Trichinella is probably best known as a parasite that humans contract from eating raw or undercooked pork. Through meat inspection programs, the incidence of trichinosis in pigs has been lowered to less than 1%, so it is unlikely (but not impossible) that pork products purchased in your local supermarket will contain \textit{Trichinella} larvae. Most recent outbreaks of trichinosis in the North America have been traced to pork products from pigs that have not been inspected and that have been slaughtered privately.

How do pigs become infected?

Because of its low host-specificity, almost any "wild" meat should be considered suspect, and hunters should be careful when preparing meat from their kills. In particular, a number of infections have been traced to contaminated bear meat.
Trichinella sp.

Intestinal: diarrhea, nausea, abdominal cramps and malaise
Muscle: fever, periorbital edema, muscle pain, swelling, weakness; damages chewing and swallowing; myocarditis
10-20%: CNS involvement
High eosinophilia: 20-90%

(by P.W. Pappas and S.M. Wardrop)
The disease in humans is zoonotic and is USUALLY a dead end for the parasite. In the domestic situation the parasite principally cycles between pigs and rats with man as the incidental host. However, it also cycles in game animals such as polar bears and black bears. Animals such as wild pigs, cats, dogs, and walrus meat have all been implicated as a source of infection.
Life Cycle

Unlike many parasites that demonstrate a high degree of host specificity, *Trichinella spiralis*, can be found in many species of carnivores and omnivores.

The definitive host becomes infected by eating raw or poorly cooked meat containing infective L1 stage larvae encysted in the muscle fibres. Stomach-small intestine- the worm is released from the cyst- rapidly penetrates the mucosa where it undergoes successive moults to become young adults, within 30 hrs post-infection. These young adults mate deep within the mucosa where they are regarded as being intracellular parasites, lying within a serial row of cells.

The female worms produce about 1500 larvae over about 4-6 weeks. Males die soon after copulation and the females die shortly after larviposition. Juveniles enter the lymphatics and mesenteric veins and are found throughout the arterial circulation between the 7th and 25th day after infection. They travel in the circulatory system through the liver, then to the heart, lungs. In striated muscles they penetrate individual muscle cells and cysts are formed. Within cysts, juveniles remain viable for many years, up to 25 years in man and 11 years in pigs. They have a predilection for highly active muscles such as the tongue, intercostal muscles, diaphragm, eye muscles and the muscles of the arms and legs.
Trichinella spiralis
The larvae absorb nutrients from the muscle cells and increase their length to about 1 mm.

Juveniles may migrate directly or be carried by circulatory system to skeletal muscle- penetrate individual fibres

Host cell gene expression is modified – contractile cell changes to a type more reminiscent of a nurse cell- that now serves to provide nutrients to the parasite. Fibre loses myofilaments, nuclei enlarge, smooth ER increases, mitochondria decrease, entire unit becomes encapsulated in collagen- so called cyst- S/E compounds produced in nematodes induce these changes

Larvae form a spiral shape, where they remain dormant until eaten and enter the digestive system of the next host. The muscle cell containing the larvae is surrounded by infiltrating lymphocytes and result in a cyst wall surrounding the cell. The cyst wall becomes gradually thicker and eventually calcified. The life span of the larvae is finite because eventually the larvae itself becomes calcified. However, this is a slow process and larvae have been known to remain viable for up to 30 years.
Material secreted from nematode stichocytes is thought to be secreted into muscle fibres- aiding in the changes- this is found in J1s but not in muscle cells- but is found in nurse cells 7 days after penetration. Injection of this S/E liquid from nurse cells into non-parasitized cells induces similar response.
Larva enters muscle, muscle proteins leak into bloodstream
Mitochondrial Damage
Secretion of parasite Proteins
DNA Synthesis and Nuclear Enlargement
Collagen Type IV Synthesis
Collagen Type VI Synthesis
Induction of VEGF
Angiogenesis
Pathology
The first symptoms of the infection occur usually about 1-2 days after ingestion, but these are generally mild and not easily diagnosed, intestinal discomfort.

When the gravid females penetrate the intestinal epithelium, the resultant trauma, coupled with secondary bacterial infection and toxic shock in response to parasite waste products, leads to inflammation of the mucosa and pain giving symptoms similar to food poisoning, nausea, vomiting and diarrhea. In some cases patients also have respiratory difficulties and red blotches appear on the skin.

This phase terminates 5-7 days following the onset of symptoms. During the migration phase, which can result in the larvae appearing in almost any tissue, they can cause pneumonia, pleurisy, encephalitis, meningitis, nephritis, deafness, peritonitis, eye damage and so on. Death can result from myocarditis (Inflammation of the heat muscle).

