

Tapeworms, Meat and Man: A Brief Review and Update of Cysticercosis Caused by *Taenia Saginata* and *Taenia Solium*

D. W. HIRD and M. M. PULLEN*

Department of Large Animal Clinical Sciences
College of Veterinary Medicine, University of Minnesota
St. Paul, Minnesota 55108

(Received for publication May 22, 1978)

ABSTRACT

Three species of tapeworms may be transmitted to man by ingestion of animal flesh: *Taenia saginata*, *Taenia solium*, and *Diphyllobothrium latum*. The first two are the subject of this brief review which concentrates on recent studies in the field and emphasizes concepts of importance in detection, control, and prevention of cysticercosis. *T. saginata* cysticercosis in beef (beef measles) continues to be a concern in developed countries such as the United States, as well as in developing areas such as East Africa where the infection is widespread. The high standards of meat inspection in the United States have not succeeded in eliminating beef cysticercosis which is seen primarily in feedlot cattle originating in the southwestern U.S. However, it should not be viewed as a strictly regional problem, due to the widespread movement of animals and meat within the United States. Beef cysticercosis is costly due to the special treatment required of infected carcasses; serious effects on human health are rare. In contrast, *T. solium* cysticercosis in swine (pork measles) is rarely reported in areas such as the U.S., Canada, and most European countries, but is still a definite human health concern in Mexico, some other Latin American nations and parts of Africa and Asia. In addition to being a financial burden, *T. solium* is a serious public health threat in those countries where it is prevalent.

Three species of tapeworms may be transmitted to man by ingestion of animal flesh: *Taenia saginata*, *Taenia solium* and *Diphyllobothrium latum*. The first two are the subject of this brief review, which concentrates on recent studies in the field and emphasizes concepts of importance in detection, control and prevention of cysticercosis.

T. saginata cysticercosis in beef (beef measles) continues to be a problem in developed countries such as the United States, as well as in developing areas such as East Africa where the infection is widespread. The high standards of meat inspection in the United States have not succeeded in eliminating this infection which is seen primarily in feedlot cattle in the southwestern U.S. It should not be viewed as a strictly regional problem, however, due to the movement of animals and meat within the country. In one outbreak of cysticercosis in a Texas feedlot (44), 7,568 potentially infected cattle were slaughtered at 20 different establishments in eight

different states and the meat was then marketed across the country from California to Rhode Island. The problem is primarily an economic one resulting from costly special treatment of infected carcasses. Serious effects on human health are rare.

T. solium cysticercosis in swine (pork measles) is rarely reported in areas such as the U.S., Canada, and most European countries, but is still a definite problem in Mexico, some other Latin American nations, and parts of Africa and Asia. As well as being a financial burden, it is a serious public health threat in those countries where it is prevalent. Much less information is available on *T. solium* cysticercosis than *T. saginata* cysticercosis, perhaps because research funding is less readily available for problems not directly affecting developed countries.

HOSTS, SYMPTOMS, AND PREVALENCE OF INFECTION

The life cycles of the two tapeworms are relatively simple and quite similar. The adult tapeworms are obligate parasites of the human intestine, and are not found in the adult form in any other species of animal in nature. The adult worms produce gravid segments which leave the human host via the intestinal tract. If gravid segments or eggs are ingested by a suitable intermediate host, the eggs hatch and eventually become larval tapeworms (cysticerci) in the muscles of that animal. The cycle is completed when a human ingests these cysts in the flesh of an intermediate host. The cycle will not be completed if the cysticerci have been rendered nonviable by freezing or adequate cooking of the meat before it is eaten.

The larval cysticerci are considerably less host-specific than the adult worms, and may be found in a variety of intermediate hosts. Domestic cattle are the usual intermediate hosts for the cysticerci of *T. saginata*, but other animals such as buffalo, African wild animals (27) and reindeer in the USSR (2) may be infected. The pig is the usual intermediate host for the larval stage of *T. solium*, but cysticerci have been reported in man and

other primates, wild carnivores and domestic dogs, wild hogs, deer, sheep and cats (18). Only in massive infections is illness noted in the intermediate host.

Despite the prodigious size of the adult tapeworm (*T. saginata* commonly grows to lengths greater than 5 meters, while *T. solium* is generally smaller), the effect on human health is usually slight, and symptoms are often vague or absent. For *T. saginata* the most frequent symptom is discharge of a proglottid which may result in a crawling sensation in the perianal region; less commonly symptoms such as abdominal pain, nausea and weakness may be noted (29). Rarely, serious complications result from lodgement of a segment in an aberrant site such as the appendix. While the adult *T. solium* tapeworm is generally innocuous, man can also become infected with the larval cysticerci, thus serving as both intermediate and definitive host for *T. solium*. Human infection with cysticerci of *T. solium* can be fatal as the brain is a frequent site of localization. It is for this reason that *T. solium* is considered a serious threat to human health even though the adult tapeworm itself seems relatively harmless.

