

Overview of food- and water-borne zoonotic parasites at the farm level

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Summary

Zoonotic parasites found in food animals include a wide variety of protozoa, nematodes, trematodes, and cestodes. Many of these parasites are emerging or already occur globally due to changes in farming practices and the increased movement of animals, food, and people. Some of the emerging or ubiquitous parasites, including *Toxoplasma*, *Cryptosporidium*, *Trichinella*, and *Taenia*, present enormous risks to global food production and consumer health. The parasite life cycle stages, such as eggs, oocysts, and cysts, typically resist adverse temperatures, desiccation, natural irradiation, chemicals, and disinfectants that are commonly used for controlling bacteria and viruses. Other important parasites include trematodes such as *Clonorchis* and *Paragonimus*, which are transmitted via fish or crustaceans and cause serious human disease in specific regions of the world. The potential for global occurrence of these parasites is increasing. Control of zoonotic parasites at the producer level requires education and the development and implementation of effective measures to eliminate the contamination of agricultural water and feed with viable stages of parasites. Standardisation, implementation, and documentation of control measures should increase confidence in global food trade.

Keywords

Control – Cysticercosis – Food safety – Parasites – *Taenia saginata* – *Taenia solium* – *Toxoplasma gondii* – *Trichinella* – Zoonosis.

Introduction

Recent shifts in farming practices, increased transportation of animals, food and people, and global warming have created environments that facilitate the rapid and widespread dissemination of water- and food-borne zoonotic pathogens. Parasites are one group of pathogens that thrive under such environmental conditions, and they continue unabated to exploit the behavioural patterns of their hosts to further transmission among animals and to humans. However, awareness of these pathogens is generally lacking among producers, regulators, and consumers. Zoonotic parasites negatively impact human health and animal production. The globalised food market has raised issues regarding food- and water-borne parasites relevant to producers and consumers around the world. Research, education, and standardised control measures are required to provide consistently safe food for global trade. This paper provides an introduction to food- and

water-borne zoonotic parasites and the unique features of these pathogens that contribute to on-farm survival and contamination of food animals. The transmission dynamics and control of toxoplasmosis, trichinellosis, and cysticercosis are reviewed, as examples of a protozoan, a nematode, and a cestode, respectively.

The parasite group

Parasites belong to a large and diverse group of eukaryotic organisms and range in complexity and size from single cells to multi-segmented organisms. The major groups of zoonotic parasites include protozoa, nematodes (roundworms), trematodes or flukes (non-segmented flatworms), and cestodes or tapeworms (segmented flatworms). In addition to establishing habitats within and deriving nutritional support from animal and/or human hosts, parasites have a variety of transmission stages that

allow them to withstand external conditions and survive in animal excreta, carrion, soil, water, feed, and invertebrate intermediate (for development) or transport hosts. Adaptive features of these stages enable a wide variety of transmission modes and facilitate contamination of food animals. The detection, identification, and destruction of these transmission stages in the environment present major obstacles to the development and execution of control programmes.

Parasite features adapted for transmission and survival

Micro-environments on animal farms are usually ideal for the long-term survival of parasite stages, such as eggs and oocysts, that are excreted by infected hosts. The protective structure of the parasite in the exogenous stages (i.e. stages outside of the host) allows many parasites to resist temperature extremes, desiccation, and irradiation, as well as chemical elements and disinfectants that are commonly used for controlling bacteria and viruses (17).

In many of the exogenous stages parasites are able to withstand freezing temperatures and are capable of overwintering, and in some of these stages parasites are even capable of surviving freeze-thaw cycles. Transmission forms of protozoa remain viable under mild weather conditions for periods of 24 days (*Giardia* cysts) to more than six months (*Cryptosporidium* oocysts). In tropical regions, thermal death points of parasites typically occur above 40°C depending upon the period of exposure, the parasite species, and the transmission stage. Tapeworm eggs, for example, survive temperatures between – 50°C and 70°C but are destroyed when exposed to – 70°C or 100°C for brief periods of time (10). In warm weather, particularly in hot arid regions, many transmission stages of parasites are susceptible to desiccation and natural sources of irradiation. Direct sunlight can kill oocysts in 4 h to 8 h and ozone and ultraviolet-irradiation can be used to control various parasite stages, such as cysts, spores, oocysts, and eggs contaminating feed and water on farms (11, 36).

The transmission stages of most parasites are disseminated within the host's faeces. Spores and cysts are infective immediately on being excreted, while most eggs and oocysts require a period of time under suitable moisture and temperature conditions to develop and become infective.

The regular removal and proper disposal of faeces from animal pens are recommended for the control of parasites, particularly in intensive farming practices. Steps should also be taken to ensure that indirect faecal route

transmission, such as via contaminated water and feed, does not occur. These aspects of food animal husbandry are particularly important since the types and concentrations of disinfectants commonly used for control of other microorganisms are rarely effective against parasites. Eggs of nematodes remain viable in 10% formalin and copper sulphate, and spores survive for brief periods of time in 70% ethanol, 0.1 N HCl, or 0.1 N NaOH (39). However, parasites in manure are eventually destroyed by heat, desiccation, or irradiation.

Free-ranging animals on farms are also susceptible to incursions of parasitic infections transmitted from wildlife. Such parasites are often zoonotic and have a broad host range, such as *Toxoplasma gondii* and *Cryptosporidium parvum*. In addition to serving as reservoir hosts, some wildlife species act as transport hosts. For example, migrating waterfowl have been shown to facilitate the distribution of zoonotic eggs, oocysts, and spores. Terrestrial insects and annelids, molluscs, shellfish, and other aquatic invertebrates have been implicated or suggested as important transport hosts for the bio-magnification and dispersal of zoonotic parasites among intermediate and final hosts (11, 18).

Parasitic infections of food animals at the farm level rely on a variety of host feeding behaviours, including garbage feeding, scavenging, and cannibalism. Specific examples of these are described below in the review of toxoplasmosis, cysticercosis, and trichinellosis.

Toxoplasmosis

Among food-borne pathogens, *Cryptosporidium*, *Cyclospora*, *Giardia*, and *Toxoplasma* are of great concern in global food production (5). These parasites are spread by water and contamination of ready-to-eat foods with oocysts or cysts, while *T. gondii* has additional transmission modes and vehicles involving food of animal origin (6).

