Review article

The presence of *Toxoplasma gondii* in the terrestrial and marine environments and its importance for public health

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ABSTRACT. *Toxoplasma gondii* occurred in terrestrial and marine environments. Many people still do not realize that how important are the role of prevention, especially hygiene and proper food processing as well as diagnostics for pregnant women. Humans become infected with *T. gondii* primarily by eating raw or undercooked meat containing tissue cysts, or by ingesting oocysts with contaminated water or food (e.g. vegetables), very rarely by blood transfusion or organ transplantation. The new risk is consumption raw mussels in this oyster or blue mussels. Other invertebrates, such as some of crustaceans, are also capable of bioaccumulation of oocysts in their body. Invertebrates are mainly responsible for the spread of toxoplasmosis among marine animals, but if eaten raw, they can also be a risk factor for humans. This indicate the need to monitor also other species of invertebrates, especially those consumed by humans, due to possibility of being vector of *T. gondii*. Most adults do not have clinical symptoms. However, primary infection is dangerous in pregnant women due to the possibility of intrauterine infection of the fetus and the occurrence of congenital toxoplasmosis. The group of people who may develop clinical symptoms also include immunocompromised persons, especially those suffering from AIDS and treated with immunosuppressive drugs. Especially this group of people should receive actual information about all potential sources of infection due to the still low public awareness of threats. This paper presents a life cycle and transmission routes of *T. gondii* in terrestrial and marine environments with an indication of the importance for public health.

Keywords: *Toxoplasma gondii*, health education, public health, terrestrial and marine routes of infection

Introduction

Toxoplasma gondii was described in 1908 by French scientists, microbiologist Charles Jules Henry Nicolle and parasitologist Luis Herbert Manceaux, working at the Pasteur Institute in Tunis. They isolated this obligate intracellular parasitic protozoan (tachyzoites) from the tissues of the rodent - common gundi. They formed the name from the Greek words toxo, meaning arc, and *plasma* = essence, which refers to the arched shape of the vegetative form of this protozoan and the name of the host [1]. At the same time, the Italian physician and microbiologist Alfonso Splendore, who working in Brazil, found this parasite in rabbit tissues. Fifteen years after the Nicolle, Manceaux and Splendore findings, the Czech ophthalmologist Josef Janků described the first case of ocular toxoplasmosis in humans [1,2]. During the autopsy of an infant with hydrocephalus and unilateral microphthalmia, he found protozoan cysts in the damaged retina of the eye. Finally, in 1939, Abner Wolf, David Cowen and Beryl Paige demonstrated that *T. gondii* is an etiological factor of congenital toxoplasmosis in humans, as well as that it is the same pathogen as in animals [1,3-5]. In 1948, Albert Sabin and Harry Feldman introduced the first serological diagnostic test, based on the color reaction, consisting in the absorption of the methylene blue dye by tachyzoites [6]. It is now known as the Sabin and Feldman color test. The exact life cycle of *T. gondii* was not known until the 1970s [7,8]. Nowadays the parasite occurs not only in the terrestrial environment of various climatic zones, but in the aquatic environment, also in the marine environment. The first reports from the marine environment concerned infections of sea otters, later seroprevalence and symptoms of toxoplasmosis were found in various species of marine mammals [9,10].

Currently, toxoplasmosis is one of the most widespread parasitic diseases of warm-blooded animals and humans in the world. Seropositive people are more in the southern than in the northern locations, which is associated with the influence of climatic conditions on the survival of oocysts in the environment (more favorable conditions are in a warm and humid climate), as well as culinary habits and the level of hygiene. The lowest seroprevalence, ranging from 10–30%, is recorded in North America, Southeast Asia, and Northern Europe. Seroprevalence of 30–50% occurs in central and southern Europe, while high, up to 90% in South America and tropical Africa [11,12].

For most people, toxoplasmosis does not pose a health risk and does not require any diagnostic and therapeutic measures. Many people are not even aware of the infection. Nevertheless, toxoplasmosis is a significant public health problem because it can have serious adverse effects on fetus and it can cause clinical manifestation in immunocompromised patient [13,14]. This infection belongs to the group of TORCH diseases (toxoplasmosis, rubella, cytomegaly, herpes), caused by microorganisms capable of overcoming the placental barrier and infecting the fetus, causing malformations, miscarriage or stimulating premature birth. Congenital toxoplasmosis can cause neurological or ocular manifestations that can lead to blindness [15,16]. Flegr et al. [11] found the occurrence of latent toxoplasmosis in 40% of women of childbearing age in Poland, mainly from rural areas. According to the reports of the European Center for Disease Prevention and Control, the largest number of intrauterine infections with toxoplasmosis in the period 2010–2018 was recorded in France. In this country rate was between 20–31, with the exception of 2012 when rate was 12.7 cases per 100,000 live births. In Poland, in the years 2010-2014, the number of intrauterine infections with toxoplasmosis increased from 1.7 to 5.3 cases per 100,000 live births and remains at a similar level in the following years [17,18]. In Annual Epidemiological Report for 2018 most of confirmed cases of congenital toxoplasmosis were reported in France (73% all cases), were rate was 19.9 cases per 100 000 live births. High rate 10.2 cases was also found in Slovenia. The number of notifications per 100 000 living newborns was 6.4 in Poland with 5.8 in the EU/EEA [18].

Therefore, screening of pregnant women for toxoplasmosis is conducted. Toxoplasmosis is the most common opportunistic infection associated with HIV infection or other primary or acquired immune deficiencies worldwide [11,13]. In immunocompromised patients infection may be acute, involving the central nervous system, ocular manifestation or other tissues and organs. An important activity for the benefit of public health is educating and providing patients with reliable information on disease prevention, including information about the actual and potential routes of *T. gondii* infection spread and risk groups.

The terrestrial life cycle of T. gondii

Despite the fact that this apicomplexan parasite was discovered at the beginning of the 20th century, its life cycle was not established until the 1970s [7,19,20]. Since then, there have been many scientific papers describing the life cycle of *T. gondii*, including local relationships between definitive hosts and intermediate hosts in various regions of the world.

Definitive host

Only Felidae, domestic cats and wild felids i.a. lynxes, bobcats, jaguars, pumas, leopards, tigers are the definitive hosts of *T. gondii* [21]. According to Montazeri et al. [21] globally average seroprevalence of *T. gondii* in domestic cats is about 30 –40% and is lower than in wild felids. The highest seroprevalence in domestic cats occurred in Australia and Africa, 52% and 51% respectively. In the case of feral cats the highest seroprevalence was observed in Africa 74%, less in Asia and Europe 67% each and South America 66%.

These coccidia have three invasive forms: tachyzoites, bradyzoites and sporozoites in sporulated oocysts. Felids become infected primarily by ingesting meat with tissue cysts containing bradyzoites, less often with other forms of parasite. An important source of infection are hunted rodents [19,20,22]. The period from infection with bradyzoites to excrete oocysts by the cat usually takes 3–10 days and with tachyzoites or oocysts over 18 days [20].

In the stomach and small intestine of felids, proteolytic enzymes dissolve the wall of tissue cysts. Released bradyzoites penetrate the epithelial cells of the small intestine, where they initiate the development of asexual and sexual generations. In

the mucosa, they undergo numerous, rapid divisions as tachyzoites. The next stage is sexual reproduction – gametogony. It is initiated by the formation of schizonts, which then divide into merozoites. Some merozoites develop into gametocytes. In the process of sporogony, microgametes and macrogametes are formed. Fusion of these gametes create zygote. The zygote develops to oocyst. Immature oocysts are released from the epithelial cells into the intestinal lumen and excreted into the environment with the host's faeces. An infected cat sheds millions of oocysts into the environment over 1–2 weeks in its lifetime [23,24]. It is unlikely that cats will shed oocysts in the next infestation, because immunity is developed and oocysts are not formed [25,26].

