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Review articles

The role of ticks in transmission cycle of *Toxoplasma gondii*

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ABSTRACT. Toxoplasmosis is globally distributed, water- and food borne zoonosis caused by the single protozoan *Toxoplasma gondii* and probably one-third of the world's human population is infected with this pathogen. Domestic and wild felids are definitive hosts of this pathogen and intermediate hosts for great variety of other homoeothermic animals. Human as other of the intermediate hosts may become infected in the main route of infection; it is the ingestion of parasite oocysts in contaminated water or soil and undercooked meat. However, the mechanism which this parasite uses to so large spread is not fully understood, because oral transmission does not explain the common event of this parasite in a variety of hosts, such as herbivorous animals or rodents and birds, as well as routes of spread to domestic hosts. Such a wide circle of hosts suggests a possibility of other paths of transmission and a role of ticks, the blood-seeking arthropods was considered in the transmission of *T. gondii*.

Key words: *Toxoplasma gondii*, genotypes, transmission ways, role of ticks

Introduction

The class Sporozoa (phylum Apicomplexa) is a large group of parasitic protozoa, comprising of organisms such as coccidia, gregarines, haemogregarines [1]. Among coccidia, there is *Toxoplasma gondii*, protozoan infecting all species of warm blooded vertebrates, including humans. In humans, the toxoplasmic infection is usually asymptomatic due to effective immune system control, however, severe complications, and even death might follow as a result of a congenital infection or in immunocompromised patients (e.g., AIDS, transplantation), what illustrates the importance of the immune system in controlling *T. gondii* infection [2]. Cases of congenital infection and human immunodeficiency virus (HIV) coinfection cause the most serious morbidity, resulting in severe neurologic and ocular diseases and in some cases lead even to abortion [3, 4]. In immunocompromised patients, the reactivation of the infection may generate additional complications including encephalitis [5]. The clinical manife - stations of toxoplasmosis are influenced, among other factors, by the parasite genotypes [6]. *T. gondii* may carry also a veterinary problem as a cause of illnesses in domestic animals, such as abortion and neonatal infections of sheep and goats [7].

Molecular methods used in epidemiological searches deliver effective tools to monitor and recognize the risk factors related with the spread of *T. gondii* in the environment, also determining the sources of infection and the genotypes involved [8,9]. Chen et al. [10] and many other authors notice that the complete knowledge about genetic diversity of *T. gondii* is crucial to better understanding the epidemiological patterns and pathogenicity, and to discover the new strategies for satisfactory diagnosis and treatment of toxoplasmosis, and for construction of vaccinations as well.

Historically, *T. gondii* was considered to be clonal with low genetic diversity and clustered into 3 genotypes: I, II, III [11]. However, later studies showed that recombinants and atypical genotypes also occur [12]. Up to now, 12 haplogroups, including the 3 initially described lineages, based on sequence analyses have been described [13–15]. These haplogroups are not totally homogenous, and more determined markers revealed subclustering that may be linked with geographical origins and phenotypic characteristics [16]. There is still lack of atypical and highly diverse isolates with many unique polymorphisms which cannot be clustered into one of these haplogroups [17]. In Europe, the population structure of *T. gondii* is evidently clonal, with majority of strains belonging to the type II

[16]. In North America, the population structure appears similar to the European one, with a predominance of type II strains, but current data shows a higher prevalence of atypical strains [15].

