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# *Taenia solium* Neurocysticercosis<sup>1</sup>

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## ABSTRACT

After many years as a relatively rare neurological disorder, *Taenia solium* (pork tapeworm) neurocysticercosis is now seen more frequently in patients in the United States. Humans are the definitive and only hosts of the tapeworm stage. The larval stage develops in the pig, the intermediate host, following ingestion of tapeworm eggs excreted in the feces of the tapeworm carrier. The larvae invade most tissues of the pig giving rise to a disease termed cysticercosis. When humans ingest raw or undercooked meat from cysticercotic pigs, taeniasis (tapeworm) results. Humans can also act as intermediate hosts if they ingest *T. solium* eggs present in contaminated food or water; cysticercosis, similar to that seen in pigs, develops. If the larvae invade the central nervous system, neurocysticercosis with ensuing neurological dysfunction results.

Cysticercotic pigs are rarely found in the United States. Only three animals out of >88 million federally inspected pigs were diagnosed as cysticercotic in 1990. In the United States, the excellent sewage disposal system prevents access of pigs to human feces and infection of pigs by *T. solium* eggs is consequently rare. In Mexico, however, the mean rate for cysticercotic pigs in inspected slaughter houses during 1980-1981 was 1.55% and there is little reason to suspect that it has decreased. In rural areas of Mexico and South America where sewage disposal is limited, the number of cysticercotic pigs can be in excess of 5% and neurocysticercosis is a common disease in the human population. In such areas, pigs are not penned or fed but depend on scavenging waste, including human waste, for food. Thus, the cycle of cysticercotic pigs infecting humans and tapeworm carriers infecting both humans and pigs is difficult to break in primitive rural areas.

The incidence of neurocysticercosis is increasing in the United States due to an influx of immigrants from areas where *T. solium* is endemic. Most patients presenting with neurocysticercosis are of Mexican origin and probably acquired their disease in Mexico. However, several cases have been reported in people who have no history of travel to endemic areas and who were probably infected through ingestion of food prepared by an unhygienic food preparer who was also a tapeworm carrier.

In this review, the life cycle of *T. solium*, parasite transmission, incidence of *T. solium*-related disease in pigs and humans, the disease process, drugs used in treatment, detection *T. solium*

and destruction of *T. solium* eggs and cysticerci in foods are discussed. Food microbiologists must be aware of the increasing importance of *T. solium* as a disease agent and how to control *T. solium*-related diseases.

Key Words: Pork tapeworm, *Taenia solium*, neurocysticercosis

Neurocysticercosis is considered to be the most common parasitosis of the human central nervous system (CNS) worldwide, particularly in less developed countries. Unfortunately, its incidence is increasing in the United States (24). Cysticercosis is caused by encystment of the larval stage of the pork tapeworm, *T. solium*, in various tissues of infected swine and humans. When the cysticerci (encysted larvae) are present in the CNS, the ensuing disease, neurocysticercosis, often results in neurological dysfunction. The adult tapeworm is found only in the human intestine; the resulting disease is taeniasis. Taeniasis is caused by eating raw or undercooked meat from cysticerci-infected pigs. The individual suffering from taeniasis excretes eggs in his feces and ingestion of *T. solium* eggs by either humans or pigs leads to cysticercosis (72). Pork cysticercosis was well known to the ancients: Aristotle (384-322 B.C.; Greek philosopher and scientist), in his *History of Animals*, described 'pork measles', which is the common name for pork meat containing *T. solium* cysticerci (82).

*Taenia solium* cysticercosis in swine is rare in the United States, Canada and western Europe, but is endemic in less-developed areas of Latin America, Africa, Asia and Europe (49). Thus, *T. solium*-related diseases are of public health significance in endemic areas. In industrialized countries, taeniasis and human cysticercosis were considered to be rare in the pre-1970s. However, with increased immigration into the United States, Canada and western Europe of individuals from countries where *T. solium* is endemic, there is now more concern about pork tapeworm-related diseases (24). In the United States, neurocysticercosis is found mainly among migrant workers and recent immigrants from Latin America and the prevalence of taeniasis and neurocysticercosis is high in California and other states bordering Mexico (27). Since the pork tapeworm can survive for 10 to 25 years in its human host (18,27), there is a good possibility that the tapeworm carrier with poor

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personal hygiene who is involved in food preparation will contaminate food with *T. solium* eggs. The consequences to consumers who eat such foods may be neurocysticercosis. This was dramatically demonstrated by Schantz et al. (103) in an interesting report of neurocysticercosis in the Orthodox Jewish community in New York City. Eleven of 17 individuals from four families were affected and some of the individuals had neurological symptoms. Evidence of taeniasis was not present in the Jewish family members as would be expected in non-pork eating individuals. However, these families employed housekeepers and/or cooks who had emigrated from Latin America and some of the caretakers were carriers of the pork tapeworm. Epidemiological evidence indicated that the afflicted individuals had been infected with *T. solium* eggs by their caretakers either from personal contact or from eating food prepared by them.

The older literature concerning *T. solium*, taeniasis and cysticercosis is summarized and discussed in the monograph, *Cysticercosis: Present State of Knowledge and Perspectives*, edited by Flisser et al. (35).

#### *Biology of Taenia solium.*

The pork tapeworm is a parasitic worm that belongs in the phylum *Platyhelminthes*, class *Cestoidea*, subclass *Cestoda*, family *Taeniidae*, genus *Taenia* and species *solium*. The life cycle of *T. solium* is diagrammed in Fig. 1. Humans are the definitive hosts of *T. solium* and are the only species in which the cestode larva (some workers refer to the larva as *Cysticercus cellulosae*) develop into the adult tapeworm under natural conditions. The adult tapeworm consists of a scolex (head), unsegmented neck and segmented body. Its total length is 2 to 4 M but can be longer. The worm is found in the human small intestine where the scolex usually attaches to the upper jejunum. The attachment to the intestinal wall is by the means of four cup-shaped suckers and a double row of hooklets located on the scolex. The neck is the region of growth that produces proglottides (segments). The body consists of 800 to 1,000 proglottides, each of which contain a single set of male and female reproductive organs. The egg-bearing terminal proglottides separate at intervals and are discharged into the feces. Each gravid proglottid contains 30,000 to 50,000 eggs. When a pig ingests the eggs, the egg membranes are digested by gastric and pancreatic enzymes with the liberation of oncospheres (fully developed and infectious embryos). The oncospheres penetrate the wall of the jejunum of the pig, enter the circulatory system and eventually spread throughout the body. The oncospheres deposit in striated muscle, liver, myocardium and nervous tissue. In 60 to 70 days, the oncospheres evolve into cysticerci which can be 5 to 20 mm in diameter. The mature cysticercus is a translucent thin-walled vesicle (bladder) with an invaginated scolex equipped with suckers and hooklets. When raw or inadequately cooked cysticerci-containing pork is ingested by humans, digestive juices dissolve the cyst walls releasing the scolices. Within 5 to 12 weeks, an adult tapeworm develops in the bowel and thereby continues the lifecycle of the cestode. Generally,

only one tapeworm is found in the human host.

Humans, similar to swine, can act as intermediate hosts and develop cysticercosis upon ingestion of water or food contaminated with *T. solium* eggs. Autoinfection can occur if the individual suffering from taeniasis places his fecally-contaminated fingers in his mouth or if he ingests food or water contaminated by his own stools. A less likely mechanism of autoinfection involves reverse peristalsis when detached gravid proglottids are transferred into more proximal parts of the small intestine.

