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***Toxoplasma gondii*: from animals to humans**

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Abstract

Toxoplasmosis is one of the more common parasitic zoonoses world-wide. Its causative agent, *Toxoplasma gondii*, is a facultatively heteroxenous, polyxenous protozoon that has developed several potential routes of transmission within and between different host species. If first contracted during pregnancy, *T. gondii* may be transmitted vertically by tachyzoites that are passed to the foetus via the placenta. Horizontal transmission of *T. gondii* may involve three life-cycle stages, i.e. ingesting infectious oocysts from the environment or ingesting tissue cysts or tachyzoites which are contained in meat or primary offal (viscera) of many different animals. Transmission may also occur via tachyzoites contained in blood products, tissue transplants, or unpasteurised milk. However, it is not known which of these routes is more important epidemiologically. In the past, the consumption of raw or undercooked meat, in particular of pigs and sheep, has been regarded as a major route of transmission to humans. However, recent studies showed that the prevalence of *T. gondii* in meat-producing animals decreased considerably over the past 20 years in areas with intensive farm management. For example, in several countries of the European Union prevalences of *T. gondii* in fattening pigs are now <1%. Considering these data it is unlikely that pork is still a major source of infection for humans in these countries. However, it is likely that the major routes of transmission are different in human populations with differences in culture and eating habits. In the Americas, recent outbreaks of acute toxoplasmosis in humans have been associated with oocyst contamination of the environment. Therefore, future epidemiological studies on *T. gondii* infections should consider the role of oocysts as potential sources of infection for humans, and methods to monitor these are currently being developed. This review presents recent epidemiological data on *T. gondii*, hypotheses on the major routes of transmission to humans in different populations, and preventive measures that may reduce the risk of contracting a primary infection during pregnancy.

1. Introduction

The tissue cyst-forming coccidium *Toxoplasma gondii* is one of the more polyxenous parasites known to date. It has a facultatively heteroxenous life cycle and can probably infect all warm-blooded animals (mammals and birds) and humans. *T. gondii* is prevalent in most areas of the world and is of veterinary and medical importance, because it may cause abortion or congenital disease in its intermediate hosts. Because of its great importance as a causative agent of a zoonosis *T. gondii* has been studied most intensively among the coccidia. To date, more than 15 000 original research articles, more than 500 reviews, and several books and book chapters have been published on this parasite (Table 1). However,

there are still many aspects of its biology, natural life cycle, and the epidemiology of *T. gondii* infections of which we know relatively little.

Asexual stages of toxoplasma-like parasites were first observed at the turn of the century in tissues of birds and mammals [1]. The first comprehensive description of *T. gondii* merozoites (i.e. tachyzoites or endozoites) in the spleen, liver, and blood of gondis, a species of North African rodents, was given in 1908 by Nicolle and Manceaux [2]. They introduced the genus *Toxoplasma* [3], and *T. gondii* became the type species of the genus. During the first half of this century, several species of *Toxoplasma* were named mainly in accordance with the host species in which they were detected [1,4,5]. It was not until the late 1930s that biological and immunological comparisons provided evidence that various isolates of animal and human origin were identical with *T. gondii* [6]. However, even then only asexual stages (merozoites and tissue cysts) of *T. gondii* were known and its classification was uncertain [5,7].

Evidence for the coccidian nature of *T. gondii* came first from EM studies carried out in the 1960s. These studies revealed ultrastructural similarities between extraintestinal merozoites of *T. gondii* and intestinal merozoites of *Eimeria* species, and thus indicated a coccidian-like life cycle for *T. gondii* [4,5,8]. The heteroxenous life cycle of *T. gondii* was elucidated in the late 1960s after it had been found that the faeces of cats may contain an infectious stage of *T. gondii* which induces infection when ingested by intermediate hosts [9]. This stage was eventually identified as an isosporan-type oocyst previously described as part of the *Isospora bigemina* complex [4,5,10]. In 1970, knowledge of the coccidian life cycle of *T. gondii* was completed by the discovery of sexual stages in the small intestine of cats [1,4,5,11–14].

Thus, knowledge on the life cycle of *T. gondii* was completed more than 60 years after the first description of its asexual stages in intermediate hosts. It was finally revealed that *T. gondii* is a tissue cyst-forming coccidium with a heteroxenous life cycle in which an asexual phase of development in various tissues of herbivorous or omnivorous intermediate hosts is linked to a sexual phase of development in the intestine of carnivorous definitive hosts. Since then, several other protozoa that had been assigned to the genus *Toxoplasma* during the first half of this century, have either been synonymised with *T. gondii*, have been reclassified into other coccidian genera, or their descriptions superseded [1,4,5,15]. Over the past 3 decades, *T. gondii* has been generally considered as the only valid species of the genus *Toxoplasma* [5,11,16–22]. More recently, molecular epidemiological studies have provided evidence that there are at least two clonal lineages within *T. gondii*, one comprising strains that are virulent in mice and another comprising strains that are avirulent in mice [23,24]. This finding has raised debate on whether or not the different lineages within *T. gondii* are indicative of ongoing speciation [23–29], and a recent hypothesis suggested that vertical transmission of *T. gondii* in the mouse-virulent lineage may have a greater epidemiological importance than has been believed so far [23,24].

In the course of evolution, *T. gondii* has developed a broad range of potential routes of transmission. However, the elucidation of these routes during the past 3 decades has not elucidated which of these routes is more important epidemiologically. For example, many studies have focussed on congenital toxoplasmosis in humans which is a result of vertical transmission of the parasite during pregnancy. By contrast, we know little about the relative importance of horizontal transmission of *T. gondii* between different host species, of the major reservoirs of the parasite in nature, or of the epidemiological impact of the different sources causing infection or disease in humans. Likewise, many studies have been carried out on the asexual stages of *T. gondii*, in particular on the tachyzoite, while much fewer studies have considered the sexual stages or their infectious product, i.e. the sporozoites

within the oocyst. Moreover, only few studies have been aimed at identifying risk factors that may be associated with acquiring an infection with *T. gondii* postnatally (Table 1).

This review focuses on probable routes of transmission of *T. gondii* from animals to humans. We review recent outbreaks of toxoplasmosis in humans and discuss the sources of infection that have been associated with them. We also review epidemiological data on *T. gondii* that have been recorded over the last decade and discuss strategies for prevention or control of *T. gondii* infections in humans. However, because of the large number of scientific papers that are being published on *T. gondii* every year it is not possible to cover all aspects of this zoonosis in this review. Therefore, we refer to the comprehensive reviews of Dubey and Towle [30], Dubey and Beattie [11], Jackson and Hutchison [12], Remington and Desmonts [31], Ho-Yen and Joss [32], Dubey [13], Remington et al. [33], and Ambroise-Thomas and Petersen [34] for more detailed information and for data recorded on this parasite prior to the 1990s.

2. Life cycle of *Toxoplasma gondii*

T. gondii is a ubiquitous parasite that occurs in most areas of the world. It is capable of infecting an unusually wide range of hosts and many different host cells [7,11,35]. The life cycle of *T. gondii* is facultatively heteroxenous (Fig. 1). Intermediate hosts are probably all warm-blooded animals including most livestock, and humans. Definitive hosts are members of the family Felidae, for example domestic cats [11–13,22,36].

In intermediate hosts, *T. gondii* undergoes two phases of asexual development. In the first phase, tachyzoites (or endozoites) multiply rapidly by repeated endodyogeny in many different types of host cells. Tachyzoites of the last generation initiate the second phase of development which results in the formation of tissue cysts. Within the tissue cyst, bradyzoites (or cystozoites) multiply slowly by endodyogeny [11–13,20,35]. Tissue cysts have a high affinity for neural and muscular tissues. They are located predominantly in the central nervous system (CNS), the eye as well as skeletal and cardiac muscles. However, to a lesser extent they may also be found in visceral organs, such as lungs, liver, and kidneys [13,14,35]. Tissue cysts are the terminal life-cycle stage in the intermediate host and are immediately infectious. In some intermediate host species, they may persist for the life of the host. The mechanism of this persistence is unknown. However, many investigators believe that tissue cysts break down periodically, with bradyzoites transforming to tachyzoites that reinvade host cells and again transform to bradyzoites within new tissue cysts [14,20,22,31,35,37,38]. If ingested by a definitive host, the bradyzoites initiate another asexual phase of proliferation which consists of initial multiplication by endodyogeny followed by repeated endopolygeny in epithelial cells of the small intestine. The terminal stages of this asexual multiplication initiate the sexual phase of the life cycle. Gamogony and oocyst formation also take place in the epithelium of the small intestine. Unsporulated oocysts are released into the intestinal lumen and passed into the environment with the faeces. Sporogony occurs outside the host and leads to the development of infectious oocysts which contain two sporocysts, each containing four sporozoites [11–13,20,35].

There are three infectious stages in the life cycle of *T. gondii*, i.e. tachyzoites, bradyzoites contained in tissue cysts, and sporozoites contained in sporulated oocysts (Fig. 1). All three stages are infectious for both intermediate and definitive hosts which may acquire a *T. gondii* infection mainly via one of the following routes (Fig. 2): (A) horizontally by oral ingestion of infectious oocysts from the environment, (B) horizontally by oral ingestion of tissue cysts contained in raw or undercooked meat or primary offal (viscera) of intermediate hosts, or (C) vertically by transplacental transmission of tachyzoites [11–13,20,31,35,39]. In

addition, in several hosts tachyzoites may also be transmitted in the milk from the mother to the offspring [11–13,20,23,31].

Thus, *T. gondii* may be transmitted from definitive to intermediate hosts, from intermediate to definitive hosts, as well as between definitive and between intermediate hosts (Figs. 1 and 2). It is currently not known which of the various routes of transmission is more important epidemiologically. However, the prevalence of *T. gondii* infections is not confined to the presence of a certain host species. Its life cycle may continue indefinitely by transmission of tissue cysts between intermediate hosts (even in the absence of definitive hosts) and also by transmission of oocysts between definitive hosts (even in the absence of intermediate hosts).

3. Zoonotic importance of *Toxoplasma gondii*

3.1. Prevalence of *T. gondii* infections in humans

Toxoplasmosis is one of the more common parasitic zoonoses world-wide. Disease in humans caused by *T. gondii* was first recognised in the late 1930s (Table 2). In 1939, Sabin [6] first proved that *Toxoplasma* isolates from humans and those previously obtained from animals belonged to the same species. In 1948, the introduction of the methylene blue dye test by Sabin and Feldman [40] enabled seroepidemiological studies in humans as well as a broad range of animal species which provided evidence for a wide distribution and high prevalence of *T. gondii* in many areas of the world. Since then, it has been estimated that up to one third of the world human population has been exposed to the parasite [1,12,14,41]. However, seroprevalence estimates for human populations vary greatly among different countries, among different geographical areas within one country, and among different ethnic groups living in the same area. Thus, over the past 3 decades antibodies to *T. gondii* have been detected in from 0 to 100% of individuals in various adult human populations [11,12,31,42,43].

When comparing seroprevalence data for infections with *T. gondii* it should be taken into account that the different serological methods used to obtain these data are not standardised. The Sabin–Feldman dye test, which is still considered as the ‘gold standard’ for detection of antibodies to *T. gondii* in humans, is labour-intensive and has the disadvantage that it requires a continuous supply of live parasites. Therefore, most epidemiological studies on *T. gondii* infections now use different tests for antibody detection. A broad range of serological tests have been developed to detect antibodies to *T. gondii* in humans and animals [11,31,44]. These tests vary in sensitivity, specificity, and predictive values. As a consequence, no two tests produce the same results in all cases, even when carried out in the same laboratory [45–54]. In addition, prevalence rates vary over time and with the age of the individuals included in the study [55–68].

Therefore, the data reviewed here do not reflect nationwide prevalences and may differ from the true prevalence of infection in the various populations. However, they are comparable if they are interpreted as estimates reflecting the different levels of prevalence among similar populations, i.e. populations that are comparable with respect to age, cultural habits, environmental factors, or other factors that may have an impact on the epidemiology of *T. gondii* infections (see Section 4). For example, in the 1990s seroprevalences in Central European countries, such as Austria, Belgium, France, Germany, and Switzerland, have been estimated to range between 37 and 58% in women of child-bearing age with no obstetric history (Table 3). Comparable seroprevalences have been observed in similar populations in Croatia, Poland, Slovenia, Australia, and Northern Africa. Seroprevalences are higher in several Latin-American countries, including Argentina, Brazil, Cuba, Jamaica, and Venezuela (51–72%), and in West African countries on the Gulf of Guinea, i.e. Benin, Cameroon, Congo, Gabon, and Togo (54–77%). Lower seroprevalences have been reported

for women of childbearing age in Southeast Asia, China, and Korea (4–39%). Seroprevalences are also low in areas with a cold climate, such as the Scandinavian countries (11–28%). However, there is no doubt that overall *T. gondii* infections are highly prevalent in adult human populations throughout the world (Table 3).

3.2. Postnatally acquired toxoplasmosis in immunocompetent humans

While infection with *T. gondii* in humans is very common, clinical disease is largely confined to risk groups (see Sections 3.3 and 3.4). Most cases of *T. gondii* infections in immunocompetent humans are asymptomatic. Occasionally, various mild symptoms may be observed of which lymphadenopathy is the most significant clinical manifestation [11,69,70]. Severe manifestations, such as encephalitis, sepsis syndrome/shock, myocarditis, or hepatitis may occur, but are very rare in immunocompetent humans [70].

Since the early 1950s, infection with *T. gondii* has also been recognised as an important cause of retinochoroiditis [71]. However, ocular toxoplasmosis has long been regarded as a result of a prenatal infection with *T. gondii*, which manifests later in life [70,72,73]. While retinochoroiditis lesions in infants with congenital toxoplasmosis are well recognised (see Section 3.3), it has been controversial whether similar ocular lesions in older children or adults result from a recently acquired, primary infection or from recurrences of prenatal infection [70,72,74–78]. However, there are now several recorded cases in which the development of ocular symptoms, such as retinitis and retinochoroiditis, was convincingly associated with acquired toxoplasmosis in humans [69,76,79–87].