Life cycle

Toxoplasma gondii is an intracellular coccidian parasite belonging to the phylum Apicomplexa (6, 7). A wide variety of terrestrial and aquatic vertebrate intermediate hosts become infected by ingesting oocysts excreted in the faeces of definitive hosts (felids) or by eating infected intermediate and definitive hosts containing tachyzoites and tissue cysts with bradyzoites (Fig. 1). Following ingestion of any of these stages, numerous tachyzoites are formed by repeated asexual multiplication (binary fission) within the host cells of most body tissues and fluids. Several days later, tachyzoites differentiate into tissue cysts, each of which mature and contain numerous bradyzoites.

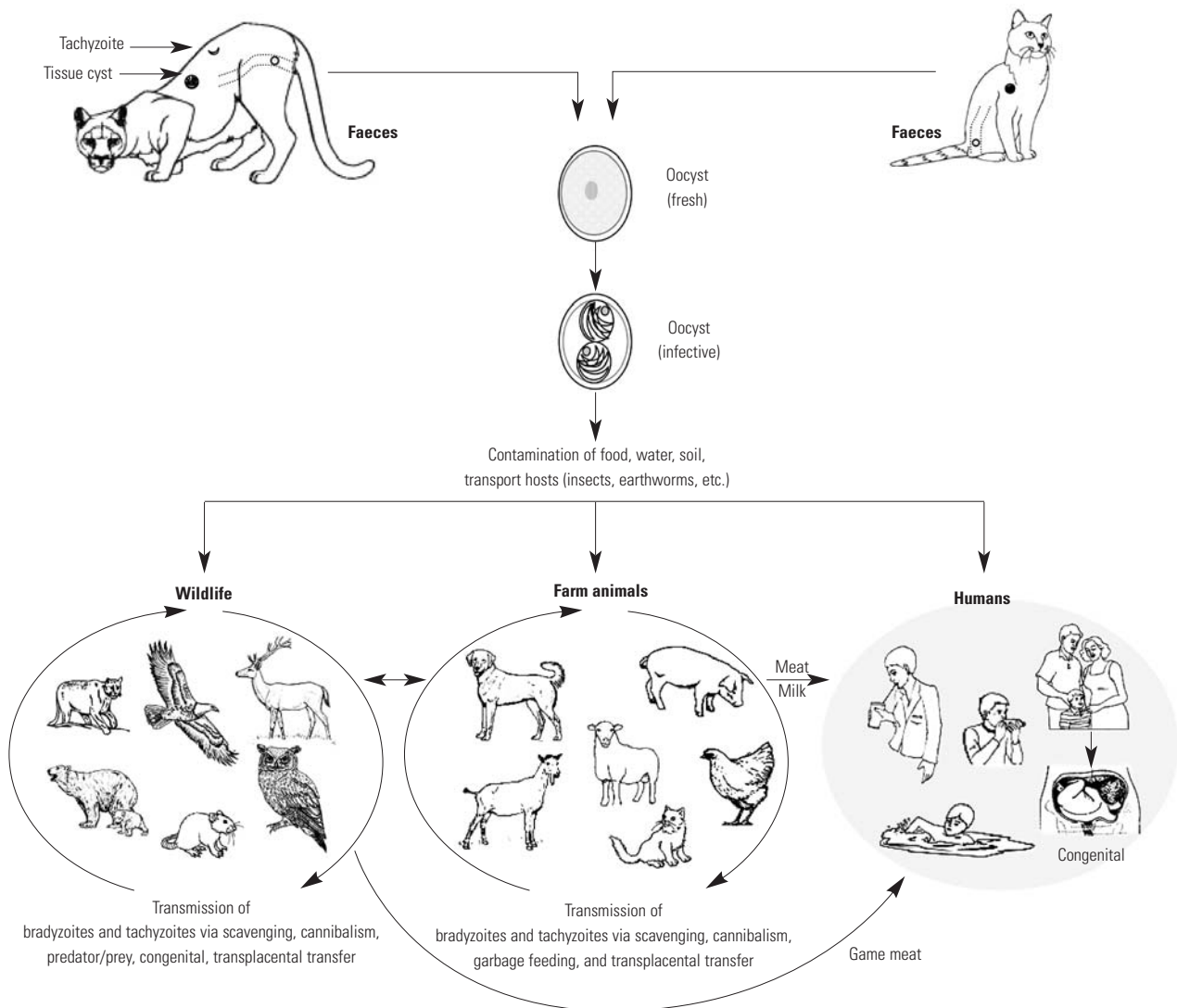


Fig. 1
Life cycle of *Toxoplasma gondii* showing transmission involving oocysts, tachyzoites, and bradyzoites (within tissue cysts) among feline definitive hosts, farm animals, wildlife, and humans

Three to ten days after ingesting tissue cysts (bradyzoites), > 13 days after ingesting tachyzoites, and > 18 days after ingesting oocysts (7) wild or domestic cats are capable of excreting millions of *T. gondii* oocysts daily. The oocysts are formed in the intestinal epithelial cells of cats following several generations of asexual multiplication (merogony) and a final phase of sexual replication (gametogony). Freshly passed oocysts require about one to five days of warmth and humidity to mature (sporulate) and become infective. Sporulated oocysts measure 11 µm by 13 µm and contain two sporocysts, each enclosing four sporozoites. Both mammals and birds serve as intermediate hosts and become infected by ingesting food contaminated with oocysts, bradyzoites in tissue cysts, or tachyzoites in animal organs and secretions, such as milk. Transplacental infection is an important mode of transmission in livestock and humans.

Disease and occurrence

Toxoplasmosis is an economically and medically important disease and is one of the most widespread of all zoonoses (1). Infection with *T. gondii* is common in both animals and humans and often remains latent, but infective, until the death of the host. Serious clinical disease is uncommon. When infection occurs in immunocompetent individuals it is self-limiting, and symptoms may include flu-like illness, swollen lymph nodes, fatigue, and joint and muscle pain. However, infection in immunosuppressed individuals is life threatening. Infection of naïve pregnant women or animals shortly before or after conception often results in congenital toxoplasmosis with consequences ranging from gross foetal abnormalities and spontaneous abortion to neonates being asymptomatic at birth but manifesting problems such as mental retardation and ocular disease much later in life.

