CHAPTER 6

Food-Borne Nematode Infections

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6.1 PREFACE

For most of human evolution, man lived in widely dispersed, small nomadic groups that subsisted on hunting and gathering. While this lifestyle undoubtedly exposed individuals to the risk of occasional zoonotic disease transmission, population size likely minimized the effect of exposure to such diseases. The Neolithic period, which saw the advent of the agricultural revolution and the eventual domestication of certain animal species, not only brought about a rapid increase and concentration in human population but also fostered an environment in which substantial increases in infectious and nutritional diseases could rapidly spread (Armelagos, 1991). In addition, man's descent from the trees, subsequent eccrine evolution, and the development of agriculture forced him to become "water-bound," thereby putting him in contact with a group of infectious agents that can be described as "water associated" (Desowitz, 1981). It is within this context that many zoonotic diseases, including those of nematode origin that are discussed in this chapter, may have gained a foothold in human populations.

When one thinks about food-borne nematode infections, the first parasites that usually come to mind are *Trichinella* and *Anisakis*. While these two are certainly important in the overall context of food-borne infections caused by nematodes, since they are directly transmitted via infected food to susceptible individuals, one also has to consider other less frequently encountered nematode infections and infections caused by rather common nematodes, but which are transferred to humans via indirect food-borne routes. The overall goal of this chapter is to provide a rather broad, general perspective of the important nematode parasites that may find their way into our food chain, either directly or indirectly, and thus may serve as a potential source of infection and disease.

6.2 *TRICHINELLA* SPP.

6.2.1 Background

The credit for the discovery of *Trichinella* infection in humans is credited to Paget who in 1835 encountered cysts in the diaphragm muscle of an Italian man who had died of tuberculosis. It was Owen (1835), however, who publicized this finding and provided the name *Trichina spiralis* for the worm encountered in the tissues of this patient. Interestingly, earlier anatomical observations conducted by others in Europe indicate that calcifications and concretions encountered within muscle tissues of deceased patients may also have been *Trichinella* cysts, but were not adequately identified (Gould, 1970). The discovery of cysts in the extensor muscles of the thigh of a hog by Leidy (1846) helped pave the way for the discovery of the main features

of the life cycle of this parasite by Leuckart (1859) and especially Virchow (1860). The first case of fatal trichinellosis in man was described by Zenker (1860), while the first instance in which this disease was clinically diagnosed during the acute phase of infection was reported by Friedriech (1862). An excellent overview of the history of *Trichinella* and trichinellosis is provided by Campbell (1983).

Man, pigs, and *Trichinella* have likely had a long-standing relationship. Just how long is anybody's guess, but encysted larvae have been recovered from a 3200-year-old Egyptian mummy (Carvalho-Gonçalves *et al.*, 2003). The origin of domestic pigs can be traced to the Eurasian wild boar, *Sus scofa*. Subspecies of this ancestor likely diverged some 500,000 years ago, providing ancestral stock for pigs of Asian and European origin. Pig domestication occurred about 9000 years ago in China, with more recent introgression of the Asian subspecies into European domestic breeds in the eighteenth and nineteenth centuries, giving us the present day domestic pig (Giuffra *et al.*, 2000). Given that pigs are highly omnivorous and that numerous animal species can harbor *Trichinella*, it is likely that sylvatic cycles preceded the inclusion of man into the category of animal species that could harbor this infection. Predation by early man on wild pigs and other wild game, and certainly domestication of pigs, closed the zoonotic loop on this parasite. Today, trichinellosis is maintained in sylvatic and urban cycles and man fits into the epidemiological picture of this infection within both of these cycles.

6.2.2 Speciation

Trichinella was considered to be a single species until the discovery in the early 1970s of distinctive biological variants and nonencapsulated forms (Gajadhar and Gamble, 2000). Today, as a result of molecular, biochemical, and experimental studies, eight distinct species are recognized and include *T. spiralis*, *T. nativa*, *T. brivoti*, *T. murrelli*, *T. nelsoni*, *T. pseudospiralis*, *T. papuae*, and *T. zimbabwensis* (Bruschi and Murrell, 2002; Murrell *et al.*, 2000; Pozio *et al.*, 2002). The first five constitute the so-called encapsulated species, while the latter three are nonencapsulated species. In addition, three distinctive genotypes, *Trichinella* T6, T8, and T9, have been identified from distinct geographical regions in carnivores, but have not yet been classified as species (Kapel, 2000a; Murrell and Pozio, 2000; Pozio, 2000a). From a human perspective, only *T. spiralis* is maintained in an urban domestic cycle (Pozio, 2000a). All other recognized species and genotypes are transmitted and maintained in somewhat distinctive sylvatic cycles, however, this certainly has not precluded their transmission to humans; as will be discussed later. In addition, sylvatic reservoirs frequently become synanthropic, thus bringing infections in contact with domestic animals and humans. Collectively, *Trichinella* spp. have a wide cosmopolitan distribution and infect a very broad range of mammals, primarily occurring in those with scavenging and carnivorous habits. In humans, *Trichinella* prevalence has been estimated at perhaps as many as 11,000,000 infections (Dupouy-Camet, 2000).

6.2.3 Life Cycle

The complete life cycle of *Trichinella* spp. occurs within the same mammalian host and involves enteral and parenteral phases. There are, however, some exceptions to this in which some species appear to have limited development in some hosts

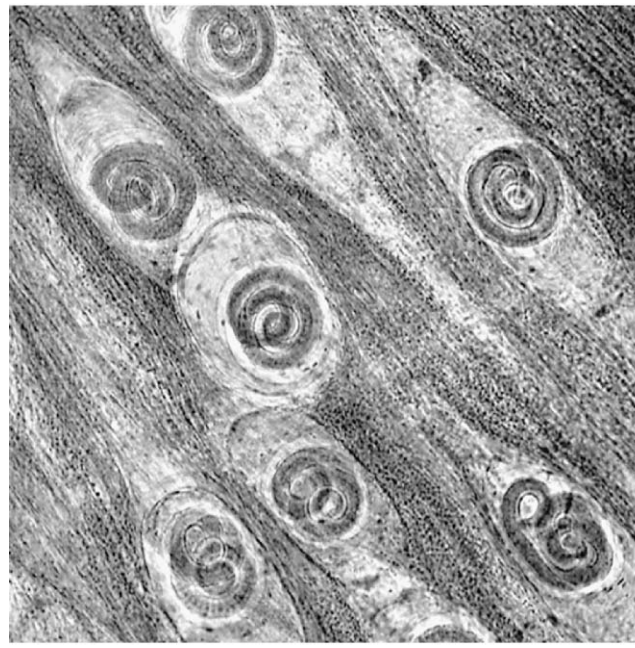

Figure 6.1. Encysted muscle larvae of *Trichinella spiralis*.