While most of the earlier studies on acquired toxoplasmic retinochoroiditis have been based on sporadic cases [77,79,84,87], some recent studies have examined the outcome of multiple cases following outbreaks of acute toxoplasmosis in adults due to various sources (see Sections 4.2.4 and 4.3.3). In those outbreaks, in which a possible source of infection was revealed and dated by epidemiological investigation, the period between primary infection and onset of ocular symptoms ranged from 1 month to 3.5 years, while the age range of the patients was much wider, i.e. 10–57 years [79,81,82,86]. In the world's largest recorded outbreak of acquired toxoplasmosis in humans (100 cases, see Section 4.3.3), 20 patients with equal gender distribution and a mean age of 54 years (range 15–83 years) presented with retinal lesions within less than 1 year after the outbreak [69,87]. In addition, a population-based household survey in a rural area in southern Brazil suggested that an exceptionally high prevalence of familial ocular toxoplasmosis in that area, which is more than 30 times higher than estimates for the same condition elsewhere, has an acquired aetiology [88,89]. These findings were supported by a recent study in France on 49 patients with acquired toxoplasmosis of whom 44 also developed ocular symptoms [76]. As a screening programme for congenital toxoplasmosis is compulsory in France since 1978, a prenatal infection with *T. gondii* could be ruled out in several of those cases based on the documented immune status of the mother. Thus, it has now become clear that ocular toxoplasmosis may be both a result of a prenatal infection or an infection that was acquired postnatally.

3.3. Congenital toxoplasmosis

In immunocompetent hosts, infection with *T. gondii* usually results in life-long immunity against toxoplasmosis. Therefore, if a primary *T. gondii* infection is acquired 4–6 months before conception or earlier, protective immunity will usually prevent vertical transmission to the foetus on subsequent exposures. The exception is seen in immunocompromised women with systemic lupus erythematosus (SLE) or acquired immunodeficiency syndrome (AIDS) where previously infected, seropositive individuals have transmitted *T. gondii* congenitally [90].

However, if first contracted during pregnancy, *T. gondii* may also be transmitted to the foetus in immunocompetent women. The mechanism of vertical transmission is not yet understood. A probable scenario is that temporary parasitaemia in a primarily infected pregnant woman may result in invasion of the placenta by tachyzoites which then multiply within cells of the placenta. Eventually, some of these may cross the placenta and enter the foetal circulation or foetal tissues [31,91]. Congenital toxoplasmosis may cause abortion, neonatal death, or foetal abnormalities with detrimental consequences for the foetus [31,33,42,92]. It may also significantly reduce the quality of life in children who survive a prenatal infection [11,93–95].

Over the past 3 decades, the incidence of prenatal infection with *T. gondii* has been estimated to vary from 1 to 100 per 10 000 births in different countries [11,12,31,42,93,94]. The risk of intrauterine infection of the foetus, the risk of manifestation of congenital toxoplasmosis, and the severity of the disease depend on the time of maternal infection during pregnancy, the immunological competence of the mother during parasitaemia, the number and virulence of the parasites transmitted to the foetus, and the age of the foetus at the time of transmission. If not treated, the risk of intrauterine infection of the foetus increases during pregnancy, i.e. from about 14% after primary maternal infection in the first trimester to about 59% after primary maternal infection in the last trimester [31,42]. Because of this, the incidence of prenatal infection with *T. gondii* varies from the incidence of primary maternal infection during pregnancy. Incidence rates also vary depending on the method of estimation. Estimates may be derived directly from surveys at birth or during infancy, or indirectly from prospective surveys of acquired *T. gondii* infection during pregnancy [31]. Recent estimates based on serological studies suggested incidences of primary maternal infection during pregnancy to range from about 1 to 310 per 10 000 pregnancies in different populations in Europe, Asia, Australia, and the Americas (Table 4). These rates are dependent on the prevalence of infection in the population under study and are slightly higher (6–410 per 10 000) if only susceptible women are taken into account, i.e. those women who have not developed immunity before conception (Table 4). Incidences of prenatal infection with *T. gondii* in the same or similar populations have been estimated to range from about 1 to 120 per 10 000 births (Table 5).

While the risk of intrauterine infection of the foetus increases during pregnancy, the effects on the foetus are more severe if transmission occurs at an early stage of pregnancy [31,42,96,97]. The most significant manifestation in the foetus is encephalomyelitis which may have severe consequences. About 10% of prenatal infections result in abortion or neonatal death [31,42]. Another 10–23% of prenatally infected newborns show clinical signs of toxoplasmosis at birth [31,42,93,98]. Signs of the classic triad of toxoplasmosis (retinochoroiditis, intracranial calcifications, and hydrocephalus) manifest in up to 10% of these newborns, while the other newborns show a variety of symptoms, ranging from central nervous symptoms to non-specific symptoms of acute infection (retinochoroiditis, convulsions, splenomegaly, hepatomegaly, fever, anaemia, jaundice, lymphadenopathy etc.). About 12–16% of these newborns die from the disease. The surviving infants suffer from progressive mental retardation or other neurological deficiencies which often require special education and residential care [11,31,93,94].

However, if transmission occurs at a late stage of pregnancy the effects on the foetus are less severe, with most infants infected during the third trimester being asymptomatic at birth. In total, in about 67–80% of prenatally infected infants the infection is subclinical and can only be diagnosed using serological and other laboratory methods. Although these infants appear healthy at birth, they may develop clinical symptoms and deficiencies later in life. These deficiencies predominantly affect the eyes (retinochoroiditis, strabismus, blindness), the CNS (psychomotorical or other neurological deficiencies, convulsions, mental retardation),

or the ear (deafness) [31,42,95]. It has been estimated that about one third of prenatally infected children will develop visual impairment later in life [11,99,100].

3.4. Toxoplasmosis in immunocompromised humans

In immunocompromised humans a previously acquired latent infection can lead to reactivated toxoplasmosis with encephalitis. Toxoplasmic encephalitis and disseminated toxoplasmosis have been observed in patients with immunodeficiencies due to various causes, such as Hodgkin's disease or immunosuppressive therapy because of other malignancies. Disseminated toxoplasmosis may also complicate transplantation of organs or bone marrow. This may result either from transplantation of an organ from a *T. gondii*-infected donor to a susceptible recipient or from reactivation of a latent *T. gondii* infection in the recipient due to immunosuppressive treatment [12,73,101].

T. gondii is also an important opportunistic pathogen in AIDS patients. World-wide, *T. gondii* causes severe encephalitis in up to 40% of AIDS patients, and 10–30% of AIDS patients infected with *T. gondii* succumb to the disease [73,101–104]. However, with highly active antiretroviral therapy (HAART) and immune reconstitution the incidence of CNS toxoplasmosis in AIDS patients is now declining in many countries, and reactivation of a latent infection can also be prevented by prophylaxis with trimethoprim-sulfamethoxazole (TMX-Sulfa).

In addition to reactivated toxoplasmosis immunocompromised patients are at risk from severe disease following primary infection, which frequently presents as pulmonary disease or diffuse encephalitis [73].

4. How do humans acquire an infection with *Toxoplasma gondii*?

With incidences of prenatal infections ranging from 1 to 120 per 10 000 births (Table 5), and seroprevalences in women of childbearing age ranging from 4 to 85% (Table 3), only a small percentage of infections with *T. gondii* in adult human populations are acquired vertically. This raises the question of how humans acquire the infection postnatally. Not all possible routes of infection are important epidemiologically, and sources of infection may vary greatly among different ethnic groups and geographical locations. Therefore, knowledge on the more probable routes of horizontal transmission to humans and on the most likely sources of infection in a given population is a pre-requisite for the development of effective strategies for prevention of infection in risk groups, such as non-immune pregnant women and immunocompromised patients, in particular those with AIDS.

4.1. Tachyzoites

Tachyzoites play the major role in vertical transmission of *T. gondii* (see Section 3.3). By contrast, they are very sensitive to environmental conditions and are usually killed rapidly outside the host. Therefore, it is generally believed that horizontal transmissions of *T. gondii* infections via tachyzoites are not important epidemiologically. However, they may occur infrequently.

In recent years, it has been found that transplantation of heart, kidney, liver, and bone marrow may be complicated by *T. gondii* infections (see Section 3.4). In these cases either tachyzoites or tissue cysts may be involved [11,101]. Tachyzoites of *T. gondii* have also been transmitted via blood products, in particular those containing the white cell fraction, and by accidental injection in the laboratory [11,20,31,73,105]. However, parasitaemia usually occurs for only a short period of time after primary infection. Therefore, it has been suggested that there is only a low risk of acquiring an infection with *T. gondii* via ordinary blood transfusion [11].

Tachyzoites of *T. gondii* have been found in the milk of several intermediate hosts, including sheep, goats, and cows [11–13,20,23,31], but thus far, acute toxoplasmosis in humans has been associated only with consumption of unpasteurised goat's milk [82,106–108]. Tachyzoites are sensitive to proteolytic enzymes and usually are destroyed by gastric digestion. However, a recent study showed that tachyzoites may occasionally survive for a short period of time (up to 2 h) in acid pepsin solutions, and that oral application of high doses of tachyzoites may cause an infection in mice and cats [109]. It has also been suggested that tachyzoites may enter the host by penetration of mucosal tissue and thereby gain access to the host's circulation or lymphatic system before reaching the stomach [23,82,106]. This may also explain a recent report of toxoplasmosis in a breast-fed infant whose mother acquired a primary infection with *T. gondii* [110]. However, tachyzoites are sensitive to temperature and, thus, it is interesting to note that in a family of goat owners *T. gondii* was transmitted to two children who frequently consumed unpasteurised goat's milk while their parents who only had small amounts of goat's milk in tea or coffee remained seronegative [20,108]. Tachyzoites are killed by pasteurisation and heating. Therefore, it is advisable that milk, in particular goat's milk, should be pasteurised or boiled before human consumption. This is particularly important for its use in infants who have a lower concentration of proteolytic enzymes in the digestive tube and who are more susceptible to toxoplasmosis than adults. A recent study assessing risk factors associated with primary *T. gondii* infections in women of childbearing age suggested that in Poland drinking milk may be a potential risk factor for horizontal transmission to humans [111]. In the past, it has often been thought that the risk of acquiring an infection with *T. gondii* by drinking cow's milk, if any, is minimal [11,12,14,39], but it cannot be excluded that any type of milk is a potential source of infection if consumed raw. Likewise, it has been suggested that the high seroprevalence of *T. gondii* (67%) in pastoral camels in Sudan may be of public health significance for nomads who consume cameline milk raw [112].

In addition to blood and milk, tachyzoites have been detected in other body fluids, including saliva, sputum, urine, tears, and semen [11,20,31], but there is currently no evidence of horizontal transmission of *T. gondii* to humans via any of these routes. An early study reported that *T. gondii* tachyzoites may be isolated from raw chicken eggs laid by hens with experimentally induced infection [113]. However, commercially raised poultry is virtually free of *T. gondii* infection (see Section 4.2.1). In addition, tachyzoites are highly susceptible to both heating and salt concentration and, thus, any type of cooking would kill tachyzoites in eggs.

In general, it is believed that the majority of horizontal transmissions to humans are caused by ingestion of one of the two persistent stages of *T. gondii*, i.e. tissue cysts in infected meat or offals (viscera) and oocysts in food or water contaminated with feline faeces [11,13,114–116].

4.2. Tissue cysts

4.2.1. Importance and prevalence of infections with *Toxoplasma gondii* in meat-producing animals—Tissue cysts of *T. gondii* contained in meat of livestock are an important source of infection for humans (Fig. 2). Tissue cysts may develop as early as 6–7 days after infection of intermediate hosts by both oocysts or other tissue cysts [35]. They probably persist for the life of the host (see Section 2). However, the number of tissue cysts that may develop inside a certain host and the locations parasitised vary with the intermediate host species [35,114,116–118]. In meat-producing animals, tissue cysts of *T. gondii* are most frequently observed in tissues of infected pigs, sheep, and goats, and less frequently in infected poultry, rabbits, dogs, and horses (Fig. 3). Tissue cysts are found only

rarely in beef or buffalo meat, although antibodies in up to 92% of cattle and up to 20% of buffaloes are evidence of past exposure to the parasite (Table 6).

In Europe and in the USA, pork has generally been considered to be a major source of *T. gondii* infection in humans [115–117,119]. This hypothesis is based on the fact that tissue cysts have been found in most commercial cuts of pork [120,121], and on estimates for prevalences of *T. gondii* infection in pigs that were made in the 1970s or 1980s [11]. However, depending on the method used to obtain such estimates, these data vary greatly among different countries and among different farms within the same country. In most countries epidemiological data on infections with *T. gondii* in livestock are not regularly monitored. Recent studies on fattening pigs raised on farms using intensive management in the Netherlands, Austria, and Germany demonstrated that the prevalence of *T. gondii* infection in pigs has decreased significantly, (i.e. to <1%) over the last decade with changes in pig production and management (Table 7) [114,118,122]. Seroprevalences of *T. gondii* infection in fattening pigs raised on farms using intensive management have now been found to be <10% in many countries (Table 8). In addition, in several countries of the European Union seroprevalences in older pigs, such as sows, which are usually kept on farms with more extensive management and, consequently, are more frequently exposed to the environment than fattening pigs, also decreased distinctly (Table 7).

