FISH MEDICINE

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ZOONOTIC DISEASES

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Zoonoses involving transmission of disease-producing agents from fish to man have been reviewed previously (Shotts, 1980, 1987; Eastaugh and Shepherd, 1989). Examination of recent literature pertaining to fishborne zoonotic diseases reveals three salient points. First, the number of reported outbreaks of fish-related diseases in the United States is increasing. Factors responsible include the increased awareness of disease symptoms; the increased rates of exposure to contaminated or infected fish via consumption or recreational activities; the increased contamination of marine, estuarine, and freshwater environments by chemical and biological pollutants; and the increased susceptibility to disease as a result of immunocompromising diseases such as acquired immunodeficiency syndrome (AIDS). Second, the overall incidence of fish-related diseases in the United States remains relatively low. Third, disease episodes in immunocompetent hosts are often self-limited bouts of gastroenteritis; however, patients may suffer serious disease and subsequent mortality if their conditions are improperly diagnosed, or if they have a preexisting disease that compromises their immune system. The purpose of this chapter is to review and summarize recently published information concerning known and potential fishborne zoonoses that occur in the United States.

BACTERIA

Bacterial diseases of fish are described in Part II of this book and in other texts (Frerichs and Roberts, 1989). The major bacterial diseases of fish in incidence and severity are caused primarily by gram-negative rods. A few of these organisms, and a small number of gram-positive species, also cause disease in humans. An important feature of many of bacterial organisms is their opportunistic nature. The development of disease in the human host often requires a preexisting state that compromises the immune system. In general, humans contract fishborne bacterial disease through ingestion of contaminated fish tissue or water, or by injection of the organisms into puncture wounds or abrasions. Exposures often result in inapparent or mild episodes of gastroenteritis or in localized infections of the skin and underlying tissue. However, a few bacteria are highly pathogenic and may produce high rates of mortality.

Gram-Positive Organisms

STREPTOCOCCUS. Members of the genus *Streptococcus* have been found to produce disease outbreaks among freshwater and saltwater fishes in the southern United States and Japan (Frerichs and Roberts, 1989). Many fish isolates are of the Lancefield group B and D serotypes (Shotts and Teska, 1989). Although no transmission from fish to man has been documented, the potential for human infection does exist among individuals who handle diseased fish.

STAPHYLOCOCCUS. Members of the genus *Staphylococcus* occasionally produce disease in fish (Shotts and Teska, 1989). *Staphylococcus* sp. has been isolated from aquarium water and has been found in aquaculture pond water in Africa (Ogbondeminu and Okaeme, 1986). The greatest potential for staphylococcal fishborne disease is probably by means of enterotoxin synthesis during improper food handling, rather than by infection with intact organisms.

CLOSTRIDIUM. Members of the genus *Clostridium* are found as commensal organisms in the intestinal tract of many marine and freshwater fishes. In fish, the bacteria rarely produce disease; however, isolated outbreaks of fish mortality have been described in salmonids (Frerichs and Roberts, 1989).

Clostridium species can cause serious and potentially fatal disease in humans who consume fish tissues in which the vegetative form has proliferated and produced heat-labile toxins. Two species (*C. perfringens* and *C. botulinum*) have produced disease outbreaks as a result of consumption of contaminated fish (Bean and Griffin, 1990). *Clostridium perfringens* causes clinical signs of gastroenteritis, which often resolve within 24 hours with minimal or no treatment. In contrast, *C. botulinum* produces five different forms of a neurotoxin that causes presynaptic blockage of acetylcholine release and consequent signs of diffuse lower motor neuron disease (e.g., generalized muscle dystonia, difficulty in swallowing or speaking, and ptosis). Death in affected individuals is usually caused by respiratory paralysis.

In fish tissues, type E toxin is produced almost exclusively (Telzak et al., 1990), although one disease outbreak involving contaminated fish was attributed to type A toxin (Rigau-Perez et al., 1982). Ingestion of uneviscerated whitefish (kapchunka) was determined to be the source of two outbreaks of type E food poisoning in the United States (Centers for Disease Control, 1987a; Telzak et al., 1990). The reasons why type E toxin is preferentially produced in fish are unknown, but it is believed to be a function of the fishes' intestinal microflora and tissue composition (Smith and Turner, 1989).

