture wounds: "Because the antimicrobials effective against vibrio species are different from the empiric therapy used after the commonly seen puncture wound or for soft tissue injuries caused by other sharp objects, one must be aware of the association of catfish spine injury with infection with Vibrio."[160] They also point out that freshwater catfish wounds often result in *Aeromonas hydrophila* infections, while wounds from marine catfish spines typically cause *Vibrio* sp. infections. Work in identifying *Vibrio* species occurring in humans, fish, and shellfish with the use of monoclonal antibodies has been done by Chen, et al.[33]

Food-workers handling fish should be aware that certain psychotropic strains of *V. parahemolyticus, V. mimicus,* and *V. fluvialis* are able to survive storage at temperatures as low as –30°C.[254] The presence of phosphates helped to enhance the bacteria's survivability.[254] People working around tropical fish aquaria also should be aware of the potential of contracting a bacterial infection; *V. cholerae* non-O1 and *V. mimicus* were found in 1 of 12 aquaria examined in a study by Hay, et al.[109]

Aeromonads

The motile *Aeromonas* complex bacteria *A. hydrophila, A. sobria, A. caviae, A. veronii* and *A. shubertii* infect wild and cultured fish.[216] Aeromonad infections in humans are rare, occurring in only one person in a million each year.[5,31] Symptoms include gastroenteritis and diarrhea typically caused by ingesting contaminated water[87] or contaminated fish, fish eggs, or shrimp.[104] When *Aeromonas* infection occurs in a localized wound, cellulitis, deep muscle necrosis, and septicemia may result, usually in immunocompromised individuals,[78] but sometimes in those who are immunocompetent.[110,126,245] Localized wounds involving *Aeromonas* bacterial infections have been associated with a freshwater catfish spine injury.[160] A recent case involved a wound caused by a stingray.[198] In another case, a woman involved in a car accident was exposed to fresh water, and her wounds became infected with *A. hydrophila*.[3]

Erysipelothrix

The bacteria *Erysipelothrix rhusiopathiae* (formerly *E. insidiosa*) causes a skin condition called erysipeloid and causes septicemia (rarely). The bacteria can be transmitted to humans from fish mucus or spines.[212] *E. rhusiopathiae* causes no disease in fish, but is commonly spread to persons such as fish filleters, butchers, veterinarians, and fish health workers who are in frequent contact with animals, especially fish.[62,255]

Clinical manifestations of this bacterium are reviewed by Reboli and Farrar.[201] Three clinical syndromes are described in people: (1) a skin infection usually on the fingers or hand, referred to as erysipeloid or "fish rose," (2) a diffuse cutaneous form that can spread from skin to adjacent tissue, and (3) a septicemic form that is rare.[94]

Gloves (e.g. latex) are helpful in preventing erysipeloid.[62] Only 49 cases of septicemia from *E. rhusiopathiae* have occurred in the U.S. between 1912 and 1988, 11 of which were caused by fish.[94] Forty four of these cases involved endocarditis, and 19 cases were fatal. A fatal case was reported in 1995 when a man became infected after gutting an eel.[113]

Erysipeloid can be confused with "seal finger," but the etiology and antibiotic treatment are different.[113] The bacteria can survive smoking, salting, and pickling, but does not survive moist heat at 55°C for 15 minutes.

Melioidosis

Melioidosis is an infectious disease in people and animals caused by the bacteria *Burkholderia (Pseudomonas) pseudomallei*, which occurs mainly in tropical and subtropical areas, notably Southeast Asia and northern Australia, but can occur in temperate areas.[52,256] The disease is reported from travelers returning from Southeast Asia, but can be autochthonous to the Americas and other areas.[52,218,252] One recent case of melioidosis that was diagnosed and contracted in Puerto Rico involved a patient with chronic granulomatous disease.[52] Another recent case was reported from Guadeloupe.[187] Mortality rates for melioidosis exceed 50%; for septicemic cases, the mortality rate is 80–90%.[200]

B. pseudomallei bacteria live in soil and water and can infect people through open wounds or by inhalation (and possibly through oral ingestion).[234] Soldiers fighting in Vietnam sometimes contracted melioidosis, giving it the name "**Vietnam tuberculosis**."[159] Recently, two tourists returning from vacations in Thailand were diagnosed with melioidosis: they had been wading in shallow water and mud (one in a rice paddy), both had been consuming four or more alcoholic drinks a day, and one of them suffered from noninsulin-dependent diabetes mellitus.[218] *B. pseudomallei* can be isolated from over two-thirds of Thailand's rice paddy fields.[44] Additionally, consuming high amounts of alcohol and being diabetic are factors that lower a person's resistance to melioidosis.[218]