These data show that it is possible to significantly reduce the risk of *T. gondii* infection in livestock using intensive farm management with adequate measures of hygiene, confinement, and prevention. These measures include: (A) to keep meat-producing animals indoors throughout their life-time, (B) to keep the sheds free of rodents, birds, and insects, (C) to feed meat-producing animals on sterilised food, and (D) to control access to sheds and feed stores, i.e. no pet animals should be allowed inside them [117]. Using such preventive measures, it is economically possible to produce pigs and poultry free of *T. gondii* infection (Tables 7–9), although this has been achieved in only a few countries, i.e. in the Netherlands, Denmark, and the former German Democratic Republic [117,122].

By contrast, production of free-ranging livestock will inevitably be associated with *T. gondii* infection. Animals kept on pastures with an increased pressure of infection due to contamination of the environment with oocysts (see Section 4.3.2), such as sheep and goats, show high seroprevalences in many areas of the world, i.e. up to 92 and 75%, respectively, (Tables 10 and 11). This is of particular importance, because tissue cysts have been found in many edible parts of sheep [123,124], and small ruminants are important in both milk and meat production throughout the world (see Sections 4.1 and 4.2.4).

Seroprevalences are distinctly lower and more varying in horses, rabbits, and poultry (Tables 9, 12 and 13). This may reflect epidemiological factors such as different types of confinement, hygiene of stables, and different types of feed. By contrast, seroprevalences are usually high in dogs, indicating their continuous exposure to a natural environment and the cumulative effect of age (Table 14).

4.2.2. Prevalence of infections with *Toxoplasma gondii* in game and wild animals—Tissue cysts of *T. gondii* in venison and other meat of wild animals, including hares, wild boars, deer and other cervids, kangaroos, and bears are another potential source of infection for humans. Hunters and their families may also become infected during evisceration and handling of game [39]. Thus, in addition to the frequent consumption of caribou meat (see Section 4.2.4), a recent outbreak of congenital toxoplasmosis in Inuits was associated with skinning of animals (wolf, fox, and marten) for fur by women during pregnancy [125]. These cases also show that toxoplasmosis in humans may occur in arctic regions, although *T. gondii* infections are less frequent in regions with a cold climate than in

regions with a warm and humid climate (see Section 3.1). In addition to higher environmental pressure of infection, there is a cumulative effect of age in many wild animals which results in very high prevalences of infection. Some recent data on *T. gondii* infections in wild mammals and birds are contained in Tables 9 and 15.

Some wild animals, such as Australian native marsupials, have evolved in the absence of *T. gondii* until cats were introduced to their environment only a few hundred years ago. As a consequence, these animals are highly susceptible to the parasite. Although seroprevalences of *T. gondii* infection in marsupials are usually lower than in mammals, kangaroo meat in particular has recently been recognised as a potential source of infection for humans, because it is very lean with little fat and, thus, is usually consumed rare or undercooked [126].

4.2.3. Survival of tissue cysts of *Toxoplasma gondii* in food for humans—

Bradyzoites of *T. gondii* are more resistant to digestive enzymes, (i.e. pepsin and trypsin) than tachyzoites (see Section 4.1) [35,109,127]. Therefore, ingestion of viable tissue cysts by a non-immune host will usually result in an infection with *T. gondii*. Although tissue cysts are less resistant to environmental conditions than oocysts (see Section 4.3.2), they are relatively resistant to changes in temperature and remain infectious in refrigerated (1–4°C) carcasses or minced meat for up to 3 weeks [123,128], i.e. probably as long as the meat remains suitable for human consumption [116]. Tissue cysts also survive freezing at temperatures between –1 and –8°C for longer than a week [129]. Most tissue cysts are killed at temperatures of –12°C or lower [129,130], but occasionally some tissue cysts may survive deep-freezing [116]. It has also been suggested that some strains of *T. gondii* may be resistant to freezing [130].

By contrast, tissue cysts in meat are killed by heating to 67°C [116,128]. Survival of tissue cysts at lower temperatures depends on the duration of cooking. For example, under laboratory conditions tissue cysts remained viable at 60°C for about 4 min and at 50°C for about 10 min [128]. It is important to note that cooking for a prolonged period of time may be necessary under household conditions to achieve the temperatures that are required to kill all tissue cysts of *T. gondii* in all parts of the meat. Some tissue cysts will remain infectious if cooking procedures are used in which the meat is heated unevenly, for example microwave cooking [124].

Some studies suggested that tissue cysts are killed by commercial procedures of curing with salt, sucrose, or low temperature smoking [124,132]. Therefore, it has previously been suggested that processed meat is an unlikely source of infection for humans [114,119]. However, the survival time of tissue cysts varies greatly with the concentration of the salt solution and the temperature of storage [132]. Under laboratory conditions, tissue cysts were killed in 6% NaCl solution at all temperatures examined (4–20°C), but survived in aqueous solutions with lower concentration of salt for several weeks [132]. It has also been shown that salting does not necessarily kill tissue cysts in home-made pork sausages [133,134]. In one study, *T. gondii* tissue cysts were killed by 3% table salt after 3–7 days [133]. This is much later than the usual storage time for pork sausages and, thus, salting alone is probably not sufficient to prevent transmission to humans via tissue cysts.

Tissue cysts are killed by gamma irradiation at a dose of 1.0 kGy which has been approved by authorities in the USA [116,130]. However, irradiation of meat has only been approved in a few countries, it is only feasible in industrialised countries, and is opposed to by consumers in many regions of the world.

4.2.4. Food-borne outbreaks of toxoplasmosis in humans, risk factors, and preventive measures that reduce the risk of food-borne infection with *Toxoplasma gondii*

Recent outbreaks of acute toxoplasmosis in humans in various regions of the world demonstrate that the sources of infection vary greatly in different human populations with differences in culture and eating habits. In Canada, an outbreak of congenital toxoplasmosis in a settlement of Inuits in northern Quebec was associated with frequent consumption of caribou meat, in addition to skinning of fur animals (see Section 4.2.2), while seropositivity in pregnant women living in the same settlement was associated with consumption of dried seal meat, seal liver, and raw caribou meat [125,135]. In Australia, an outbreak of acute and congenital toxoplasmosis was associated with rare kangaroo meat and undercooked lamb satay which were consumed during a cocktail party in Queensland [136]. Consumption of raw mutton at a party has also been reported as a source of acute toxoplasmosis in humans in Brazil [85]. It is also important to consider that, in addition to meat, tissue cysts of *T. gondii* may form in visceral organs (see Section 2). Thus, an outbreak of acute toxoplasmosis in humans occurred after consumption of raw spleen and liver of a wild boar, and a second outbreak after consumption of raw liver of a domestic pig, in Korea where raw liver is believed to have special nutritional value [86]. In the latter cases, either tachyzoites or tissue cysts may have been involved.

While these reports highlight that the risk of acquiring an infection with *T. gondii* via meat or other edible parts of animals varies with cultural and eating habits in different human populations, data derived from outbreaks of acute toxoplasmosis are usually linked to an occasional point source of infection and, thus, do not necessarily reflect the major, epidemiologically important sources of infection for the whole population. For example, kangaroo meat has only recently become commercially available for human consumption in Australia [126], yet 35% of women of child-bearing age in Western Australia show serological evidence of previous exposure to *T. gondii* [137]. It has to be kept in mind that most infections with *T. gondii* in immunocompetent humans are asymptomatic and, thus, will not be recorded unless systematic screening programs for *T. gondii* infections are carried out in the population under study (see Section 5).

Only recently, comprehensive case-control studies have been aimed at identifying the different sources of infection with *T. gondii* in different human populations. In several studies associated with the European Research Network on Congenital Toxoplasmosis [138], a large number of women were screened for seroconversion during pregnancy [139–142]. A European multicentre study including selected cities in Belgium, Denmark, Italy, Norway, Switzerland, and the UK identified the consumption of undercooked lamb, beef, or game, contact with soil, and travel outside Europe and North America as strong risk factors for acquiring an infection with *T. gondii*, with 30–63% of infections in the various regions being attributed to consumption of undercooked or cured meat products [142]. Likewise, consumption of raw pork and tasting of raw meat during meal preparation were the principle risk factors for acquiring a *T. gondii* infection in a similar population in Poland [111]. Frequent consumption of meat or consumption of undercooked meat have also been associated with seroconversion or seropositivity for *T. gondii* in case-control studies on healthy adults in France, Yugoslavia, and the USA [141,143,144].

However, while consumption of raw or undercooked meat was consistently identified as a risk factor in all of these studies, the relative importance of the risk factor and the type of meat associated with it varied among different countries [142]. For example, in France consumption of undercooked beef was a stronger risk factor than consumption of undercooked lamb [141], in Norway consumption of undercooked lamb was a stronger risk factor than consumption of undercooked pork [140], whereas in Poland consumption of undercooked pork was the principle risk factor identified in the study [111]. These findings

may reflect differences in eating habits of consumers or different prevalences of infection in meat-producing animals in these regions. Thus, in Norway up to 18% of sheep, but only 3% of slaughter pigs are infected with *T. gondii* [145,146], whereas 36% of slaughter pigs are infected in Poland [147].

To prevent food-borne horizontal transmission of *T. gondii* to humans, meat and other edible parts of animals should not be consumed raw or undercooked, i.e. they should be cooked thoroughly (67°C) before consumption. Although freezing alone is not a reliable means of rendering all tissue cysts non-infectious (see Section 4.2.3), deep-freezing meat (−12°C or lower) before cooking can reduce the risk of infection. In addition, meat should not be tasted during seasoning or cooking [111,142], which is of particular importance for non-immune pregnant women (see Sections 3.3 and 5.1). It is also essential that preventive measures to reduce the risk of horizontal transmission of *T. gondii* to humans via tissue cysts include a high standard of kitchen hygiene. Thus, in a case-control study in Norway, washing kitchen knives infrequently after preparation of raw meat was independently associated with an increased risk of primary infection during pregnancy [140]. Both tissue cysts and tachyzoites are killed by water [127] and, thus, hands and all kitchen utensils used for the preparation of uncooked meat or other food from animals should be cleaned thoroughly with hot water and soap [116].

4.3. Oocysts

4.3.1. Importance of cats in the epidemiology of infections with *Toxoplasma gondii*—Infections with *T. gondii* in cats are usually asymptomatic, and vertical transmissions occur only infrequently [11,36,148]. However, latent infections with *T. gondii* are common in domestic cats and wild felines throughout the world [11,36]. At least 17 species of wild felines have been reported to shed oocysts of *T. gondii*, i.e. European and African wild cats, Pallas cat, bobcat, leopard cat, Amur leopard cat, iriomote cat, ocelot, Geoffroy's cat, Pampas cat, jaguarundi, cougar, leopard, jaguar, tiger, lion, and cheetah [149], and there is serological evidence of *T. gondii* infection in servals [150]. In domestic cats, antibodies to *T. gondii* may be detected in up to 74% of adult cat populations, depending on the type of feeding and whether cats are kept indoors or outdoors (Table 16). Seroprevalences are usually higher in stray or feral cats than in cats living in an urban or suburban environment. However, between 9 and 46% of pet cats in Europe, South America, and the USA show serological evidence of past exposure to the parasite, while seroprevalences of *T. gondii* infections in Asia have been estimated to range between 6 and 9% (Table 16).

Domestic cats and other feline species may become infected with *T. gondii* either by ingesting infectious oocysts from the environment or by ingesting tissue cysts from intermediate hosts. For example, cats may ingest tissue cysts when feeding on food scraps containing meat or viscera of livestock or game animals. Cats that are allowed to hunt may become infected by feeding on carcasses of small mammals or birds infected with *T. gondii* (Fig. 2). Depending on the host species, the geographical area, and the season of the year, up to 73% of small rodents and up to 71% of wild birds may be infected with *T. gondii* (Table 9) [12,151–164].

After primary infection of cats with tissue cysts of *T. gondii*, the bradyzoites immediately initiate a phase of asexual proliferation which consists of numerous cycles of endopolygony in the small intestine. The terminal stages of this proliferation initiate the sexual phase of reproduction which results in the formation of oocysts (see Section 2, Fig. 1). Almost all cats that have been infected primarily with tissue cysts shed oocysts after a prepatent period of 3–10 days, with patency lasting for up to 20 days [12,22,36,165]. By contrast, after primary infection with oocysts of *T. gondii*, the sporozoites first initiate a phase of asexual

multiplication, which is similar to the development in intermediate hosts, in extraintestinal tissues (see Section 2, Fig. 1). In the course of this development, some parasites migrate to intestinal tissues and initiate a sexual phase of reproduction. About one third of cats that have been infected primarily with oocysts shed other oocysts after a prolonged prepatent period of 18–49 days for up to 10 days [166,167]. Cats may also become infected by ingesting large numbers (≥ 1000) of tachyzoites (see Section 4.1) which may result in shedding of oocysts after 15–19 days for up to 7 days [109].

Cats usually only shed large numbers of oocysts after a primary infection with *T. gondii*. It has previously been believed that shedding of oocysts after reinfection or reshedding of oocysts in the absence of reinfection with *T. gondii* is rare. However, recent studies showed that this immunity does not persist for the life of the cat. A second shedding of oocysts could be induced in cats that were challenged with *T. gondii* about 6 years after primary infection [168,169]. In addition, in some cases short-term reshedding of oocysts has been observed without reinfection of the cat. It is currently not known which factors induce a reshedding of oocysts under natural conditions. Experimentally, reshedding of oocysts may be induced by superinfection with other coccidia, for example *Isospora* species, as well as after immunosuppression, for example due to application of high doses of corticosteroids [11,12,36,119,149,170].