Toxoplasma gondii is considered the most common cause of retinochoroiditis worldwide, and it has been shown that 24% of people with ocular disease due to infection with *Toxoplasma* eventually become legally blind (2).

The prevalence of *T. gondii* infection among humans and animals varies greatly in different regions of the world. The parasite even occurs in harsh environments, such as the Arctic, but the prevalence of infection is lower than in regions with a cold but humid climate (42). Seroprevalence of human toxoplasmosis varies between 16% and 40% in North America and the United Kingdom and 50% to 80% in continental Europe and Latin America (1). Over the last three decades the prevalence of *T. gondii* infection in humans in many countries has declined markedly. However, the decline in infection and the corresponding rise in naïve populations have led to concerns about increased risks of human congenital toxoplasmosis (4). The prevalence of *T. gondii* infection differs between wildlife and farm animals. Among wild carnivores, the prevalence of infection is driven primarily by predator-prey behaviour and scavenging; whereas, husbandry practices influence the rate of infection among farmed animals. Pigs, sheep, and goats are more commonly infected than other livestock and represent a significant source of *T. gondii* for humans and other animals. Evidence of *T. gondii* infection in cattle and buffalo has been found only rarely (42). Cats and wild small mammals on farms have a high prevalence of infection and probably contribute to *T. gondii* infections in food animals raised outdoors. The increasing use of intensive indoor management practices has lowered the prevalence of *T. gondii* infection in pigs from 75% to < 10% in many countries and to < 1% in a few European countries (42).

Source and transmission

Transmission of *T. gondii* to animals occurs via ingestion of feed or water contaminated with sporulated oocysts and the consumption of meat, viscera, blood, milk, and other animal products containing tachyzoites and/or bradyzoites in tissue cysts. Wild and domestic cats play a central role in the epidemiology of *T. gondii* infections. Following primary infection, felines contaminate the environment with millions of oocysts, each of which is capable of infecting a new host. Infections in wildlife and farm animals can be established via cannibalism, scavenging, and garbage feeding. Although bradyzoites within tissue cysts are less resistant to environmental conditions than oocysts, they remain viable over a wide range of temperatures, including mild freezing conditions. Bradyzoites survive in carcasses or minced meat at 1°C to 4°C for three weeks but are usually killed at temperatures of –12°C or lower (8). Heat also kills bradyzoites when the tissue cysts are subjected to temperatures of 67°C or higher. Transplacental infection occurs in animals, and although *Toxoplasma*-induced abortions in sheep are

common, the proportion of *T. gondii* infections in food animals due to vertical transmission is not known. Likewise, the role of rodents and other small mammals in maintaining *T. gondii* on farms is unclear. Nevertheless, wildlife remains a significant source of infection for food animals and humans (Fig. 1).

Diagnosis and control

In recognition of the public health importance of *T. gondii*, public health organisations, including the World Health Organization, encourage the collection of epidemiological data for use in developing comprehensive programmes to control *T. gondii* infections in food animals and humans. However, few countries have implemented active surveillance programmes for toxoplasmosis in humans, and food animals are rarely monitored (42). Differences in farming practices, animal populations and interactions, ecosystems and climates, as well as the epidemiological versatility and complexity of *T. gondii*, make it difficult to develop an effective global control strategy. For example, *T. gondii* infections in some livestock species can be prevented by the elimination of cats from the environment; although this is possible in controlled biosecure operations, it is not feasible in most parts of the world. Furthermore, the increasing practice of outdoor housing of livestock in Europe and North America is an added challenge for the control of *T. gondii* on farms. Outdoor housing has been shown to be associated with an increase in the rate of *T. gondii* infections in animals (26). Organic farming practices present additional difficulties in the control of *T. gondii* by limiting the use of several control methods. The availability of resources and technology in many developing countries also limits the relevance of a single global control strategy.

Numerous methods have been developed for the detection of *T. gondii* in the environment and animals, but the reliability of many of these techniques is unclear. Diagnostic approaches include serology, parasite isolation and identification by traditional parasitological methods, bioassays, polymerase chain reaction (PCR) and other molecular assays (7). Selection of the appropriate direct or indirect method of detection is important in specific situations, such as surveillance, disease outbreak investigations, and routine diagnostics. The use of properly validated assays in laboratories operating as part of a recognised quality assurance system for testing parasites is essential for producing reliable data for use in control systems (16).

Trichinellosis

Trichinellosis is a disease that affects animals and humans and is caused by small intramuscular larval nematodes of

the genus *Trichinella*. Eleven genotypes (T1-T11) are currently recognised, eight of which have species status (Table I). Infective larvae are transferred from host to host by the consumption of raw or undercooked meat. Historically, human trichinellosis has been associated with the consumption of meat from *Trichinella*-infected swine, and regulations to detect and control trichinellosis in pigs have been in place in many countries for over 100 years. Consequently, human trichinellosis associated with pork slaughtered in abattoirs operating under modern inspection systems is rare. However, *Trichinella* infection in livestock and wildlife remains common in different regions of the world and the potential for transmission to humans makes this disease a significant human zoonosis (9, 15).

Life cycle

Trichinella have a simple direct life cycle with all stages occurring within one host. Following ingestion of infected raw or undercooked meat, larvae are released by digestion of the meat in the stomach and mature in the small intestine within a few days. Adult female worms survive and shed larvae for about two to three weeks. The newborn larvae enter the blood circulatory system and invade skeletal muscle where most of the genotypes become encapsulated (except T4, T10 and T11) and survive for three to five years or, in some cases, for longer periods of time until the infected meat is consumed by a new host. The tongue, diaphragm, and masseter muscles of the host often harbour higher numbers of larvae than other muscle sites, but these predilection locations may vary according to the host species (25). Clinical disease is rarely observed in naturally infected animals. The severity of human trichinellosis is dependent upon the number of

infective larvae ingested, the genotype of *Trichinella*, and the immune status of the host. Small numbers of larvae usually result in asymptomatic infections. Clinical signs are specific to the stage of infection and may include diarrhoea and/or gastrointestinal upset, periorbital and facial oedema, myalgia, fever, conjunctivitis, photophobia, headache, and skin rash. Myocarditis, endocarditis, encephalitis or meningitis, if these symptoms occur, are serious and may be life-threatening (28).