Information on the prevalence of *T. saginata* infection in man is sparse, but more complete data for presence of the larval cysticerci in cattle at slaughter indicate that the prevalence of infection has increased since World War II in many countries (1,29,46). Pawlowski and Schultz (29) reported that the prevalence in Europe in general rose after World War II, and Silverman (47) noted the sharp rise in Britain after the war. The reasons for this apparent increase are not clear, but several hypotheses have been advanced, such as the upheaval and displacement of the human population due to the war, the increased consumption of beef, especially raw beef, and the overtaxing of sewage treatment facilities in many countries. In the United States, U.S. Department of Agriculture statistics (55) show that in federally inspected slaughter houses for the fiscal year 1976, that of approximately 37,000,000 beef inspected, 135 were condemned for cysticercosis, and 9,628 less severely affected carcasses were passed after freezing. It is noteworthy that in the past, over 80% of parasitized cattle have originated from California, Arizona, New Mexico and Texas, the four states bordering Mexico (43). The prevalence of infection in California is 20 times that for the rest of the nation (45). Whether this higher prevalence of infection is due to infected workers from Mexico, importation of Mexican cattle as feeders, the climate, use of irrigation water, and/or the numbers and management of the cattle feedlots in the region is not entirely known. *T. saginata* cysticercosis is also prevalent in less highly developed regions. Six Latin American countries reported a combined prevalence of 0.27% from 1968-1973 (41) while in some African countries the prevalence may be about 10% (29). Pawlowski and Schultz (29) make the interesting observation that *T. saginata* is a problem in poor countries because they are poor, i.e. with comparatively lower standards of hygiene,

and in rich countries because they are rich, i.e. with increased beef consumption and overtaxed sewage treatment facilities.

T. solium cysticercosis, on the other hand, remains a problem of less highly developed countries where sanitary conditions and systems of pig raising permit direct human to pig transmission. In the United States only four of approximately 70,000,000 hogs slaughtered in federally inspected slaughter-houses were reported to be infected with cysticercosis during the fiscal year 1976 (55). In Mexico, however, 8,820 of 814,000 pigs (1.1%) slaughtered in Mexico City during 1970 were infected (42). Schenone (40) reported that of approximately 10,000,000 pigs slaughtered in some abattoirs of Central and South America between 1960 and 1973, 1.9% were infected with cysticercosis. Acha and Aguilar (3) found that cysticercosis was responsible for 68% of 17,100 condemnations of slaughtered pigs in six Central American countries during 1959-1961. Likewise, *T. solium* cysticercosis remains a serious problem in parts of South Africa, and in India and other Asian countries.

MECHANISMS OF TRANSMISSION

Man acquires the adult tapeworm by ingestion of raw or inadequately cooked meat containing cysticerci. Beef and pork are the usual vehicles for *T. saginata* and *T. solium*, respectively, but dog meat may transmit *T. solium* in countries where it is eaten (59). Adult worms appear to be long-lived; the length of life of *T. saginata* appears to be limited only by that of the host (29) and *T. solium* may live in excess of 10 years (52). Both are prolific egg producers. *T. saginata* may release 6-9 proglottids daily, containing an approximate total of three-quarters of a million eggs (30); *T. solium* may extrude five segments daily, containing a total of 250,000 or more eggs (61).

Under proper conditions taeniid eggs can survive for long periods and are resistant to most chemical disinfectants. Sufficient moisture seems to be the most important factor to maintain viability, and eggs survive longer at lower than at higher temperatures. For example, eggs of *T. saginata* have been kept alive for at least 168 days at 4-5 C (9), and some maintain that they can survive for even longer periods. Jepsen and Roth (19) found that *T. saginata* eggs survived at least 16 days at 18 C in a dish filled with liquid manure, for 71 days in liquid manure in an underground cistern, and up to 159 days on grass. Recent studies (4) have suggested that eggs when shed may be at different stages of their life span, being in a continuing transition from juvenile (non-infective) to mature (infective) to senescent (diminished ability to invade and develop). Higher temperatures speed the process, suggesting differing potentials for transmission at different seasons of the year.

Transmission from infected humans to the intermediate host is usually indirect via contaminated feed or water, but is occasionally direct as in the infection of

newborn calves by contaminated hands of herdsmen in East Africa (53).

