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Author for correspondence:
J. P. Dubey, E-mail: jitender.dubey@ars.usda.gov

A review of toxoplasmosis in humans and animals in Turkey

Z. Kolören[1,2] and J. P. Dubey[2]

[1]Faculty of Science and Literature, Department of Molecular Biology and Genetics, Ordu University, 52200, Ordu, Turkey and [2]United States Department of Agriculture, Agricultural Research Service, Animal Parasitic Diseases Laboratory, Building 1001, Beltsville, Maryland 20705-2350, USA

Abstract

Infections by the protozoan parasite *Toxoplasma gondii* are widely prevalent in humans and animals in Turkey but little is known of the burden of their clinical toxoplasmosis. Many early papers on toxoplasmosis in Turkey were published in Turkish and often not available widely. Here, we review prevalence, clinical spectrum, epidemiology and diagnosis of *T. gondii* in humans and animals in Turkey. This knowledge should be useful to biologists, public health workers, veterinarians and physicians. Although one-third of the human population in Turkey is seropositive, the rate of congenital toxoplasmosis is unknown and no information is available in children 12 years old or younger. One large outbreak of acute toxoplasmosis has been reported in 14–18-year old school children in Turkey. An alarming rate (36%) of *T. gondii* tissue cysts were reported in tissues of sheep and water buffalo meats destined for human consumption; these reports require verification. Genetically, *T. gondii* strains from domestic cats and wild birds in Turkey were generally classical type II and III, like those prevalent in Europe. A separate genotype, Type 1 Africa, was isolated from two congenitally infected children and a domestic cat in Turkey.

Introduction

Turkey is a geographically important link between Asia, Europe and Africa and is surrounded by seas (Fig. 1). It has a population of nearly 83 million. Much of the literature on toxoplasmosis in Turkey is in Turkish. Here, we review the available literature on *Toxoplasma gondii* infection in humans and animals in Turkey.

Methods for review

We consulted original manuscripts to minimize mistakes in translation. Detailed historical, serological, parasitological, clinical and genetic information on *T. gondii* infections in humans and other animals are summarized in tables throughout the review.

Most of the reports on toxoplasmosis relate to serological surveys with no uniformity with respect to tests used and cut-off values. In Table 1, information on serological assays used in Turkey is summarized and noted throughout the paper where applicable.

History

Although *T. gondii* was discovered in 1908, there was no information from Turkey until 1950 (Dubey, 2008). Professor Hayati Ekmen poineered research on toxoplasmosis in Turkey in 1970's. He first isolated viable *T. gondii* from a dog (Ekmen and Altıntaş, 1970) and from a child (Ekmen *et al.*, 1974). The report on the baby was unfortunately published only as an abstract (Döşkaya *et al.*, 2013). Toxoplasmosis was suspected in a child born to a mother from Ankara in 1972. The mother had been treated with anti-*T. gondii* therapy (sulfadiazine and pyrimethamine) during the last 2 months of pregnancy. Nothing was reported concerning the symptoms of toxoplasmosis in the newborn but the child must have had neurological signs because child's cerebrospinal fluid (CSF) was bioassayed in mice for the isolation of viable *T. gondii*. A virulent strain of *T. gondii* was recovered from the mice and designated the Ankara strain. This strain has been maintained since 1972 and used for molecular studies (Döşkaya *et al.*, 2013).

Another noteworthy publication is an outbreak of acute toxoplasmosis in school children in Turkey (Doganci *et al.*, 2006). In 2002, a boarding school in Izmir, Turkey, saw 171 (9.5%) of 1797 students, aged 14–18-year, develop mild flu-like illness. All students were examined physically, including ophthalmic testing. The symptoms were typical of acquired toxoplasmosis (cervical lymph adenopathy, fever, myalgia, headache and dizziness). Antibodies to *T. gondii* were found in all 171 students by means of several serological techniques; all were positive for IgM antibodies and 40 of 43 randomly selected students had low-avidity *T. gondii* antibodies. The IgM and low-avidity antibodies are indicative of recent infection. None of the students had ocular lesions. Epidemiological investigation revealed no common source. Near the dining hall, there was a sheltering place for large numbers of stray cats. However, the school

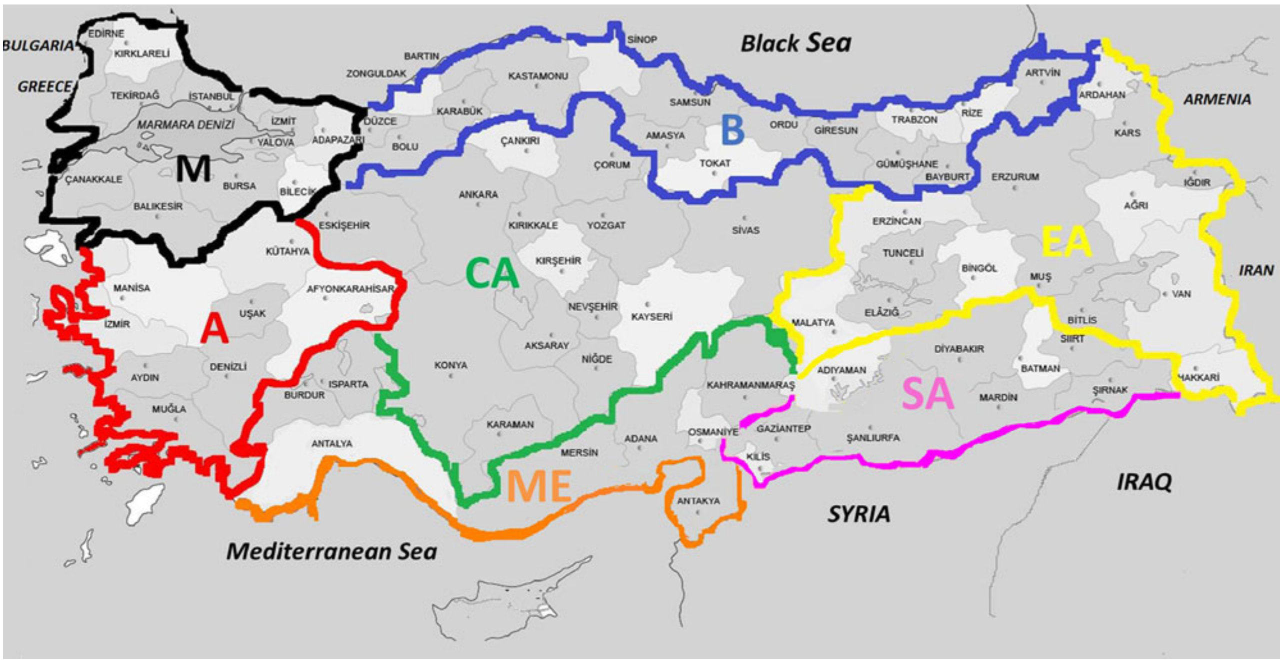

Fig. 1. Map of Turkey with seven regions and human population. Marmara region (M), Central Anatolia (CA), Aegean region (A), Mediterranean region (ME), Black Sea region (B), Eastern Anatolia (EA) and Southern anatolia (SA).

authorities removed the cats before they could be tested for *T. gondii*. This outbreak provided reference sera for acute toxoplasmosis for other investigations (Liang *et al*., 2011).

Toxoplasmosis in humans

Toxoplasma gondii infections are prevalent worldwide but are mostly asymptomatic. However, *T. gondii* can cause severe illness in humans, particularly in congenitally infected children, and those with suppressed immunity, and even immunocompetent persons have died of toxoplasmosis (Robert-Gangneux and Dardè, 2012; Torgerson and Mastroiacovo, 2013; Peyron *et al*., 2016).