Water used for importing exotic aquarium fishes from Southeast Asia was found to contain *B. pseudomallei*.[34] Fish dealers and aquarium owners should take precautions, such as disinfecting aquaria between batches of fish and wearing gloves to protect open hand or arm wounds. Melioidosis also is considered a major problem in Australian animals, including kangaroos, cattle, sheep, and goats.[159]

A wide range of symptoms occur with melioidosis: septicemia, pneumonia, chronic fever, and abscesses in the liver, spleen, lungs, skin, and soft tissue, as well as in the salivary glands of children[159] and in the brain.[100] Muscle pain, folliculitis, septic shock, hypoventilation, meningitis, anuria, dysarthria, ataxia, double vision, soporose, and dysfunction of cranial nerves also are symptoms.[218] Relapse after several months of treatment is characteristic. Melioidosis imitates many other diseases, including typhoid fever, leptospirosis and tuberculosis.[200] Acute cases of melioidosis involving sepsis and pneumonia are usually fatal in a few days if untreated. Rapid death from acute cases of melioidosis may be due to uncontrolled cytokine production, which induces multiple organ failure and disseminated intravascular coagulation,[136] or possibly due to a recently described *B. pseudomallei* exotoxin referred to by Haase, et al.[100] as cytolethal toxin. Persons with compromised immune systems or ailments such as diabetes or renal disease are especially susceptible to melioidosis, as are consumers of high amounts of alcohol.[100] A physician at a Malaysian hospital who deals with many melioidosis cases has noted that *all* of his patients are diabetic.[159] The disease can suddenly manifest after being dormant in the infected

individual, sometimes for as long as 26 years, giving it a common name of "**time-bomb disease**."[100]

Limited success has been achieved using antibiotics to treat melioidosis. Even the most potent antibiotic, ceftazidime (with or without co-trimoxazole), cures only 50–70% of those with septic melioidosis.[44] Several antibiotics are effective in inhibiting the growth of *B. pseudomallei* in the laboratory, but are not as successful in vivo, perhaps because the bacteria is harbored within organ and tissue abscesses or in macrophages.[100,136] *B. pseudomallei* usually is sensitive to tetracycline, co-trimoxazole, amoxicillin plus clavulanic acid, ampicillin plus sulbactam, cefoperazone plus sulbactam, piperacillin, ceftazidime, imipenem, meropenem and co-amoxiclav.[44,159] Imipenem, meropenem, and piperacillin were, at one time, thought to be potentially superior to ceftazidime based on laboratory results, but apparently did not perform as well in patients.[44] Relapse in spite of several months of antibiotic therapy occurs in up to a quarter of all melioidosis cases. The relapse rate was reduced to only 4% after treatment with doxycycline + co-trimoxazole + chloramphenicol, while co-amoxiclav + amoxycillin had a 16% rate of relapse.[44]

B. pseudomallei colonies have a "wrinkled" appearance and have the smell of wet soil after a rain.[159] The bacterium is a gram-negative rod that exhibits bipolar staining and is rather slow growing. There is a great need for rapid identification tests for *B. pseudomallei*; the culture of the bacteria takes several days, during which time the patient will worsen and may expire. Polymerase chain reaction (PCR) tests can bypass the need to culture the suspected bacterial pathogen. Rattanathongkom, et al. have developed a PCR test that can detect a single *B. pseudomallei* cell in blood samples.[200] Additionally, indirect hemagglutination tests and enzyme-linked immunosorbent assays are being developed that detect the *B. pseudomallei* antibodies in the suspected carrier.[159] But Rattanathongkom, et al. claim that tests designed to detect antibodies are of limited value, because many people in melioidosis-endemic areas carry high background levels of these antibodies. Leakey, et al.[136] recently have demonstrated effective uses of two different mouse strains as models for studying various forms of melioidosis.

PARASITES

Larval parasites in uncooked or undercooked fish pose a human health risk. The problem-causing nematode genera include *Anisakis, Pseudoterranova, Contracaecum,*[121] *Eustrongylides, Philometra, Gnathostoma, Angiostrongylus* and *Capillaria*; the cestodes include *Diphyllobothrium*; the digenetic trematode genera involved are *Clonorchis, Opisthorchis, Heterophyes,* and *Nanophyetes*;[238] the acanthocephalan is *Bolbosoma* sp.;[125] and the protozoa *Cryptosporidium,*[173] *Enterocytozoon,*[49] and *Giardia lamblia.*[62] Ogawa states that about five species of parasites from marine organisms are known to be zoonotic.[174] Deardorff, however, reports that over 50 species of helminth parasites from fishes, crabs, crayfishes, snails, and bivalves are known to produce human infections.[45] Although prevention of parasitic infections is thought to be more difficult in net-pen culture systems in coastal waters than in freshwater ponds, infection of maricultured species with zoonotic trematodes, cestodes, and nematodes has not been confirmed.[174] The parasites causing the most significant problems are covered in the following sections.

