Taeniasis and Cysticercosis

Investigative Guidelines

December 2015

1. DISEASE REPORTING

1.1 Purpose of Reporting and Surveillance

1. To identify potential sources of disease transmission and arrange for treatment.
2. To determine incidence and risk factors for illness.
3. To refer persons for appropriate medical care.

1.2 Laboratory and Physician Reporting Requirements

1. Physicians are required to report confirmed, presumptive and suspect cases within 1 week of diagnosis.
2. Laboratories are required to report positive test results within one day.

1.3 Local Health Department Reporting and Follow-Up Responsibilities

Report all confirmed and presumptive (but *not* suspect) cases as soon as possible, but no later than the end of the calendar week of the initial physician or laboratory report.

1. Begin follow-up investigation within 3 working days. Complete the *Taenia* case report form and send a copy to Oregon Public Health Department (PHD) within 7 days of initial report. Fax or attach a copy to case file in Orpheus.
2. Case definitions and routine investigation steps are not the same for taeniasis and cysticercosis. Refer to the appropriate section in the guidelines below.

2. THE DISEASE AND ITS EPIDEMIOLOGY

There is some potential for confusion with these reportable conditions, as they do not follow the usual one bug:one illness paradigm. In effect we have 2 different parasites causing 3 distinct clinical syndromes. Deep down we really only care about one of the syndromes from one of the bugs, but the vagaries of diagnostic testing and the potential for multiple syndromes in the same person mean that we have to track all of these combinations. With luck (and a few re-readings) this will all make perfect sense.

2.1 Etiologic Agent

These are infections with cestode parasites in the genus *Taenia.* There are two species of interest: *T. solium* (the “pork” tapeworm) and *T. saginata* (the “beef” tapeworm). *T. solium* has a complex life cycle that can lead to humans being infected by one or multiple forms of the parasite. *T. saginata* has a simpler life cycle and chooses not to venture beyond the confines of the human intestine. Sometimes these various forms are referred to with names that make them sound like different species; they aren’t—just different stages in the life cycle. So “taeniasis” is having the adult tapeworm and “cysticercosis” is having the larval stage (cysticerci); some people have both, or they may have just tapeworms but be at risk for developing cysticercosis later.

If it were just about tapeworms in the gut, these would be pretty benign bugs, with some abdominal discomfort and occasional feelings of disgust or horror when visible tapeworm fragments emerge with stool. Cysticercosis, however, is potentially life-threatening, because the *T. solium* (but not *T. saginata*) larvae have a tendency to encyst in the brain, leading to a variety of neurological problems.

The other problem is that common laboratory methods rarely distinguish *T. solium* from *T. saginata*. The eggs look exactly the same, so if the diagnosis is based on seeing eggs in an O&P you won’t know which parasite they have; even mixed infections are theoretically possible.

Except as noted, these guidelines presume that we are talking about *T. solium* infections.

2.2 Description of Illness

Intestinal infection with the adult form of the parasite (the tapeworm) is called **taeniasis**. Taeniasis is usually asymptomatic, although mild, non-specific abdominal symptoms including nausea, anorexia and abdominal pain are sometimes reported. Persons with intestinal taeniasis may pass readily visible worm segments (proglottids) in stool. Proglottid fragments are typically white and measure about 5 x 10 mm.