The domestic cat is the only domestic animal that is used as a definitive host by *T. gondii*, and thus appears to play a key role in the epidemiology of *T. gondii* infections. After primary infection with *T. gondii* cats that are kept inside houses may shed large numbers of oocysts into the household, thereby putting their owners at risk of infection. Stray cats or cats that are roaming on farms may contaminate the environment with oocysts which may infect livestock that will later be slaughtered for human consumption (Fig. 2). However, oocysts shed by cats are unsporulated and, thus, are not immediately infectious (see Section 4.3.2, Fig. 1). Therefore, direct contact with cats usually does not result in an infection with *T. gondii*. Cat-ownership and keeping of cats inside houses or flats does not necessarily provide a risk of contracting a *T. gondii* infection, if preventive measures are effective and cat faeces are removed daily from the household (see Section 4.3.3).

4.3.2. Importance and survival of oocysts of *Toxoplasma gondii* in the environment—By contrast, sporulated oocysts contained in the environment are a potential source of infection for humans and other intermediate hosts. The epidemiological importance of oocysts is highlighted by the fact that, despite the worldwide distribution of *T. gondii*, infections with this parasite are virtually absent on small islands and atolls which have never been inhabited by cats [171,172]. Contamination of the environment with oocysts of *T. gondii* may be due to infected domestic cats or wild felines. After primary infection with tissue cysts or oocysts of *T. gondii*, a single cat may shed more than 100 million oocysts into the environment [12,22,167,173]. Under environmental conditions with sufficient aeration, humidity, and warm temperature oocysts sporulate and become infectious within 1–5 days [12,20,36], while sporulation may be delayed under microaerophilic conditions [22]. Depending on the strain of *T. gondii*, ingestion of as few as 10 sporulated oocysts may cause an infection in intermediate hosts, such as pigs [174], and ingestion of 100 or more sporulated oocysts may result in a patent infection in felines [167], thereby further contributing to the contamination of the environment with oocysts.

Sporulated oocysts of *T. gondii* are very resistant to environmental conditions. They survive short periods of cold and dehydration, and remain infectious in moist soil or sand for up to 18 months [22,165]. Under laboratory conditions, sporulated oocysts survived storage at 4°C for up to 54 months and freezing at –10°C for 106 days [175]. However, they were killed

within 1–2 min by heating to 55–60°C [175]. Sporulated oocysts also are highly impermeable and, therefore, are also very resistant to disinfectants [11,20,22,36,130].

Oocysts are distributed in the environment through wind, rain, and surface water, or harvested feeds. Hay, straw, and grain which had been contaminated with cat faeces have been identified as sources of infection for livestock [11,176]. In addition, oocysts may be spread via earthworms, coprophagous invertebrates, or manure [11,177]. In the gut of cockroaches, which may act as transport hosts of *T. gondii*, oocysts remain infectious for up to 19 days [178]. Humans may become infected via contact with contaminated soil, for example through gardening (see Section 4.3.3). Oocysts of *T. gondii* have been isolated from samples of soil in various areas of the world [22,36,165]. It has also been suggested that the fur of dogs that have come in contact with cat faeces may be a vector for transmission of oocysts to humans [155].

4.3.3. Oocyst-transmitted outbreaks of toxoplasmosis in humans, risk factors, and preventive measures that reduce the risk of oocyst-transmitted infection with *Toxoplasma gondii*

—There are several preventive measures that may reduce the risk of horizontal transmission of *T. gondii* infections to humans via oocysts. In order to advise appropriate measures for prevention of oocyst shedding by pet cats, cat owners belonging to risk groups, i.e. non-immune pregnant women and immunocompromised patients (see Sections 3.3 and 3.4), should have their cat examined with respect to infection with *T. gondii*. In this context, examination of faeces is not an appropriate procedure as most cats will shed the majority of oocysts during only 1–2 days, while the whole period of patency may last for up to 20 days (see Section 4.3.1). On those days on which only few oocysts are shed and in cases of reshedding, koproscopic methods may provide a negative result although faeces are still infectious for intermediate hosts as shown in bioassays. Therefore, examination of faeces is only meaningful for the cat owner if the result is positive, i.e. if toxoplasma-like oocysts have been detected in the faeces of the cat.

In epidemiological situations it is more advisable to examine the cat serologically for *T. gondii*-specific antibodies to find out the immune status of the cat [148]. A serologically negative result suggests that the cat has not yet been exposed to *T. gondii* and is still susceptible to infection in the future. In such cases cats should be fed on dry, canned, or cooked food and should be prevented from hunting to prevent a primary infection with *T. gondii*. In addition, the environment of the cat should be controlled with respect to potential intermediate hosts, such as mice and rats, as well as transport hosts, such as cockroaches and other invertebrates. A serologically positive result suggests that the cat already has been infected with *T. gondii* in the past. The majority of cats with detectable levels of IgG antibodies to *T. gondii* are likely to be immune and, thus, will not shed oocysts in the near future. Cats that have been infected via tissue cysts usually seroconvert (IgG) between 2 and 5 weeks post-infection, which is after the period of patency (see Section 4.3.1) [150,169,173,179–183]. However, in some cats that have been infected by ingestion of oocysts, IgG antibodies are already detectable during the prolonged period of prepatency [167]. Thus, while detection of IgG antibodies in the serum of cats is indicative of immunity in most cases, it does not exclude that in some rare cases shedding of oocysts may still occur. In addition, some previously infected cats may reshed oocysts over short periods of time (see Section 4.3.1), and consequently immuno-suppressive treatment of cats owned by individuals belonging to risk groups should be avoided. It may also be advisable that immunocompromised individuals should not keep a cat in their household.

In all cases, faeces of pet cats should be removed daily from the household. Cat litter boxes and all items that may have come in contact with cat faeces should be cleaned thoroughly with hot water (>70°) and detergents wearing gloves, but preferably not by

immunocompromised individuals or pregnant women. In a case-control study on primary maternal infection in Norway, cleaning the cat litter box was identified as a strong risk factor [140]. However, with the appropriate preventive measures the risk of acquiring an infection with *T. gondii* from a pet cat can be highly controlled by its owner. Accordingly, in the same case-control study in Norway as well as in a European multi-centre case-control study on primary *T. gondii* infections in humans, neither direct daily contact with cats nor living in a household or neighborhood with cats were associated with *T. gondii* infection [140,142].

On the other hand, while consumption of undercooked meat was identified as the principle risk factor in several recent case-control studies on *T. gondii* infections in humans (see Section 4.2.4) [111,139,140,142,144], this finding does not explain the high rate of seropositivity (24–47%) in some populations of vegetarians [144,184]. However, a few risk factors identified in those studies point to the importance of oocysts in the transmission of *T. gondii* infections to humans. For example, contact with soil was identified as a strong risk factor in the European multi-centre case-control study, and 6–17% of primary infections in humans were attributed to this factor [142], while in the case-control study in Norway, eating unwashed raw vegetables or fruits was associated with an increased risk of primary infection during pregnancy [140]. Thus, it is advisable that pregnant women and immunocompromised individuals should wash or cook vegetables and fruits, which may be contaminated with cat faeces, before consumption. These individuals should also wear gloves when working in gardens. In addition, *T. gondii* oocysts in sand pits used as childrens' playgrounds may be a source of infection. Geophagia was strongly associated with an outbreak of acute toxoplasmosis in six of 11 preschool-aged children of an extended family who played in the same sandy yard of their grandmother's house, which was also visited by cats [185]. Where ever possible, measures should be taken to prevent cats from defecating into childrens' playgrounds.

At least another three well-documented outbreaks of acute toxoplasmosis in humans have been associated with contamination of the environment by oocysts. In 1977, an outbreak of acute toxoplasmosis in 37 of 86 patrons of a riding stable in Atlanta, USA, was linked to inhalation of aerolized oocysts which were shed by cats in the stable [81,186–188]. Another outbreak of toxoplasmosis in 35 of 98 military trainees was linked to ingestion of oocyst-contaminated water during training in a jungle environment in Panama [189,190]. However, the largest and best documented outbreak of acute toxoplasmosis in humans to date occurred in 110 individuals in Vancouver, Canada, in 1995. Comprehensive, retrospective epidemiological studies provided strong evidence that this outbreak was caused by contamination of municipal drinking water with oocysts [69,191–194]. It has been suggested that both domestic cats and wild felines (cougars) may have been the source of this outbreak by shedding *T. gondii* oocysts into the environment of a reservoir that was the source of the municipal water system, which used unfiltered chloraminated surface water [69,193,195].

5. Strategies for surveillance and control of toxoplasmosis in humans

Preventive measures such as the ones described above (see Sections 4.2.4 and 4.3.3) can significantly reduce the risk of acquiring an infection with *T. gondii*, but cannot always prevent the infection. Therefore, because of the great impact that *T. gondii* has on the quality of human life several authorities, including the World Health Organisation, have advised strategies for surveillance and control of toxoplasmosis in humans. Such strategies are particularly important for risk groups and are directed at prevention of symptomatic congenital toxoplasmosis and long-term sequelae in children with prenatal *T. gondii* infection, and at prevention of fatal disease in immunocompromised patients.

In some European countries and some states of the USA, screening programmes have been launched that are aimed at either early detection of primary maternal infection with *T. gondii* during pregnancies or at detection of prenatal infection in neonates at birth. However, these programmes are not standardised and vary greatly among different countries. Such variation is largely due to controversy that exists on whether treatment of the mother during pregnancy is effective in reducing the risk of vertical transmission to the foetus, in reducing the risk of symptomatic congenital toxoplasmosis in the neonate, or in preventing long-term sequelae in the child. Consequently, there is debate on the cost-effectiveness of such programmes.

In Austria and France, prenatal screening programmes are mandatory and were already established in 1975 and 1978, respectively, [68,196,197]. More recently, prenatal screening has been initiated in parts of Italy (Campania region) [198], whereas Denmark, Poland (Poznan region), and parts of the USA (Massachusetts and New Hampshire) have established neonatal screening programmes [199–202]. While the establishment of these programmes has not clarified the debate about their costs and benefits in public health, they have significantly improved our knowledge on congenital toxoplasmosis as well as on many aspects of the management of, and outcome for, children with prenatal *T. gondii* infection. In addition, they have provided invaluable data on the epidemiology of infections with this parasite in humans.

5.1. Congenital toxoplasmosis

In 1923, Janků first described tissue cysts of *T. gondii* in the retina of an 11-month-old infant with congenital hydrocephalus and microphthalmia [203,204]. This was later recognised as the first recorded case of congenital toxoplasmosis in humans [205]. In the late 1930s, studies by Wolf and Cowen [206–208] led to the recognition of *T. gondii* as a causative agent of encephalomyelitis in human neonates. In the early 1940s, it was recognised that this disease had a congenital origin and, thus, resulted from vertical transmission of the parasite from the mother to her child [209]. The major symptoms of congenital toxoplasmosis in neonates and infants were described during the 1940s and early 1950s, but it was not before the 1960s that prenatal infection with *T. gondii* was also recognised as a cause of major sequelae later in life (Table 2).

Prenatal infection with *T. gondii* occurs in from about 1 to 120 per 10 000 live births depending on the geographic location and the population studied (Table 5). This rate can be modelled as a function of the seroconversion rate of the population at risk, e.g. women of childbearing age [210,211]. Thus, a seroconversion rate of 3% per year would suggest that 15% of women between the ages of 20 and 30 would seroconvert with an expected prenatal infection rate of about 46 per 10 000 live births.

Transmission of *T. gondii* to the foetus in an infected mother has an excellent correlation with isolation of the organism from the placenta. This transmission occurs during the initial acquisition of the infection by immunologically naive pregnant women. There is some debate if chronic infection can increase the rate of spontaneous abortion in subsequent pregnancies, but congenital toxoplasmosis is not a risk in subsequent pregnancies. There are only rare reports of transmission occurring in pregnancies of women who were seropositive at the time of conception [90,212]. The majority of these reports involve women who are immunocompromised with either steroid use and SLE or infection with human immunodeficiency virus (HIV) in the presence of a low CD4 count (less than 300) [90]. It is believed that reactivation of latent infection, (e.g. circulating tachyzoites) in an immunocompromised woman results in a seeding of the placenta and subsequent transmission to the foetus despite the seropositive status of the mother. In a study of 112 placentas from immunocompetent women who were seropositive for *T. gondii* before

pregnancy, no organisms could be found in the placenta [33]. In another study of 177 pregnancies that were terminated, women were separated into two categories; 62 women who acquired infection before or soon after conception and 115 women who acquired infection during pregnancy [213]. Of the women who acquired infection during pregnancy 10 out of 115 had organisms in the placenta, while no organisms were found in the placenta of the 62 women who acquired infection before or at conception. These data confirm that vertical transmission of *T. gondii* may occur if infection is acquired during pregnancy and that immunity acquired before pregnancy, as reflected by maternal IgG seropositivity, is protective for the foetus.

Vertical transmission of *T. gondii* during pregnancy is a function of when primary maternal infection occurs during the 40-week gestation period. The later maternal infection is acquired during pregnancy, the more frequently parasites are transmitted to the foetus. In the last few weeks of gestation the transmission rate rises to greater than 90%, while in the first 6 weeks after conception the rate is under 2% [213–215]. An average transmission rate for each trimester in which seroconversion occurs is 14% for the first trimester, 30% for the second trimester, and 59% for the third trimester. In prospective surveys of women who seroconvert during pregnancy the average incidence of prenatal infection in this population is 30% of the maternal seroconversion rate, (e.g. if the rate of maternal seroconversion is three per 1000 pregnancies the incidence of prenatal infection will be one per 1000 pregnancies). While the transmission rate rises during gestation, the risk of symptomatic congenital toxoplasmosis and the severity of the disease are inversely related to the week of gestation in which transmission occurs. Thus, the highest frequency of severe abnormalities at birth is seen in children whose mother acquired a primary infection with *T. gondii* between the 10th and 24th week of gestation [215]. Several studies have demonstrated that treatment of pregnant women who show evidence of recently acquired infection with *T. gondii* can decrease the severity of congenital toxoplasmosis in their children. In a study of 542 pregnancies in which spiramycin was given to women who seroconverted during pregnancy (388 women given treatment and 154 without treatment) the percentage of children without prenatal infection increased from 39 to 77%, and the percentage of children with severe congenital disease or intrauterine death decreased from 11 to 3% [214]. If transmission to the foetus can be documented in utero (by polymerase chain reaction (PCR) or other techniques [33]) the administration of pyrimethamine and sulfadiazine to the mother can treat the infection in utero and further decrease the severity of symptoms in infected children [216,217].