The virulence of this parasite was defined in laboratory mice [11,18] or in murine models [13]. Type I strains cause lethal infection in all strains of laboratory mice even at low inocula (lethal dose (LD100) approx. 1), whereas types II and III strains are much less virulent (median lethal dose (LD50) ≥105). All three main genotypes were isolated from both humans and animals, but the majority of human toxoplasmosis cases are caused by type II and III that was isolated more often from AIDS patients [6] than from patients with congenital toxoplasmosis. Type I was associated mainly with congenital toxoplasmosis but it was detected also in AIDS patients [11]. This genotype is connected also with ocular toxoplasmosis in immunocompetent adults [12]. Genotyping of *T. gondii* DNA isolates from Polish population of *Ixodes ricinus* revealed that detected strains represent genotype I [18,19]. *Toxoplasma* I genotype was identified in all nested PCR+ of *I. ricinus* with the use of primers complementary to B1 gene and amplified gene fragments were sequenced. Genotyping of *T. gondii* isolated from *I. ricinus* conducted by Sroka et al. [20] with the use of RFLP PCR based on B1 gene fragment identified I or II/III clonal type. Genotype I was found as dominant in the water bodies from eastern Poland examined by Sroka et al. [21] and as unique type from north-western part of the country by Adamska [22]. Estimated occurrence of *T. gondii* in northern Polish soil and vegetable samples and determining the genotype was conducted by Lass et al. [23,24] and showed type I and II in some isolates. Thus, there is higher genetic divergence among *T. gondii* strains isolated from environmental samples as well as from *I. ricinus* ticks in other part of Poland than in the north-western part of our country and it is hard to compare it because there is no data about studies on this type of samples from other parts of the world.

Epidemiology and transmission ways of *T. gondii*

The definitive hosts for *T. gondii* are members of the Felidae family [25] and the felids are the only animals capable to shed infective oocysts into the environment in their feces. Human is one of the intermediate hosts and may become infected in the main route of infection, during ingestion of parasite oocysts in contaminated water or food (meat mainly), and additionally through transplantation, or transplacentally from mother to fetus [25–27].

Recently, usage of the molecular methods in epidemiological searches allows monitoring and recognizing the risk factors related with the spread of *T. gondii* in the environment, especially in determining the sources of infection [8,9]. *T. gondii* is a tissue cyst-forming parasite with complex life cycle that includes sexual and asexual stages. Asexual replication occurs in a wide variety of intermediate host species and is characterized by two stages: the rapidly growing "tachyzoites", and the slowly dividing encysted "bradyzoites" [28,29]. The sexual phase development of *T. gondii* takes place in definitive hosts, which are felids. Oocysts containing highly infectious sporozoites are shed by infected felids, and for example, a single infected cat can shed millions of oocysts into the environment [30]. Sporulated oocysts become infective in the environment, and if digested can infect both intermediate hosts and other definitive hosts. After succeeding ingestion, sporozoites excyst from oocysts, enter the gut epithelium and transform into tachyzoites. Tachyzoites reproduce asexually and may colonise many host tissues, inducing immune response. Tachyzoites differentiate into bradyzoites, which produce tissue cysts, but they are resistant to the immune response. Bradyzoites may be transmitted to a definitive host, or another intermediate host, upon digestion of infected tissues. These hosts may also be infected via vertical transmission from infected mother to fetus [25,28]. Thus, the common horizontal transmission to humans is caused by the ingestion of *T. gondii* cysts in infected meat or by the ingestion of sporulated oocysts from water, soil or food contaminated directly from feline feces or, less frequently, directly from feline feces [31]. However, toxoplasmosis is one of the most prevalent parasitic infections worldwide, affecting at least one-third of human population and big part of meat-production animals [32]. This would indicate that a frequent contact with the definitive host (cats) is necessary to explain the very high prevalence found in many human and animal populations, and it is worth considering that cat can shed large number of oocysts into the environment once for approximately 2 weeks only [33].

Such a wide circle of hosts suggests a possibility of other paths of transmission except eating raw or

half-cooked meat with the oocysts or contaminated soil or water [34]. The possible role of arthropods in transmission of *T. gondii* was considered earlier [35] and Polish studies showed that *Ixodes ricinus* ticks may be involved in the spread of this protozoan [34,36]. In Europe, the tick *I. ricinus* is the main vector of numerous pathogens considered as agents of emerging human diseases. Among them there are viruses, bacteria and protozoa, but the most frequently described is *Borrelia burgdorferi* sensu lato, bacteria causing Lyme disease, and less frequently some species of protozoa.