In the transmission of *T. saginata* cysticercosis, contamination of cattle feed may occur in a variety of ways such as defecation of field workers in vegetable or hay fields, or in storage areas for grain, hay, silage, and other feeds used in cattle feedlots. Several recent outbreaks in feedlots in the Southwestern U.S. have been traced to feed contaminated in this manner (44,50). It is important to note, however, that the act of defecation or the presence of feces may not be necessary to transmit the infection. Proglottids and eggs are usually passed in the feces, but the proglottids of *T. saginata* may be motile and may pass through the anus and expel eggs on the perianal skin (36). Schultz (44) hypothesized that infection of feedlot cattle could have occurred by proglottids from an infected worker dropping unobserved to the ground, thus contaminating cattle feed.

Water can also be a vehicle for transmission of cysticercosis. Infected workers may defecate in irrigation water for crops used as animal feed, or animal drinking water may itself become contaminated. In tests with 10 persons carrying *T. saginata*, Ockert (28) recovered up to 68 oncospheres from fingernail dirt, up to 700 from water for hand washing, and over 50,000 per liter from water used to soak underwear. Effluent from sewage plants is an important mechanism of transmission of cysticercosis. Silverman (48) showed that tapeworm eggs can survive most urban and rural sewage treatment processes and can then pass on in the final effluent or in air-dried sludge; even under ideal circumstances sedimentation and rapid sand filtration will not remove all eggs. Many sewage plants now are old and overworked; increasing numbers of users and increased water usage per user add to the problem, as does the increased use of detergents and other agents which interfere with sedimentation, putrefaction, and oxidation (48). Recent outbreaks of cysticercosis in the U.S. (44) and Australia (34) have been traced to sewage effluent. It must be emphasized that use of sewage effluent in agriculture is not without considerable danger.

Birds have been implicated in transmission of the disease, apparently by ingesting the eggs in sewage or sewage effluent, and by disseminating them in their feces. Gotzsche (16) was able to infect calves with seagull droppings, and it has been suggested that other species of birds may also be involved in transmission of *T. saginata*.

Intrauterine infection of calves with *T. saginata* has been reported (26,29,49) but is of unknown importance in the epidemiology of the disease.

Transmission of *T. solium* between man and pigs appears to be less subtle. *T. solium* proglottids do not seem to be motile, and are frequently shed as connected segments presenting less opportunity for dispersion and a greater likelihood of massive infection in a single intermediate host such as a scavenging pig which consumes human feces.

DEVELOPMENT AND SURVIVAL OF THE CYSTICERCUS AND HOST IMMUNITY

Under proper circumstances, ingestion of an infective egg by the proper intermediate host will give rise to a larval cysticercus in the muscle of the host. McIntosh and Miller investigated development of *T. saginata* cysticerci in muscles of cattle. At 11 days post infection, the cysticerci and surrounding connective tissue were 3×2 mm in diameter and visible to the naked eye. At 18 days they were 4×2 mm. They are believed to be infective at 10-12 weeks and are fully developed at about 16 weeks when they may be about the size of a large pea (5×8 mm). Cysticerci of *T. solium* may attain approximately the same size. There is much variation in the size of cysts, and size alone cannot be used as a criterion of age. The date of infection can be estimated for young cysts, being accurate only until approximately 10 weeks post-infection for *T. saginata* (25).

The longevity of cysts is variable even within the same animal, and may be partially dependent on the tissue invaded (51,57) and host age at time of first infection (1). It is not unusual to find both living and dead cysts in the same animal (7,20,31). *T. saginata* cysticerci have been shown (57) to live for as long as 3 years after experimental infection. Detailed data are not available for cysticerci of *T. solium*, but degenerated cysts are seen less commonly in pigs than in cattle (59), perhaps because pigs are usually slaughtered at an earlier age than cattle.

In contrast to the adult tapeworm, the larval cysticercus produces an active immune response, perhaps because of its more intimate association with host tissues (62). This aspect of the biology of *T. saginata* and *T. solium* is currently receiving a great deal of attention, and much of the work in the field is concerned with the study of immunity to infection, to development of a vaccine, and to development of procedures which will permit accurate detection of infection in the living animal.

How and when cysticerci produce an immune response in the intermediate host is at best incompletely understood. A complicating factor is the ability of cysticerci to survive in the tissues of immune animals; this has become a central issue in immunoparasitology (15). Calves experimentally infected a few days to several months after birth with a single dose of *T. saginata* eggs will harbor cysticerci from that infection, and are susceptible to reinfection (10-13). However, if calves receive multiple doses of eggs from birth, or if they are infected at 3-4 months of age, they develop a strong resistance to reinfection at a later date (10-13,51) even though cysticerci from the original exposures may still be present.