Occurrence

Members of the genus *Trichinella* have a worldwide distribution, except for the Antarctic (Table I). Domestic animals are infected by deliberate or unintentional feeding of raw tissues containing the parasite or by scavenging infected carcasses of domestic or wild animals. In poorly managed farm operations, domestic cycles involving pigs and rats can readily be established. Human disease associated with the ingestion of horsemeat, dog meat, and farmed wild boar meat is frequently reported, particularly in Europe and Asia (34). In addition, trichinellosis is common in many carnivorous and omnivorous mammalian wildlife populations worldwide, and animals such as bears, foxes, and walrus are frequently the source of disease in humans (13). It has been estimated that as many as 11 million people are infected worldwide, and it is recognised that significant underreporting occurs for reasons relating to test methodology and health care infrastructure (9). The recently recognised non-encapsulated species of *Trichinella* that infects pigs and humans could be missed by traditional tissue compression techniques that rely on capsule formation for detection.

Table I
Genotype, biological characteristics and geographical distribution of *Trichinella* species

| Genotype and species | Host | Capsule | Freeze tolerance | Geographical distribution |
|--|-----------------|---------|------------------|---------------------------|
| T1 – <i>Trichinella spiralis</i> | Mammal | Yes | None | Worldwide |
| T2 – <i>Trichinella nativa</i> | Mammal | Yes | High | Worldwide |
| T3 – <i>Trichinella britovi</i> | Mammal | Yes | Moderate | Europe, Asia, west Africa |
| T4 – <i>Trichinella pseudospiralis</i> | Mammal, bird | No | None | Worldwide |
| T5 – <i>Trichinella murrelli</i> | Mammal | Yes | Moderate | North America |
| T6 ^(a) | Mammal | Yes | High | North America |
| T7 – <i>Trichinella nelsoni</i> | Mammal | Yes | None | East Africa |
| T8 ^(b) | Mammal | Yes | Unknown | South Africa, Namibia |
| T9 ^(b) | Mammal | Yes | Unknown | Japan |
| T10 – <i>Trichinella papuae</i> | Mammal, reptile | No | Unknown | New Guinea |
| T11 – <i>Trichinella zimbabwensis</i> | Mammal, reptile | No | Unknown | Zimbabwe |

a) T6 is closely related to *Trichinella nativa*

b) T8 and T9 are closely related to *Trichinella britovi*

Diagnosis and control

Methods for the detection of trichinellosis include artificial digestion of muscle tissue to release larvae, compression techniques to visualise cysts in muscle (trichinoscopy), serology, PCR, histology, and bioassay. The digestion assay allows the intensity of infection to be quantified and is the most sensitive technique for testing individual animal carcasses (19). Single animals may also be tested using muscle biopsies examined by either digestion assay or trichinoscopy, but quantification of parasite load is not reliable due to the small sample size. The identity of recovered larvae can be confirmed by molecular genotyping (PCR). Although generally reliable for herd testing, serological tests may not detect all infected animals, particularly animals in the early stages of infection, and are therefore not recommended for use in testing individual carcasses for food safety purposes.

Control measures for trichinellosis can be instituted at the producer, abattoir (slaughter and processing), or consumer level. Appropriate identification of animals and products during transport (farm-abattoir, abattoir-marketplace) is necessary for trace-back purposes and is an important component of on-farm certification programmes for producing *Trichinella*-free pigs.

On-farm control at the producer level requires preventing access of domestic animals to any source of raw muscle tissues. Uncooked animal parts may be fed intentionally as a high protein finishing ration prior to slaughter or unintentionally as a cheap food source in the form of household or commercial (restaurant or food processing) garbage. Human trichinellosis has frequently been associated with pork and horsemeat produced using such practices. Poorly managed or primitive farming conditions may create opportunities for cannibalism, predation, and the scavenging of carrion. Lack of rodent control can result in the carcasses of infected small mammals, such as rats, becoming accidentally incorporated into mixed feed. A swine-rat cycle of infection is well known. Prevention of garbage feeding, rodent control, and enhanced management practices are required to break the chain of infection at the producer level (19). Although many countries have legislation regulating some aspects of on-farm control, such as garbage feeding, strict regulations, and enforcement, procedures are required to achieve and maintain an effective integrated system at the producer level. A detailed list of good production practices for use in an on-farm certification programme for pork has been described (35). The programme includes guidelines for animal source, food source, food storage, rodent control, wildlife control, garbage feeding, carcass disposal, general hygiene and sanitation (solid waste, spilled feed, etc.), animal arrival/departure documentation, and detailed and updated record keeping. Additionally, regular audits are conducted and an official non-industry regulatory body to

govern the programme exists. Appropriate quality assured laboratory programmes are recommended for testing and surveillance to monitor and verify control programmes (14).

Abattoir control involving digestion testing of individual carcasses for food safety purposes is recommended in endemic areas (19). Freezing for food safety purposes should be used with caution in areas where freeze-tolerant genotypes exist (Table I). A certified and actively monitored *Trichinella*-free herd or region could reduce or eliminate control procedures at the abattoir level. Regular sero-surveys and digestion testing of a representative proportion of slaughtered animals for surveillance purposes is required in support of the control programmes (45).

Consumer control includes thoroughly heating meat or meat products to 71°C before consumption. In the case of traditional preparations that are consumed raw or undercooked, the source meat should be acquired from an assured safe supplier. Consumer education and a high degree of consumer responsibility are required for meat obtained from wildlife and farm animals that are slaughtered privately.

