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Internal Parasites

Helminths

Matthew T. Brewer and John H. Greve

Introduction

Internal parasites are common pests of swine worldwide. Controlled trials with nematode infections consistently demonstrate a reduction in average daily gain (ADG) and increase in feed to gain ratio (F/G) of infected pigs compared with their controls. In addition, internal parasites can generally compromise vigor and may act synergistically with other endemic potential pathogens. The extent of these losses depends on the quality of nutrient intake, type of housing, climate in the geographic area concerned, genetics of the swine, and veterinary costs associated with these infections. Premises heavily contaminated with thousands of infectious eggs or larvae from previous endemic infections can cause severe disease when encountered by a naïve animal. More often, subtle subclinical infections lead to insidious production losses that are substantial over time. This chapter addresses helminths, whereas ectoparasites and protozoa are addressed in Chapters 65 and 66, respectively.

Historically, schemes for parasite control incorporate sanitation, anthelmintics, and management practices aimed to reduce condemnations and production losses. While modern swine production facilities have decreased parasitism by denying pigs access to soil and/or parasite intermediate hosts, some helminths still persist due to their high fecundity and environmental stability. For example, despite the availability of efficacious anthelmintics, *Ascaris suum* remains as one of the most prevalent swine parasites throughout the world. On the other hand, pigs raised with access to soil continue to serve as hosts for a variety of internal parasites. Recent resurgence of extensive or outdoor production practices offers risk for reemergence of parasite concerns. Application of specific management practices from the last century and previous editions of this book may have merit, particularly because effective alternatives to licensed anthelmintics have not been documented.

Digestive system

The digestive system offers the most convenient entry into and exit from the host; therefore a myriad of parasites have evolved using this system as their niche. The mouth is a chaotic environment and hence is not often parasitized. However, all other levels for the digestive tract harbor helminth parasites.

Esophagus

Gongylonema pulchrum, the esophageal worm, is a spiruroid nematode occurring in tunnels burrowed into the epithelium covering the esophagus and occasionally the lingual or buccal mucosa. The tunnels are usually aligned with the longitudinal axis of the esophagus and form a sine wave appearance along their length. The males are 60 mm long, and the females about 90 mm long when removed from their tunnels. Oval-shaped eggs (55–65 × 30–35 µm) are transparent, contain a developed larva, and are passed in the host's feces. Infective L3 larvae develop when the eggs are ingested by coprophagous beetles or cockroaches, and pigs become infected when they ingest the insect intermediate host. *Gongylonema* cause minor inflammation as they glide back and forth in their tunnels. Their main importance is at slaughter, where tissue is trimmed if the lesion is discovered. Ruminants and humans are also susceptible to *G. pulchrum*, but they must ingest the intermediate host insect to become infected, so trimming of affected tissue is for appearance sake.

Stomach

Five nematode genera occur within the stomach. One, *Hyostrongylus*, is moderately common, but the other

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four (*Ascarops*, *Physocephalus*, *Gnathostoma*, and *Simondsia*) are less common and limited geographically.

Hyostrongylus

Hyostrongylus rubidus, the red stomach worm, is a trichostrongyloid nematode occurring unattached on the mucosa of the lesser curvature of the stomach. Adults are the width of a hair and less than 10 mm in length. The eggs have typical strongyle structure (ovoid, thin shelled, transparent, 60–76 × 30–38 µm) containing the 16- to 32-cell stage when laid. These strongyle eggs resemble those of *Oesophagostomum* and *Globocephalus*; their differentiation requires careful measurement of eggs and harvest of infective ensheathed L3 larvae from incubated fecal cultures (Honer 1967). Eggs are passed in the feces and develop to infective larvae in about 7 days. The larvae migrate away from feces and onto grass where they are subsequently ingested by swine. Thus, the life cycle is direct, and hyostrongylosis is a disease of pastured swine. The ingested larvae enter gastric glands, undergo two molts, and reemerge into the gastric lumen. Some larvae may remain in the gastric glands in a state of hypobiosis, causing nodular distension of the affected glands. Hypobiotic larvae may enter the gastric lumen at a later time and mature into adult worms.

Although *H. rubidus* is reported from many areas of the world, little is known about the pathogenicity of this parasite. They suck a small amount of blood, leading to catarrhal gastritis and potential mucosal erosion. *H. rubidus* may be a factor in the pathogenesis of gastric ulceration, and these changes in the gastric mucosa can impact feed conversion and weight gains (Stewart et al. 1985).

Spiruroid stomach worms

Other stomach worms, such as *Physocephalus sexalatus*, *Ascarops strongylina*, *Gnathostoma spinigerum*, and *Simondsia paradoxa,* are spiruroid nematodes. They are stouter in appearance than *Hyostrongylus* and approximately 20 mm in length. The adults are attached by mouth to the mucosa, but this attachment causes no visible damage except for excessive mucus production. Female *Simondsia* have anterior ends that enter the gastric glands so that only their bulbous posterior is visible.

The life cycles of these four spiruroids, so far as is known, are all similar to one another. The typical spiruroid eggs (thick shelled, transparent, ovoid, contain a larva) pass in the feces where they are ingested by coprophagous beetles. The eggs (30–40 × 15–20 µm) resemble, but are slightly smaller than, eggs of *Gongylonema* (55–65 × 30–35 µm). Infective L3 larvae develop in beetles that are subsequently ingested by pastured swine.

Small intestine

Strongyloides

Strongyloides ransomi, the minute threadworm, is a rhabditoid nematode that has a cosmopolitan distribution but is more rarely present in modern indoor swine production facilities. It is particularly pathogenic in suckling pigs and has a greater importance in tropical and subtropical climates.

These minute (3–5 mm long) hairlike nematodes live embedded in the epithelium of the small intestine. Parasitic females are parthenogenetic and lay thin-shelled, transparent, embryonated eggs that are shed in feces. Rhabditiform larvae hatch in a few hours and may progress to either infective third-stage female filariform larvae (homogonic cycle) or free-living rhabditiform males and females (heterogonic cycle). The offspring of the free-living heterogonic cycle may become either rhabditiform free-living individuals or filariform infective parasitic females. Male individuals are not parasitic. Factors that determine whether individual nematodes undergo heterogonic or homogonic development are studied but poorly defined, with environmental factors such as availability of food and moisture suspected to influence which developmental pathway is taken.

Infective larvae typically infect the next host by penetration of the skin or the oral mucosa to gain access to the capillaries and carried by the bloodstream to the lungs where they are coughed up and swallowed (tracheal migration). This process results in a patent infection in 6–10 days. The most important route of infection in neonatal pigs is transcolostral (Moncol 1975). In sows, larvae accumulate in a hypobiotic state in the mammary fat until the time of parturition, whereupon the larvae become mobilized, enter mammary alveoli, and are shed in colostrum and milk. *S. ransomi* acquired by the lactogenic route produce a patent infection in 2–4 days. It is thought that piglets may also acquire larvae transplacentally; however, the lactogenic route is probably the most important. Piglets separated from their dam at birth are worm-free as opposed to nursing littermates.

