

# Pork as a source of human parasitic infection

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## Abstract

Foodborne zoonoses have been estimated to annually affect 10% of the global population, among which zoonotic parasites constitute an important class of aetiological agents. The major meatborne parasites include the protozoa *Toxoplasma gondii* and *Sarcocystis* spp., and the helminths *Trichinella* spp. and *Taenia* spp., all of which may be transmitted by pork. The significance of zoonotic parasites transmitted by pork consumption is emphasized by the prediction by the Food and Agriculture Organization of an 18.5% increase in world pork production over the next 10 years. Of all the porkborne parasites, the three 'T' parasites have been responsible for most porkborne illness throughout history; they are still endemic, and therefore are important public-health concerns, in developing countries. Although the risk of porkborne parasites, particularly helminths, may currently be considered insignificant in developed countries, the modern trend of consuming raw meat favours their re-emergence. This paper overviews the main parasites transmitted to humans by pork, and outlines the main lines of prevention.

**Keywords:** Pork, prevention, taeniasis/cysticercosis, toxoplasmosis, trichinellosis, zoonotic parasites

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## Introduction

The beginning of the twenty-first century is marked by an ever-high public interest in nutrition and, by extension, in food safety. This is reflected in an increasing awareness of foodborne biological hazards. Foodborne zoonoses are defined as diseases naturally transmitted between animals and humans through food. In developed countries, it has been estimated that up to 10% of the population annually suffer from foodborne zoonoses [1], among which zoonotic parasites constitute a very important class of aetiological agents. Humans acquire foodborne parasites through meat, water or faecal contamination of both food and water. The major meatborne parasites include the protozoa *Toxoplasma gondii* and *Sarcocystis* spp., and the helminths *Trichinella* spp. and *Taenia* spp. Interestingly, although consumption of other meat types may be a transmission route for some of these parasites, only pork can be a source of all

four. Although pigs can play host to a much wider range of zoonotic parasites (Table 1), only the species named here can be transmitted by eating pork and pork products. Of these, the three 'T' parasites are 'responsible for the lion's share of the burden of porkborne illness throughout the history of mankind' [8] and are still important public-health concerns in developing countries. According to the Food and Agriculture Organization, pork is the world's most consumed meat from terrestrial animals, with the demand rising in recent decades in developing countries with fast-growing economies. The pig production sector is (along with poultry) the fastest growing livestock subsector. It is estimated that there will be 1 billion pigs by 2015, which is double the number in the 1970s. Pig production is global (excluding the regions with religious limitations regarding the consumption of pork) and is characterized by an increasing dichotomy of production systems—specialized industrial farming versus traditional subsistence-driven small-scale production (backyard pigs).

**TABLE 1. Pig parasites with zoonotic potential: transmission routes from pigs to humans**

| Parasite  | Porkborne infection | Faeco-oral infection (waterborne, contaminated food etc.) | References |
|---|---------------------|---|------------|
| <i>Toxoplasma gondii</i>                          | +                   | –   | 2          |
| <i>Sarcocystis suihominis</i>                     | +                   | –   | 2          |
| <i>Balantidium coli</i>                           | –                   | +   | 2          |
| <i>Blastocystis hominis</i>                       | –                   | +   | 2          |
| <i>Cryptosporidium</i> spp.                       | –                   | + <sup>a</sup>  | 3          |
| <i>Entamoeba polecki</i>                          | –                   | +   | 2          |
| <i>Giardia</i> spp.                               | –                   | + <sup>a</sup>  | 3          |
| <i>Alaria</i> spp.                                | + <sup>b</sup>      | –   | 4          |
| <i>Taenia solium</i>                              | +                   | –   | 3          |
| <i>Taenia asiatica</i>                            | +                   | –   | 5          |
| <i>Ascaris suum</i>                               | –                   | + <sup>b</sup>  | 6          |
| <i>Trichinella</i>                                | +                   | –   | 3          |
| <i>Trichuris suis</i>                             | –                   | + <sup>b</sup>  | 6          |
| <i>Larva migrans</i><br>( <i>Toxocara canis</i> ) | + <sup>c</sup>      | –   | 7          |

<sup>a</sup>Not definitely confirmed.  
<sup>b</sup>Extremely rare.  
<sup>c</sup>Experimentally shown.

