MARINE FISH PARASITES OF PUBLIC HEALTH IMPORTANCE

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SUMMARY

The most commonly encountered marine fish parasites with human infection potential are larval anisakid nematodes that normally parasitize adult marine mammals. Cestodes in the genus Diphyllobothrium also infect humans, who may acquire the worms from ocean-caught salmon. Usual cooking procedures render fish preparations safe because parasites are killed after 1 min at 60°C. Freezing to -20°C for 24 hr also kills the parasites. Although the recent popularity of raw fish preparations has resulted in an increased occurrence of human infections from this source, the total number of cases reported in North America and Europe is very low. This contrasts with Asia where cultural factors lead to a greater risk of infection.

INTRODUCTION

Parasitism is extremely common, and virtually all animals host a variety of parasitic organisms from viruses to helminths. Traditionally, the science of parasitology treats the animal parasites, organisms with representatives from most animal groups and excluding microorganisms such as viruses and bacteria. Control of parasites in domestic animals has met with substantial success and consumers rarely encounter macroscopic parasites in products from these animals. There is no similar opportunity to control parasitic infections in most seafood products that are harvested from the wild, and marine fishes caught by commercial fishermen are actually small communities consisting of a host and its parasites.

Fortunately for the sensitivities of most seafood consumers, the bulk of the parasitic organisms that live in or on marine fishes occupy those portions, such as visceral organs, that are disposed of when the fishes are prepared for market. There are, however, a number of parasites that commonly occur in fish flesh and of these, there are several species that can infect humans. Such infections only occur if the fish is consumed raw or lightly marinated and has not been frozen before preparation. Cooking also kills the parasites and renders infected fish safe to eat.

Because potential human pathogens occur in marine fish flesh, there is need for parasite detection and identification, for the effective diagnosis and treatment of human infection, and for methods of ensuring the safety of marine fish products.

Recognition of parasitic infections associated with marine fish consumption has a short history primarily concerning infections caused by nematodes belonging to the family Anisakidae. The emphasis of this review
will be on those helminths. Other reviews of anisakid nematodes that are potential human pathogens include those of Oshima, of Margolis, and of Smith and Wootten [refs. 1-3]. Also considered in this review are cestodes (tapeworms) in the genus *Diphyllobothrium*. Although the species known to infect humans have freshwater life cycles, they may be acquired from salmon caught during the marine phase of their life cycle. Species that infect pinnipeds may have entirely marine cycles. Other human parasitic infections that result from ingesting raw fish are usually associated with freshwater fishes in Asia and are outside the scope of this review.

Although larval stages of anisakid nematodes have probably been known in fishes since the thirteenth century [ref. 4], it is generally agreed that these nematodes were not recognized as potential human pathogens until 1955 [refs. 3,5] when an individual in the Netherlands became infected by eating lightly salted herring [ref. 6]. Since that time, a great deal has been learned about parasite biology, about the frequency and severity of human infections, and about the steps required for the prevention of human infections.

**PARASITES OF CONCERN—IDENTIFICATION AND BIOLOGY**

As indicated, marine fishes host a vast array of parasitic organisms. Occasionally these parasites cause pathological effects on fish hosts, but more often parasites in moderate numbers are accommodated by marine fish hosts without obvious effect. Parasites of concern from a human health standpoint are worm, or helminth, parasites and belong to the roundworm group known as nematodes or to the tapeworm group of flatworms known as cestodes. A third group, the trematodes, are flatworms related to cestodes that are commonly found in marine fishes and which may infect humans, but there is little documentation of this possibility.

All of these parasitic helminths have complicated, indirect life cycles. They do not spread directly from fish to fish, but rather must pass through one or more intermediate hosts and in some cases one or more paratenic or transport hosts. Many nematodes and cestodes have life cycles that involve crustacean first-intermediate hosts, marine fish second-intermediate hosts, and marine vertebrate final hosts in which the sexually mature adult worms are found. Trematode cycles are even more complicated, with an asexually multiplying stage in a molluscan first-intermediate host, a fish or invertebrate second-intermediate host, and a marine vertebrate final host. In all three groups, the possibility of human infection is raised when larval stages of the parasite lodged in fish intermediate or paratenic hosts are ingested in fish flesh that has not been cooked or frozen to kill the parasites.