The simultaneous presence of both diseases — taeniasis and cysticercosis — may be seen. However, Nash and Neva (80) state that most patients with CNS symptoms do not have the adult tapeworm. The egg membrane is digested by gut enzymes and the released oncospheres penetrate the gastric or intestinal wall; once they reach the general circulation, the oncospheres deposit in various tissues (often the CNS) where they form cysticerci. Cysticercosis in humans represents a dead end since the cestode lifecycle can not be completed. The life cycle of *T. solium* given above is a composite of the descriptions of Del Brutto and Sotelo (24), Loo and Braude (68), Markell et al. (72) and Murrell et al. (79).

#### *Transmission of T. solium to swine and humans.*

Coprophyagy of human feces is the predominant mode of transmission of *T. solium* eggs to the pig — the major intermediate host (5,99). *Taenia solium* eggs are infectious when excreted and do not require a period of soil incubation for activation (6). The mature proglottid contains 30,000 to 50,000 eggs and approximately five proglottides are released. The proglottides are non-motile and are usually shed as connected segments in the feces from an infected human (18,79). Ingestion of the connected segments by a scavenging pig means that the animal receives a massive infectious dose of *T. solium* eggs (49). While most *T. solium* eggs are present in the excreted proglottides, individual eggs also are present in the intestinal lumen and perianal area, on the clothes and on the hands of the tapeworm carrier (91). Therefore, pork tapeworm eggs may be transmitted to humans via personal contact. Any food or equipment used in food preparation handled by a pork tapeworm carrier may be contaminated with eggs. Uncooked foods such as salads or sandwiches pose a significant risk (14,18). Even water or other drinks can be contaminated if the carrier handles ice cubes. Eggs can be transmitted to individuals from vegetables grown on lands that have been fertilized with human excrement, particularly if the vegetables are uncooked (68). If human feces are not disposed of properly, flies may be a mode of transfer of *T. solium* eggs to human and swine foodstuffs (66).

Cysticerci are transmitted to humans via the ingestion of raw or undercooked meat from infected pigs (18). The only other animal source of *T. solium* cysticerci is the infected human; cannibalism of flesh from a person with cysticercosis could lead to taeniasis.

#### *Incidence of T. solium disease in swine and human populations.*

The incidence of *T. solium* cysticercosis in swine in the

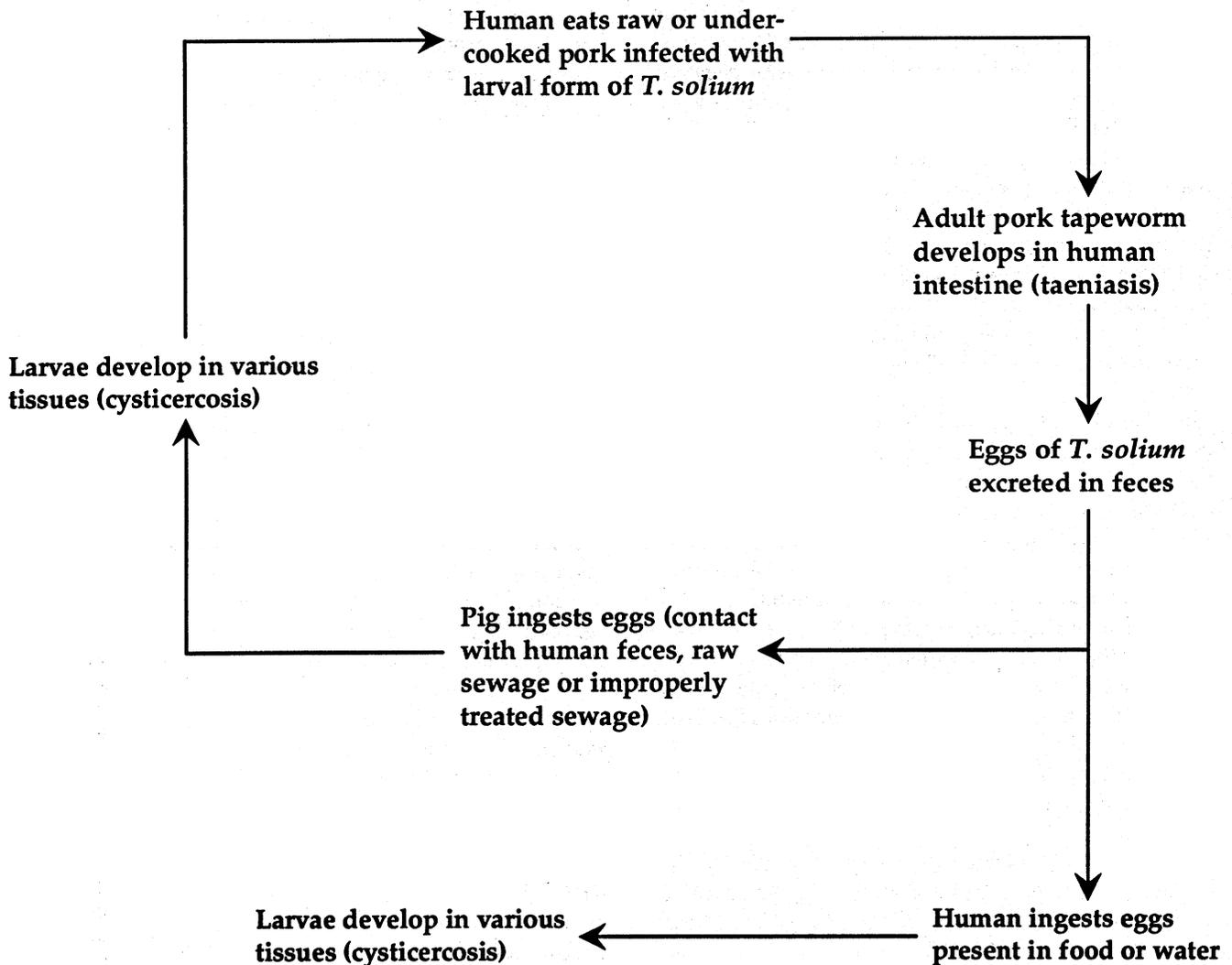


Figure 1. Lifecycle of *Taenia solium*.

United States is low. In 1976, 4 out of approximately 70 million hogs slaughtered in federally inspected slaughter houses were reported to be infected with cysticercosis (49); while in 1981, 6 of approximately 88 million pigs were positive (79). In 1990, 3 pigs were diagnosed with *T. solium* cysticercosis out of more than 83 million (103). Therefore, it is virtually impossible to contract taeniasis from eating pork in the United States, especially if the meat was obtained from a federally-inspected abattoir.

The mean rate of incidence of *T. solium* cysticerci in pigs in 75 slaughter houses in 22 states of Mexico was 1.55% for the years 1980-1981 (5). Acevedo-Hernandez (2) stated that 17,058,300 hogs were slaughtered in Mexico during 1980; at an incidence of 1.55%, 264,404 of those slaughtered pigs would be infected with *T. solium*. However, it is not certain how many infected animals were actually condemned and were not allowed to enter the food chain. It is probable that swine cysticercosis in Mexico has not changed much since the 1980 data were obtained and the incidence is still high.