Although the risk of porkborne parasites, particularly helminths, may currently be considered insignificant in developed countries, the modern trend of consuming raw/rare meat and an increased demand for organic pork favours their re-emergence.

This paper provides a brief overview of the main parasitic infections transmitted to humans by pork, and outlines the main lines of prevention and preventive measures.

## Toxoplasmosis

Toxoplasmosis is a globally distributed zoonosis estimated to infect one-third of the global human population, and hence *Toxoplasma gondii* is the most significant protozoan foodborne agent and obviously the most significant protozoan that may be meat-borne. The disease burden of toxoplasmosis is by far the greatest of all parasitic infections, and has been estimated to be similar to that of the major foodborne diseases, salmonellosis and campylobacteriosis [9]. Despite the low incidence of toxoplasmosis, the disease burden of ~ 620 disability-adjusted life-years per year parallels that of *Salmonella* species (670 disability-adjusted life-years per year) [10]. In the USA, congenital *T. gondii* infection occurs in up to 5000 of the 4.2 million US newborns annually, which costs the society US\$8.8 billion [11]. It has been estimated that 50% of cases in the USA are foodborne [12].

The life cycle of *T. gondii* involves the Felidae as the definitive host and all other mammals (including both humans and pigs) as intermediate hosts. Unlike other parasites, the genus is comprised of a single species able to infect all hosts and all types of host cells. The fact that *T. gondii* can circulate between intermediate hosts only makes pigs a convenient source of human infection.

Worldwide, the prevalence ranges widely, from 8 to 92% [13,14]. Although generally higher in warmer and more humid areas, it has been shown to depend on the population's nutritional habits, and so varies even between geographically close areas [15]. In Europe, the last decades have been characterized by a continuing decreasing trend in the prevalence of infection [16–18]. Studies have convincingly shown undercooked meat as a major risk factor for infection [19–21]. Given the worldwide seroprevalence rate in pigs and the leading proportion of pork among all other meats globally, pork has traditionally been considered as the highest risk meat. Since pigs are continuously exposed to infection, the prevalence and risk of infection increase with age [22–26], making pork products that are traditionally made from meat from older animals especially risky for consumers.

*Toxoplasma gondii* infection is generally mild and self-limiting in immunocompetent individuals, but its clinical impact is traditionally associated with the unborn fetus, in which primary maternal infection in pregnancy can be detrimental, and the immunocompromised individual where reactivation of latent infection can cause life-threatening disease. A major consequence of congenital *T. gondii* infection is chorioretinitis, with toxoplasmosis being a major cause of infectious posterior uveitis worldwide [27], but in the past decades it has become clear that ocular disease of toxoplasmic origin may result at least as frequently from acquired disease [27,28]. This sheds new light on the need to prevent postnatal disease as well.

Diagnosis of *T. gondii* infection may be difficult. Although serological screening is widely available and reasonably simple, what may be tricky is the diagnosis of acute infection (more precisely, the dating of acute infection against conception), as well as proving the *T. gondii* aetiology of ocular or neurological disease. This is today largely resolved by the detection of parasite DNA in bodily fluids (amniotic fluid, ocular humour etc.)

There is currently no vaccine (except one for sheep used locally), whereas available therapies are unable to eliminate parasite cysts from the infected host. Hence, treatment is limited to acute and reactivated disease, characterized by the proliferative parasite form, and involves old drugs such as pyrimethamine and sulfa drugs, and newer ones such as clindamycin, azithromycin and atovaquone. Although the last of these has shown activity against cysts [29,30], prevention remains the best treatment for toxoplasmosis.

## Sarcocystosis

Three species of *Sarcocystis* have been recognized in pigs: *S. miescheriana*, *S. porcifelis* and *S. suihominis*. However, only

*S. suihominis* can cause an intestinal infection in humans upon consumption of raw pork [31].

The life cycle of *S. suihominis* includes humans or chimpanzees, rhesus and cynomolgus monkeys as definitive hosts (excretion of sporocysts with faeces) and pigs as the intermediate host [32]. In pigs the parasite encysts in muscular tissues but generally without causing pathological changes or symptoms [33]. Cysts can reach macroscopic size.