Anisakidosis

Anisakidosis (formerly called anisakiasis) is caused when humans ingest the third-stage nematode of *Anisakis simplex* or *Pseudoterranova decipiens.*[62] It is

considered the most important zoonotic disease spread from fish to man; over 20,000 cases have been reported from Japan.[174] But there have been no cases of *Anisakis* or *Pseudoterranova* infections originating from cultured salmonids.[46] Marine mammals (e.g., whales, dolphins, seals) are normally the definitive hosts, but people assume this role when infected fish (such as salmon, herring, tuna, and Pacific cod) are consumed raw or undercooked.[121,131] Reports from Japan include mussels, oyster, crawfish, lobster, and prawns as sources of anisakid infections.[121] In the U.S., anisakidosis is found mostly in the western states and around the Great Lakes.[209] The disease is common in the Netherlands, Scandinavia, Russia, and Japan (which has about 1,000 new cases of anisakidosis a year).[121,176] No parasites of public health significance, including *Anisakis simplex*, occur in net-pen–reared salmon fed commercially prepared diets.[46]

The anasakid nematode causes serious inflammation of the definitive host's stomach wall.[76] The larva can be from 1–5 cm long. Symptoms of anisakidosis include abdominal pain from the round worm penetrating the stomach or intestine, fever, nausea, vomiting, diarrhea, blood in the stool, abdominal dilation caused by ascites, intestinal gas, swelling of lymph nodes, and edema.[41,121] Eosinophilia, leukocytosis, and pyrexia can be seen during the first 3 weeks. The disease sometimes is misdiagnosed as gastric ulcers or appendicitis.[169] Sometimes patients cough up the worm and can feel the worm in their throat causing "**tingling throat syndrome**."[158] Some reports exist of tumors in the lung and stomach that suddenly and mysteriously vanish.[121]

Treatment involves surgically removing the parasite and/or affected intestinal tissue. If the worm is in the stomach, fibergastroscopy can be used.[76,125] Gastroscopy, which allows the anisakid worms to be visualized and removed, is in much wider use in Japan than in most other countries.[121] Interestingly, the Japanese custom of drinking sake wine with raw fish may have some health implications; some evidence exists that consumption of sake wine along with sashimi may prevent the establishment of anisakid worms.[121]

Eustrongylides sp.

Eustrongylidiasis is caused by consumption of fish infected with the nematode *Eustrongylides* sp. Three fishermen in Maryland became infected when they ate live bait minnows.[32] Symptoms included severe abdominal pain within 24 hours, followed by progressive abdominal cramping. It was misdiagnosed as appendicitis, but during the appendectomy two fourth-stage *Eustrongylides* parasites were found. One worm was in the abdominal cavity and the other was penetrating the intestinal cecum. In 1989, a single *Eustrongylides* sp. was discovered in the flesh of a farm-raised catfish in Mississippi, but was considered an isolated case and never developed into a problem.[62] Any parasite found in farm-raised catfish would have little opportunity to infect a person due to the routine practice of cooking these fish before consumption (they are not customarily used in raw fish products).

Philometra sp.

A *Philometra* sp. nematode penetrated 2.5 cm into the hand of a person filleting a jack fish.[48] It was discovered 3 hours later when he noticed the distal end of the red worm protruding 1.1 cm out of his hand.

Gnathostomiasis

Several species of the nematode genus *Gnathostoma* cause infections in fish and humans. Before 1980, *G. spinigerum* was the only known cause of gnathostomiasis

in many parts of the world.[208] Since 1980, migrating larvae of *G. hispidium* were found in the skin of patients who had swallowed small, live loaches.[125] The larvae caused creeping eruptions in the patients' skin. Similar lesions were caused by *G. nipponicum* larvae in another group of patients that consumed loaches and in those eating kokanee (*Salmo nerka*), carp (*Cyprinis carpio*), crucian carp, and common ice-fish.[125,208] *G. doloresi* caused dermatitis in Japanese patients and in one case, brook trout (*Salmo masou masou*) were the suspected source.[125,208] The freshwater catfish *Clarias fuscus* and *C. batrachus* have been reported as hosts for *G. spinigerum.*[133]

