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Source: Journal of Wildlife Diseases, 6(4) : 305-312

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-6.4.305

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Toxoplasmosis: Epidemiology and Medical Importance

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Abstract

Developmental stages of *T. gondii* are described. The recent studies on fecal transmission of *T. gondii* are reviewed with the suggestion explored that *T. gondii* is a coccidian parasite which produces an intestinal cyst previously named *Isopora bigemina,* the intestinal reproductive stages of which are specific for the felines.

Human illness associated with this parasite is reviewed and its importance described.

Toxoplasma gondii remained a parasitological curiosity for 30 years after its discovery, in 1908, independently by Nicolle and Manceaux in Tunis and by Splendore in Brazil. Many more reports of its occurrence in animals were published, and for the parasite in each host a new specific name was proposed. Eventually, as a result of cross-infection studies, it became clear that this protozoan is much less specific than most organisms in the Sporozoa, the group it most resembled. Only one name, *T. gondii,* remained valid.

After its discovery in man, *Toxoplasma* attracted somewhat more attention as a pathogenic agent. The relation of toxoplasmosis in lower animals to the human infection became the concern of various investigators, and a considerable body of knowledge was built upon the prevalence of the organism.

Toxoplasma gondii is, on the basis of these reports, a highly successful parasite. It is world-wide in distribution, except for Antarctica. As I have pointed out in previous publications, the epidemiological clues we have had gave us little help in regard to its mechanisms of transmission. It is more prevalent in warm moist climates than in hot dry areas, but it exists abundantly throughout the temperate zone as well. Because it occurs in so many different situations, it has been difficult to identify regional or area characteristics which would help us in determining its methods of spread in human and animal populations. As we discuss the stages of *Toxoplasma gondii* we will see that there is still a great deal of epidemiological work to do to assess their relative importance, in various locales, in the dissemination of infection.

The history of the recent development of knowledge concerning the stages of *T. gondii* is itself so interesting and exciting that I could dwell on it throughout the remainder of this paper. However, I will try to go through it quickly in order to leave time for the discussion of the other subjects I should cover. Let us go back prior to 1965, to set the stage.

At that time we knew two different stages of *Toxoplasma gondii.* One of these, which I designated the trophozoite, is an actively growing and multiplying form which requires an intracellular environment. It reproduces by a process called endodyogeny, first described by Goldman, et al.[11] on the basis of silver-stained specimens and confirmed and extended in detail by Gavin, Wanko, and Jacobs[10] by

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electron microscopy, and subsequently by other ultra-micrographic studies.[21,22] Endodyogeny is a process of cytoplasmic reorganization preceding division of the nucleus. It appears to be a form of schizogony in which only two daughter organisms are formed. Its discovery itself hastened acceptance of the idea that *T. gondii* is a sporozoan; previous observations based on light microscopy had suggested longitudinal fission as the method of reproduction and shed doubt on the systematic position of the organism.

The trophozoite is a very delicate organism. It survives only a short time outside of cells, even in nutrient media. It is killed by changes in osmotic pressure, by drying, freezing, exposure to pepsin, and so on. Because it is so labile, it was considered unlikely that the trophozoite could be involved in transmission, unless vector organisms were involved. Since a parasitemia does occur during the acute stage of toxoplasmosis, studies on transmission by blood-sucking arthropods were carried out by a number of investigators. This work was almost universally unsuccessful and was eventually dropped.

Following the acute stage of the infection, *Toxoplasma* becomes encysted within many tissues of the body. This form was originally called a pseudocyst, because it was believed that the parasites massed within it were dormant inside the original wall of the host cell, from which the nucleus had been extruded. We now know, from many studies, that the cyst wall is formed within the host cell. Some workers consider that the parasite makes the entire contribution of material to the cyst wall, others that the deposition of wall material on the inner surface of a vacuole, within which the parasites lie, is mediated also by some substances from the host cell. The former group believes that the cyst can grow outside the host cell. These are interesting points, worthy of further study, but I will not digress to deal longer with them here.