(Kapel, 2000a). Infection and the enteral phase commence when first stage larvae contained within muscle tissues (Fig. 6.1) are ingested by another host. The larvae are released from the "nurse cell" by the action of gastric fluids and enzymes and pass into the small intestine, where they penetrate the epithelium to become intramulticellular organisms (Despommier *et al.*, 1978). Larvae molt four times at this site within 30 h to become sexually active adults. Mating ensues and female worms give birth to an estimated 500 to 1500 live newborn larvae (ovoviparous) until host immunity brings about their expulsion (Wakelin and Denham, 1983). The parenteral phase begins with larval entry into the circulatory system and distribution to multiple body organs. Only larvae that invade muscle cells are destined to survive and do so by establishing a unique, albeit highly modified, intracellular niche. This may take from 3 to 4 weeks to complete, at which time they are infective to another host. The complex of parasite and host cell becomes a remarkably stable unit termed the nurse cell (Despommier, 1990, 1998), which in some instances may survive for years. This may especially be true in natural hosts. In three species, *T. pseudospiralis*, *T. papuae*, and *T. zimbabwensis*, however, the larvae do not induce cyst or capsular formation and it is not known how long such larvae can survive in host muscle tissue.

6.2.4 Epidemiology

There is no easy way to discuss the overall epidemiology of *Trichinella* without providing information on each of the species involved in this complex picture. Likewise, it has to be kept in mind that most of these species have the potential of

infecting man and that in many instances, the species responsible for initiating an infection or outbreak has never been determined. In addition, those species occurring in sylvatic cycles can exist in areas where the domestic cycle involving *T. spiralis* predominates, further complicating the epidemiological picture. Because of this, it is appropriate to discuss the biology and epidemiology unique to each *Trichinella* sp. and then put it in the context of human infection and the overall epidemiological picture. Overviews of these species are provided in the reviews of Kapel (2000a) and Pozio (2000a; 2001a).

6.2.4.1 *Trichinella spiralis*

Trichinella spiralis is responsible for the domestic cycle of trichinellosis because of its primary association with the domestic pig. It also occurs in sylvatic pigs and other synanthropic animals such as rats. Infections occur in many countries of temperate and tropical regions but are unlikely to occur in colder regions because muscle larvae are incapable of surviving in frozen animal carcasses (Pozio, 2001a). Exceptions to this, however, might occur in situations where wildlife may harbor this infection near human settlements. Geographic distribution of this species within temperate and tropical zones, therefore, is frequently determined by the extent to which domestic, synanthropic, and potentially sylvatic cycles may overlap. *T. spiralis* is highly pathogenic to humans and is more infective to pigs and other domestic animal species such as the horse, cow, sheep, and goats than are other *Trichinella* spp. or genotypes (Kapel, 2000a; Kapel *et al.*, 1998). This species is infrequently encountered in wild animals. For these reasons, and because of human influences, *T. spiralis* has become the predominant species encountered within humans. Epidemiologically, transmission routes of *T. spiralis* in farm animals such as the pig are usually the result of poor breeding and rearing habits instituted by humans. Identified transmission routes include the feeding of pigs with infected scraps of meat from other pigs, tail-biting among pigs, ingestion of feces from pigs fed infected meat, ingestion of infected rats on poorly managed pig farms where effective barriers of rodent control have not been installed, and ingestion of other synanthropic or sylvatic animals under similar circumstances (Pozio, 2000a).

6.2.4.2 *Trichinella nativa*

This species is interesting because of its geographic distribution, being restricted to arctic and subarctic regions where it is largely a parasite of sylvatic and scavenging carnivores such as bears, foxes, wolves, and mustelids. Biological features that help distinguish this species include the variable freeze tolerance of muscle larvae and the inability to maintain long-term infections in pigs, boars, and other livestock (Kapel, 2000a). The most freeze-resistant larvae, which can remain viable for years in some instances, are found in animals such as the polar bear and arctic fox that live at the more northern latitudes of this parasite's range (Dick and Pozio, 2001; Kapel *et al.*, 1999). *T. nativa* is highly pathogenic in humans and occurs where sylvatic animals are hunted and consumed as a food source.

6.2.4.3 *Trichinella brivoti*

Trichinella brivoti is a sylvatic species infecting wolves, foxes, raccoon dogs, mustelids, and wild boars in more temperate parts of Europe and Asia. It has also

been encountered in synanthropic rats, domestic pigs, and horses in mainland Europe and other parts of Asia. Encysted larvae have been reported to survive up to 6 months in the frozen (−20°C) muscle tissues of carnivores (Pozio *et al.*, 1989). When encountered in wild boars and domestic pigs, its prevalence is always lower than for *T. spiralis* (Pozio, 2001a). Infectivity is comparable in the wild boar and domestic pig and the former has been cited as a frequent source of infection to humans where infection is moderately pathogenic (Kapel, 2000a, 2000b).

6.2.4.4 *Trichinella murrelli*

This parasite appears to have a predominantly sylvatic cycle in carnivores such as foxes, coyotes, raccoons, black bears, and bobcats of the more temperate regions of the United States. Very low infectivity to noninfectivity has been reported from domestic pigs (Kapel and Gamble 2000; Yao *et al.*, 1997). Muscle larvae are moderately tolerant to freezing, but less so than for *T. brivoti* (Pozio *et al.*, 1994a). Infections in humans have been reported in France following the consumption of raw horsemeat imported from the United States (Ancelle *et al.*, 1988; Dupouy-Camet *et al.*, 1994).

6.2.4.5 *Trichinella nelsoni*

Trichinella nelsoni infections predominate in scavenging carnivores, south of the Sahara region in Africa. Jackals, hyenas, cheetahs, lions, leopards, and wild pigs have been found to harbor infections. The latter are thought to be a common source of human infection (Pozio *et al.*, 1994b). Infectivity is moderate for both domestic pigs and wild boar (Kapel and Gamble, 2000). Interestingly, this genotype is very sensitive to freezing, yet somewhat resistant to elevated temperatures such as might be encountered in decaying meat in the tropics of Africa (Sokolova, 1979). This genotype is not highly pathogenic in humans (Bura and Willett, 1977).