*Anisakid nematodes*

Anisakid nematodes exhibit typical nematode morphology. They are cylindrical, elongate worms tapering at each end. A body cavity
(pseudocoel) is present and the worms are covered by a secreted, resistant cuticle that is shed when the animal molts. Like most parasitic nematodes, anisakids found in fish are easily visible with the unaided eye.

Within the family Anisakidae, species with the greatest potential for human infection are those that use fishes as either intermediate or paratenic hosts and warm-blooded vertebrates, usually marine mammals, as final hosts. In the marine environment these include *Anisakis simplex*, *Pseudoterranova (=Phocanema = Porrocaecum = Terranova) decipiens*, and *Phocascarisi/Contraecaecum* spp. [refs. 7-9]. These species probably all use crustacean intermediate hosts and occur in fish as larvae that can infect the final host.

A fourth species, *Hysterothylacium aduncum*, has a similar life cycle except that fishes host both larval and adult nematodes [ref. 10]. This species normally matures in a cold-blooded vertebrate, suggesting that they may be less of a hazard for humans, although at least one study has shown that a larval *Hysterothylacium* caused lesions when experimentally administered to rhesus monkeys [ref. 11].

It is unclear how many species of anisakid nematode larvae occur in marine fishes [ref. 3], because the morphological characteristics that distinguish them are often subtle. The following information, provided by Smith and Wootten [refs. 7-9], is helpful in recognizing three of the most commonly encountered species. *Anisakis simplex* larvae (often referred to as *Anisakis* larva type 1) are from 9 to 36 mm long, off-white, and encysted in capsules of host origin, coiled like a watch spring. In the fish host, *A. simplex* is found in the body cavity, on or in the viscer, and in musculature. The proportion of worms located in flesh is low in some host species (herring) and high in others (gadoid fishes). Larvae of *P. decipiens* in fish are from 9 to 58 mm long, vary in color (creamy white, yellow, brown, or reddish brown) and lie coiled irregularly in capsules of host origin. According to Margolis [ref. 2], this species is usually located in fish musculature, but may occur in the body cavity. Larvae of *Phocascarisi/Contraecaecum* are indistinguishable and may be synonymous. They are between 7 and 30 mm long, greenish brown (sometimes tinged with red), and occur coiled in capsules of irregular shape, often in the liver and only rarely in the musculatures of fish hosts.

Like all nematodes, anisakids have five growth stages separated by four molts. The first four stages are larval and are designated L1, L2, L3, and L4. The fifth stage is the sexually mature adult.

The life history of anisakid nematodes is most intensively studied and best known for *A. simplex*. In spite of this, the number of potential hosts has complicated studies, preventing a complete understanding of parasite population dynamics according to Smith [ref. 12], who has provided the most comprehensive ecological picture to date. Smith concludes that eggs are passed from the digestive tract of vertebrate final hosts into seawater where the developing larva molts once before hatching as a second stage larva (L2) that is sheathed in the cuticle of the L1. This sheathed larva is
ingested by a crustacean (usually a euphausiid in the case of A. simplex) where it molts to the L3 stage that is infective for the final host. Whales that feed primarily on euphausiids are known to be heavily infected with A. simplex. When fish or squid feed on infected euphausiids, the nematodes penetrate the digestive tract and migrate to viscera or musculature where they are encapsulated by the host. They do not molt in the fish or squid host, rather they remain in the third larval stage (L3) that is infective for the cetacean host. Usually, toothed whales that feed upon fish and squid serve as hosts for A. simplex larvae that occur in those paratenic hosts.

Life cycles of other anisakids in the marine environment are less well known, but it is likely that they are similar to that of A. simplex. The life history of P. decipiens has been summarized by Margolis and by Smith and Wootton [refs. 2,8]. Adult worms occur primarily in seals, sea lions, and walruses. Second stage larvae (L2) hatch from eggs and become L3 when eaten by small crustaceans. Fishes serve as paratenic hosts and as such facilitate the life cycle ecologically if not physiologically. Pseudoterranova decipiens larvae routinely locate in muscle of the fish host and molt to L4 and adult in the pinniped stomach.