The main point about the cyst in which we are interested is its possible role in transmission. As I have mentioned, cysts may be demonstrated in many tissues of the body. They have been found in brain, eye, lung, uterus, heart, intestine, liver, and skeletal muscle, as well as other organs. There is no assurance, however, that the tissue distribution of cysts is the same in all animals.

The cyst wall of the parasite is rapidly destroyed by artificial gastric juice or by trypsin. The organisms within the cyst are, however, resistant to the action of pepsin for at least two hours, in marked contrast to the trophozoites. The cysts also remain viable for considerable periods of time in refrigerated tissue so long as they are undisturbed. If they are separated from the tissue, as can be done with brain material merely by shaking with glass beads, they do not survive so well. They will not withstand freezing in tissue, nor when separated are they resistant to osmotic change (distilled water) or to drying.

These observations on cysts have been useful in studying the distribution of the organisms in tissues of various animals by techniques that we have described. They are also important as indicating one means by which carnivorous animals may acquire toxoplasmosis. Indeed, Desmonts et al.[5] have documented the seroconversion of children who are fed raw mutton in an institution in Paris, and other workers have reported individual or small groups of people who apparently have acquired the infection from raw or rare meats.

I have no doubt that this is one method of spread of *T. gondii*. However, we have needed other explanations for the high prevalence of the parasite in some herbivores, in birds, and in human beings who are vegetarians. Furthermore, prevalence studies on human toxoplasmosis have shown the highest rates in such areas as Tahiti, where fresh meat is rarely eaten and then only when well cooked, and the Caribbean area, where meat is scarce and expensive and when eaten is again well cooked. Thus neither the characteristics of the trophozoite nor the cyst indicate that they can serve as the sole transmittal stages of toxoplasmosis.

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Because of the liability of the cyst, it was considered possible that organisms which consume dead tissue or feces might be involved in transmitting such cysts, in a more protected state, from one host to another. For this reason we did numerous experiments in my laboratory, to test the ability of various free-living invertebrates to ingest and carry *Toxoplasma* cysts. All of these gave negative results. We also tested the possibility of fecal transmission, using fresh or stored fecal material from dogs and other animals, but never obtained a positive finding. Because we were trying to do many things along these lines, we probably missed some factors, and did not test this method of transmission adequately, as, for instance, at various intervals after oral administration of *Toxoplasma* cysts. We used principally chronically infected animals because of the finding of *Toxoplasma* cysts in the epithelial lining of the intestine from which they could conceivably be shed into the lumen. We also studied concomitant infections of hookworms and *Toxoplasma* in dogs, with the idea that *Toxoplasma* could be spread by nematodes.

When Hutchison[12] reported the transmission of *T. gondii* by material recovered from zinc sulfate flotation of feces of a cat previously fed *Toxoplasma* cysts, we were, therefore, ready to welcome this as an important clue to the transmission problem. Hutchison's cat had *Toxocara cati* eggs and large *Isospora* sp. oocysts in its feces. The fecal float material was washed and stored in water at room temperature for three months. Then, and at later intervals for over 1 year, it was fed to mice, and produced toxoplasmosis.

In our laboratory we hastened to test Hutchison's observations and we were able to confirm the infectivity of the feces of cats fed *Toxoplasma* cysts. Our cats had *Toxocara cati* but no *Isospora* that we could identify. Three cats with natural *Toxocara cati* infections were fed two or three mice which were chronically infected with *T. gondii*. Flotations of their feces were prepared in $ZnSO_4$ solution and suspended in tap water. Some parts of these were fed immediately to mice, all with negative results. Other parts were kept for various periods of time and then fed to mice. Some of these, from fecal specimens collected on days 8 to 14 after the cats were fed *Toxoplasma* cysts, produced toxoplasmosis when administered orally to mice 48 days to 7 months after collection.