A recent report by Sarti-G. et al. (99) indicated that porcine cysticercosis in a rural community, Angahuan, in Michoacan State of Mexico was 6.5% as revealed by tongue examination (cysticercotic nodules are present on the underside of the pig's tongue). It has been shown by

Gonzalez et al. (42) that there is a reasonably good correlation between the presence of cysticerci in the tongues of living infected pigs and the presence of cysticerci in muscular tissue at antemortem inspection. They found that 24/77 pigs were positive by necropsy whereas 18/77 pigs showed tongue cysticerci. Therefore, the 6.5% prevalence figure obtained by Sarti-G. et al. (99) represents a minimum and the real prevalence of cysticercotic pigs in Angahuan was probably higher. Owners who believe their pigs to be positive for cysticercosis do not send them to controlled markets but either kill the pigs for family consumption or sell them through clandestine markets (99). Sarti et al. (98) examined 571 pigs (out of a total of 2,200) in the village of Xoxocotla in Morelos State in Mexico and found that 4% were positive for cysticerci by tongue examination. Infection increased with age of the pigs and was higher in pigs that were allowed to roam and feed on human feces. In the village of Los Sauces in Guerrero State, Mexico, Keilbach et al. (54) found that 6% of 440 pigs were positive by the tongue test. The pigs were not confined and were allowed to eat garbage and human waste.

For the years 1981-1986, the frequency of cysticercosis in 366,712 pigs slaughtered at the main abattoir in Tegucigalpa, Honduras was 4.8% (52). The prevalence of

cysticercosis (determined by serology) in pigs in Peruvian slaughter houses was approximately 12% (42). Gonzalez et al. (42) stated that small farmers send pigs with negative tongue tests to regular slaughter houses; positive pigs were either used for home consumption or were sold through informal markets at greatly reduced prices. In the large coastal cities of Peru, pork is inspected and supplied through regulated slaughter houses (8). However, in the Huancayo region in the Sierra highlands of Peru, pigs are not processed through government slaughter houses but are sold through informal markets; 43% of the informally traded pork is believed to be cysticercotic. Approximately 65% of the pork consumed in Peru is obtained from non-inspected sources (8).

In Uttar Pradesh State of India, during the period July 1980 to December 1985, 8.3% of 3550 slaughtered pigs were infected with cysticercosis (84). One-hundred-and-sixty of the infected pigs were considered fit for consumption but 136 were condemned. However, Pathak and Gaur (84) did not state the conditions under which the infected pork was rendered suitable for human consumption.

There is little information on the incidence of taeniasis and cysticercosis in human populations. At the Los Angeles County Hospital, only 14 cases of cerebral cysticercosis were found among 82,700 consecutive autopsies (0.02%) for the years 1918-1965 (71) whereas for the period 1950-1984, cerebral cysticerci were found in 481 of 20,026 autopsies (2.4%) performed in Mexico City (98). Thus, autopsy data indicate a much higher incidence of neurocysticercosis in Mexico as compared to the United States. Schenone et al. (105) believed that the incidence of neurocysticercosis in Latin America was approximately one case per 1,000 inhabitants (0.1%).

Recent data have appeared which give some idea of the incidence of taeniasis and cysticercosis in humans residing in rural areas of Mexico where primitive swine-raising is practiced and where the hygienic conditions of the population are low. In the village of El Sotano in Hidalgo State, 4/127 individuals (3.1%) were *Taenia* tapeworm carriers; four other individuals said that they had seen proglottides in their stools within the past year (100). Since there were no cattle in the village, the tapeworm carriers were believed to be infected with *T. solium*. Seven of 117 individuals (6.0%) in El Sotano were seropositive for cysticercosis and 41% of the population gave a history of at least one sign or symptom indicative of neurocysticercosis (100). In the village of Los Sauces in Guerrero State, 24/760 people (3.1%) were found to be carriers of *Taenia* spp. tapeworms (54) and in the village of El Salado, Culiacan State, Camacho et al. (15) found 6/516 persons (1.2%) had stools positive for *T. solium*. Only four of 1,531 residents (0.3%) of the village of Xoxocotla, Morelos State had stools positive for *Taenia* species (98). However, 90/1,552 individuals said that they had passed tapeworm proglottides in the past. By using enzyme-linked immunoelectrotransfer blotting, Sarti et al. (98) found that 167 of 1546 Xoxocotla villagers (10.8%) were seropositive for cysticercosis.

Tapeworm carriers were present in these rural Mexican villages at rates ranging from 0.3 to 3.1%. In Xoxocotla, which had the lowest incidence (0.3%) of tapeworm carri-

ers, approximately half of the households had inside toilets or outdoor privies (98). Such sanitary facilities were either lacking or in short supply in the other villages.

Taeniasis and cysticercosis data for Honduras presented by de Kaminsky (52) indicated that 1,728/365,400 (0.47%) individuals visiting health facilities (in 12 provinces) for various reasons during 1983-1989 were carriers for *Taenia* spp. tapeworms. In a survey during 1985-1988 of 15 rural areas in Honduras, 68 out of 3,288 randomly selected individuals (2.1%) were tapeworm carriers. De Kaminsky (52) identified 74.6% of 181 proglottides in fecal specimens as *T. solium*; it is probable that most of the *Taenia* spp. carriers from rural areas of Honduras were infected with *T. solium*. The number of cysticercosis cases was 129 out of 79,545 (0.16%) during the period 1980-1988 in patients at the University Hospital of Tegucigalpa, Honduras (52). The number of cysticercosis cases in rural areas of Honduras is probably much higher considering the fact that the incidence of tapeworm carriage is higher. In a survey conducted by Cruz et al. (21) in areas of southern Ecuador, 1.6% of 9,529 individuals reported finding a taenia in their feces after treatment with the anthelmintic drug, praziquantel. Since *Taenia saginata* taeniasis is not present in this area of Ecuador, the expelled worms were considered to be *T. solium*. A random sample of 242 individuals from two villages of the Ekari tribe from Indonesian New Guinea indicated that 42 (17.4%) had palpable cysticerci and 7/242 (2.9%) had *T. solium* eggs in their feces (78). A survey of schoolchildren from rural areas of South Africa indicated that 2.49% of 736 children in the Transkei area and 0.23% of 677 children from the KwaZulu area were seropositive for cysticerci (83). Shasha and Pammenter (107) demonstrated that 1,352 school children from rural areas of Transkei, South Africa had a mean cysticercotic seropositivity rate of 5.5%. Interestingly, these workers found that children with good nutrition had a rate of 7.3% whereas it was 3.3% in children with poor nutrition. The decreased seropositivity found in undernourished children reflects impaired antibody response associated with low intake of protein and energy (107). On the whole, there appears to be a close relationship between incidence of cysticercosis in swine and cysticercosis (and taeniasis) in humans in underdeveloped areas of the world. The more rural and primitive areas show a higher incidence of cysticercosis in both swine and humans and this is related to the primitive conditions of swine raising and sanitation facilities as well as to poor hygienic behavior.

Neurocysticercosis is rare in the United States population but few surveys have been done. However, some information on neurocysticercosis in Los Angeles County (California) is available since cysticercosis is a reportable disease in California (91). During the period 1973-1983, 497 patients were treated for cysticercosis in Los Angeles hospitals (91). Approximately 90% of the patients were Hispanics with the majority being Mexican in origin. There were 11 deaths (2.2%) attributed to cerebral cysticercosis. The disease was identified in 12 United States citizens who had no history of travel to endemic areas which indicated that these individuals were probably infected by local *T. solium* carriers (91). Fecal parasite data from stool

specimens recorded at Los Angeles County Department of Health Services revealed that approximately 0.5% of stools were positive for *Taenia* spp. for the period 1976-1983. The national average for the presence of *Taenia* spp. in stools for 1978 was 0.06% (91).