ERYSIPELOTHRIX. The genus *Erysipelothrix* has only one member, *E. rhusiopathiae* (formerly *E. insidiosa*). It is a facultative anaerobic rod with worldwide distribution. The organism appears to cause no disease in fish and is thought to be present in the external mucus of many freshwater and marine species (Wood, 1975).

Erysipelothrix rhusiopathiae does cause disease in humans, swine, sheep, avian, and other species. The clinical syndrome in man has recently been reviewed (Reboli and Farrar, 1989). Infections with *E. rhusiopathiae* are considered to be an occupational disease of individuals who handle animal tissues containing the organism. Butchers, abattoir workers, veterinarians, and cooks, both home and professional, have a higher risk of infection than the general population. The disease is considered to be particularly prevalent among persons handling fish. Three forms of the disease have been described in humans: a localized skin infection (erysipeloid, or "fish rose") often involving the fingers or hands, a diffuse cutaneous form in which a localized infection spreads to adjacent tissues, and a septicemic form.

The literature concerning *E. rhusiopathiae* septicemia has recently been summarized (Gorby and Peacock, 1988). Only 49 cases have been reported in the United States since 1912; however, the syndrome produced a 38% mortality rate among affected individuals. Endocarditis was the most common and serious sequela, occurring in 90% of patients. Fish or shellfish were believed to be the source of infection in 22% of reported cases. A history of alcohol abuse was found to be a predisposing factor to septicemia (33% incidence).

MYCOBACTERIUM. Organisms in the genus *Mycobacterium* are nonmotile acid-fast rods. Three species are recognized as pathogens of fish (Frerichs and Roberts, 1989). *Mycobacterium fortuitum* produces disease in a variety of fish found in tropical and temperate climates. *Mycobacterium chelonei* has been isolated primarily from salmonids. *Mycobacterium marinum* is a pathogen of tropical species of freshwater and saltwater fishes. Of interest is the fact that *M. fortuitum* grows at 30° and 37°C, whereas the other two species grow only at 30°C (Frerichs and Roberts, 1989). *Mycobacterium chelonei* and *M. marinum* may not be detected in some instances because the incubation of human isolates is commonly done at 37°C.

The advent of AIDS has spurred considerable interest in these and other mycobacteria other than tuberculosis (MOTT) species. Mycobacterial infections are common sequelae to AIDS, and MOTT species (primarily *M. avium* complex, formerly *M. avium-intracellulare*) are currently isolated at a higher frequency than *M. tuberculosis* in human immunodeficiency virus (HIV)–infected individuals (Centers for Disease Control, 1987b). The increased number of mycobacterial infections is of concern because the rates of infection have also risen in nonimmunocompromised individuals (Prince et al., 1989), and because inadequate diagnosis and treatment may result in excessive morbidity and mortality (Iseman, 1989).

In contrast to *M. avium*, only a low number of human infections with the three fish pathogens has been reported. Clinical data regarding these and other MOTT species have been reviewed (Woods and Washington, 1987). Persons infected with *M. fortuitum* and *M. chelonei* demonstrate a variety of clinical presentations that are correlated with the presence or absence of immunocompromising diseases such as AIDS. Typically, HIV-positive individuals show signs of disseminated or respiratory disease, whereas immunocompetent patients are more likely to exhibit circumscribed cutaneous lesions acquired through penetrating wounds. No cases of infection in the reported survey resulted from exposure to fish. However, *M. fortuitum* has been reported to produce cutaneous lesions on the hands of individuals handling contaminated aquaria (Shotts, 1987; Frerichs and Roberts, 1989).

Case histories of human infections with *M. marinum* have been reported (Donta et al., 1986; Gray et al., 1990). This organism is the agent of swimming pool and fish tank granuloma in humans and tuberculosis of fish. Humans are infected with *M. marinum* by contamination of lacerated or abraded skin during activities such as swimming or cleaning tropical fish aquaria. Two clinical syndromes are observed (Woods and Washington, 1987). A single granulomatous nodule may form at the site of infection, most commonly on the hands or fingers (Fig. 17–1). This lesion often resolves over a period of weeks to months without antibiotic treatment. The customary lack of dissemination to adjacent tissues is believed to be a reflection of the organism's inability to grow at 37°C (Brown and Sanders, 1987).