Dubey[5] working in Beverley's laboratory in Sheffield, England, also confirmed the transmission of toxoplasmosis by feces of *T. cati* infected cats fed the tissues of chronically infected mice. Hutchison[13] was unable to produce this result with cats that were not infected with *Toxocara cati*. Furthermore, when Hutchison used a filter to remove embryonated *T. cati* eggs from float material which was *Toxoplasma*-infective, the filtrate did not produce the infection in mice. Our experiments and those of Dubey, which showed the survival of the infective forms after exposure to 5% NaOCL, in addition to their ability to withstand the $ZnSO_4$ solution, suggested that the form of the protozoan in the infective cat feces was either highly resistant itself or was protected within the egg of the nematode. In addition, our experiments had shown that the forms were not immediately infective after they had been shed in the feces, but required some time in water before they became infective. We at that time thought this might be due to the need for the embryonation of the nematode egg, so that it would hatch, or alternatively for the maturation of the *Toxoplasma* form (which we had not seen) itself.

All of these observations seemed to point to the involvement of the nematode egg in the transmission of toxoplasmosis. Indeed, Hutchison's second paper[13] was entitled *"The nematode transmission of Toxoplasma gondii."* However, we had some discrepant results in our laboratory with two cats in which we could not demonstrate a patent *T. cati* infection. One of these cats was fed two *Toxoplasma*-infected mice and its feces were collected daily from the third through the twenty-first day following this meal. Floats of feces collected on days 4, 5, 7-8 and 11 after the feeding, and incubated for 6-9 weeks, produced toxoplasmosis in mice. No nematode eggs were seen in the floats from any of the fecal specimens. These floats were

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examined immediately after they were prepared and the water suspensions were examined at the time they were fed to mice. When the cat was autopsied 27 days after it received the infective *Toxoplasma* meal, only one small and immature *T. cati* male was found in its intestine.

The other cat had received, 3 months prior to a *Toxoplasma*-infective meal, 300 embryonated eggs of *Toxocara cati*. However, the nematode infection either never became established or those worms which eventually reached the intestine after larval development were lost. No *T. cati* eggs were ever seen in its feces and no *T. cati* adults were found in its intestine when it was killed 5 months later. Feces collected from this cat on days 8 and 9 following the *Toxoplasma*-infective meal, floated and incubated in water, produced toxoplasmosis in mice.

These results were difficult to interpret in comparison with Hutchison's[12] report. I stated, in my summary of the problem: "In both of these cases it is possible that small numbers of *T. cati* eggs were present in the infective floats and were missed on microscopic examination of the preparations; otherwise we must resort to the hypothesis that a developmental form of *T. gondii* is produced in the intestinal tract of cats fed the encysted protozoan, that this form is not immediately infective to mice when it is discharged in the cat feces but requires an incubation period in water, and that it is small enough to be easily missed on microscopic examination. Furthermore, this form must be resistant to $ZnSO_4$ and must float in a solution of this salt at a specific gravity of 1.180. It would also have to be resistant to a 30-minute exposure to 5% NaOCL. These characteristics seem remarkably similar to those of the nematode egg. At the present time, there is no explanation available to account for these apparently discrepant findings. We can only wait for further work to determine whether or not the infective form of *T. gondii* in the feces of cats is always associated with *T. cati* eggs."

With the acuity of hindsight, I know now that I should have said "These characteristics seem remarkably similar to those of the nematode egg *or the coccidian oocyst.*" Hutchison's results with filtration, and similar tests in our laboratory, however, had indicated that large pore size filters, that retained the nematode egg, removed infectivity of fecal float preparations. A series of papers by Dubey[6], Hutchison et al.[15], Dubey[7], Work and Hutchison[29], Sheffield and Melton[24], Frenkel, Dubey, and Miller[9] and Hutchison, Dunachie, Slim, and Work[14] and Overdulve[17] established that the transmission of toxoplasmosis takes place on feeding to mice, the feces of cats fed *Toxoplasma*-infective meals 3 days previously without the presence of *Toxocari cati* eggs in the feces. Also, the infective form of *Toxoplasma* in the feces is a coccidian oocyst of very small size, probably *Isospora bigemina*. These reports have essentially confirmed each other. They also may possibly be used to explain other reports that appeared in the intervening years suggesting the spread of *T. gondii* in association with the eggs or larvae of other helminths.[27,2] These latter reports, of *Toxoplasma* infection carried by swine lungworms in pigs and guinea pigs, and by *Hymenolepis fraterna* in rats must be subjected to a great deal of scrutiny. In contrast to the positive results in these host-parasite combinations, Von Rommel[28] failed to transmit *Toxoplasma* infections with the following combinations of helminths and hosts:

Toxoscaris leonina in cats.