Ten days after the initial symptoms, the larvae penetrate the muscle fibres, the resultant symptoms are dependant on the intensity of the infection. However, symptoms such as intense muscular pain, difficulty breathing or swallowing, swelling of the neck muscles can occur. In addition weakening of the pulse accompanies, lower blood pressure, damage to the heart and the onset of nervous disorders such as hallucinations. Death can occur through heart failure, respiratory complications, or kidney failure.
Abdominal symptoms can occur 1-2 days after infection. Further symptoms usually start 2-8 weeks after eating contaminated meat. Symptoms may range from very mild to severe and relate to the number of infectious worms consumed in meat. Often, mild cases of trichinosis are never specifically diagnosed and are assumed to be the flu or other common illnesses.

<table>
<thead>
<tr>
<th>TIME post-infection</th>
<th>Stage of infection</th>
<th>Progressive disease symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>12 hrs-2 days</td>
<td>Initial penetration and development of larval stages</td>
<td>First symptoms, mild non-descriptive</td>
</tr>
<tr>
<td>30-32 hrs</td>
<td>Copulation and female penetration of mucosa</td>
<td>Intestinal inflammation and pain, nausea, vomiting, diarrhoea. Terminates with facial edema and fever</td>
</tr>
<tr>
<td>5-7 days</td>
<td>New born larvae released into tissues and start migration</td>
<td>Focal or localized oedema - face and hands. Pneumonia, pleurisy, encephalitis, meningitis, nephritis, deafness, peritonitis. Death from myocarditis</td>
</tr>
<tr>
<td>5 days - 6 wks</td>
<td>Larvae start to penetrate muscle cells</td>
<td>Muscular pain, breathing difficulties, swelling of masseter muscles, weak pulse and low blood pressure, damage to heart. Death as a result of heart failure, respiratory failure, toxemia or kidney damage</td>
</tr>
</tbody>
</table>
Nausea, dysentery, colic, vomiting, fatigue, fever, and abdominal discomfort are the first symptoms due to invasion by adult worms.

Headaches, fevers, chills, cough, eye swelling, aching joints and muscle pains, itchy skin, diarrhea, or constipation follow the first symptoms. If the infection is heavy, patients may experience difficulty coordinating movements, and have heart and breathing problems. In severe cases, death can occur.

Migrating juveniles cause pain as they invade muscle tissue; there may also be edema, delirium, cardiac and pulmonary difficulty, pneumonia, nervous disorders, deafness and delayed or lost reflexes.

Many cases are never diagnosed because of the vagueness of the symptoms. Symptoms may range from very mild to severe and relate to the number of infectious worms consumed in meat. Muscle biopsy can be conducted and involves pressing muscle between glass plates to look for cysts. Xenodiagnosis involves feeding suspected muscle to laboratory rats.
Trichinella can infect virtually all mammals. The adult worm lives in the gastrointestinal tract, while the infectious larval form is present in cysts in the striated muscles of these animals. Trichinosis is a zoonotic infection transmitted by eating undercooked meat which contains the Trichinella cysts.

The female produces larvae which migrate throughout the body and encyst in striated muscle. This causes inflammation and muscle pain. In fact, the larvae can invade almost any part of the body producing inflammation and disease which can mimic many different clinical syndromes.

The disease varies considerably in severity, depending on the number of organisms present and the location of the migrating larvae. Patients with 10 or less larvae are likely to be asymptomatic. 100 will cause significant disease while 1000 to 5000 can cause death.
Prevention –

Cook pork thoroughly (also flesh of bear, walrus, wild pigs). Cook all garbage fed to hogs.
Proper meat handling, ordinary curing and salting of pork products will not kill encysted juveniles.
Freezing is effective if carried out properly. The freezing requirements differ with the size of the meat. Pieces not exceeding 6 inches in thickness require 20 days at 5F, 10 days at -10F, 6 days at -20F. Larger pieces require longer periods. Quick freezing and storage for 2 days is effective.
**Epidemiology**

The incidence of trichinellosis, particularly in the US and Canada is low, mainly because of the rigorous meat inspection procedures, and the practice of freezing carcasses. The occasional outbreaks that do occur, are however frequently fatal.

These are usually traced to uncooked meats which are often found in European style sausage made by small backyard butchers, who are often supplied directly from small farms, instead of via the large regulated abattoirs.