Serological prevalence in the general population

The seropositivity of *T. gondii* antibodies in the general population, including 15–40 years old women and those pregnant is summarized in Tables 2 and 3, respectively; seroprevalence varied between 18–100%.

Most of these serological studies were retrospective and based on convenience samples. There were no data on children less than 14 years of age. Notable among these surveys are reports that assayed more than 7,000 sera from different regions of Turkey. A study found *T. gondii* IgG antibodies in 28.8% and IgM antibodies in 1.9% of 10 295 patients from the Marmara region (Alver *et al*., 2014). Another study reported *T. gondii* IgG antibodies in 21.3% of 4,048 and IgM antibodies in 1.2% of 13 605 patients from Bolu province in the Black Sea region (Aydın Türkoğlu *et al*., 2018). From the Central region of Turkey, IgG antibodies were found in 29.5% and IgM antibodies in 2.4% of the 7051 hospital patients (Maçin *et al*., 2018). The prevalence of *T. gondii* antibodies in pregnant women varied a great deal (Table 3). In the largest sample of 30 863 women tested, IgG antibodies were detected in 25.5% and IgM antibodies were present in 0.3% (Çelen *et al*., 2013). Several authors emphasized the need to use multiple serological tests for diagnosis of acute infection and serological conversion during pregnancy (Tanyuksel *et al*., 2004; Bahar *et al*., 2005; Ertug *et al*., 2005; Doğan *et al*., 2012; Uysal *et al*., 2013; Karacan *et al*., 2014; Dinçgez *et al*., 2018).

Limited risk factor analysis data indicated that eating raw meat (Tekay and Özbek, 2007; Doğan Toklu, 2013; Gencer *et al*., 2014), contact with soil (Doğan Toklu, 2013; Gencer *et al*., 2014), and consumption of raw eggs (Gencer *et al*., 2014) were the main factors associated with seroprevalence. One study found seroprevalence of *T. gondii* IgG in 64.6% of 84 Syrian refugees (Bakacak *et al*., 2015).

Mother to foetus transmission of T. gondii *and congenital toxoplasmosis*

There are no firm data with respect to transmission of *T. gondii* during pregnancy. *Toxoplasma gondii* DNA was detected in amniotic fluid in one of 300 foetuses tested between 15th and 18th week of gestation (Gunel *et al*., 2012). Evidence of recently acquired infection (based on low-avidity antibodies) in one of 4651 women tested during the first trimester (Uysal *et al*., 2013). The intrauterine growth of the foetus was retarded with deficient amniotic fluid; there was no follow up. Chorioretinitis was diagnosed in one of the seven babies born to mothers that had IgM antibodies.

One study followed the outcomes of pregnancy in 13 women who had seroconverted to *T. gondii* infection during pregnancy; all were given anti-*T. gondii* therapy (Samanci *et al*.,1995). One of the childrens had classical symptoms of congenital toxoplasmosis: chorioretinitis, intracerebral calcification and hydrocephalus at birth. Thus, the authors estimated the risk of foetal infection at 7.1% (1 of 13), based only on symptoms; there was no confirmation of congenital toxoplasmosis.

Evidence was presented that spiramycin therapy during pregnancy can reduce congenital transmission of *T. gondii* (Avci *et al*., 2016). Of the 61 women who acquired *T. gondii* infection during pregnancy, 55 (90.2%) received spiramycin prophylaxis while six (6.6%) refused it. Obvious lesions of congenital toxoplasmosis were evident by ultrasonographic examination of foetuses of two mothers who refused spiramycin therapy. Both foetuses had intracranial calcification, enlarged ventricles and

Table 1. Details of serological tests used for the seropositivity of *T. gondii* in humans and animals in Turkey

Abbreviation of test	Antigen	Cut-off titer	Manufacturer	Tables referred
Sabin-–Feldman dye test (SFDT)				
1. SFDT	Live tachyzoites	⩾1: 16	In-house	2, 5, 6, 10
2. SFDT		⩾1: 4	In-house	5, 7, 10
Immunofluorescence assay (IFA)				
1. IFA	Inactivated	IgM ⩾ 1:16	Euroimmun GmbH	2
		IgG ⩾ 1:64		
2. IFA	Inactivated	IgG ⩾ 1:16	In-house	3, 4, 5
Indirect fluorescent antibody test (IFAT)				
1. IFAT	Inactivated	IgM ⩾ 1:16	Euroimmun GmbH	2
2. IFAT		IgG ⩾ 1:64	In-house	6
		IgM ⩾ 1:16		
3. IFAT		IgG ⩾ 1:16	In-house	4, 5
Indirect haemagglutination assay (IHA)	Soluble	IgG ⩾ 1:80	Toxo-HAI Fumouze kit	2
		IgM: NS		
Latex agglutination test (LAT)	Soluble	NS	Toxolatex Fumouze Diagnostics	6, 10
Chemiluminescence microparticle immunoassay (CMIA)	p30 antigen	IgG ⩾ 3 IU mL^{-1}	ARCHITECT i1000 System, Abbott	3
		IgM ⩾ 0.6 IU mL^{-1}		
Chemiluminescence immunoassay (CLIA)				
1. CLIA	Solid	>1 IU mL^{-1}	UniCel DxI 800, Beckman Coulter	3
		>6 IU mL^{-1}		
2. CLIA	Solid	IgG ⩾ 8.8 IU mL^{-1}	Cobas 6000, Roche Diagnostics	3
		IgM ⩾ 1.0 IU mL^{-1}		
3. CLIA	Solid	IgG ⩾ 8.8 IU mL^{-1}	LIAISON, DiaSorin S.p.A	3
		IgM ⩾ 1.0 IU mL^{-1}		
4. CLIA	Solid	IgG ⩾ 3 IU mL^{-1}	Cobas 6000, Roche Diagnostics	3
		IgM ⩾ 0.49 IU mL^{-1}		
5. CLIA	Solid	NS	Vitros Eciq ABD	3
Electrochemiluminescence method (ECLIA)				
1. ECLIA	Soluble	IgG ⩾ 3.0 IU ml^{-1}	Cobas e-170 analyser, Roche Diagnostics	2
		IgM ⩾ 1.0 IU mL^{-1}		
2. ECLIA	Soluble	IgG ⩾ 3.0 IU ml^{-1}	ECLIA-Roche, Elecsys	3
		IgM ⩾ 1.0 IU mL^{-1}		
3. ECLIA	Soluble	IgG ⩾ 3.0 IU mL^{-1}	Cobas e-601 analyser, Roche Diagnostics	3
		IgM ⩾ 1.0 IU mL^{-1}		
Enzyme linked immunosorbent assay (ELISA)				
1. ELISA Kit	NS	⩾1,1 IU mL^{-1}	Atlas Link Microwell	2
2. ELISA Kit	Soluble	IgG and IgM > 1 IU mL^{-1}	IgG (Equipar)	2
		VIDAS IgM indices >0.65 IU mL^{-1}	IgM (Equipar) and (VIDAS TOXO IgM; Biomerieux)	
3. ELISA	Soluble whole tachyzoites	NS	In-house	2
4. IgG ELISA	Whole tachyzoites	NS	Abbott Axsym System	2
5. ELISA	Whole tachyzoites	IgG ⩾ 10 IU mL^{-1}	Meddens Diagnostica BV	2, 3
6. ELISA	Soluble	IgG > 8 IU mL^{-1}	VIDAS, Biomerieux	2
		IgM > 0.65 IU mL^{-1}		
7. Macro ELISA	Inactivated	IgG = 3 IU mL^{-1}	Cobas E411, Roche Diagnostics	2
		IgM = 1 IU mL^{-1}	Abbott Architect system, Wiesbaden	