Substantially less is known about other marine anisakid species. Phocascaris/Contraeaeum spp. probably have life cycles similar to those already described, occur in seals as adults, and usually locate in fish liver rather than muscle [ref. 9]. The anisakid genus Hysterothylacium contains species that mature in marine fishes and most are not thought to infect warm-blooded animals. Nevertheless, they occur commonly in marine fishes as L3. They are very difficult to distinguish from species that are potential human health hazards, and include species (Hysterothylacium type MB larvae) that have been shown to penetrate the stomach of primates in spite of the fact that they normally become adult worms in the digestive tract of Gulf of Mexico fishes [ref. 12].

It is difficult to give a precise summary of the larval anisakid infection levels that occur in marine fishes due to the vast number of reports in the literature, to the uncertainties of dealing with a group of parasites that is in a state of taxonomic flux, and to the difficulty of identifying specimens. In general, it appears that specificity for the fish host on the part of anisakid larvae is low or absent and that virtually any fish that ingests either infected crustaceans or other infected fishes will become infected with the larval nematodes. This means that the factors dictating which fish species serve as host and the levels of infection that occur are ecological rather than physiological. Examples of these types of factors are proximity to areas where marine mammal final hosts concentrate, and the food habits of the fish.

The common occurrence of anisakid larvae in marine fishes that do not typically feed on zooplankton suggests that these species acquire infections when preying upon plankton-feeding fishes that are already infected with the larvae. Reports that indicate the wide range of fish species that serve as hosts for larval anisakid nematodes include: 34 fish hosts of Pseudo-
and 53 of Anisakis in Canadian marine waters [ref. 13]; 123 species of fish hosts and one squid host of Anisakis type 1 larvae in Japanese waters [ref. 1]; 57 fish host species of Anisakis larvae in New Zealand marine waters [ref. 14]; and Myers' report [ref. 15] that 38 of 42 species caught off the United States Pacific Coast were hosts of anisakids and that each of the four negative species were represented in the sample by only a single individual. Anisakids are also common in Alaskan marine fishes [refs. 16, 17] and in fishes caught in the vicinity of the Hawaiian Islands [ref. 18]. Rhode [ref. 19] provides further references documenting the worldwide occurrence of anisakid nematode larvae in marine fishes.

Numerous studies have provided a picture of the variations in the intensity of infection that often occur among individual fish within a certain geographic area. In these studies, prevalence and abundance of worms have been associated with host age as reflected by length [refs. 20-22].

The exhaustive study by Arthur et al. [ref. 20] dealt primarily with tissue parasites of walleye pollock (Theragra chalcogramma) and results give a comprehensive view of the economic parasitology of this commercial fish species. The authors found four species of anisakid nematodes parasitizing walleye pollock from two sampling areas, and three of the four were the potential human pathogens already discussed. The occurrence and abundance of these parasites varied with geographic location and with fish length (age). The increased infection levels with fish age reflects the long life span of larval anisakid nematodes, which possibly live as long as the fish host. Infection prevalences in pollock were highest for A. simplex (99 percent at one sampling site) and infection intensities ranged from 2 to 269 worms per host. P. decipiens was least common (6.1 and 15.0 percent prevalences in two fish stocks) and lowest in intensity (1 to 8 worms per host). Phocascaris/Contraearcem and Hysterothyacum aduncum were found at intermediate infection levels.

The tendency of A. simplex to become encapsulated in viscera of the body cavity rather than in musculature was also demonstrated by Arthur et al. [ref. 20]. They reported worms in the flesh of 24 percent of the walleye pollock that were infected with A. simplex and that these were usually few in number compared with the number occupying the body cavity. In contrast, these authors found P. decipiens only in the musculature, where the worms were distributed randomly, unlike A. simplex which occurred primarily in the body cavity. The distribution of A. simplex larvae within the fish host may be related to the feeding habits of the fish. Smith [ref. 23] has recently shown that most encapsulated larvae occur in the body cavity of zooplankton-feeding fishes such as herring (Clupea harengus), but are more widely distributed throughout the tissues of piscivorous fishes such as cod (Gadus morhua). Smith [ref. 12] suggests that the zooplankton (primarily euphausiids) feeders are probably on the "mainstream" of the A. simplex life cycle involving a transfer of worms upward from euphausiid to euphausiid-feeding fish to cetaceans. Piscivorous
fishes probably acquire most of their *A. simplex* from prey fishes and may not be an efficient mechanism of transfer to final hosts [ref. 23].