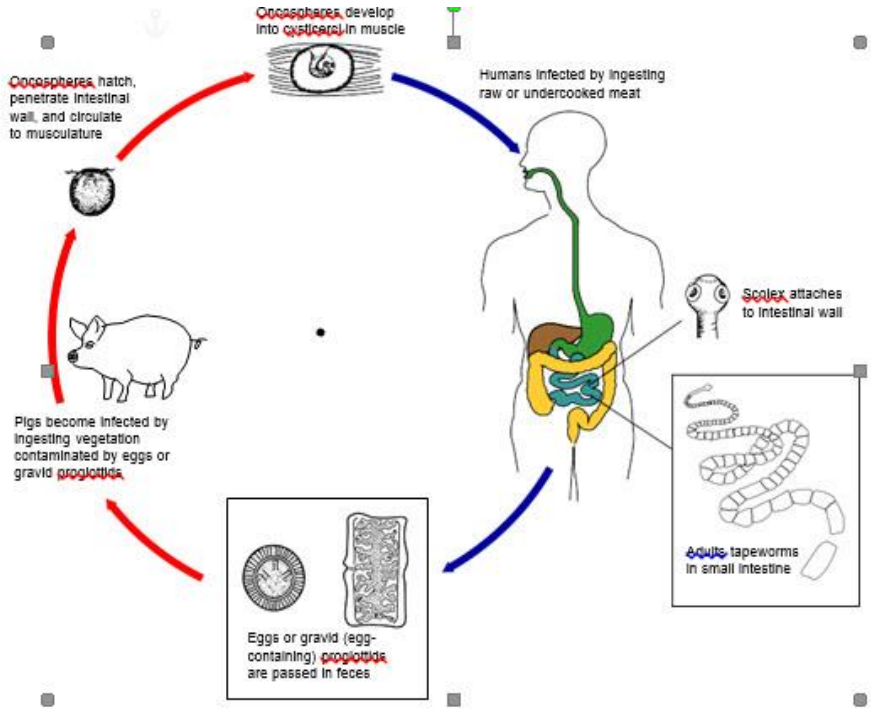
Cysticercosis is defined as infection of any tissue, usually in the central nervous system or muscle, with the larval form of the parasite. The corresponding symptoms depend on the number, location, and stage of development of the larval cysts, as well as on the host’s immune response. Neurocysticercosis is common, occurring when the larvae encyst in the brain or other CNS tissue. Neurologic signs and symptoms can be quite variable. Seizures result from inflammation surrounding dead or dying cysts in the brain parenchyma, and occur in up to 80% of neurocysticercosis cases. Headaches—often severe and protracted—are also common and present in around 40% of cases. Hydrocephalus and increased intracranial pressure, with associated chronic or recurrent headaches, nausea and vomiting can result from blockage of cerebrospinal fluid flow by cysts and/or inflammation in the cerebral ventricles. Untreated hydrocephalus may result in sudden death due to brain herniation.

Other neurologic symptoms of neurocysticercosis include psychiatric disturbances, balance problems, cognitive impairment, visual disturbance, focal weakness or paresthesias. Cysts may also develop in skeletal muscle, skin, or other organs, but these usually cause few if any symptoms.

2.3 Reservoirs and Life Cycle

To understand the epidemiology of these infections, it is necessary to master at least the rudiments of the pork tapeworm life cycle. The "natural" cycle of *T. solium* alternates between humans and pigs. Humans get tapeworms by eating undercooked and "measly" pork, which is tissue from pigs with cysticercosis—i.e., parasite cysts had formed in their muscles (the meat) at least. (Whether they also had bad headaches is hard to say.) Once ingested, the larvae "excyst" and attach to the wall of the small intestine as baby tapeworms. Over a period of months they develop into hermaphroditic adult tapeworms that may attain several meters in length. Tapeworms can survive in the human intestine for many years, intermittently shedding eggs and worm fragments in stool; these are immediately infectious to both humans and pigs. If ingested, these eggs develop into larvae, invade the intestinal wall and are then carried via the bloodstream throughout the body. Eventually the larvae embed themselves in soft tissues and become encapsulated: cysticercosis.

Figure 1: T. solium Life Cycle, Version 1



Study the first life cycle above (Figure 1)—if you are more fascinated than repelled, consider becoming a parasitologist. Notice that since we don't usually eat other people, humans with cysticercosis are a dead-end host in the *T. solium*