(*Continued*)

Table 1. (*Continued.*)

Abbreviation of test	Antigen	Cut-off titer	Manufacturer	Tables referred
8. Macro ELISA	Inactivated	IgG ⩾ 8 IU mL^{-1}	Immulite®	2, 3
		IgM ⩾ 1.1 IU mL^{-1}	2000 XPi™ İmmunoassay System (Siemens)	
9. ELISA	NS	IgG 3 IU mL^{-1}	(Axsym, Abbott)	3
		IgM 0.490 IU mL^{-1}		
10. MEIA	Soluble	⩾3 IU mL^{-1}	(Axsym Plus immünoanalizör)	3
11. Micro ELISA	Soluble	IgG > 20 IU mL^{-1}	DYNEX technologies, inc.	3
12. ELISA	NS	IgG 3 IU mL^{-1}	Cobas 4000 e411 (Roche)	3
		IgM 0.6 IU mL^{-1}		
13. ELISA	Inactivated	IgG ⩾ 8 IU mL^{-1}	Immulite®	3
		IgM ⩾ 1.1 IU mL^{-1}	2000 XPi™ İmmunoassay System Siemens Healthcare Diagnostics Inc.	
14. Micro ELISA	Soluble	IgG > 3.0 IU mL^{-1}	Cobas e-601 analyser, Roche Diagnostics	3
		IgM < 1.0 IU mL^{-1}		
15. ELISA	Soluble	NS	In house	3
16. ELISA	Soluble whole tachyzoites	NS	Institut Pourquier	5
17. ELISA	Sonicated tachyzoite	NS	In-house	9
Enzyme-Linked fluorescence assay (ELFA)	Membrane and cytoplasmic Toxoplasma RH strain	IgG ⩾ 8 IU mL^{-1}	VIDAS TOXO IgG II (BioMerieux)	2, 3
		IgM 0.65 IU mL^{-1}		
Enzyme immunoassay (EIA)				
1. EIA	NS	>1.1 IU mL^{-1}	Euroimmun Labordiagnostica	2
2. EIA	Soluble	IgG ⩾ 3 IU mL^{-1}	Axsym, Abbott	3
		IgM ⩾ 0.600 IU mL^{-1}		
3. EIA	Soluble	IgA = 10–40 AU mL^{-1}	Different[a]	3
Microimmunoflorescence (MIF) IgG kit	Tachyzoites in the solid phase	NS	Euroimmun Labordiagnostica	2

[a]IgG ve IgM antibodies (Cobas Core, Roche), IgA antibodies (ETI-TOXOK-A reverse); IgG Avidity EIA Well-RADIM.

hepatomegaly. Pregnancy was terminated in foetuses from four of the six mothers who refused treatment; autopsy was not permitted. In summary, there are no estimates of the rate of congenital toxoplasmosis in Turkey.

There are only two proven cases of congenital toxoplasmosis, as documented by the isolation of viable *T. gondii.* The first report by Ekmen *et al.* (1974) was discussed earlier in the history section. The second case of congenital toxoplasmosis was a child who had bilateral chorioretinitis, hepato-splenomegaly and jaundice at birth and died 2 weeks later (Döşkaya *et al.*, 2013). The CSF collected from the baby at postmortem was inoculated intraperitoneally into mice and viable tachyzoites were found in the peritoneal fluid. This highly mouse-virulent strain was designated Ege-1 strain. Retrospectively, *T. gondii* IgG and IgM antibodies were found in serum samples of the child and mother; low-IgG avidity in mother serum indicating recent infection. Additionally, *T. gondii* DNA was detected by the polymerase chain reaction (PCR) in CSF. There was no screening for *T. gondii* antibodies in mother before or during pregnancy.

Clinical toxoplasmosis in adults

Ocular disease is a common sequelae of toxoplasmosis but definitive diagnosis is difficult. There are several reports of ocular toxoplasmosis in Turkey (Ozcan, 1975; Küçükerdönmez *et al.*, 2002; Atmaca *et al.*, 2004; Tanyuksel *et al.*, 2004; Tugal-Tutkun *et al.*, 2005; Avkan Oguz *et al.*, 2012; Celebi *et al.*, 2015; Oray *et al.*, 2015; Türkcü *et al.*, 2017). However, most of these reports were based on lesions and the presence of *T. gondii* antibodies in the serum. One of the shortcomings of serological diagnosis is that chronically infected patients have low-antibody titers, not different than in the general population; a negative serological test, however, rules out ocular toxoplasmosis. Among the reports of ocular toxoplasmosis, two studies are discussed here. Atmaca *et al.* (2004) reported 189 cases of ocular toxoplasmosis from 1972 to 1999; 140 (74%) were considered congenital toxoplasmosis and 49 (26%) acquired toxoplasmosis. At the first examination, 65 active lesions were detected in 65 eyes. Lesions were seen in macula of 59%. Manifestations of congenital toxoplasmosis included were: strabismus in 15%, nystagmus in 9.2%, microphthalmia in 2.6%, optic nerve atrophy in 3.1% and lens opacities in 1%. The second study (Tugal-Tutkun *et al.*, 2005) reported on 109 consecutive patients with active ocular disease in the Department of Ophthalmology, Istambul Faculty of Medicine in the last decade. The patients had IgG but no IgM antibodies to *T. gondii.* Retino-choroidal scars were present in 90 (83%) of 189 patients. Bilateral lesions, suggestive of congenital toxoplasmosis, were seen in 21 patients and active lesions were detected in 55 patients. All patients were treated with anti-*T. gondii* drugs with favourable prognosis.

Table 2. Serological prevalence of *T. gondii* in the general human population of different regions in Turkey