Haemonchus contortus, Ostertagia circumcincta, Chabertia ovina, and Oesophagostomum venulosum in sheep.

Ascaris suum, Oesophagostomum quadrispinulatum, and *O. dentatum* in pigs.

Toxocara canis, Toxascaris leonina, Ancylostoma caninum, and *Uncinaria stenocephala* in dogs.

Syphacia obvelata in mice.

None of these last positive reports are well documented; the reported transmission of toxoplasmosis associated with these helminths could be explained by the

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independent occurrence of coccidian oocysts. However, what may be of significance is the host specificity of the intestinal stages of *Toxoplasma*. Miss Melton and I failed to transmit toxoplasmosis with the dog-*Toxocaris canis* association, and Overdulve[17] also had negative results with this combination. Nakayama et al.[16] had no success with *Ascaris suum* in pigs. The only well documented reports of transmission by fecal forms involve the cat. It appears possible that the lack of host specificity of *Toxoplasma gondii* is a characteristic of the first two stages we recognized, the trophozoite and the cyst, but not of the intestinal stage. The development of *T. gondii* through a sexual cycle may be specific for the intestinal epithelium of the cat, or for felines in general. This is something that requires further research. It is a very interesting biological problem.

We must await additional work on the life cycle of *Toxoplasma* to classify this organism definitively, and to interpret these recent findings in relation to the epidemiology of the human infection and disease. Now I should like to discuss the infection in man. To a great extent, the generalities apply also to lower animals.

A very interesting observation has been made in two laboratories working on fecal transmission of toxoplasmosis. Five experienced professional workers who had handled the trophozoite and cyst forms of *Toxoplasma* for years without acquiring the infection have all become serologically positive since working with the oocysts. The observation indicates that people working with large numbers of oocysts should be studied regularly to monitor infection. No episode of illness was identified as related to the sero-conversion. The occurrence of infection without disease is the rule rather than the exception. Only a small percentage of infections result in disease.

Nevertheless, because infection is so prevalent, there are enough instances of disease to make toxoplasmosis a serious medical problem. Siim[28] in Denmark, Beverley and Beattie[1] in England, and Remington et al.[20] in California have estimated that 3% to 6% of cases of lymphadenopathy of unknown etiology are due to toxoplasmosis. The main sign of this illness is lymphadenopathy, cervical, axillary, and inguinal, although cases with only cervical node enlargement are not uncommon. There may be fever, anemia, anorexia, muscular pain, and sore throat, occasionally splenomegaly and/or hepatomegaly with some abnormal liver function tests. On the one hand, the lympadenopathy may be subclinical, not of such import to the patient as to lead him to seek medical attention. On the other hand, the disease may be so generalized that a prolonged convalescence ensues, in which there may be relapses accompanied by re-enlargement of nodes or by recurring fever. Sometimes the lymph node enlargement may last for months and cause suspicion of Hodgkin's disease or chronic lymphatic leukemia. There may be a lymphocytosis with abnormal lymphocytes in the peripheral blood. Occasionally the mesenteric lymph nodes are the ones chiefly involved.

The acute stages of acquired toxoplasmosis in adults rarely grade into a fulminating fatal illness involving many organs. There may be atypical pneumonitis, myocarditis, myositis, hepatitis, enlargement of the liver and spleen, encephalomyelitis, and a rickettsial-like exanthem. Only a few cases of this nature have been observed in the U.S.[19,10,22] A few similar reports have appeared in other countries. Thus the spectrum of acquired acute toxoplasmosis is wide, and there is always the possibility of involvement of one or another organ or organ system accompanying the lymphadenopathy.

