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Nematodes Transmitted to Man by Fish and Aquatic Mammals

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Abstract

Zoonotic nematodes may cause disease in man through migrating larva (larva migrans), through direct infection or possibly through allergic responses. The parasitic genera *Ancylostoma, Uncinaria, Bunostomum* and *Toxocara* can cause larva migrans. The cod worm *(Phocanema decipiens)* a parasite found in fish and seals, can infect man, as can *Anisakis, Dioctophyme renale* and *Gnathostoma hispidium* larvae obtained from eating raw fish.

Trichinella spiralis occurs in marine mammals.

Man serves as host to over 200 species of parasites (Cameron[4]). Of the parasites infecting man there are a few which he acquired in the process of human evolution; others he acquired from domestic animals and wild animals, and some he is still acquiring.[5] At present the known zoonotic nematodes man has acquired from fish, and marine and fresh water vertebrates can be grouped into two categories (1) larval parasites of animals which infect man and can often cause serious disease *(e.g.,* "larva migrans") and (2) accidental parasites of man of which the natural final host is another animal. In time others could be added to these two categories.

Among the parasitic nematodes of man are those which show a normal larval migration through the body before reaching an adult stage in the stomach, intestine or body tissue *(e.g., Ascaris, Strongyloides,* hookworms, filarids, etc.*), and still others in which man is the accidental rather than the natural host. In most of the latter cases the larval stages are able to survive for varying periods but may not develop to maturity**. These larvae usually produce a series of obscure host reactions which pass unnoticed or are regarded either as symptoms of disease of unknown etiology or wrongly assigned to other diseases. A parasite etiology becomes clear only when one can demonstrate the presence of these nematodes and host damage due to migration.

The term "larva migrans" is used to describe the disease caused by migrating larvae of nematodes which occur in hosts other than man. Larva migrans has been further classified as "cutaneous larva migrans" or "visceral larva migrans".

In man "cutaneous larva migrans" is caused by larvae of the dog and cat hookworms *Ancylostoma braziliense, A. caninum, Uncinaria stenocephala* and

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* In parasites for which man is reported to be the natural host, an abnormality in larval migration sometimes causes symptomatology, *e.g., Ascaris,* etc.

** *Toxocara, Anisakis, Gnathostoma,* etc.

Bunostomum phlebotomum of cattle. The natural parasites of humans *Ancylostoma duodenale, Necator americanus* and *Strongyloides sterocoralis* produce a transient cutaneous larva migrans.

"Visceral larva migrans" is perhaps best known in the infections of *Toxocara canis* (which is a natural parasite of the small intestine of dogs) and *T. cati* (which is a natural parasite of cats). These larvae have been found in man on autopsy in the lungs, liver, brain, eye and other organs. These larvae migrate extensively but ultimately become encapsulated. During this migration they produce a high degree of eosinophilia and may contribute to general poor health. Acute symptomatic situations usually occur in children in which case histories involve dirt eating or contact with infected dogs (Beaver[2]).

Sometimes "visceral larva migrans" follows "cutaneous larva migrans" both being produced by the same parasite, *e.g.*, hookworms. In some cases of human gnathostomiasis these two syndromes often replace each other.

There are no definite clinical signs which could alone assure the diagnosis of "visceral larva migrans". Biopsy may reveal the presence of larvae in tissue; however, at times host reaction may obscure the recognition of the parasite.

The presence of a parasitic infection in either an accidental or a natural host involves an interaction of factors depending on the species of parasite and its life cycle, as well as the biology and ecology of the host or hosts.

Many environmental factors limit the distribution of a parasitic infection, including geographic distribution of the natural host, or intermediate and/or transport host. In case of human infection perhaps the most important of all are the social customs and habits of the people. For example the high incidence of *Opisthorchis* (a trematode found in the liver) infection in certain areas of Japan, South Korea, Southern China and Indo-China; of *Heterophyes* (a trematode of the intestine) in the coastal regions of Egypt and parts of the Far East and *Diphyllobothrium* (a cestode of the intestine) in North America and Europe, are all attributed to the eating of raw or undercooked fish. A classic example is the absence of *Taenia solium* of man in Moslem countries where abstention is practiced in the consumption of pork.

Differences in diet and food preparation may be associated with the presence or absence of an infection, even though the parasite may be included in the food consumed by the population.

With the growing awareness of providing food for the increased human population, in recent years more attention has been directed to the utilization of invertebrates and vertebrates from salt and fresh water. The presence of parasites in the fish consumed can materially reduce the commercial value as well as be a threat to human health, especially in regions where fish and other vertebrates are consumed raw or undercooked.