Group/age	Region/province	No. tested	Positive %	Test	Important findings/coinfections with other pathogens	References
Hospital patients	**Aegean**	50	26 (52)	IHA IgG		Ertuğ *et al.* (2000)
	Izmir		28 (56)	ELISA-IgG		
			28 (56)	IFA-IgG		
People in car accident	**Aegean**	185	45 (24.32)	ELISA[1] IgG		Yereli *et al.* (2006)
	Izmir		6 (3.24)	ELISA[1] IgM		
	Manisa					
Hospital patients	**Aegean**	72	36 (50)	IFA IgG		Delibas *et al.* (2006)
	Aydin			ELISA[2] IgG		
Dustmen worker	**Eastern Anatolia**	150	37 (24.6)	SFDT[1]	Sweepers had higher prevalence, coinfection with listeriosis	Çelik *et al.* (2008)
	Malatya					
Drivers	**Marmara**	243	130 (53.5)	EIA[1] IgG, MIF IgG		Kocazeybek *et al.* (2009)
	Istanbul and its suburbs		2 (0.82)	EIA[1] IgM		
			130 (53.5)	SFDT		
Prisoners	**Central Anatolia**	628	236 (37.58)	IFA[1]-IgG		Yaman *et al.* (2009)
	Kayseri		11 (1.75)	IFA[1]- IgG /IgM		
Hospital patients	**Eastern Anatolia**	4908	1522 (31.01)	ELISA[5] IgG	IgG was found in 171 (31.09%) newborn whereas no positives for IgM	Kuk and Ozden (2007)
	Elazig		38 (0.77)	ELISA[5] IgM		
Hospital patients	**Marmara**	10295	2761 (26.8)	ELFA IgG		Alver *et al.* (2014)
	Bursa		202 (1.9)	ELFA IgM		
Hospital patients	**Aegean**	1887	452 (24)	ECLIA[1] IgG	IgG was positive in 40 (18%) pregnant women whereas no positives for IgM	Aşcı and Akgün (2015)
	Afyonkarahisar	446	27 (1.4)	ECLIA IgM		
Hospital patients	**Aegean**	2942	954 (32.4)	Macro ELISA[8]-IgG		Pektaş *et al.* (2015)
	Izmır	3899	106 (2.7)	Macro ELISA[8]-IgM		
Hospital patients	**Black Sea**	4048	863 (21.3)	Macro ELISA[7]-IgG	IgG was found in 18 (24.3%) of 74 newborn whereas no positives in 93 newborn for IgM	Aydın Türkoğlu *et al.* (2018)
	Bolu	**13 605**	162 (1.2)	Macro ELISA[7]-IgM		
Hospital patients	**Central Anatolia**	7051	576 (29.53)	ELISA[6]-IgG		Maçin *et al.* (2018)
	Konya		120 (2.44)	ELISA[6]-IgM		
Women from urban area	**Central**	321	(44.9)	ELISA IgG	Risk assessment	Nas *et al.* (2007)
	Ankara	1732	(40.7)			
Women[a]	**Marmara**	96	33 (34.4)	ELISA[4]-IgG	Risk assessment	Tansel *et al.* (2009)
	Edirne					
Women[a]	**Marmara**	17 751	(24.61)	EIA IgG	TORCH pathogens	Akyar (2011)
	Istanbul, Bursa, Kocaeli		(1.34)	IgM		
	Mediterranean					
	Adana					
	Central					
	Kayseri					
Women[a]	**Central Anatolia**	1314	376 (28.6)	ELISA-IgG	Risk assessment	Aral Akarsu *et al.* (2011)
	Ankara		1 (0.07)	ELISA-IgM		
Women[a]	**Mediterranean**	2986	(32.64)	Macro ELISA[7]		Pekintürk *et al.* (2012)

(*Continued*)

Table 2. (*Continued.*)

Group/age	Region/province	No. tested	Positive %	Test	Important findings/coinfections with other pathogens	References
	Antalya	5013	(1.8)	IgG		
				IgM		
Women[a]	**Marmara**	5073	1559 (30.7)	ELFA IgG		Alver *et al.* (2014)
	Bursa					
Women[a]	**Marmara**	1101	(31)	ELISA	TORCH pathogens	Numan *et al.* (2015)
	İstanbul			IgG		
				IgM		
Students	**Aegean**	171	171 (100)	ELISA[3] IgG/IgM	Risk assessment	Doganci *et al.* (2006)
	Izmir					
School students	**Marmara**	388	61 (18)	ELISA-IgG	Risk assessment/cystic echinococcosis	Tamer (2009)
	Kocaeli			ELISA-IgM		
Students	**Central Anatolia**	347	81(23.3)	IFAT[1] IgG	Foreign student	Çetinkaya *et al.* (2011)
	Kayseri		6 (1.72)	IFAT[1] IgG /IgM		

[a]15–50 year old.

Confirmatory tests, such as the detection of *T. gondii* antibodies in aqueous humour or *T. gondii* DNA, were not used in any reports on ocular toxoplasmosis.

Lymphadenitis, dermal rash, fever, headaches, myalgia, hepatitis and chorioretinitis are some of the common symptoms of acquired toxoplasmosis. A 46-year old woman who had a history of eating undercooked meat developed maculopapular rashes, hepatomegaly and elevated liver enzymes (Atilla *et al.*, 2015). She had both IgG and IgM *T. gondii* antibodies, and *T. gondii*-like bodies were seen in histological sections of liver biopsy.

Reactivation of chronic *T. gondii* infection in patients with human immunodeficiency virus (HIV) has been reported worldwide, but rarely from Turkey. We are aware of only two reports of toxoplasmosis in HIV-infected patients in Turkey. Cerebral toxoplasmosis was diagnosed in a 29-year-old woman with HIV infection who had fever, vomiting, headache, weakness of the right side and seizure (Yapar *et al.*, 2005). Cranial MRI revealed four mass lesions in the cerebellar cortex. The patient had *T. gondii* IgG antibodies but no IgM antibodies. She was treated with clindamycin and antiviral therapy. After 2 months, the neural lesions were resolved.

The second patient, a 37-year-old male, who had neurological signs, cranial MRI revealed multiple cerebral lesions and the diagnosis of toxoplasmosis was confirmed by brain biopsy (Midi *et al.*, 2008). Despite antitoxoplasmosis, antiretroviral and antidopaminergic treatments, the patient died because of bacterial septicemia.

There is only limited information concerning toxoplasmosis in patients with other immunosuppressive disorders or transplant recipients. Possible toxoplasmosis was reported in five of 170 allogenic haematopoietic stem cell transplant patients, based on MRI, response to treatment with anti-*T. gondii* therapy and the PCR on CSF (Hakko *et al.*, 2013). Two of these patients died of toxoplasmosis; postmortem was not performed.

Toxoplasmosis was detected in four of 40 liver transplants in a transplant service in Turkey (Caner *et al.*, 2008). Both the donors and the recipients were seropositive to *T. gondii*. Toxoplasmosis was suspected when symptoms of fever, headaches and nausea were noted. Diagnosis was supported by positive DNA detection in blood of patients using PCR and anti-*T. gondii* therapy; all four patients recovered.

A rare case of spinal cord arachnoiditis was reported by Cosan *et al.* (2011). *Toxoplasma gondii*-like tachyzoites and bradyzoites were said to be detected but images are too low power to be convincing.

There are several reports of higher *T. gondii* serological prevalence in patients with schizophrenia (Cetinkaya *et al.*, 2007; Tamer *et al.*, 2008; Dogruman *et al.*, 2009; Tanyüksel *et al.*, 2010; Çelik *et al.*, 2015; Cevizci *et al.*, 2015; Karabulut *et al.*, 2015; Yuksel *et al.* 2010) and patients with chronic renal failure (Yazar *et al.*, 2003; Ocak *et al.*, 2005), neoplastic disorders (Yazar *et al.*, 2004), cirrhosis (Ustun *et al.*, 2004; Atilla *et al.*, 2015), chronic heart failure (Yazar *et al.*, 2006), reactive arthritis (Sert *et al.*, 2007), idiopathic Parkinson's disease (Celik *et al.*, 2010), Alzheimer's disease (Kusbeci *et al.*, 2011) and Multiple Sclerosis (Koskderelioglu *et al.*, 2017); however, the sample size and appropriate controls were lacking for cause effect relationship.

Toxoplasmosis in animals

Companion animals

Cats

Cats are key in the epidemiology of *T. gondii* because they are the only hosts that can excrete environmentally resistant oocysts. Seroprevalence varied with age and life style of the cat (Table 4). Viable *T. gondii* was isolated from homogenates of hearts and brains in 20 of 100 cats by bioassay in mice (Can *et al.*, 2014). Additionally, *T. gondii* DNA was detected in tissues of 12 additional cats. These 32 *T. gondii* strains were used for genotyping – (see molecular epidemiology and genotyping analysis section).

To our knowledge, there is only one report dealing with prevalence of *T. gondii*-like oocysts in cats from Turkey. *Toxoplasma gondii*-like oocysts measuring 9–14 μm in size were found in faeces of three of 36 (8.3%) cats from farms, however, results were not confirmed by bioassay or by PCR (Muz *et al.*, 2013).