For the period, January 1988 to December 1990, 138 cases of cysticercosis were reported for Los Angeles County (108). Approximately 90% of the cases were Hispanic and most were Mexican immigrants. Nineteen of the infected individuals were born in the United States; nine of these individuals were probably infected when they traveled to Mexico but the other ten United States citizens appear to have acquired their disease locally. Cysticercosis was responsible for eight deaths (5.8%); however, during the study, eight other people died whose disease onset appeared before January 1988 (108). *Taenia* spp. eggs were present in approximately 0.2% of the stool samples submitted to the Los Angeles County Public Health Laboratory during the study period (108). The 1987 national average for stools containing *Taenia* spp. was <0.1% (53).

It is reasonable to assume that cysticercosis in the United States is not limited to Los Angeles County or to California. Other areas that border Mexico such as New Mexico, Arizona and Texas probably have increased incidence of cysticercosis. It can be assumed, also, that there is an increased incidence of the disease in any city or area that has a large immigrant population.

#### *Diseases caused by T. solium.*

Taeniasis results when a human ingests raw or improperly cooked pork containing the cysticerci of *T. solium*. The adult tapeworm is found only in humans; it has been estimated that 4 million people worldwide are carriers (129). The disease is difficult to detect since the patient is usually asymptomatic and tapeworm eggs and proglottides are not always found in feces (32). There may be occasional gastrointestinal symptoms, such as abdominal pain, hunger pains or chronic indigestion, but most carriers are unaware that they are infected (72). While *T. solium* taeniasis may be a mild or unapparent disease, the carrier poses a potentially dangerous threat since the ingestion of worm eggs leads to cysticercosis in both humans and swine. Due to the long life span of the tapeworm, the carrier can serve as a source of eggs for many years. In Los Angeles County (California), Richards et al. (91) found that 1.5% of the parasitism in females was due to taeniasis (*Taenia* spp.) in contrast to 0.5% for men. De Kaminsky (52) also found that women were infected with tapeworm more frequently than men. This higher incidence of tapeworm in women may be due to sampling raw meats during meal preparation. Thus, the role of women as traditional food preparers may be of epidemiological significance in the transmission of pork tapeworm eggs.

Whereas ingestion of cysticercotic pork leads to development of tapeworm, cysticercosis results when humans ingest *T. solium* eggs. Many clinicians in the United States do not see cysticercotic patients whereas others may see several cases a year. The frequency depends on the geographical location, socioeconomic status, and the ethnic

background of the patients.

The encysted larvae (cysticerci) of *T. solium* can lodge anywhere in the body and Despommier (27) stated that the parasite does not have tropism for any tissue. However, Sotelo et al. (110) and Martinez et al. (73) asserted that there is a marked tendency for encystment to occur in the CNS, muscle and eyes. The preferential location of the cysticerci is probably due to the extensive vascular network and to the high glucose (or glycogen) concentration found in these areas of the body. Also, the larvae may survive best in the brain since it is an environment relatively safe from immunological reactions (75).

Many cases of human cysticercosis are asymptomatic and the individuals, as well as their physicians, may not be aware of the infection. Symptomatic cysticercosis is divided into subgroups depending on the localization of the parasite: disseminated cysticercosis, ocular cysticercosis and neurocysticercosis.

Disseminated cysticercosis may localize in the viscera (heart, liver, lung, etc.), muscles, connective and subcutaneous tissue and bone (93,132). Subcutaneous cysticerci may be prominent and the patient's skin presents a nodulated appearance. Cysticerci can develop in any voluntary muscle tissue and the infection is generally asymptomatic. However, there may be pain and muscle weakness with fever and eosinophilia (72). More rarely, there may be massive muscle enlargement (muscular pseudohypertrophy) where the individual presents with apparent increased muscle mass (126). Invasion of muscle is believed to occur in approximately 5% of infections by *T. solium* larvae (45).

Cysticerci present in the ocular system may float in the aqueous or vitreous humors, or adhere to retinal tissue and the infection can be severe enough to lead to decreased vision or loss of the eye. The lachrymal glands and eyelids may be invaded, also (18,72,132). It is estimated that the eye is affected in approximately 3% of *T. solium* larvae infections (45).

Invasion of the CNS occurs in approximately 60% of *T. solium* larvae infections and symptoms are seen in about 50% of the cases (18,45). Neurological disease symptoms may appear 2 months to 30 years (mean of 5 years) after infection; most of the afflicted individuals are aged 20 to 50 years (18,45).

Any part of the CNS may be infected. The larvae may lodge in the cerebral parenchyma (usually gray matter), subarachnoid space, ventricles and/or spinal cord (18,24,50). The organisms may be viable for years; death of the cysticerci leads to onset or aggravation of symptoms. Often, the dead cysticerci calcify (18,44). Patients with neurocysticercosis present with a wide variety of neurological signs and symptoms including cranial nerve palsies (partial paralysis), dementia (mental deterioration), encephalitis (inflammation of the brain), headache, hydrocephalus (fluid accumulation in CNS), involuntary movement disorders, meningitis (inflammation of membranes of brain or spinal cord), psychosis, seizures (epilepsy) and stroke (18,24,50). Many patients develop combinations of the above symptoms. Death may be the outcome in some cases of neurocysticercosis. How the patient is affected depends on the location of the cysts, size of the cysts, numbers of cysts (cyst load) and extent of host-

cyst inflammation (104).

Epilepsy or seizures appears to be a major response to *T. solium* larvae infection of the CNS. In a total of 2,821 cases of neurocysticercosis from China, Mexico and the United States, epilepsy was seen in 56.4% (29,70,104,110,128). Medina et al. (74) investigated late-onset epilepsy (occurs after age 25) in Mexico City and found that neurocysticercosis or its sequelae was the underlying cause for at least 50% of seizure cases. In areas where *T. solium* infections are uncommon, brain tumors, cerebrovascular disease, trauma and alcoholism are major causes of late-onset epilepsy (74). Garcia et al. (39) studying epileptic patients in Peru, found 22/189 (11.6%) were seropositive for *T. solium* whereas only 8/309 (2.6%) of non-epileptic patients were positive. In late-onset epileptic patients, 15/74 (20.3%) were seropositive for *T. solium*, thereby indicating that neurocysticercosis can be an important cause of late-onset epilepsy in Peru (39).

A particularly dangerous type of neurocysticercosis — racemose neurocysticercosis — is caused by a larval form of *T. solium* termed *Cysticercus racemosus* (in contrast to the normal larval form, which is called *C. cellulosae*). The racemose form of the larvae can reach a large size (4-12 cm) with greatly varied appearance. It is sterile, e.g., there is no scolex. It can appear as a large bladder with few lobations or as a complex of unequally sized bladders arranged in grape-like clusters (14,89,122). The life span of the typical cysticercus is 2 to 5 years followed by calcification whereas the racemose form can grow for longer periods and if unchecked, may grow to overwhelming dimensions with unfortunate consequences to the patient. The origin of the racemose form is uncertain but probably represents a degenerated form of *T. solium* larvae (73,89,122). Racemose cysticercosis is characterized by parasite invasion of nonconfining areas of the brain such as the subarachnoid spaces or ventricles where unchecked growth of the racemose form can occur. Frequently, hydrocephalus results and patients suffer visual loss, dementia, arachnoiditis (inflammation of arachnoid membrane and subarachnoid space) meningitis and/or cranial nerve palsies (104,122). The course of racemose disease is invariably grave and often ends in death.