Capillariasis

Capillaria philippinensis is a nematode that utilizes birds and fish as hosts, and humans become accidental hosts when they eat raw or undercooked fish.[42] Diarrhea, borborygmi, and abdominal pain are symptoms. If left untreated, conditions can worsen to include weight loss, weakness, malaise, anorexia, edema, cachexia, and eventually death. During autopsy, many thousands of worms can be found in the intestine.[42] *C. philippinensis* has been treated successfully with mebendazole at 200 mg twice a day for 20 days or albendazole at 200 mg per day for 10 days.[42] Chronic infections may require fluid and electrolyte replacement and a high-protein diet.[42]

Diphyllobothrium latum

Larvae, also called plerocercoids or grubs, of the broad tapeworm or fish tapeworm (*Diphyllobothrium latum*) can cause disease in humans if host fish are consumed raw or undercooked.[62] Bears, dogs, cats, coons, and other mammals consuming infected fish serve as the normal hosts for the worm.[169] Dick et al. also mention two other species of *Diphyllobothrium* that infect humans: *D. ursi* and D. *dendriticum.*[50]

Cold smoking of fish is not hot enough to kill the *D. latum* plerocercoids, so people consuming infested, cold-smoked fish are susceptible to infection. The adult cestode develops in the person's intestine, can reach lengths of 50 to 60 feet, and can live for up to 10 years.[164] It is commonly found in Russia and Scandinavian countries, especially Finland.[41] In Canada, the disease is primarily restricted to the northern regions.[50]

Symptoms include abdominal discomfort, diarrhea, nausea, and weakness.[209] The presence of the adult broad tapeworm in the intestine can contribute to pernicious anemia in the infected person due to the parasite's absorption of vitamin B_{12}.

Prevention is achieved by killing the *D. latum* plerocercoid in the fish with heat $\geq 56°C$ (133°F) for 5 minutes, or freezing at –18°C (0°F) for 24 hours or –10°C (14°F) for 72 hours.[253] Incidence of *D. latum* can be reduced by treating sewage from lakeside homes and ships, and by deworming domestic pets.[164]

Clonorchis sinensis

C. sinensis, the liverfluke, is prevalent in Southeast Asia, and an estimated 7 million people are infected worldwide.[203] This parasite is the most common zoonosis in Hong Kong, where 36–60% of autopsies are positive for *C. sinensis.*[133] Indigestion, epigastric discomfort, diarrhea, and acute pancreatitis are among the symptoms of the disease.[41] Chronic infections of *C. sinensis* sometimes can lead to cholangiocarcinoma. The life cycle of this digenetic trematode utilizes snails, fish, and mammals as hosts.[164] Prevention of *C. sinensis* infections can be attained by the usual cooking and freezing procedures. The common practice in many Asian countries of using human

manure for fertilizing aquaculture ponds should be discontinued.[62] Fish hosts of the liverfluke include silver carp (*Hypothalamicthys molitrix*) and grass carp (*Ctenophargyndon idellus*),[133] both of which are among aquaculture species.

Opisthorchiasis

The digenetic trematodes *Opisthorchis viverrini* and *O. felineus* infect people in Southeast Asia and Eastern Europe, respectively. Infection with this fluke causes symptoms similar to those caused by *C. sinensis* but is considered more threatening, because it causes a higher incidence of cholangiocarcinoma. Hinz, et al. list liver complaints, obstructive jaundice, and liver cirrhosis as symptoms of the disease.[111] *Opisthorchis* parasitizes the bile ducts of piscivorous mammals and humans. Opisthorchiasis is a major cause of death in northeast Thailand, and it is estimated that 7 million people are infected in Thailand.[108] There is also a very high prevalence of the disease in Laos; 51% of 224 Laotian students examined in one study were carrying this parasite or some kind of small fluke.[88] Opisthorchiasis is transmitted (and controlled) similarly to infections caused by *Clonorchis*, and the life cycle also involves snails, fish, and piscivorous mammals. The broad-spectrum antihelminthic praziquantel is discussed as a treatment by Hinz, et al.[111] They theorize that by treating opisthorchiasis in Thailand during the time of year when reinfection is lowest, the disease can be eliminated. Most likely, however, the only means of controlling it is praziquantel followed by abstaining from eating raw or undercooked fish.

Nanophyetus salmincola

N. salmincola is a digenetic trematode of salmon that can infect people that consume raw or undercooked salmon.[83] It also has caused infection by being transferred from the hand to the mouth of a person working with a contaminated salmon.[106] *N. salmincola* imbeds in the wall of the small intestine and causes abdominal pain, diarrhea, nausea, vomiting, weight loss, and fatigue.[65] Although people can serve as the definitive host, the natural definitive mammalian hosts are raccoons and skunks.[62] Snails, fish, and mammals serve as hosts in the parasite's complex life cycle.

N. salmincola also serves as a vector of the pathogenic rickettsia *Neorickettsia helminthoeca*, which is capable of infecting dogs.[62] Dogs contract the disease after eating raw salmon infected with *N. salmincola* carrying *N. helminthoeca*.