It should be appreciated that children who have been prenatally infected but are asymptomatic at birth will develop manifestations of this infection such as retinochoroiditis later in life [218]. Treatment of prenatally infected children in a recent clinical trial suggests that early treatment of children diagnosed with prenatal *T. gondii* infection can decrease these late manifestations and improve outcome for these children [219].

Prevention of congenital toxoplasmosis can thus be accomplished by preventing infection in seronegative pregnant women or by treatment of those women who acquire a primary infection with *T. gondii*, as reflected in maternal seroconversion, during pregnancy. In some cases, education programmes aimed at reducing the risk of primary maternal infection during pregnancy also have been successful in decreasing maternal seroconversion rates [220]. For example, a study at Saint Antoine Hospital demonstrated a decrease of the maternal seroconversion rate from 3.7 to 1.1% [221]. In these programs, women were informed about the epidemiology of *T. gondii* infections as well as preventive measures, such as the need to cook meat thoroughly, to wash hands after handling raw meat, to wash kitchen utensils that have come in contact with raw meat, to wash fruits and vegetables before consumption, to avoid contact with items contaminated with cat faeces, to use gloves

if cleaning a cat litter box cannot be avoided, to remove any cat faeces from such litter boxes on a daily basis, and to clean all litter boxes with hot water between litter changes (see Sections 4.2.4 and 4.3.3).

Serological screening of pregnant women is an effective strategy to prevent prenatal infections with *T. gondii* in their children. The rate of maternal seroconversion in a given population will determine the cost-effectiveness of such a screening strategy. For example, a seroconversion rate of 7.5% per year would indicate only 20% of women would be at risk of seroconversion during the childbearing years, while a seroconversion rate of 1% per year would indicate 80% of the women would be at risk of seroconversion during the childbearing years; yet in both cases the overall rate of prenatal infections for those populations would be 27 per 10 000 live births. Thus, to identify these 27 cases, only 20% of the women would need repeated screening if the seroconversion rate was 7.5% per year vs. 80% of the women in the other scenario. Nonetheless cost-benefit analysis suggests that even in areas with low incidence of *T. gondii* infections screening for primary maternal infections during pregnancy is economically worthwhile [222]. An effective screening strategy would involve serological examination of pregnant women for presence or absence of antibodies (IgG and IgM) to *T. gondii* at the time of presentation and in each subsequent trimester. Seroconversion should be followed up with confirmatory tests as well as determination by amniocentesis and PCR if transmission to the foetus has occurred. This has been extensively reviewed by Remington and co-authors [33]. Women who seroconvert during pregnancy should be treated with spiramycin, and if transmission to the foetus is documented, they should be treated with pyrimethamine and sulfadiazine. Once born, all children of mothers who seroconverted during pregnancy should be evaluated for congenital toxoplasmosis and treated if infection is still evident [214,217–219,223–231].

An alternative strategy for the identification of prenatal infections with *T. gondii* is serological screening of neonates [58,199,201,232–234]. The most common technique in neonatal screening has been to use dried blood collected on filter paper in serological tests for IgM antibodies to *T. gondii* [199,201,232–234], although neonatal screening utilising cord blood has also been effective [58]. Dried blood on filter paper is utilised for phenylketonuria (PKU) screening programmes in many countries and techniques have been published for the elution of antibodies from such samples [235]. As programmes that utilise such PKU samples for *T. gondii* serology can piggyback onto pre-existing collection systems for screening for genetic disorders, such neonatal screening programmes are cost-effective even in areas with a low incidence of prenatal infections with *T. gondii* [201,232]. The majority of neonatal screening programmes use IgM capture enzyme-linked immunosorbent assay (ELISA) techniques for screening, which have a sensitivity of only 70–90% (with some reports on sensitivities as low as 40% [236]). Thus, neonatal screening for only IgM antibodies to *T. gondii* will only identify a subset of the children with prenatal infection. To some extent this may explain the lower observed than expected prevalence of prenatal infections with *T. gondii*, compared to historical data, seen in such screening programmes [201,233]. Nonetheless, the experience with most of these programmes is that neonatal screening identifies prenatally infected children that would otherwise have gone undiagnosed and untreated. False-positive IgM serology occurs in about 0.19 per 1000 pregnancies. Therefore, the evaluation of a positive IgM result in a neonate should include serological tests for IgG and IgM antibodies to *T. gondii* in both the mother and the neonate. In some cases serial serological testing, serological tests for IgA antibodies to *T. gondii*, or immunoblots may be needed to clarify if congenital transmission has occurred [33,237]. Treatment of children identified with prenatal *T. gondii* infections has been demonstrated to decrease adverse sequelae [214,217–219,223–231]. Therefore, neonatal screening programmes for *T. gondii* infection should result in a net health care cost savings.

5.2. Immunocompromised patients

The other human population in which *T. gondii* causes severe disease is in immunocompromised patients where reactivation of latent infection causes symptomatic disease [238]. In heart transplantation, seronegative recipients are at high risk of toxoplasmosis, and primary prophylaxis with pyrimethamine and sulfadiazine for 6 weeks (at full treatment dosages) has been demonstrated to prevent this infection [239,240].

T. gondii has been an important opportunistic pathogen in AIDS patients, where the development of toxoplasmic encephalitis was reported to occur in up to 40% of seropositive patients during the course of their HIV infection (see Section 3.4). This infection has declined in the era of HAART due to the improved immune status of HIV infected patients, and CNS toxoplasmosis is now rarely seen in patients with CD4 counts over 200 [238]. Once CNS toxoplasmosis has been diagnosed in an AIDS patient, treatment should be continued indefinitely as stopping treatment is associated with a high relapse rate. Primary prevention of reactivated toxoplasmosis in HIV patients who are serologically positive for *T. gondii* has been successful with trimethoprim-sulfamethoxazole, dapsone-pyrimethamine, or fansidar (administered to prevent *Pneumocystis carinii* pneumonia) [241–243]. In HIV infected patients who are serologically negative for *T. gondii*, preventive measures as described in Section 4.2.4 and 4.3.3 are prudent as a primary infection with *T. gondii* in the setting of AIDS can result in pneumonia or other severe symptoms (see Section 3.4).

6. Conclusions

Because of the great importance of *T. gondii* as a causative agent of a zoonosis, public health organisations, such as the World Health Organisation, have repeatedly advised the collection of accurate epidemiological data on this parasite. Such data are essential to elucidate the relative importance of the various sources of infection for humans, to control disease, and to prevent reduction in quality of human life caused by this parasite. However, only few countries of the world regularly monitor toxoplasmosis in humans, and even less countries monitor *T. gondii* infection in animals.

Although it is now 3 decades ago that the heteroxenous life cycle of *T. gondii* was elucidated, we still know very little about the relative epidemiological importance of the various routes of horizontal transmission to humans. Because tachyzoites only survive for a short period of time outside the host, it has been generally accepted that postnatal infections in humans are acquired by ingesting one of the two persistent stages of *T. gondii*, i.e. tissue cysts contained in meat or viscera of many animals and oocysts shed into the environment by domestic cats or wild felines. However, their relative importance in the epidemiology of *T. gondii* infections remains obscure. On one hand, consumption of undercooked meat has been identified as the principle risk factor in several recent case-control studies on primary infection of *T. gondii* or seropositivity in humans, on the other hand up to 47% of strict vegetarians have been shown to possess antibodies to *T. gondii* [184].

It is likely that the major sources of *T. gondii* infections are different in human populations with differences in culture and eating habits. However, it is also important to take into account that epidemiology is in a state of flux, in particular in the case of a versatile parasite, such as *T. gondii*. There are many factors that have an impact on the epidemiology of *T. gondii* infections, such as the type of management and production of livestock, hygienic standards of abattoirs, food processing and technology, the density of cats or wild felines in the environment, environmental conditions that have an influence on the sporulation of oocysts in the environment, (i.e. temperature, humidity, wind), geographical location (with respect to latitude) as well as the different habits of human consumers, to name just a few.

Because of the versatility of *T. gondii* and its complex epidemiology, it is not possible to advise strategies for control or prevention of disease that are effective worldwide or are effective for all ethnic groups in one location. In recent case-control studies on pregnant women in six European countries, travel outside Europe was identified as a risk factor for acquiring a primary infection with *T. gondii* [140,142]. This finding points to the fact that people change their habits when entering different environments, and thus become at risk from sources of infection, if only temporarily, that are not important epidemiologically in their home situation. In addition, changes in the habits of consumers may have an impact on potential sources of infection with *T. gondii*. For example, very recently meat from kangaroos and other marsupials, which are highly susceptible to *T. gondii*, has become available to consumers outside Australia, for example in Europe. Because this meat is very lean it is usually served undercooked in restaurants, and thus has the potential of a new source of infection for European consumers. Examples such as these highlight the fact that the epidemiology of *T. gondii* infections in human populations may change when individuals change their eating habits or develop preferences for new food types.

Several recent studies have raised the hypothesis that water-borne transmission of oocysts to humans may have a greater epidemiological importance than has been believed previously. While the risk of acquiring an infection with *T. gondii* via oocysts that are shed by pet cats into the household of their owners may be significantly reduced by preventive measures, it is currently unknown whether measures for quality control of drinking water are sufficient for preventing its contamination with infectious oocysts of *T. gondii* from the environment. As a consequence, the Vancouver outbreak of toxoplasmosis in humans (see Section 4.3.3) has now initiated research on the oocyst stage of the parasite, and methods are being developed to facilitate their detection in drinking water [244]. It is desirable that future epidemiological studies on *T. gondii* should consider the role of oocysts as potential sources of infection for humans and there is a need for methods to monitor these in the environment.

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Fig. 1.

Life cycle of *T. gondii*. Development in the intermediate host is illustrated below the horizontal bar, development in the definitive host is illustrated above the horizontal bar. The infectious stages, i.e. tachyzoites, bradyzoites contained in tissue cysts, and sporozoites contained in sporulated oocysts, have been shaded (modified from Ref. [245]).

Fig. 2.
Major routes of transmission of *T. gondii*.

Fig. 3.

Relative importance of meat-producing and game animals in the transmission of *T. gondii* to humans, adapted from the Report on the WHO Workshop on Public Health Aspects on Toxoplasmosis, Meeting of the Working Groups 'Husbandry, Household and Environment' and 'Food Hygiene', Bilthoven, The Netherlands, 23rd-24th October, 1989 and from Refs. [116–118].

Table 1Entries on *T. gondii* or toxoplasmosis in different publication databases

Search terms ^a	ParasiteCD ^b	VETCD ^c	PubMed ^d
Toxoplasma*	10 753	6615	12 605
AND human*	8546	3915	9086
AND animal*	10 567	6611	6186
AND (zoonosis OR zoonoses)	553	604	253
AND (congenital OR pregnancy)	1400	377	3051
AND (immunocompromised OR immunosuppression)	1027	168	525
AND AIDS	882	49	1754
AND (prevalence OR seroprevalence)	1585	942	2275
AND epidemiology	1305	777	2141
AND transmission	707	473	859
AND (source* OR route*) AND infection	200	160	212
AND (tachyzoite* OR endozoite*)	980	791	782
AND (tissue cyst* OR bradyzoite* OR cystozoite*)	454	404	231
AND (oocyst* OR sporozoite*)	687	662	407
AND (therapy OR treatment)	2670	1040	3725
AND control	1632	894	1498
AND ((prevention OR preventive) NOT control)	252	120	120
AND (risk factor*)	99	36	36
AND (economy OR economic impact)	15	15	46

^a Boolean operators (in capitals) and truncations (*) were as shown. In PubMed phrase searching with double quotes was used to search for entries on 'tissue cyst*', 'risk factor*' and 'economic impact'.

^b ParasiteCD, 1973–2000/04 (CAB International); searches were carried out using the search and retrieval software WinSPIRS, version 2.0 (SilverPlatter International, N.V.).

^c VETCD, 1973–2000/05 (CAB International); searches were carried out using the search and retrieval software WinSPIRS, version 2.0 (SilverPlatter International, N.V.).

^d PubMed, 1966–2000/07; searches were carried out using the advanced search options of the new PubMed system of the National Center for Biotechnology Information (NCBI) at the National Library of Medicine (NLM), USA.