Neurocysticercosis is not usually seen in children since the time from infection to overt neurological symptoms averages 5 years but can range from a few months to 30 years (18,45). At hospitals in the Los Angeles areas, Mitchell and Crawford (76) and Percy et al. (88) studied 61 patients ranging in age from age 21 months to 20 years. Two patients had headaches (one of these had visual problems also) but the other 59 presented only with seizures. In a study of 89 Mexican children, Lopez-Hernandez and Garaizar (69) found that 55% of the children had intracranial hypertension due to cerebral edema and 81% had seizures. In South Africa, 43.8% (n = 89) of children from rural areas presented with epilepsy and 31.5% with increased intracranial pressure (22,119). Kalra et al. (51) studied 11 neurocysticercotic children (mean age = 9.5 years) at a pediatric clinic in New Delhi, India. They found increased intracranial pressure in 10 and seizures in 7 of the 11 children. An increase in intracranial pressure is rare in

neurocysticercotic children in the United States. Thus, pediatric patients in the United States appear to have a milder disease than those from underdeveloped countries. The difference in disease symptoms between the two sets of children may be due to repeated exposure of children in endemic areas to large numbers of *T. solium* eggs whereas children in the United States are exposed to smaller infecting doses (29,76).

There does not appear to be an association of human cysticercosis with major histocompatibility complex (MHC) antigens nor are immunosuppressed individuals more susceptible to infection (63). However, women do develop a greater degree of inflammation when the cysticerci are located in the brain parenchyma. There is a higher prevalence of cysticercotic encephalitis in women (male-female ratio of 1:7); also, their prognosis is worse for other forms of neurocysticercosis (25).

#### *Immune system evasion by T. solium.*

Many patients infected with *T. solium* larvae are often asymptomatic and lack a detectable immune response to cysticerci as long as the larvae remain viable (70). The immune refractoriness may be due, in part, to encapsulation of the larvae (50,61). Encapsulation of the parasite by progressive host-derived fibroblastic responses leads to a dense collagenous capsule surrounding the larvae. There may be a narrow zone of granulation tissue consisting of various leukocytic cells at the surface of the capsule. The fibrous capsule can be separated from the living larvae, thereby indicating that there is a clear line of demarcation between host and parasite (50,61). The larval wall may be beneficial to both host and parasite. The parasite is protected from the immune system and the host is protected from parasitically derived toxic products (92).

Nemeth and Juhasz (81) found a protease inhibitor in *Taenia pisiformis* (dog tapeworm, with rabbits as intermediate hosts) which inactivated trypsin and chymotrypsin and probably acts to protect the larvae during their transit in the human body. However, it is not known if *T. solium* possesses such a protease inhibitor.

*Echinococcus multilocularis* (tapeworm of dogs, cats or foxes with rodent prey as intermediate hosts) larvae can induce the generation of CD8<sup>+</sup> suppressor (T<sub>s</sub>) cells, which inhibit the production of interleukin-2 and expression of interleukin-2 receptors by lymphocytes; thus, the parasite is immunosuppressive (56). Flisser (33) suggested that neurocysticercosis patients are immunosuppressed: there is decreased intradermal reaction to *Mycobacterium tuberculosis* purified protein derivative, increased numbers of CD8<sup>+</sup> T<sub>s</sub> cells, and decreased blastoid transformation of blood lymphocytes. Pigs infected with *T. solium* eggs are immunosuppressed, also. There were decreased numbers of CD4<sup>+</sup> T helper cells in peripheral blood and an inverse relationship between the number of CD4<sup>+</sup> cells and the number of cysticerci found in pigs after slaughter (129). Grisolia (44), however, stated that there is no real evidence that *T. solium* larvae infection leads to immunosuppression.

While only 50% of patients produce anti-cysticercal antibodies, 84% of the antibody responders react against

Antigen B produced by the parasite (34). Antigen B (paramyosin) is synthesized by the larva and is secreted through the parasite membrane into host tissues. Secretion of Antigen B may be an evasive mechanism used by the parasite, since immune complexes will be formed at a distance from the parasite surface (60). Therefore, host attack mediated by anti-Antigen B antibodies will not damage the larvae. Lacleite et al. (61) proposed that Antigen B facilitates survival of pork tapeworm cysticerci by inhibiting the classical pathway of the host's complement cascade. Antigen B inhibits complement function by binding C1<sub>q</sub> (62). Other parts of the classical complement pathway are not inhibited; thus, Antigen B is directed at inhibiting the initiation of the classical pathway. If Antigen B can bind C1<sub>q</sub> *in vivo*, then it protects the parasite from antibody-mediated damage by the membrane attack complex of complement. Antigen B, by inhibiting initiation of the classical pathway, may decrease the formation of complement mediators, which contribute to potentiation of inflammation (62). It is probable that *T. solium* larvae have other specific mechanisms, which either inhibit immune responses or inhibit other anti-parasite reactions of the host (129), but little or no information is available concerning these possible mechanisms.

Often, there is no inflammation around the cysticerci and the larvae survive for many years. Flisser et al. (34) found that approximately 50% of patients with cysticercosis were immunologically unaware of the presence of the parasite since they did not have circulating antibodies against cysticerci. But, when the cysticerci began to degenerate and die, a marked inflammatory reaction occurred in the surrounding brain tissue suggesting that the organism may be more immunogenic when dead than when alive (75). Thus, onset or worsening of symptoms coincides with death of the cysticerci and may be due to release of larvae antigens (44). In addition, the worsening condition of the patient upon death of the cysticerci suggests that immunosuppression of the host defenses by the parasite is no longer possible (129).

#### *Medical treatment for taeniasis and cysticercosis.*

The tapeworm stage of *T. solium* can be eliminated by administration of niclosamide or quinacrine. Niclosamide is not always efficacious and has the additional disadvantage of causing maceration and digestion of worm segments so that eggs are released into the intestine (1,68). Eggs released by niclosamide action may enter into the general circulation by passage through the intestinal wall (68). However, the anti-helminthic, praziquantel, is more useful since it kills both tapeworms and larvae of various taenids and does not cause maceration and digestion of the worm. Praziquantel is now widely used to treat *T. solium* related diseases (12,55,106,118).

Before the 1980s, once the CNS had been invaded by *T. solium*, there was little that medical science could do for the neurocysticercosis patient. The patient's symptoms could be relieved with the administration of corticosteroids (to reduce inflammation due to dead or dying parasites), antiepileptic drugs (to control seizures), surgical extirpation

of single cysts or ventricular shunting in cases of cysticercotic-induced hydrocephalus (45,68,80). But the introduction of praziquantel into cysticercosis management provides more hope for patients with disseminated and cerebral cysticercosis.

Praziquantel is well-tolerated and has no long-term toxicity. Toxic effects are transient and include mild gastrointestinal disturbances, headaches and at high doses, a feeling of decreased sense of well-being (64,87). Praziquantel does not possess organ toxicity (e.g., the drug does not produce abnormalities in liver, bone marrow or CNS functions), does not interfere with fertility, and is not teratogenic, carcinogenic or mutagenic (38,87). Side-effects are seen with praziquantel use in neurocysticercosis (nausea, fever, vomiting and exacerbation of many of the neurological reactions seen in the disease) and are mainly due to inflammatory reactions to dying and dead larvae rather than to the drug itself (18). These side-effects are controlled by the use of analgesics (pain relievers), anticonvulsants, corticosteroids or antihistamines (123).

Praziquantel completely cured cases of subcutaneous (dermal) cysticercosis (12,93,116) but treatment of ocular cysticercosis was unsuccessful (12).