HUMAN INFECTION

Since infection in man with the adult tapeworm of *T. saginata* or *T. solium* is frequently not accompanied by

clinical signs, diagnosis frequently depends on laboratory examinations. Although eggs can be detected by fecal examination, this method is considered unreliable. The favored diagnostic method is the adhesive cellulose tape or paddle and swab procedure in which a sticky surface is pressed to the perianal skin causing the eggs to adhere to it. The procedure should be repeated several times over several days and will not detect all infections. It is not possible to visually distinguish eggs of *T. saginata* from those of *T. solium*. Distinction between the two species based on segments of the tapeworm is difficult and controversial (32).

The treatment of choice for the adult tapeworm is niclosamide (Yomesan), a safe and effective drug (29).

As previously mentioned, man can be infected with the larval cysticercus as well as the adult tapeworm of *T. solium* and can thus act as both definitive and intermediate host. Human infection with the cysticerci of *T. solium* occurs either by ingestion of contaminated food or drink or by auto-infection, in which it is presumed that mature proglottids from an established adult *T. solium* are carried by reverse peristalsis from the small intestine to the stomach where they are stimulated to hatch and subsequently invade the extra-intestinal tissues to become cysticerci. The relative importance of auto-infection is not known, but it would seem reasonable to suppose that pork eaters have a greater chance of acquiring the tapeworm and hence would also have a greater chance of developing *T. solium* cysticercosis if auto-infection were of major importance. Acha and Aguilar (3) found no evidence of an increased prevalence of clinical cysticercosis in individuals parasitized with the adult *T. solium* as opposed to those free of it, and Heinz and Macnab (17) found chances of a non-pork eater becoming infected with cysticercosis to be as great as those of a pork eater. Human illness due to *T. solium* cysticercosis is a significant problem in many countries. For example, in 1972 Biagi (5) reported that human cysticercosis was found in 3-4% of autopsies in Mexico City, and for fully half of these it was the principal disease and probable cause of death. Many clinical pictures are seen, depending on the sites of localization of the cysticerci, and diagnosis can be quite difficult. Serological tests may be used as an aid to diagnosis; the indirect hemagglutination test is employed at the Center for Disease Control (39).

DETECTION, CONTROL AND PREVENTION

The life cycles of both *T. saginata* and *T. solium* are relatively simple; man transmits the infection to an animal which in turn transmits the infection to man. It is logical that the chain of infection may be broken in two places: the transmission from man to animal or the transmission from animal to man.

Man to animal

One method of halting the transmission from man to animal is to diagnose and eliminate human infections.

Programs of mass diagnosis and treatment aimed at eliminating the adult tapeworm from infected persons have reportedly had some success in Bulgaria, Poland and parts of the U.S.S.R. (29). Another measure that has been suggested (45) is the routine checking of feedlot workers for taeniasis before employment. Another method is proper protection of animal feed and water from human feces or from sewage treatment plant effluent.

Because a strong immunity is provoked by the cysticercus, animal vaccination has been investigated as a means of control; successful vaccination of the intermediate host would break the man-to-animal cycle of infection. Most recent work has been done with cattle and *T. saginata*. Immunization in cattle has been achieved by giving irradiated eggs by mouth (54), by the intramuscular injection with onchospheres (14,63), by induction of heterologous immunity with *Taenia hydatigena* (63), by inoculation with a homogenate of *T. saginata* strobila (14) and by a "parasite free" vaccine of antigens produced during in vitro cultivation of cysticerci (33,35). Many of these vaccines produce an excellent immunity to later challenge with *T. saginata* eggs. Young calves, however, do not respond to vaccination, and would be susceptible to infection for the first months of life under a program of control by vaccination. Intramammary vaccination to elicit maternal immunity which would be passed to the calf via colostrum has been accomplished (24). It has been recently reported that a single vaccination of heifers with *T. saginata* culture antigens "parasite free" vaccine during the last month of pregnancy resulted in the transfer of a considerable degree of colostral immunity to young calves. These calves, when subsequently vaccinated at 8-10 weeks of age, achieved a high degree of immunity to infection (35).

Animal to man (T. saginata)

To prevent the animal-to-man transmission of *T. saginata*, it is necessary to first detect infection in cattle either before or after slaughter, and then to prevent human consumption of infective meat.