A "sporotrichoid" form of infection with *M. marinum* is sometimes seen. Localized infection is followed by spread to nearby lymph nodes, resulting in the appearance of nodules at the original site of infection and in regional lymphatic tissues. The bacterium has also been observed to occasionally spread deeper into adjacent tissues and produce arthritis, osteomyelitis, and tenosynovitis. Such infections may be the result of T-cell unresponsiveness to specific mycobacterial antigens in immunoincompetent patients (Dattwyler et al., 1987). More disseminated forms of the disease are likely in patients infected with HIV, and those individuals are advised
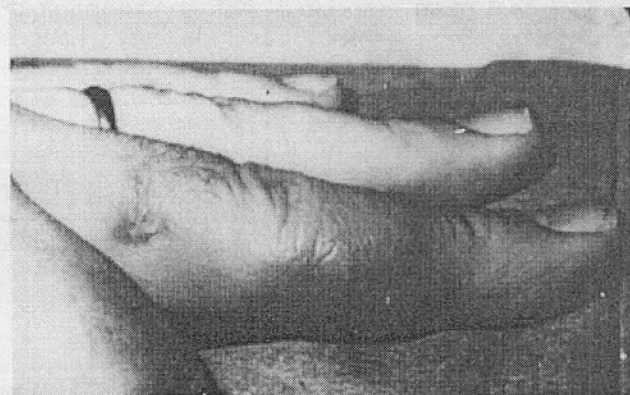

FIGURE 17–1. Human infection with *Mycobacterium marinum*.

to avoid cleaning tropical fish aquaria (Ries et al., 1990).

NOCARDIA. *Nocardia asteroides* and *N. kampachi* have been isolated at very low frequencies from tuberculoid lesions of both fish and man (Shotts, 1980, 1987; Frerichs and Roberts, 1989). Infections with these organisms may be misdiagnosed as mycobacterial disease because of the similarity of clinical signs and the positive reaction to acid-fast staining.

Gram-Negative Organisms

VIBRIO SPP. Members of the genus *Vibrio* are facultatively anaerobic, pleiomorphic rods that are commonly isolated from marine and estuarine waters; however, some species are also found in fresh water. They are considered to be the most significant bacterial pathogens of marine fish (Frerichs and Roberts, 1989). Nine species have been associated with foodborne diseases in humans, the most important species of which are *V. cholerae* 0 group 1, *V. cholerae* non-01, *V. parahemolyticus*, and *V. vulnificus* (Eastaugh and Shepherd, 1989).

Foodborne infections with *Vibrio* spp. in the United States are most often associated with consumption of raw or inadequately cooked shellfish. Clinical signs include vomiting and diarrhea that vary in severity according to the bacterial species causing the disease. In general, *V. cholerae* 0 group 1 causes the most severe signs. *Vibrio cholerae* non-01 infections are seen less frequently and are less debilitating. *Vibrio parahemolyticus* infections are frequently inapparent (Morris et al., 1981; Lowry et al., 1989). The apparent ubiquity of this organism in marine water gives credence to the idea that ingestion of improperly cooked fish may also be a source of human infection. In Japan, *V. cholerae* non-01 has been isolated at relatively high frequencies from market fish (Murase et al., 1988). One case of septicemia caused by ingestion of catfish contaminated with *Vibrio hollisae* has been reported in the United States (Lowry et al., 1986).

Human infections with *V. vulnificus* have been the object of some scrutiny (Johnston et al., 1985; Klontz et al., 1988; Hoffmann et al., 1988; Vartian and Septimus, 1990). The disease is one of low incidence and high mortality. Two clinical syndromes have been described. The first is a primary septicemia and is most often associated with consumption of raw oysters. Signs of infection include fever, changes in mental status, ecchymotic hemorrhages, bulla formation, and pain in the lower extremities. The mortality rate among affected individuals is approximately 50%, even with prompt diagnosis and treatment. A prominent predisposing factor is preexisting liver disease, especially cirrhosis, which is believed to adversely affect leukocyte migration (Morris, 1988).

The second clinical syndrome produced by *V. vulnificus* is one of wound infections and is characterized by cellulitis, edema, hemorrhages, bulla formation, and extensive tissue necrosis. Despite prompt and aggressive treatment, the mortality rate ranges from 25 to 30% among affected individuals. In most cases, infection is acquired by introduction of contaminated seawater into skin wounds.