The other source of contaminated meat is from wild game, killed and eaten on the trail by hunters. It is estimated that heavily infected meat can contain about >1 million infective larvae in a single mouthful of meat. A concentration of 5 gms/ Kg body weight is a fatal dose for humans. In general humans are incidental hosts and are susceptible to all the sylvatic species.
Trichinellosis

*Trichinella spiralis* is an interesting parasite for a number of reasons:

1. It is one of the smallest nematode parasite of humans
2. Although it has a single host life cycle, the host acts as both the definitive and intermediate host.
3. There are no free-living stages

There are four species of *Trichinella* recognised:

<table>
<thead>
<tr>
<th>Species</th>
<th>Hosts</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>T. spiralis</em></td>
<td>Domestic strain</td>
</tr>
<tr>
<td><em>T. nativa</em></td>
<td>Northern arctic sylvatic</td>
</tr>
<tr>
<td><em>T. nelsoni</em></td>
<td>Southern sylvatic</td>
</tr>
<tr>
<td><em>T. pseudospiralis</em></td>
<td>Avian strain</td>
</tr>
</tbody>
</table>

The epidemiological importance of *T. pseudospiralis* has not been assessed but would appear to cycle through rodents and birds of prey. This species appears to have low infectivity for pigs.
Four persons became ill with trichinellosis after eating meat from a wild boar hunted in Camargue, France. Nonencapsulated larvae of Trichinella pseudospiralis were detected in meat and muscle biopsy specimens. The diagnoses were confirmed by molecular typing. Surveillance for the emerging T. pseudospiralis should be expanded.
TRICHINELLOSIS - CANADA (REPULSE BAY)
Trichinellosis, Repulse Bay, Nunavut. After a significant outbreak of trichinosis in Repulse Bay, Nunavut's department of health is establishing a program to test walrus meat for the parasite that causes the disease. John Raven, an environmental health officer based in Rankin Inlet, said that after 16 people contracted the disease in Repulse Bay in December, the department decided to use that community in a pilot project. Hunters will soon be able to send the tongues of walrus they harvest to the lab in Rankin Inlet and find out within 24 hours whether the meat is infected or safe to eat. "The tongue has the highest concentration of trichinosis," Raven explained. "A walrus can have light infection, moderate, heavy, whatever, and if there's any infection in a walrus it will appear in the tongue."

Algeria
We report here a single case acquired in Algeria, by a Muslim worker. Shortly after returning to France, in November 2004, the patient developed the typical clinical and biological signs of the disease. At first, he claimed to have eaten only mutton - a most unusual host for Trichinella but the subsequent inquiry revealed that he had eaten barbecued leg of jackal (Canis aureus), which he captured while wandering in the countryside. The muscular biopsy of the patient was positive. Trichinellosis is rather infrequent in Muslim countries such as Algeria, as pork is forbidden, but the parasite circulates in wildlife (wild boars and jackals).

Between 1982 and 1999 blood samples were collected from 500 polar bears (*Ursus maritimus*) captured in the Beaufort and Chukchi seas, to determine the seroprevalence of Brucella species, *Toxoplasma gondii*, and *Trichinella* species infections. The bears were classified into four age groups, cubs, yearlings, subadults and adults. *Brucella* and *Toxoplasma* antibodies were detected and ELISA was used to detect *Trichinella* antibodies.

The overall seroprevalence of Brucella species was 5 per cent, and subadults and yearlings were 2-62 times (95 per cent confidence interval 1.02 to 6.82) more likely to be seropositive for Brucella species than adults and their cubs.

The antibody prevalence for *Toxoplasma gondii* was 6 per cent, and for *Trichinella* species 55.6 per cent. The prevalence of antibodies to *Trichinella* species increased with age (P<0.001).
Summary:

Ingested worms enter small intestine- released from muscle, enter intestinal mucosa

4 moults, growth, copulation, occur within 2 days

Adults lie in cytoplasm and thread through a series of epithelial cells

Females produce thousands of juveniles over 4 months

Juveniles may migrate directly or be carried passively by circulatory system

Reach skeletal muscle- penetrate individual fibres

Host cell gene expression is modified – contractile cell changes to type more reminiscent of a nurse cell- that now serves to provide nutrients to the parasite

Fibre loses myofilaments, nuclei enlarge, smooth ER increases, mitochondria decrease, entire unit becomes encapsulated in collagen- so called cyst

S/E compounds produced in nematodes induce these changes
Despommier called *Trichinella spiralis*:

1) The largest intracellular parasite: Adults are intra multicellular parasites in the host intestinal epithelium, juveniles live in nurse cells in skeletal muscle

2) The worm that would be a virus
Nematodes:

1) Mermithids: always kill their invertebrate hosts, free living and parasitic stage, transmission via ingested eggs or penetration by juveniles

2) Ascarids have no free living stage. Eggs are released into environment. Immature stages go on migratory walks in the host before returning to the GI tract.

3) *Trichinella* sp. can kill hosts (density dependent), never a free living stage, transmission via predation