Little information on the prevalence of *S. suihominis* infection is available but its distribution is probably worldwide. In pigs, it has been detected in Germany, Japan, Thailand and India [34–37].

In humans, one study in Germany reported a 2% prevalence of *Sarcocystis* spp. infection [34]. *Sarcocystis suihominis* was found in stools of up to 7% of 926 persons in Tibet [38], and in 14 of 20 examined children who consumed raw salted pork tails [39].

Infection can be asymptomatic or symptomatic. Volunteers infected by pork containing *S. suihominis* had dramatic symptoms 6–48 h later, including bloat, nausea, loss of appetite, stomachache, vomiting, diarrhoea, difficulty in breathing, and rapid pulse [40]. In contrast, a scientist who infected himself began excreting sporocysts 12 days later and continued to excrete sporocysts for > 120 days, but remained asymptomatic throughout [41]. Sarcocystosis is a self-limiting infection, and prophylaxis and treatment are not known.

Diagnosis is based on microscopy, stool examination and epidemiological data. *S. suihominis* cannot be morphologically distinguished from *S. hominis* by routine microscopy and so data on consuming raw pork are important. In pork, *S. suihominis* may be detected by direct observation of macroscopic cysts, although microscopic examination will allow for more frequent detection.

## Trichinellosis

Trichinellosis is a worldwide zoonosis caused by the nematode *Trichinella* and at the world level is mostly transmitted by pork from backyard pigs. It can be a serious disease, particularly in the elderly, where severe complications such as myocarditis or encephalitis can lead to death.

Contamination occurs after consumption of raw meat containing coiled larvae 0.5 mm in length. The parasitic cycle can be divided into two phases: an intestinal (or enteral) phase and a muscular (parenteral or systemic) phase, which can co-exist for a period lasting from a few days to weeks. After gastric digestion of infected meat the larvae are released in the stomach, penetrate the mucosa of the small intestine, and mature into adult worms (5 days post-infection). The larval

penetration of the intestinal mucosa causes modifications in the epithelial cells, specifically, the brush border of villi, the lamina propria, and the smooth muscles of the jejunum. After mating in the intestine, adult females shed 100- $\mu$ m long newborn larvae (NBL) into the blood and lymphatic vessels. These larvae migrate in the general circulation to find their definitive niche, the musculoskeletal fibre. The circulating larvae induce parasitic vasculitis in their host. After penetrating the muscle fibre the larvae take control of the muscle fibre and, for most *Trichinella* species, induce the constitution of a collagen capsule surrounding the larva. The NBL will increase its volume by 600-fold within 2 weeks and become infective. The larvae will stay alive in the modified muscle fibre (called a 'nurse cell') for months or even years. Mature females release NBL for 3–4 weeks; although this estimate was based only on experimental data from pigs, it has been confirmed by the observation of a *Trichinella* female containing embryos on a duodenal section of a patient infected 3–4 weeks earlier and presenting with fever, myalgia and high eosinophilia [42]. The females then die or are expelled by smooth muscle hypercontractility elicited by the immune response. The prolonged diarrhoea observed in the Inuit population in the Canadian Arctic outbreaks suggests persistence of adult worms in the intestine of people frequently exposed to infection, showing that in humans immunity plays a crucial role in host protection [42].

Trichinellosis remains a globally important zoonotic disease. In an extensive review of published cases, Murrell and Pozio analysed 261 reports [43]. From 1986 through to 2009, they identified 65 818 cases causing 42 deaths in 41 countries. The WHO European Region accounted for 87% of cases; 50% of those occurred in Romania, mainly during 1990–99. Most outbreaks are related to the consumption of raw pork containing larvae of *Tr. spiralis*, the cosmopolitan and most prevalent species. Meats from wild animals (wild boar, dogs, bears, etc.) are a source of small outbreaks among hunters and their associated social groups (friends, relatives, etc.), and horsemeat has been implicated in a number of larger outbreaks. In these cases, species different from *Tr. spiralis* may be involved: *Tr. britovi* (Old World mountainous areas), *Tr. nativa* (wildlife from cold northern regions of the northern hemisphere), and *Tr. pseudospiralis* (cosmopolitan non-encapsulated species). *Trichinella zimbabwensis* in east Africa and *Tr. papuae* in Papua New Guinea are recently described species infecting reptiles. The most frequent species found in pigs is the cosmopolitan and domestic *Tr. spiralis*. However, *Tr. britovi* is frequently found in pigs, particularly wild boar. *Trichinella nelsoni* (African genotype) could be found in warthogs but this has not really been proven and *Tr. papuae* could be present in pigs from Papua New Guinea. The