Environment

Oocysts become invasive only after sporulation. This process always occurs in the external environment, at least after 24 hours, most often within 2–5 days, after excretion with cat's faeces, depending on temperature, humidity and oxygenation [20,27]. Each invasive oocysts contains two sporocysts with two sporozoites. Mature oocysts may remain invasive under favorable conditions, in soil for over a year, and in water, including sea water, up to 2 years [28–33].

Intermediate host

Over 350 species of birds and mammals, i.e. mice, rats, squirrels, bears, raccoons, foxes, rabbits and hares, beavers, deer, wild boars, and many other animals found locally, are intermediate hosts worldwide [12,34]. Among companion and farm animals, the most frequently mentioned are cats, dogs, ferrets, minks, horses, sheeps, goats, cattle, pigs, camels, llamas and alpacas, rabbits and birds such as turkeys, chickens, ducks, geese and pigeons [10,35,36]. Toxoplasma gondii infection has also been reported among wild birds, i.a. in representatives of the order Galliformes, including wild turkeys, pheasants, partridges, Anseriformes with various species of ducks and geese, Accipitriformes, Passeriformes, Columbiformes, Strigiformes, Charadriiformes and Gruiformes [10,34]. Infections of marine mammals [9,37–39], such as sea otters, seals [40,41], dolphins [42,43], and sea lions [44], are becoming more frequent in last decades.

The intermediate hosts become infected after ingesting plants or drinking water contaminated with sporulated oocysts [7,34]. Carnivores become

infected mainly by eating herbivores, which tissues containing tissue cysts with bradyzoites. The main intermediate host and reservoir of *T. gondii* are rodents [22,45], especially mice. Not only are they the main food of hunting cats, but infected individuals do not even avoid them, because the parasite modifies the behavior of rodents in such a way that the distance to escape is shortened, making them easier to prey to predators [46–49]. House mice play the most important role in the transmission of this parasite in the urban environment [22,50]. Another source is access carnivores to unprotected leftovers from slaughter process.

In the intermediate host stomach the sporozoites from the oocysts and the bradyzoites from the tissue cysts are released and transformed into tachyzoites. Tachyzoites intensively multiply in the reticuloendothelial system and in the epithelial cells of the small intestine, from where they spread through the blood and lymph throughout the body, penetrating subsequent cells in various tissues and organs. Intensively multiplying tachyzoites break the infected cell and release the next generation of tachyzoites, which infect other cells. This is the acute phase of invasion where inflammation arises. Then tachyzoite multiplication is inhibited, transformed into bradyzoites and tissue cysts are formed, most often in skeletal muscles and the brain [12]. In some animals, such as sheeps, intrauterine infection with tachyzoites has been documented, which can cause fetal death, miscarriage or neonatal death [24,51].

The presence of tachyzoites strongly stimulates the immune system response and there is a transition from the acute to the chronic phase. The multiplication of tachyzoites is inhibited and transformed into bradyzoites, forming tissue cysts. Tissue cysts are surrounded by their own envelope and then are surrounded by the connective tissue of the host, and over time they become saturated with calcium salts. A significant decrease in immunity can cause bradyzoites to convert to tachyzoites and cause reactivation [19,22].

The marine life cycle of T. gondii

For several years, infections among many species of marine mammals are becoming more frequent [38,39]. Seroprevalence and clinical symptoms of toxoplasmosis have been described primarily in the southern sea otters (*Enhydra lutris nereis*), but also seals [40,41], dolphins [42,43], sea

lions [44], porpoises, walruses and manatees [10]. In sea otters, numerous deaths due to infection have been observed [52].

The beginning of the increase in otters mortality was observed in the early 1990s. Staff of the National Wildlife Health Center (NWHC), Madison, Wisconsin, USA conducted a study of the dead animals in an attempt to determine the causes. *T. gondii* was isolated from tissues, especially from the brain and heart of the animals. In laboratory conditions, using mice and cats, it was shown that isolated parasites were capable of infecting and producing oocysts [53]. Similarly, high sero-prevalence is found in another subspecies *E. lutris kenyoni* living on the US East Coast [38].

The highest prevalence of antibodies against T. gondii is recorded in sea otters and varies depending on the region from 47% to 100%. High seroprevalence has also been reported in *Tursiops* truncatus in California and Florida waters [54,55], Erignathus barbatus and Pusa hispida in Arctic waters [56] or Leptonychotes weddellii and Mirounga leonina from Antarctica [57]. Toxoplasma gondii infection also has been found in other marine mammals like Sousa chinensis, Stenella longirostris, Stenella coeruleoalba, Tursiops aduncus, Grampus griseus, Delphinapterus leucas, Balaenoptera physalus [10], Odobenus rosmarus [58], Callorhinus ursinus [10], Phocarctos hookeri [44], Zalophus californianus [10], Cystophora cristata, Halichoerus grypus [40], Mirounga angustirostris [10], Neomonachus schauinslandi [10], Phoca largha [40], Phoca vituline [10], Pagophilus groenlandicus [40], Trichechus manatus [10].

In the waters around European continent *T. gondii* seroprevalence has been found in *Delphinus delphis* [59,60], *S. coeruleoalba*, *Tursiops truncates*, *Globicephala melas*, *Grampus griseus* [42,61–63], *Phocoena phocoena* [59,60], *Megaptera novaeangliae* [60], *Erignathus barbatus* [56], *P. vitulina* [40], *Pusa hispida* [56] and *H. grypus* [40].

Some of the infected sea mammals show symptoms of the central nervous system, behavioral changes, less frequently miscarriages, fetal and neonatal death, and congenital toxoplasmosis [64,65]. Cases of placental infection in sea otters [65], *T. aduncus* [64] and *G. griseus* [61] were observed. Symptomatic form of toxoplasmosis has been described in *S. chinensis*, *S. longirostris*, *S. coeruleoalba* and *T. truncatus* [10,63]. Documented fatal cases of toxoplasmosis except sea otter include

T. truncatus [66], P. vitulina, Z. californianus [10,67]. Michael et al. [44] indicate that infection with T. gondii may be the reason for the poor reproductive success observed in Phocarctos hookeri.

The diet of sea otters consists of marine invertebrates such as clams, sea snails, crabs and sea urchins [38]. This excludes the possibility of infection with tissue cysts because they occur only in warm-blooded animals. Infection with oocysts can occur either by swallowing them directly in contaminated water, or by eating invertebrates, e.g. filter feeding species that can accumulate oocysts in their body [24,68]. Conrad et al. [38] suggest the possibility of otters becoming infected by collecting and ingesting oocysts when they cleaning their fur.

Oocysts can get into the marine environment from land with flowing river waters, domestic wastewater or, more often, by surface runoff, especially in times of thaw or heavy rainfall [41,69]. Wild cats, e.g. pumas, lynxes, feral cats and, to a lesser extent, domestic cats contribute to contamination with dispersion forms of the terrestrial environment and further aquatic ecosystems [41,69,70].

In the marine environment, oocysts captured and accumulated by marine filter feeding invertebrates, e.g. clams, can then be transported through the food chain. The invertebrate bioaccumulation of invasive oocysts from contaminated water has been confirmed in the case of Mytilus californianus [68], Mytilus galloprovincialis, Crassostrea virginica [71–73], Crassostrea rhizophorae [74], Crassostrea gigas, Tapes decussatus [75], Dreissena polymorpha [76], Haliotis rufescens [77], Perna canaliculus [78,79], also marine snails like Chlorostoma and Promartynia [80]. Bioaccumulation of oocysts has also been found in the freshwater crustacean Gammarus fossarum [81]. This observation indicates that other crustaceans, including marine species, may have also the potential to accumulate oocysts and be able to transmit them through the food chain.