6.2.4.6 *Trichinella pseudospiralis*

A distinguishing feature of this species is that muscle larvae are not encapsulated. In addition, this species can infect birds as well as mammals. A broad diversity of animal species, including wild carnivores, meat eating birds, marsupials, rodents, pigs, and man have been found to harbor infection with *T. pseudospiralis* (Kapel, 2000a; Pozio, 2001a). It appears to be a very biologically diverse species with molecular and biochemical differences having been detected in isolates from varying regions (La Rosa *et al.*, 2001). Larvae are more difficult to detect on necropsy and in digests than for the encapsulated species, so this species may be underreported. This species does not appear to tolerate heat or freezing as well as encapsulating species (Sokolova, 1979). Severe disease has been reported in humans from Australia, Asia, and Europe. In each instance, infection from wild pigs was implicated (Kapel, 2000a).

6.2.4.7 *Trichinella papuae*

Trichinella papuae, another nonencapsulated species, has recently been described from wild and domestic pigs of Papua New Guinea (Pozio *et al.*, 1999). The distribution of this parasite is unknown; as are reservoirs. It is not infective to birds, as is *T. pseudospiralis*, has an intermediate sized larvae, and has a low infectivity to mice (Pozio *et al.*, 1999). This parasite has recently also proven infective to several

reptilian species (Pozio *et al.*, 2004). A focus of human infection with this parasite has been described from New Guinea (Pozio, 2001b).

6.2.4.8 *Trichinella zimbabwensis*

The third nonencapsulated *Trichinella* species was recently described from farmed crocodiles in Zimbabwe and is similar in morphology to *T. papuae* (Pozio *et al.*, 2002). This species is also infective to mammals. It represents the first nonencapsulated species described from Africa and because it can infect both reptiles and mammals suggests a more ancient lineage for this parasite than previously thought (Pozio *et al.*, 2002). Infections have not been reported in humans to date.

In addition to the above-recognized species, the following genotypes have also been described but not yet classified.

6.2.4.9 *Trichinella T6*

This genotype is related to *T. nativa* and has been detected in numerous carnivores (bears, wolves, foxes, mountain lions) of subarctic regions of North America. Like *T. nativa*, this genotype can survive in frozen tissue for several years (Worley *et al.*, 1990). Human infections have resulted from eating game meat (Dworkin *et al.*, 1996). *Trichinella* T6 and *T. nativa* can interbreed and live in sympatry in Arctic wolf populations (La Rosa *et al.*, 2003). This has led to the suggestion that these two genotypes may have diverged and are still diverging as a result of glaciation episodes in North America.

6.2.4.10 *Trichinella T8 and T9*

These two genotypes are related to *T. brivoti*, but with disparate geographical distributions. The former has been isolated from carnivores of South Africa and Namibia whereas the latter has only been seen in a dog and bear from Japan (Kapel, 2000a). Very little is known of the biological characteristics of these two genotypes.

6.2.5 Human Trichinellosis–Epidemiology

In the not too distant past, all trichinellosis was thought to be caused by a single species, *T. spiralis.* This view had been propagated in many circles because of the well-known and ingrained association of this infection with the domestic pig. The advent of molecular tools and biochemical methods capable of distinguishing species has greatly impacted our knowledge of *Trichinella* as a species complex and has contributed to a more thorough understanding of the epidemiology of this parasite.

The modern day epidemiology of human trichinellosis is governed by complex interactions involving changes in human activity as well as by interactions that bring humans into contact with sylvatic species of the disease. Most human trichinellosis is still caused by *T. spiralis* and is maintained in the domestic cycle because of human activity linked to pig breeding and rearing. Transmission routes of epidemiological importance that likely impact the domestic cycle include: (1) pigs eating scraps composed of other infected pigs, (2) predation on other infected synanthropic animals, such as rats, (3) pig habits such as tail-biting and coprophagy where infections are endemic, and (4) occasional ingestion of infected sylvatic animals; although this latter route does not lead to persistent cycling because the domestic pig, in many

instances, becomes a dead end host (Pozio, 2000a). *T. spiralis*, however, can easily cross back into the sylvatic habitat and become a persistent problem where poor management practices prevail (Murrell and Pozio, 2000).

The problem of human infection with *T. spiralis* can usually be linked to factors related to either nonexistent public health or veterinary standards that might help prevent disease transmission, or to a breakdown in such standards. In China and many areas of Latin America, pigs have become a part of the cash economy for the family unit. As such, they are often butchered in backyard operations and their meat is sold without benefit of any formal inspection. The lack of health education and an awareness of *Trichinella* itself in such environments contribute to a sustained spread of disease (Pozio, 2000a). In eastern Europe and parts of the former USSR, political change and economic destabilization have resulted in a diminished quality of veterinary care and controls that have led to a sharp rise in the prevalence of domestic trichinellosis. Pig production on large well-controlled collective farms has diminished significantly and fallen back to small individual farms that lack adequate feed, technology, and veterinary services. In such situations, the prevalence of *Trichinella* infection in pigs is frequently 10 times higher than on the collective farms (Bessonov, 1994), ultimately resulting in more human infections. In countries such as Lithuania and Romania, the incidence of trichinellosis has increased nine-fold and 17-fold, respectively, over the past two decades, making it a truly reemergent disease problem (Olteanu, 1997; Rockiene and Rocka, 1997).

Globalization of trade is another risk factor that has resulted in the spread of trichinellosis from endemic to largely nonendemic regions. This type of transference results in meat products ending up in markets where inspection services may not be familiar with this disease problem. Nowhere has this been more evident than in France and northern Italy, where infected horsemeat from North America, Mexico, and especially eastern European countries has contributed to more than 3300 human infections (Pozio, 2000b). *T. spiralis* infection in horses carries with it the implication that such noncarnivorous animals are being fed infected food products and again underscores the need for adequate veterinary control practices and regulations (Dupouy-Camet *et al.*, 1994).

In countries such as the United States, where pig production has increased and control programs and surveillance have demonstrated a dramatic reduction in the annual number of cases of trichinellosis in humans due to *T. spiralis*, there has been an increase of cases associated with consumption of infected game meat (Moorhead *et al.*, 1999; Murrell and Pozio, 2000). Many of these new infections have been attributed to *T. nativa*, *T. murrelli*, or *Trichinella* T6 (Gajadhar and Gamble, 2000). Regions of Canada, and particularly the Northwest territories and Quebec, have experienced human rates of infection 200-fold over that of the general population in spite of a decline in domestic trichinellosis, once again underscoring the likely connection with consumption of infected game meat (Murrell and Pozio, 2000). In many countries the impact of sylvatic *Trichinella* transmission to humans cannot be adequately addressed because of the lack of appropriate diagnostic tests.