The fulminating disease in adults is similar to what is frequently seen in congenital toxoplasmosis. The acute disease ensues in the infant because of the acquisition of the infection by the mother shortly before delivery. The infant presents with a severe febrile illness involving practically all organs. There may be severe jaundice, involvement of the liver, spleen, heart, lung, brain, and eyes. When the transmission to the foetus takes place earlier in pregnancy, the acute infection has waned at the time of birth, and the infant may be born afebrile, with signs of

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central nervous disease developing later, or hydrocephalus, chorioretinitis and other c.n.s. signs may already be present. Desmonts et al.[4] have shown, in 47 cases of pregnant women, that even in acute infection of the mothers,[27] or over 50% of the foetuses escaped infection, and that among the infected foetuses only 4 neonatal deaths and 3 living children with signs of congenital toxoplasmosis were produced. There were thirteen infants proven to be infected on the basis of serological tests but without signs of disease.

It is possible that some of the latter infants would later be found to have chronic ocular toxoplasmosis. Chorioretinitis is the most common manifestation of acute or subacute congenital toxoplasmosis, and there are many case reports of ocular lesions flaring up some years after birth of apparently normal children. Chorioretinitis is also a manifestation of chronic toxoplasmosis in adults. Some of this may be attributable to congenital infection. However, there is no doubt that chorioretinitis in adults can result from the acquired infection. Individual cases of chorioretinal lesions following lymphadenopathic disease have been documented. Furthermore many cases of chorioretinitis in adults are unilateral, while congenital *Toxoplasma*-induced chorioretinitis in infants is usually bilateral. It is estimated that between 15% and 35% of cases of chorioretinitis (posterior uveitis) in the United States are due to *T. gondii*.

Other forms of disease, such as chronic idiopathic myocarditis have been attributed to chronic *Toxoplasma* infection. The proof is not yet demonstrated. *Toxoplasma* encysts in heart muscle but until there is a demonstration of necrotizing reactions around cysts in cases of myocarditis, the mere demonstration of the cysts is not adequate evidence that they are the etiological agents of the heart muscle disease. Other reports of chronic lymphadenitis, persistent over many years, are very difficult to substantiate. Habitual abortion due to chronic toxoplasmosis has been claimed to be a frequent occurrence especially in central and eastern Europe. The best data we have, in the United States, England, and France, speak against such conclusions. There is some evidence that Toxoplasma can occasionally be found in the products of abortion in a woman with evidence of long-past infection. This is not surprising, since *Toxoplasma* cysts do reside in the uterus. However, most of our experience points to the acute infection in women as the principal circumstance under which transplacental transmission occurs.

With the increasing use of anti-tumor drugs which are also immuno-suppressive agents, we are seeing more cases of toxoplasmosis along with more cases of exacerbated herpes virus and cytomegalic virus infections in leukemia and solid tumor patients. Similarly, toxoplasmosis may occasionally be a problem in organ transplant cases, in which immuno-suppressive drugs are used to prevent rejection.

I have given you some estimates of the percentages of lymphadenopathy and chorioretinopathy of unknown etiology that may be due to toxoplasmosis. For the congenital infection, I can say that estimates of congenital toxoplasmosis per 1000 live births range between 0.25 to 6 - 7 in various countries. The other conditions I have mentioned are of lesser importance statistically. However, as an old parasitologist who has always had to justify work on diseases other than the major killers, I'd like to point out that the victim of a rare disease entity is not especially comforted by statements that he is a statistical curiosity.

The disease in animals, as I have said, is similar to that in man. The abortion problem in sheep in New Zealand and England is illustrative. It is noteworthy, however, that cats frequently suffer a granulomatous disease of the intestinal tract, and that dogs have similar signs and pathology in acute toxoplasmosis. Again with 20:20 hindsight, I can say that we should have investigated fecal transmission from cats earlier.

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