Cysticercosis

Cysticercosis is a parasitic disease characterised by infection of the muscle with larvae of the cestodes *Taenia solium*, *T. saginata*, and *T. saginata asiatica*. Historically, the adult tapeworms, found in the intestine of humans, were assigned to the genus *Taenia* and the larvae (cysticerci), found in pigs or cattle, to the genus *Cysticercus*. Consequently, *T. solium* cysticerci became known as *Cysticercus cellulosae* and *T. saginata* cysticerci as *C. bovis*. *Taenia saginata asiatica*, described more recently, has the same name for both the larval and adult stages. Both bovine and porcine cysticercoses are recognised worldwide as the cause of human taeniosis (taeniasis). *Taenia solium* also causes human neurological disease (neurocysticercosis). Due to the public health implications of taeniosis and neurocysticercosis, and the negative aesthetics of infested meat, cysticercosis of cattle and swine causes significant economic loss through condemnation of infested meat and offal and trade restrictions for endemic regions.

Life cycle

Taenia tapeworms have an indirect life cycle and are relatively host specific. Humans are the only natural hosts of the adult tapeworms. The tapeworms measure up to several metres in length and consist of an anterior scolex

for attachment to the intestinal mucosa and a chain of progressively maturing hermaphroditic reproductive segments, or proglottids. Thick walled eggs released from mature gravid proglottids are approximately 30 µm to 45 µm in diameter and morphologically indistinguishable among species. The eggs are discharged from infected humans spontaneously or in the faeces. Upon ingestion by a suitable intermediate host, an oncosphere hatches from the egg and eventually develops in the muscle of the host into a cysticercus, which can remain viable for up to several years. The cysticercus consists of a larval tapeworm (with a fluid-filled bladder and invaginated scolex) contained within a connective tissue capsule (cyst) which is oval in shape, and up to 1 cm long. The intermediate hosts for *T. saginata* and *T. saginata asiatica* cysticerci are domestic cattle and swine, respectively. Reindeer have also proven to be suitable intermediate hosts for *T. saginata* (27). In cattle, *T. saginata* cysticerci are found mostly in the tissues of the cardiac and skeletal musculature, whereas *T. saginata asiatica* cysticerci localise on the serosal surface and within the parenchyma of the liver of pigs. The normal intermediate host of *T. solium* is domestic swine, although humans, and occasionally dogs, can serve as intermediate hosts. *Taenia solium* cysticerci localise in the tissues of the tongue, skeletal muscle, subcutis, and central nervous system of pigs. Human consumption of infected pork or beef completes the cycle.

Disease and occurrence

Cysticercosis in cattle does not typically cause detectable disease, but swine with heavy infections can manifest clinically. Human taeniosis is often asymptomatic or manifests as mild non-specific gastrointestinal illness. However, *T. solium* human cysticercosis can result in potentially fatal neurocysticercosis and is the most common cause of acquired epilepsy.

There are no accurate prevalence data on a global scale for any *Taenia* species. Both *T. saginata* and *T. solium* occur worldwide, with the highest occurrence in developing regions where poor sanitation and animal husbandry, as well as some cultural practices, facilitate parasite transmission among hosts. Only sporadic cases of *T. saginata* taeniosis and epizootic outbreaks of bovine cysticercosis occur in North America and other non-endemic areas. *Taenia solium* taeniosis and neurocysticercosis 'imported' from endemic regions are increasingly recognised, and endemic foci resulting from immigration are now established in the United States of America, with approximately 1,000 cases reported annually (44). Neurocysticercosis is an emerging global disease causing an estimated 50,000 deaths annually (37).

Distribution of *T. saginata asiatica* is believed to be limited mostly to South-East Asia and Korea. Prevalence estimates

for this species must be interpreted with caution, however, as the distinction between *T. saginata* and *T. saginata asiatica* has only been recently recognised.

Source and transmission

A person harbouring a single tapeworm can contaminate the environment with up to half a million eggs per day over the course of an infection, which, if left untreated, can persist for years. Eggs can be further disseminated by water, wind, scavenging birds (e.g. gulls feeding on raw sewage), oribatid mites, earthworms, or inanimate objects, such as boots or farm machinery. Infective eggs can persist under a variety of environmental conditions. As with most parasite environmental stages, cool and moist conditions favour long-term survival. *Taenia saginata* eggs can overwinter on pasture and can survive for several months in sewage and sludge, as well as in fresh, brackish, or salt water. The eggs are also resistant to most conventional chemical disinfecting agents (33).

Transmission to livestock occurs via ingestion of food or water contaminated with infective eggs. Scavenged human faeces are a major source of infection in pigs. Parasite transmission from humans to pigs is facilitated by the common practice in many endemic rural regions of allowing pigs to roam free in areas without human latrines. Since cattle do not intentionally consume human faeces, bovine cysticercosis occurs via inadvertent ingestion of contaminated feedstuffs or water, including pasture fertilised with human sewage.

Diagnosis and control

Globalisation poses an increasing threat of incursions of cysticercoses and taenioses via the immigration of people and importation of animals and animal products and potentially contaminated produce or other fomites from endemic regions. Eradication of these infections in humans and livestock is possible, as pigs and cattle are the only significant reservoir of cysticercosis, humans can be easily and inexpensively treated with anthelmintics, and education about the parasite life cycle and mitigating measures (such as proper hygiene and latrine use, preventing access of livestock to human faeces, and thorough cooking or freezing of meat) will reduce overall parasite transmission (12).

Detection of cysticercosis in cattle and pigs usually occurs *post mortem*, although pigs with heavy infections may be presumptively diagnosed *ante mortem* by observation and/or palpation of cysts in the tongue. Many countries regulate *post mortem* screening for cysticercosis, requiring examination of the so-called 'predilection sites'. Affected carcasses are condemned or treated by cooking or freezing

to kill the parasite. Such screening is insensitive, particularly for lightly infected carcasses (38). Dead or dying cysticerci elicit inflammatory lesions that are easier to detect than viable parasites. Since carcasses can harbour both viable (infective) and degenerated cysticerci, detection of degenerated cysts is still significant. However, definitive diagnosis of degenerated lesions using gross examination or histology is often difficult. A recently developed immunohistochemical assay for bovine cysticercus excretory-secretory antigen will help in this regard (32). Molecular methods have been adapted for detecting cysticerci but require further validation (23). Although commercially available enzyme-linked immunosorbent (ELISA) and enzyme-linked immunotransfer blot (EITB) assays have high sensitivity and specificity when used on human serum or cerebrospinal fluid samples, reliability has been low for samples collected from naturally infected animals (41, 43). Such assays have value as epidemiological tools for screening herds for cysticercosis but not for assessing individual animals.