Lesions are dependent on the number of infective larvae acquired and on the host's resistance. It is not uncommon to find a small number of *Strongyloides* without associated lesions. However, the adult nematodes are so small that they may be difficult to identify amid ingesta. Heavy infections in juvenile animals can lead to poor rate of gain, hemorrhagic diarrhea, and death. Immunity develops following exposure to larvae and is typically age related.

Diagnosis of patent infections is by observation of embryonated (larvated) eggs in fecal flotation procedures (Figure 67.1). However, these larvated eggs must be differentiated from other parasites such as spiruroids. This can be accomplished by culturing eggs to allow

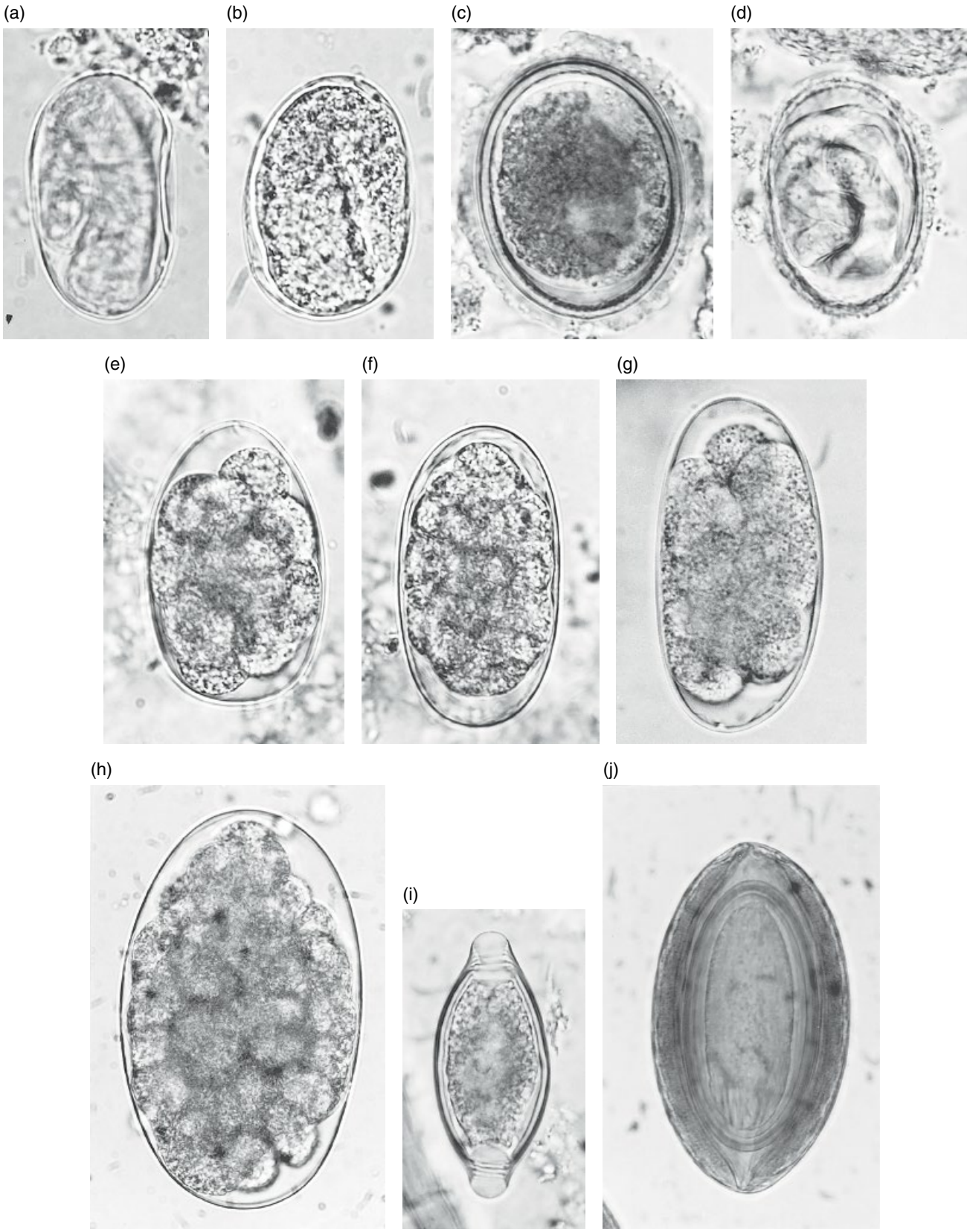

Figure 67.1 (a) *Strongyloides* egg, thick shelled, lacking one of three layers, and larvated. (b) *Ascarops* egg, larvated and similar morphologically to those of *Physocephalus* and *Gongylonema*. (c) The *Ascaris* egg has an outer proteinaceous layer, often missing. (d) *Metastrongylus* egg. (e) *Oesophagostomum* egg. (f) *Hyostrongylus* egg. (g) *Globocephalus* egg. (h) *Stephanurus dentatus* egg passed in the urine. (i) *Trichuris* egg. (j) *Macracanthorhynchus* egg. (All eggs photographed and printed at the same magnification.)

hatching and observation of rhabditiform L1 larvae. Adult *Strongyloides* may be found in mucosal scrapings, but the adults are very small (3–5 mm) and may be confused with larval stages of other nematodes. Squash preparations revealing typical eggs help address this problem as *Strongyloides* larvae do not have eggs.

Ascaris

Ascaris suum is the large roundworm and most cosmopolitan parasite of swine. *Ascaris* has persisted despite decades of pharmaceutical intervention and modern confinement systems. *A. suum* is a close relative to the human ascarid, *Ascaris lumbricoides*, and it is proposed that these two nematodes are a single species. *Ascaris* isolated from pigs is capable of infecting humans, and recent analysis of nuclear and mitochondrial genes from worms obtained from pigs suggests that human, swine, and hybrid genotypes are present (Jesudoss et al. 2017). If swine have access to soil, one can assume they are infected with *Ascaris* because of the ubiquitous nature of the nematodes and the extreme longevity of the eggs in the environment. Producers often assume that pigs are infected and administer anthelmintics, even without specific proof.

Ascaris suum adults can be easily observed at necropsy due to their large robust size (Figure 67.2): females are up to 40 cm in length, and males can be up to 25 cm. Adults live unattached in the lumen of the small intestine, swimming against peristalsis. It is proposed that *A. suum* maintains its position in the intestine by pushing up against opposing sides of the lumen. Adults mate and produce eggs that are thick and barrel shaped (50–80 × 40–60 µm). The colorless thick shell is coated with a sticky, brownish, mammillated proteinaceous layer and contains a single large cell. Females shed thousands of eggs over their lifetime of approximately 6 months. The eggs are resilient and long lived, so the environment is likely to be heavily contaminated wherever swine exist.