Infections with *T. gondii* also occur in other marine mammals, which, unlike sea otters, feed on fish or cephalopods. In their case contaminated water or fish appear to be the possible route of infection. Sea mammals e.g. dolphins drink little or no drink water [42]. Fish, like invertebrates, do not belong to intermediate hosts, because *T. gondii* parasitizes only in warm-blooded animals. Therefore, they can only be a vectors if they are able

to bioaccumulate in their gastrointestinal tract oocysts taken from the environment. Massie et al. [82] demonstrated this possibility in filter fish such as California anchovy (Engraulis mordax) and Pacific sardine (Sardinops sagax). These fish have the potential to be the vectors capable of invading T. gondii oocysts for approximately 8 hours after exposure. Marino et al. [83] found T. gondii DNA in some fish species like Engraulis encrasicolus, Boops boops, Trachurus trachurus, Mullus barbatus, Pagellus acarne, Pagellus erythrinus, Merluccius merluccius, Arnoglossus laterna, Diplodus sargus, Raja clavata, Scorpaena scrofa and Spicara maena, captured in Mediterranean area. Some of them are demersal species feeding on benthic invertebrates and other fish feeding on zooplankton or are omnivores.

Transmission of *Toxoplasma gondii* in the terrestrial and marine environment. Importance for public and environmental health.

Man can become infected with all invasive forms of T. gondii [2,69]. Most adults don't have clinical symptoms [24,55,84]. However, primary infection is dangerous in pregnant women due to the possibility of intrauterine infection of the fetus and the occurrence of congenital toxoplasmosis [12,24]. The group of people who may develop clinical symptoms also include people with immune deficiencies, especially those suffering from AIDS and treated with immunosuppressive drugs [12,13,19,85]. Especially this group of people (women of childbearing age and immunocompromised patients) should receive information about all potential sources of infection due to the still low public awareness of threats. Most often, the infection occurs through ingestion, by the placenta, less frequently iatrogenic or other routes (Fig. 1). Animals infected through ingestion or some of them via placenta, i.e. sheeps, goats, cattle [86-88], sea mammals [61,64,65].

The routes of infection in the terrestrial environment of animals and humans are well known in the scientific literature. However, in the common

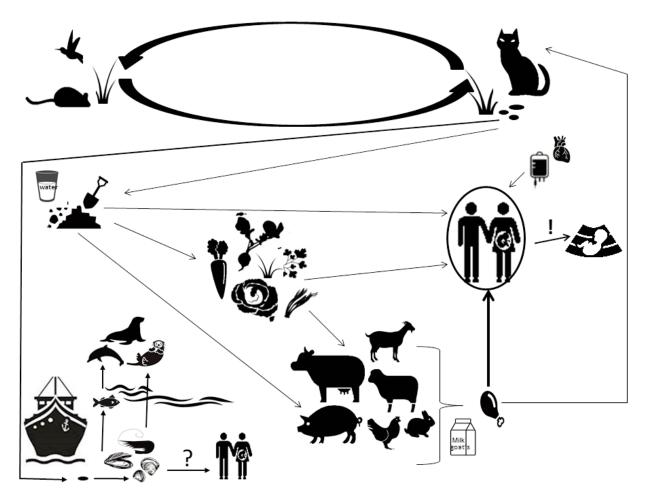


Figure 1. Potential sources of Toxoplasma gondii infection

knowledge of society, it is still not so obvious. For many people, infecting humans is equated with having a cat and not with the main route of infection, which is contaminated meat. The health risk associated with contamination of the marine environment with dispersive stages of parasites are even less known to the public.

Terrestrial routes of infection

People become infected primarily by eating raw or undercooked meat that contains tissue cysts, or by not following the rules of hygiene when preparing meals, e.g. knives, cutting boards and other kitchen utensils that have come into contact with raw meat are used in the processing of other foods. An important link in the epidemiology of toxoplasmosis is pork, lamb, goat, game and freerange poultry, e.g. chickens and ducks [27,34]. On the one hand, intensive breeding of animals (e.g. pigs, cattle, poultry) contributed to the reduction of animal contamination due to keeping them indoors, feeding them with processed fodder, controlling insects and rodents, limited entry of humans and animals, preventive vaccinations [89]. On the other hand, the fashion for ecological breeding, due to wider contact with the environment, may again increase the risk of meat contamination [84].

Fresh fruit and vegetables that have been contaminated with felids faeces can also pose a health risk if not carefully washed, peeled or cooked. Oocysts are most often found in carrots, lettuce, radishes [90], spinach (e.g. baby spinach) [91], raspberries and blueberries [92]. The importance of this source in the epidemiology of toxoplasmosis is demonstrated by the high seroprevalence among vegetarians [93]. Oocysts can be transferred to unprotected food by flies, cockroaches, caprophagic invertebrates, and earthworms [23].

Oocysts and parasite DNA are found in drinking water samples all over the world [37,94–97]. There are known cases of water-derived epidemics of toxoplasmosis related to the contamination of drinking water intake, e.g., in Victoria (British Columbia, Canada) [85], India, Brazil or Panama [12,13,34,55]. Oocysts are sensitive to temperature (cooking, freezing), gamma radiation and UV radiation [33,55,98,99].

Another risk factor is the consumption of raw, unpasteurized goat's milk or cheese [84,101–103]. Tachyzoites are also found in sheep's and cow's milk, but cases of toxoplasmosis have been reported

so far only after consuming goat's milk [104].

Infection can also occur through dirty hands or by drinking or choking water from open water bodies. Contamination of sandboxes and playgrounds for children is of particular epidemiological importance [12,23,100,101]. Contact with oocysts may also occur during gardening [83]. Oocysts are also commonly detected in freshwater and seawater, into which they get with sewage and surface runoff [31,41,43].

The risk of tachyzoites passing through the placenta and infection of the developing fetus exists in the case of primary infection in a woman during pregnancy, less frequently in the preconception period [12,105]. If a woman had contact with the parasite before, she developed immunity before reinfection. Similarly, undergoing toxoplasmosis in the first pregnancy does not pose a threat to a subsequent pregnancy. Reactivation in the case of a pregnant woman with immunosuppression may be an exception [106]. The risk of congenital toxoplasmosis changes with gestational age and is highest in the third trimester [15,106].

Rare cases of infection may occur during blood transfusion, through tachyzoites contained in blood or blood products (*e.g.* leukocyte concentrate), during transplantation together with transplanted organs or tissues [13], as heart, lung, bone marrow transplant [11,107].

Occasionally, accidental inoculation conjunctival infection may result in the infection of employees of diagnostic laboratories, while working with infectious material, or people performing procedures on sick animals [12,108]. Work in the meat industry (slaughterhouses, butchers), hunting, due to contact with carcasses of animals, their slaughter, skinning, carcass cutting and further processing, especially carcasses of pigs, sheeps, goats and game (deer, wild boars, hares, birds) and pigeons [35,45], is also indicated as a potential route of infection if occupational hygiene is not followed. In this case, infection can occur through dirty hands, conjunctiva or through injured skin.

Marine routes of infection

Contamination of the environment, including water, with various pathogens is a global public health and animal health problem [33]. The new problem is occurrence of *T. gondii* in marine environment and surface runoff is the most important factor influencing the contamination of

sea water and the possibility of infection of marine animals [33,41,69,70]. The presence of *T. gondii* in the marine environment creates local problems not only for the health of marine mammals, but also for terrestrial predators like polar bears hunting them [56,109] or small predators that can feed on carcass, *e.g.* arctic foxes and carnivorous birds in Arctic regions. In the far Arctic, where felids do not exist, migrating birds such as *Branta leucopsis* can be a second source which can infect at wintering grounds in the North Sea and Irish Sea [56,58].