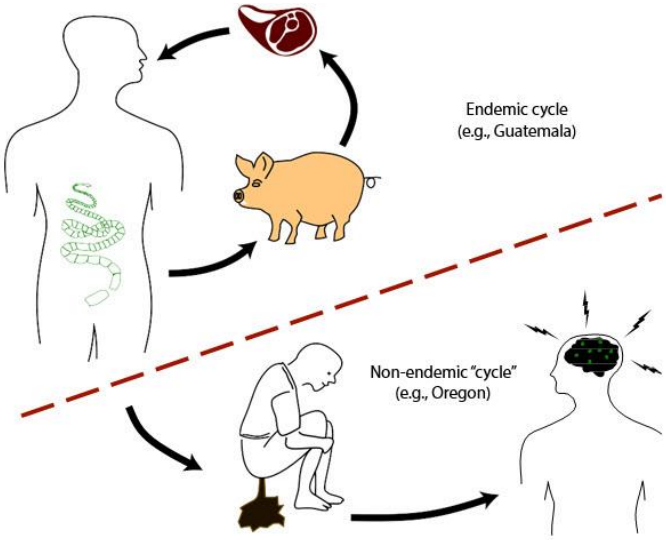
lifecycle. Some people do eat pigs though, and sometimes these are pigs with cysticercosis. To summarize, while both pigs and humans can develop cysticercosis, tapeworms only develop in humans; thus, only humans can shed eggs. In other words, while the most common scenario is a pig-human cycle, it is possible (and very dangerous) to have a human-human cycle. It is not possible to have a pig-pig cycle. In other words, although not recommended for other reasons, eating pig feces is *not* a risk factor for cysticercosis. Eating human feces is quite another matter, as that is how both people and pigs get cysticercosis.

Interestingly, tapeworm carriers are a danger not only to other persons, but also to themselves. Since the eggs shed in stool are immediately infectious, ingestion of human feces (including your own) can lead directly to *in vivo* larval development (i.e., cysticercosis). The risk of auto-infection is high enough that most tapeworm carriers also have cysticercosis at some point, though often it is asymptomatic.

Animal husbandry practices in the United States usually prevent pigs from eating human feces, so *T. solium* contaminated pork is rare in this country. (Factory farms are not all bad.) Where pigs and humans commingle, pigs can and do eat human waste and pork is often infested. This is tapeworm heaven. Conversely, *T. solium* infections are rarely seen in countries where pork is not consumed.

Consider the *T. solium* life cycle as from the perspective of an Oregon (public) health practitioner. Note that while tapeworms are almost invariably acquired in endemic areas, cysticercosis transmission can and occasionally does occur in non-endemic places, like Oregon—wherever human tapeworm carriers are found. Primary cysticercosis in Oregon is almost invariably the result of exposure to a carrier from an endemic country (see Figure 2). The public health priority is identifying and treating these tapeworm carriers before they cause cysticercosis.

Figure 2: *T. solium* Life Cycle, Version 2



Why are we so blasé about infections with the beef tapeworm (*T. saginata*)? Well, as evolution would have it, there is a crucial distinction between these two closely related parasites. People can develop the tapeworms if they eat measly beef, but the eggs and segments shed in human feces are not infectious to humans—only to cattle. So there is an obligatory human-bovine life cycle. Maybe cows go mad if they develop cysticercosis, but that isn't our problem.

Reread this section and study the figures until it makes sense to you. Both versions of the life cycle portray essentially the same story; we just couldn't decide which one was better.

2.4 Modes of Transmission

To recap, you get the tapeworm infection (taeniasis) by eating undercooked pork that was contaminated with larval cysts. You get cysticercosis via the fecal-oral route by consuming the feces of a human tapeworm carrier, either directly or indirectly. Tapeworm carriers often infect themselves via the fecal-oral route and develop concurrent cysticercosis as a result; this is called "auto-infection."

2.5 Incubation Period

Eggs may appear in human stools within 2–3 months after ingestion of larvae. Onset of symptoms of cysticercosis is delayed and variable. Most present within 5 years of exposure, but latencies of up to 30 years have been reported.

2.6 Period of Communicability

The life span of adult worms (and concomitant egg shedding) is usually less than 5 years. Some experts think that infections occasionally persist in some individuals for decades, but this is controversial. Eggs can survive in the environment for several months. Again, persons with cysticercosis are infectious only if they are simultaneously infected with adult worms.

2.7 Treatment

Treatment of taeniasis is simple and effective. There are two single-dose options:

- Niclosamide, adults 2 g once, children 50 mg/kg once, or
- Praziquantel, 5-10 mg/kg once

Some recommend treatment with a purgative if niclosamide is used; others do not. This is because the tapeworm can regenerate completely if the scolex is retained. (The scolex is the "head" end of the tapeworm with the hooklet structures that attach to the bowel wall.)

Niclosamide is arguably the safer drug because it is not absorbed by the GI tract and, therefore, has no activity against cysts. Inadvertently killing cysts in the CNS can cause inflammation and precipitate seizures. Unfortunately, niclosamide is not easy to get; local pharmacists will not carry it and most will tell you that it is "unavailable." It is available from some compounding pharmacies and ACDP staff will help you get it, if required.