Potential role of ticks for spreading of *Toxoplasma gondii*

Toxoplasmosis, caused by the protozoan *T. gondii*, is a worldwide parasitic zoonosis and as it was described above, domestic and wild felids are the definitive hosts of this pathogen and intermediate hosts for great variety of other homoeothermic animals. Herbivores obtain infection mostly by consuming oocysts with sporozoites that are in water or contaminated herbal food, but carnivores and omnivores, including human beings, can also become infected after ingesting meat with cysts (bradyzoites) or even pseudocysts (tachyzoites). Vertical transmission is an additional route, which may be rare [37]. Generally, all *T. gondii* risk factors for human infection are related to any contact with cat fecal material (water, food, soil) or contact with meat (in the case of working with meat or consuming improperly cooked meat) potentially infected with *T. gondii* [38]. However, studies of Boyer et al. [39] demonstrated that 31% of patients transmitting toxoplasmosis congenitally to their children indicated none of these common risk factors for *T. gondii* exposure. Although the exposure to cat feces is known as very significant basis of contact with oocysts, ownership of a pet cat was not a significant risk factor [39]. Additionally, studies of Berger-Schoch et al. [40] on prevalence of *T. gondii* in feline faeces (oocysts) and in meat from sheep, cattle and pigs in Switzerland showed only one of the cats shed *T. gondii* oocysts, what makes a *T. gondii* occurrence of 0.4% (n=252) of examined samples. In meat-producing animals, prevalence was also low in sheep and pigs (0.1-4.8%), but the highest prevalence was found in cattle (2.8-7.2%). Such low prevalence in a cat, the only definitive host for T. *gondii* makes one wonder how appears such a wide spread of this parasite.

The presence of *T. gondii* antibodies, for example in equine sera indicating contact with the parasite was showed in many countries in the world. Horses can become infected with *T. gondii* and in certain regions of the world up to 90% of the animals were shown to be seropositive [41]. In recent studies from Europe ratio is lower and for example in southern Italy, antibodies against *T. gondii* were detected in 3% of healthy horses [42] and in 13.3% of equids from Spain [43] what can testify about regional distribution but also about the fact that comparison of seroprevalence among studies is not possible because of using different methods. Above data and high human exposure (*T. gondii* infects around 30% of the global human population) [44], breeding and wild animals exposure to *T. gondii* with the estimated serological prevalence rates [16,45,46] indicate that there are more ways of infection with *T. gondii* and additional risk factors.

First time detection of *T.gondii* DNA in questing *I. ricinus* had place in Poland in 2003 by Sroka et al. [34] Results of those studies showed the hypothetical vector role of *I. ricinus* ticks for this protozoan and it was first suggestion concerning the alternative route of the infection than oral transmission. The later study of Sroka et al. [36] provided new information about the occurrence of *T. gondii* DNA in *I. ricinus* and confirmed the possibility of live parasite transmission from ticks to mice. The authors detected DNA of parasite (with analysis of B1 gene) in mice inoculated with homogenate of *I. ricinus* ticks collected from vegetation. Samples from brains and/or other organs of 60 inoculated mice showed 44 positive results for the presence of *T. gondii* DNA and sequence analysis confirmed the identity of the *T. gondii* DNA.

The results of the current Polish study also showed the presence of *T. gondii* DNA in *I. ricinus* and it was found both in feeding (on ponies) and questing (on vegetation) ticks [19]. This study showed that *T. gondii* DNA was detected in adults, nymphs and larvae in field-collected *I. ricinus* ticks and the total percentage was not low (10.2 %). The last paper about toxoplasmic molecular infection shows that *Dermacentor reticulatus,* tick species common in Europe should be also considered as a potential vector of *T. gondii* [47].