Visual observation of cysticerci in slaughtered animals by meat inspectors is the most practical and widely used method of detection presently available. Current meat inspection procedure for federally inspected slaughterhouses in the U.S. is multiple incisions in the cardiac muscle and muscles of mastication together with visual examination of the muscle surfaces exposed by splitting the carcass. If one or more cysts are found a more extensive search is undertaken consisting of incisions into each round and into each forelimb 2 or 3 inches above the elbow. Selection of inspection sites is based on the likelihood of finding cysticerci, but there is a diversity of opinion regarding predilection sites for the organism. In addition, it is important for the meat industry that incisions be kept at a minimum and that mutilation of valuable cuts of meats be avoided. It is generally accepted that the muscles of mastication and the heart

are prime targets for *T. saginata* cysticercosis. In a recent study of feedlot cattle from the Southwest U.S., Juranek et al. (20) found that the heart and muscles of mastication were the most profitable sites in terms of numbers of cysticerci per pound of lean meat, and that the diaphragm was not as helpful. Investigators in South Africa and Rhodesia (37,59) have emphasized the importance of examining the shoulder muscles above the elbow. Robinson (37) reported that in 1,810 cases of *T. saginata* seen in slaughter cattle in Rhodesia 41.8% showed cysts only in the shoulder muscles. There is probably no universally appropriate predilection site. Rather, there may be differences due to geographic area, breed of cattle, age and activity of muscle groups; cysts have been hypothesized to settle in deeper muscles with a higher level of metabolic activity and greater blood supply (22).

Postmortem inspection for detection of *T. saginata* cysticercosis has serious drawbacks. For example, cattle frequently harbor only a few cysticerci, and current inspection procedures miss many light infections. Dewhirst et al. (8) reported that an inspector using the regular incision sites (masticatory muscles and heart) missed 22 of 80 infected carcasses, even though he was expecting to encounter cysticercosis. In addition, the infection is often detected months after it has occurred, making determination of the source difficult. If the source of infection were continuously operating, many animals could be infected in the long lag period between the start of the contamination and the first detection of infection at slaughter.

Use of antemortem techniques for detection could theoretically circumvent some of the defects of postmortem detection and current research efforts are being made in this direction. Serological methods such as complement fixation, latex, indirect hemagglutination, immunofluorescence, and gel diffusion techniques (1) have been tried for the antemortem diagnosis of cysticercosis but at present no good serologic method is available (15,21,29). There is no problem in detecting specific antibodies in heavily infected experimental animals, but the specificity of serologic tests in naturally infected animals is low, and the level of cross reactivity with other cestode species is high (15,21). The enzyme-linked immunosorbent assay (ELISA) technique, a highly promising test for serodiagnosis of parasitic infections, may find application for the detection of *T. saginata* cysticercosis (60). Intradermal tests have been tried, and Dewhirst et al. (6), using saline extracts of lyophilized and defatted portions of adult *T. saginata*, found an accuracy of 75% in naturally infected animals. This method, however, necessitates extra handling of the animals before slaughter to inject the reagent and would require a source of supply of adult *T. saginata*.

Once the cysticerci are detected in an animal, the carcass is condemned if the infection is severe, or in light infections the meat may be subjected to various

procedures to destroy the cysticerci. Cysticerci in meat can be killed by a variety of methods and the meat thus rendered safe for human consumption. Freezing at -10 C for 10 days will kill cysticerci in beef (23) and this is the most common current procedure in the United States. Shorter freezing periods would reduce expense, but current studies are lacking (29). Heating beef to temperatures above 56 C will inactivate *T. saginata* cysticerci, as will salting under appropriate conditions, and gamma radiation has also been proposed (29). U.S. Department of Agriculture regulations (56) permit freezing or cooking of lightly infected animals; cattle with one or more live or dead cysts must be frozen at 15 F (-10 C) or less for 10 days, or heated to at least 140 F (60 C). Until 1970, U.S. federal regulations permitted passage of a beef carcass without freezing or cooking if only one dead cyst was found under the assumption that (a) dead cysts occur singly in carcasses and (b) all cysts have the same life expectancy so that if there were more cysts they would also be dead. Minute dissection of 20 heavily infected animals (more than two cysts found on preliminary inspection) and 19 lightly infected animals (one or two cysts) by Juranek et al. (20) has shown these assumptions to be incorrect. Of 11 carcasses where only one cyst was found on initial (routine) inspection, five revealed more cysts on dissection; living and dead cysts were found together in some carcasses. It is obvious that if all beef were adequately cooked before eating that *T. saginata* would be effectively controlled or eliminated, but many people continue to prefer rare or raw beef, and human dietary habits are difficult to change.