There are three reports of clinical toxoplasmosis in cats from Turkey. The first report concerns three cats (one, 8-month old male necropsied in 1988, one 3-month old female and one 6 month old female, both necropsied in 1984) (Haziroglu

Table 3. Seroprevalence of *T. gondii* in pregnant women tested in hospital from different regions in Turkey

Region/province	No. tested	Test	IgG (%)	IgM (%)	References
Central Anatolia/Ankara	37	ELISA[5]	35 (94.6)	2 (5.4)	Tanyuksel *et al.* (2004)
Agean/Aydin	389	ELISA[15] IFA[2]	(30.1)	None	Ertug *et al.* (2005)
Aegan/Izmir	52	EIA[5]	14 (26.9)	14 (26.9)	Bahar *et al.* (2005)
Southeastern Anatolia/ Şanliurfa	2586	CLIA[1]	1798 (69.5)	78 (3)	Tekay and Özbek (2007)
Mediterranean/Hatay	1652	ELISA[9]	860 (52.1)	9 (0.54)	Ocak *et al.* (2007)
Marmara/Kocaeli	1972	ELISA[9]	952 (48.3)	8 (0.4)	Tamer *et al.* (2009)
Eastern Anatolia/Van	625	EIA[4]	225 (36)	2 (0.3)	Efe *et al.* (2009)
Central Anatolia/Kayseri	2235	MEIA[10]	(33.42)	(2.95)	Inci *et al.* (2009)
Central Anatolia/Kayseri	1676	EIA[4] IgG	568 (33.9)	46 (2.5)	Kayman and Kayman (2010)
	1813	EIA[4] IgM			
Marmara/Edirne	1646	ELISA[9]	426 (31.95)	13 (0.97)	Varol *et al.* (2011)
Mediterranean/Antalya	7520	CLIA[2]	1262 (31)	22 (0.5)	Çekin *et al.* (2011)
Aegean/Denizli	1102	Automated Vitros ECiQ system	408 (37)	15 (1.4)	Karabulut *et al.* (2011)
Eastern/Malatya	312	ELISA	117 (37.5)	104 (33.3)	Doğan *et al.* (2012)
Mediterranean/Hatay	3340	ELISA	1910 (57)	120 (3.6)	Okyay *et al.* (2013)
Central/Ankara	30 863	CLIA[3] ELISA	7869 (25.5)	83 (0.3)	Şevki *et al.* (2013)
Aegean/Uşak	1465	Micro ELISA[11]	268 (18.3)	44 (3.0)	Doğan Toklu (2013)
Marmara/Istanbul	1258	ELFA	291 (23.1)	5 (0.4)	Karacan *et al.* (2014)
Marmara/Canakkale	196	ELISA	(28.8)	(2.7)	Gencer *et al.* (2014)
Black Sea/Artvin	1133	CMIA	343 (30.3)	15 (1.3)	İnci *et al.* (2014)
Central/Ankara	4758	ELFA	1278 (26.9)	8 (0.2)	Mumcuoglu *et al.* (2014)
Black Sea/Tokat	3162	ELISA	1011 (32)	36 (1.1)	Çeltek *et al.* (2014)
Mediterranean/Kahraman Maraş	4113	Micro ELISA IgG	84 (64.6)	12 (4.8)	Bakacak *et al.* (2015)
	7201	Micro ELISA IgM			
Easten Anatolia/Van	457	ELISA[12] IgG	172 (37.6)	99 (1.1)	Parlak *et al.* (2015)
	9156	ELISA[12] IgM			
Black Sea/Zonguldak	910	CLIA[4]	(43.9)	(2.5)	Aynioglu *et al.* (2015)
Black Sea/Amasya	1838	CLIA[5] IgG	430 (23.39)	19 (1.02)	Kılınç *et al.* (2015)
	1852	CLIA[5] IgM			
Aegean/Afyonkarahisar	1091	EIA[5] IgG	256 (23.4)	16 (1.5)	Şimşek *et al.* (2016)
	1020	EIA[5] IgM			
Black Sea/Ordu	1394	ECLIA[2] IgG	385 (27.6)	22 (1.6)	Çalgın *et al.* (2017)
	1397	CMIA IgM			
Aegean/Mugla	191	Automated analyser	36 (18.8)	7 (3.7)	Kasap *et al.* (2017)
Aegean/Izmir	7513	CLIA[3] IgG, IgM	2427 (32.3)	138 (1.9)	Sirin *et al.* (2017)
Mediterranean/Hatay	11.564	ECLIA[3] IgG, IgM	(48.70)	(3.9)	Çetin and Çetin (2017)
		CMIA IgG			
Mediterranean/Isparta	1937	Macro[8] ELISAIgG	344 (28.4)	34 (1.8)	Akpınar *et al.* (2017)
	1203	Macro[8] ELISAIgM			
Eastern Anatolia/Bingöl	10 178	ECLIA[3]	6155 (63)	196 (2)	Nazik *et al.* (2017)
Mediterranean/Adana	11.313	ELISA[13]	5233 (46.3)	200 (1.8)	Bozok (2017)
Marmara/Bursa	412[a]	Micro ELISA[14]	125 (30.6)	27 (6.6)	Dinçgez Çakmak *et al.* (2018)[a]
	828[a]		157 (19.2)	35 (4.2)	
Marmara/Adapazarı	1007	CMIA IgG	261 (25.9)	No positivity	Aydemir *et al.* (2018)

[a]Pregnancies with abortion (412), normal pregnancies (828).

Table 4. Serological prevalence of *T. gondii* at cats in different regions of Turkey

Category	Region (place)	No. tested	Test	Positive %	Cut-off titer	References
Stray	Central	77	SFDT	16 (21)	⩾1:16	Ekmen (1970)
	Ankara					
Pets	Central	65	SFDT	28 (43)	⩾1:16	Inci *et al.* (1996)
	Ankara					
Pets[a]	Central	99	SFDT	40 (40.3)	⩾1:16	Taylan Özkan *et al.* (2008)
	Ankara		IFAT[3] IgG	34 (34.3)		
Stray[b]	Central	72	SFDT	55 (76.4)	⩾1:16	Karatepe *et al.* (2008)
	Nigde					
Stray and pets[c]	Izmir	1121	ELISA[27] IgG	33.4–34.4	–	Can *et al.* (2014)
			IFA[2] IgG	42–48	⩾1:16	
Indoor[d]	Eastern Anatolia	102	SFDT[1]	45 (44.1)	⩾1:16	Erkılıç *et al.* (2016)
	Kars					
Indoor[e]	Central Anatolia	102	SFDT[1e]	49 (48.03)	⩾1:16	Yasa Duru *et al.* (2017)
	Kırıkkale					
	Ankara					

[a]The seropositivity of *T. gondii* in cats older than 1 year (47.8%) was more about three times than cats less than a year old (13.6%). The seropositivity in indoor cats was 23.1% by IFAT and 30.8% by SFDT. The seropositivity in stray cats was 41.7% by IFAT and 52.8% by the SFDT. The presence of antibodies was significantly related to outdoor access.
[b]Seropositivity in male cats (48.6%) was lower than in female cats (51.4%).
[c]Tissues of 100 cats were used to isolate viable *T. gondii* (shown in the text).
[d]No significant differences for the seropositivity between 20 (44.4%) of 45 males and 25 (43.9%) of 57 females.
[e]Nested PCR was positive for four cats (8.2%). Foetal toxoplasmosis was diagnosed in one cat.

et al., 1988; Haziroglu, 1993). All three cats had disseminated toxoplasmosis and *T. gondii* was detected in multiple tissues of these cats histologically and by transmission electron microscopy.