In summary, marine fishes are very commonly infected with anisakid nematodes that may occur in the fish flesh. Although not all have the same potential to be human pathogens, gross morphological similarities prevent ready separation of species macroscopically. From a practical standpoint, all should be considered potentially harmful if ingested alive. Infection levels in fish depend upon the availability of infective stages (which depends on mammalian final hosts and crustacean intermediate hosts), upon food habits of the fish, and upon the age of the fish host. These parasites do not multiply in the fish host; each worm is the result of ingesting an individual larva. Physiological host specificity for the fish host has not been demonstrated and ecological factors are the primary determinants of infection levels.

*Diphyllobothriid cestodes*

Cestodes, or tapeworms, are flatworms characterized morphologically by the presence of an anterior attachment structure, termed a scolex, and a body consisting of a chain of segments, termed proglottids. Tapeworms acquired by humans when ingesting raw fish are in the taxonomic order Pseudophyllidea and the genus *Diphyllobothrium* and have life cycles involving crustacean (usually copepod) first-intermediate hosts, fish second-intermediate hosts, and mammal or bird final hosts. Crustacean first-intermediate hosts become infected when they feed on a ciliated larva called a coracidium that emerges from the *Diphyllobothrium* egg in water. The coracidium burrows through the wall of the crustacean digestive tract and develops into a proceroid larva in the hemocoel. No further development takes place until the crustacean is eaten by a fish, where the second tapeworm larval stage, the pleuroceroid, develops in the tissue. When the appropriate warm-blooded host eats the fish, the pleuroceroid larva develops into a sexually mature adult tapeworm in the intestine.

It is probable that most diphyllobothriids that infect man have freshwater cycles involving freshwater fishes. However, the marine environment serves as habitat for anadromous salmonid fishes that may carry freshwater-acquired pleuroceroids of *Diphyllobothrium* for their entire lives. It is in this marine context that the subject is considered here.

The taxonomy of diphyllobothriid cestodes is difficult, and identifying these worms requires an expert [ref. 24]. The species that usually infects humans is *D. latum*. Called the broad fish tapeworm, it is common in a variety of fish-eating animals throughout the more northerly portions of the northern hemisphere, although in western Alaska the only important final hosts are humans [ref. 25]. Humans become infected when they eat raw or undercooked fish containing pleuroceroids that are usually found unencysted and coiled in the musculature, which may occur encysted in viscera. Infective pleuroceroids vary in length from a few millimeters to
several centimeters and have few morphological features that allow species identifications.

Another species of *Diphyllobothrium* that has infected humans in Alaska and Canada is *D. ursi* [refs. 25, 26], a common parasite of bears. The pleurocercoid of this species does not occur in musculature, but only in small cysts on fish viscera, especially sockeye salmon (*Oncorhynchus nerka*). Adult *Diphyllobothrium* spp. in humans are large worms reaching a length of 30 ft, consisting of several thousand proglottids and releasing vast numbers of eggs in feces each day.

Reports of *D. latum* pleurocercoid infection levels in fish intermediate hosts are sparse due to the difficulty of identifying the larvae species. For example, diphyllobothriid pleurocercoid prevalences of 66 percent in Alaska sockeye salmon smolts and 13 percent in adults have been observed [ref. 27], but the larvae species was not determined. Although infection levels in fish were not known, an outbreak of diphyllobothriasis in the United States in 1980 was associated with an unusually large harvest of sockeye salmon that resulted in widespread shipment of fresh salmon to fish markets [ref. 28].

Diphyllobothriasis in Japan was once common, but is now believed to be rare [ref. 29].

*Other parasites acquired when eating raw fish*

Like cestodes, trematodes are flatworms in the phylum Platyhelminthes. *Nanophyetus salmincola*, the so-called salmon poisoning fluke, is a trematode of fish-eating carnivores in eastern Russia and the northwestern United States. The parasite has long been known to infect people in Siberia [ref. 30] and has been reported to cause human infections in the United States [ref. 31]. Salmon are among the most commonly infected fishes and metacercariae remain viable through the marine phase of the salmon life cycle. Since metacercariae locate in virtually any fish tissue, ingestion of raw salmon flesh could lead to infection. The enzootic area in North America is defined by the distribution of the snail host and includes western Washington, Oregon, and northern California [ref. 32]. In humans, these infections may be asymptomatic or may cause abdominal discomfort and diarrhea and are readily treated with praziquantel [ref. 31].

