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Trichinosis and Meat

Trichinosis is an exceedingly painful disease and is among the most dreaded of human illnesses. In 1835 a British medical student first discovered the parasite which was subsequently named *Trichinella spiralis* (figure 1); however, it was not until 25 years after the parasite's discovery that the source and importance of human infection was realized. In 1860 a German professor of pathology found both muscle and intestinal trichinae in the body of a 20-year-old girl who had died. Further investigation revealed that the girl's trichina infection originated from eating raw or partially cooked pork from an infected pig.

LIFE CYCLE – TRICHINELLA SPIRALIS

Human trichinosis may occur from ingesting infected meat that is raw or inadequately cooked. A level of one trichina per gram is generally considered a sufficient dose to produce clinical illness in man. Human symptoms are generally related to dosage and closely follow the stages of the parasite life cycle. The incubation period (time from consuming infective *T. spiralis* larvae to onset of clinical symptoms) is usually 9 days (ranging from 2 to 28 days).

The life cycle of *T. spiralis* infection in man, swine, and other meat-eating animals consists of three stages: intestinal, larval migration, and muscular.

Intestinal Stage. When viable trichinae are consumed with infected meat they are digested free from the meat in the stomach. The liberated larvae then enter the small intestine where they mature into adult worms within 2 days and then mate. The fertile female worm burrows into the intestinal lining where she deposits numerous young larvae into the lymph spaces. A single female may produce up to 1,500 larvae and persist in the intestinal tract for as long as 4 months. During the intestinal phase human symptoms may include nausea, vomiting, and diarrhea.

Larval Migration. The larvae are then transported to the heart by way of lymph and blood vessels. They enter the arterial blood supply and are distributed throughout the body in the skeletal muscles. Adequate blood supply enhances larval survival; therefore, they have an affinity for the diaphragm, tongue, and muscles of mastication. During larval migration fever, eosinophilia, muscular pains, edema, and enlarged lymph nodes may be apparent. Sudden appearance of edema of the upper eyelids is a common and early characteristic sign noted about 10 or 11 days postinfection.

Muscular. Upon penetrating muscle, larvae grow in size and begin to coil. In the muscle phase (the most critical stage) cachexia, generalized toxemia, edema, and dehydration occur. Death may follow. Treatment is directed toward the relief of symptoms. The course of the disease ranges from 2 to 8 weeks. Gradually the host tissues form a cyst around the parasite. The encysted larvae are trapped in the muscle tissue and cannot develop further until the tissue is digested by another susceptible animal.

Living larvae are generally considered an indication of recent infection. Calcification of cysts, an indication of death of the trichina larvae, may occur 6 to 18 months after infection of the host. However, viable larvae have been detected in man for as long as 40 years postinfection.

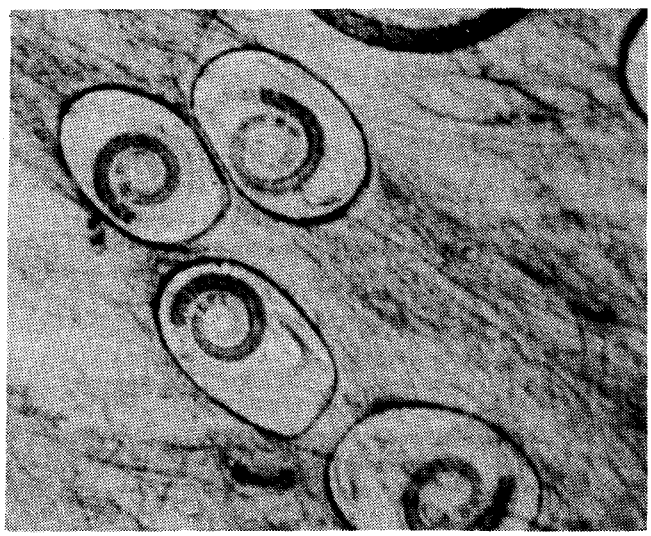

Figure 1. *Trichinella spiralis*, **the microscopic parasite that can cause trichinosis.**

HUMAN TRICHINOSIS – UNITED STATES

During the 10-year interval from 1967 to 1976, 1,308 human trichinosis cases were reported in the United States, with an average of 131 cases per year (ranging from 67 to 284). During the same interval, a case fatality rate of less than 1 percent, or 7.6 deaths per 1,000 cases was diagnosed.

A gradual decline in the incidence of human trichinosis in the U.S. has occurred since 1947. In contrast, during the preceding 20-year period (1947-1966) the annual average for human trichinosis was 277 cases. The increase in 1975 was related to an unusually large number of multiple case outbreaks. For example, in 1975 an investigation in Postville, Iowa revealed 67 human trichinosis cases; however, no deaths were reported. These patients were among at least 242 individuals who had consumed a locally produced pork/venison sausage. Studies confirmed the presence of viable *T. spiralis* larvae in the sausage. The suspect sausage had been produced by a custom locker plant using venison from deer shot by hunters during the 1975 hunting season. Since deer are rarely or never infected with trichinae, the pork purchased from a commercial source was suspected. Further investigation revealed that the locker plant operator had failed to heat the pork/venison sausage to 137°F or higher—the temperature necessary to destroy trichina. The Postville, Iowa epidemic is the largest single-source epidemic of human trichinosis ever reported in the United States.