Animal to man (T. solium)

Postmortem detection of cysticercosis in swine is easier than in cattle because lightly infected carcasses are uncommon, and detection is therefore easier. Little work has been done with antemortem serodiagnosis although there are some indications that the ELISA test is not as promising in *T. solium* cysticercosis as in *T. saginata* cysticercosis (60). U.S.D.A. regulations permit pig carcasses with light infections of *T. solium* cysticerci to be passed for cooking (56). On the basis of his studies in Rhodesia, Robinson (38) recently recommended that statutory freezing times for *T. solium*-infected pork carcasses should be reduced to 3 days. Verster (58) has recently stated that infested pork carcasses can be rendered fit for human consumption by exposure to gamma radiation at doses between 20 and 60 Krad. In general, control of *T. solium* infection in both animals and man is more easily effected by postmortem inspection and simple hygienic measures than is *T. saginata* infection.

RESEARCH TRENDS

At meetings in Germany in 1974 and in Kenya in 1976, experts from around the world discussed current problems in cysticercosis and identified certain areas where research was to be encouraged. The following are

some of the research needs identified (1):

1. Purification and isolation of species-specific antigens and their evaluation with techniques such as the enzyme-linked immunosorbent assay (ELISA), the soluble antigen fluorescent antibody test, immunoelectrophoresis, and counter-current electrophoresis.
2. In vitro culture of various developmental stages for the production of diagnostic and immunizing agents.
3. In vitro or laboratory animal model techniques to assess the infectivity of eggs of *T. saginata* and *T. solium*.
4. Factors that modify survival of eggs.
5. Chemical agents that have ovicidal effect during processing of sewage.
6. The contribution of prenatal and neonatal infection to survival of cysticerci in cattle.
7. The prevalence of antenatal infection in endemic areas.
8. Factors involved in the survival of cysticerci.
9. Active immunization by means of homologous and heterologous species of cestode.
10. Passive immunization with locally induced antibody in the mammary gland.

This list provides a good indication of fields in which research is now being carried out and probable areas of future concentration.