Table 2History of *T. gondii* and its emergence as a human pathogen

Year	Event	Reference
1900	Description of toxoplasma-like parasites in Java sparrows	[246,247]
1908	First description of toxoplasma-like tissue cysts in humans (as sarcosporidiosis)	[248]
1908	Description of <i>T. gondii</i> merozoites in gondi (first named <i>Leishmania gondii</i>)	[2]
1909	Introduction of the genus <i>Toxoplasma</i> (type species: <i>T. gondii</i>)	[3]
1923	First recorded case of toxoplasmosis in an 11-month-old infant with congenital hydrocephalus and microphthalmia (recognised retrospectively)	[203,204,249]
1928	First description of the tissue cyst as a persistent stage in intermediate hosts	[205]
1937	First recorded case of fatal disseminated toxoplasmosis in an adult (22-year-old) human	[250]
1937–39	Recognition of <i>T. gondii</i> as a causative agent of encephalomyelitis in human neonates	[206–208]
1939	Description of classic triad of symptoms of congenital toxoplasmosis in humans (retinochoroiditis, hydrocephalus, encephalitis followed by cerebral calcification)	[208]
1939	Identity of isolates from humans and animals based on biological and immunological similarities	[6]
1940–41	Recognition of <i>T. gondii</i> as a causative agent of acute, acquired disease in adult humans	[250,251]
1941–42	Comprehensive description of toxoplasmic encephalitis in children with acquired toxoplasmosis	[252,253]
1942	Vertical transmission recognised in humans	[209]
1948	Methylene blue dye test introduced for detection of antibodies to <i>T. gondii</i> (gold standard for <i>T. gondii</i> -specific serology in humans)	[40]
1951–52	Recognition of <i>T. gondii</i> as a causative agent of lymphadenopathy in humans	[254,255]
1952	Description of <i>T. gondii</i> as a causative agent of retinochoroiditis in humans	[71]
1952	Description of classic tetrad of symptoms of congenital toxoplasmosis in humans (retinochoroiditis, cerebral calcification, hydrocephalus or microcephalus, and psychomotor disturbances)	[256]
1953–54	First recorded case of toxoplasmic encephalitis in a patient with Hodgkin's disease	[257]
1954–56	Hypothesis that horizontal transmission to humans may occur via tissue cysts in undercooked meat (pork)	[258,259]
1959	Serological evidence of <i>T. gondii</i> infections in vegetarians	[260]
1960	Discovery that tissue cysts are resistant to proteolytic enzymes	[127,261]
1960	Description of major sequelae of congenital toxoplasmosis in humans	[262]
1965	Recognition of the coccidian nature of <i>T. gondii</i> based on the ultrastructure of extraintestinal merozoites	[263,264]
1965	Epidemiological evidence that horizontal transmission to humans occurs via undercooked meat	[265]
1965	Hypothesis that an infectious stage of <i>T. gondii</i> is passed into the environment via the faeces of cats	[9]
1968	Recognition of <i>T. gondii</i> as a complication in patients with malignancies	[266]
1969	Identification of the oocyst of <i>T. gondii</i>	[267–273]
1970	Description of the sexual phase of the life cycle in the small intestine of cats	[270,274–277]
1969–72	Recognition of the epidemiological role of cats in the spread of <i>T. gondii</i> in different geographical areas	[171,172]
1981–82	First recorded cases of CNS toxoplasmosis in AIDS patients	[278]
1984	Recognition of <i>T. gondii</i> as an opportunistic pathogen in AIDS patients	[102]
1995–99	Largest recorded outbreak of acute toxoplasmosis in humans (100 individuals aged 6–83 years) associated with oocysts in municipal drinking water	[69,191,279]

Table 3

Seroprevalences of *T. gondii* infection in women of childbearing age (1990–2000)

Country	Year of sampling ^a	BOH ^b	Seroprevalence (%) ^c	Number of samples tested (n)	Method ^d	Reference
Argentina	1992–94	No	59	3049	IFAT	[280]
Australia	1986–89	No	35	10207	DAT	[137]
Austria	1981–91	No	43	167041	SFDT	[196]
	1993–94	No	50	8596	<i>e</i>	[281]
	1994–95	No	37	2413	<i>e</i>	[282]
	1995–96	No	43	18227	<i>e</i>	[281]
	1997	No	42	4601	<i>e</i>	[281]
Bangladesh	1991	Yes	16	302	LAT	[283]
	1994–95	No	11	617	LAT	[284]
	< 1998	No	38	286	ELISA	[285]
Belgium	1979–90	No	56	11286	IFAT	[286]
	1990	No	50	784	MEIA	[287]
Benin	1993	No	54	211	ELISA	[288]
Brazil	1997	No	72	185	ELISA	[289]
Cameroon	1989–90	No	77	192	ELISA	[290]
China	< 1995	No	39	1211	ELISA	[291]
	1996	No	4	557	IHAT	[292]
Colombia	1991–92	No	60	937	IFAT	[67]
Congo	1986–90	No	60	2897	IHAT	[293]
Croatia	1989–93	No	46	2778	ELISA	[294]
Cuba	1990–91	No	71	362	ELISA	[295]
	1990–91	No	71	5537	ELISA	[296]
	< 1993	No	51	3196	–	[297]
Czech Republic	1984–86	No	35	3392	SFDT	[298]
	1984–86	No	25	3392	CFT	[298]
	< 1999	No	29*	191	CFT	[299]
Denmark	1990	No	27	5402	ELISA	[300]
	1992–96	No	28	89873	ELISA	[232]

Country	Year of sampling ^a	BOH ^b	Seroprevalence (%) ^c	Number of samples tested (n)	Method ^d	Reference
Egypt	< 1990	Yes	72	200	SFDT	[301]
	< 1990	Yes	59	200	IFAT	[301]
	< 1991	Yes	28	72	IFAT	[302]
	< 1993	Yes	65	100	ELISA	[303]
	< 1995	Yes	42*	62	ELISA	[304]
	< 1990	No	38	100	SFDT	[301]
	< 1990	No	32	100	IFAT	[301]
	< 1991	No	12	34	IFAT	[302]
	< 1992	No	31	70	IFAT	[305]
	< 1993	No	27	600	IHAT	[306]
	< 1993	No	6	100	ELISA	[303]
	1992–93	No	43	150	IHAT	[307]
Ethiopia	< 1994	No	20	94	ELISA	[308]
Finland	1988–89	No	20	16733	<i>e</i>	[309]
France	1993–94	No	58	987	–	[310]
	1995	No	54	13459	–	[65]
Gabon	1995–97	No	71	767	LAT	[311]
Germany	1987–90	No	73	4355	ELISA	[62]
	1989–90	No	42	2104	DAT	[312]
	< 1992	No	39	5670	ISAGA	[313]
	1991–95	No	30	1242	ELISA	[314]
Greece	< 1996	No	37	914	ELISA	[315]
India	1986–91	Yes	8	2075	IFAT	[316]
	1990	Yes	22	100	IHAT	[317]
Iraq	< 1997	Yes	8	540	LAT	[318]
	1994–95	Yes	19	81	IHAT	[319]
	1994–95	No	6	119	IHAT	[319]
Israel	1988–89	No	21	213	IFAT	[320]
Italy	< 1990	No	73	691	DAT	[321]
	1987–91	No	49	19432	ELISA	[322]
	1992–93	No	60	1800	ISAGA	[323]

Country	Year of sampling ^a	BOH ^b	Seroprevalence (%) ^c	Number of samples tested (n)	Method ^d	Reference
	1993	No	40	3518	–	[324]
	1993–94	No	18	2295	ELFA	[325]
	1992–97	No	23	9029	ELISA	[326]
Jamaica	1986	No	57	1604	ELISA	[327]
Korea	1990	No	7	618	IFAT	[328]
	1990	No	7	618	ELISA	[328]
	1993–94	No	4	899	ELISA	[329]
	1993–94	No	< 1	899	LAT	[329]
Libya	< 1991	No	47	369	IHAT	[330]
Madagascar	1992	No	84	599	ELISA	[331]
Mexico	< 1995	Yes	35	350	ELISA	[332]
Nepal	1995–96	No	55	345	LAT	[333]
	1995–96	No	55	345	ELISA	[333]
Nigeria	< 1990	No	40	834	DAT	[334]
	< 1990	No	39	834	IFAT	[334]
	< 1992	No	78	352	SFDT	[335]
Norway	1992–93	No	11	35940	ELISA	[336]
Pakistan	< 1996	Yes	17	240	IFAT	[337]
	< 1997	Yes	33	105	ELISA	[338]
Papua New Guinea	1989–90	No	18	197	DAT	[339]
Poland	1991–92	No	59	3734	DAT	[340]
Saudi Arabia	< 1991	Yes	100	219	IHAT	[341]
	< 1991	No	32	921	IHAT	[342]
Senegal	< 1990	No	33	60	LAT	[343]
	1993	No	40	353	ELISA	[344]
	1993	No	40	720	IFAT	[345]
Slovenia	1989–91	No	51	3959	SFDT	[346]
Spain	< 1991	No	39	1221	DAT	[347]
	1991–93	No	13	299	IFAT	[348]
	1991–93	No	30	6454	ELISA	[349]
	1994–95	No	42	109	ELISA	[350]

Country	Year of sampling ^a	BOH ^b	Seroprevalence (%) ^c	Number of samples tested (n)	Method ^d	Reference
Sweden	1992–93	No	14	3094	DAT	[351]
Switzerland	1990–91	No	46	9059	ELISA	[352]
Tanzania	1989–91	No	35	549	SFDT	[353]
Thailand	< 1991	No	13	690	LAT	[354]
	1996	No	13	1181	SFDT	[355]
Togo	< 1991	No	75	620	ELISA	[356]
Trinidad	1991–92	No	43	300	ELISA	[357]
Tunisia	1988–91	No	64	3288	IFAT	[358]
	1991–93	No	57	809	IFAT	[359]
	1994–96	No	43	2231	ELISA	[360]
Turkey	< 1993	Yes	47	1160	IFAT	[361]
	< 1993	Yes	47	1146	ELISA	[361]
	< 1995	Yes	77	314	IHAT	[362]
	< 1995	Yes	35	100	IFAT	[363]
	< 1996	Yes	82*	140	ELISA	[364]
	< 1996	Yes	38	954	ELISA	[365]
	< 1997	Yes	63	314	ELISA	[366]
	< 1993	No	27	187	ELISA	[367]
	< 1993	No	19	187	SFDT	[367]
	1991–95	No	55	2287	ELISA	[368]
	1992–95	No	40	996	ELISA	[369]
	< 1995	No	62	100	IFAT	[363]
	< 1995	No	47	152	IHAT	[362]
	< 1995	No	32	150	–	[370]
	1995–96	No	43	258	–	[371]
	< 1996	No	81	72	ELISA	[364]
	< 1996	No	80*	420	ELISA	[372]
	< 1996	No	71*	420	IHAT	[372]
	< 1996	No	34	324	–	[373]
	< 1998	No	61	326	ELISA	[374]
	< 1999	No	85	86	IFAT	[375]

Country	Year of sampling ^a	BOH ^b	Seroprevalence (%) ^c	Number of samples tested (n)	Method ^d	Reference
United Arab Emirates	< 1997	No	23	1503	ELISA	[376]
United Kingdom						
Sheffield	1989–92	No	10	1621	LAT	[377]
East England	1992	No	8	13000	ELISA	[378]
Wales	< 1992	No	22	192	SFDT	[335]
Venezuela	1976–92	No	54	7696	IHAT	[379]
Yugoslavia	1988–91	No	77	1157	SFDT	[380]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bBOH, women with bad obstetric history.

^cSeroprevalences marked with ‘*’ were calculated from the published data.

^dCFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; ELFA, enzyme-linked fluorescent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; ISAGA, immunosorbent agglutination assay; LAT, latex agglutination test; MEIA, microparticle capture enzyme immunoassay; SFDT, Sabin–Feldman dye test; –, not reported.

^eData were derived from screening programs using various diagnostic methods.

Table 4

Incidence of *T. gondii* infection in women of childbearing age (1990–2000)

Country	Year of sampling ^a	Incidence per 1000 pregnancies ^b	Incidence per 1000 susceptible mothers ^b	Number of pregnant women tested (n) ^b	Prevalence (%)	Reference
Argentina	1992	7	–	–	40	[381]
Australia	1986–89	1.08*	1.6	10207	35	[137]
Austria	1989–91	0.08	–	–	37	[196]
Colombia	1991–92	3.75–15	10–40	937	60	[67]
Czech Republic	1982–94	2.2	3.70*	50023	40	[382]
Denmark	1990	0.44*	0.61	5402	27	[299]
	1992–96	1.5	2.1	89873	28	[232]
Finland	1988–89	1.49*	2.4	16733	20	[308,383]
Germany	1987–90	2.53	9.28*	4355	73	[62]
	1990	4.9–6.1	–	126733*	–	[384]
Greece	< 1996	6	–	914	37	[314]
Israel	1988–89	14	20*	213	21	[319]
Norway	1992–94	1.31*	1.47	35940	11	[385]
Slovenia	1991–94	4.73*	7.5	8254	37	[386]
Spain	< 1996	0.56	1.3	3580	57	[387]
Sweden	1992–93	1.29*	1.51*	3094	14	[350]
United Arab Emirates	< 1997	31	41*	1503	23	[375]
United Kingdom	1989–92	0.62*	0.68*	1621	10	[376]
	1992	3.97*	4–6	13328	8	[377]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bFigures marked with ‘*’ were calculated from the published data. –, not reported.

Table 5

Incidence of prenatal infection with *T. gondii* in human neonates (1990–2000)

Country	Year of sampling ^a	Incidence per 1000 births ^b	Incidence per 1000 births to non-immune mothers ^b	Number of samples tested (n) ^b	Prevalence of infection in mothers (%)	Reference
Australia	1986–89	0.16*	0.23	18908	32	[137]
Austria	1991	<0.10	–	–	37	[196]
Denmark	1992–96	0.30	0.42	89873	28	[232]
Germany	1990	1.1	–	126733*	–	[384]
Guatemala	1987	10.9	20*	550	44	[388]
Norway	1992–94	0.31*	0.34*	35940	11	[385]
Poland	1996–98	0.55	1.33	27516	59	[199]
Switzerland	1986–90	0.73	–	15000	–	[389]
	1991–94	0.33	–	15000	–	[389]
United Arab Emirates	< 1997	12	16	1503	23	[375]
United Kingdom	1992	0.3–1.6	–	13328	8	[377]
USA	1986–91	0.08*	–	530000	–	[233]
	1986–92	0.08*	–	635000	–	[201]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bFigures marked with ‘*’ were calculated from the published data. –, not reported.