Reliable methods for recovering *Taenia* eggs from various environmental matrices are not available. In most sporadic outbreaks of bovine cysticercosis in low prevalence regions, such as North America, a definitive source of the infection is not identified. Even if a particular feed or water source is suspected, processing of relatively large volumes of test samples with low level contamination is problematic. Modified flotation methods have been attempted, but the high specific gravity of *Taenia* eggs and confounding debris in the assayed matrix decreases the sensitivity of detection (40). Eggs of the taeniid family cannot be speciated based on morphology, and there are no baseline data available for levels of environmental contamination with taeniid species from other infected domestic animals or wildlife species. Reliable molecular methods for detecting low numbers of *Taenia* eggs are still being developed (20, 31). Since *Taenia* eggs are resistant to many environmental conditions and most practical and conventional chemical treatments, efforts should be aimed at preventing environmental contamination. If sewage must be used as fertiliser, measures should be taken to reduce the number of viable eggs in the applied sludge (3).

Anthelmintic treatment of livestock is effective but does not quickly or reliably eliminate cysticerci and is not economically feasible for cattle (21). Vaccines hold more promise but are not yet commercially available. Recombinant subunit vaccines based on oncosphere antigens have proven highly effective in protecting cattle and pigs from experimental challenge with *T. saginata* and

T. solium eggs, respectively (29, 22). A synthetic peptide vaccine against *T. solium* cysticercosis has been shown to significantly reduce the prevalence and intensity of natural infections in pigs (24).

Other parasites

Other zoonotic parasites also infect domestic or wild animals, particularly animals in aquatic environments. Among the nematodes, *Anisakis simplex*, *Pseudoterranova decipiens*, and *Gnathostoma* species are fish-borne, while *Angiostrongylus cantonensis* is transmitted by fresh water molluscs, crab, and shrimp (1). Although humans are not normal hosts for these parasites, infection can result in serious disease. Many species of fresh water fish in Asia and elsewhere serve as intermediate hosts for several trematodes that use humans and other mammals as primary definitive hosts (30). These parasites, which include *Clonorchis sinensis* (Chinese liver fluke), *Heterophyes heterophyes*, *Metagonimus yokogawi*, and *Opistorchis* spp., develop in the intestine or bile duct of humans. *Paragonimus westermani*, another trematode that infects humans, causes pulmonary disease following the ingestion of freshwater crab and crayfish containing the infective stage (metacercaria). Since specific aquatic invertebrates on fish farms serve as intermediate hosts, control of these hosts may be a useful strategy for reducing trematode zoonoses.

Many zoonotic parasites use water as a vehicle for transmission to their definitive or intermediate hosts, either directly or via aquatic plants, invertebrates, and/or fish. For example, large numbers of animals become infected with *T. gondii* and *Taenia* spp. when they consume common-source water infected with oocysts and eggs, respectively. Implementation of on-farm control measures to eliminate the contamination of agricultural water with human faeces is a key factor in the control of many zoonotic parasitic infections.

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Un aperçu des parasitoses zoonotiques d'origine alimentaire ou hydrique présentes dans les élevages

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Résumé

Parmi les parasites présents dans les denrées alimentaires d'origine animale, on retrouve une grande variété de protozoaires, de nématodes, de trématodes et de cestodes. L'émergence ou la distribution mondiale de la plupart de ces parasites sont liées à la transformation des pratiques d'élevage et à la mobilité croissante des animaux, des personnes et des produits alimentaires. Certains de ces parasites émergents ou ubiquistes, notamment *Toxoplasma*, *Cryptosporidium*, *Trichinella* et *Taenia* représentent une menace considérable pour la production alimentaire et la santé des consommateurs au niveau mondial. À chacun des stades du cycle parasitaire (œufs, oocystes, kystes), la plupart des parasites résistent aux traitements thermiques, par dessiccation, par radiation, ainsi qu'aux produits chimiques et aux désinfectants habituellement utilisés pour inactiver les bactéries et les virus. Autres parasites importants, les trématodes du genre *Clonorchis* et *Paragonimus* sont présents dans les poissons et les fruits de mer et occasionnent chez l'homme des maladies graves bien que circonscrites à certaines régions du globe. Le risque d'extension de l'aire de répartition de ces parasites au niveau mondial va en augmentant. La maîtrise des parasitoses zoonotiques au niveau des élevages passe par une meilleure formation des éleveurs et par la conception et la mise en place de mesures efficaces visant à empêcher que l'eau et les aliments distribués aux animaux dans les exploitations ne soient contaminés par des parasites parvenus à un stade viable. La normalisation, la mise en œuvre et la diffusion d'informations sur les mesures de prophylaxie devraient améliorer la confiance dans le commerce mondial de denrées alimentaires.

Mots-clés

Cysticercose – Parasites – Prophylaxie – Sécurité sanitaire des aliments – *Taenia saginata* – *Taenia solium* – *Toxoplasma gondii* – *Trichinella* – Zoonose.



Parásitos zoonóticos transmitidos por los alimentos y el agua en las granjas

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Resumen

Los parásitos zoonóticos que se encuentran en animales para consumo incluyen una amplia variedad de protozoarios, nematodos, trematodos y céstodos. Muchos de estos son parásitos emergentes, o que aparecen en todas partes del mundo debido al incremento de los movimientos de animales, alimentos y personas, así como a la evolución de las prácticas pecuarias. Algunos de los parásitos emergentes o ubicuos, incluidos *Toxoplasma*, *Cryptosporidium*, *Trichinella* y *Taenia*, representan enormes amenazas para la producción de

alimentos y la salud de los consumidores mundiales. Habitualmente, las distintas etapas del ciclo biológico de los parásitos – huevo, ooquiste y quiste – resisten a las temperaturas elevadas, la desecación, la radiación natural, los productos químicos y los desinfectantes que suelen utilizarse para controlar bacterias y virus. Otros importantes parásitos incluyen los trematodos, como *Clonorchis* y *Paragonimus*, transmitidos por los peces y crustáceos y que provocan enfermedades graves a los seres humanos de determinadas regiones. La posibilidad de que se extiendan por todo el mundo es cada vez mayor. Para controlar los parásitos zoonóticos en el nivel de la producción es preciso formar a sus responsables y, también, formular y aplicar medidas eficaces para eliminar sus etapas viables del agua y los alimentos destinados a los animales. La estandarización, implementación y documentación de medidas de control, debería aumentar la confianza en el comercio mundial de alimentos.