Cryptosporidiosis

Infections in humans caused by the protozoan *Cryptosporidium* were first reported in 1976, but occurred only in immunocompromised patients.[173] In 1982, however, it was discovered infecting an immunocompetent person, and since then has been diagnosed in a wide range of patients from over 50 countries on all the continents.[173] *Cryptosporidium* infects the intestines, causing diarrhea (profuse and watery, often containing mucus), copious and frequent bowel movements contributing to weight loss, crampy abdominal pain, low-grade fever, nausea, vomiting, malaise, weakness, fatigue, headache, myalgia, and anorexia.[173] Prolonged infection can lead to chronic diarrhea and proximal small intestinal enteropathy. Immunocompromised patients sometimes experience extraintestinal cases of cryptosporidiosis in the gall bladder, liver, pancreas, and even in macrophages in the lungs of one patient.[173]

Nine species of fish have been found to be carriers of *Cryotosporidium*. They are mostly cultured, captured, or ornamental, fish including tropical marine fish,

brown trout, tilapia, *Clarias* catfish, and common carp (e.g., ornamental koi).[173] They typically do *not* cause disease in the infected fish or pathological changes in the fish's intestine. Although there has not been a report of fish-to-man transmission of *Cryptosporidium*, the parasite's ability to infect man and its presence in fish make it a potential zoonotic problem for people working around aquarium, ornamental, or cultured fish.

Enterocytozoon sp.

The microsporidian species *E. bieneusi* is a parasite that occurs in AIDS patients. The closely related species *E. salmonis* is found in the salmonid fish chinook salmon and rainbow trout. Desportes-Livage, et al.[49] found that the two *Enterocytozoon* species are closely related, opening possibilities for a model by which to study the AIDS-associated species. The ability of *E. salmonis* to infect salmonids may point to possible animal origins of the closely related *E. bieneusi* species (known thus far to occur only in humans).

Giardia lamblia

The flagellated protozoan *G. lamblia*, is an intestinal parasite that sometimes is contracted by hikers/campers who drink directly from mountain streams without disinfecting the water. It has been found in canned feral salmon, due to fecal contamination.[177] The infection sometimes is called "**beaver fever**," and symptoms include lethargy, intense hunger followed by satiation after eating very little, nausea associated with certain odors, and yellowish-to-white diarrhea caused by blockage of the bile duct.[62] Malabsorption of fats and fat-soluble vitamins may occur. The disease is spread easily to family members; thus, infected individuals must practice strict sanitation.[62]

Prevention and Treatment of Parasite Problems

Deardorff outlines various foods that include raw fish as an ingredient, all potential sources of parasitic infection.[45,47] Sushi is raw seafood surrounded by rice and nori wrap; sashimi is thinly sliced raw fish; Japanese salad is raw fish, lettuce, and soya sauce; Hawaiian lomi lomi salmon is chopped salmon, bell peppers, and tomatoes; tako poki is a raw cephalopod dish; palu is meat from a fish head and visceral organs that have been allowed to ripen in a closed container; Latin American ceviche is a fillet marinated in lime juice; Philippine bagoong is uncooked fish viscera often in a deteriorated state; and Pacific Island poisson cru is a fish fillet marinated in coconut juice.

The recommended procedures of freezing and heating are the basic ways to reliably prevent parasite infections.[238] Other techniques such as brining and physical removal of the parasites offer partial control. Fish can be frozen at –20°C (–4°F) for 7 days or at –35°C (–31°F) for 15 hours[238] to kill any parasites present. Parasites also can be killed by heating the host fish to 60°C (140°F) for at least 5 minutes.[253] Preserving the fish with salt in 20–30% brine for 10 days has been shown to kill resident parasites.[164] However, the U.S. Food and Drug Administration (FDA) points out that larvae nematodes have survived in a 21% brine solution for 28 days.[238] Candling and physically removing parasites from the fillet are semieffective, but do not eliminate the hazard. Consumption of aquacultured fish also reduces the threat of contracting a parasite, especially when they are exclusively fed a formulated, pelleted diet. Aquacultured fish that are fed processing waste and by-catch fish pose a potential parasite hazard, as do fish that are exposed to snails and birds (which occur

naturally in and around most ponds).[238] Cooking or freezing the fish before consumption, as described, eliminates these parasite dangers.