cosmopolitan non-encapsulated species *Tr. pseudospiralis* has been responsible for outbreaks linked to pork in Thailand and to wild boar in France [44].

At the world level, pigs are the main vectors of the trichinellosis but their role depends on the mode of breeding. In large controlled farms with rodent and refuse control programmes, the incidence of the disease is minimal. Pigs at risk are backyard and free-ranging pigs; they can be a source of small family outbreaks. Feral pigs (wild boar, warthogs) can be a source of the disease in hunters and their families. In the past 25 years, outbreaks linked to pork consumption have been reported in, among others, Eastern European countries (Romania, Serbia, Croatia, Poland), Argentina, China, Laos. Social upheavals resulting in abandoning control at farm level and slaughterhouse veterinary inspection are often key factors for the emergence of these outbreaks. People at risk are those consuming raw or rare pork prepared as sausages, smoked ham or local recipes such as *lap mou* in Laos. The infective dose for humans is historically estimated to be around 50 larvae but mathematical models have estimated this infective dose to be lower [45].

In pigs, the parasite does not provoke any clinical signs. In humans, after an intestinal phase lasting for 3–7 days associated with abdominal pain and diarrhoea, the invasive phase of the disease is characterized by high fever, facial oedema and muscular pain. Cutaneous rash and subconjunctival haemorrhages may occur. This phase will last for 1 week but muscular pain will persist for several weeks. Severe, life-threatening complications may occur during the invasive phase. Although observed mainly in severe cases, complications have also been reported in moderate cases, in individuals who were improperly treated (including those for whom treatment was begun too late) and, particularly, in the elderly. A positive correlation has been reported between age and the frequency and severity of complications. Encephalitis and myocarditis are often simultaneously present. Cardiovascular disturbances occur usually later in the infection (between the 3rd and 4th weeks after infection). Myocarditis develops in 5–20% of all infected persons. The symptoms include pain in the heart region, tachycardia and abnormalities on electrocardiogram. Another cardiovascular complication is thromboembolic disease, specifically, deep thrombophlebitis, intraventricular thrombi, and pulmonary embolism, all of which can lead to death. Cardiovascular complications may be accompanied by oedema of the lower limbs due to hypoalbuminaemia. Sudden death may result from embolism of the pulmonary artery or from paroxysmal tachycardia. Neurological complications include a variety of signs and symptoms; patients with severe disease can show consciousness disorders or excessive excitement, and frequently somnolence and apathy, sometimes associated with

meningitis or encephalopathy. Dizziness, nausea and tinnitus are transient. Within a few days after the onset of fever, development of diffuse encephalopathy or focal signs such as disorientation, memory disturbances, frontal syndrome, behavioural disturbances, transient hemiparesis or hemiplegia, oculomotor dysfunction, aphasia and cerebellar syndrome is possible. Most computed tomography or magnetic resonance image brain abnormalities as well as the clinical signs and symptoms disappear in 4–8 weeks after infection. Although long-disputed, chronic trichinellosis may occur in untreated or inappropriately treated patients [42].

In pigs, the diagnosis is performed by searching for the larvae after chlorhydropeptic digestion of a sufficient amount of meat. Trichinoscopy with a sensitivity of around one larva per gram is not a recommended method. Serology can be used to detect infected pigs but false-positive results are possible, particularly with ELISA tests using crude antigens. The regulations vary according to country and details can be found in the guidelines produced by the Food and Agriculture Organization–World Organization for Animal Health–WHO collaboration, which are available online [42].