Life cycle

The life cycle is direct, with eggs passing in feces and developing infectivity over a period of approximately 3–4 weeks. The infective larva remains inside the protective egg, sheltered from potentially lethal environmental extremes until the egg is ingested. After ingestion, larvae hatch from the egg, penetrate the jejunal wall, and are carried by portal circulation to the liver. A few larvae may be found wandering in the mesenteric lymph nodes, peritoneal cavity, and elsewhere, but such wandering larvae probably do not complete their life cycle. Most larvae reach the liver 1–2 days postinfection and are then carried to the lungs via the blood 4–7 days postinfection. After molting and spending a few days in the lungs, larvae exit the pulmonary capillaries, breaking into the airways. Larvae are coughed up, carried to the pharynx by the mucociliary escalator, and then swallowed. Adult nematodes can be found in the small intestine 10–15 days after ingestion of infective eggs. Oviposition begins around 6–7 weeks after infection.

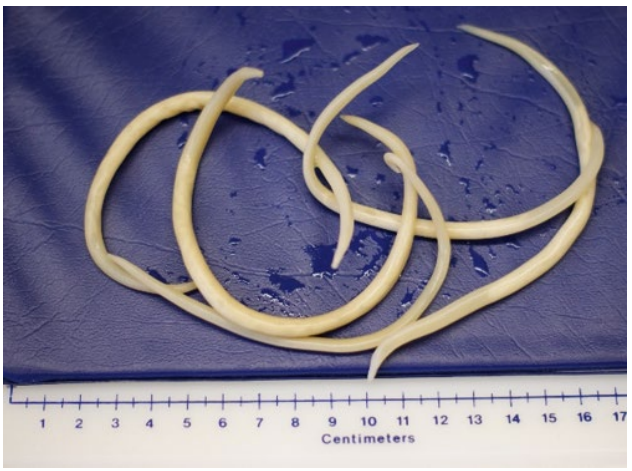

Figure 67.2 *Ascaris suum* adults removed from the intestine of a pig.

Eggs are very resistant to temperature extremes and may remain infective for years. Most chemicals have no effect on eggs, but steam may affect their viability. Because of the sticky outer layer of the egg, eggs are easily transported by boots, insects, and other means. It is thought that most piglets are exposed when they ingest eggs in manure stuck to the sows' udder.

Senescent ascarids are expelled in feces, but swine may continue to carry a small number of worms for several months. Ascarids often occur in young pigs, and by the time swine are 5–6 months old, they are often resistant to infection due to previous exposure to migrating larvae and/or by reason of age resistance. Pigs previously exposed mount an immune response, leading larvae to be trapped in foci of granulomatous inflammation. The larvae of *A. suum* are aggressive migrators and can cause visceral larva migrans in accidental hosts. The migrating larvae may cause serious verminous pneumonia or other inflammatory disease due to a prolonged migration or larval death, for example, in cattle housed in facilities previously dedicated to swine.

Pathology

Adult ascarids compete with the host for nutrients and probably interfere with nutrient absorptions as they browse on the tips of the villi, thereby causing insidious production losses. Adult ascarids swim against peristaltic waves to maintain presence and can occasionally wander up and occlude the bile duct or even approach the liver capsule. It has been hypothesized that bile duct occlusion can occur postmortem; however, ascarid occlusion of the bile duct should be considered as a potential cause of icterus in swine.

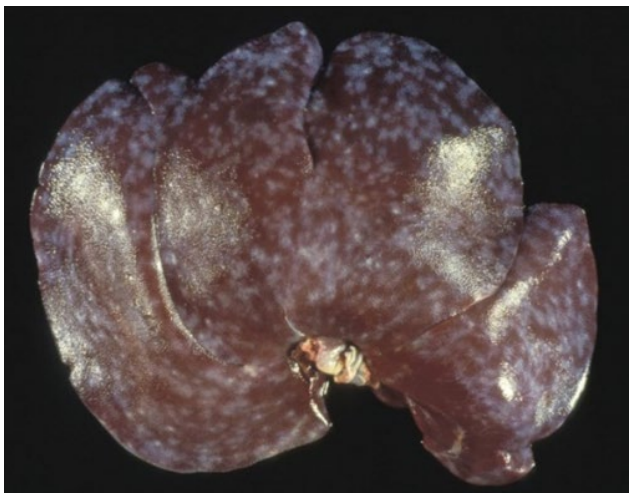

Figure 67.3 Multiple foci of fibrosis, the result of ascarid larval migrations ("milk spot liver").

In contrast to adult worms, the pathology damage associated with the migrations of larval stages of *A. suum* to the liver and lungs is substantial. Repeated waves of migrating larvae lead to an exuberant inflammation and immune response against larval antigens. In the liver, larvae migratory paths are infiltrated by eosinophils and fibrotic connective tissue that appears grossly as "milk spots." Lesions in the liver become visible 7–10 days postinfection and can expand or be variable in size if continued migrations occur. In low infestations, the "milk spots" can regress within 25 days if the liver does not experience further larval insult (Figure 67.3). Severe and ongoing infections result in diffuse liver fibrosis. Liver enzymes may be increased as a result of infection. In the lungs, larval migration is associated with petechial hemorrhages due to larvae breaking out of the capillaries and into the alveoli. Interstitial pneumonia, bronchiolitis, and alveolar edema also occur in the areas of larval migration. In mild cases, pigs are asymptomatic; however, more severely affected pigs have a characteristic expirational abdominal lift known as "thumping," sometimes with increase in cough. Most pigs become infected early in life, with immune mitigation of clinical signs. If naïve pigs are 20 kg or larger before first exposure, the pulmonary reaction may be extreme with life-threatening clinical signs similar to those of respiratory disease complex. Indeed, ascarid larval migrations may compromise the lung and exacerbate impact of other endemic viral and bacterial pathogens in the herd. The ingestion of infective eggs often occurs at low level over time, so lesions of larval migration can occur simultaneously with the presence of adults, which will continue until an immunological response develops.

Diagnosis

The diagnosis of *Ascaris* is straightforward when dealing with patent infections since the prodigious oviposition rate of the female makes eggs easy to find by standard flotation methods. At necropsy, the presence of milk spots in the liver is evidence that the animal has been infected with ascarid eggs within the past month. Early punctate lesions gradually expand in size before resolving in about 30 days. In areas where the swine kidney worm, *Stephanurus dentatus*, is present, the milk spots due to ascarid migration must be differentiated from fibrosis associated with kidney worm migration. Scars of the latter are more extensive with similar lesions occurring in other organs such as the lungs and kidney. Milk spots may also be caused by other helminths such as *Toxocara* undergoing visceral larva migrans in swine (Helwigh et al. 1999). Adult *Ascaris* are usually found in the small intestine at the time of necropsy or slaughter, but occasionally are found wandering in other portions of the gastrointestinal tract. The adults are large, often 20–30 cm in length and the diameter of a pencil with three large lips. Adults can be visible or palpable through the wall of the intestine.

When naïve pigs are exposed to egg-contaminated premises, morbidity or mortality can occur, while the infection is in the prepatent phase in which case fecal flotation is of no diagnostic value. Diagnosis is by observation of abundant milk spot lesions at necropsy. Careful examination may reveal small immature ascarids in the jejunum. Larvae migrating in lungs are difficult to observe grossly, but histopathology can offer a tentative diagnosis. Larvae can be collected by suspending snips of lung tissue in water. The motile larvae will migrate out of the lung tissue but are not strong swimmers, so they sink and can be collected with a funnel and tubing system similar to the Baermann apparatus often used to detect lungworm larvae in other domestic animal species.