Treatment with the broad-spectrum antihelminthic praziquantel (Biltricide) is mentioned for use against a wide range of trematode and cestode infections.[111] There is a need for improved diagnostic methods, including the use of monoclonal antibodies in ELISAs, the gastrofiberscope, and ultrasound, to detect the parasites as early as possible for prompt treatment.[125]

VIRUSES

Viruses are the most common cause of shellfish-associated diseases in humans.[145] **Norwalk viruses** (a group of small, round-structured viruses) caused 33% of shellfish-related diseases reported in New York between 1981 and 1992.[145] These viruses were first linked with gastroenteritis in 1972.[134] Symptoms include nausea, diarrhea, vomiting, headache, fever, and abdominal cramps. Treatment involves replacing lost fluids. Sources of infection are contaminated food (including raw shellfish, salad, and cake frosting), water and ice, airborne droplets, and other persons infected with the Norwalk virus.[134,243]

A 1993 Norwalk virus outbreak was traced to an oyster bed in Louisiana; oyster harvesters in this area had been routinely discarding their sewage into the water. The sewage included feces of one waterman who was infected by a Norwalk virus. Even though the dilution factor of discarding sewage overboard is high, spread of the disease in this manner is possible due to the fact that oysters concentrate viruses during their filter feeding, and because the infectious dose of Norwalk virus needed to cause disease is very low.[134] Although Louisiana laws prohibit such practices, enforcement was not being actively practiced at that time due, in part, to the remoteness of most oyster beds.[134] Educating harvesters on the damage that this practice can inflict on their own industry may prevent such incidences from occurring in the future.

People affected by this 1993 outbreak had consumed oyster stew that may not have been heated sufficiently to inactivate the Norwalk virus.[134] Studies have shown that Norwalk virus can survive temperatures of 60°C for 30 minutes. Similar findings revealed that simply steaming oysters may not be sufficient to inactivate Norwalk virus.[129] In the 1993 outbreak, persons who had consumed large quantities of the contaminated oysters prepared by frying were unaffected.[134]

In 1996, an outbreak of 97 cases of Norwalk virus gastroenteritis occurred in the Queensland and New South Wales areas of Australia over a 4-week period.[223] Ninety-two of the patients had eaten raw oysters 3 days prior to developing the illness.

Romalde and Green review new molecular methods (such as polymerase chain reaction) for detecting food-borne viruses, such as Norwalk virus, that normally are difficult to detect.[98,204]

SAFETY

The general public's awareness of the necessity for food and workplace safety is increasing. Evidence of this can be observed with the establishment of the Working Group for Quality Assurance in Aquaculture Production.[122] Also, OSHA inspects rainbow trout aquaculture facilities employing more than 25 persons and oversees worker safety during construction of metal frame and netting structures used to protect trout from bird depredation.[62] There is even a newly published book, *Spawn, Spat, and Sprains: A Manual for Aquaculture Safety in Alaska* (1998), which covers small boat safety, injuries from fish, electrical hazards on the water, cold injuries, cold water survival, and threats from bears.[63]

Antibiotic Safety

Localized skin reactions, respiratory problems, or generalized anaphylactic shock may result from repeated exposure to antibiotic.[120] Accidental injection or contact of the antibiotic powder with the skin can lead to the development of an allergy.[210] Fish health workers, researchers, fish producers, and feed mill operators are susceptible.

Misuse of antibiotics by fish producers, such as administering the antibiotic at lower-than-therapeutic doses or shorter-than-therapeutic periods of time, can allow the bacteria to adapt to the drug and develop resistance. This poses a problem for the producer, who then has an ineffective antibiotic against future infections caused by the resistant pathogen, but more importantly, humans infected with a resistant bacterial strain are in danger of prolonged infection if the ineffective antibiotic is prescribed initially.[257] The pathogens *Salmonella* sp., *Aeromonas hydrophila*, and *Plesiomonas shigelloides*, which are capable of infecting humans, were isolated from a Southeast Asian fish farm and showed resistance to nalidixic acid, oxolinic acid, chloramphenicol, neomycin, oxytetracycline, tetracycline, furazolidone, and sulphamethoxazole plus trimethoprim.[236] This occurrence was due, in part, to the Southeast Asian practice of using human sewage to fertilize fish ponds.