In humans, the diagnosis is suspected if grouped cases of patients with fever, facial oedema and muscular pain are observed. When cases are sporadic or the clinical course is atypical, it is more difficult to suspect infection or less likely that the infection will be suspected. Once infection is suspected, information on the consumption of raw or undercooked meat or meat products should be collected, including the place and time of purchase and consumption [42]. Table 2 gives an algorithm that can help in the diagnosis. High eosinophil counts associated with high levels of muscular enzymes (creatine phosphokinase, aldolase) are highly suggestive of the diagnosis. At disease onset, antibody detection (ELISA, indirect fluorescence) can be negative and should be repeated after several days. Specific antibodies can be detected earlier by Western blot analysis. Antibodies will be detectable in the serum months or years after acute disease. Muscular biopsy is useless during the acute phase of the disease because larvae are not encapsulated and not easily seen, but modifications of muscle fibres (disappearance of myofibrils, enlargement of nuclei with prominent nucleoli) can suggest the diagnosis. One month after the disease onset, larvae are easily seen in the muscles and can be used to precisely identify the parasite genotype. Detection in blood of parasitic DNA by PCR is efficient in experimental models but its sensitivity and specificity have not been proven in human infections [42].

The sooner specific treatment is introduced, the fewer cardiovascular and neurological complications occur. Drug treatment is based on mebendazole or, better, on albendazole (15 mg twice daily during a fatty meal) for 10–15 days.

**TABLE 2.** Algorithm for the diagnosis of acute trichinellosis

| Group A                     | Group B   | Group C  | Group D   |
|-----------------------------|---|--|---|
| Fever                       | Diarrhoea   | Eosinophilia (> 1 G/L) and/or increased total IgE levels | Positive serology (with a highly specific test) |
| Eyelid and/or facial oedema | Neurological signs  | Increased levels of muscular enzymes                     | Seroconversion                                  |
| Myalgia                     | Cardiological signs<br>Conjunctivitis<br>Subungual haemorrhages<br>Cutaneous rash |  | Positive muscular biopsy                        |

The diagnosis is:  
 Very unlikely: one A or one B or one C.  
 Suspected: one A or two B or one C.  
 Probable: three A or one C.  
 Highly probable: three A or two C.  
 Confirmed: three A, two C, and one D; any of groups A or B and one C and one D.

Prednisolone at 1 mg/kg once a day is administered for several days to alleviate symptoms and prevent complications [42].

## Taeniasis and Cysticercosis

Worldwide around 50 million people are estimated to be affected by cysticercosis and every year 50 000 deaths are caused by neurocysticercosis [46]. Among people with epilepsy, the proportion of neurocysticercosis is almost 30% [47]. *Taenia solium* infection is on the B list of infections of the World Organization for Animal Health.

For *Ta. solium*, humans are both the definitive and the intermediate hosts, whereas intermediate hosts also include pigs and dogs. As definitive hosts, humans carry the adult worm attached to the small intestine from which eggs are detached and excreted with faeces, contaminating the environment (up to 300 000 eggs per day can be excreted). Ingestion of these eggs via contaminated food and water by intermediate hosts will lead to tissue cyst formation—cysticercosis. Human taeniasis occurs by ingestion of *Ta. solium* cysts from infected undercooked pork.

The prevalence of infection depends on the level of economic development and hygienic conditions [48]. *Taenia solium* infection is traditionally endemic and a serious emerging disease in developing countries in South and Central America, sub-Saharan Africa and Asia and the Pacific Region, except in Islamic countries. Since 2000, in Africa the prevalence has been up to 64% in pigs and 10% in humans [49,50], in South and Central America up to 65.4% in pigs and 22% in humans [51,52], whereas in Asia it is up to 26% in pigs and 1% in humans [37]. This, however, does not mean that infection is necessarily associated with poverty and eradicated in developed countries. The increasing economic immigration from areas of endemicity into developed countries, as well as increased tourism in developing countries, confirm *Ta. solium* as 'a parasite with international health implications and the capability of spreading to non-endemic areas' [53]. Immigrants

and tourists alike may be just registered cases of cysticercosis [54], or they may also acquire taeniasis and as such present a source of eggs for autochthonous cases in developed or Islamic countries [55,56]. In the USA, cysticercosis is included among the five parasitic diseases that have been targeted by the CDC for public health action as neglected parasitic infections. A study of cysticercosis-related deaths in the USA has shown that at least one death of a US-born person was reported in each year of the study period (1990–2002) [57]. Within the European Union, *Ta. solium* infection can be acquired locally in some countries; pig infection has been reported in Austria, Estonia, Hungary, Lithuania, Poland and Romania [58], whereas only sporadic imported cases are registered in other countries.