Economic importance

Numerous studies have detailed economic importance of various aspects of ascarosis in swine. The results of experimental infections demonstrate some compromise of ADG and feed efficiency, even at low infection levels. *Ascaris* infection may also decrease the host response to vaccination against other pathogens. Metabolic studies show detrimental effects on nitrogen metabolism during the rapid growth phase of ascarids, about 1 month postinfection. At slaughter, there are losses due to condemnation or trimming of livers as well as condemnation carcasses due to icterus. The monetary value of these losses is enormous, though difficult to quantify. Historical estimates of losses are in the hundreds of millions of dollars in lost revenue annually (Stewart and Hale 1988). *A. suum* has continued to persist in commercial swine; a

visit to a slaughterhouse confirms the presence of these parasites on a daily basis.

Trichinella

Trichinellosis occurs in most mammals, including humans. The life cycle of this parasite is confusing as the adult nematodes live in the intestine and are seldom seen. Detection efforts focus on the larvae that live within muscle and cause disease. *Trichinella* has been found to be a diverse genus, but most cases of trichinosis are caused by *Trichinella spiralis*. Molecular characterization has revealed potential of at least eight different species in the genus with several genotypes that may achieve species status (Gottstein et al. 2009). *T. spiralis* has a global distribution, is a swine-adapted species, and is often detected in rats. *Trichinella britovi* can be found in pigs in Europe, Asia, and Africa and grouped in a single clade because they induce a thick collagen capsule during the muscle phase of infection; other species produce a capsule only detectable by electron microscopy. Examples of non-encapsulating species infecting pigs are *Trichinella pseudospiralis*, which has a cosmopolitan distribution, and *Trichinella papuae*, which can be found in Southeast Asia. Reduced incidence of *Trichinella* infections is attributed to regulation of garbage feeding to swine, public health programs, and improved detection techniques. In developed countries, trichinosis cases are now more often associated with ingestion of poorly cooked wild game meat.

Life cycle

Adult *Trichinella* are tiny (2–4 mm long) nematodes that are short lived and typically never encountered. The adults tunnel within the villi of the intestine. Within 5 days after mating, the viviparous females continuously deposit larvae in the lamina propria for duration of life, which is 2–3 weeks. The larvae enter the circulation and are distributed throughout the body until opportunity to penetrate the sarcolemma of skeletal muscle cells. After entering the myocyte, the myocyte becomes a "nurse cell" that supports the quiescent larva for months or years (Figure 67.4). Circulating larvae that do not enter myocytes eventually are arrested within granulomas. When cysts from the muscle are ingested, the larvae excyst and develop into adults within 48 hours. Examples of transmission within swine herds include tail biting, scavenging (rats, raccoons, etc.), and eating of garbage containing uncooked meat.

Pathology

The intestinal adults may be associated with enteritis although this is not typically observed clinically. Pathology and disease is associated with the larvae in the nurse cell. As the nurse cells form, there is malaise, pyrexia, and myalgia accompanied by eosinophilia.

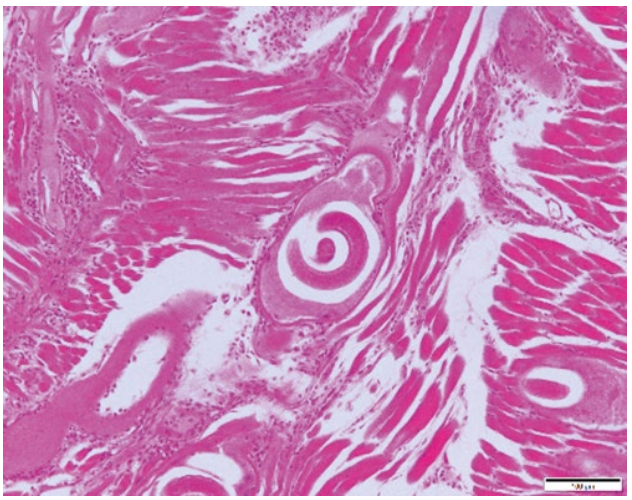

Figure 67.4 *Trichinella* larvae in a nurse cell.

Growth rates may be reduced, but the disease is often undetectable. Ten larvae per gram of body weight are thought to be a lethal infectious dose in a pig. As the nurse cell forms, the myocyte is walled off by collagen. Once the development of the nurse cell is complete, clinical signs regress, and normal growth rates resume.

Diagnosis

Traditional methods of diagnosis are aimed at finding nurse cells, which are not distributed homogeneously throughout the musculature but tend to be concentrated in particular muscle groups (diaphragm, extrinsic muscles of the eye, and muscles of posture). Both of the two main methods for detecting muscle cysts are labor intensive and produce false negatives. The first is by microscopic examination of bits of muscle compressed between glass plates, and the other is by digestion of several grams of muscle in artificial gastric juice (1% pepsin, 1% hydrochloric acid at 37 °C) and microscopy of sediment. These techniques have been largely replaced by a serum ELISA that detects pig antibodies directed at a larval secretory antigen (Murrell et al. 1986). False-negative ELISA results can occur when low populations (<5 larvae/g of muscle) of parasites are present.

Public health

Trichinella are zoonotic, infecting humans as well as other mammals. The source of *Trichinella* varies but can include pork products. Ground pork or sausage is particularly of concern because a single infected carcass may find its way into numerous sausages or can be used to dilute venison or beef sausage. In recent years, more cases of human trichinellosis in the United States have been caused by eating undercooked bear, wild game meat, or home-slaughtered pork. The incidence of swine trichinellosis in the United States has varied from 0.1 to

0.3% since the mid-twentieth century because the use of control measures (prohibition of feeding raw garbage, raising pigs in confinement, docking of tails, etc.) has practically eliminated trichinellosis in commercial pork. In the United States, there is a voluntary National Trichinae Certification Program aimed to continued efforts to eliminate trichinellosis.

Trichinella larvae cooked to a uniform temperature of 60 °C are not infective; therefore the US Department of Agriculture recommends that fresh pork be cooked to an internal temperature of 63 °C, allowing it to rest for 3 minutes between cooking and cutting. This temperature includes a safety factor that allows for variation in cooking methods (e.g. microwave ovens do not heat evenly). Freezing of fresh pork products less than 15 mm thick at −15 °C for 20 days or at −29 °C for 6 days will kill most larvae, but not isolates adapted to cold (e.g. *Trichinella nativa*, which is not a problem in commercial pork industry). Salt curing does not reliably kill larval *Trichinella*, so cured products should be cooked.

As a result of public health and veterinary food safety efforts, the number of cases of human trichinellosis in the United States has dropped from 450 cases per year in 1947 to about 12 cases per year 50 years later (Kennedy et al. 2009), and similar results have been achieved elsewhere. *Trichinella* is still present at a low level in swine as well as in wildlife in some regions, which is justification for continued vigilance.