The presence of *T. gondii* in marine ecosystems can pose both local and global problems for public health. An example of local problems can be arctic areas, where the habits of eating raw or dried seal and liver seal meat along with raw caribou meat and skinning of hunted animals (e.g. foxes, wolves, martens) are a source of infection in Canadian Inuit [13,58,69,104]. Eating raw oysters and clams is identified as a new global health risk [75,84,112]. Marine invertebrates such as molluscs and crustaceans act as transport hosts. Absorption of oocysts has been found in filtering invertebrates like molluses, such as abalone, oysters, blue mussels and other [11,68,74,75,77,90,110,111]. Crustaceans may also be a vectors, especially that G. fossarum, are capable of bioaccumulation of oocysts in their body [81]. Invertebrates are mainly responsible for the spread of toxoplasmosis among marine animals, but if eaten raw, undercooked, they can also be a risk factor for humans [38]. Coastal invertebrates are most vulnerable to contamination from surface runoff or sewage. If they demonstrate the ability to transmit oocysts from seawater to humans, they could become a global public health problem because of the popularity of shellfish dishes worldwide. Jones et al. [112] and Putignani et al. [75] indicate that eating raw seafood – oysters, blue mussels and other bivalve is now a new risk of T. gondii infection. Hence the warnings, especially in areas with high consumption of shellfish, that eating them should be avoided raw, but only after proper cooking. It is especially important that that people from risk groups,, such as pregnant women, immunocompromised people and those taking immunosuppressive drugs, be aware of the new potential risks. The possible risk from filter fish such as sardines and anchovies is negligible as, unlike invertebrates, because the fish are gutted prior to consumption.

Prevention summary

The risk of toxoplasmosis infection is primarily shaped by hygiene and eating habits. The main route of infection is undercooked meat, dirty hands and utensils, unwashed vegetables and fruit growing in/near the ground. The basis of our health is knowledge about the pathways of transmission of the parasite, adherence to basic hygiene rules and diagnostics. Personal hygiene and food preparation - frequent washing of hands with soap and water, especially before meals, after contact with raw meat, unwashed vegetables and fruit as well as soil and sand (garden, park, sandbox), taking care of the cleanliness of knives, cutting boards, dishes, kitchen worktops, washing them thoroughly with detergents after contact with raw meat, seafood and unwashed vegetables and fruit. Food should be protected against the access of insects (e.g. flies, cockroaches).

Food and drinking water – eating meat that has undergone proper heat treatment, cooking, stewing, frying at a minimum temperature of 67°C (e.g. using meat thermometers), deep-freezing meat at a temperature of at least -12°C for a minimum of 3 days [104]. Under refrigerated conditions (1–4°C), cysts in meat may remain invasive for up to 3 weeks [12]. Avoiding eating raw and semi-raw products, long-ripening or low-temperature smoked meats (e.g. tartare, Parma ham, salami), unwashed fruit and vegetables, and drinking unpasteurized milk. People at risk should also avoid eating raw oysters and other mussels, tasting food while cooking, and eating grilled meat that may not have reached the required temperature during cooking. Drinking only boiled or bottled water, especially when traveling to countries with low hygiene standards.

Toxoplasmosis is a disease around which a myriad of myths have emerged, like this fake that people can be infected directly from a cat. For this reason cat owners are concerned and have an unnecessary dilemma whether their cat threatens the health of their infants. Against, people who don't have daily contact with cats feel safe and they don't appreciate the role of proper hygiene rules and proper food processing reduces risk of infection. The presence of a cat in the immediate vicinity, with the observance of basic hygiene rules and regular cleaning of the litter box, does not pose a risk of infection with toxoplasmosis [12,113]. Cats that do not leave the house and are not fed raw meat have a negligible chance of becoming infected with the parasite and thus pose a threat to human health. Hunting cats, usually young, can only contribute to

environmental contamination with oocysts for a few weeks in their lifetime. Later they develop immunity and no oocyst sowing occurs. The litter box and its surroundings should be cleaned daily, while wearing protective gloves, and remember to wash your hands after completing these activities. For safety, if a pregnant woman or an immunocompromised person has a cat, they have someone else to clean the litter box.

- Environment work in the garden in protective gloves, protect sandboxes and playgrounds for children against animal access, always wash hands with soap and water after contact with soil and sand. Prevent geophagia in children. People with reduced immunity should avoid swimming in recreational waters (ponds, lakes, sea) that could be contaminated with oocytes from sewage or runoff. In order to limit the spread of *T. gondii*, the remaining carcass remains from the slaughterhouse must be kept away from domestic and wild animals [45].
- Pregnancy perform preventive blood tests for T. gondii. According to the Regulation of the Minister of Health of August 16, 2018 on the organizational standard of perinatal care, it is recommended to carry out the toxoplasmosis test (IgM, IgG) up to the 10th week of pregnancy unless the pregnant woman shows a result confirming the presence of pre-pregnancy IgG antibodies and in women with a negative results in the first trimester, repeat test (IgM) between 21 and 26 weeks of pregnancy [114]. Follow the rules of prevention, especially take care of personal and food hygiene, avoid eating unwashed vegetables and fruits, unpasteurized milk, unboiled water, avoid contact with raw meat (preferably using frozen meat), limit the consumption of raw seafood.
- Educational programs because it is one of the most common opportunistic infection in the world, it is important for public health specialists to disseminate updated educational materials concerning on the risk factors, routes of infection from terrestrial and marine environment, and prevention of toxoplasmosis, proper food processing and hygienic behaviors, aimed at young people, especially girls and women of childbearing age, as well as immuncompromised people.
- Environmental monitoring The presence of invasive forms of *T. gondii* in terrestrial and aquatic environments, including marine

environment, should be monitored. Especially the monitoring of invertebrates that could be potential vectors of *T. gondii* and the health of marine mammals.

References

- [1] Weiss L.M., Dubey J.P. 2009. Toxoplasmosis: a history of clinical observations. *International Journal of Parasitology* 39: 895-901. doi:10.1016/j.ijpara.2009.02.004
- [2] Morrissette N.S., Ajioka J.W. 2009. The early years of *Toxoplasma* research: What's past is prologue. *International Journal of Parasitology* 39: 865-869. doi:10.1016/j.ijpara.2009.02.010
- [3] Wolf A., Cowen D., Paige B.H. 1940. Toxoplasmic encephalomyelitis. IV. Experimental transmission of the infection to animals from a human infant. *Journal of Experimental Medicine* 71: 187-214.
- [4] Wolf A., Cowen D., Paige B.H. 1939.Toxoplasmic encephalomyelitis. III. A new case of granulomatous encephalomyelitis due to a protozoon. *The American Journal of Pathology* 15: 657-694.
- [5] Wolf A., Cowen D., Paige B.H. 1939. Human toxoplasmosis: occurrence in infants as an encephalomyelitis verification by transmission to animals. *Science* 89: 226-227. https://doi.org/10.1126/science.89.2306.226
- [6] Sabin A.B., Feldman H.A. 1948. Dyes as microchemical indicators of a new immunity phenomenon affecting a protozoon parasite (*Toxoplasma*). *Science* 108: 660-663.
 - https://doi.org/10.1126/science.108.2815.660
- [7] Dubey J.P. 2009. History of the discovery of the life cycle of *Toxoplasma gondii*. *International Journal of Parasitology* 39: 877-882. doi:10.1016/j.ijpara.2009.01.005
- [8] Ferguson D.J.P. 2009. Identification of faecal transmission of *Toxoplasma gondii*. Small science, large characters. *International Journal of Parasitology* 39: 871-875. doi:10.1016/j.ijpara.2009.01.003
- [9] Mazzillo F.F.M., Shapiro K., Silver M.W. 2013. A new pathogen transmission mechanism in the ocean: the case of sea otter exposure to the land-parasite *Toxoplasma gondii*. *PLoS ONE* 8: e82477. doi:10.1371/journal.pone.0082477
- [10] Lindsay D.S., Dubey J.P. 2014. Chapter 6. Toxoplasmosis in wild and domestic animals In: *Toxoplasma gondii. The model apicomplexan perspectives and methods.* (Eds. L.M. Weiss, K. Kim). 2nd ed. Academic Press: 193-215.
- [11] Flegr J., Prandota J., Sovičková M., Israili Z.H. 2014. Toxoplasmosis a global threat. Correlation of latent toxoplasmosis with specific disease burden in a set of 88 countries. *PLoS ONE* 9: e90203. doi:10.1371/journal.pone.0090203
- [12] Robert-Gangneux F., Darde M.L. 2012. Epidemio-