Withdrawal times required by U.S. FDA for the antibiotics Terramycin (oxytetracycline), Romet-30, and Romet-B (sulfadimethoxine + ormetoprim) allow residues to fall to safe concentrations in edible portions of catfish and salmonids.[193] A 21-day withdrawal period is required after the last feed application of Terramycin. This allows the residue level to drop below 0.1 mg/L in the fish flesh, providing a safe level for the consumer.[195] For Romet-30, a 3-day withdrawal for catfish and a 6-week withdrawal for salmonids allow drug residues to drop below the 0.1 mg/L concentration in both species.[59,194] The FDA also checks for drug residues in imported fish. The use of drugs in the salmonid aquaculture industry is reviewed by Burka, et al.[23]

Vaccine Safety

In the salmon farming industry, individual fish are vaccinated against the bacterial diseases furunculosis, caused by *Aeromonas salmonicida* subspecies *salmonicida*, and vibriosis.[62] Vaccinators hold each salmon smolt (weighing about 30–100 g) and use an automatic repeating syringe to inject the vaccine intraperitoneally.[140] A skilled vaccinator can vaccinate 2500 or more fish per hour. The furunculosis vaccine may contain a formalin-killed culture of *A. salmonicida* subsp. *salmonicida*, and adjuvants such as glucan, aluminium hydroxide, aluminium phosphate, and mineral oil.[140] Accidental autoinjections (usually in the hand or fingers) have caused inflammatory reactions, fever, muscle ache, general malaise, edema, infiltration, lymphagitis and swollen axillary glands, dizziness, and headache.[139,140] A few anaphylactic reactions have developed, with coma, dyspnea, tachycardia, and confusion. Two documented anaphylaxis cases involved the vaccinators being injected previously, with only mild local reactions.[140] Both cases featured furunculosis vaccine with either aluminium phosphate or glucan adjuvants. Adrenaline or adrenaline plus steroids were both effective treatments.

In one case, a furunculosis vaccine with mineral oil adjuvant was accidentally injected into a finger, which had to be amputated. The mineral oil was probably the major contributor to the severe local reaction in this patient.[140]

A double safety bow, which is a metal safety guard surrounding the needle, was introduced in 1992 and has been effective in reducing the risk of self-injection.[140] Other safety measures now recommended are: (1) keep a source of adrenaline

available to vaccinators, (2) educate local physicians near vaccination sites about injection accidents, and (3) provide thorough training for vaccinators.

Anesthetic Safety

The anesthetic 2-phenoxyethanol has been reported to cause problems in people. Three women anesthetizing small salmon developed headaches and intoxication symptoms.[163] Shortly after exposure to the 2-phenoxyethanol, all three women had diminished sensation and strength in their hands and fingers, and the problems were worse in the dominant hand. After 1–2 years of exposure to the anesthetic, the women developed focal cognitive impairments, and one of them had hearing problems.[163] Skin and eye protection should help to avoid problems from contact with anesthetics. This anesthetic is not a problem in aquaculture, because it is not approved by the FDA for use on food fish. Tricaine methanesulfonate (MS-222), the anesthetic approved for use in aquaculture, has not been associated with any human health problems.

Chemicals for Testing Water Quality

Aquaculturists must routinely monitor the quality of water where they are growing their fish in order to assure the health and optimum growth of their crop. Caution must be taken to prevent skin and eye contact when working with chemicals for testing water. The chemical mercuric iodide plus sodium hydroxide (also called Nessler Reagent), used to test for ammonia concentrations in water, especially should be handled with care. Other chemicals in standard aquaculture test kits (such as sulfuric acid) contain warnings about flammability, toxicity if ingested, and ability to irritate if contacted with skin or eyes. These neatly packaged chemicals are convenient and are used with much repetition by fish culturists. It is easy, therefore, for the water quality tester to assume that there is no risk, which may lead to careless contact and possible ill effects.

Shrimp Preservatives

Sodium metabisulfite (sometimes called "shrimp dip") is a preservative used in the shrimping industry. In sulfite-sensitive individuals, bronchospasm, oculonasal symptoms, and urticaria/angioedema may result from exposure to this chemical.[7] Several years ago, two shrimp trawler crewmen were found asphyxiated to death in the hold of the ship after using sodium metabisulfate. Their deaths were due to inhalation of sulfur dioxide, which resulted from a reaction of the sodium metabisulfite with acid and water.[7] Although this kind of incident has not occurred in the marine peneid shrimp aquaculture industry, those working with shrimp preservatives should be aware of such potential dangers.

There have been some reports of allergic reactions to sodium bisulfite, which is applied to marine shrimp to prevent melanosis (black spot).[178] FDA guidelines that restrict sulfite concentrations to 100 mg/L are helpful in minimizing the occurrence of these type of reactions.