Palabras clave

Cisticercosis – Control – Inocuidad de los alimentos – Parásito – *Taenia saginata* – *Taenia solium* – *Toxoplasma gondii* – *Trichinella* – Zoonosis.

References

1. Acha P.N. & Szyfres B. (2003). – Zoonoses and communicable diseases common to man and animals, 3rd Ed., Vol. 3, Parasitoses. Pan American Health Organization, Washington, DC.
2. Bosch-Driessen L.E., Berendschot T.T., Ongkosuwito J.V. & Rothova A. (2002). – Ocular toxoplasmosis: clinical features and prognosis of 154 patients. *Ophthalmology*, **109**, 869-878.
3. Cabaret J., Geerts S., Madeline M., Ballandonne C. & Barbier D. (2002). – The use of urban sewage sludge on pastures: the cysticercosis threat. *Vet. Res.*, **33** (5), 575-597.
4. Cook A.J., Gilbert R.E., Buffolano W., Zufferey J., Petersen E., Jenun P.A., Foulon W., Semprini A.E. & Dunn D.T. (2000). – Sources of *Toxoplasma* infection in pregnant women: European multicentre case-control study. European Research Network on Congenital Toxoplasmosis. *BJM*, **321**, 142-147.
5. Dawson D. (2005). – Foodborne protozoan parasites. *Int. J. Food Microbiol.*, **103**, 207-227.
6. Dubey J.P. (1993). – *Toxoplasma*, *Neospora*, *Sarcocystis*, and other tissue cyst-forming coccidia of humans and animals. In Parasitic protozoa (J.P. Kreier, ed.), 2nd Ed., Vol. 6. Academic Press, London, 1-158.
7. Dubey J.P. (2004). – Toxoplasmosis – a waterborne zoonosis. In Waterborne zoonotic parasites (A.A. Gajadhar, ed.). *Vet. Parasitol.*, **126** (special issue), 57-71.
8. Dubey J.P., Kotula A.W., Sharar A., Andrews C.D. & Lindsay D.S. (1990). – Effect of high temperature on infectivity of *Toxoplasma gondii* tissue cysts in pork. *J. Parasitol.*, **76**, 201-204.
9. Dupouy-Camet J. (2000). – Trichinellosis: a worldwide zoonosis. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 191-200.
10. Eckert J., Gottstein B., Heath D. & Liu F.J. (2001). – Prevention of echinococcosis in humans and safety precautions. In WHO-OIE Manual on echinococcosis in humans and animals: a public health problem of global concern. OIE, Paris, 238-247.
11. Fayer R. (2004). – *Cryptosporidium*: a waterborne zoonotic parasite. In Waterborne zoonotic parasites (A.A. Gajadhar, ed.). *Vet. Parasitol.*, **126** (special issue), 37-56.
12. Flisser A., Sarti E., Lightowlers M. & Schantz P. (2003). – Neurocysticercosis: regional status, epidemiology, impact and control measures in the Americas. *Acta trop.*, **87** (1), 43-51.
13. Forbes L.B. (2000). – The occurrence and ecology of *Trichinella* in marine mammals. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 321-334.