Praziquantel is active against both tapeworms and cysts, so one must be cautious in using it to treat tapeworms in patients who may have coexisting neurocysticercosis (NCC). Patients should be evaluated for signs and symptoms of NCC before treatment and be monitored for adverse reactions (e.g., headache and seizures) following treatment that may indicate an acute inflammatory response to dying parasites.

Neither treatment is always 100% effective and re-treatment may be necessary. Documentation of parasite clearance, either by visualization of the scolex in a post-treatment fecal specimen or by fecal antigen performed at CDC is desirable, although that may be easier said than done.

Cysticercosis treatment is complex and expert medical consultation is recommended. Albendazole and praziquantel are both effective against CNS cysts. Not everyone benefits from anti-helmenthic therapy, however. Cysts often do not cause symptoms until they are already dead or dying and treatment may paradoxically increase symptoms by killing viable cysts, resulting in an increased inflammatory response. Control of inflammation around dying cysts may require prolonged corticosteroid therapy. Seizures generally require anticonvulsant medications, often lifelong. Neurological intervention for excision of cysts or shunting of cerebrospinal fluid may be required.

3. CASE DEFINITIONS, DIAGNOSIS AND LABORATORY SERVICES

3.1 Taeniasis

Case definitions for taeniasis are more straightforward. As has been noted, we can rarely distinguish beef from pork tapeworm infections unless the person also has cysticercosis.

Confirmed Case Definition

- Presence of *Taenia* eggs, proglottids or scolex in a fecal or pathologic specimen *or*
- Positive ELISA test for *Taenia* antigens in stool (“coproantigens”). (This test will likely be unavailable as it is no longer offered at the CDC.)

Presumptive Case Definition

- History of passing worm segments *and* close contact with a cysticercosis case (confirmed or presumptive) *or*
- Positive EITB serologic assay for antibodies against *T. solium.*

Diagnosis of taeniasis

Taeniasis is diagnosed by identification of eggs or worm segments in a stool ova and parasite (“O&P”) microscopic exam. The O&P exam is available at the OSPHL. Send fresh stool transported in both Formalin and PVA media for the O&P testing panel. Unfortunately, *T. solium* eggs cannot be distinguished from *T. saginata* eggs, nor can proglottid segments unless they are gravid (egg-

containing). In the rare case that a tapeworm scolex (head) is found, the species can be determined by the mouth parts. Unfortunately, most O&P exams are at best generic: typically reported as undifferentiated "*Taenia* sp." or something like that. Due to the intermittent excretion of eggs and proglottids, O&P exams are rather insensitive, positive in only 30-40% of cases detected by other methods. If indicated, obtain and submit stool samples from up to three different stool episodes.

Coproantigen ELISA tests can detect *Taenia* antigens in stool, but do not distinguish between *T*. *solium* and *T. saginata*. The sensitivity and specificity of these tests are approximately 95% and 99% respectively, making them the preferred "test-of-cure." A positive coproantigen test 2 months after treatment for taeniasis should prompt repeat treatment. Unfortunately, these tests are no longer available at the CDC.

Serologic (antibody) tests for *T. solium* have also been developed at CDC and have high sensitivity and specificity—but the catch, again, is that they are not commercially available (see §3.1.D).

3.2 Cysticercosis

Case definitions for cysticercosis depend on combinations of diagnostic criteria outlined in the table below. If it seems complicated, it may be because it is. We didn't make this stuff up; these are time-honored classifications developed by experts that apparently work reasonably well in practice.

Criterion Type	Finding
Absolute	• Histologic identification of the parasite • Visualization of the parasite by fundoscopic examination • Evidence of cystic lesions showing the scolex on CT or MRI
Major	• Evidence of lesions suggestive of neurocysticercosis on neuroimaging studies • Detection of *T. solium* cysticercal antibodies • Characteristic "cigar-shaped" calcification in thigh and calf muscles on X-ray
Minor	• Presence of subcutaneous nodules (without histologic confirmation) • Evidence of punctuate soft-tissue or intercranial calcification on X-ray • Clinical manifestations suggestive of neurocysticerosis • Disappearance of intracranial lesions after treatment with anticysticercal drugs
Epidemiological	• History of living in or frequent travel to a presumptively endemic area • Close (e.g., household) contact with a tapeworm carrier

Confirmed Case Definition

- 1 absolute criterion *or*
- 2 major criteria *or*
- 1 major *and* 2 minor *and* 1 epi criterion.