Histologically confirmed disseminated toxoplasmosis was detected in a 2.5 year-old queen and her two kittens (Atmaca *et al.*, 2013). The queen was hospitalized because of dystocia. Ultrasound examination revealed that three of five kittens had died in utero. After cesarean section, the queen and the two live kittens died. The queen had pneumonia and hepatitis. The presence of *T. gondii* was confirmed by both immunohistochemical examination and PCR testing in the kittens and the queen, thus, confirming transplacental toxoplasmosis.

Dermal toxoplasmosis was diagnosed in 2-year old female immunocompetent Angora cat (Kul *et al.*, 2011). The cat had an antibody titer of 1:256 using the dye test and diagnosis was confirmed immunohistochemically and by PCR testing. The dermal lesions resolved after treatment with azithromycin.

Dogs

Serologic reports are summarized in Table 5. Viable *T. gondii* was isolated from lungs of a dog (Ekmen and Altıntaş, 1970) but details of the dog's condition are missing. Clinical toxoplasmosis was diagnosed postmortem in a dog owned by an American visitor to Turkey (Akcay *et al.*, 1950); in retrospect it is not clear if the dog had toxoplasmosis or neosporosis (Dubey *et al.*, 2017).

Toxoplasmosis in livestock

Sheep and goats

Serological surveys indicate widespread exposure to *T. gondii* in sheep and goats in Turkey (Tables 6 and 7). Although toxoplasmosis is a major cause of abortion in sheep and goats worldwide (Dubey, 2010), there is little information from Turkey. *Toxoplasma gondii*-associated abortion cannot be diagnosed alone by serological testing of ewes; a negative serology rules out toxoplasmosis but positive result does not establish aetiology because seroprevalence is high in general population and antibodies can remain elevated in the next pregnancy (Dubey, 2010). To our knowledge, there is no confirmed report of *T. gondii*-associated abortion in sheep or goats from Turkey. Although *T. gondii* DNA was found in five of 20 sheep tissues (Ergin *et al.*, 2009), there is no report of isolation of viable *T. gondii* from sheep and goats in Turkey.

Horses

Serologic data are summarized in Table 8. There is no report of clinical toxoplasmosis in horses.

Cattle

There is limited information concerning toxoplasmosis in cattle in Turkey (Table 9). Additionally, the dye test used in several surveys is nonspecific and gives erratic results with cattle sera (Dubey and Beattie, 1988). Finding of *T. gondii* DNA in five of 10 beef samples (Ergin *et al.*, 2009) needs confirmation.

Pigs

Toxoplasma gondii infection is not a significant direct risk for human in Turkey because pork is not eaten in Turkey due to religious restrictions, 99.8% of population is muslim ((Library of Congress. Federal Research Division website: https://en.wikipedia.org/wiki/Islam_in_Turkey#cite_note-11 (accessed 29 August 2018)).

Various other animals

Serologic prevalence of *T. gondii* in miscellaneous animals is shown in Table 10. There is no confirmed report of clinical toxoplasmosis in animals, except in a zoo animal. Fatal toxoplasmosis was diagnosed in a captive kangaroo (*Macropus* sp.) from a zoo in Ankara (Kabak *et al.*, 2011). *Toxoplasma gondii*

Table 5. Serological prevalence of *T. gondii* in dogs from different regions of Turkey

Type of dog	Region (place)	No. tested	Test	Positive %	Cut-off titer	References
Healthy stray	Central Anatolia Ankara	116	SFDT[1] IFAT3	72 (62.0)	1:16	Aslantaş *et al.* (2005)
Stray and pets	Marmara Izmit (Kocaeli)	116	SFDT[1]	81 (69.8%)	⩾1:16	Simşek *et al.* (2006)
Stray	Marmara Istanbul	150	IFAT[3]	77 (51.3)	1:64	Öncel *et al.* (2007)
Stray	Southern Anatolia Şanlıurfa	80	SFDT[1]	78 (97.5)	⩾1:16	Babür et al. (2007*a*)
Stray	Eastern Anatolia Van	69	SFDT[2]	40 (57.9)	⩾1:4	Babür *et al.* (2007*b*)
Stray	Central Anatolia Kırıkkale	35	SFDT[1]	19 (54.3)	⩾1:16	Yıldız *et al.* (2009*b*)
Stray	Central Anatolia Ankara	107	SFDT[1]	58 (54)	⩾1:16	Şahal *et al.* (2009)
Stray	Southeastern Anatolia Diyarbakir	100	SFDT[2]	94 (94)	⩾1:4	Içen *et al.* (2010)
Stray	Eastern Anatolia Erzurum	72	SFDT[1]	70 (97%)	⩾1:16	Balkaya *et al.* (2010)
Pets and sheep dog	Eastern Anatolia Kars	179	SFDT[1]	172 (96.1)	No data	Gicik *et al.* (2010)
Military dogs	Central Anatolia Nevsehir	140	SFDT[1]	81 (57.86)	⩾1:16	Kırbaş *et al.* (2011)
Pets and stray	Central Anatolia Sivas	120	SFDT[1]	115 (95.8)	⩾1:16	Altay *et al.* (2013)
Shepherd dogs	Mediterranean Hatay	46	ELISA[16] IgG	27 (58.7)	No data	Muz *et al.* (2013)

DNA was demonstrated in the brain of a badger (*Meles meles*); indicating contamination of local waters by *T. gondii* (Karakavuk *et al.*, 2018*a*).

Meat as source of *T. gondii* infectıon

Humans acquire *T. gondii* infection postnatally by eating undercooked meat containing tissue cysts or ingesting food and water contaminated with oocysts (Dubey, 2010). There are reports of very high prevalence of *T. gondii* tissue cysts in sheep in Turkey destined for human consumption (Yildiz *et al.*, 2014, 2015). In the first of these two reports, antibodies to *T. gondii* were found in 88 of 100 sheep; tissue cysts were detected in 46, in 36 by using the Percoll concentration technique in 5 g of ovine brain. Additionally, by using immunohistochemical staining with *T. gondii* antibodies, tissue cysts were found in 17% of tissue sections. However, the parasites assumed to be *Toxoplasma* depicted in Fig. 2 of their paper are not *T. gondii* and are most likely *Sarcocystis* spp. (J. P. Dubey, own opinion). Polyclonal *T. gondii* antibodies can cross react with *Sarcocystis* (Dubey, 2010). In the second report by these authors, tissue cysts were detected in 21.2% of 250 sheep meat samples by the Percoll method (Yildiz *et al.*, 2015).

There is a similar report of high prevalence of *T. gondii* tissue cysts in buffalo meat imported from India into Turkey (Gencay *et al.*, 2013). Tissue cysts were found in meat in three of 20 (15%) buffaloes tested by the Percoll method. The diagnosis was reported to be confirmed by PCR in both the buffalo and sheep-derived tissue cysts. However, the parasites assumed to be *T. gondii* depicted in Fig. 1 of their paper (Gencay *et al.*, 2013) are most likely pollen grains and not *T. gondii* tissue cysts (J. P. Dubey, own opinion). Additionally, water buffaloes are considered resistant hosts of *T. gondii,* and *T. gondii* has not been isolated from buffalo meat in any country (Dubey, 2010).

These studies need confirmation because the density of *T. gondii* cysts in adult sheep is one tissue cyst in more than 100 g sheep tissue (Dubey, 2010). Reports of the presence of DNA in tissues of food animals are summarized in Table 11. However, detection of DNA does not relate to the presence of live organisms.