14. Forbes L.B., Scandrett W.B. & Gajadhar A.A. (2005). – A program to accredit laboratories for reliable testing of pork and horsemeat for *Trichinella*. *Vet. Parasitol.*, **132**, 173-177.
15. Gajadhar A.A. & Gamble H.R. (2000). – Historical perspectives and current global challenges of *Trichinella* and trichinellosis. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 181-189.
16. Gajadhar A.A. & Forbes L.B. (2002). – An internationally recognized quality assurance system for diagnostic parasitology in animal health and food safety, with example data on trichinellosis. *Vet. Parasitol.*, **103**, 133-140.
17. Gajadhar A.A. & Allen J.R. (2004). – Factors contributing to the public health and economic importance of waterborne zoonotic parasites. In *Waterborne zoonotic parasites* (A.A. Gajadhar, ed.). *Vet. Parasitol.*, **126** (special issue), 3-14.
18. Gajadhar A.A., Measures L., Forbes L., Kapel C. & Dubey J.P. (2004). – Experimental *Toxoplasma gondii* infection in grey seals (*Halichoerus grypus*). *J. Parasitol.*, **90**, 255-259.
19. Gamble H.R., Bessonov A.S., Cuperlovic K., Gajadhar A.A., van Knapen F., Noeckler K., Schenone H. & Zhu X. (2000). – International Commission on trichinellosis: recommendations on methods for the control of *Trichinella* in domestic and wild animals intended for human consumption. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 393-408.
20. Gonzalez L.M., Montero E., Harrison L.J.S., Parkhouse R.M.E. & Garate T. (2000). – Differential diagnosis of *Taenia saginata* and *Taenia solium* infection by PCR. *Clin. Microbiol.*, **38** (2), 737-744.
21. Gonzalez A.E., Gavidia C., Falcon N., Bernal T., Verastegui M., Garcia H.H., Gilman R.H. & Tsang V.C.W. (2001). – Protection of pigs with cysticercosis from further infections after treatment with oxfendazole. *Am. J. trop. Med. Hyg.*, **65** (1), 15-18.
22. Gonzalez A.E., Gauci C.G., Barber D., Gilman R.H., Tsang V.C.W., Garcia H.H., Verastegui M. & Lightowlers M.W. (2005). – Vaccination of pigs to control human neurocysticercosis. *Am. J. trop. Med. Hyg.*, **72** (6), 837-839.
23. Harrison L.J.S., Garate T., Bryce D.M., Gonzalez L.M., Foster-Cuevas M., Wamae L.W., Onyango-Abuje J.A. & Parkhouse R.M.E. (2005). – Ag-ELISA and PCR for monitoring the vaccination of cattle against *Taenia saginata* cysticercosis using an oncospherical adhesion protein (HP6) with surface and secreted localization. *Trop. anim. Hlth Prod.*, **37** (2), 103-120.
24. Huerta M., De Aluga A.S., Fragoso G., Toledo A., Villalobos N., Hernandez M., Gevorkian G., Acero G., Diaz A., Alvarez I., Avila R., Beltran C., Garcia G., Martinez J.J., Larralde C. & Sciuotto E. (2002). – Synthetic peptide vaccine against *Taenia solium* pig cysticercosis: successful vaccination in a controlled field trial in rural Mexico. *Vaccine*, **20**, 262-266.
25. Kapel C.M.O. (2000). – Host diversity and biological characteristics of the *Trichinella* genotypes and their effect on transmission. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 263-278.
26. Kijlstra A., Eissen O., Cornelissen J., Munniksma K., Eijck I. & Kortbeek T. (2004). – *Toxoplasma gondii* infection in animal-friendly pig production systems. *Investigative Ophthalmol. visual Sci.*, **45** (9), 3165-3169.
27. Kirichek V.S. (1985). – Peculiarities of the biology of *Taenia saginata* and of the disease that it causes. *Veterinariya (Kiev) or (Moscow)*, **2**, 50-52.
28. Kociecka W. (2000). – Trichinellosis: human disease, diagnosis and treatment. In *Trichinella* and trichinellosis (A.A. Gajadhar & H.R. Gamble, eds). *Vet. Parasitol.*, **93** (special issue), 365-383.
29. Lightowlers M.W., Rolfe R. & Gauci C.G. (1996). – *Taenia saginata*: vaccination against cysticercosis in cattle with recombinant oncosphere antigens. *Experim. Parasitol.*, **84**, 330-338.
30. Nithiuthai S., Anantaphruti M.T., Waikagul J. & Gajadhar A. (2004). – Waterborne zoonotic helminthiasis. In *Waterborne zoonotic parasites* (A.A. Gajadhar, ed.). *Vet. Parasitol.*, **126** (special issue), 167-193.
31. Nunes C.M., Dias A.K.K., Dias F.E.F., Aoki S.M., De Paula H.B., Lima L.G.F. & Garcia J.F. (2005). – *Taenia saginata*: differential diagnosis of human taeniasis by polymerase chain reaction-restriction fragment length polymorphism assay. *Experim. Parasitol.*, **110** (4), 412-415.
32. Ogunremi O., Macdonald G., Geerts S. & Brandt J. (2004). – Diagnosis of *Taenia saginata* cysticercosis by immunohistochemical test on formalin-fixed and paraffin-embedded bovine lesions. *J. vet. diagn. Invest.*, **16** (5), 438-441.
33. Pawlowski Z.S. (1994). – *Taeniasis* and *cysticercosis*. In *Foodborne disease handbook* (Y.H. Hiu, J.R. Gorham, K.D. Murrel & D.O. Cliver, eds), Vol. 2. Marcel Dekker, Inc., New York, 199-254.
34. Pozio E. (2005). – The broad spectrum of *Trichinella* hosts: from cold to warm-blooded animals. *Vet. Parasitol.*, **132**, 3-11.
35. Pyburn D.G., Gamble H.R., Wagstrom E.A., Anderson L.A. & Miller L.E. (2005). – Trichinae certification in the United States pork industry. *Vet. Parasitol.*, **132**, 179-183.
36. Quintero-Betancourt W. & Rose J.B. (2004). – Drinking water processes for removal of *Cryptosporidium* and *Giardia*. In *Waterborne zoonotic parasites* (A.A. Gajadhar, ed.). *Vet. Parasitol.*, **126** (special issue), 219-234.

37. Roman G., Sotelo J., Brutto O.D., Flisser A., Dumas M., Wadia N., Botero D., Cruz M., Garcia H., De Bittencourt P.R.M., Trelles L., Arriagada C., Lorenzana P., Nash T.E. & Spina-Franca A. (2000). – A proposal to declare neurocysticercosis an international reportable disease. *Bull. WHO*, **78** (3), 399-406.
38. Saini P.K., Webert D.W. & McCaskey P.C. (1997). – Food safety and regulatory aspects of cattle and swine cysticercosis. *J. Food Protec.*, **60** (4), 447-453.
39. Santillana-Hayat M., Sarfati C., Fournier S., Chau F., Porcher R., Molina J.-M. & Derouin F. (2002). – Effects of chemical and physical agents on viability and infectivity of *Encephalitozoon intestinalis* determined by cell culture and flow cytometry. *Antimicrob. Agents Chemother.*, **46** (6), 2049-2051.
40. Scandrett W.B. & Gajadhar A.A. (2004). – Recovery of putative taeniid eggs from silt in water associated with an outbreak of bovine cysticercosis. *Can. Vet. J.*, **45**, 758-760.
41. Sciuotto E., Martinez J.J., Villalobos N.M., Hernandez M., Jose M.V., Beltran C., Rodarte F., Flores I., Bobadilla J.R., Fragoso G., Parkhouse M.E., Harrison L.J.S. & De Aluja A.S. (1998). – Limitations of current diagnostic procedures for the diagnosis of *Taenia solium* cysticercosis in rural pigs. *Vet. Parasitol.*, **79**, 299-313.
42. Tenter A., Heckeroth A. & Weiss L. (2000). – *Toxoplasma gondii*: from animals to humans. *Int. J. Parasitol.*, **30**, 1217-1258.
43. Van Kerckhoven I., Vansteenkiste W., Claes M., Geerts S. & Brandt J. (1998). – Improved detection of circulating antigen in cattle infected with *Taenia saginata* metacestodes. *Vet. Parasitol.*, **76**, 269-274.
44. White A.C. (2000). – Neurocysticercosis: updates on epidemiology, pathogenesis, diagnosis, and management. *Annu. Rev. Med.*, **51**, 187-206.
45. World Organisation for Animal Health (OIE) (2004) – Terrestrial Animal Health Code, 14 Ed. OIE, Paris, 110-111.
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