Hookworm

Globocephalus urosubulatus is distributed throughout the world, but in North America it is limited to swine pastured in southern states. Adults attach to the jejunal mucosa by way of a large buccal capsule that lacks teeth or cutting plates. Young pigs are more likely to become anemic than older, more resistant individuals. However, clinical disease attributed to hookworms in pigs is rare. Adult *Globocephalus* are about 7 mm in length and lay strongyle-type eggs (52–56 × 25–35 µm) similar to those of *Hyostrongylus*. Infective larvae develop in the environment and infect other pigs by ingestion or skin penetration. Geographic distribution is limited to areas that do not freeze. Unlike hookworms infecting dogs and cats, *Globocephalus* is not associated with cutaneous larva migrans in humans.

Tapeworms

Although the swine gut is home to a myriad of helminth parasites, they are not typically definitive hosts for the adult stages of cestodes (tapeworms). Swine can, however, be important cestode intermediate hosts for *Taenia* and *Echinococcus* as discussed elsewhere in this chapter.

Acanthocephalans

Acanthocephala ("thornyhead") is a phylum distinct from Nematoda, characterized by parasites with protrusible proboscis. *Macracanthorhynchus hirudinaceus* is a large acanthocephalan up to 40 cm in length and has a dusty coral pink color, and the anterior end has a spiny proboscis with which it attaches to the jejunal wall. Its body may be turgid or wrinkled and flattened depending on hydration status and hence may be mistaken for an ascarid or a cestode. Ascarids lack a proboscis and do not attach, while cestodes have true segments (not mere wrinkles) and have a scolex that is not as dramatic as the proboscis of an acanthocephalan.

Eggs of *M. hirudinaceus* (70–100 × 40–65 µm) are passed in the hosts' feces and have a three-layered shell that is almond shaped, is brown, and contains a larva (acanthor). The acanthor has an ellipsoidal shape and needlelike hooks on one end. When ingested by white grubs of certain beetles (June bugs, dung beetles, etc.), an infective larva (cystacanth) develops in about 3 months, surviving metamorphosis to be present in the adult beetle. Pigs are infected by ingesting a grub or adult beetle while rooting. Once ingested, the cystacanths mature into adults in the small intestine, mate, and lay eggs after about 2–3 months. Since commercial swine are generally raised in confinement that denies pigs access to intermediate host, acanthocephalans have become rare. There are a few reports of *M. hirudinaceus* in humans in cultures that eat raw beetles. Because the life cycle is indirect, there is no danger in handling eggs or tissues of the adults.

Macracanthorhynchus hirudinaceus inserts its proboscis into the wall of the jejunum. The proboscis is large and capable of penetrating through the entire thickness of the intestinal wall, resulting in a perforation. This lesion may be encapsulated with nodular fibrous connective tissue that can be seen at necropsy, or they may rupture causing peritonitis. There are typically more nodules than adult worms, suggesting that release and reattachment may occur. Clinical disease associated with acanthocephalans is not reported often, but intestinal perforation may lead to abdominal pain, diarrhea, fever, and emaciation.

Large intestine

Two kinds of nematodes occur in the cecum and colon of pigs: nodular worms and whipworms. Both are very common, and both can lead to important clinical disease.

Whipworm

Trichuris suis, the swine whipworm, occurs in the cecum and colon. Much like ascarids, whipworms have persisted as an important parasite of pigs raised in confinement

facilities. Adult *Trichuris* are approximately 60 mm long, and about two-thirds of their length consists of a filamentous esophageal portion of the body that is stitched into the mucosa and not readily noticed. The thick posterior portion of the body protrudes from the mucosal surfaces. Intact nematodes are difficult to collect at necropsy because the delicate, long esophageal portion breaks. The eggs are thick shelled and lemon shaped (50–58 × 21–35 µm), each containing a single large cell. The egg shell is smooth and brown with a translucent plug filling an opening at the poles of each egg.

Life cycle

The life cycle of *Trichuris* is direct. Eggs pass in the feces, and once outside the body the first-stage infective larva develops within the egg in 3–4 weeks. The infective larva remains within the egg until ingested by a pig. Eggs can remain viable for several years in the environment. After ingestion, the plugs dissolve, perhaps stimulated by fimbria of intestinal bacteria. The first-stage larva penetrates the mucosa of the colon and undergoes four molts over a period of about 2 weeks. The posterior of the whipworm's body extends out into the lumen, and oviposition begins 6–7 weeks postinfection, while the worm's life span is approximately 4–5 months (Beer 1973).

Pathology

Low populations of adult *Trichuris* cause minimal lesions, although they may provide an entry point for other pathogens. Heavy infections with *Trichuris* are associated with ulceration of the mucosa, mucosal edema, and hemorrhage. Much of this tissue damage is caused by the histotrophic larvae, before adults are grossly present. Clinical signs include diarrhea, hematochezia, and rectal prolapse.

Diagnosis

The presence of adult *Trichuris* can be confirmed by demonstrating the characteristic eggs in fecal flotations; however, false negatives may occur due to sporadic egg laying by females, as well as the long (6–7 weeks) prepatent period. Adult worms can be detected at necropsy but are often overlooked due to their small size. Often, the most severe clinical signs, including dysentery, are produced by migrating larvae that may not be grossly visible for the first 3 weeks after infection. The tiny larvae and immature worms can be found in mucosal scrapings or recognized by histopathology. Whipworms can be recognized by their unusual esophageal structure of a single line of large glandular cells known as a stichosome. The uterus of a mature female contains eggs of typical morphology often visualized by histology. Commercial *Trichuris* antigen detection tests have been developed for use in other domestic animal species, but their utility has not yet been proven for *T. suis*.

Nodular Worms

Nodular worms (*Oesophagostomum* spp.) are common although less prevalent in swine raised indoors. These strongyloid nematodes inhabit the mucosal surface of the cecum and colon. Adults range from 8 to 15 mm in length, and males possess a copulatory bursa. There are several species, but their appearance and life cycle is similar. Eggs are typical strongyle eggs (70 × 40 µm) containing a morula stage.

The life cycle is direct; eggs passed in feces develop into first-stage larvae that hatch from the egg and molt twice in the environment to produce an infective third-stage larva that is ingested by swine. Infective larvae retain their last molted cuticle as a sheath, so they are moderately resistant to environmental extremes and can survive for several months or more. Ingested larvae exsheath and enter the mucosal glands of the ileum, cecum, and colon. They penetrate into the lamina propria, molt, and remain for about 2 weeks. They emerge into the intestine to become adults, and eggs begin to appear 3–6 weeks after infection.

Adults cause minimal damage to the mucosa and do not cause much in the way of clinical signs. The migration and molting of larvae in the lamina propria cause the characteristic nodule that is usually small (about 2 mm) but may be much larger, especially in animals with repeated infections. The larvae may remain within the nodules for several weeks before escaping into the lumen.