Ascarid nematodes are large fleshy forms which occur in the intestine of a great variety of animals. The best known is *Ascaris lumbricoides* of man and pig. The group is a large one and is divided into several families and subfamilies of which the Anisakidae are found mainly in fish-eating vertebrates, *i.e.*, mammals, birds, and fish. The larval stages of certain genera occur commonly in the flesh and viscera of marine fish and have attracted greater attention since their presence lowers the marketability of the fish for human consumption.

During the 1950's extensive studies were conducted in the eastern maritimes of Canada under the auspices of the Fisheries Research Board of Canada on the "cod worm" [*Phocanema decipiens* (Krabbe, 1878) Myers, 1959]. The larval stage occurs in many species of ground fish and the definitive host, the seal, is infected by feeding on infected fish. These larvae which are yellow, in contrast to the white flesh of the fish fillet, are easily detected with the naked eye.

The life cycle of *Phocanema* is unknown but, presumably, it could utilize a crustacean as the first intermediate host and a series of fish as transport hosts (a host in which the larvae remain alive but undergo no further development) before reaching the seal host. Larvae from the fish fed to seals will develop to adults in the stomach in 21 days after infection (Myers[20]). *Phocanema*-type larvae are common in cod, smelt, hake, plaice, flounder, etc., in the Northern Atlantic. The fish which are predominantly fish feeders and which are older are more heavily infected (Templeman *et al.*[26]). Yamaguti[22] reported 18 species of Pacific fish to be infected and Kahl[11,12] reported smelt, redfish, cod and other species of fish to be infected in the North Sea.

Buckley[3] reported finding an immature form of *Porrocaecum* [*Phocanema*-type larva (Myers, 1959)] in the mouth of a man and suggested it had come from a piece of fish eaten by the patient. Hitchcock[10] in a fecal study of Eskimos from the Bethel Area of Alaska found nematodes which were identified as immature forms of *Anisakis* and probably *Porrocaecum* in 10% of the stools examined.

The first human infection of *Porrocaecum* was reported by Chitwood.[6]

Another genus, *Anisakis,* reaches its adult stage in marine mammals and elasmobranchs and like other nematodes which live in sea animals requires an intermediate host to carry the larvae to the final host. Like *Phocanema,* the complete life cycle is unknown, but the larval stage occurs in the viscera and flesh of herring, salmon, tuna and other fish. It has a cosmopolitan distribution. If the fish containing the larvae are eaten raw the larvae could be a potential threat. Studies in guinea pigs have shown the larvae could penetrate the stomach wall and be recovered from the body cavity (Myers).[21] Human infections were first brought to attention by Van Theil *et al.*[28] who reported this larva as *Eustoma* sp. from man. Eleven cases were reported from Holland, and two deaths were attributed to the infection. A study of the case histories revealed that all patients had ingested "green" herring, a lightly salted raw herring which is commonly consumed raw in the Netherlands. Over 100 cases have been reported from Japan (Yokogawa and Yoshimura[23]), where infection is acquired by eating either raw or pickled fish. The majority of the Japanese cases involved the stomach, whereas, those in the Netherlands were intestinal (Van Thiel and Van Houten[27]). The clinical symptoms varied with the location of the larvae. Symptoms of gastric complaint in the Japanese cases varied from one to several months and cases were frequently diagnosed as gastric tumor. The cases involving the intestines were of shorter duration and involved surgery. Ashby *et al.*,[1] in reviewing case histories of eosinophilic granulomas of the gastro-intestinal tract, concluded that many of these granulomas could be attributed to *Anisakis* larvae. Emphasis should be placed on the demonstration of the larvae in the tissue for confirmed diagnosis. Many times, identification is difficult, if not impossible, because only a section of the larva is present which may not show the morphological characteristics necessary for the specific identification of the nematode.

Kuipers[16] and Kikuchi *et al.*[15] have suggested that the gastro-intestinal eosinophilic phenomena and histopathology indicate an allergic etiology. The first larval infection may not bring about a clinical picture but sensitizes the individual against future infections, and the subsequent infection may cause a severe allergic reaction.

Gnathostomiasis in man is caused by the larval stage of the spirurid nematode, *Gnathostoma spinigerum* Owen, 1836, the adult of which is found in tumors in the stomach of domestic and wild felidae, raccoons and dogs in the warmer regions of the world. It is an especially important parasite of man in Thailand and Japan. Human infections have been reported from Thailand, Malaya, Indo-China, Japan, China, Indonesia and India (Miyazaki[19]). Another species, *Gnathostoma hispidium* Fedtschencko, 1872, is found in wild and domestic hogs in Asia. Human infections have been reported once from Japan and China, and twice from India (Faust *et al.*[8]).