T. gondii asexually reproduces in intermediate hosts, in cells of many diverse organs and by

penetrating to monocytes and neutrophils it can move to other tissues of the host by blood and lymph and it can create possibility of transmission to other animals via blood-sucking arthropods. The presence of invasive *T. gondii* forms in blood suggests the possibility of human infection by transfusion [48]. Theoretically, almost each of intermediate host of *T. gondii* may serve as a source of blood for each stage of *I. ricinus,* blood-sucking ticks using many of wild and domestic animal species as hosts. In this way, ticks may contribute to the spread of *T. gondii* in the environment through the transmission of this parasite between links of the environmental food chains. These links being intermediate hosts, including small rodents and birds will get into a cat – a definitive host in which the life cycle of *T. gondii* is ended with the full sexual cycle in that host. In this way, a tick may increase infected populations among all intermediate host species, including humans and domestic animals. Thus, these biting arthropods may help to explain the wide spread of this parasite over large species of host animals and geographical areas.

The latest paper, derived from China evaluated the role of *Haemaphysalis longicornis*, Asiatic tick, in the epidemiology of toxoplasmosis [49]. *H. longicornis* is known in Asia and Australia as a tick transmitting diverse pathogenic species of *Theileria, Babesia* and *Rickettsia* genera. In this study Zhou et al. [49] showed a high infection rate of adult (11.26%) and nymph ticks (5.95%) from the field, what in opinion of authors indicates on the natural infection or survival of *T. gondii* in the *H. longicornis* ticks. The survival of the *T. gondii* infection in *H. longicornis* was examined with microinjection of a *T. gondii* and its survival for at last 15 days. It is an indication of parasite growing, suggest authors and it is in contrast with the presented theory that *T. gondii* infection is possible in warm-blooded hosts only [50]. To confirm tick infection of *T. gondii* by tick blood feeding, they evaluated existent *T. gondii* infection in adult *H. longicornis* developed from nymphs feeding on infected mice in laboratory condition. The obtained results demonstrate that *T. gondii* can be transmitted to ticks by ticks feeding on the blood of infected mammal host.

Guo et al. [31] and many other authors link the infection ratio of farm animals with environmental oocyst-contamination and direct contact with cats only. However, when we consider oral transmission as being the main route of infection [32,33], it is not clear in which way the common rate of *T. gondii* becomes for example in herbivorous animals [45,49]. However, Zhou et al. [49] suggested an alternative oral transmission (except traditional oral way). Their studies demonstrated that *T. gondii* infection can be transmitted to the other hosts through the ingestion of infected ticks (*Haemaphysalis longicornis*). The authors conclude that ingesting infected ticks as an oral route of transmission may explain the common occurrence of *T. gondii* in a variety of hosts, including mainly herbivorous. They demonstrated, in laboratory conditions the possibility that these species of ticks constitute a reservoir for transmission of toxoplasmosis and concluded that these tick species do not transmit *T. gondii* on other hosts, thus toxoplasmosis probably do not belong to tick-borne disease, and these tick species are not vectors but reservoir hosts for *T. gondii*.

Conclusions

I. ricinus as a blood-sucking tick uses many of wild and domestic animal species as hosts, it means that, almost each of intermediate host of *T. gondii* may serve as a source of blood for each tick stage. In this way, an infected tick may take part in the spread of *T. gondii* in the environment through the transmission of this parasite between links of the environmental food chain. These links being intermediate hosts, including small rodents and birds will get into a cat – a definitive host. In this way, ticks may increase infected populations among all intermediate host species, including humans and animals. Therefore, the life cycle of *T. gondii* should be completed with an arthropod vector, probably as a mechanical vector or even as biological vector, which means that it is infected for longer time and can be a source of infection to others. Second hypothesis tells, the tick does not transmit *T. gondii* to other host mammals, but takes infection from them after molting and increases populations of infected ticks.The mammalian hosts digest infected ticks and in this way *T. gondii* is transmitted, what explains herbivorous infection, rodents and birds infection. In this case, ticks do not transmit *T. gondii* to other hosts but serve as reservoir host for *T. gondii*, however it requires more studies. Thus, these biting arthropods may help to explain the wide spread of this parasite in almost all geographical areas and warm blooded animals, but there is need

to establish whether they are an important infection source for toxoplasmosis.

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