In many instances, human behavior influences the transmission of certain *Trichinella* genotypes within defined habitats. International travel and the fashionable consumption of undercooked meat can explain outbreaks in certain population

groups (Dupouy-Camet, 2000). Likewise, outbreaks have occurred in defined ethnic communities within countries where the overall prevalence rates are low, but where in their native countries the prevalence rates are much higher. This situation is likely linked to customs of food preparation (Pozio, 2000a; Shantz and McAuley, 1991). In Arctic regions, hunting habits of native peoples not only contribute to high rates of infection among human populations, but help sustain infection in wildlife populations as well. Among the Inuit culture, sled dogs are fed the remains of killed animals including polar bears, which have some of the highest rates of trichinellosis recorded because of their carnivorous and scavenging habits. Dogs that die of trichinellosis are left as carcasses that become available to infect other scavenging carnivores (Kapel *et al.*, 1997).

Ecological modifications can also impact the distribution of *Trichinella* genotypes. A decrease in the number of farms and reforestation in parts of Europe has led to an increase in the wild boar population and maintenance of sylvatic trichinellosis (Pozio *et al.*, 1996). Likewise, food access of pigs to potential wildlife reservoirs is likely to increase as human populations expand and with it the need for increased food production in certain areas of the world. Such situations have already been implicated as a major risk factor for transmission to sylvatic animals (Dame *et al.*, 1987; Murrell *et al.*, 1987).

Another factor that likely plays a role in the overall epidemiology of trichinellosis and certainly in the reemergence of this disease problem is the issue of misdiagnosis. Clinical manifestations of disease such as fever, myalgia, and fatigue, particularly in the early stages of infection, can easily be mistaken as influenza. Other features of infection, such as the appearance of facial edema, could also be mistaken as manifestations of an allergic response brought on by the flu. Because of this issue, the disease is likely to be underreported in many areas where health care systems are incapable of providing optimal service, especially with respect to infectious disease diagnosis.

6.2.6 Clinical Manifestations

Clinical aspects of trichinellosis have been aptly described and are summarized in excellent reviews by Capo and Despommier (1996) and Bruschi and Murrell (2002). The course of disease depends, in part, on the infecting species and may result in mild, to moderate, to severe symptoms. The incubation period may be variable, ranging from 7 to 30 days. During the enteral phase, which usually lasts about 6 weeks, patients may exhibit upper abdominal pain, diarrhea or constipation, vomiting, malaise, and low-grade fever. Variations depend on the severity of the infection, age, sex, ethnicity, and immune status of the infected individual. Such symptoms, which are not uncommon with other enteral infections, often make diagnosis difficult at this time during the infection. Gastrointestinal symptoms may overlap with the parenteral phase of the infection during which time the larvae are migrating and invading muscle tissue. This phase, which may begin as early as 2 weeks following infection, is characterized by inflammatory and allergic responses that may be manifest as facial edema, diffuse myalgia, conjunctivitis, fever, headache, and urticaria. The most severe cases usually serve as index cases of an outbreak and prompt epidemiological investigations. Today, death occurs in only

rare instances because of improved therapies, and the long-term consequences of harboring larvae are somewhat controversial since many will ultimately undergo complete calcification.

6.2.7 Diagnosis and Treatment

An important aspect of dealing effectively with trichinellosis is to affect a diagnosis as early as possible during the course of an infection. As already mentioned, clinical signs of disease early in the infection can often be misleading. Obtaining a history of food consumption can play an important role in suspecting trichinellosis and this should be followed up with blood work to demonstrate a hypereosinophilia and an elevation of certain muscle enzymes, such as creatine phosphokinase and lactate dehydrogenase. Taken collectively, the food consumption history and laboratory findings are highly pathognomonic for the disease (Bruschi and Murrell, 2002; Despommier *et al.*, 2000). A definitive diagnosis can be made by finding encysted muscle larvae or by using immunodiagnostic procedures, but the latter are not likely to be positive until several weeks into the course of an infection, by which time the larvae are fully ensconced in the muscle tissue (Bruschi and Murrell, 2002).

An important goal of early treatment is to limit muscle invasion by larvae and barring that, to limit muscle damage after larvae have invaded. Drugs such as mebendazole, albendazole, and thiabendazole, members of the benzimidazole family, work quite effectively against intestinal worms, although the latter has been noted to have frequent side effects (Kociecka, 2000). These drugs are not likely to control infections once they have reached the muscle encapsulated stage and in severe cases, hospitalization is required (Pozio *et al.*, 2001). In severe cases, treatment may involve administration of glucocorticosteroids, analgetic drugs, or even immunomodulating drugs, depending on the individual patient (Kociecka, 2000).

6.2.8 Prevention and Control

Effective prevention and control of trichinellosis depends not only on reducing transmission of the disease to humans via livestock and wild game, but also on reducing transmission of infection between sylvatic and synanthropic animals and domestic livestock. In highly industrialized countries where pig farming is more tightly regulated and where inspection or processing methods are in place to insure trichina free meat, trichinellosis has been more or less confined to the few who acquire infection via consumption of wild game, or who because of culinary habits have acquired a taste for food prepared in ways inadequate to insure safety. In many parts of the world, however, economic problems, the erosion or lack of appropriate veterinary and medical infrastructures, and poor education translate into virtually no control of this disease at the level of the farm and abattoir and into no general information about the disease being disseminated to the general public or to the medical profession (van Knapen, 2000).

The discovery of the association of trichinellosis in humans with the consumption of infected pork in the nineteenth century led to the advent of microscopy and later trichinoscopy as a means of detecting infection in pigs. Virchow, who did much to advance our knowledge of *Trichinella*, has been credited with initiating inspection of pork for the control of trichinellosis in provincial provinces of Germany in the

mid-1860s (Gould, 1970; Nöckler *et al.*, 2000). Trichinoscopy, which relies on the postmortem examination of select muscle samples by compression between glass slides, and is viewed as having rather limited sensitivity, served as the mainstay of diagnosing infected meat from its inception until 1978 when the pooled artificial digestion method was introduced (Nöckler *et al.*, 2000). This technique, which utilizes pepsin digestion of select muscles to release encysted larvae, results in a threefold or greater increase in sensitivity when compared to trichinoscopy (Forbes *et al.*, 2003). The pooled digestion assay permits testing of up to 100 carcasses at the same time and for sensitivity in the range of 1 larva per gram of muscle tissue, digestion of at least 5 grams of muscle tissue is recommended (Gamble, 1996; Gamble *et al.*, 2000; Nöckler *et al.*, 2000). Identification of muscle to be tested may vary because of differences in predilection sites by the various *Trichinella* species and because of infected host differences. In addition, not all *Trichinella* species encyst and thus trichinoscopy may be unable to detect such infections (Nöckler *et al.*, 2000). In spite of this, trichinoscopy is still in use, particularly in the examination of muscle tissue from wildlife in epidemiological studies. The International Commission on Trichinellosis has strongly urged that all laboratories that test for the presence of Trichinella adapt quality assurance guidelines and the Canadian Food Inspection Agency's Centre for Animal Parasitology has taken the lead in this regard (Gajadhar and Forbes, 2002).