During the same 10-year period there were six human trichinosis cases reported in Minnesota—2, 1, and 3 cases in 1969, 1970, and 1976, respectively. The two cases in 1969 were acquired in Mexico after consuming a meat product containing pork.

SOURCE OF HUMAN TRICHINOSIS – UNITED STATES

Humans most often become infected from eating raw or improperly prepared, trichina-infected pork or pork products. To a lesser extent man is infected from eating bear, seal, wild pigs, and other meats containing trichina larvae.

During the interval from 1967 to 1976, 969 human cases (74 percent) were attributed to the consumption of pork products; 190 cases (14.5 percent) were attributed to nonpork products; and 149 cases (11.5 percent) had an unidentified source of infection (table 1). The nonpork source was hamburger meat or bear meat. Although hamburger meat is generally considered to be pure beef, it may be adulterated either by contamination from a common meat grinder that was previously used to grind pork or the intentional mixing of beef and pork. With the American tendency to undercook beef products, the infective trichinae are not destroyed during food preparation. In 1975 New Jersey health authorities identified 28 cases of trichinosis in which the implicated meat source was ground beef.

Table 1. Sources of Infection–Human Trichinosis, United States, 1967-1976.

Food	Cases
Pork Products	
Fresh sausage	433
Wild pig	15
Other preparation	245
Unspecified	276
Nonpork Products	
Hamburger	95
Bear meat	40
Other beef products	12
Walrus meat	32
Unspecified	11
Unknown	149
TOTAL	1,308

In 1976 the Center for Disease Control, U.S. Public Health Service, initiated a preliminary survey in 12 states to obtain an estimate of the degree of hamburger adulteration with pork. Minnesota was not one of the 12 states sampled. Participating local and state health department officials submitted 231 ground beef samples purchased from 136 retail markets. Overall, 6 percent of samples were shown to contain pork, and 8 percent of stores had at least one sample that contained pork. Therefore, the consumption of raw or rare hamburger meat is discouraged. State and federal regulations prohibit the addition of pork to meat products labeled as ground beef or "hamburger."

Of the wildlife species known to harbor *Trichinella spiralis* in the U.S., none are more important as a direct source of human trichinosis than bears. A recent survey of 454 black bears from 10 states revealed that 3.1 percent were infected. Therefore, every bear should be considered a potentially infected animal and, thus, a human health threat. Hunters should be aware of the possibility of contracting trichinosis from the consumption of bear meat. *All bear meat should be thoroughly cooked.* From 1967 to 1976, 40 human cases of trichinosis were contracted from inadequately cooked bear meat.

TRICHINOSIS – SWINE

Swine and wild animals are the reservoirs of trichinae. Swine are usually infected by consuming viable larvae in pork scraps found in uncooked garbage and, to a lesser degree, by meat from carcasses of infected animals such as swine, dogs, cats, rats, foxes, and other carnivorous wildlife.

The prevalence of trichina infections in U.S. farm-raised swine has been declining steadily since 1898 from 14 per 1,000 hogs slaughtered to 1 per 1,000. In a recent study (1976) conducted in a Kentucky abattoir, only one of 513 sow diaphragm samples examined contained trichina. The prevalence was 2 per 1,000. The status of these sows relative to garbage feeding was not determined. There are three major reasons for the decline in swine trichinosis: 1) the required cooking of garbage fed to swine, 2) improvements in swine management, and 3) the decline in garbage feeding of swine. It should be emphasized that this decline of infection in swine is also reflected in the decreasing number of human trichinosis cases reported annually.

TRICHINOSIS PREVENTION

In the absence of a mandatory carcass-by-carcass inspection of pork for the presence of trichinae, the consumer must treat all uncooked pork as if it is infected. The stamp "U.S. Inspected and Passed" on raw pork products does not guarantee the product is free from infective trichina larvae. USDA specifications do require that "ready-to-eat" pork preparations be heated to an internal temperature of at least 137°F, which is sufficient to kill the parasite. For maximum tenderness, juiciness, and flavor with a minimum amount of cooking loss it is recommended that pork be cooked to an internal temperature of 170°F.

Freezing pork also will kill trichina larvae. When pork cuts less than 6-inches thick are frozen at 5°F for a minimum of 20 days, trichinae that may be present are rendered nonviable. Care should be taken to insure that home freezers actually will maintain a temperature of 5°F or less before total reliance is placed on this method as a measure to prevent trichinosis.

Dry curing, which is the interaction of salt and drying for relatively long periods, will devitalize trichinae cysts, if proper time-temperature relationships are established.

REFERENCES

The following readings are recommended for further information on the subject:

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