Antemortem diagnosis of nodular worms depends on finding the eggs in fecal flotations. However, this is complicated by the fact that eggs of *Hyostrongylus* and *Globocephalus* resemble them. For definitive identification, larval culture to the L3 stage is needed. At necropsy, the finding of nodules in the cecum and colon is suggestive but must be differentiated from other causes of inflammation, diverticulitis, or abscessed lymphoglandular complexes.

Respiratory system

Lungworms

Metastrongylus spp. have a worldwide distribution, with *Metastrongylus apri* being the predominant species, but mixed infections are common. The adults are slender, 40–50 mm in length, and occur in the bronchi and bronchioles, usually in the diaphragmatic lung lobes. Masses of entwined worms covered with mucus may occlude the peripheral airways. The eggs bear a thick rough coat, are colorless, contain a larva, and measure 50–60 × 35–40 µm.

The life cycle is indirect. Eggs are coughed up, swallowed, and passed in the feces. Certain earthworms, notably *Eisenia* and *Allolobophora* spp., ingest the eggs. Larvae hatch and invade the earthworm's tissues (calciferous

glands, heart, dorsal blood vessel, and crop). When swine eat the earthworm, the lungworm larvae migrate to the lungs via the lymphatic system and begin laying eggs in 4–5 weeks. Clinical signs are not pronounced, but heavy infections and infections complicated with bacterial infections cause coughing and "thumping."

Lungs with metastrongylosis have wedge-shaped areas of emphysema or atelectasis, usually at the tips and about midway along the length of the diaphragmatic lobes where major bronchi approach the lobe periphery. Often, hypertrophic bronchial muscle, hyperplastic epithelium, and nodular lymphoid hyperplasia are associated with the nematodes. Concurrent bacterial pneumonia is not uncommon.

Diagnosis of metastrongylosis is accomplished by finding the characteristic eggs on flotation, but the eggs do not float well. Postmortem diagnosis is accomplished by trimming 1 cm strips from the edge of the diaphragmatic lung lobe and squeezing to express adults from the bronchi.

Lung fluke

Paragonimus kellicotti is a trematode fluke that occurs in bronchial cysts of a wide range of hosts, including swine, in North America. *Paragonimus westermani* is a similar species occurring in Southeast Asia and a few areas of South America. *Paragonimus* are large, fleshy, brownish flukes that measure 8–12 mm long, 4–6 mm wide, and 3–4 mm thick. They are often found in pairs. The cysts are about 2–3 cm in diameter and are easily seen and palpated at necropsy. Usually there are few clinical signs because a few cysts do not compromise lung function. On the other hand, heavy infections can produce a productive cough with dyspnea and hemoptysis.

The life cycle is indirect. Miracidia hatch from the egg and penetrate the first intermediate host, a snail. Cercaria exit the snail and are consumed by the second intermediate host, which is a crayfish for *P. kellicotti* or either crayfish or crab for *P. westermani*. When eaten by a swine, the metacercariae in the crustacean excyst, penetrate the intestinal walls, burrow through the diaphragm, and enter the pulmonary parenchyma. They settle in pairs, mature, and begin oviposition in about 60 days. Pairs live in cysts that communicate with bronchi. Worms are hermaphroditic, but it is thought that they may either mate with another individual or self-fertilize. Eggs are coughed up, swallowed, and passed in feces.

At necropsy, the characteristic cysts can be found in the lungs. Adult flukes may be teased from the cysts or may be observed by histopathology. The eggs are relatively heavy but will float in a saturated sugar solution with centrifugation. The eggs are brown, vase shaped, and large (80–110 × 50–60 µm) and have a thickening at the junction of the operculum and shell. The number of eggs appearing at any one time varies because their presence in the feces depends on being coughed up and swallowed. Eggs may also be detected in smears of sputum. Some migrating excysted metacercariae wander, and adults may be found ectopically in lymph nodes, liver, peritoneum, etc.

Liver and pancreas

Aside from being the site of larval migration (e.g. *Ascaris* and *Stephanurus*), the liver and pancreas are the niches for only a few helminths.

Liver fluke

Fasciola hepatica is a cosmopolitan trematode fluke that has a broad host range that includes swine. The adult fluke is large (30 × 10 mm) and leaf shaped, with a conical anterior end. The eggs are large (130–150 × 65–90 µm), oval, yellowish brown, and operculated.

Eggs passed in the feces develop in water. A ciliated miracidium hatches and penetrates a lymnaeid snail that undergoes asexual reproduction and results in the release of hundreds of cercaria. Each cercaria encysts on vegetation to form an infective metacercaria. Metacercariae may remain infective for a long period of time but are killed quickly under dry conditions. After being ingested by the final host, the metacercaria penetrates the intestinal wall and the liver capsule and migrates in the liver parenchyma for 6 or more weeks. It eventually reaches the biliary duct system and migrates to the larger bile ducts and sometimes the gallbladder. The prepatent period is 10–12 weeks.

Migrating adolescent flukes cause necrohemorrhagic tracts in the liver parenchyma. These tracts regress once the flukes have entered the bile ducts. Adults cause bile duct hyperplasia and fibrosis, leading to the characteristic "pipestem liver" lesions that resemble the stems of clay pipes. Adult flukes and brownish exudate can be expressed if the affected bile ducts are incised. Clinical signs associated with fasciolosis are anemia and hypoproteinemia due to liver damage and hematophagia by adult flukes. Infected individuals may have weight loss or poor gains, but infections are often silent. The liver is condemned at slaughter. Ruminants and humans can also be infected. A similar fluke, *Fasciola gigantica*, infects swine in Africa and Asia.

Echinococcus (hydatid cyst disease)

Adult *Echinococcus* cestodes occur worldwide in carnivores, and their larval cysts (hydatids) occur in various herbivores and omnivores, including humans and swine. The adults are very small (3–6 mm) and typically occur

by the hundreds in the definitive host's intestine. Egg-laden proglottids are shed into the environment via feces. The eggs are identical to those of other taeniids (e.g. *Taenia*), and, when ingested by a pig, the oncosphere hatches from the egg, penetrates the intestinal wall, and enters the circulation. A large proportion becomes trapped in the liver where they nidate and develop into hydatids. Unilocular hydatid cysts (*Echinococcus granulosus*) have an external fibrous cuticle and an inner germinal membrane, while multilocular hydatids (*Echinococcus multilocularis*) have a germinal layer on both the inner and outer surfaces, which are capable of producing a more invasive lesion similar to that of a neoplasm. The germinal layers give rise to many thin-walled brood capsules, and each brood capsule contains several dozen protoscolices, often referred to as "hydatid sand," which can be visualized ultrasonographically. The end result is that numerous protoscolices are produced from each egg ingested. When ingested by a carnivore, each protoscolex develops into an adult cestode.

There are no clinical signs that imply hydatid cyst disease, and diagnosis is usually done at necropsy. Aspirates of hydatid cysts contain protoscolices that contain calcareous corpuscles and hooks. Histologically the brood capsules and protoscolices can also be identified. One challenge for diagnosis is that some hydatids developing in pigs, particularly those of *E. granulosus*, are "sterile" (e.g. contain no protoscolices), which makes for a more difficult diagnosis. Hydatid cyst disease is uncommon in the United States, but sylvatic cycles of the parasite mean that it is an ever-present threat. In areas of the world where pigs are free roaming or where offal is fed to carnivores, hydatid cyst disease is both an economic and a public health concern since humans can develop hydatids.