The use of immunological techniques applicable to the serodiagnostic detection of Trichinella infections in pigs and other animals has gained in popularity, but have yet to become a permanent diagnostic fixture because they have not been standardized (Gamble *et al.,* 2004). Such techniques, and particularly the enzyme-linked immunsorbent assay (ELISA), have the advantage of permitting testing of pre and postmortem animals and with greater sensitivity. However, the ELISA may not be able to detect very early or late stage infections and for this reason has not replaced digestion testing (Nöckler *et al.*, 2000). Still, the ELISA has proven invaluable as a test for pig herd surveillance and for detection of infection in humans (Gamble, 1996; Gamble *et al.*, 2004).

Processing methods to control trichinellosis have also been recommended. Such methods are particularly applicable where meat from pigs or other animals are not tested by recommended inspection procedures, these include cooking, freezing, and irradiation (Gamble *et al.*, 2000). Cooking of meat to an internal temperature of 71°C (160°F) should be sufficient to kill any *Trichinella* larvae present. In the absence of temperature monitoring, meat texture and color should be monitored. Freezing guidelines call for cuts of meat up to 15 cm thick to be frozen solid for no less than 3 weeks, rising to 4 weeks for meat up to 69 cm thick. Irradiation, where permitted, at 0.3 kGy has proven to inactivate *Trichinella* larvae and is recommended for sealed packaged food only. Interestingly, irradiation was reported to be effective in killing *Trichinella* cysts in pork by scientists of the USDA as early as 1921 (Steele, 2000). The key element associated with these recommendations is good consumer education.

In addition to the above, prevention and control of trichinellosis requires utilization of strict rules of good production practices on farms along with good veterinary practices to minimize the transmission risks of *Trichinella* spp. The following

summarizes guidelines required for *Trichinella* free pig production (Gamble *et al.*, 2000; Murrell and Pozio, 2000; van Knapen, 2000):

- Construction of animal containment facilities that will exclude rodents and wildlife.
- Provisions for feed storage that do not permit rodent or animal entry and purchase of food from approved facilities.
- Adherence to garbage feeding regulations and cooking of meat products to inactivate any larvae that may be present.
- Maintenance of effective rodent and bird control programs.
- Purchase of new animals only from certified *Trichinella*-free farms.
- Absence of garbage dumps within a 2 km radius of the farm.
- Appropriate disposal of pig and other animal carcasses to minimize infection risks on the farm and with other animals.

Eradication of *Trichinella* from wild animal populations may never be possible due to predatory and scavenging habits, but certainly could be impacted upon if hunters would properly dispose of offals and carcasses.

Finally, pig vaccination against *T. spiralis* using antigens of the newborn larvae of the same or different species of *Trichinella* has been attempted and could have applications in situations where the above-mentioned management practices are not used or have not been successful (Marinculic *et al.*, 1991; Marti *et al.*, 1987; Smith, 1987). Such an approach, however, is likely to be very expensive and have limited use.

6.3 *ANISAKIS SIMPLEX* AND RELATED SPECIES

6.3.1 Background

Worms parasitizing fish were recorded as early as the thirteenth century. It was not until 1845, however, that Dujardin described the taxonomic position of *Anisakis* from dolphins, a common worm parasite encountered in fish with marine mammals as definitive hosts (Bouree *et al.*, 1995). This nematode and the relatives *Pseudoterranova decipiens* and *Contracaecum* spp. are all anisakids belonging to the same superfamily of worms as the common human intestinal roundworm, *Ascaris lumbricoides.* All are likely to cause human infection through the consumption of raw or undercooked fish (Sakanari and McKerrow, 1989). Additional anisakid worms have been implicated in causing human disease but case reports are rare. *A. simplex* and *P. decipiens* are the only anisakids reported from human cases in the United States http://vm.cfsan.fda.gov/~mow/chap25.html.

The first case involving human infection with *Anisakis* was recorded in Holland from an eosinophilic intestinal lesion in a patient with severe abdominal pain. This case was important because it linked the eating of infected raw herring with observed symptoms (van Thiel *et al.*, 1960). Endoscopy was used to directly view larvae of *Anisakis* in an infected individual in 1968 (Namiki, 1989) and in 1975 the first case of human infection was recorded from the United States (Pinkus *et al.*, 1975).

6.3.2 Life Cycle

The life cycles of the various anisakids usually involve two intermediate hosts with the final definitive host being a marine mammal such as dolphins, sea lions, and whales (Bouree *et al.*, 1995; Sakanari and Mckerrow, 1989; Smith, 1983). Adult worms reside within the stomach of their definitive host and eggs are passed in the feces to embryonate in seawater. Eggs containing first stage larvae can hatch or be ingested. In either case, planktonic crustaceans ingest the larvae and serve as first intermediate hosts for this infection. Larvae develop to the second stage in these intermediate hosts, which are then consumed by fish where they become infective third stage larvae. Distribution of these third stage larvae within the fish hosts can vary from the body cavity to encystment in various tissues. In the normal life cycle, marine mammals consume infected fish at which point the larvae molt within the stomach to become adult worms. In some instances, it is possible for a larger predatory fish or animal, such as man, to eat a smaller infected fish in which case the life cycle is completed. In these hosts, the worms do not mature. It is also theoretically possible for marine mammals to be infected via ingestion of infected crustaceans, which might result in larval development in their body cavities or tissues, but this has not been reported.

6.3.3 Epidemiology

Anisakiasis, or disease caused by anisakid worms, is frequently reported in the literature as simply due to *Anisakis* spp. This is probably because of the fact that attempts are often not made to distinguish between the infecting anisakid species. There is little doubt that more attention needs to be paid to which distinct species is causing infection and to try to discern which fish species is responsible for the cause of infection so that the epidemiology of these infections can be better defined. Having made this statement, there is no doubt that the transmission of these anisakid infections to humans is associated with the food-borne route via the consumption of raw and undercooked fish. Fish dishes from various parts of the world that are considered to pose high risk of infection include sushi, sashimi, pickled or smoked herring, dry cured salmon, lomi-lomi, cebiche, and pickled anchovies (Audicana *et al.*, 2002).