In the natural host the adult worm lives embedded in a nodule in the wall of the stomach. The life cycle involves two intermediate hosts and frequently addi-

tional facultative hosts. The first host is a *Cyclops* which ingests the larva and the second host is usually a fish, but may be other cold blooded vertebrates in which it reaches the infective stage and encysts. In endemic areas of Japan an incidence of 99% of the food fish, *Ophicephalus* sp., are found to be infected, and some fish will contain over 300 cysts. The natural final host is infected by feeding on the infected fish. In the final host the larva undergoes a migration to the liver before it returns to the stomach to become an adult.

If the infected fish is consumed by snakes, birds and/or mammals other than the natural host they will migrate from the intestinal tract to the cutaneous or subcutaneous tissue and/or somatic musculature where they produce a "larva migrans" picture, or form a granulomatous lesion or a stationary abscess. The disease syndrome involves mechanical injury produced by the migrating larvae, inflammation and toxic and allergic eosinophilia. The only effective treatment is the removal of the worms; prevention consists of informing people of potential dangers in consumption of fishes known to serve as intermediate hosts.

The giant kidney worm, *Dioctophyme renale* (Goeze, 1782) Stiles, 1901, is widely distributed throughout the world in fish eating mammals. The adult nematode lives in the kidney and gradually consumes the parenchyma finally leaving only the renal capsule. Diagnosis is usually made by detection of the eggs in the urine.

The life cycle involves a succession of hosts. The egg is consumed by an annelid worm in which the larva hatches and encysts. These annelids sometimes attach to the gills of crayfish which are consumed by the fish. In the fish host the larva develops to the infective stage and the infection of the final host is by consumption of infected fish (Woodhead[31]). The life cycle has been studied recently in the U.S.S.R. by Karmanova.[13,14] The freed larva migrates through the intestinal wall to the kidney (Hallberg[9]). Eleven human cases have been reported (Simenonoff[25]).

Perhaps the best known and most important nematode transmitted from animal to man is *Trichinella spiralis* (Owen, 1835) Railliet, 1895. This nematode shows little host specificity and can mature in any mammal which will eat the infective meat in a raw or undercooked condition. Upon ingestion of the flesh, the female worm will produce from 500-600 embryos which enter the blood stream where they are carried to the muscles and become encysted. In this stage they are capable of infecting another host. It has been shown to be common in the Arctic regions (Davies and Cameron[7]) where fuel is scarce and meat is eaten raw or undercooked. Especially significant in the Arctic is the occurrence of *Trichinella* in marine mammals. Although the incidence is low they can contribute to human infection.

Trichinelliasis has been reported from the walrus (Kuitunen-Ekbaum[17]); bearded seal (Rausch *et al.*,[22] Roth[23], Madsen[16], Thorberg[29]) and the ring seal (Roth[24]). Although the walrus generally feeds on marine invertebrates there is evidence that it will attack and kill seals and in many places has access to polar bear, dog and other carcasses which are left on the ice. Flensing of animals in the Arctic is performed in the intertidal zone and the debris discarded in the sea. The feeding habits of the walrus (digging for molluscs) would give ample opportunity for infection. Vibe[30] considered the amphipods which feed on carcasses of dead animals to be a carrier host to marine mammals.

Discussion

Although this review presents the obvious manifestations of zoonotic nematodes of marine and fresh water vertebrates there are undoubtedly others produced by the parasites which elicit host responses.

The harmful effect of larval migration depends largely on where they go and where they develop. This migration presents a multiplicity of pathogenicity.

Little is known of the role migrating larvae can play as a transport of bacterial or viral infections, as well as serving to open pathways to other organs for these organisms.

Granulomatous responses of the host to the presence of the larvae needs further investigation.

The first infection of man could produce a sensitization and subsequent infections with larvae of the same or related species may result in severe allergic reactions. *Ascaris lumbricoides* of man has a life cycle which involves a considerable somatic migration by the larvae after it has hatched from the egg in the small intestine. This migration involves mainly the liver and the lungs but can also involve other organs. Only those larvae which reach the lungs can become mature in the intestine; all others die in the organ they have reached. Nothing is known of the effect of these dead larvae in man and it is quite possible they could be responsible for sensitization. In many countries where people show varied symptoms of allergies such as eosinophilias, the population is or probably has been infected with intestinal nematodes, *e.g., Ascaris,* hookworms, etc., involving a visceral migration, as well as some perhaps exhibiting a "visceral larval migrans" *(e.g., Toxocara,* anisakid nematodes, *Gnathostoma* and even those showing a "cutaneous larva migrans" *(e.g.,* dog and cat hookworms, etc.). There is a possibility that the presence of these or the superimposed infection may result in certain allergic responses which have been diagnosed as syndromes of unknown etiology. Therefore, zoonotic nematodes may be involved in other than the more obvious pathological conditions discussed in this review.

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