Environmental contamination by *T. gondii* oocysts

Cats can excrete millions of *T. gondii* oocysts and oocysts that can survive harsh conditions in the environment (Dubey, 2010). *Toxoplasma* oocyst DNA has been detected in water samples from different regions in Turkey but not in drinking water (Table 12). Overall, sedimentation and filtration procedures are efficient in trapping *T. gondii* oocysts, water used for irrigation and farm animals may not be filtered.

Additionally, *T. gondii* oocysts can be concentrated by molluscs and fish. *Toxoplasma gondii* DNA was detected in 39.6% of edible shellfish (*Mytilus galloprovincialis*) in Izmir (Aksoy *et al.*, 2014).

Table 6. Serological prevalence of *T. gondii* in sheep in different regions of Turkey

Region/province	No. tested	Test	Positive %	Antibody titer range	References
Central Ankara	123	SFDT[1]	48 (29.1)	1:16	Ekmen (1967)
–	250	SFDT[1]	(38)	1:16–1:256	Weilland and Dalchow (1970)
Eastern Anatolia Elaziğ	295	IHA	(27.7)	1:32–1:256	Dumanli *et al.* (1991)
Central Ankara	1050	LAT	(14.6)	1:64–1:154	Zeybek *et al.* (1995)
Mediterranean Adana	42[a]	IHA	(9.5)	1:64–1:154	Oz *et al.* (1995)
–	603	SFDT	(31.1)	1:16–1:256	Altıntaş (1996)
Black Sea Samsun	62	SFDT	(88.7)	1:16–1:256	Babür *et al.* (1997)
Central Kayseri	154	SFDT	(33.8)	1:16–1:256	Inci *et al.* (1999)
Eastern Anatolia Elaziğ	154[b]	SFDT[1b]	72 (46.8)	1:16–1:1024	Aktas *et al.* (2000)
Central Konya	1110[c]	IFA[2]	(13.78)	1:64–1:2048	Sevinç *et al.* (2000)
Eastern Anatolia Kars	103[d]	SFDT[1]	53 (51.45)	No data	Aslantaş and Babür (2000)
Central Yozgat	152	SFDT[1]	(45.4)	1:16	Babür *et al.* (2001)
Aegean Afyon	172	SFDT	(54.6)	1:16–1:256	Çicek *et al.* (2004)
Central Niğde	110	SFDT	(50.9)	1:16–1:256	Karatepe *et al.* (2004)
Marmara Yalova	63	SFDT[1]	42 (66.66)	1:16–1:256	Oncel *et al.* (2005)
		LAT[e]	41 (65.08)		
South-eastern Şanlıurfa	300[f]	SFDT[1]	167 (55.66)[f]	1:16–1:1024	Sevgili *et al.* (2005)
Marmara Istanbul	182[g]	ELISA[19] IgG	56 (31)	–	Oncel and Vural (2006)
Eastern Anatolia Kars	460	ELISA	(95.7)	–	Mor and Arslan (2007)
Aegean Afyonkarahisar	186	SFDT[1]	184 (98.92)	1:16–1:256	Çiçek *et al.* (2011)
Mediterranean Hatay	184	ELISA[10] IgG	99 (53.8)	–	Muz *et al.* (2013)
South-eastern Anatolia Silopi	100[h]	IFAT[2]	97 (97)	1:16–1:256	Leblebicier and Yıldız (2014)
Different[i]	610	Indirect ELISA[21] IgG	122 (20.0)	–	Zhou *et al.* (2017)
Central Nevşehir	180[j]	ELISA[22] IgG	18 (10)	–	Özmutlu Çakmak and Karatepe (2017)

[a]25.5% seropositivity of 259 aborted sheep with titers of 1:64–1:256.
[b]Pregnant 56, aborted 57 in the previous year, aborted 41 within the period of the study. Inactivating temperature for the ovine complement was 56 °C. Ovine sera should be heated to 60°C to inactivate complement (Dubey, 2010).
[c]Healthy ewes 827, ewes aborted 283 (10.16%). No significant difference between the two groups for the presence of antibody titers.
[d]5 of 10 aborted sheep were seropositive for *T. gondii*.
[e]63 sheep older than one year of age were tested. SFDT was accepted as a reference test. The specificity and sensitivity of LAT were 61.90 and 78.57%, respectively. The correlation between two tests was 73.01%.
[f]The seropositivity of 0–1 year old was 58.34% and >1 year age was 47.23%, respectively. The seropositivity of male (47.45%) and female (57.67%) and the seropositivity of Akkaraman breed (54.8%), Ivesi (55.71%) and Morkaraman (63.63%). No significant correlation between serum titers of age, sex and breed.
[g]No dissimilarity between female (31.4%) and male ewes (30). The significant difference between 0.6–1 year age (12.5%) and >1 year age (41%),
[h]Seropositivity of aborted 25 sheep was 96%. No significant difference between 2–4 year (96%) and 5–10 year (100%) sheep.
[i]Karaman, Konya provinces from Central region and Zonguldak province from Black Sea. Identify specific antibodies to *T. gondii* (rTgSAG2-ELISA).
[j]The seropositivity 1 to 2 years sheep (11.53%) and 5 to 7 years (8.51%). No statistically significant differences between the two age groups. *Toxoplasma gondii* antibodies were detected in 18 (11.1%) out of 162 ewes, while there was no seropositivity in the 18 rams tested.

Molecular characterization

Although *T. gondii* infections are prevalent worldwide, there is a geographic distribution of *T. gondii* genetic types (Shwab *et al.*, 2014). Except for minor variations, *T. gondii* strains are broadly divided into four types: I, II, III and atypical. In general, Type I stains are rare worldwide (Dubey, 2010). Type II and Type III strains are prevalent worldwide, except Brazil where they are rare. The *T. gondii* strains in Europe are similar to those in North America. An unusual strain, designated Africa 1 genotype, has been identified in humans from sub-Saharan Africa (Döşkaya *et al.*, 2013), and more recently in Denmark (Jokelainen *et al.*, 2018).

As stated in the Introduction section, Turkey is geographically unique, a bridge between Asia, Africa and Europe therefore, providing an avenue for cross mixing of *T. gondii* strains.

For genotyping of *T. gondii*, it is important to have good quality *T. gondii* DNA with minimal contamination of host tissue. For

Table 7. Serological prevalence of *T. gondii* in goats from different regions of Turkey

Goat type	Region/province	No test	Test	Positive %	Cut-off titer	References
Mohair of Siirt[a]	South-eastern Anatolia (Siirt)	181	SFDT[2]	137 (75.7)	⩾1:4	Ataseven *et al.* (2006)
Norduz of Van[a]	Eastern Anatolia (Van)	94		63 (67)[b]		
Unspecified	Eastern Anatolia Van	98	SFDT[1]	79 (80.61)	1:16	Karaca *et al.* (2007)
Saanen × Kilis	Central Anatolia	74	SFDT[1]	60 (81. 1)[c]	⩾1:16	Ural *et al.* (2009)
Angora goats	Ankara	63		52 (82. 53)[c]		
Unspecified	Mediterranean Hatay	184	ELISA[20] IgG	66 (35.9)	–	Muz *et al.* (2013)
Unspecified[d]	South-eastern Anatolia	105	SFDT[1]	100 (95.24)	⩾1:16	Beyhan *et al.* (2013)
	Kilis					
Unspecified	Central Anatolia	249	Indirect ELISA[21] IgG	32 (12.9)	–	Zhou *et al.* (2017)
	Karaman and Konya provinces					

[a]Mohair goats used for the production of mohair and blankets and Norduz are dairy breed.
[b]42.85% seropositive at 1:16, 27.55% at 1:64, 7.17% at 1:256 and 3.06% at 1:1024.
[c]38.33% seropositivity of 60 Saanen × Kilis goats at 1:16, 50% at 1:64, 6.67% at 1:256 and 5% at 1:1024. 36.5% seropositivity of 52 Angora goats at 1:16, 44.2% at 1:64, 11.53% at 1:256 and 7.7% at 1:1024.
[d]100% of the Shami goats were seropositive with titers of 1:16 in 40 of 53, 12 at 1:64 and 1 at 1:256. 90.38% of Kilis goats were seropositive with titers of 1:16 in 36 of 52, 10 at 1:64 and 1 at 1:256.