Only active cases of neurocysticercosis (cases with viable cysticerci) can be treated with praziquantel; cases in which the larvae are dying or dead cannot be improved by the drug (46). Praziquantel with or without pharmacological control of side-effects has been shown to either cure or improve cases of uncomplicated neurocysticercosis (24,26,29,55,95,124).

Pharmacological control of side-effects during praziquantel treatment depends on the severity of the effects and probably should be determined on a patient-to-patient basis (123). In complicated neurocysticercosis (extraparenchymal involving ventricular, spinal or subarachnoid types) presenting with hydrocephalus, praziquantel may be used along with shunting or other surgery (10,123). The treatment of racemose cysticercosis with praziquantel was only partially successful in resolving the disease (9). King and Mahmoud (55) point out that despite the effectiveness of praziquantel in treatment of taeniasis and cysticercosis, its relatively high cost limits its use in many developing countries — the very countries that need the drug the most.

Albendazole, with or without pharmacological control of side-effects, appears to be as effective as praziquantel in treating neurocysticercosis (3,30,90,96,109,113,124). Clinicians may use albendazole and praziquantel sequentially in treatment of some patients whose disease is difficult to resolve by use of only one of the drugs. Similarly to praziquantel, albendazole acts against both the tapeworm and larval stages of *T. solium* (117).

The modes of action of praziquantel and albendazole against *T. solium* are different. Praziquantel acts on the scolex of the cysticercus by paralyzing the parasite's musculature whereas albendazole inhibits the uptake of glucose by the parasitic membrane leading to energy depletion (23). Since the racemose form of cysticercosis consists of membranes without a scolex, albendazole should be effective against that form of the disease.

Thus, the use of praziquantel and/or albendazole al-

lows the clinician to successfully manage cerebral cysticercosis but their use does not always eliminate the need for surgery. It is necessary to remove cysticerci from some CNS locations (intraventricular or spinal cysticerci) and to shunt in order to relieve obstructive hydrocephalus and intracranial pressure (26,29). It is important, too, when a case of cerebral cysticercosis is found, to screen other members of the household, relatives, and close friends for neurocysticercosis. It is imperative to check for taeniasis in contacts of neurocysticercosis patients in order to remove the source of infection.

Some cases of neurocysticercosis resolve spontaneously and it is difficult to be certain that treatment is really effective. Cook (17) has pointed out that no large randomized double-blind trials with praziquantel have been performed (nor have such studies been done with albendazole). But in today's political climate, what may be good science may not be good ethics.

#### *Detection of T. solium and cysticercosis in humans and swine.*

Fecal examination of the suspect tapeworm carrier may reveal the presence of proglottides or eggs of *T. solium*, but the excretion pattern of proglottides and eggs by infected individuals is inconsistent; thus, fecal examination is frequently negative (32). Eggs of *T. solium* and *T. saginata* can not be differentiated on the basis of morphology; however, the proglottides are morphologically different (102). Tapeworm coproantigens can be detected in feces but *T. solium* and *T. saginata* coproantigens cross-react (37). At present, no specific DNA hybridization technique is available for detection of *T. solium* eggs. Rishi and McManus (94) have developed DNA probes that discriminate between the proglottides of *T. saginata* and *T. solium*; however, these probes are not in routine use.

Palpation of the body can reveal the presence of subcutaneous and intramuscular cysticercotic nodules and identification of an excised cysticercus can be done histologically (18). However, diagnosis of neurocysticercosis depends on more sophisticated techniques.

*Computed tomography and magnetic resonance imaging.* The most sensitive and highly specific technique for demonstrating the presence of neurocysticercotic lesions is cranial computed tomography (CT). This radiologic procedure allows the detection (and eventual treatment) of cases of neurocysticercosis that were missed by the use of older radiographic imaging techniques (24,29). Almeida-Pinto et al. (4) reported that after obtaining a CT scanner, the rate of detection of neurocysticercosis at their hospital increased seven-fold. Kramer et al. (59) used CT to study the progression of lesions during the course of untreated cysticercosis and CT has been used to follow the course of recovery from cysticercosis during chemotherapy (3,11,12,95,112). Magnetic resonance (MR) imaging of the cranium also has been found useful for detecting cysticercosis (73,131). Magnetic resonance imaging may demonstrate lesions that are no longer visible on CT scans (59). According to Martinez et al. (73), MR is more effective than CT in diagnosing active neurocysticercosis but CT is superior in

detecting inactive and calcified lesions.

Computed tomography has been used to detect cysticercosis in pigs, also. Flisser et al. (36) used CT to determine the effectiveness of praziquantel treatment in cysticercotic pigs.

*Serology.* While imaging techniques have increased the detection of neurocysticercosis, serological techniques are desirable in order to exclude non-*T. solium* related lesions. Serological procedures would be useful, also, in areas of the world where advanced imaging techniques are too expensive.

Many patients with clinically proven neurocysticercosis lack circulating anticysticercotic antibodies. According to Corona et al. (19), antibodies are present in 60 to 80% of the clinically proven cases whereas Flisser et al. (34) found that only 50% of cases responded immunologically. Gottstein et al. (43) demonstrated that 25% of clinically confirmed cysticercosis cases did not produce anticysticercotic antibodies. Corona et al. (19) and Zini et al. (133) showed that patients with benign (little or no symptoms) cysticercosis had lower levels (or none) of antibody than those with malignant cysticercosis. The varied antibody response in patient populations with neurocysticercosis demonstrated by these different investigators is probably due to the varied number of benign cases present in the populations studied.

Tsang et al. (120) have developed an enzyme-linked immunotransfer blot assay (EITB) for diagnosing human cysticercosis using glycoprotein antigens isolated from pig cysticerci. Seven antigenic bands from the glycoprotein preparation were recognized by antibodies present in sera of cysticercotic patients. According to Tsang et al. (120), reaction to one or more of the seven bands is indicative of cysticercosis. Examination of patients with confirmed cysticercosis revealed that sera of 108/111 and cerebral spinal fluid (CSF) of 37/37 patients reacted to one or more of the antigenic bands in the EITB assay. The sera of healthy individuals or individuals infected by other parasitic diseases did not react with any of the diagnostic bands (120). The EITB, thus, appeared to be both sensitive and specific. Other workers have confirmed the sensitivity and specificity of the glycoprotein EITB for the detection of cysticercotic antibodies (28,31,130). Antibody was present in sera of cysticercotic patients more often than it was present in CSF. The EITB assay for cysticercotic antibodies was highly sensitive for patients with multiple, CT or MR enhancing intracranial lesions; however, the assay was less likely to be positive with sera from patients with a single lesion or with calcified lesions, e.g., benign cysticercosis (130). Feldman et al. (31) found that saliva could be used in the EITB assay; a positive EITB was found in 70% of patients' saliva as compared with 100% of their sera. While EITB on saliva was not as sensitive in detecting cysticercosis, it is an excellent adjunct to serum assays and should prove useful in epidemiological studies since obtaining saliva is painless and non-invasive.

Gonzalez et al. (42) found that the EITB assay could be used for detection of cysticercosis in pigs. The prevalence of swine cysticercosis in Huancayo, Peru was 23.4% by tongue assay, 31.2% by necropsy, 37.7% by enzyme-

linked immunosorbent assay (ELISA) and 51.9% by EITB. Thus, EITB was the most sensitive assay for determining the presence of cysticercosis in pigs. The assay was quite specific since the sera of non-infected pigs or pigs with other parasitic diseases were non reactive and all pigs that were confirmed to be cysticercotic by parasitic examination were positive by EITB (121).