The remaining parasites that are acquired from fish and that are known to cause human infections are primarily trematodes involving freshwater fishes in Asia, including *Clonorchis sinensis*, *Heterophyes heterophyes*, and *Metagonimus yokogawai* [ref. 24] and are beyond the scope of this review.

**PARASITES COMMON IN FLESH OF MARINE FISHES, BUT NOT OF PUBLIC HEALTH CONCERN**

One aspect of macroscopic parasites in marine fishes, other than the human health concern, is aesthetic. Although this topic is also beyond the scope of this review, brief mention will be made of commonly encountered
parasites that may affect the quality of marine fish flesh, but not safety from a public health standpoint.

Trypanorhynchn  **cestodes**

Trypanorhynchn tapeworms occur as adult worms only in the spiral intestine of elasmobranchs (sharks, skates, and rays), but use as transport hosts virtually any marine teleost that ingests crustacean intermediate hosts. In marine fish, these macroscopic (several mm) whitish larval worms bearing four spiny tentacles locate in the body cavity, digestive tract wall, and in musculature. They have never been reported to infect humans [ref. 24], but are very common as larvae in many species of marine fishes [refs. 23,17,20].

**Myxosporidian protozoans**

Although myxosporidians are classified as protozoan parasites and have characteristic spores that can only be seen under a microscope, they often occur in fish tissue within cysts that are macroscopic and readily visible. Members of this group are not known to infect humans or any other warm-blooded animal, so they do not constitute a public health hazard. However, several species of myxosporidians are of major economic importance because they cause large, unsightly cysts that may render fish flesh unmarketable. Species that are examples of this include *Henneguya salminicola* in salmon and *Unicapsula muscularis* in halibut (*Hippoglossus stenolepis*) [refs. 33,34]. Other species locate in smaller, less noticeable cysts, but are associated with an enzymatic breakdown of the fish muscle that prevents the marketing of affected individuals. This problem has had a major impact on use of Pacific hake (*Merluccius productus*) and other fish species that are infected with myxosporidians in the genus *Kudoa*. This situation has been extensively studied [refs. 35-37] but many questions remain.

Myxosporidians may be common in some fish stocks and rare in others. For example, the prevalence of *H. salminicola* infection reaches 67 percent in certain British Columbian sockeye salmon stocks while in other stocks and salmon species the parasite is less common or absent [ref. 33]. In Oregon, an infection prevalence in coho salmon (*O. kisutch*) of 2.5 percent has been reported [ref. 38].

**Microsporidian protozoans**

A final parasite group that occurs in the flesh of commercially important marine fishes is the Microspora. In fish, these protozoans usually occur within whitish, elongate, cyst-like structures termed xenomas that may be macroscopic. These xenomas contain vast numbers of very small microscopic spores that transmit the parasite to other fish when the spores are released into the water, presumably after the infected fish dies. The microsporidians that parasitize fishes do not infect humans or other warm-blooded vertebrates. These parasites usually locate in tissue other than
muscule, but an undescribed species of *Pleistophora* sometimes occurs in high prevalence and intensity in walleye pollock in the northwestern Pacific [ref. 20] and in Pacific cod (*Gadus macrocephalus*) near Kodiak Island, Alaska [ref. 17].

**HISTORY, PATHOLOGY, DIAGNOSIS, AND TREATMENT OF NEMATODE AND CESTODE INFECTIONS ACQUIRED BY EATING MARINE FISHES**

**Nematodes**

As discussed in an earlier section, the nematodes occurring in marine fishes that have demonstrated potential human health hazards are members of the family Anisakidae. Most reports of human infection have implicated two species of anisakis, *Anisakis simplex* and *Pseudoterranova decipiens*. Both of these species have been extensively reviewed, *A. simplex* by Oshima [ref. 1] and by Smith and Wootten [ref. 3], and *P. decipiens* by Margolis [ref. 2]. *Anisakis simplex* has been termed "herringworm" and *P. decipiens" codworm" to reflect hosts in which they commonly occur, but both also occur in a great variety of other marine fish species.