Table 8. Serological prevalence of *T. gondii* in horses in different regions of Turkey

Region/place	No. tested	Test	Positive %	Cut-off titer	References
Central Anatolia	125	SFDT[1]	9 (7.2)	⩾1:16	Karatepe *et al.* (2010)
Niğde					
East Anatolia	74	IHA	13.5	1:160	Göz *et al.* (2007)
Hakkari		SFDT[1]	28.3	1:16	
Central Ankara	100	SFDT[1]	28	⩾1:16	Güçlü *et al.* (2007)
Central Ankara	168	SFDT[1]	62 (36.9)	⩾1:16	Gazyağci *et al.* (2011)
Mediterranean	616	Indirect ELISA[21] IgG	285 (46.3)	–	Zhou *et al.* (2017)
Adana					
Central Anatolia					
Konya					
Aegaen					
Izmir					
Marmara					
Bursa and Istanbul					
South-eastern Anatolia					
Gaziantep					

Table 9. Serological prevalence of *T. gondii* in cattle in different regions of Turkey

Region (place)	No. tested	Test	Positive %	Cut-off titer	References
East Anatolia	112	SFDT[1]	32 (22.3)	⩾1:16	Ekmen (1967)
Kars					
Central Anatolia					
Ankara					
East Anatolia	115[a]	SFDT[1]	57 (49.56)	No data	Aslantaş and Babür (2000)
Kars					
Central Kirikkale	100	SFDT[1]	53 (53)	⩾1:16	Öcal *et al.* (2008)
East Anatolia	216	ELISA[17]	202 (93.5)	–	Akca and Mor (2010)
Kars					
Kirikkale, Tokat, Izmir	557[b]	SFDT[1]	138 (24.77)	⩾1:16	Yıldız *et al.* (2009*a*)
Adana	132	SFDT[1]	(56.06)	⩾1:16	Yağci *et al.* (2014)
Mediterranean Hatay	184	ELISA[16] IgG	112 (60.9)	–	Muz *et al.* (2013)

[a]13 of 30 aborted cattle were seropositive for *T. gondii*.
[b]Aborted 234, pregnant cows 323.

Table 10. Serological prevalence of *T. gondii* in miscellaneous animals in different regions of Turkey

Animal	Region (place)	No. tested	Test	Positive %	Cut-off titer	References
Goitered gazelles (*Gazella subgutturosa*)	Southern Anatolia Şanliurfa	82	SFDT[1]	23 (28.04)	⩾1:16	Gokcen *et al.* (2007)
Camels	Central Anatolia Nevşehir	11	SFDT[1]	10 (90.9)	⩾1:16	Utuk *et al.* (2012)
Layer hens[a]	Central Anatolia Konya	287	SFDT[2]	1(0.34)	1:16	Altinöz *et al.* (2007)
Domestic pigeon (105)	Central Anatolia	105	SFDT[1]	1 (0.95)	1:16	Karatepe *et al.* (2011)
Wild pigeons (111)	Niğde	111		1 (0.90)		
Water buffaloes (*Bubalus bubalis*)	Black Sea Samsun Central Anatolia Afyon	131	SFDT[1]	115 (87.79)	⩾1:16	Beyhan *et al.* (2014)
Wild boars (*Sus scrofa*)	Eastern Anatolia (Erzurum)	12	SFDT[1]	4 (33.3)	1:16	Balkaya *et al.* (2015)
Quails (*Coturnix coturnix japonica*)	Central Anatolia Niğde	144	SFDT	0	No data	Kılıç *et al.* (2017)
Geese	Eastern Anatolia Kars	400	LAT	1 (0.25)	No data	Tasçi *et al.* (2018)

[a]The SFDT does not work with chicken sera (Dubey, 2010).

Table 11. Detection of *T. gondii* from tissues of food animals in different regions of Turkey

Samples	Regions (place)	No. tested	Test	Positive %	References
Slaughterhouse cattle	Marmara	50[a]	Nested PCR targeting B1 gene	5 (10)	Ergin *et al.* (2009)
Slaughterhouse sheep	İstanbul	20[a]		5 (25)	
Imported meat samples	Marmara Istanbul	20	Nested PCR targeting B1 gene	3 (15)	Gencay *et al.* (2013)
Shellfish mussels (*M. galloprovincialis*)	Agean Izmir	53	EvaGreen® real time PCR and high resolution melting (HRM)	21 (39.6)	Aksoy *et al.* (2014)
Brain and skeletal muscles from sheep	Central Anatolia Kirikkale	100	Nested PCR targeting B1 gene	46 (46)[b]	Yildiz *et al.* (2014)
Boneless sheep meat	Central Anatolia Kirikkale Ankara	250	Nested PCR for B1 gene	102 (40.8)	Yildiz *et al.* (2015)

[a]*Toxoplasma gondii* was found in 2% of 50 bovine brains, 6% of 50 bovine muscles, 4.17% of 120 ovine brains, 20% of 20 ovine muscles and 19% of 100 fermented sausage samples.
[b]Tissue cysts were at 78.2% of 36 brain and at 69.5% of 32 of skeletal muscles (masseter, tongue, diaphragm, intercostal and leg).

Table 12. Detection of *Toxoplasma gondii* from water samples in different region of Turkey

Samples	Regions (place)	No. tested	Test	Positive %	References
Water samples	Sinop, Ordu Rize, Amasya,	30	LAMP n-PCR	30 (100) 13 (43.33)	Koloren, 2013
Water samples	Ordu	56	LAMP Conventional PCR n-PCR	20 (35.7) 12 (21.42) 16 (28.57)	Koloren and Demirel 2013a
Water samples	Amasya	120	n-PCR	48 (40)	Koloren and Demirel 2013b
Water samples	Giresun	76	LAMP Conventional PCR	10 (13.2)	Demirel *et al.*, 2014

this, it is often necessary to extract DNA from viable strains. This has been achieved in three studies from Turkey. Döşkaya *et al.* (2013) found that both the *T. gondii* isolates obtained from CSF of diseased children were Africa 1 genotype. This Africa 1 genotype has been found in a cat but not yet identified in other hosts in Turkey. Of the 22 isolates of *T. gondii* genotyped from domestic cats in Turkey, 19 were classical Type II, two were Type III, and one was Africa 1 genotype (Can *et al.*, 2014). Of the five viable isolates of *T. gondii* genotyped from wild birds in Turkey, four were Type II and one was Type III (Karakavuk *et al.*, 2018*b*). Thus, the predominant distribution of genetic types of *T. gondii* in Turkey was like Europe.

Conclusion

It is evident from this review that there are many gaps in our knowledge of toxoplasmosis in humans and animals, particular data on clinical infections are lacking. There is no centralized facility for advice and confirmation of diagnosis. Little is known about the rate of congenital toxoplasmosis, and follow up of subclinically infected children for clinical toxoplasmosis. No information is available concerning modes of transmission. Little information is available concerning the presence of viable *T. gondii* in edible meats, and *T. gondii* oocysts in the environment. Educational programs are needed to prevent *T. gondii i*nfection in humans and animals.

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