Table 6Seroprevalences of *T. gondii* infection in cattle and buffaloes (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Cattle</i>					
Argentina	< 1990	39	249	IHAT	[390]
Bangladesh	< 1993	16	205	LAT	[391]
Brazil	< 1994	32	334	IFAT	[392]
	1996	1	194	LAT	[393]
	< 1999	26	400	IFAT	[394]
China	< 1990	4	90	IHAT	[151]
	< 1991	1	208	IHAT	[395]
Costa Rica	1991	34	601	IFAT	[396]
Czech Republic	1979–90	4	1926	SFDT	[397]
	1979–90	2	1238	CFT	[397]
	1981–90	22	218	SFDT	[397]
	1981–90	3	176	CFT	[397]
Djibouti	< 1994	3	499	IHAT	[398]
Egypt	< 1990	21	19	IHAT	[399]
	< 1997	49	39	IHAT	[400]
	< 1997	49	39	IFAT	[400]
France	< 1997	69	364	IFAT	[401]
Greece	< 1992	40	1890	CFT	[402]
India	< 1991	43	102	DAT	[403]
	< 1992	9	32	LAT	[404]
Iran	1984–88	15	142	LAT	[405]
	< 1996	0	2000	LAT	[406]
	< 1996	0	2000	IHAT	[406]
Iraq	1989–90	48	204	CFT	[407]
Israel	1985–90	15	172	IFAT	[408]
Italy	< 1993	92	255	DAT	[409]
Malaysia	< 1990	0	132	IHAT	[410]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Mexico	1990–91	28	300	SFDT	[411]
	< 1993	12	397	ELISA	[412]
Netherlands	< 1995	13–43**	6976*	ELISA	[122]
Norway	1989	5	1053	ELISA	[145]
Pakistan	1993	25	100	LAT	[413]
Portugal	1988–90	43	60	DAT	[414]
Reunion	1987	54	780	ELISA	[415]
Saudi Arabia	< 2000	2	60	IHAT	[416]
Spain	< 1991	41	304	MAT	[417]
	< 1991	40	304	IFAT	[417]
Switzerland	1994	14	148	ELISA	[418]
Thailand	1996–97	3	119	LAT	[419]
Trinidad	< 1996	27	55	DAT	[420]
Turkey	< 1994	9	272	IHAT	[421]
	< 1995	4	280	ELISA	[422]
	< 1995	5	280	IHAT	[422]
	< 1997	63*	203*	SFDT	[423]
	1997–98	66	106	SFDT	[424]
Vietnam	1995	11	200	DAT	[425]
<i>Buffaloes</i>					
Brazil	1996	4	104	LAT	[393]
China	< 1990	0	83	IHAT	[151]
Egypt	< 1990	20	15	IHAT	[399]
	< 1998	0	75	DAT	[426]
India	< 1992	10	48	LAT	[404]
Iran	1995–96	9	385	IFAT	[427]
Vietnam	1995	3	200	DAT	[425]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bFigures marked with '**' were calculated from the published data. Seroprevalences marked with '***' varied with the herd examined.

^c CFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDI, Sabin–Feldman dye test.

Changes in seroprevalences of *T. gondii* infection in fattening pigs and sows between 1960 and 2000 in selected European countries now using intensive farm management

Table 7

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n)	Method ^c	Reference
<i>Fattening pigs</i>					
Austria	< 1975	32	100	SFDT	[428]
	1982	12	2238	IFAT	[429]
	< 1990	4	2755	CFT	[430]
	1992	< 1	2300	IFAT	[429]
Germany	1962–64	12–97**	500	SFDT	[431]
	1974	9	1366	SFDT	[432]
	1980	16	834	IFAT, IHAT	[433]
	1993–95	< 1	60	ELISA	[434]
Netherlands	< 1969	54	50	SFDT	[435]
	< 1982	0	196	ELISA	[436]
	< 1991	2	23348	ELISA	[437]
	< 1995	< 2	994	ELISA	[122]
<i>Sows</i>					
Austria	< 1990	3	1162	CFT	[430]
	1992	4	46	IFAT	[429]
Germany	< 1982	32	95	IFAT, IHAT	[433]
	1993–95	8	90	ELISA	[434]
	1997–99	18	2000	ELISA	[438]
Netherlands	< 1969	86	50	SFDT	[435]
	< 1982	11	36	ELISA	[436]
	< 1995	31	1009	ELISA	[122]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’.

^bSeroprevalences marked with ‘***’ varied with the cut-off titre used in the SFDT.

^cCFT, complement fixation test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; SFDT, Sabin–Feldman dye test.

Table 8

Seroprevalences of *T. gondii* infection in pigs (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Fattening/slaughter pigs</i>					
Argentina	< 1998	43	388	IHAT	[439]
	< 1998	43	388	IFAT	[439]
Austria	< 1990	4	2755	CFT	[430]
	1992	< 1	2300	IFAT	[429]
Brazil	< 1992	90	198	IFAT	[440]
	< 1997	7–54**	792*	IFAT	[441]
	< 1998	9–61**	792*	ELISA	[442]
Canada	1990	9	1443	MAT	[443]
	1991–92	9*	2800	LAT	[444]
Chile	1984	30	1474	IHAT	[445]
	1984	28	1474	SFDT	[445]
Czech Republic	1979–90	6	2616	SFDT	[446]
	1979–90	< 1	1179	CFT	[446]
	1981–90	32	287	SFDT	[447]
	1981–90	11	215	CFT	[447]
	1988–90	35	57	SFDT	[446]
	1988–90	14	57	CFT	[446]
Finland	1984	3	1847	ELISA	[448]
Germany	1993–95	0	60	ELISA	[434]
Italy	< 1991	64	90	IFAT	[449]
Japan	1992–93	3	423	LAT	[450]
Mexico	< 1993	9	1203	ELISA	[412]
Netherlands	< 1991	2	23348	ELISA	[437]
	< 1995	2	994	ELISA	[122]
Norway	1993–94	3	1605	ELISA	[145]
Poland	< 1991	36	925	ELISA	[147]
Portugal	1988–90	5	300	DAT	[414]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (<i>n</i>) ^b	Method ^c	Reference
Trinidad	1992–95	6	55	CAT	[420]
USA					
Illinois	1992	3	1885	MAT	[451]
Illinois	1992–93	21	4252	MAT	[452]
Iowa	< 1990	5	2029	ELISA	[453]
Iowa	< 1995	22	1000	MAT	[454]
N Carolina	1994–95	< 1*	2312	MAT	[455]
N Carolina	< 1998	1	3990	MAT	[456]
Tennessee	1991–92	3	437	MAT	[455]
Zimbabwe	< 1992	1	211	LAT	[457]
	< 1992	0	211	ELISA	[457]
	1995	9	97	MAT	[458]
Sows					
Austria	< 1990	3	1162	CFT	[430]
	1992	4	46	IFAT	[429]
Germany	1993–95	8	90	ELISA	[434]
	1997–99	18	2000	ELISA	[438]
Japan	1992–93	13	141	LAT	[450]
Netherlands	< 1995	31	1009	ELISA	[122]
USA					
17 states	1990	20	3479	MAT	[455]
Illinois	1992	21	5080	MAT	[451]
Illinois	1992–93	15	2617	MAT	[452]
Iowa	< 1990	10	587	ELISA	[453]
Iowa	< 1992	14	273	MAT	[459]
Tennessee	1991–92	29*	3841	MAT	[460]
Zimbabwe	< 1992	10	100	ELISA	[457]
<i>Not classified</i>					
Brazil	< 1990	38	1131	IFAT	[461]
	< 1995	100	200	IHAT	[462]
	< 1999	24	267	IFAT	[394]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
China	< 1990	10	816	IHAT	[151]
	< 1991	20	525	IHAT	[395]
Costa Rica	1991	44	496	IFAT	[396]
Czech Republic	1981–90	31	230	SFDT	[446]
	1981–90	10	158	CFT	[446]
Ghana	1997–98	39	641	ELISA	[463]
Malaysia	< 1990	16	122	IHAT	[410]
Poland	< 1991	36	925	ELISA	[147]
Taiwan	1978–88	28	3880	LAT	[464]
USA					
Hawaii	< 1992	49	509	DAT	[465]
New England	< 1999	47	1897	MAT	[466]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bFigures marked with '*' were calculated from the published data. Seroprevalences marked with '**' varied with the herd examined.

^cCAT, card agglutination test; CFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDT, Sabin–Feldman dye test.

Table 9

Seroprevalences of *T. gondii* infection in domestic and wild fowl (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Chickens</i>					
Brazil	< 2000	10	155	IFAT	[467]
China	< 1995	3*	109	–	[468]
Czech Republic	1981–90	1–5**	4458*	SFDT	[469]
India	< 1998	40	185	MAT	[470]
Iran	< 1990	33*	101	IHAT	[471]
Japan	1995	6*	50	LAT	[472]
Malaysia	< 1990	17	48	IHAT	[410]
Pakistan	1993	0	64	LAT	[413]
Turkey	1995–96	2	140	SFDT	[473]
<i>Ducks</i>					
China	< 1995	4*	82	–	[468]
Czech Republic	1981–90	2	297	SFDT	[469]
Iran	< 1990	0	8	IHAT	[471]
Turkey	1995–96	0	55	SFDT	[473]
<i>Geese</i>					
Czech Republic	1981–90	16	32	SFDT	[469]
Iran	< 1990	50*	8	IHAT	[471]
Turkey	1995–96	4	45	SFDT	[473]
<i>Pigeons</i>					
Iran	< 1990	33*	12	IHAT	[471]
Turkey	1996–97	0	60	SFDT	[474]
USA (New Jersey)	1986–87	6*	34	MAT	[152]
<i>Turkeys</i>					
Iran	< 1990	24*	25	IHAT	[471]
Turkey	1995–96	0	60	SFDT	[473]
<i>Wild turkeys</i>					
USA (Alabama)	< 1994	71	17	MAT	[475]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
USA (West Virginia)	1993	10	130	–	[153]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bFigures marked with ‘*’ were calculated from the published data. Seroprevalences marked with ‘***’ varied with the herd examined.

^cIFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDT, Sabin–Feldman dye test; –, not reported.

Table 10

Seroprevalences of *T. gondii* infection in sheep (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Lambs</i>					
Zimbabwe	< 1992	6	107	IFAT	[457]
	< 1992	3	107	ELISA	[457]
<i>Slaughter sheep</i>					
Djibouti	< 1994	10	486	IHAT	[398]
Egypt	< 1990	29	17	IHAT	[399]
Indonesia	< 1998	60	123	IHAT	[476]
Iran	1984–88	14	138	LAT	[405]
Norway	1993	18	2070	ELISA	[145]
	1993	16	1940	ELISA	[146]
Pakistan	1993	3	40	LAT	[413]
Saudi Arabia	< 1997	39	100	IHAT	[477]
Trinidad	< 1996	36	14	CAT	[420]
Turkey	1993–94	37	712	SFDT	[478]
USA (North East)	< 1990	59	654	ELISA	[479]
<i>Farmed sheep</i>					
Austria	< 1991	72	531	CFT	[480]
	< 1996	66	4079	IFAT	[114]
Bangladesh	< 1993	64	56	LAT	[481]
Brazil	< 1999	52	228	IFAT	[394]
Cameroon	< 1994	32	211	LAT	[482]
Canada	1988	58	3872	ELISA	[483]
Chile	< 1999	28	408	IFAT	[484]
	< 1999	12	408	IHAT	[484]
China	< 1991	7	202	IHAT	[395]
Croatia	< 1994	4	95	DAT	[485]
Czech Republic	1982–89	55	886	SFDT	[486]
	1982–89	40	484	CFT	[486]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (<i>n</i>) ^b	Method ^c	Reference
	1986–90	46–74**	661 *	SFDT	[486]
	1986–90	13–23**	650 *	CFT	[486]
France	< 1997	92	642	IFAT	[401]
Germany	1993–95	33	1122	ELISA	[434]
	< 1997	21	151	IFAT	[487]
Greece (Crete)	< 1995	23	8700	ELISA	[488]
India	< 1993	23	88	DAT	[489]
Ireland	< 1990	56	837	IHAT	[490]
Israel	1985–90	25	372	IFAT	[408]
Jordan	1989–90	21	176	LAT	[491]
	< 1993	21	559	LAT	[492]
Malaysia	< 1990	23	106	IHAT	[410]
Mexico	< 1990	30	495	IFAT	[493]
Niger	< 1991	14	70	LAT	[494]
Nigeria	< 1993	12	206	LAT	[495]
Slovakia	1988–91	10	1939	CFT	[496]
Spain	< 1991	40	550	DAT	[497]
	< 1991	35	550	IFAT	[497]
	1992–93	12	541	MAT	[498]
	< 1996	38	3212	DAT	[499]
	< 1996	35	2306	MAT	[500]
	< 1996	34	2306	IFAT	[500]
Suriname	1994	67	106	MAT	[501]
Sweden	< 1992	19	704	ELISA	[502]
Turkey	1990–92	26	259	IHAT	[422]
	1990–92	22	259	ELISA	[422]
	< 1992	23–31**	295 *	IHAT	[503]
	< 1997	89	62	SFDT	[504]
	< 1997	40	531	SFDT	[505]
	1997–98	34	154	SFDT	[424]
United Kingdom	1990	29	202	LAT	[506]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Uruguay	1991	14–29**	573*	LAT	[507]
	1992	28	422	DAT	[508]
	1992–94	39	1613	DAT	[509]
Zimbabwe	< 1992	9	109	ELISA	[457]
<i>Unclassified</i>					
Bangladesh	< 1993	18	17	LAT	[391]
Benin	< 1996	0	21	IHAT	[510]
Brazil	< 1995	48	370	IFAT	[511]
	1996	19	240	IFAT	[393]
Burkina Faso	< 1996	23	65	IHAT	[510]
China	< 1996	29	56	IHAT	[512]
Côte d'Ivoire	< 1996	68	62	IHAT	[510]
Djibouti	< 1996	13	183	IHAT	[510]
Ethiopia	< 1996	26	94	IHAT	[510]
Ghana	1997–98	33	732	ELISA	[513]
Iran	< 1996	25	1102	IHAT	[406]
	< 1996	24	2209	LAT	[406]
Mexico	1988	38	702	IFAT	[514]
Niger	< 1996	20	77	IHAT	[510]
Saudi Arabia	< 2000	3	150	IHAT	[416]
Senegal	< 1993	55	190	ELISA	[515]
	< 1993	46	190	IFAT	[515]
	< 1996	12	52	IHAT	[510]
Turkey	1994	72	414	IFAT	[516]
	1994	69	414	SFDT	[516]
	1994	37	414	LAT	[516]
	1994–95	15	1050	LAT	[517]

^a Years of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^b Figures marked with '*' were calculated from the published data. Seroprevalences marked with '**' varied with the herd examined.