Hydrogen Sulfide Gas

Another gas posing a threat to people is hydrogen sulfide (H_2S), which is naturally produced during anaerobic reactions on pond bottoms. Fish are highly susceptible to H_2S; channel catfish fry die when exposed to concentrations as low as 0.005 mg/L.[235] In a rare incident, three of four men who fell into a flatfish culture pond drowned; autopsies revealed that the men had been exposed to sublethal levels of H_2S, which led to their drownings.[128]

Fish Processing Injuries

Processing equipment, as well as the fish being processed, pose safety risks to processors.[62] U.S. government regulations require workers at cultured rainbow trout processing plants to rotate periodically from jobs where carpal tunnel syndrome is a risk. The band saw used to remove the head of farm-raised catfish can cause severe cuts and even severing of the fingers in spite of metal-link gloves worn by the band saw operator.[62] Some skilled operators remove the heads of over 60 fish a minute. The live catfish are electrically stunned as they enter the processing plant. This usually prevents them from moving during the beheading process, but occasionally the fish will jerk, which can pull the operator's finger(s) into the open band saw. Cuts also can be received from the skinning machine blade and fillet knives. Any injury that can be expected when working around **heavy machinery** can occur in fish processing facilities.

The sharp spines of channel catfish pose a threat to processing plant personnel. The dorsal and pectoral spines are sharp and serrated, and can lock into an erect position.[99] These spines can transmit a **toxin** that produces a stinging sensation in persons who are punctured.[17] The spines of tilapia, especially those raised in closed, recirculating systems, can "inject" *Streptococcus iniae* bacteria into the fish handler, causing infection.[215]

Corneal erosion can occur when fish bile accidentally comes into contact with the eyes. In Norway, one worker did not give proper attention to his eyes after coming in contact with fish bile; this led to serious corneal opacity.[35] Eye protection should be worn during fish processing, and an eye-wash station should be close by for prompt rinsing when necessary.[62]

Occupational asthma (OA) to snow crab extracts was diagnosed in 16% of workers in a snow crab processing plant.[27] An increase in crab-specific serum IgE was found in the asthma patients,[28] and it was observed in one study that OA patients can remain symptomatic for prolonged periods after exposure to the crabs stops.[117] It was suspected that inhalation of crab boiling water was the cause for the asthma.[153] A later study confirmed that the snow crab allergens did become airborne near the site where the crabs were boiled, and affected workers in this area of the processing plant.[154] Other reactions in affected individuals include rhinoconjunctivitis and urticaria.[27]

Electrical Accidents

Electrically powered aerators that float in the water are commonly used around fish culture ponds. Disconnect and circuit breaker boxes are safety features used in such set-ups. The electricity travels from the power line to a meter and "disconnect" box with circuit breakers and then to individual circuit breaker boxes at each aerator.[62] If the circuit becomes overloaded, circuit breakers trip and interrupt the flow of electricity as long as the correct fuse size is used. Power can also be shut off manually from any circuit breaker box.

The electrical cord connecting the floating aerator to the circuit box is encased in metal conduit or PVC pipe and buried in the pond levees. Qualified electricians often are hired to do the electrical work around the water. Power to the circuit breaker boxes is shut off when work needs to be done in the water. And when heavy harvesting equipment is being used near overhead electrical lines, power to the lines is usually cut off by the electric company.[62] Almost every pond in the Mississippi Delta in the west-central part of the state (where there are more than 90,000 acres of channel catfish aquaculture ponds) has a floating electric aerator. The author is

aware of only one fatality due to these aerators, which occurred in the early 1980s. The electrical cord powering the aerator had not yet been buried in the pond levee when it became entangled underneath a farm manager's truck. When he tried to dislodge it, he was electrocuted.[62]

Other Safety Risks in Aquaculture

All farm tractors, including those used in aquaculture, should be equipped with rollover protection systems (roll bars) in compliance with OSHA guidelines. The power take-off (PTO) of tractors has a strong torque capacity that is used for powering fish pond aerators. Protective shields covering PTOs help to prevent clothing from getting caught, thus reducing the number of injuries.

Potentially dangerous torque also is found in electrically powered catfish hatching troughs that turn metal paddles attached to an axle. The on-off switch typically is too far from the paddles for a person to reach if he or she becomes entangled in the paddles or axle. A design built by Dr. Robert Busch (personal communication) at Indianola, Mississippi in the early 1990s had lines connected to the on-off switch stretching the length of the hatching trough. They could be pulled from anywhere along the trough to turn off the paddles.

Foam-padded floatation vests worn around water help to protect workers against drownings as well as hypothermia and chest and rib injuries.[55] They appear to be superior to life jackets that utilize CO_2 cylinders; in one study, 10 of 15 life jackets utilizing CO_2 cylinders failed to inflate when the subjects fell into the water.[55]

Drowning also may occur among SCUBA divers at salmon net-pen culture facilities. Divers have become entangled in netting while clearing dead and dying fish from the pens; at least one fatality has been reported.[55] Additionally, divers are continuously diving and resurfacing (called yo-yo diving) while performing their underwater tasks. No decompression tables are available for this kind of diving, and it has led to decompression (dysbaric) illness.[55] Ice-related accidents, as well as sunburn, also occur in aquaculture settings.

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