Urinary system

Kidney worm

Stephanurus dentatus adults occur in perirenal cysts that open by a fistula into the ureters. It is common to discover ectopic cysts in other organs such as the spleen, stomach, urinary bladder, and spinal cord. Renal parenchyma per se is seldom involved. Adult *S. dentatus* are large stout worms, measuring 20–40 × 2 mm with visible internal organs. Their eggs are typical for strongyles but can be differentiated from those of hookworms, nodular worms, and other strongyles because they are found in urine.

First-stage larvae develop in the egg, hatch, and molt twice into an infective third-stage larva. Infective larvae may survive on pasture for up to several months and can either be ingested or penetrate the skin, migrate to mesenteric lymph nodes, molt, and enter the liver via portal veins. There they migrate for several weeks, growing to a size of 5–6 cm before exiting the liver and migrating to retroperitoneal tissues where adults cluster within an inflammatory cyst that fistulates to the ureters or kidneys. The prepatent period is long (9–12 months), and adults may live for several years. Migrating larvae are aggressive and can be found in ectopic sites including the epaxial muscles and spinal column, and because early migration takes place in blood vessels, it is thought that some pigs may become infected *in utero*. Migrating larvae produce hemorrhagiconecrotic tracts wherever they go and hence produce considerable abscessation, eosinophilia, and fibrosis, including much more prominent hepatic "milk spots" than those caused by ascarids.

The infection is limited geographically to areas that do not experience severe winters, and confinement rearing has dramatically reduced the incidence of kidney worm. In systems where the problem persists, a "gilts-only" breeding system can be implemented in order to control the parasite. This measure is successful due to the long prepatent period; gilts are slaughtered prior to developing a patent infection.

Musculoskeletal system

The body musculature harbors immature stages of only a few helminths. Already mentioned above are the larvae of *Trichinella* in "nurse cells" and aberrantly migrating larvae of *S. dentatus*. In addition to these nematodes, the cysticercus of *Taenia solium* lives within muscle tissue.

Cysticercosis ("pork measles")

The larval stage (metacestode or cysticercus) of *T. solium* occurs in the skeletal muscles and myocardium of swine as a fluid-filled cyst. Prior to a complete understanding of the life cycle, this organism (stage) was named *Cysticercus cellulosae*; some authors still use this nomenclature to describe the metacestode of *T. solium*. The adult form of this cestode occurs in the intestine of humans. Although once widespread in distribution, the infection is now limited by methods of human hygiene and cooking of pork. However, human cases from endemic areas occur, and diaspora assures continued cases and public health concerns.

Taenia hydatigena, "the long-necked bladder worm," is another source of cysticercosis in swine. Unlike *T. solium*, *T. hydatigena* infects canine definitive hosts and does not develop in humans. The cysts are larger (approximately 8 cm) and found in the omentum and mesentery. The cyst bears a long armlike extension (neck) that holds the inverted protoscolex.

Life Cycle

Humans infected with adult *T. solium* shed proglottids in the feces. The egg-filled proglottids release eggs when the proglottids are desiccated or damaged as no uterine pore occurs in *Taenia*. The eggs or proglottids are ingested by swine. The oncospheres hatch from the egg, penetrate the small intestine, and are distributed throughout the body by the circulation. They become trapped in capillary beds associated with muscle tissue and develop into infective cysticerci. This condition is sometimes called "pork measles" by packers. The term measles refers to the spot that is the cysticercus. Cysticerci are subspherical, whitish, translucent, fluid-filled cysts about 1 cm in diameter. Visible through the surface is a white, 1–2 mm spot that is an inverted protoscolex that will become the scolex of the adult tapeworm when ingested. Cysts remain infective for up to 2 years, but eventually they die and undergo caseation and mineralization. Cysticerci develop in any muscle of the body but are most commonly found in the heart, psoas, masseter, tongue, and limbs. Humans can become accidental intermediate hosts and develop cysticercosis following ingestion of eggs from human feces.

Diagnosis

There are no characteristic clinical signs associated with cysticercosis. Risk factors for infection include pigs that are free roaming and have access to domestic waste and human feces. The presence of a cysticercus is revealed at necropsy or slaughter. Palpation of the tongue may reveal deeply seated cysts. Microscopy of squash preparations of cysticercus can reveal the rostellum containing hooklets; hooklets do not disintegrate despite caseation. Several ELISA assays have been developed to detect *Taenia* antigens as well as anti-*Taenia* antibodies. These assays have been applied in humans but are not regularly used for surveillance in swine herds.

Public health

Viable *T. solium* cysticerci are infective for humans, so meat inspectors search for them regularly. If inspection reveals only a few cysticerci, they may be excised and discarded. Finding several organisms requires the entire carcass be cooked at 170 °C for 30 minutes (which kills the cysts) prior to releasing for consumption; however, finding high numbers of cysticerci leads to condemnation of the entire carcass. Freezing cuts of pork is another method of killing cysticerci; the temperature and time held at that temperature depends on the size and thickness of the piece of pork. Discovery of cysticerci should prompt an investigation of how pigs are coming into contact with human waste.

Besides acting as the definitive host for *T. solium*, humans can become accidental intermediate hosts after ingesting the eggs. In humans the cysticerci have a predilection for the central nervous system; neurocysticercosis is thought to be the cause of cognitive dysfunction in humans wherever *T. solium* is endemic. Humans infected with adult *T. solium* can be treated with praziquantel. Swine intermediate hosts are not treated; elimination of the life cycle is accomplished by sanitation, treatment for infected humans, and proper disposal of human feces.

There are many other helminths reported to infect swine but are considered of lesser importance (Table 67.1).

Control of helminth parasites

Parasite control methods may be broadly categorized as preventative or therapeutic. Preventative methods focus on interrupting critical points in the parasite life cycle, whereas therapeutic approaches utilize pharmaceutical agents to remove parasites from the pig. Seldom will therapeutic approaches alone eliminate a helminth parasite from a herd.

Prevention

Those parasites requiring an intermediate host can be successfully prevented by removing pigs from contact with the intermediate host (e.g. dung beetles, earthworms, snails, etc.). Therefore, maintaining pigs on concrete will prevent infection with spiruroids, acanthocephalans, and metastrongyloids. An added benefit would also be the reduction or prevention of infection by strongyles, such as *Hyostrongylus*, *Globocephalus*, and *Trichostrongylus*, that are transmitted more efficiently under pasture conditions.

Good sanitation is a critical element for controlling parasitic infections. The major mode of transmission of internal parasites is through contaminated food, soil, or bedding with feces or urine. Since parasite eggs need moisture and warmth to develop and survive, direct sunlight or dry conditions shorten egg and larval survival. Thorough cleaning of buildings, pens, and equipment with detergent and steam is the best way to control parasite eggs and larvae. Steam penetrates cracks and crevices and kills the tiny eggs and larvae. The common disinfectants used on the farm do not kill eggs of *A. suum*.