Presumptive Case Definition

- 1 major *and* 2 minor criteria *or*
- 1 major *and* 1 minor *and* 1 epidemiologic criterion *or*
- 3 minor *and* 1 epidemiologic criterion.

Suspect Case Definition

- 1 major *or*
- 2 minor *or*
- 1 minor *and* 1 epidemiologic criterion.

Diagnosis of cysticercosis

CT (non-contrast) or MRI scans of the brain or spinal cord can confirm a diagnosis of neurocysticercosis if a clear parasite scolex is visualized within a cyst. CT scanning is more sensitive for detecting brain calcifications and is usually the first imaging modality; MRI is better at detecting viable intraventricular cysts. CT and MRI findings are often non-specific, however.

Plain radiographs of skeletal muscle may show numerous calcifications, which suggests but does not confirm the diagnosis without other supportive data.

EITB (enzyme-linked immunoelectrotransfer blot, ""immunoblot") serological assays are available at CDC. The sensitivity is 94-98% in persons with two or more non-calcified cysts, but <50% in those with a single cyst. Specificity approaches 100%, but positive results may indicate past exposure rather than currently active disease. For Immunoblot testing, obtain blood or serum and refrigerate before transporting to the OSPHL. Save time and headaches by following the simplified instructions for submitting specimens to CDC on OSPHL's "Cysticercosis, Total Antibody" page: (http://public.health.oregon.gov/LaboratoryServices/Pages/AllLabTests.aspx).

Commercial ELISA tests are available, but they lack the sensitivity and specificity of the EITB. ELISAs work better for CSF specimens than for blood.

The diagnosis can be confirmed by pathologic exams of biopsy or autopsy material — or even by fundoscopic examination — if retinal cysts are present, but this isn't common.

3.3 Services Available at the Oregon State Public Health Laboratory

O&P exams are readily available, although the limitations of such tests have been noted. Where indicated, the ACDP epidemiology staff will arrange other testing (e.g. immunoblot (IB)) through the CDC.

4. ROUTINE CASE INVESTIGATION

The most pressing public health need is to identify and treat *T. solium* tapeworm carriers (i.e., egg shedders). This is straightforward if they have taeniasis, as the person with this diagnosis is the one you are looking for. With cysticercosis the

tapeworm carrier *might* be that same person, it might be a close contact or it might be a street vendor in Michoacan that they bought a tamale from several years before—i.e., someone unknowable and far away in space and time. These latter carriers are lost causes for us.

Interview the case and others who may provide pertinent information. Use the appropriate case investigation form for taeniasis or cysticercosis to help structure your interview(s). Make sure you are using a current form from the ACDP website or its electronic equivalent.

4.1 Taeniasis

1. Evaluate the Diagnosis

If the tapeworm has been identified as *T. saginata*, then no further public health intervention is required; this is a benign condition that can be treated by the physician without our help. This will rarely if ever happen, however. If the report specifies *T. solium* or simply undifferentiated *Taenia* sp., pray continue.

2. Identify Source of Infection

Determine whether the person infected with the tapeworm has traveled or lived in an endemic area; this will usually be the case. “Endemic areas” can be defined loosely as almost any developing (non-Muslim) country. Recall that tapeworms are long-lived and the exposure could have occurred years—possibly decades—prior to diagnosis. If there is no history of international travel or residence, further inquiries for the source of contaminated pork may be warranted. Discuss with ACDP staff.

3. Facilitate and Document Appropriate Treatment

Verify treatment details with the tapeworm carrier or physician, noting the medication name and the date taken. The goal is to encourage, facilitate and verify appropriate treatment; ultimately we are not in a position to supervise their clinical management. Niclosamide is the treatment of choice, but it is only available commercially from a few compounding pharmacies. The ACDP will facilitate acquiring the medication, if requested. If the care provider plans to use praziquantel, encourage appropriate screening for co-existing neurocysticercosis prior to treatment. This could be as simple as asking about symptoms of neurocysticercosis or may require imaging such as a CT of the head or MRI of the brain.