REFERENCES

1. Abdussalam, M., M. Gemmell, R. Griffiths, et al. 1976. Research needs in taeniasis-cysticercosis (memorandum). Bull. World Health Org. 53:67-73.
2. Abuladze, K. 1964. Taeniata of animals and man and diseases caused by them. p. 154. In K. Skrjabin (ed.), Essentials of cestodology. Academy of Sciences of the U.S.S.R. Helminthological Laboratory, Israel Program for Scientific Translations, 1970.
3. Acha, P., and F. Aguilar. 1964. Studies on cysticercosis in Central America and Panama. Am. J. Trop. Med. Hyg. 13:48-53.
4. Anonymous. 1976. Report of the joint FAO/UNEP/WHO consultation on field control of taeniasis and echinococcosis - Nairobi, Kenya. June 2-4, 1976. p. 5.
5. Biagi, F. 1972. Cerebral cysticercosis as a public health problem. II. Epidemiology in Mexico. Gac. Med. Mex. 103:227-230.
6. Dewhirst, L. 1967. Antemortem diagnosis of bovine cysticercosis due to *Taenia saginata*. Proc. U.S. An. Health Assoc. 71:540-545.
7. Dewhirst, L., J. Cramer, and W. Pistor. 1963. Bovine cysticercosis. I. Longevity of cysticerci of *Taenia saginata*. J. Parasitol. 49:297-300.
8. Dewhirst, L., J. Cramer, and J. Sheldon. 1967. An analysis of current inspection procedures for detecting bovine cysticercosis. J. Am. Vet. Med. Assoc. 150:412-417.
9. Froyd, G. 1962. Longevity of *Taenia saginata* eggs. J. Parasitol. 48:279.
10. Froyd, G. 1964. The artificial oral infection of cattle with *Taenia saginata* eggs. Res. Vet. Sci. 5:434-440.
11. Gallie, G., and M. Sewell. 1972. The survival of *Cysticercus bovis* in resistant calves. Vet. Res. 91:481-482.
12. Gallie, G., and M. Sewell. 1974. The serological response of three month old calves to infection with *Taenia saginata* (*Cysticercus bovis*) and their resistance to infection. Trop. Anim. Health Prod. 6:163-171.
13. Gallie, G., and M. Sewell. 1974. The serological response of calves infected neonatally with *Taenia saginata* (*Cysticercus bovis*). Trop. Anim. Health Prod. 6:173-177.
14. Gallie, G., and M. Sewell. 1976. Experimental immunization of six-month old calves against infection with the cysticercus stage of *Taenia saginata*. Trop. Anim. Health Prod. 8:233-242.
15. Gemmell, M. and P. Johnstone. 1977. Experimental epidemiology of hydatidosis and cysticercosis. p. 311-369. In B. Dawes (ed) Advances in parasitology, Vol. 15. Acad. Press, N.Y.
16. Gotzsche, N. 1951. Bidrag til *Taenia saginata*'s epidemiologi. Nord. Vet. Med. 3:957-983.
17. Heinz, H., and G. Macnab. 1965. Cysticercosis in the Bantu of Southern Africa. S. Afr. J. med. Sci. 30:19-31.
18. Herbert, I., and C. Oberg. 1974. Cysticercosis in pigs due to infection with *Taenia solium*, Linnaeus, 1758. p. 199-211. In E. Soulsby (ed.) Parasitic zoonoses. Acad. Press, N.Y.
19. Jepsen, A., and H. Roth. 1952. Epizootiology of *Cysticercus bovis* - resistance of the eggs of *Taenia saginata*. Intern. Vet. Congr., 14th, London, Proceedings 2:43-50.
20. Juranek, D., L. Forbes, and U. Keller. 1976. *Taenia saginata* cysticerci in muscles of beef cattle. Am. J. Vet. Res. 37:785-789.
21. Kagan, I. 1974. Advances in the immunodiagnosis of parasitic infections. Z. Parasitenkunde. 45:163-195.
22. Kearney, A. 1970. *Cysticercus bovis* - some factors which may influence cyst distribution. J. Parasitol. 56:183.
23. Landi, A., and A. Monzini. 1954. Osservazioni sul compartimento e sulla vitalita del *Cysticercus bovis* e del *Cysticercus cellulosae* alle basse temperature. Clinica Vet. Milano 77:264-268.
24. Lloyd, S., and E. Soulsby. 1977. Passive transfer of immunity to neonatal calves against the metacestodes of *Taenia saginata*. Vet. Parasitol. 2:355-362.
25. McIntosh, A., and D. Miller. 1960. Bovine cysticercosis, with special reference to the early developmental stages of *Taenia saginata*. Am. J. Vet. Res. 21:169-177.
26. McManus, D. 1960. Prenatal infection of calves with *Cysticercus bovis*. Vet. Res. 72:847-848.
27. Nelson, G., F. Pester, and R. Rickman. 1965. The significance of wild animals in the transmission of cestodes of medical importance in Kenya. Trans. R. Soc. Trop. Med. Hyg. 59:507-524.
28. Ockerl, G., and J. Obst. 1973. Dissemination of encapsulated oncospheres by tapeworm carriers. Monatshefte für Vet. 28:97-98.
29. Pawlowski, Z., and M. Schultz. 1972. Taeniasis and cysticercosis (*Taenia saginata*), p. 269-343. In Advances in parasitology, Vol. 10. Acad. Press, N.Y.
30. Penfold, H. 1937. The signs and symptoms of *Taenia saginata* infestation. Med. J. Aust., 24th year 1:531-535.
31. Penfold, H. 1937. The life history of *Cysticercus bovis* in the tissues of the ox. Med. J. Aust., 24th year 1:579-583.
32. Proctor, E. 1972. Identification of tapeworms. S. African Med. J. 46:234-238.
33. Rickard, M., and A. Adolph. 1976. Vaccination of calves against *Taenia saginata* infection using a "parasite free" vaccination. Vet. Parasitol. 1:389-392.
34. Rickard, M., and A. Adolph. 1977. The prevalence of cysticerci of *Taenia saginata* in cattle reared on sewage-irrigated pasture. Med. J. Aust. 1:525-527.
35. Rickard, M., A. Adolph, and S. Arundel. 1977. Vaccination of calves against *Taenia saginata* infection using antigens collected during in vitro cultivation of larvae: Passive protection via colostrum from vaccinated cows and vaccination of calves protected by maternal antibody. Res. Vet. Sci. 23:365.
36. Rijpstra, A., A. Smit, and N. Swellengrebel. 1961. How and where to search for the ova of *Taenia saginata*. Trop. Geogr. Med. Amsterdam 13:160-166.
37. Robinson, J. 1976. Observation on cysticercosis in Rhodesia. I. The distribution of cysts disclosed in beef carcasses at meat inspection. Rhodesian Vet. J. 7:2-5.
38. Robinson, J., and P. Chambers. 1976. Observations on cysticercosis in Rhodesia. II. The survival time for *Cysticercus cellulosae* cysts at low temperatures. Rhodesian Vet. J. 7:32-35.
39. Rydzewski, A., E. Chisholm, and I. Kagan. 1975. Comparison of serologic tests for human cysticercosis by indirect hemagglutination, indirect immunofluorescent antibody and agar gel