PLESIOMONAS SHIGELLOIDES. The taxonomy of the genus *Plesiomonas* is uncertain, but at present it is considered to be a member of the family Vibrionaceae. *Plesiomonas shigelloides* is a facultatively anaerobic rod that causes septicemia in various species of fishes (Shotts and Teska, 1989). The organism is found in fresh water and salt water and has been isolated from the intestinal tract of tropical freshwater fishes (Centers for Disease Control, 1989).

Human infections with *P. shigelloides* are acquired through ingestion of contaminated water or uncooked fish and shellfish. Two clinical syndromes have been described (Brenden et al., 1988). The first and most common syndrome is one of gastroenteritis, which is characterized by fever, abdominal pain, vomiting, and diarrhea. The low incidence of positive cultures among asymptomatic individuals is believed to reflect the absence of a carrier state in humans. A majority (70%) of patients with this disease have either a preexisting debilitating disease such as cancer or cirrhosis or a risk factor such as recent foreign travel or seafood consumption. Contaminated water from a tropical aquarium has been implicated as the source in one case (CDC, 1989).

The second clinical syndrome caused by *P. shigelloides* is one of extraintestinal disease and is rarely encountered. Affected individuals may demonstrate meningitis, sepsis, and cellulitis. A recent clinical report described treatment of a pancreatic abscess from which *P. shigelloides* was cultured (Kennedy et al., 1990).

AEROMONAS. Aeromonad organisms are members of the family Vibrionaceae and are facultatively anaerobic rods. *Aeromonas salmonicida* is the agent of furunculosis in salmonids and is not considered to be a human pathogen. Five other species (*A. hydrophila, A. sobria, A. caviae, A. schubertii,* and *A. veronii*) are currently considered to be members of the motile aeromonas complex (Shotts and Teska, 1989). These organisms can produce septicemia in infected fishes and are routinely found in the aquatic environment. The species most commonly isolated is *A. hydrophila*. It is found worldwide in tropical and temperate fresh water and is considered to be part of the normal intestinal microflora of healthy fish (Frerichs and Roberts, 1989). *Aeromonas* spp. have been found in nearly 100% of freshwater tropical aquaria in a survey of English pet shops (Sanyal et al., 1987).

Despite the ubiquity of *Aeromonas* spp., available data suggest that motile aeromonads produce a low number of human infections. California reported a 1-year disease rate of 0.7/million population (Centers for Disease Control, 1990). Individuals infected with *Aeromonas* may show a variety of clinical signs, but the two most common syndromes are gastroenteritis and localized wound infection. Aeromonad gastroenteritis is produced by ingestion of contaminated water and is characterized by diarrheic states that

range from acute and self-limiting to chronic and unresolving (George et al., 1985). Wound infections due to aeromonads are sporadically reported and result from introduction of the organism via laceration or puncture wound. Such infections may be superficial or may progress to cellulitis, deep muscle necrosis, or septicemia. Although such infections have occasionally been documented in the nonimmunocompromised host (Heckerling et al., 1983; Karam et al., 1983), the primary concern from a public health standpoint is with individuals who have an immune deficiency as a result of AIDS or other chronic diseases and who acquire an aeromonad infection as a result of wound contamination (Flynn and Knepp, 1987).

PSEUDOMONAS. The genus *Pseudomonas* is a member of the family Pseudomonadaceae. Pseudomonads are motile aerobic rods that are commonly found in soil and water. *Pseudomonas fluorescens* infrequently produces a septicemic disease in pond, aquarium, and marine fish that is similar to the one seen with *Aeromonas* (Frerichs and Roberts, 1989). Although the potential exists for this organism to produce infections in humans with preexisting immunocompromising disease, no documented cases related to fish exposure have been reported.

THE ENTEROBACTERIACEAE. Members of the genera *Escherichia*, *Salmonella*, *Klebsiella*, *Edwardsiella*, and *Yersinia* are included in the family Enterbacteriaceae, which occur as facultatively anaerobic rods. Enteropathogenic *Escherichia coli* has been isolated from fish but apparently does not produce disease (Shotts, 1987). *Salmonella* spp. have been isolated from freshwater aquaria, and fish are apparent carriers of the organism (Shotts, 1987; Sanyal et al., 1987). Similarly, *Salmonella* spp. have been isolated from numerous marine species, including fishes, shellfishes, cetaceans, and pinnipeds (Minette, 1986). At present, the primary risk of infection with *E. coli* and *Salmonella* appears to be through the ingestion of improperly prepared contaminated fish food products. Isolated instances of such food poisonings have been reported (Bean and Griffin, 1990; Bean et al., 1990). *Klebsiella* spp. are found in aquatic environments, and one case of *K. pneumoniae* septicemia has been reported as a consequence of handling contaminated fish (Reagan et al., 1990).