Most of the studies for the serological determination of cysticercosis rely on the detection of anti-cysticercotic antibodies in patient sera or CSF. Few studies have been done on detection of cysticercotic antigens in patients. Tellez-Giron et al. (114,115), using ELISA, found that 13/17 cysticercotic patients had serum antibodies against cysticerci. Determination of cysticercotic antigens in CSF using Dot-ELISA indicated that 10/17 were positive and 13/17 of patients were antigenically positive when a standard ELISA for cysticercotic antigens was used (114,115). Use of one of the assays did not detect all of the patients; however, combined use of the three ELISA procedures allowed detection of all 17 patients.

Correa et al. (20), utilizing ELISA with a combination of monoclonal and polyclonal antibodies against *T. solium* cysticerci, were able to detect parasitic antigens in the CSF of 90 to 93% of cysticercotic patients. An ELISA with monoclonal antibody was useful for the detection of parasitic antigens in pig sera, also (20). A monoclonal antibody-based ELISA was used by Wang et al. (127) to demonstrate circulating antigen in 95/116 neurocysticercotic patients. The patients lacking antigen appeared to have very few or dead cysticerci. A sensitive assay for the detection of cysticercotic antigens in sera or CSF could be based on the new technique called immuno-PCR (polymerase chain reaction) in which a specific antibody-PCR conjugate is used to detect antigen (97). The authors determined that immuno-PCR is sufficiently sensitive to detect a few hundred antigen molecules.

The International Task Force for Disease Eradication (7) stated that one of the chief obstacles to eradication of *T. solium* is the lack of simple diagnostic methodology for humans and pigs. Enzyme-linked immunotransfer blot assay is the most specific and sensitive method for serological detection of cysticercosis available at present, but EITB is surely not the inexpensive and simple diagnostic test that the Task Force envisions. Enzyme-linked immunotransfer blot assay utilizing saliva (31), if it could be made more sensitive, could be the basis of a simple and non-invasive method for detecting cysticercosis in both humans and pigs.

#### *Destruction of T. solium in foods.*

Little information is available on the stability of *T. solium* eggs and cysticerci to food processing conditions. Lawson and Gemmell (65) have reviewed certain aspects of the stability of the eggs of various taeniid species to temperature and drying conditions, but there is no information on *T. solium* eggs in that review. In a number of other taeniid species, infectivity of eggs is eliminated by treatment with moist heat: in  $\leq 10$  min at 55 to 70°C and in  $\leq 1$  min at 85 to 100°C (65). It is probable that *T. solium* eggs inadvertently present in foods will be inactivated if

that food is adequately heated.

A pork carcass "heavily" infected with *T. solium* cysticerci must be destroyed (16); however, the Food Safety and Inspection Service (FSIS) of the United States permits the cooking (passed for cooking) of "lightly" infected pork at an internal temperature of  $\geq 76.7^\circ\text{C}$  for 30 min before being used to prepare pork products (16). A beef carcass "lightly" infected with *T. saginata* (beef tapeworm) cysticerci may be passed for human consumption if cooked to an internal temperature of 60°C (16). The great difference in the "passed for cooking" temperatures required by FSIS regulations suggests that *T. solium* cysticerci are more difficult to inactivate with heat than *T. saginata* cysticerci. Experimental data were not found concerning heat destruction of either *T. solium* or *T. saginata* cysticerci in meats.

Freezing pork at  $-10^\circ\text{C}$  for at least 14 days is considered to be an effective means for eliminating infectivity of *T. solium* cysticerci (79). Using 5 to 7 kg samples of heavily infected pork stored at 11, 4,  $-5$ ,  $-15$  or  $-24^\circ\text{C}$ , Sotelo et al. (111) found that *T. solium* cysticerci survival was decreased with storage at 4 or  $11^\circ\text{C}$ , but storage at these temperatures did not lead to complete inactivation after 15 days. At  $-5$ ,  $-15$  or  $-24^\circ\text{C}$ , viable cysticerci were not found after 4, 3 or 2 days of storage, respectively. Thus, the 14 days freezing period at  $-10^\circ\text{C}$  appears to be more than adequate for the destruction of *T. solium* cysticerci. It is recommended that beef infected with *T. saginata* be stored at  $-9.4^\circ\text{C}$  for 10 days to destroy infectivity (16). Beef infected with *T. saginata* cysticerci was rendered non-infectious when frozen for 15 days at  $-5^\circ\text{C}$ , for 9 days at  $-10^\circ\text{C}$ , and for 6 days at  $-15$  to  $-30^\circ\text{C}$  (48). These data suggest that *T. saginata* cysticerci are more resistant to freezing than *T. solium* cysticerci.

No information was found on the effect of curing conditions (drying, sodium chloride [NaCl] or other curing salts, organic acids, etc.) or fermentation on the survival of *T. solium* cysticerci in meats. Hird and Pullen (49) stated that salting under the "appropriate conditions" will inactivate *T. saginata* cysticerci but there do not appear to be any experimental data indicating to what extent *T. saginata* or *T. solium* cysticerci are inactivated by curing conditions used in meat processing.

Immunosuppressed Golden hamsters infected by *T. solium* cysticerci develop non-egg producing tapeworms. Using this animal model, Verster et al. (125) studied the effect of  $\gamma$ -irradiation (0.2-1.4 kGy) on *T. solium* cysticerci in pork. Thirty days after infection, the mean length of worms, which developed in hamsters fed non-irradiated cysticercotic pork was 173.8 mm; cestodes that developed in animals fed meat irradiated with 0.2-0.4 kGy consisted of only the scolex. No worms were recovered from hamsters fed cysticercotic meat treated with 0.6 kGy. Verster et al. (125) recommended that pork carcasses containing cysticerci be rendered fit for human consumption by treatment with irradiation doses of 0.2-0.6 kGy. Veal infected with *T. saginata* cysticerci and subjected to  $\gamma$ -irradiation doses of 0.3-0.6 kGy was shown to be non-infective for humans volunteers (40). The radiation data of Verster et al. (125) and Geerts et al. (40) indicate that the infectivity of cystic-

erci from both *T. solium* and *T. saginata* would be eliminated if carcasses were treated with a radiation dose of approximately 0.6 kGy.

Do treatments of pork which inactivate *Trichinella spiralis* larvae inactivate *T. solium* cysticerci? Freezing pork containing *T. spiralis* to an internal temperature of -20°C eliminated infectivity within 48 min (58). Food Safety Inspection Services regulations stipulate storage at -20.6°C for 82 h (16). Kotula et al. (57) found that trichinae larvae were non-infectious when pork was heated to an internal temperature of 60°C for 2 min. Food Safety Inspection Services regulations indicate that the internal temperature of the meat must be at 60°C for at least 1 min (16). Irradiation of *T. spiralis*-infected pork with 0.15-0.30 kGy inactivated the larvae (13). Food Safety Inspection Services regulations also allow the inactivation of *T. spiralis* by various curing conditions (16). While the amount of information concerning the inactivation of *T. solium* cysticerci is scanty, the data do indicate that freezing or radiation conditions that eliminate infectivity of trichinae larvae in pork will probably inactivate *T. solium* cysticerci. It is not certain, however, whether heating or curing conditions that render pork trichinae-free will inactivate *T. solium*.

#### Control of *T. solium*.

Prior to the 20th century, *T. solium* was probably hyperendemic in much of the world where swine raising was practiced; however, the organism has virtually disappeared in Western Europe and North America (United States and Canada) due to the introduction of high standards of meat inspection, development of modern methods of swine management, closing of clandestine markets, improved sanitation and improved standards of living (41). In areas where porcine cysticercosis is still common, the presence of *T. solium* is associated with poor sanitation, low living standards, primitive swine-raising practices, high levels of illiteracy and lack of meat inspection.