Epidemiological studies from Japan, where 95% of the cases of anisakiasis have been reported from, indicate that the main sources of *A. simplex* are the spotted chub mackerel and Japanese flying squid. In Western Europe, *A. simplex* is principally found in herring and in Spain, anchovies and sardines are the main culprits (Audicana *et al.*, 2002). Fish found in North American waters that may harbor *A. simplex* infections include wild caught and farm reared salmon (Deardorff and Kent, 1989), Pacific herring (Moser and Hsieh, 1992), and Atlantic cod (Chandra and Khan, 1988). Halibut, mackerel, rockfish, and squid may also be infected (Sakanari and Mckerrow, 1989). *A. simplex* has also been reported from ahi off Hawaiian shores (Deardorff *et al.*, 1991). Recently, *A. simplex* was reported for the first time from the American shad in Oregon rivers (Shields *et al.*, 2002). This report is significant because they are an introduced species that was derived from stock from the Hudson River in New York where they are normally not infected. In addition, infected fish were found in rivers distant from ocean sources, suggesting an ecological expansion

of this infection into a new environment. Interestingly, *A. simplex* and other anisakids have recently been reported from river otters in the Pacific Northwest at considerable distances from the ocean (Hoberg *et al.*, 1997). Otters have been observed feeding on living and dead shad during their spawning runs and outmigrations (Shields *et al.*, 2002). Taken collectively, these observations suggest that this new host—parasite relationship within the Northwestern United States has led to infections spreading within a new habitat that also pose risks to certain wildlife species and potentially to humans as well. It is also a reminder that the introduction of stock fish species into new areas, whether infected or not, can alter host—parasite relationships.

Pseudoterranova decipiens, the other anasakid associated with human infection in North America, is most commonly encountered in cod and can be found in cod worldwide (Oshima, 1987). Red snapper, another popular fish consumed in the United States, has been thought to be the main source of codworm anisakiasis (Oshima, 1987). In Mexico, where cebiche is a popular dish, five fish species commonly used in its preparation were found to be frequently infected with this parasite. They included the lane snapper, yellow fin mojarra, barracuda, red grouper, and white grunt (Laffon-Leal *et al.*, 2000).

As of 2002, a total of ~14,000 cases of anisakiasis have been reported, with Japan accounting for about 2000 new cases annually (Audicana *et al.*, 2002). Epidemiological studies conducted in Japan, have shown that cases of anisakiasis were more likely to be encountered in coastal areas where individuals were involved in the fish industry (Asaishi *et al.*, 1980). Cases from Europe, the United States and elsewhere also appear to be on the rise, but are more likely the result of culinary habits associated with ethnic groups or restaurants. The increased recognition of anisakiasis as a problem is probably due in large part to an increased awareness of this infection and the means by which to diagnose it.

6.3.4 Clinical Manifestations

The clinical symptoms of acute anisakiasis include sudden epigastric pain, nausea, vomiting, diarrhea, and frequently urticaria (Audicana *et al.*, 2002; Bouree *et al.*, 1995; Sakanari and Mckerrow, 1989;). In fact, most of the symptoms are probably the result of digestive tract allergic reactions (Asaishi *et al.*, 1980; Audicana *et al.*, 2002; Moreno-Ancillo *et al.*, 1997). Onset of symptoms is usually rapid and may persist for 1–5 days. Infections may frequently be misdiagnosed at this time. Chronic anisakiasis results from larval invasion that causes abscess or eosinophilic granulomas. This form of disease can mimic other conditions such as appendicitis, gastroduodenal ulcer, colitis, inflammatory bowel disease, and intestinal obstruction (Moreno-Ancillo *et al.*, 1997). Some *Anisakis* allergens are heat and freeze resistant. Cooking, therefore, which kills the parasite, might not prevent potent allergic responses (Audicana *et al.*, 1997; Moreno-Ancillo *et al.*, 1997). In some individuals, sensitization may even lead to severe anaphylactic shock (Audicana *et al.*, 2002; Moreno-Ancillo *et al.*, 1997).

6.3.5 Diagnosis and Treatment

Vague symptoms during the early stages of the disease can often lead to a misdiagnosis (Sakanari and Mckerrow, 1989). Because clinical manifestations of allergy

often accompany anisakiasis, it is clinically relevant to link consumption of fish with the attendant disease symptoms and then to rule out allergic responses to fish as the likely cause of disease. The diagnosis of allergy brought on by an anisakid infection can be assessed using the following criteria: (1) a history of urticaria, angioedema or anaphylaxis following fish consumption, (2) a positive skin-prick test with a somatic extract of larvae, (3) a radioimmunoassay which detects *Anisakis*-specific IgE responses, and (4) a lack of reaction to proteins from the ingested fish host (Audicana *et al.*, 2002). Immunoblotting techniques that detect the presence of antigen specific antibody responses may also be of use (Del Pozo *et al.*, 1996; Montoro *et al.*, 1997). The value of immunoassays is likely increased in populations previously sensitized to *Anisakis* antigens. Many serologic assays, such as Ouchterlony tests, indirect haemagglutination, complement fixation, and ELISA can yield false positive reactions due to the cross-reactivity with other parasite antigens (Kennedy *et al.*, 1988). It must also be kept in mind that many of these assays are of limited use in the case of acute disease.

Endoscopy, applied early during the course of infection, is still one of the most important tools in both the diagnosis and treatment of anisakiasis (Bouree *et al.*, 1995; Sakanari and Mckerrow, 1989). Worms cannot only be visualized, but removed with the biopsy forceps for identification. Worm removal also minimizes the chance of allergic responsiveness characteristic of more chronic infections. Even in chronic cases, this technique may identify larval cuticular debris within granulomatous lesions (Gomez *et al.*, 1998). Chemotherapeutic treatments have not been described for anisakiasis.

6.3.6 Prevention and Control

Visual examination of fish is required in Europe with extraction of visible parasites and removal of heavily parasitized fish from the markets (Audicana *et al.*, 2002). Evisceration of fish soon after they are caught is recommended since larvae are known to leave the digestive tract and go into the muscles (Declerck, 1988; Smith and Wooten, 1975). Salting, smoke curing, and marinating are not likely to affect larval viability (Bouree *et al.*, 1995). The United States Food and Drug Administration has listed guidelines for seafood not cooked to temperatures above 140° F throughout, which includes blast freezing to −31°F or below for 15 h, or regular freezing to −10°F for 7 days (Deardorff *et al.*, 1991). These measures should reduce the risk of anasakid infection but may not totally eliminate the risk of allergic responses. For that, one would have to consider a fish-free diet!