- logy of and diagnostic strategies for toxoplasmosis. *Clinical Microbiology Reviews* 25: 264-296. doi:10.1128/cmr.05013-11
- [13] Tenter A.M., Heckeroth A.R., Weiss L.M. 2000. *Toxoplasma gondii*: from animals to humans. *International Journal of Parasitology* 30: 1217-1258. https://doi.org/10.1016/s0020-7519(00)00124-7
- [14] Dabritz H.A., Conrad P.A. 2009. Cats and *Toxo-plasma*: implications for public health. *Zoonoses Public Health* 57: 34-52. doi:10.1111/j.1863-2378.2009.01273.x
- [15] Paquet C., Yudin M.H. 2013. Society of Obstetricians and Gynaecologists of Canada. Toxoplasmosis in pregnancy: prevention, screening, and treatment. *Journal of Obstetrics and Gynaecology Canada* 35: 78-81. doi:10.1016/s1701-2163(15)31053-7
- [16] Khan K., Khan W. 2018. Congenital toxoplasmosis: An overview of the neurological and ocular manifestations. *Parasitology International* 67: 715-721. doi:10.1016/j.parint.2018.07.004
- [17] European Centre for Disease Prevention and Control. Congenital toxoplasmosis. In: ECDC. Annual epidemiological report for 2018. ECDC; 2021 (Homepage | European Centre for Disease Prevention and Control (europa.eu)
- [18] European Centre for Disease Prevention and Control. Annual epidemiological report for 2014 – Toxoplasmosis. ECDC; 2016 Homepage | European Centre for Disease Prevention and Control (europa.eu)
- [19] Dubey J.P., Lindsay D.S., Speer C.A. 1998. Structures of *Toxoplasma gondii* tachyzoites, bradyzoites, and sporozoites and biology and development of tissue cysts. *Clinical Microbiology Reviews* 11: 267-299.
- [20] Dubey J.P. 1998. Advances in the life cycle of *Toxoplasma gondii*. *International Journal of Parasitology* 28: 1019-102. https://doi.org/10.1016/S0020-7519(98)00023-X
- [21] Montazeri M., Galeh M.T., Moosazadeh, M., Sarvi S., Dodangeh S., Sharif M., Daryari A. 2020. The global serological prevalence of *Toxoplasma gondii* in felids during the last five decades (1967–2017): a systematic review and meta-analysis. *Parasites & Vectors* 13: 82.
 - https://doi.org/10.1186/s13071-020-3954-1
- [22] Yan C., Liang L.J., Zhang B.B., Lou Z.L., Zhang H.F., Shen X., Wu Y.Q., Wang Z.M., Tang R.X., Fu L.L., Zheng K.Y. 2014. Prevalence and genotyping of *Toxoplasma gondii* in naturally-infected synanthropic rats (*Rattus norvegicus*) and mice (*Mus musculus*) in eastern China. *Parasites & Vectors* 7: 591. doi:10.1186/s13071-014-0591-6
- [23] Hill D.E., Dubey J.P. 2002. *Toxoplasma gondii*: transmission, diagnosis and prevention. *Clinical Microbiology and Infection* 8: 634-640. doi:10.1046/j.1469-0691.2002.00485.x

- [24] Dubey J.P., Jones J.L. 2008. *Toxoplasma gondii* infection in humans and animals in the United States. *International Journal of Parasitology* 38: 1257-1278. https://doi.org/10.1016/j.ijpara.2008.03.007
- [25] Dubey J.P., Frenkel J.K. 1974. Immunity to feline toxoplasmosis: modification by administration of corticosteroids. *Veterinary Pathology* 11: 350-379. https://doi.org/10.1177/030098587401100407
- [26] Doubey J.P. 1995. Duration of immunity to shedding of *Toxoplasma gondii* oocysts by cats. *Journal of Parasitology* 81: 410-415.
- [27] Hill D.E., Dubey J.P. 2016. Toxoplasma gondii as a parasite in food: analysis and control. Microbiology Spectrum 4: PFS-0011-2015. doi:10.1128/microbiolspec
- [28] Lindsay D.S., Blagburn B.L., Dubey J.P. 2002. Survival of nonsporulated *Toxoplasma gondii* oocysts under refrigerator conditions. *Veterinary Parasitology* 103: 309-313. https://doi.org/10.1016/s0304-4017(01)00554-4
- [29] Dumetre A., Darde M.L. 2003. How to detect *Toxoplasma gondii* oocysts in environmental samples? *FEMS Microbiology Reviews* 27: 651-661.
- [30] Dabritz H.A., Conrad P.A. 2009. Cats and *Toxo-plasma*: Implications for public health. *Zoonoses and Public Health* 57:34-52. doi:10.1111/j.1863-2378.2009.01273.x
- [31] Lindsay D.S., Dubey J.P. 2009. Long-term survival of *Toxoplasma gondii* sporulated oocysts in seawater. *Journal of Parasitology* 95: 1019-1020. doi:10.1645/GE-1919.1
- [32] Tenter A.M., Heckeroth A.R., Weiss L.M. 2000. *Toxoplasma gondii*: from animals to humans. *International Journal of Parasitology* 30: 1217-1258. https://doi.org/10.1016/s0020-7519(00)00124-7
- [33] VanWormer E., Fritz H., Shapiro K., Mazet J.A., Conrad P.A. 2013. Molecules to modeling: *Toxoplasma gondii* oocysts at the human-animal-environment interface. *Comparative Immunology, Microbiology and Infectious Diseases* 36: 217-231. doi:10.1016/j.cimid.2012.10.006
- [34] Dubey J.P. 2002. A review of toxoplasmosis in wild birds. *Veterinary Parasitology* 106: 121-153. https://doi.org/10.1016/s0304-4017(02)00034-1
- [35] Witkowski L., Czopowicz M., Nagy D.A. et al. 2015. Seroprevalence of *Toxoplasma gondii* in wild boars, red deer and roe deer in Poland. *Parasite* 22: 1-4. https://dx.doi.org/10.1051/parasite%2F2015017
- [36] Dubey J.P., Lindsay D.S., Lappin M.R. 2009. Toxoplasmosis and other intestinal coccidial infections in cats and dogs. *Veterinary Clinics of North America: Small Animal Practice* 39: 1009-1034. doi:10.1016/j.cvsm.2009.08.001
- [37] Dubey J.P. 2004.Toxoplasmosis a waterborne zoonosis. *Veterinary Parasitology* 126: 57-72. https://doi.org/10.1016/j.vetpar.2004.09.005
- [38] Conrad P.A, Miller M.A, Kreuder C., James E.R.,