Two species of *Edwardsiella* are known pathogens of fish. *Edwardsiella ictaluri* is the agent of enteric septicemia in channel catfish. No instance of human infection with this bacterium has been reported. *Edwardsiella tarda* is the agent of emphysematous putrefactive disease in catfish and is found in the intestinal microflora of rats, birds, amphibians, fish, and other species (Frerichs and Roberts, 1989). *Edwardsiella tarda* infections in humans have recently been reviewed (Wilson et al., 1989). Infection with this organism is by ingestion or a penetrating wound. Infected fish and contaminated water are sources of infection. A variety of disease states may develop, including gastroenteritis (the most common symptom), localized infection, an asymptomatic carrier

state, or septicemia. Documented infections are rare. Persons with serious preexisting illnesses are predisposed to infection with *E. tarda*, and as a consequence the mortality rate is high (44%).

Yersinia ruckeri is the agent of enteric red-mouth disease in salmonids and is considered to be highly pathogenic to infected fish. In contrast, only one case of human infection with this organism has been reported (Farmer et al., 1985).

LEPTOSPIROSIS. Three cases of *Leptospira icterohaemorrhagica* infection among fish farmers in England in 1981 caused concern that fish and other aquatic species could harbor *Leptospira* and transmit the disease to humans. An epidemiologic survey concluded that English fish farmers were at increased risk of developing *leptospirosis* (Gill et al., 1985). However, the authors were unable to identify any factors or husbandry practices that predisposed to development of the disease.

Definitive data are lacking concerning the existence of *Leptospira* infections in fish and the possible role of fish in disease transmission. An early report (Maestrone and Benjaminson, 1962) stated that *Leptospira* could be isolated from goldfish tissues up to 17 days after experimental infection. Since that time, little has been published concerning the possible role of fish in the etiology of human disease. Studies of leptospirosis in aquatic species have concluded that fish can harbor the organism. However, more field and experimental data are needed to accurately determine the risk of human infection (Minette, 1983).

TOXINS

A large number of marine organisms produce toxic substances that can cause illness and death in man. In most instances, however, poisonings caused by marine species occur as isolated events. The toxicology and clinical syndromes produced by these agents have been comprehensively reviewed (Halstead, 1988). Ciguatera and scombroid food poisonings are worthy of discussion because they are frequently implicated as causes of foodborne intoxications in the United States and other countries. Statistics compiled by the Centers for Disease Control showed that chemical poisonings accounted for 26% of foodborne disease outbreaks and 2% of all clinical cases from 1983 to 1987 (Bean et al., 1990). Fish poisonings resulting from ingestion of ciguatoxin and scombrotoxin accounted for 73% of these outbreaks.

Ciguatera Poisoning

The term *ciguatera* is believed to be derived from *cigua*, the common name for a Cuban gastropod that produces similar clinical signs when ingested (Lee, 1980). The intoxication is highly endemic in tropical regions (Morris et al., 1982). However, modern transportation of fish and man has made ciguatera a condition of worldwide occurrence (Lawrence et al., 1980; Halstead, 1988).

Ciguatera poisoning is caused by ingestion of the meat of carnivorous reef fishes such as grouper,

snapper, kingfish, and barracuda. Ciguatoxin is probably produced by the dinoflagellate *Gambierdiscus toxicus*. The dinoflagellate adheres to the surfaces of reef plants, which are consumed by herbivorous fishes. These fishes are in turn consumed by predatory species. Ciguatoxin accumulates in the liver, intestines, reproductive organs, and muscles, and affected fish may demonstrate clinical signs of neurotoxicity (Halstead, 1988). Factors resulting in increased rates of dinoflagellate growth (storms, rains, chemical and organic pollutants) also produce increased levels of ciguatoxin in fish meat (Lee, 1980).