It is worthwhile to do follow-up exam; collect a follow-up stool sample 2 months after treatment. Send the specimen to the OSPHL for an O&P exam. Note that O&P exams are *not* sufficient to verify cure. If the 2 month follow-up stool sample shows an infection, work with the patient’s clinician to facilitate and verify repeat treatment. (Note: the only definitive way to do a test-of-cure is via the coproantigen ELISA testing. Unfortunately, this testing is no longer offered at the CDC.)

4. Identify Potentially Exposed Individuals

Screen the tapeworm carrier, household members and close contacts for symptoms of neurocysticercosis. Symptoms consistent with neurocysticercosis include: seizures, chronic or persistent headaches, cognitive changes, vision changes and focal neurologic deficits. Recommend medical evaluation for neurocysticercosis in any person with consistent symptoms. The person with symptoms is ultimately responsible for following through; however, they should at least be made aware of the possibility of tapeworm larvae growing in their brains.

5. Environmental Evaluation

Although not covered by current rules, we strongly recommend that commercial food handlers not return to work until they have been treated with niclosamide or praziquantel. Make sure that infected persons understand the importance of thorough hand-washing. This isn't Norwalk; tapeworm carriers can kill people if they aren't careful. As with all cases, arrange for a follow-up O&P exam 2 months after treatment. Note that the O&P exam is *not* sufficient to verify cure.

4.2 Cysticercosis

1. Evaluate the Diagnosis

Review available clinical details, recording signs, symptoms, laboratory and pathologic results and epidemiologic criteria. Classify the case as confirmed, presumptive or suspect according to the diagnostic criteria listed above. Continue with the public health guidelines outlined below for **confirmed** and **presumptive** cases only.

2. Identify Source of Infection

Determine whether the case has traveled or lived internationally; this will usually be the case. If that is NOT the case, contact ACDP epidemiologists immediately; this suggests the presence of a tapeworm carrier domestically (possibly a household contact).

In all cases a search for the tapeworm carrier should begin with the cysticercosis patient; remember the "auto-infection" cycle—that doesn't mean they got it from a car. Next, consider other household members and close contacts. This is fecal-oral transmission, so think about sexual partners, food preparers and others that may require screening. Screen the index case and all identified close contacts for symptoms of tapeworm infection, including abdominal discomfort, diarrhea and passing worm segments in the stool. All close contacts should also be screened for laboratory evidence of tapeworm infection. This involves obtaining a fecal specimen from each individual and sending it to the OSPHL for O&P exam by light microscopy. Complete a separate taeniasis report for any individual identified as a tapeworm carrier.

Again, the goal is to encourage and verify appropriate treatment of these tapeworm carriers so they cannot infect others. Treatment of neurocysticercosis can be extraordinarily complex—leave this to the experts.

3. Identify Other Potentially Exposed Individuals

Because they may share the same exposure risk as the index case, all household and close contacts should be screened for symptoms of neurocysticercosis. Symptoms consistent with neurocysticercosis include: seizures, chronic or persistent headaches, cognitive changes, vision changes and focal neurologic deficits. Document these screening symptoms and any relevant comments on the case investigation form. Recommend medical evaluation for neurocysticercosis in any person with consistent symptoms. The person with symptoms is ultimately responsible for following through; however, we feel they should at least be made aware of the possibility of tapeworm larvae growing in their brains.

5. CONTROLLING FURTHER SPREAD

5.1 Identify and Treat Tapeworm Carriers

As indicated above in “Routine Case Investigation” instructions.

5.2 Patient/Household Education

Provide basic instruction to cases and close contacts about the parasite lifecycle; about hand-washing after defecation, diaper changing and before food preparation; about the importance of proper food handling and adequate cooking of pork; in general, provide pointers about minimizing fecal consumption in daily life.

UPDATE LOG

December 2015: Updated testing methods and restructured IG. (Kathleen Rees)

November 2015: Transferred IGuide into new template. (Leslie Byster)

September 2014: Update submission of case report form §1.3. (Genevieve Buser)

June 2014: Update testing methods. (Genevieve Buser)

May 2008: New guideline written. (Seth O’Neal, John Townes & Bill Keene)