- Mazet J., Dabritz H., Jessup D.A., Gulland F., Grigg M.E. 2005. Transmission of *Toxoplasma*: Clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. *International Journal of Parasitology* 35: 1155-1168. https://doi.org/10.1016/j.ijpara.2005.07.002
- [39] Jensen S.K., Nymo I.H., Forcada J., Godfroid J., Hall A. 2012. Prevalence of *Toxoplasma gondii* antibodies in pinnipeds from Antarctica. *Veterinary Record* 171: 249-250. doi:10.1136/vr.100848
- [40] Cabezon O., Hall A.J., Vincent C., Pabón M., García-Bocanegra I., Dubey J.P., Almería S. 2011. Seroprevalence of *Toxoplasma gondii* in Northeastern Atlantic harbor seal (*Phoca vitulina vitulina*) and grey seal (*Halichoerus grypus*). *Veterinary Parasitology* 179: 253-256. https://doi.org/10.1016/j.vetpar.2011.01.046
- [41] VanWormer E., Carpenter T.E., Singh P., Shapiro K., Wallender W.W., Conrad P.A., Largier J.L., Maneta M.P., Mazet J.A. 2016. Coastal development and precipitation drive pathogen flow from land to sea: evidence from a *Toxoplasma gondii* and felid host system. *Scientific Reports* 6: 29252. doi:10.1038/srep29252
- [42] Pretti C., Mancianti F., Nardoni S., Ariti G., Monni G., di Bello D., Marsili S., Papini R. 2010. Detection of *Toxoplasma gondii* infection in dolphins stranded along the Tuscan coast, Italy. *Revue de Médecine Vétérinaire* 161: 428-431.
- [43] Fayer R., Dubey J.P., Lindsay D.S. 2004. Zoonotic protozoa: from land to sea. *Trends in Parasitology* 20: 531-536. https://doi.org/10.1016/j.pt.2004.08.008
- [44] Michael S.A. Howe L., Chilvers B.L., Morel P.C.H., Roe W. 2016. Seroprevalence of Toxoplasma gondii in mainland and sub-Antarctic New Zealand sea lion (Phocarctos hookeri) populations. New Zealand Veterinary Journal 64: 293-297.
- [45] Turčeková L., Hurníková Z., Spišák F., Miterpáková M., Chovancová B. 2014. Toxoplasma gondii in protected wildlife in the Tatra National Park (TANAP), Slovakia. Annals of Agricultural and Environmental Medicine 21: 235-238.
- [46] Hrdá S., Votýpka J., Kodym P., Flegr J. 2000. Transient nature of *Toxoplasma gondii* - induced behavioral changes in mice. *Journal of Parasitology* 86: 657-663. doi:10.1645/0022-3395(2000)086
- [47] Gatkowska J., Wieczorek M., Dziadek B., Dzitko K., Dlugońska H. 2012. Behavioral changes in mice caused by *Toxoplasma gondii* invasion of brain. *Parasitology Research* 111: 53-58. http://dx.doi.org/10.1007/s00436-011-2800-y
- [48] Webster J.P. 2001. Rats, cats and parasites: the impact of latent toxoplasmosis on behaviour. *Microbes and Infection* 3: 1037-1045. doi:10.1016/s1286-4579(01)01459-9
- [49] Kannan G., Moldovan K., Xiao J.Ch. et al. 2010. *Toxoplasma gondii* strain-dependent effects on mouse

- behaviour. Folia Parasitologica 57: 151-155.
- [50] Murphy G., Oldbury D., Hughes J., Hide G., Thomasson D., Williams H. 2008. Role of urban mice in transmission of *Toxoplasma gondii*. Proceedings of the Sixth International Conference on Urban Pests Budapest, Hungary, 13-16 July 2008. (Eds. W.H. Robinson, D. Bajomi): 319-323.
- [51] Hill D.E., Chirukandoth S., Dubey J.P. 2005. Biology and epidemiology of *Toxoplasma gondii* in man and animals. *Animal Health Research Reviews* 6: 41-61.
- [52] Miller M.A., Grigg M.E., Kreuder C., James E.R., Melli A.C., Crosbie P.R., Jessup D.A., Boothroyd J.C., Brownstein D., Conrad P.A. 2004. An unusual genotype of *Toxoplasma gondii* is common in California sea otters (*Enhydra lutris nereis*) and is a cause of mortality. *International Journal of Parasitology* 34: 275-284. doi:10.1016/j.ijpara.2003.12.008
- [53] Cole R.A., Lindsay D.S., Howe D. K., Roderick C.L., Dubey J.P., Thomas N.J., Baeten L.A. 2000. Biological and molecular characterizations of *Toxoplasma gondii* strains obtained from southern sea otters (*Enhydra lutris nereis*). *Journal of Parasitology* 86: 526-530. https://doi.org/10.1645/0022-3395(2000)086[0526: bamcot]2.0.co;2
- [54] Miller M.A., Gardner I.A., Kreuder C., Paradies D.M., Worcester K.R., Jessup D.A., Dodd E., Harris M.D., Ames J.A., Packham A.E., Conrad P.A. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (Enhydra lutris nereis). International Journal of Parasitology 32: 997-1006. https://doi.org/10.1016/s0020-7519(02)00069-3
- [55] Jones J.L., Dubey J.P. 2010. Waterborne toxoplasmosis – Recent development. *Experimental Parasitology* 124: 10-25. https://doi.org/10.1016/j.exppara.2009.03.013
- [56] Jensen S.K., Aars J., Lydersen C., Kovacs K.M., Asbakk K. 2010. The prevalence of *Toxoplasma gondii* in polar bears and their marine mammal prey: Evidence for a marine transmission pathway? *Polar Biology* 33: 599-606. doi.org/10.1007/s00300-009-0735-x
- [57] Jensen S.K., Nymo H., Forcada J., Godfroid J., Hall A. 2012. Prevalence of *Toxoplasma gondii* antibodies in pinnipeds from Antarctica. *Veterinary Record* 171: 249-250.
- [58] Simon A., Chambellan T.M., Ward B.J., Simard M., Proulx J.F., Levesque B., Bigras-Poulin M., Rousseau A.N., Ogden N.H. 2011. Spatio-temporal variations and age effect on *Toxoplasma gondii* seroprevalence in seals from the Canadian Arctic. *Parasitology* 138: 1362-1368.
- [59] Van De Velde N., Devleesschauwer B., Leopold M., Begeman L., Ijsseldijk L., Hiemstra S., Ijzer J.,

- Brownlow A., Davison N., Haelters J., Jauniaux T., Siebert U., Dorny P., De Craeye S. 2016. *Toxoplasma gondii* in stranded marine mammals from the North Sea and Eastern Atlantic Ocean: Findings and diagnostic difficulties. *Veterinary Parasitol*ogy 230: 25-32. http://dx.doi.org/10.1016/j.vetpar.2016.10.021
- [60] Forman D., West N., Francis J., Guy E. 2009. The sero-prevalence of *Toxoplasma gondii* in British marine mammals. *Memórias do Instituto Oswaldo Cruz* 104: 296-298.
 - http://dx.doi.org/10.1590/S0074-02762009000200024
- [61] Resendes A.R., Almeria S., Dubey J.P., Obon E., Juan-Salles C., Degollada E., Algre F., Cabezon O., Pont S., Domingo M. 2002. Disseminated toxoplasmosis in a Mediterranean pregnant Risso's dolphin (*Grampus griseus*) with transplacental fetal infection. *Journal of Parasitology* 88: 1029-1032. doi:10.1645/0022-3395(2002)088[1029:dtiamp]2.0. co:2
- [62] Cabezón O., Resendes A.R., Domingo M., Raga J.A., Agustí C., Alegre F., Mons J.L., Dubey J.P., Almería S. 2004. Seroprevalence of *Toxoplasma* gondii antibodies in wild dolphins from the Spanish Mediterranean Coast. *Journal of Parasitology* 90: 643-644. https://doi.org/10.1645/ge-257r
- [63] Di Guardo G., Proietto U., Di Francesco C.E., Marsilio F., Zaccaroni A., Scaravelli D., Mignone W., Garibaldi F., Kennedy S., Forster F., Iulini B., Bozzetta E., Casalone C. 2010. Cerebral toxoplasmosis in striped dolphins (Stenella coeruleoalba) stranded along the Ligurian Sea coast of Italy. Veterinary Pathology 47: 245-253. https://doi.org/10.1177/0300985809358036
- [64] Jardine J.E., Dubey J.P. 2002. Congenital toxoplasmosis in Indo-Pacific bottlenose dolphin (*Tursiops aduncus*). *Journal of Parasitology* 88: 197-199. https://doi.org/10.1645/0022-3395(2002)088[0197:
- [65] Miller M., Conrad P., James E.R., Packham A., Toy-Choutka S., Murray M.J., Jessup D., Grigg M. 2008. Transplacental toxoplasmosis in a wild sea otter (*Enhydra lutris nereis*). *Veterinary Parasitology* 153: 12-18. https://doi.org/10.1016/j.vetpar.2008.01.015

CTIAIP]2.0.CO;2

- [66] Inskeep W., Gardiner C.H., Harris R.K., Dubey J.P., Goldston R.T. 1990. Toxoplasmosis in Atlantic Bottle-Nosed Dolphins (*Tursiops truncatus*). *Journal of Wildlife Diseases* 26: 377-382.
- [67] Carlson-Bremer D., Colegrove K.M., Gulland F.M.D., Conrad P.A., Mazet J.A.K., Johnson C.K. 2015. Epidemiology and pathology of *Toxoplasma* gondii in free-ranging california sea lions (Zalophus californianus). Journal of Wildlife Diseases 51: 362-373.
- [68] Miller M.A., Miller W.A., Conrad P.A., James E.R., Melli A.C., Leutenegger C.M., Dabritz H.A., Packham A.E., Paradies D., Harris M., Ames J., Jessup D.A., Worcester K., Grigg M.E. 2008. Type X