Ciguatera poisoning in man is primarily a syndrome of high morbidity, although deaths have been reported (Bagnis et al., 1979). The clinical syndrome has recently been reviewed (Frenette et al., 1988). Frequently affected individuals develop signs of gastroenteritis (nausea, vomiting, and diarrhea) within 5 to 6 hours of ingestion. These symptoms usually resolve within 1 to 2 days, although diarrhea may persist for longer periods.

A second phase of the intoxication usually starts 18 to 24 hours after ingestion of contaminated food. It is characterized by neurologic signs such as paresthesias of the mouth and extremities, pruritus, myalgia and arthralgia, headaches, and weakness. Cold sensitivity of the mouth and extremities is highly characteristic of the condition. Patients often report a paradoxical burning sensation upon exposure to cold food or objects. Bradycardia, shock, and coma may develop in cases of severe toxicity. These neurological signs may in part be attributable to cerebral edema. This could account for the dramatic clinical improvement seen in some patients after intravenous administration of mannitol (Palafox et al., 1988).

The biphasic clinical syndrome may be related to the presence of more than one toxin in contaminated fish (Halstead, 1988). Ciguatoxin has been characterized as a lipid-soluble polyether. It is believed to produce neurological symptoms by acting on the sodium channels of nerve cells and effecting changes in their permeability and electrical potential (Palafox et al., 1988). The lipid nature of ciguatoxin may render it soluble in body fluids. In two recently described cases of ciguatera poisoning, the sources of exposure were believed to be sexual contact and breast feeding (Lange et al., 1989; Blythe and de Sylva, 1990).

Diagnosis of ciguatera poisoning is based on the clinical signs and a history of recent ingestion of carnivorous marine fish. Although the toxin is currently not detectable in human samples, an enzyme immunoassay system can screen for its presence in fish tissues (Hokama, 1985). Treatment of the toxicity is largely symptomatic. In addition to mannitol, other recently tested drugs include the sodium channel blocker amitryptiline and the calcium channel blocker nifedipine (Calvert et al., 1987). Prevention is hampered by the fact that ciguatoxin is heat and cold stable and does not impart an odor or taste to contaminated meat.

Scombroid Poisoning

Scombroid toxicity derives its name from the fact that most cases are seen after ingestion of marine fishes of the family Scombroidae. Representative species implicated in food poisonings include tuna, mackerel, bonito, and skipjack. Other nonscombroid species (bluefish, mahi-mahi, herring, and sardines) have also been identified as vectors in outbreaks (Taylor et al., 1989). Although the reported incidence of scombroid poisoning is less than that of ciguatera, it is believed to be underreported and often misdiagnosed as food allergy (Eastaugh and Shepherd, 1989).

The exact mechanism of scombroid food poisoning is not completely understood (Halstead, 1988). Surface contamination of fish with bacteria such as *Proteus* and *Klebsiella* spp. results in the accumulation of histamine. Tissues of scombroid fish contain high levels of histidine, and the bacteria convert this amino acid to histamine via the enzyme histidine decarboxylase. Histamine may accumulate to toxic levels if contaminated fishes are left unrefrigerated for as little as 3 to 4 hours (Kow-Tong and Malison, 1987).

Ingestion of histamine has previously been believed to produce the clinical signs of scombroid food poisoning. These include a rapid onset of nausea, vomiting, diarrhea, a burning sensation of the mouth, hives, pruritus, flushing, and skin rash (Taylor et al., 1989). Levels of histamine in fish tissue are positively correlated with the occurrence and severity of clinical signs and are used to diagnose cases of scombroid poisoning. However, histamine is degraded during ingestion and does not produce signs of food poisoning when administered orally (Halstead, 1988; Eastaugh and Shepherd, 1989). It has been suggested that scombrotoxin is not histamine per se, but rather a potentiator of histamine activity (Taylor et al., 1984).

The diagnosis of scombroid poisoning is based on clinical signs and a history of recent ingestion of fish. Treatment is symptomatic and consists of supportive therapy (including antihistamines). The disease is best prevented by refrigeration of fish during all stages of handling (Bartholomew et al., 1987).

PARASITES

In the United States, the incidence of parasitic zoonoses attributable to fish is quite small. This is in contrast to other countries and geographic regions (e.g., Japan, Russia, the Far East) where rates of infection reach significant levels (Wootten, 1989). In the United States, most reported cases involve consumption of fish, which are intermediate hosts for intestinal parasites of fish predators. The incidence of fishborne parasitism has recently increased, and this is thought to be the result of dietary changes; i.e., greater consumption of raw or undercooked dishes such as sushi or sashimi (McKerrow et al., 1988; Wittner, et al., 1989).