Since parasites compete with the host for available nutrients, adequate nutrition aids in reducing the adverse effects on feed efficiency and ADG. Pigs on a good plane of nutrition are less likely to exhibit clinical parasitic disease as opposed to individuals with a negative energy balance. However, improved nutrition alone will not abrogate a serious helminth problem in a herd.

A management system in which gilts only are used as breeders has been shown to be effective in eradicating

Table 67.1 Gastrointestinal helminths of lesser importance.

	Locale	Description	Comments
Gastrodiscus aegyptiacus Gastrodiscoides hominis	Small and large intestine in Africa and southern Asia	Flaky paramphistome flukes 5 × 14 mm; operculated eggs 150 × 70 µm	Infects humans; infection acquired by ingesting metacercariae on vegetation
Fasciolopsis buski	Small intestine in southern Asia and India	Large fluke 20–75 mm long; operculated eggs 135 × 85 µm	Infects humans; cycle similar to that of *Fasciola hepatica*
Gnathostoma spp.	In nodules in gastric wall in Eurasia and Africa	20–40 mm long, spinose cuticle; eggs with one polar plug, 70 × 40 µm, brown pitted shell	Second intermediate host is small vertebrate (reptile, bird, etc.); prepatent period about 3 months
Ascarops strongylina	Mucosal surface of stomach	15–20 mm; eggs thick shelled, transparent, embryonated, ellipsoidal 30–40 × 15–20 µm	Intermediate hosts are coprophagous beetles; prepatent period 4–6 weeks
Physocephalus sexalatus	Mucosal surface of stomach	15–20 mm; eggs similar to those of *Ascarops*	Cycle similar to that of *Ascarops*
Simondsia paradoxa	Stomach mucosal glands	15–20 mm; posterior end of female bulbous and filled with eggs	Cycle similar to that of *Ascarops*
Eurytrema pancreaticum	Pancreatic ducts	Flukes 10–15 × 2 mm; eggs 40–50 × 25–35 µm; dark brown; contain miracidium	Second intermediate host is grasshopper
Clonorchis sinensis	Bile ducts	Flukes 10–25 × 4 mm; eggs 27–35 × 12–20 µm; light brown; contain miracidium	Second intermediate hosts are freshwater fishes

kidney worms. This is possible because of the lengthy prepatent period of 9 months or more. Selling breeders as soon as first litters are weaned and maintaining boars separately or replacing them with young stock will prevent contamination of the premises. Using the gilts-only breeding system was used to achieve eradication of kidney worm in less than 2 years (Stewart et al. 1964).

For some parasites, prevention is the most effective method for eliminating the organism. For example, elimination of feeding raw meat, scavenging, and cannibalism is effective in preventing the transmission of *Trichinella*. Similarly, denying pigs access to human fecal material is efficacious for halting the transmission of *T. solium*.

Treatment

Therapeutic anthelmintics present only a temporary solution unless the conditions under which the parasites were acquired are altered. No drug is effective against all stages of all parasites, and the tissue damage prior to therapy results in slower growth rates and increased nutrient requirements. A good management system will incorporate practices, such as sanitation, genetic selection, and nutrition, to prevent infections and will not use treatment as the sole method of controlling parasites. The choice of anthelmintic is dependent on the parasite species present and the relative cost of the products. Routine surveillance and strategic treatment for parasites are advisable on farms with a previous history of clinical disease. Treatment reduces the number of egg-producing parasites and keeps further premise contamination to a minimum. For pigs placed in high-risk environments, the use of prophylactic compounds pyrantel tartrate and fenbendazole fed for several weeks can control infections and reduce lesions from ascarids and nodular worms, reduce worm populations, or may enhance immunity against *A. suum* (Southern et al. 1989; Stankiewicz and Jeska 1990). Treatment of sows 10–14 days before farrowing has been shown to be effective in preventing transmission of nematodes and *Sarcoptes scabiei*.

Macrocyclic lactones

Macrocyclic lactones, such as avermectins and milbemycins, are fermentation products of the fungus *Streptomyces avermitilis* and are thought to act by preferentially binding glutamate-gated chloride ion channels, a class of ion channel that is only found in invertebrates, which leads to paralysis and death of susceptible invertebrates. Avermectins are available as injectable and pour-on formations or as feed additives and have stated slaughter withdrawals. In general, they control *A. suum* (adults and fourth-stage larvae), *H. rubidus* (adults and fourth-stage larvae), *Oesophagostomum* (adults and fourth-stage larvae), *S. ransomi* (adults), and

Metastrongylus (adults). Doramectin is labeled for *A. suum* (adults and fourth-stage larvae), *Oesophagostomum* (adults and fourth-stage larvae), *S. ransomi* (adults), *Metastrongylus* (adults), and *Stephanurus* (adults) and has variable efficacy for *Trichuris*.

Benzimidazoles

Benzimidazoles preferentially bind nematode beta-tubulin and disrupt cell division. This class of drugs is thought to have a wide margin of safety as it preferentially binds the nematode protein as opposed to mammalian tubulin. Thiabendazole is well known but less efficacious in swine than fenbendazole, which is the only benzimidazole labeled for helminths in swine in the United States, available for administration in an oral suspension or feed additive with a slaughter withdrawal. Fenbendazole products are labeled for the treatment of *A. suum*, *Metastrongylus*, *Oesophagostomum*, *Hyostrongylus*, *Stephanurus*, and *Trichuris* in the United States. It should be noted that fenbendazole is thought to be one of the few anthelmintics effective for removal of *Trichuris*.

Imidazothiazoles

Levamisole binds nematode nicotinic acetylcholine receptors, leading to paralysis and death. It has been used in water or feed. It is effective for the treatment of *Ascaris*, *Oesophagostomum*, *Metastrongylus*, *Strongyloides*, and *Stephanurus*. It has a slaughter withdrawal in the United States.

Tetrahydropyrimidines

Pyrantel tartrate is the only tetrahydropyrimidine labeled for use in swine; it also acts by binding nematode nicotinic acetylcholine receptors. It is available for incorporation into feed, most commonly used as a continuous dewormer for 30 days in starter and growing pigs as an aid in the prevention of larval migration and establishment of *A. suum* and *Oesophagostomum*. Pyrantel has a slaughter withdrawal in the United States.

Piperazine

Piperazine salts are an older generation of antiparasitic purge dewormer that causes a neuromuscular blockade by disrupting GABA neurotransmission. Piperazine is administered in feed or water and should all be consumed in an 8–12 hour period; withholding feed or water the previous night is beneficial for stimulating consumption. It is currently approved for treatment of adults of *Ascaris* and *Oesophagostomum*.

Organophosphate compounds

Dichlorvos is an organophosphate compound added to feed with good efficacy against *Ascaris, Oesophagostomum, Trichuris,* and *Hyostrongylus* with slightly lower efficacy against *Strongyloides* (Marti et al. 1978). It can be incorporated into slow-release polyvinyl chloride pellets that allows for continued effect in the cecum, producing the desired removal of whipworms.

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