- Toxoplasma gondii in a wild mussel and terrestrial carnivores from coastal California: new linkages between terrestrial mammals, runoff and toxoplasmosis of sea otters. International Journal of Parasitology 38: 1319-1328. https://doi.org/10.1016/j.ijpara.2008.02.005
- [69] Simon A., Poulin M.B., Rousseau A.N., Ogden N.H. 2013. Fate and transport of *Toxoplasma gondii* ocysts in seasonally snow covered watersheds: a conceptual framework from a melting snowpack to the Canadian Arctic Coasts. *International Journal of Environmental Research and Public Health* 10: 994-1005.
- [70] Vanwormer E., Conrad P.A., Miller M.A., Melli A.C., Carpenter T.E., Mazet J.A.K. 2013. *Toxoplasma gondii*, source to sea: higher contribution of domestic felids to terrestrial parasite loading despite lower infection prevalence. *EcoHealth* 10: 277-289.
- [71] Lindsay D.S., Phelps K.K., Smith S.A., Flick G., Sumner S.S., Dubey J.P. 2001. Removal of Toxoplasma gondii oocysts from sea water by Eastern Oysters (Crassostrea virginica). Journal of Eukaryotic Microbiology 48: 197s-198s. https://doi.org/10.1111/j.1550-7408.2001.tb00517.x
- [72] Lindsay D.S., Collins M.V., Mitchell S.M., Wetch C.N., Rosypal A.C., Flick G.J., Zajac A.M., Lindquist A., Dubey J.P. 2004. Survival of *Toxoplasma gondii* oocysts in eastern oysters (*Crassostrea virginica*). *Journal of Parasitology* 90: 1054-1057. doi:10.1645/ge-296r
- [73] Arkush K.D., Miller M.A., Leutenegger C.M., Gardner I.A., Packham A.E., Heckeroth A.R., Tenter A.M., Barr B.C., Conrad P.A. 2003. Molecular and bioassay-based detection of *Toxoplasma gondii* oocyst uptake by mussels (Mytilus galloprovincialis). International Journal of Parasitology 33: 1087-1097. https://doi.org/10.1016/s0020-7519(03)00181-4
- [74] Esmerini P.O., Gennari S.M., Pena H.F.J. 2010. Analysis of marine bivalve shellfish from the fish market in Santos city, Sao Paulo state, Brazil, for *Toxoplasma gondii. Veterinary Parasitol*ogy 170: 8-13. https://doi.org/10.1016/j.vetpar.2010.01.036
- [75] Putignani L., Mancinelli L., Del Chierico F., Menichella D., Adlerstein D., Angelici M.C., Marangi M., Berrilli F., Caffara M., di Regalbono D.A., Giangaspero A. 2011. Investigation of *Toxoplasma* gondii presence in farmed shellfish by nested-PCR and real-time PCR fluorescent amplicon generation assay (FLAG). Experimental Parasitology 127: 409-417. https://doi.org/10.1016/j.exppara.2010.09.007
- [76] Palos Ladeiro M., Bigot-Clivot A., Aubert D., Villena I., Geffard A. 2015. Assessment of *Toxo-plasma gondii* levels in zebra mussel (*Dreissena polymorpha*) by real-time PCR: an organotropism study. *Environmental Science and Pollution Research* 22: 13693-13701.doi:10.1007/s11356-015-4296-y
- [77] Schott K.C., Krusor C., Tinker M.T., Moore J., Conrad P.A., Shapiro K. 2016. Concentration and

- retention of *Toxoplasma gondii* surrogates from seawater by red abalone *(Haliotis rufescens)*. *Parasitology* 143: 1703-1712. doi:10.1017/s0031182016001359
- [78] Coupe A., Howe L., Burrows E., Sine A., Pita A., Velathanthiri N., Vallée E., Hayman D., Shapiro K., Roe W.D. 2018. First report of *Toxoplasma gondii* sporulated oocysts and *Giardia duodenalis* in commercial green-lipped mussels (*Perna canaliculus*) in New Zealand. *Parasitology Res*earch 117: 1453-1463. doi:10.1007/s00436-018-5832-8
- [79] Coupe A., Howe L., Shapiro K., Roe W.D. 2019. Comparison of PCR assays to detect *Toxoplasma gondii* oocysts in green-lipped mussels (*Perna canaliculus*). *Parasitology Research* 118: 2389-2398. doi:10.1007/s00436-019-06357-z
- [80] Krusor C., Smith W.A., Tinker M.T., Silver M., Conrad P.A., Shapiro K. 2015. Concentration and retention of *Toxoplasma gondii* oocysts by marine snails demonstrate a novel mechanism for transmission of terrestrial zoonotic pathogens in coastal ecosystems. *Environmental Microbiology* 17: 4527-4537.
 - http://dx.doi.org/10.1111/1462-2920.12927
- [81] Bigot-Clivot A., Palos Ladeiro M., Lepoutre A., Bastien F., Bonnard I., Dubey J.P., Villena I., Aubert D., Geffard O., François A., Geffard A. 2016. Bioaccumulation of *Toxoplasma* and *Cryptosporidium* by the freshwater crustacean *Gammarus fossarum*: involvement in biomonitoring surveys and trophic transfer. *Ecotoxicology and Environmental Safety* 133: 188-194.
- https://doi.org/10.1016/j.ecoenv.2016.07.006 [82] Massie G.N., Ware M.W., Villegas E.N., Black M.W.
- [82] Massie G.N., Ware M.W., Villegas E.N., Black M.W. 2010. Uptake and transmission of *Toxoplasma gondii* oocysts by migratory, filter-feeding fish. *Veterinary Parasitology* 169: 296-303.
 - https://doi.org/10.1016/j.vetpar.2010.01.002
- [83] Marino A.M.F., Giunta R.P., Salvaggio A., Castello A., Alfonzetti T., Barbagallo A., Aparo A., Scalzo F., Reale S., Buffolano W., Percipalle M. 2019. *Toxoplasma gondii* in edible fishes captured in the Mediterranean basin. *Zoonoses and Public Health*. 66: 826-834. doi:10.1111/zph.12630
- [84] Jones J.L., Dubey J.P. 2012. Foodborne toxoplasmosis. Clinical Infectious Diseases 55: 845-851. https://doi.org/10.1093/cid/cis508
- [85] Alfonso Y., Fraga J., Jiménez N., Fonseca C., Dorta-Contreras A.J., Cox R., Capó V., Bandera F., Pomier O., Ginorio D. 2009. Detection of *Toxoplasma gondii* in cerebrospinal fluid from AIDS patients by nested PCR and rapid identification of type I allele at B1 gene by RFLP analysis. *Experimental Parasitology* 122: 203-207. doi:10.1016/j.exppara.2009.03.009
- [86] Esteban-Redondo I., Maley S.W., Thomson K., Nicoll S., Wright S., Buxton D., Innes E.A. 1999. Detection of *T. gondii* in tissues of sheep and cattle