^c CAT, card agglutination test; CFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFT, Sabin–Feldman dye test.

Table 11

Seroprevalences of *T. gondii* infection in goats (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Kids</i>					
Jordan	1989–90	19	69	LAT	[491]
<i>Slaughter goats</i>					
Bangladesh	1994–95	13	528	LAT	[518]
Djibouti	<1994	6	554	IHAT	[398]
	<1996	21	176	IHAT	[510]
Egypt	<1990	29	14	IHAT	[399]
Indonesia	<1998	40	38	IHAT	[476]
Iran	1984–88	13	130	LAT	[405]
Pakistan	1993	0	58	LAT	[413]
Saudi Arabia	<1997	28	100	IHAT	[477]
Zimbabwe	<1992	5	156	ELISA	[457]
<i>Farmed goats</i>					
Austria	<1996	69	687	IFAT	[114]
Bangladesh	<1993	54	33	LAT	[481]
	<1993	12	306	LAT	[391]
Botswana	1994–96	10	345	IHAT	[519]
Brazil	1993	16	202	IFAT	[520]
	<1994	31	153	IFAT	[521]
Croatia	1992	4–14**	179*	MAT	[522]
Czech Republic	1981–90	61	54	SFDT	[486]
	1981–90	21	54	CFT	[486]
Djibouti	<1996	31	35	IHAT	[510]
Ethiopia	<1996	20	133	IHAT	[510]
Germany	1993–95	42	69	ELISA	[434]
	<1997	19	829	IFAT	[487]
Greece (Crete)	<1995	14	2320	ELISA	[488]
Jordan	<1993	17	305	LAT	[492]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (<i>n</i>) ^b	Method ^c	Reference
Malaysia	1991–92	35	400	MAT	[523]
	< 1996	18	107	IHAT	[410]
Mexico	< 1993	3	707	ELISA	[412]
New Zealand	< 1991	35	185	IFAT	[524]
	< 1991	32	185	LAT	[524]
Netherlands	< 1998	47	189	DAT	[525]
Nigeria	< 1993	5	248	LAT	[495]
Reunion	1987	75	395	ELISA	[415]
Senegal	< 1996	4	144	IHAT	[510]
Spain (Grand Canary Island)	< 1995	63	1052	ELISA	[526]
Turkey	1990–92	15	66	IHAT	[422]
	1990–92	12	66	ELISA	[422]
	< 1997	63	38	SFDT	[504]
	1997	44	98	SFDT	[527]
USA	< 1990	65	99	MAT	[528]
	< 1990	55	99	IHAT	[528]
Venezuela	< 1998	6	438	IHAT	[529]
<i>Unclassified</i>					
Brazil	1996	29	439	LAT	[393]
China	< 1996	26	1028	IHAT	[512]
Czech Republic	1994	20	247	CFT	[530]
	1994–96	30*	202	CFT	[531]
	1996	60–66**	159*	IFAT	[531]
	1994–97	45*	203	CFT	[531]
Egypt	< 1997	51	78	IFAT	[400]
	< 1997	49	78	IHAT	[400]
France	< 1997	0–77**	765	ELISA	[532]
Ghana	1997–98	27	526	ELISA	[513]
India	< 1993	68	95	DAT	[489]
Iran	< 1993	20	530	LAT	[406]
Mexico	< 1990	44	211	IFAT	[493]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Saudi Arabia	< 2000	4	56	IHAT	[416]
Sri Lanka	1989	22	139	MAT	[533]
Turkey	1996	54	68	SFDT	[534]
Uganda	1996	31	784	ELISA	[535]
USA	1982–84	22	1000	MAT	[536]

^a Years of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^b Figures marked with ‘*’ were calculated from the published data. Seroprevalences marked with ‘***’ varied with the herd examined.

^c CFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDT, Sabin–Feldman dye test.

Table 12

Seroprevalences of *T. gondii* infection in horses (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n)	Method ^c	Reference
Argentina	< 1990	20	20	IHAT	[390]
	1986–98	13	76	IFAT	[537]
Brazil	1994–96	32	561	IFAT	[538]
	< 1997	8*	430	SFDT	[539]
	< 1999	16	101	MAT	[540]
	< 1999	12	173	IFAT	[394]
China	< 1991	2	132	IHAT	[395]
Czech Republic	1987	8	2886	SFDT	[541]
Sweden	1986–87	< 1	219	ELISA	[542]
Turkey	1995	2	103	SFDT	[543]
	< 1997	8*	60	SFDT	[544]
	< 1998	8	194	SFDT	[545]
	< 1998	6	194	LAT	[545]
	< 1998	2	50	SFDT	[546]
USA	1998	16	339	SFDT	[547]
	1998	7	1788	MAT	[547]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bSeroprevalences marked with '*' were calculated from the published data.

^cELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDT, Sabin–Feldman dye test.

Table 13

Seroprevalences of *T. gondii* infection in rabbits (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%)	Number of samples tested (n)	Method ^b	Reference
Chile	< 1990	13	143	IHAT	[548]
China	< 1990	8	12	IHAT	[151]
Czech Republic	1981–86	53	366	SFDT	[549]
Egypt	< 1991	20	100	CIA	[550]
France	< 1990	6	187	IFAT	[551]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^bCIA, carbon immunoassay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; SFDT, Sabin–Feldman dye test.

Table 14

Seroprevalences of *T. gondii* infection in dogs (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Argentina	1988–94	60	232	IFAT	[552]
Brazil	1988–90	47	243	IFAT	[553]
	1994	53	218	IHAT	[554]
	< 1998	63	276	ELISA	[555]
	< 1998	55	327	IFAT	[556]
	< 1998	46	276	IFAT	[555]
	< 1999	84	189	IFAT	[557]
Chile	< 1991	12	178	IHAT	[558]
China	< 1997	5	101	ELISA	[559]
Czech Republic	1982–84	33–39**	1393*	SFDT	[560]
	1982–84	12–15**	1393*	CFT	[560]
France	< 1998	39	3580	IFAT	[561]
Iran	< 1993	31	100	SFDT	[562]
Israel	< 1996	36	220	IFAT	[563]
Italy	1996	17	104	IFAT	[564]
Pakistan	< 1992	17*	12	LAT	[565]
Spain	< 1997	47	97	IFAT	[566]
Sweden	< 1994	30	398	DAT	[567]
Taiwan	1995–96	25	289	LAT	[568]
	1995–96	8	658	ELISA	[569]
	< 1998	20	105	LAT	[570]
	1997	20	51	LAT	[571]
Turkey	< 1996	85	52	IFAT	[572]
	< 1996	79	52	SFDT	[572]
	< 1996	69	70	SFDT	[573]
	< 1996	48	52	LAT	[572]
	< 1997	72	50	SFDT	[574]
	< 1997	46	50	LAT	[574]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
USA (Kansas)	< 1998	75*	53	SFDT	[575]
	< 1990	25	229	DAT	[576]

^a Years of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^b Figures marked with '*' were calculated from the published data. Seroprevalences marked with '**' varied with the herd examined.

^c CFT, complement fixation test; DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; SFDT, Sabin–Feldman dye test.

Table 15

Seroprevalences of *T. gondii* infection in game and wild animals (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Wild boars</i>					
Austria	1990–93	19	269	IFAT	[577]
Czech Republic	1981–90	15	124	SFDT	[154]
Germany	1993–94	25	130	IFAT	[578]
	1997	21	81	IFAT	[579]
	1997	19	81	ELISA	[579]
	1997	15	81	SFDT	[579]
Japan	< 1999	6	108	LAT	[580]
<i>USA</i>					
South Carolina	1993	37	149	MAT	[581]
Tennessee	1990	31	108	MAT	[581]
Zimbabwe	< 1999	0	3	MAT	[458]
<i>Deer</i>					
Brazil	1990	27	66	IHAT	[582]
	1995	12	41	IFAT	[582]
	1995	12	41	ELISA	[582]
Czech Republic	1981–90	14–100**	401	SFDT	[154]
<i>USA</i>					
Alabama	< 1991	44	16	DAT	[583]
California	1987–91	7	276	LAT	[584]
Kansas	1989–93	44	106	MAT	[585]
Minnesota	1990–93	30	1367	MAT	[586]
Ohio	1996–98	44	147	MAT	[587]
Pennsylvania	1991	60	593	MAT	[588]
<i>Moose</i>					
Canada	< 1990	15	125	IHAT	[589]
<i>Reindeer</i>					
Finland	1993–96	0–25**	900*	DAT	[590]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Norway	1993–94	< 1	1 677*	DAT	[590]
<i>Gazelles and antelopes</i>					
Saudi Arabia	1990–91	0–6**	608*	IHAT	[591]
Zimbabwe	< 1999	12–37**	86*	MAT	[458]
<i>Bears</i>					
USA					
Alaska	1988–91	15–18**	520	LAT	[592]
Florida	1993–95	56	66	LAT	[593]
N Carolina	1996–97	84	143	MAT	[594]
Pennsylvania	1989–92	80	665	DAT	[595]
Pennsylvania	1992	80	322	MAT	[596]
Pennsylvania	1993	79	28	MAT	[597]
Pennsylvania	1993	75*	28	DAT	[597]
Pennsylvania	1993	32*	28	LAT	[597]
Pennsylvania	1993	21*	28	IHAT	[597]

^aYears of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by ‘<’.

^bFigures marked with ‘**’ were calculated from the published data. Seroprevalences marked with ‘***’ varied with the geographical area or the species examined.

^cDAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDI, Sabin–Feldman dye test.

Table 16

Seroprevalences of *T. gondii* infection in cats (1990–2000)

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
<i>Pet cats</i>					
Argentina	1993	20	169	IHAT	[598]
Brazil	1996–97	18	248	IFAT	[599]
Chile	< 1992	40	65	IHAT	[600]
France	< 1997	43	519	IFAT	[401]
	< 1998	43	506	IFAT	[561]
Germany	1989–90	46	231	IFAT	[601]
	1989–90	31–47**	218*	ELISA	[602]
	1989–90	34–51**	218*	IFAT	[602]
	1992–94	34–61**	465*	ELISA	[603]
Italy	1996	9	113	IFAT	[564]
Japan	1994–95	9	471	LAT	[604]
	< 1998	6	800	LAT	[605]
Mexico	< 1999	71	24	ELISA	[606]
Singapore	< 1992	7	15	LAT	[607]
Sweden	1986–87	42	241	ELISA	[542]
Taiwan	< 1990	8	117	ELISA	[608]
Turkey	1994	43	65	SFDT	[609]
USA (Oklahoma)	1987–88	22	618	LAT	[610]
<i>Stray or feral cats</i>					
Australia	< 1997	50	18	LAT	[611]
	< 1999	40	103	ELISA	[612]
Bangladesh	1995	33	24	LAT	[282]
Brazil	< 1999	73	173	IFAT	[557]
Germany	1989–90	66	61	IFAT	[601]
	1989–90	58	88	ELISA	[602]
	1989–90	56	88	IFAT	[602]
	1992–94	59	112	ELISA	[603]

Country	Year of sampling ^a	Seroprevalence (%) ^b	Number of samples tested (n) ^b	Method ^c	Reference
Italy	1998	66	259	ELISA	[613]
	< 1997	33	490	ELISA	[614]
	< 1997	33	490	DAT	[614]
Japan	1990–91	19	231	LAT	[615]
Korea	< 1999	13	198	ELISA	[131]
Singapore	< 1992	31	706	LAT	[607]
United Kingdom (Oxfordshire)	< 1996	62	45	IHAT	[616]
USA					
Illinois	1992–93	68	391	MAT	[452]
Iowa	< 1992	42	74	MAT	[459]
Turkey	< 1995	70	53	IHAT	[617]
<i>Not classified</i>					
Argentina	1988–94	61	145	IFAT	[552]
	< 1997	35	55	IFAT	[618]
Austria	< 1996	48	456	IFAT	[114]
Czech Republic	1995–97	59	390	IFAT	[619]
Japan	1989–91	11	726	LAT	[620]
Panama	1990–92	32–61**	141*	DAT	[155]
Poland	1992–96	71	357	DAT	[621]
Slovenia	< 1997	70	54	IFAT	[622]
	< 1997	59	54	ELISA	[622]
	< 1997	57	54	DAT	[622]
Turkey	< 1998	56	36	SFDT	[623]
	< 1998	38	24	SFDT	[624]
United Kingdom (Scotland)	< 1993	19*	158	–	[625]
USA	< 1992	74	124	ELISA	[626]

^a Years of sampling are listed as published in the references. In cases where this information was not available, the year listed here is the year when the study was published, as indicated by '<'. Data from the 1980s are included if the study was published in the 1990s and if no recent data were available for the area.

^b Figures marked with '*' were calculated from the published data. Seroprevalences marked with '**' varied with the type of keeping or the year of examination.

^c DAT, direct agglutination test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect immunofluorescent antibody test; IHAT, indirect haemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test; SFDT, Sabin–Feldman dye test; –, not reported.