6.4 *ANGIOSTRONGYLUS CANTONENSIS* AND *ANGIOSTRONGYLUS COSTARICENSIS*

Angiostrongylus cantonensis and Angiostrongylus costaricensis are metastrongyle nematodes. As a group, the metastrongyles usually occur in the lungs of mammals. *A. cantonensis*, a parasite of the lungs of rats, was first observed in China in 1935 (Chen, 1935). The first reported human infection described a 15-year-old boy in Taiwan in 1945 with symptoms of meningitis, and from whom worms were recovered from

cerebrospinal fluid (Beaver and Rosen, 1964). Humans are accidental hosts for this infection, as the worms never reach maturity. It is now recognized as a major cause of eosinophilic meningitis worldwide, although most of the cases still occur in areas around Southeast Asia (Alicata, 1991; Cross, 1987; Lo Re and Gluckman, 2001; Rosen *et al.*, 1967). Snails and slugs serve as intermediate hosts, while shrimp, crabs, and land planarians serve as transport hosts (Alicata, 1991). Oysters and clams have been experimentally infected and from which juvenile worms produced infections in rats (Cheng, 1965). Adult worms reside within the pulmonary arteries of rodents where females lay their eggs. Eggs hatch at this site and first stage larvae work their way into the lungs, up the trachea, are swallowed, and subsequently passed in the feces. Snails and slugs consume these larvae, which then undergo two molts to become infective third stage larvae. Rodents and man get infected by ingesting raw snails, vegetables contaminated with snail or slug slime, or transport hosts that have consumed infected snails or slugs (Lo Re and Gluckman, 2001). In rodents, larvae migrate to the lungs to become adults, while in man the larvae make it as far as the central nervous system where they die and produce inflammation. Increased dispersal of infected rodents worldwide via shipborne routes is likely contributing to a spread of this disease (Kliks and Palumbo, 1992). Infections have recently been reported in humans in the United States and Caribbean (New *et al.*, 1995; Slom *et al.*, 2002) and the parasite has been reported as endemic in Louisiana wildlife (Kim *et al.*, 2002). Clinical manifestations develop from 2 to 35 days after infection and most frequently present as headache (Lo Re and Glickman, 2001). Fever and vomiting are less frequently observed. Abnormal skin sensations of the extremities, trunk, or face are also frequently encountered and can persist for weeks. Diagnosis is difficult at best and usually involves a history of exposure in an endemic area along with a clinical presentation suggestive of infection with this parasite. An elevated CSF eosinophilia along with serologic testing will confirm infection (Lo Re and Glickman, 2001). Unfortunately, commercially available serologic tests are not available. Therapy is controversial and in some instances treatments have resulted in inflammatory reactions due to responses directed against antigens of dying worms (Pien and Pien, 1999). Use of glucocorticosteroids may reduce illness duration (Chau, 2003). Control is difficult because attempts to eliminate snail hosts may harm native or beneficial snail species (Alicata, 1991). Avoidance of eating uncooked or undercooked snails and transport hosts is the best way to prevent infection.

Angiostrongylus costaricensis is also a parasite of rodents, but adult worms reside within the mesenteric arteries of the ileocecal region (Morera, 1973; Waisberg *et al.*, 1999). Abdominal angiostrongyliasis, due to infection with this worm, was first reported from humans in 1967 in Costa Rico (Morera, 1967; Céspedes *et al.*, 1967) and is endemic in Central and South America where it primarily infects children, perhaps due to their play habits (Loría-Cortés and Lobo-Sanahuja, 1980; Morera, 1973; Morera *et al.*, 1982). Unlike *A. cantonensis*, this parasite can mature to adults in humans. Infections lead to inflammation and thrombosis in the terminal branches of the superior mesenteric artery, and possibly to perforation and peritonitis (Waisberg *et al.*, 1999). The life cycle of this parasite parallels that of *A. cantonensis* with the exception of adult worm site location in the definitive host. Adult worms residing within the mesenteric arteries deposit eggs that hatch in the

feces; releasing first stage larvae. These are ingested by land slugs in which they undergo two molts to become infective third stage larvae. These larvae pass from the slugs in mucous secretions that may contaminate food or other surfaces. Accidental human infection results from eating inadequately cleaned and/or cooked vegetables and contact with surfaces contaminated with slug mucous containing infective larvae (Morera, 1973). Experimental infections conducted in dogs have led to the suggestion that they may serve as reservoir hosts of this infection (Rodriquez *et al.*, 2002). In human infections, adult worms damage the vascular endothelium causing thrombosis and necrosis. Eggs, larvae, and excretory/secretory products produced by adult worms also contribute to inflammatory processes in infected individuals (Morera, 1988). The clinical picture is one of ileitis, often mimicking acute appendicitis (Waisberg *et al.*, 1999). Included in this picture are abdominal pain, fever, anorexia, nausea, and vomiting. Continued low-grade fever and pain may persist for several weeks and infections may become chronic. Leucocytosis and eosinophilia are prominent features of infection (Morera, 1988) and as for *A. cantonensis*, the clinical diagnosis often depends on the clinical picture being tied to a history of travel to an endemic area. Ectopic localization of adult worms and eggs has also been seen in children, leading to visceral larval migrans-like symptoms (Morera *et al.*, 1982). Sensitive ELISA serologic tests do exist, but they lack high specificity (Geiger *et al.*, 2001; Hulbert *et al.*, 1992). An outbreak involving 22 cases of abdominal angiostrongyliasis was linked epidemiologically to the consumption of mint used in preparing ceviche in Guatemala (Kramer *et al.*, 1998). This was the first instance linking a specific food item to this disease and points to the importance of thorough washing or cooking of all food items that may have been contaminated by slug secretions. Cases of angiostrongyliasis have been reported from the United States but they are rare and usually linked to travel to an endemic region (Wu *et al.*, 1997). The parasite, however, is enzootic in Texas cotton rat populations (Ubelaker and Hall, 1979). Therapy for this infection is also controversial since it may result in worm migration that could aggravate lesions (Waisberg *et al.*, 1999). A laparotomy may have to be performed to rule out suspicion of neoplasms. In severe cases, surgery may have to be performed. Preventive measures include thorough cooking of mollusk hosts and foods that may have been contaminated by slug secretions. Rodent control might be helpful, but is frequently difficult to maintain in endemic areas.

6.5 *GNATHOSTOMA* SPP.