Nematodes

ANASAKIASIS. Anasakiasis is a rare but increasingly observed clinical disease in the United States. Only 50 cases have been reported since 1958, but 70% of these have occurred since 1980 (McKerrow et al., 1988). Humans are infected by eating fish meat containing third-stage larvae of *Anasakis simplex* and *Pseudoterranova* (formerly *Phocanema*) *decipiens*. These nematodes normally parasitize marine mammals that consume infected fish. Many cases of anasakiasis occur in the western United States, and this is believed to reflect the geographical location of marine mammals and intermediate hosts such as salmon, herring, and Pacific cod (Kliks, 1986).

In many instances, human infection with anasakid parasites produces no ill effects. Larvae are often regurgitated 1 to 2 days after ingestion. However, larvae may burrow into the walls of the stomach or intestines and produce acute illness that may be clinically indistinguishable from gastric ulceration or appendicitis. Laparotomy often reveals the presence of inflamed portions of the gastrointestinal tract and the parasite. Anaskiasis is prevented by cooking or freezing fish meat.

EUSTRONGYLOIDES. Human infections with other nematode parasites have been described. *Eustrongyloides* causes an anasakiasislike syndrome of acute abdominal pain. Infection has been reported to occur after ingestion of live bait minnows and sushi (Centers for Disease Control, 1982; Wittner et al., 1989). An unusual case involved the migration of a dracunculoid nematode (*Philometra* sp.) into an open hand wound (Deardorff et al., 1986).

CESTODES. Human infections with cestode parasites such as *Diphyllobothrium latum* have been noted (Wootten, 1989; Eastaugh and Shepherd, 1989). In contrast to other fishborne parasites, most cases involve ingestion of freshwater fishes such as pike or walleye. Typical clinical signs include mild intestinal upset (cramps, loose stools). Megaloblastic anemia is occasionally found during diagnostic evaluation. Diagnosis is by demonstration of ova in feces.

TREMATODES. Trematode infections in humans are rare in the United States. One case report documented infection with *Heterophyes heterophyes* as a consequence of eating sushi (Adams et al., 1986). A number of human infections with *Nanophyetus salmincola* have recently been diagnosed (Fritsche et al., 1989; Harrell and Deardorff, 1990). This intestinal fluke is of interest to veterinarians because it is the biological vector for *Neorickettsia helminthoeca*, the agent of salmon poisoning in dogs. Human infection with *Nanophyetus* is caused by the ingestion of raw or partially cooked salmon. Clinical signs may include abdominal pain and diarrhea, but patients are often asymptomatic. Diagnosis is by demonstration of ova through fecal flotation.

PROTOZOA. Human infection with fishborne protozoans are very rare. The coccidial organism *Cryptosporidium* is known to have a wide range of hosts, including man and fish (O'Donoghue, 1985). Humans are predisposed to infection by congenital or acquired immunodeficiency diseases. Although no cases involving transmission from fish to man have been reported, it is possible that these may occur. One outbreak of giardiasis has been attributed to fecal contamination of canned salmon (Osterholm et al., 1983).

VIRUSES AND FUNGI

No human infections with fish viruses have been reported (Wolf, 1988). However, San Miguel sea lion virus, a calicivirus known to produce vesicular disease in both marine mammals and pigs, has been shown to elicit antibody production in humans and vesicular lesions in primates. The virus is believed to be transmitted by various marine fishes, and it has been suggested that humans may become infected during handling of fish vectors (Barlough et al., 1986a, 1986b). The practice of integrated fish farming may contribute to the development of human influenza pandemics (Scholtissek and Naylor, 1988). New strains of human influenza virus are thought to be produced by genetic reassortment of human and avian influenza viruses, and this reassortment is believed to occur in pigs. The proximity of humans, pigs, and birds is most notably seen in polyculture systems in Asia, where duck and/or pig feces are used to fertilize aquaculture ponds. These ponds may be fertile areas for the growth of both fish and new strains of human influenza (Scholtissek and Naylor, 1988).

No incidence of human fungal infection with fish pathogens has been described (Roberts, 1989). However, *Candida albicans* has been cultured from skin lesions of mullet (Macri et al., 1984).

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