Jackson [ref. 5], Myers [ref. 4], and Smith and Wootten [ref. 3] have provided a history of knowledge concerning anisakid nematodes as human pathogens. The first observed human case apparently occurred in 1876 and involved an "ascarid" now believed to have been *P. decipiens*, which was vomited by a child in Greenland. In succeeding years, many larval anisakis were described from fish hosts and eventually concern for aesthetic problems caused by *P. decipiens* in the 1950s led to efforts to control the parasite. In the Gulf of St. Lawrence these efforts involved fishing in areas of low parasite prevalence and reducing the seal (final host) population. These efforts were unsuccessful because of the size and movements of the seal population.

Difficulty of diagnosis probably prevented recognition of the human anisakid problem. The first case of severe anisakiasis was not detected until 1955 in the Netherlands and was not reported in the literature until 1960 [ref. 6]. The first cases of anisakiasis in Japan were reported in 1965 [ref. 39]. These cases, like those with similar clinical characteristics observed previously, might have been attributed to other parasitic nematodes if the report of Van Thiel, Kuipers, and Roskam [ref. 6] had not appeared in the scientific literature. After these reports of human anisakid infection were published, physicians became aware of possible anisakid involvement if raw fish had been eaten by patients complaining of gastrointestinal pain and many more cases were recorded, including more than 1,000 cases in Japan by 1976 [ref. 4].

In North America, the first observations of anisakid nematodes in humans were asymptomatic, noticed only because worms were coughed up or vomited, and were not recognized as infections [refs. 40-42]. The first case of intestinal anisakiasis in North America involved a woman who regularly prepared a salted salmon recipe, sampling the fish periodically
during the salting process. Intestinal pain led to an operation in which an anisakid nematode was discovered in an intestinal lesion [ref. 43]. By 1982, 23 cases of anisakiasis had been reported from North America, 11 of these in California and five in Alaska [ref. 44]. By 1986 the total number of cases reported in North America remained less than 30 [ref. 45].

The typical human infections caused by *A. simplex* and *P. decipiens* differ, a fact that has caused some confusion when clinical characteristics of anisakiasis are described. The clinical characteristics of anisakiasis caused by larvae in the genus *Anisakis* have been described and reviewed by numerous authors [refs. 1,3,43,46-48]. Anisakiasis caused by *Pseudoterranova* larvae has also been described [refs. 44,46] and reviewed [ref. 2]. The following summary is based upon those reports and reviews.

Intestinal anisakiasis is caused only by larval *Anisakis* while gastric anisakiasis may be due to either *Anisakis* or *Pseudoterranova*. Gastric infections result in sudden stomach pains, nausea, and vomiting 4-6 hr after ingesting seafood containing viable larvae. Gastric anisakiasis has been confused with other stomach disorders such as peptic ulcers, neoplasia, gastritis, and food poisoning. After the initial episode, the condition may recur over a period of several years. Gastric anisakiasis is most often reported from Japan where it usually involves *A. simplex*. The first United States cases of gastric anisakiasis involving tissue penetration and caused by *A. simplex* were recently reported in Hawaii. Patients apparently acquired infections from fresh Pacific Northwest salmon, locally caught tuna, and imported squid [refs. 49, 50]. A common observation in *Pseudoterranova*-caused gastric anisakiasis is for the worms to be coughed up and expelled after varying lengths of time following infection. These cases are often asymptomatic and may involve larvae that have not penetrated into tissue. Gastric anisakiasis may cause eosinophilia, but leucocytosis is not characteristic.

Intestinal anisakiasis is characterized by severe abdominal pain that may occur between 10 hr and several weeks after eating infected fish. The symptoms may mimic acute appendicitis, neoplasia, regional enteritis, or food poisoning. Nausea, vomiting, fever, diarrhea, and occult blood in stools are typical with eosinophilia absent and leucocytosis present. Pathogenesis is not well understood and a number of ideas concerning the development of pathological conditions have been proposed. These deal with the extent of tissue penetration by larvae and the possibility that hypersensitivity reactions based on repeated infections may lead to the development of extensive lesions and the persistence of symptoms long after the worms have died.