Gnathostoma spp. normally occur as parasites in the stomach of carnivorous mammals. Human disease may result from the ingestion of an infected fish or other intermediate host or from ingestion of infected paratenic hosts that have not been adequately cooked (Rojekittikhun *et al.*, 2002). The highest prevalence of disease is seen in several areas of Asia, but recently gnathostomiasis has been reported with increasing frequency in Mexico and other parts of South America (Miyazaki, 1966; Nawa, 1991; Pelaez and Perez-Reyes, 1970; Rojas-Molina *et al.*, 1999). In this life cycle, adults live in the stomach of infected hosts and pass eggs which embryonate

in water. Free-swimming first stage larvae are then ingested by copepods where they mature to second stage larvae in the body cavity. Fish or other hosts then ingest these first intermediate hosts. In these hosts, the parasites develop into the third stage infective larval form in the musculature or connective tissue. The final host acquires the infection by eating infected fish or other second intermediate or paratenic hosts (Miyazaki, 1954). Humans behave like paratenic hosts in that consuming raw or undercooked infected foods leads to no further development of the larvae. This is true whether the consumed host is intermediate or paratenic. More than 125 species of hosts serve as second intermediate hosts and more than 35 species of paratenic hosts are known. These include crustaceans, fish, amphibians, reptiles, birds, many mammals, and man (Miyazaki, 1966; Rusnak and Lucey, 1993). One of the interesting aspects of this life cycle is that many hosts seem to have the ability to serve as both intermediate and paratenic hosts. In man, the larvae tend to migrate through subcutaneous tissue or other organs, producing symptoms dependent on site location. Symptoms may develop within 24 h of larval ingestion and include many of the common indications of intestinal upset. Eosinophilia accompanied by cutaneal or visceral migrans symptoms are also seen (Crowley, 1995; Rusnak and Lucey, 1993). Cutaneal gnathostomiasis, which is frequently observed, is likely to manifest as a creeping eruption commonly associated with hookworm infections (Magana *et al.*, 2004). Visceral gnathostomiasis is usually more severe with symptoms dictated by the site location of migrating larvae (Rusnak and Lucey, 1993). Infections in humans involving persistence of juvenile worms for up to 10 years in humans have been reported (Taniguchi *et al.*, 1991). Outbreaks of disease involving consumption of ceviche or raw fish have recently been reported in Myanmar and Mexico (Chai *et al.*, 2003; Diaz Camacho *et al.*, 2003). Rarely are cases seen in the United States and Europe, but when reported, they are likely the result of travel to endemic countries where raw or undercooked fish was consumed (Menard *et al.*, 2003). Clinical presentation that involves creeping eruption along with a travel history that involves consumption of raw fish are early indications of gnathosomiasis. ELISA and western blot follow-ups are also useful, but as for many parasitic infections, absolute confirmation usually depends on worm isolation and identification (Rojas-Molina *et al.*, 1999). Most anthelminthic treatments have proven ineffective (Ruiz-Maldonado and Mosqueda-Cabrera, 1999), although albendazole may cause worms to migrate to the skin where they can be extracted (Ogata *et al.*, 1998). In the absence of success using chemotherapy, surgical removal would be the treatment of choice (Feinstein and Rodriguez-Valdes, 1984). In the context of many other parasite problems, this is clearly a localized emerging infection. Cultural food practices no doubt impact disease prevalence in certain areas. Indigenous peoples and travelers to endemic areas need to be educated to the threat and better understand good cooking and eating practices.

6.6 *GONGYLONEMA* SPP.

Gongylonema pulchrum has been the main species associated with the 50+ human infections reported in the literature (Gutierrez, 1999). This parasite normally occurs

in ruminants and swine, with man being an accidental host. Interestingly, another related parasite, *G. neoplasticum*, which normally occurs in rodents, was associated with tumor research at the beginning of the twentieth century that led to a Nobel Prize (Campbell, 1997). Adult worms live in the esophageal epithelium of their normal hosts. Eggs passed in the feces are consumed by cockroaches and dung beetles where larvae mature to the infective third stage. Infection of the definitive host occurs after ingestion of the insect host (Waite and Gorrie, 1935). As unthinkable as it may sound, it has been suggested that most human infections follow ingestion of cockroaches (Beaver *et al.*, 1984). It could also follow that infection might occur while drinking water in which larvae had been released from disintegrating insect intermediate hosts. Unbeknownst to most of us, we do consume insect parts on a regular basis, and the USDA even has guidelines for allowable numbers of insect parts, eggs, etc., that can be present in food (http://www.cfsan.fda.gov/~dms/dalbook.html). In case report studies on human infections, the patient usually complains of sensations associated with something moving in the skin around the mouth area. Worms can be extracted following local anesthesia and albendazole therapy is usually prescribed (Eberhard and Busillo, 1999; Wilson *et al.*, 2001). In most cases, patients do not recall knowingly ingesting insects and in many there may have been travel to areas where sanitation might have played a role in transmission of this disease. In the cases reported from the United States the majority occurred in individuals living in southeastern states, although a few occurred in individuals from large northeastern cities (Wilson *et al.*, 2001). It is obvious that risk factors for acquiring this disease are not fully understood.

6.7 OTHER NEMATODE INFECTIONS WITH FOOD-BORNE ASSOCIATIONS

Nematodes, as a group, cause more human infections that any other group of parasites (Cromptom, 1999; Stoll, 1999) and the Disability Adjusted Life Years (DALYs) lost due to the three major ones (Ascaris, Trichuris, and the hookworms) surpass that due to malaria infections (Chan, 1997). While this chapter has primarily focused on nematode infections acquired directly from food sources, it would be a mistake to neglect infections that may be acquired indirectly from food or via other non-conventional routes considered to be food sources. In the latter category could be included potential transmammary routes of infection as have been suggested for *Ancylostoma duodenale* (Navitsky *et al.*, 1998; Nwosu, 1981; Prociv and Luke, 1995) and *Strongyloides fuelleborni* (Brown and Girardeau; 1977). While larvae of these parasites were not found in mothers' milk, the frequency of infection in mothers and their nursing children highly suggests lactogenic infections. It also has to be kept in mind that nematodes are very prodigious egg producers and that in many parts of the world, human feces and raw sewage is used as fertilizer to grow crops for human consumption (Cai *et al.*, 1988; Choi and Chang, 1967; Needham *et al.*, 1998). In such instances, it is certainly feasible for eggs to end up on vegetables that if consumed raw or poorly cooked could contribute to a substantial number of human infections. One also has to consider the role of flies as transport hosts in disseminating helminth eggs to food that people might consume. Flies have recently

gained a good deal of notoriety in transmitting protozoan infections (Graczyk *et al.*, 1999a, 1999b), but their role in the transport of helminth eggs requires study. Lastly, there was a recent report on the zoonotic risk of *Toxocara canis* infection through consumption of pig or poultry viscera (Taira *et al.*, 2004). While this study was an assessment of the potential risk using pigs as a model, it does point to the possibility that human infection could occur via ingestion of undercooked foods from these sources that may harbor *T. canis* larvae. As we gain knowledge of more of these infections and as more epidemiological studies are undertaken to determine where they come from, it is likely we will become more respectful of food-borne nematode infections.

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