Pigs are the source of *Ta. solium*, with cysticerci commonly found in muscles (heart, tongue, masseter muscle, diaphragm, shoulder muscles, intercostal muscles, oesophagus), whereas lymph nodes, liver, spleen, lungs and the brain are rare localizations. Infection is rarely associated with any symptoms.

Taeniasis in humans is of minor clinical significance, causing only mild inflammation at the implantation site, usually asymptomatic, or symptoms are mild and non-specific (abdominal pain, distension, nausea, diarrhoea or constipation). However, taeniasis leads to the spreading of eggs, which if ingested give rise to cysticercosis, which does have major clinical significance. Neurocysticercosis is the most important neurological parasitic disease in areas where *Ta. solium* is endemic [59]. Epilepsy and intracranial hypertension are the most common clinical manifestations but neurocysticercosis can cause almost any neurological symptom, and is the leading cause of late-onset epilepsy in endemic areas [59,60]. If intracranial hypertension and hydrocephalus develop, mortality may be considerable [61,62]. *Taenia solium* is the most common intraorbital parasite, with ocular localization occurring in 3–3.5% of all infections [63]. Retinal tissue damage, chronic uveitis or papilloedema, or chiasm compression related to cerebral cysticercosis, can cause loss of vision. Subcutaneous (rare in Latin America but very common in Asia and Africa) or muscular cysticercosis (including in the heart

in ~ 17% of patients) [64] is usually of no major clinical importance.

Diagnosis in pigs includes tongue palpation and serology, but as most pigs in endemic areas have been shown to carry only light infections [65] these methods are not sensitive enough. Routine meat/carcass inspection is of low sensitivity too, as only 11–18% of cysticerci [65,66] were located in organs indicated for routine meat inspection (heart, tongue and masseter muscle).

Examination of stool for *Ta. solium* eggs is the basic diagnostic method for taeniasis, but for differential diagnosis within the *Taenia* species, coproantigen detection by ELISA or PCR is required [67]. For cysticercosis, imaging techniques provide objective evidence on the number and topography of lesions and their stage of involution [61,68]. Serological tests are the most common diagnostic method, but it must be noted that they may be positive in patients who have been exposed to the adult parasite without developing cysticercosis [68], whereas even highly sensitive variants including enzyme-linked immunoelectrotransfer with highly purified cysticercus antigens may give false-negative results in up to 50% of patients with a single cerebral cyst or in those with calcifications alone [69]. Detection of circulating antigens has a poor sensitivity but may be of value to monitor the response to cysticidal therapy [67]. A relationship between genetic polymorphisms among *Ta. solium* metacestodes and differences in antibody detection in patients with neurocysticercosis has been established [70].

Taeniasis is treated with a single dose of niclosamide, and praziquantel or albendazole can also be used. For cysticercosis, however, a single therapeutic approach is not useful and management includes antiparasitic drugs (albendazole, praziquantel), symptomatic therapy (analgesics, antiepileptics, anticonvulsant drugs), anti-inflammatory drugs (corticosteroids), and surgery [71]. A standard regimen does not exist and treatment depends not only on viability and number of cysts but also on

the localization and number of lesions as well as on the host immune response [72].

Pigs are also intermediate hosts for *Ta. asiatica* whose cysticerci are predominantly located in liver or other viscera. However, *Ta. asiatica* is not known to cause human cysticercosis. Geographical distribution is presumed to be restricted to South-East Asia [5] but new data on infection in Nepal [73] point to a wider distribution. Generally, infection is possible in areas where raw pork liver or other viscera are habitually consumed. In the differential diagnosis, *Ta. asiatica* can be misclassified as *Ta. saginata* in the absence of molecular confirmation.