Anisakiasis is often difficult to diagnose due to ambiguous symptoms, infrequent occurrence (except in Japan), and because worms do not mature to produce eggs that could be detected in stools. When gastric anisakiasis is probable, endoscopy and worm removal with a gastrofiberscopic biopsy forceps allows diagnosis. X-ray analysis is not definitive, but may be highly suggestive of anisakiasis [refs. 48,51]. Acute intestinal anisakiasis cannot
be diagnosed by endoscopy, and surgical techniques have often been employed for diagnosis and treatment [ref. 53]. Serological techniques have been used to detect both active and resolved cases of anisakiasis [refs. 49, 53]. A probable case of pulmonary anisakiasis has been diagnosed in a patient who developed gastric symptoms soon after eating raw salmon and who subsequently developed a cough and thoracic pain. X-rays revealed pleural effusion, and immunoelectrophoresis resulted in a band of identity with *Anisakis* antigen [ref. 54]. In general, the development of serological diagnostic techniques for anisakiasis has been hindered by its cross-reactivity with other nematode parasites [ref. 3].

Anthelminthic drug treatment of humans with anisakiasis has apparently not been attempted, but some success with thiabendazole and piperazine in treating marine mammals has been reported [ref. 53,55]. Cases of spontaneous cure have been noted with anisakiasis caused by *Pseudoterranova* and larvae are believed to have a short life span in the human body [ref. 2].

**Cestodes**

*Diphyllobothrium latum* has a long history as a human pathogen, especially in northern Europe [ref. 50], but it is also common in northern North Americans who eat raw fish [ref. 57]. Although human infections may be characterized by abdominal pain, diarrhea, constipation, and occasional megaloblastic anemia caused by absorption of vitamin B<sub>12</sub> by the parasite, most cases are asymptomatic [ref. 24]. Demonstration of characteristic eggs or proglottids passed with feces allows diagnosis of the genus *Diphyllobothrium*, but not of the species that can only be determined by examination of the scolex [ref. 48].

Other species of *Diphyllobothrium*, including *D. chordatum* and *D. pacificum* are marine, normally infect seals and sea lions [refs. 24,58], would be expected to use marine fishes as intermediate hosts, and could potentially infect humans.

Treatment of diphyllobothriasis is by administration of the anthelminthic drugs nicosamide or praziquantel [ref. 24].

**Prevention of Infection**

Efforts to detect and remove anisakid nematodes from fish products predate recognition of the potential public health hazard. These efforts have increased in recent years, but no completely effective method has been found. Many fish processors candle fish fillets over an illuminated screen to locate worms. This method is fairly effective in locating the larger and more highly pigmented *Pseudoterranova* larvae, but it is only of limited value in detecting the colorless larvae of *A. simplex* [ref. 3].

The fact that anisakid larvae migrate from fish host viscera to muscle under certain circumstances was a factor leading to the discovery of the extent of the human health hazard. In the Netherlands after World War II,
lightly salted herring (green herring, 4 percent NaCl) was increasingly consumed. Most often the herring were processed aboard the boat, but with increased demand the gutting and salting were done ashore [ref. 4]. This allowed anisakids to move from the viscera to the musculature during storage of iced fish. Because the salting did not kill the larvae, human infections resulted [ref. 6]. In a recent study, Smith [ref. 23] has documented the conditions under which anisakid nematodes migrate from viscera to muscle in certain species and has reviewed the literature on the subject. Based upon his and other studies, Smith points out that A. simplex migrates from viscera to flesh during storage of ungutted fish on ice in herring and mackerel (Scomber scomber), but does not migrate to flesh under similar conditions in walleye pollock, whiting (Merlangus merlangus), and blue whiting (Micromesistius poutassou). Although the reasons for this are not clear, Smith suggests that it relates to the fact that herring and mackerel are "fatty" species with lipid concentrated in the flesh and the gadiform species are "non-fatty" with lipids stored mainly in the liver and mesenteries. He speculates that the stimulus for worms to leave capsules and migrate might be associated with the presence of adjacent "fatty" muscle tissue.

The relative proportion of A. simplex larvae in the fish body cavity and in the musculature varies with the host species, which can be divided into two groups. Pelagic species that feed on euphausiids (the most important intermediate hosts of A. simplex) are infected primarily in the body cavity. Fishes in which a high proportion of nematode larvae occur in the flesh tend to prey on other fish, and most of the larvae they contain were likely to have been acquired from prey fishes that had fed on infected euphausiids [ref. 23]. In any event, most of the larval A. simplex that occur in fish flesh are located within the hypaxial muscles surrounding the body cavity (belly flaps) and can be trimmed by processors so that most worms are removed [ref. 3].