- precipitin tests. *J. Parasitol.* 61:154-155.
40. Schenone, H. 1973. Some considerations on the occurrence of cysticercosis in swine in Latin America. *Bol. Chil. Parasitol.* 28:106-107.
 41. Schenone, H., and T. Letonja. 1974. Swine and bovine cysticercosis in Latin America. *Bol. Chil. Parasitol.* 29:90-98.
 42. Schnaas, G. 1972. Sanitary control of cysticercosis. *Gac. Med. Mex.* 103:246-249.
 43. Schultz, M. 1971. Parasitic diseases along the Mexican - United States border. *Salud Publica Mex.* 13:377-380.
 44. Schultz, M., L. Halterman, A. Rich, and G. Martin. 1969. An epizootic of bovine cysticercosis. *J. Am. Vet. Med. Assoc.* 155:1708-1717.
 45. Schultz, M., J. Hermos, and J. Steele. 1970. Epidemiology of beef tapeworm infection in the United States. *Publ. Health. Rep.* 85:169-176.
 46. Sewell, M., and G. Gallie. 1974. Immunological studies on experimental infections with the larval stage of *Taenia saginata*. p. 187-193. In E. Soulsby (ed.) *Parasitic zoonoses*. Acad. Press, N.Y.
 47. Silverman, P. 1955. Bovine cysticercosis in Great Britain from July, 1950 to December, 1953, with some notes on meat inspection and the incidence of *Taenia saginata* in man. *Ann. Trop. Med. Parasitol.* 49:429-435.
 48. Silverman, P., and R. Griffiths. 1955. A review of methods of sewage disposal in Great Britain, with special reference to the epizootiology of *Cysticercus bovis*. *Ann. Trop. Med. Parasitol.* 49:436-450.
 49. Slais, J., and I. Mann. 1976. Morphological determination of the age of *Cysticercus bovis* in very young calves with cysticercosis. *Folia Parasitol. (Praha)* 23:321-326.
 50. Slonka, G., J. Moulthrop, L. Dewhirst, P., Hotchkiss, B. Vallaza, and M. Schultz. 1975. An epizootic of bovine cysticercosis. *J. Am. Vet. Med. Assoc.* 166:678-681.
 51. Soulsby, E. 1963. Immunological unresponsiveness to helminth infections in animals. *Int. Vet. Congr. 17th, Hannover*, p. 761-767.
 52. Soulsby, E. 1965. Textbook of veterinary clinical parasitology. Volume I. Helminths. F. A. Davis Co., Philadelphia, PA. p. 1068.
 53. Urquhart, G. 1961. Epizootiological and experimental studies on bovine cysticercosis in East Africa. *J. Parasitol.* 47:857-869.
 54. Urquhart, G. 1966. Bovine cysticercosis. *Int. Congr. Parasitol. (1st) Rome, Sept. 21-26, 1964. Proceedings, Vol. II*, p. 829. (Discussion p. 839).
 55. U.S. Department of Agriculture. 1976. Statistical summary, Federal meat and poultry inspection, MPI-1.
 56. U.S. Department of Agriculture. 1977. Code of Federal regulations. Title 9: Animals and animal products. p. 512-513.
 57. Van den Heever, L. 1967. On the longevity of *Cysticercus bovis* in various organs in a bovine. *J. Parasitol.* 53:1168.
 58. Verster, A., J. DuPlessis, and L. Van den Heever. 1976. The effect of gamma radiation on the cysticerci of *Taenia solium*. *Onderstepoort J. Vet. Res.* 43:23-26.
 59. Viljoen, N. 1937. Cysticercosis in swine and bovines, with special reference to South African conditions. *Onderstepoort J. Vet. Res.* 9:337-570.
 60. Walls, K., D. Allain, P. Arambulo, S. Bullock, and A. Dykes. 1977. The use of enzyme linked immunospecific assay for the serodiagnosis of parasitic infections. *Abstr. Annual Meet. Am Soc. Microbiol.* 77:37.
 61. Webbe, G. 1967. The hatching and activation of taeniid ova in relation to the development of cysticercosis in man. *Z. Tropenmed. Parasitol.* 18:354-369.
 62. Weinmann, C. 1966. Immunity mechanisms in cestode infections. p. 301-320. In E. J. L. Soulsby (ed.) *Biology of parasites*. Acad. Press, N.Y.
 63. Wikerhauser, T., M. Zukovic, and N. Dzakula. 1970. Vaccination against bovine cysticercosis. *J. Parasitol.* 56:369.