### Larval Alariosis

*Alaria alata* is a fluke, a few millimetres long, that is frequently found in the intestines of wild canids (foxes) or other carnivores and as a larval stage (mesocercaria) encysted in the muscles of frogs [4]. The cycle involves freshwater snails (planorbids). In Europe, *A. alata* mesocercariae have been found after digestion of wild boar (as paratenic hosts) muscles during *Trichinella* inspection [74]. The prevalence in wild boar is low (< 2%) but could be higher in wetland regions. In humans, seven cases of mesocercariasis (one fatal generalized and six ocular or subcutaneous cases) have been reported but only in North America with the species *A. americana* after consumption of raw frog legs [4]. The precautionary principle recommends considering boar meat parasitized by *A. alata* unfit for consumption.

### Prevention and Control

Porkborne zoonotic parasitic infections are a global problem, but control programmes must be decided in relation to the

**TABLE 3.** Methods for rendering pork and pork products safe for consumption

| Parasite species                     | Cooking <sup>a</sup>  | Freezing  | Curing   | Irradiation ( $\gamma$ -radiation source)High-pressure processing | Enhancement procedures  |
|--------------------------------------|---|---|--|---|---|
| <i>Toxoplasma gondii</i>             | Internal temp. 66°C [77]<br>time varies w/thickness and type of cut | -12°C/3 days [78]<br><0°C/7 days [79]   | Variability in standards, no safety recommendation [79–81] | 0.5 kGy [82,83]<br>400 MPa [84]                                   | 2% sodium chloride or 1.4% potassium or sodium lactate/8 h [79] |
| <i>Sarcocystis</i> spp.              | 60°C/20 min; 70°C/15 min; 100°C/5 min [85]                          | -4°C/48 h<br>-20°C/24 h [85]  |  |   |   |
| <i>Taenia</i> spp.                   | 60°C, or until it loses pink colour [86]                            | -5°C/4 days;<br>-15°C/3 days;<br>-24°C/1 day [87]                                     |  |   |   |
| <i>Trichinella</i> spp. <sup>b</sup> | Internal temp. 71°C, or until it loses pink colour [88]             | -15°C/20–30 days<br>-23°C/10–20 days<br>-29°C/6–12 days (depending on thickness) [88] | Unreliable [88]  | 0.3 kGy [88]  |   |

<sup>a</sup>Not in a microwave!

<sup>b</sup>Depending on legislation.

situation in a specific region or even at the local level. For instance, whereas *Ta. solium* has been virtually eliminated from developed countries, it is still endemic in a number of developing regions, where the choice of prevention strategy is limited by economic reasons.

Generally, prevention may be carried out at four levels, including farm, slaughter, post-slaughter processing and consumer level. At farm level, implementation of good management measures may lead to parasite-free farms. Strategies to reduce the level of parasite infection in pigs include raising animals in controlled zoo-hygienic conditions in strictly managed intensive-type farms (rodent control, no access for cats, feed and water control, no access for pigs to refuse). In Western Europe, raising pigs indoors with proper sanitation has been responsible for complete elimination of *Ta. solium* [75], and has been associated with the decrease of human toxoplasmosis cases. However, it should be kept in mind that the modern approach in farm management (to provide for the welfare of the animals) involving animal-friendly (organic) farms may in itself provide the environment for reappearance of parasitic infections, as illustrated by an increase in *T. gondii* infection in such farms in the Netherlands [76].

Prevention at slaughterhouse level comprises veterinary inspection at slaughter. This may, however, be limited by the applicability and sensitivity of the described diagnostic methods. Training of technicians, quality assurance and accreditation are compulsory measures. Organic pork and wild boar meat should be systematically examined for *Trichinella*.

Post-slaughter methods refer to pork pre-market processing, and include procedures such as freezing and curing to inactivate the parasites; performance of these procedures varies with the species of parasite (Table 3).

The last front is prevention at individual consumer level, which relies on sufficient cooking of meat, i.e. until pork turns light brown. Microwave cooking is not sufficient to inactivate all parasite larvae or cysts. For *Trichinella*, various European and international regulations and guidelines have been developed to protect consumers from exposure [42,88].

In conclusion, reasonably safe pork is not an unachievable goal, but to maintain this major protein source in human nutrition as safe as possible continuous monitoring and control of porkborne parasites at different levels is required in both developed and developing countries.

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## Transparency Declaration

The authors declare no conflict of interest.

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