Making fish processors aware that certain fish species are more likely to be infected than others, and that anisakids tend to migrate into musculature in some host species after fish are caught, can help reduce anisakid infection in fish products. Scientists continue to search for ways to detect parasites found within the musculature of processed fish without destroying tissue. Some experimentation has been done using ultrasonic sound, but a practical technique has not yet been developed [refs. 59,60]. Recently, biochemical techniques have shown that adenosine triphosphate activity when stimulated by Mg\(^{2+}\) was greater in muscle mitochondria of fish infected with larval A. simplex than in uninfected fish. The activity in infected fish was also dose-dependent, increasing with the number of worms per fish [ref. 61]. In spite of this progress, practical methods for detecting and removing all parasites in processed fish flesh are not yet available. Fish products should be treated to kill all worm larvae before human consumption.
Anisakid larvae have been reported to die at temperatures about 60°C [ref. 3], a temperature normally attained during cooking. Although some cold-smoking procedures do not reach temperatures hot enough to kill anisakid nematode larvae [ref. 62], hot-smoked fish should be brought to at least 82°C to prevent growth of food-poisoning bacteria [ref. 63]. If this is done, all parasites will be killed.

When raw or lightly marinated fish are prepared, it is best to freeze the fish first. Several researchers have reported that anisakid larvae are killed if held between -17°C and -20°C for 24 hr [refs. 3,64]. In the Netherlands, regulations requiring freezing have been developed to prevent anisakiasis due to consumption of lightly salted herring [ref. 65]. According to Myers [ref. 4], fish should be held at -20°C for 60 hr to ensure that a lethal temperature is reached throughout the entire fish product. According to the review of Smith and Wootten [ref. 3], anisakid larvae are resistant to most curing and marinating and to a wide variety of chemicals, spices, and food additives. For these reasons, only deep-frozen fish should be used in recipes for various raw fish dishes.

Methods that kill anisakid nematodes will also kill larval diphyllobothriid cestodes [ref. 28], so both tapeworm and nematode infections are prevented by the same safety measures.

CONCLUSIONS

Consuming fish has few negative aspects; however, it is impossible to avoid dealing with the parasitic organisms that fishes acquire during the normal course of their lives. The majority of these parasites are not infective to humans or to other homeotherms. However, as has been discussed, several commonly occurring parasites can cause infections in humans.

Although human infections with diphyllobothriid tapeworms have long been recognized, they are primarily associated with freshwater fishes. Reports of infections obtained from salmon caught during the marine phase of their life cycle have been increasingly recognized only in recent years. The more common problem involves anisakid nematodes that normally infect marine mammals. Infections in humans have been routinely diagnosed for only the past 25 to 30 years.

The extent of the public health problem depends upon biological, geographic, and cultural factors. Biological factors include the health of marine mammal populations and the accompanying efficiency with which parasite life cycles operate. It would be expected that high mammal host populations would be reflected in increased incidence and intensity of larval stages in fishes. Biological factors also include preferred food habits of the fish hosts. Fishes feeding heavily on parasite intermediate hosts (for example, herring) will probably carry heavier infections than will fishes that acquire infections at higher levels in the food web and only occasionally feed on prey carrying transferable parasites. Geographic factors are related
to biological ones and involve proximity of the harvested fishes to areas where marine mammal populations are high, and on the part of the human population to areas where fresh fish is routinely available. Cultural factors are more important than the other two. The majority of human anisakiasis cases have occurred in Japan where fish consumption per capita is very high and where raw fish preparations are common. Although adequate freezing or cooking prevents human infection by worm parasites that occur in fish flesh, traditional practices of marketing, storing, and preparing fish products have great inertia. Complete elimination of the human health hazard may be difficult to attain. In areas where raw fish consumption is not widespread or culturally ingrained, such as North America and Europe, documented cases of anisakiasis are rare and considered accidental parasitic infections that are readily manageable [refs. 45,66,67].

ACKNOWLEDGEMENTS

Appreciation is extended to Pam Rogers for typing the manuscript and to Marilyn Guin for assistance with the literature. This report was sponsored by the Oregon State University Sea Grant College Program, supported by NOAA Office of Sea Grant, U.S. Department of Commerce under Grant Number NA85-AA-D-5G095.

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