- following oral infection. *Veterinary Pathology* 86: 155-171. doi:10.1016/s0304-4017(99)00138-7
- [87] Katzer F., Brülisauer F., Collantes-Fernández E., Bartley P.M., Gunn G., Maley S.W., Cousens C., Innes E.A. 2011. Increased *Toxoplasma gondii* positivity relative to age in 125 Scottish sheep flocks; evidence of frequent acquired infection. *Veterinary Research*. 42: 121. doi:10.1186/1297-9716-42-121
- [88] Dubey J.P., Murata F.H.A., Cerqueira-Cézar C.K., Kwok O.C.H., Su C. 2020. Economic and public health importance of *Toxoplasma gondii* infections in sheep: 2009-2020. *Veterinary Parasitology* 286: 109195. doi:10.1016/j.vetpar.2020.109195
- [89] Davies P.R. 2011. Intensive swine production and pork safety. *Foodborne Pathogens and Disease* 8: 189-201. doi:10.1089/fpd.2010.0717
- [90] Lass A., Pietkiewicz H., Szostakowska B., Myjak P. 2012. The first detection of *Toxoplasma gondii* DNA in environmental fruits and vegetables samples. *European Journal of Clinical Microbiology and Infectious Diseases* 31: 1101-1108.
- [91] Lalonde L.F., Gajadhar A.A. 2016. Detection of *Cyclospora cayetanensis, Cryptosporidium* spp., and *Toxoplasma gondii* on imported leafy green vegetables in Canadian survey. *Food Waterborne Parasitology* 2: 8-14. doi:10.1016/j.fawpar.2016.01.001
- [92] Kniel K.E., Lindsay D.S., Sumner S.S., Hackney C.R., Pierson M.D., Dubey J.P. 2002. Examination of attachment and survival of *Toxoplasma gondii* oocysts on raspberries and blueberries. *Journal of Parasitology* 88: 790-793. https://doi.org/10.1645/0022-3395(2002)088[0790:
- [93] Hall S.M., Pandit A., Golwilkar A., Williams T.S. 1999. How do Jains get toxoplasma infection? *Lancet* 354: 486-487. doi:10.1016/s0140-6736(99)02587-8

eoaaso]2.0.co;2

- [94] Bowie W.R., King A.S., Werker D.H., Isaac-Renton J.L., Bell A., Eng S.B., Marion S.A. 1997. Outbreak of toxoplasmosis associated with municipal drinking water. For the British Columbia Toxoplasma Investigation Team. *Lancet* 350: 173-177. https://doi.org/10.1016/s0140-6736(96)11105-3
- [95] Wainwright K.E., Lagunas-Solar M., Miller M.A., Barr B.C., Gardner I.A., Pina C., Melli A.C., Packham A.E., Zeng N., Truong T., Conrad P.A. 2007. Physical inactivation of *Toxoplasma gondii* oocysts in water. *Applied and Environmental Microbiology* 73: 5663-5666. https://doi.org/10.1128/aem.00504-07
- [96] Sroka J., Wojcik-Fatla A., Dutkiewicz J. 2006. Occurrence of *Toxoplasma gondii* in water from wells located on farms. *Annals of Agricultural and Environmental Medicine* 13: 169-175.
- [97] Wells B., Shaw H., Innocent G., Guido S., Hotchkiss E., Parigi M., Opsteegh M., Green J., Gillespie S.,

- Innes E.A., Katzer F. 2015. Molecular detection of Toxoplasma gondii in water samples from Scotland and a comparison between the 529bp real-time PCR and ITS1 nested PCR. Water Research 87: 175-181. http://dx.doi.org/10.1016/j.watres.2015.09.015
- [98] Wainwright K.E., Lagunas-Solar M., Miller M.A., Barr B.C., Gardner I.A., Pina C., Melli A.C., Packham A.E., Zeng N., Truong T., Conrad P.A. 2007. Physical inactivation of *Toxoplasma gondii* oocysts in water. Applied and Environmental Microbiology 73: 5663-5666. https://doi.org/10.1128/aem.00504-07
- [99] Ware M.W., Augustine S.A..J., Erisman D.O., See M.J., Wymer L., Hayes S.L., Dubey J.P., Villegas E.N. 2010. Determining UV inactivation of Toxoplasma gondii oocysts by using cell culture and a mouse bioassay. Applied and Environmental Microbiology 76: 5140-5147.
 - https://doi.org/10.1128/aem.00153-10
- [100] Lass A., Pietkiewicz H., Modzelewska E., Dumètre A., Szostakowska B., Myjak P. 2009. Detection of Toxoplasma gondii oocysts in environmental soil samples using molecular methods. European Journal of Clinical Microbiology and Infectious Diseases 28: 599-605.https://doi.org/10.1007/s10096-008-0681-5
- [101] Torrey E.F., Yolken R.H. 2013. Toxoplasma oocysts as a public health problem. Trends in Parasitology 29: 380-384. https://doi.org/10.1016/j.pt.2013.06.001
- [102] Dubey J.P., Verma S.K., Ferreira L.R., Oliveira S., Cassinelli A.B., Ying Y., Kwok O.C., Tuo W., Chiesa O.A, Jones J.L. 2014. Detection and survival of Toxoplasma gondii in milk and cheese from experimentally infected goats. Journal of Food Protection 77: 1747-1753.
 - doi:10.4315/0362-028X.JFP-14-167
- [103] Riemann H.P., Meyer M.E., Theis J.H., Kelso G., Behymer D.E. 1975. Toxoplasmosis in an infant fed unpasteurized goat milk. Journal of Pediatrics 87: 573-576. doi:10.1016/s0022-3476(75)80825-0
- [104] Tenter A. 2009. Toxoplasma gondii in animals used for human consumption. Memórias do Instituto Oswaldo Cruz 104: 364-369. https://doi.org/10.1590/s0074-02762009000200033
- [105] Długońska H. 2007. Transmisja pierwotniaków Toxoplasma gondii i Neospora caninum przez łożysko. Wiadomości Parazytologiczne 53: 9-13 (in Polish with summary in English).
- [106] Biedermann K., Flepp M., Fierz W., Joller-Jemelka H., Kleihues P. 1995. Pregnancy, immunosupression and reactivation of latent toxoplasmosis. Journal of

- Perinatal Medicine 23: 191-204. https://doi.org/10.1515/jpme.1995.23.3.191
- [107] Villard O., Cimon B., L'Ollivier C., Fricker-Hidalgo H., Godineau N., Houze S., Paris L., Pelloux H., Villena I., Candolfi E. 2016. Serological diagnosis of Toxoplasma gondii infection. Recommendations from the French National Reference Center for Toxoplasmosis. Diagnostic Microbiology Infectious Disease 84: 22-33. doi.org/10.1016/j.diagmicrobio.2015.09.009
- [108] Herwaldt B.L. 2001. Laboratory-acquired parasitic infections from accidental exposures. Clinical Microbiology Reviews 14: 659-688.
- [109] Oksanen A., Asbakk K., Prestrud K.W., Aars J., Derocher A.E., Tryland M., Wiig O., Dubey J.P., Sonne C., Dietz R., Andersen M, Born E.W. 2009. Prevalence of antibodies against Toxoplasma gondii in polar bears (Ursus maritimus) from Svalbard and East Greenland. Journal of Parasitology 95: 89-94. doi:10.1645/GE-1590.1
- [110] Graczyk T.K., Girouard A.S., Tamang L., Nappier S.P., Schwab K.J. 2006. Recovery, bioaccumulation, and inactivation of human waterborne pathogens by the Chesapeake Bay nonnative oyster, Crassostrea ariakensis. Applied and Environmental Microbiology 72: 3390-3395.
 - https://doi.org/10.1128/aem.72.5.3390-3395.2006
- [111] Ward J.E., Kach D.J. 2009. Marine aggregates facilitate ingestion of nanoparticles by suspensionfeeding bivalves. Marine Environmental Research 68: 137-142.
- [112] Jones J.L., Dargelas V., Roberts J., Press C., Remington J.S., Montoya J.G. 2009. Risk factors for Toxoplasma gondii infection in the United States. Clinical Infectious Diseases 49: 878-884. https://doi.org/10.1086/605433
- [113] Cook A.J.C., Gilbert R.E., Buffolano W., Zufferey J., Petersen E., Jenum P.A., Foulon W., Semprini A.E., Dunn D.T. 2000. Sources of Toxoplasma infection in pregnant women: European multicentre casecontrol study. BMJ 321: 142-147. doi:10.1136/bmj.321.7254.142
- [114] Rozporządzenie Ministra Zdrowia z dnia 16 sierpnia 2018 r. w sprawie standardu organizacyjnego opieki okołoporodowej. Dziennik Ustaw 2018, poz. 1756.

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