Methods of control in endemic areas must focus on the pork tapeworm carrier, tapeworm eggs in the environment, the diseased pig, stringent meat inspection, and education (18,32,66,86,111):

1. Tapeworm carriers must be detected and treated with anti-helminthics.
2. Excretion of *T. solium* eggs into the environment by indiscriminate human defecation should not be permitted and adequate sanitary sewage disposal should be initiated. Swine husbandry practices should be changed so that pigs do not come in contact with human feces.
3. High standards of meat inspection and proper disposal of infected pig carcasses (closing of clandestine markets) will aid in eliminating infected pigs as a source of human infection.
4. An important part of control is education. People must be made aware of the life-cycle of *T. solium*, how tapeworm-related diseases are transmitted and how these diseases can be controlled and/or prevented. There must be emphasis on what constitutes good personal hygiene and on the proper handling of food. The individuals in the commu-

nity must be trained in the proper cooking of pork to ensure destruction of cysticerci. While freezing is another means of cysticerci destruction, it is probable that most households in pork tapeworm endemic areas will not have access to freezers.

The key factor in control of *T. solium* in endemic areas is proper disposal of human waste so that it is not available to scavenging pigs (66). In non-endemic areas, the key factor for control is detection and treatment of tapeworm carriers (103). Immigrants from *T. solium* endemic areas who prepare food in homes or restaurants or who work in or around swine-raising facilities should be tested for the presence of pork tapeworm. Infected immigrant workers were the suspected source of *T. solium* eggs in an outbreak of swine cysticercosis, which occurred in a feedlot in Colorado in the early 1980s (101). Schantz et al. (103) have shown that pork tapeworm-infected housekeepers/cooks pose a risk to individuals residing in those households. Carriers who are family members have been shown to infect other family members and tapeworm-infected visitors to a household may also be a source of infection (6).

Travelers to *T. solium* endemic areas should be cautious about eating pork. Since food and water may be served by individuals suffering from taeniasis, travelers must also be cautious about everything that they eat or drink. All vegetables should be thoroughly cooked since they may have been grown on lands fertilized with human excrement.

There are currently no vaccination procedures that can control *T. solium* in either pigs or humans. Molinari et al. (77) using antigenic material from cysticerci and Pathak and Gaur (85) using antigens isolated from oncospheres found that vaccinated pigs were protected against infection when challenged with *T. solium* eggs. The sample sizes were small and protection was not complete but the results were sufficiently encouraging to indicate that further research should be done. A recombinant vaccine against *Taenia ovis* (dog tapeworm with sheep as intermediate host) has been developed, which protected sheep against infection when they were challenged with *T. ovis* eggs (47,67). Thus, it is possible that a recombinant antigen from *T. solium* could be used as a vaccine. Vaccination, along with other control measures, could lead to the elimination of pork tapeworm and cysticercosis in endemic areas.

#### Animal models.

The understanding of *T. solium*-related diseases has been hampered by the lack of a small animal model. Therefore, there are only two choices: pigs or humans. The pig is an excellent model to use in the study of cysticercosis since the same organs are affected as in humans. The pig would be suitable for determining various aspects of host immune behavior toward *T. solium* larvae and the reaction of the parasite to the immune system.

It is difficult to be certain that drugs, such as praziquantel or albendazole, are effective in treatment of neurocysticercosis since some cases appear to resolve spontaneously. Since no large randomized double-blind drug

trials for treatment of neurocysticercosis have been done (17), the use of the cysticercotic pig model could give the needed information on the true effectiveness of the drugs currently in use. For example, Flisser et al. (36) treated pigs suffering from cysticercosis with praziquantel (50 mg/kg for 15 days); at 57 days after the end of treatment, no muscle cysticerci were seen. The drug was not as effective against cerebral cysticerci and while many cysts had degenerated, viable cysticerci were still present in pig brain tissue at 57 days after the end of therapy. A longer term of treatment or higher concentrations of the drug might have succeeded in eliminating the brain cysticerci. In fact, Bittencourt et al. (11) have suggested the use of high concentrations (100 mg/kg rather than 50 mg/kg) of praziquantel in treatment of human neurocysticercosis. The results that Flisser et al. (36) obtained with drug use in swine cysticercosis could be the basis of a thorough study in determining the effectiveness of praziquantel and albendazole in treatment of cysticercosis. In addition, infected pigs could be used to screen and evaluate new anti-cysticercotic drugs and to determine the effectiveness of vaccines.

#### Economics.

Due to lack of systematic studies on the epidemiology of *T. solium*-related diseases, there is little information concerning the economics of cysticercosis in humans and swine. Diseased pigs represent an economic loss to the animal industry in endemic areas (32). Pig-raising is an extremely important source of income for the small farmer in much of the developing world. Free-ranging pigs feeding on domestic refuse and fecal matter require minimum investment in terms of feeding and housing costs. These marginal farmers do not welcome governmental regulations such as meat inspection since any regulations concerning cysticercotic animals would reduce their already slender incomes (86).

Individuals with neurocysticercosis in both endemic and non-endemic areas may require repeated hospitalizations, expensive surgery and/or prolonged chemotherapy (10,91,101). The protracted treatment required for cysticercotic patients ultimately leads to an overall increased cost in health care. Additionally, most of the ill individuals are 20 to 50 years of age, their most productive years (45), and their illness results in the incapacity for full-time work and represents a loss in productive and remunerative labor. When death occurs from neurocysticercosis, the economic loss is incalculable. The International Task Force for Disease Eradication estimates that, worldwide, there are 50 million cases of *T. solium* taeniasis/cysticercosis with an annual death rate of 50,000 (7). These statistics are probably conservative and the incidence of taeniasis and cysticercosis is much higher. It is obvious that the global economy can ill afford the continued presence of *T. solium* in the environment.

#### CONCLUSIONS

The major hindrance to eliminating *T. solium* is the

lack of understanding by residents in endemic areas (particularly in the more rural and primitives districts) about the lifecycle of *T. solium* and the consequences of *T. solium*-induced diseases to pigs and humans. An example, which indicates how difficult it is to educate people about the pork tapeworm, has been provided by Keilbach et al. (54). In a rural area of Mexico, they initiated an educational program in which the lifecycle of *T. solium*, the importance of the role of human feces in propagating the parasite, the danger involved in eating undercooked pork, and the consequence of cysticercosis to humans were discussed. Most of the men refused to participate; however, women and children did attend the educational sessions. Two years after initiating the program, a questionnaire revealed that only 3/150 adults understood the lifecycle of the parasite and the cause of cysticercosis in pigs and humans. However, 46/60 children did show a good understanding of *T. solium* (54). In spite of the difficulties, education is absolutely essential as a motivating factor in eradicating the parasite from the environment. A vigorous insistence on education along with drastic changes in pig-raising practices, sanitation, personal hygiene and pig inspection requirements will do much in achieving elimination of *T. solium* in endemic areas.

Reduction or elimination of neurocysticercosis in the United States and other developed nations will, of course, depend on the effectiveness of control programs in *T. solium* endemic areas, since the major sources of infection in developed countries are immigrants from those areas. But until economic, social, sanitary and education conditions improve, control or elimination of *T. solium* in underdeveloped countries will not have high priority. It is probable that neurocysticercosis will continue to be a health problem in North America and Western Europe for some time to come.

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