Congenital Toxoplasmosis: A Review

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Toxoplasmosis is caused by infection with the protozoan parasite *Toxoplasma gondii*. In the United States, approximately 85% of women of childbearing age are susceptible to acute infection with *T. gondii*. Acute infections in pregnant women may cause serious health problems when the organism is transmitted to the fetus (congenital toxoplasmosis), including mental retardation, seizures, blindness, and death. An estimated 400 to 4000 cases of congenital toxoplasmosis occur in the U.S. each year. Manifestations of congenital toxoplasmosis may not become apparent until the second or third decade of life. Serologic tests are used to diagnose acute infection in pregnant women, but false-positive tests occur frequently, therefore, serologic diagnosis must be confirmed at a reference laboratory before treatment with potentially toxic drugs should be considered. Much of congenital toxoplasmosis can be prevented by educating women of childbearing age and pregnant women to avoid eating raw or undercooked meat, to avoid cross-contamination of other foods with raw or undercooked meat, and to use proper cat-litter and soil-related hygiene.

**Target Audience:** Obstetricians & Gynecologists, Family Physicians

**Learning Objectives:** After completion of this article, the reader will be able to outline the biology of toxoplasmosis, to explain the methods of transmission of toxoplasmosis, and to identify the methods used to diagnose toxoplasmosis in pregnancy.

Toxoplasmosis is caused by infection with the protozoan parasite *Toxoplasma gondii*. In the United States, a serological survey from the Third National Health and Nutrition Examination Survey found that an estimated 23% of adolescents and adults have laboratory evidence of infection with *T. gondii* (15% among women of childbearing age) (1; Centers for Disease Control, unpublished data, 1994). Although these infections are usually either asymptomatic or associated with self-limited symptoms in adults (e.g., fever, malaise, and lymphadenopathy), infections in pregnant women can cause serious health problems in the fetus if the parasites are transmitted (i.e., congenital toxoplasmosis) and cause severe sequelae in the infant including mental retardation, blindness, and epilepsy. Practicing obstetricians may be confronted with a number of issues regarding toxoplasmosis, including diagnosis, laboratory testing, screening practices, clinical presentation, and prevention.

Although congenital toxoplasmosis is not a nationally reportable disease, extrapolation from regional studies indicates that an estimated 400 to 4,000 cases occur in the U.S. each year (2–4). The most recent data from New England indicate that congenital toxoplasmosis occurs in approximately 1 in 10,000 live births (4).

**BIOLOGY, TRANSMISSION, CLINICAL SYMPTOMS, AND EPIDEMIOLOGY**

* T. gondii has a complex life cycle (Fig. 1) consisting of three stages: 1) tachyzoite—during the acute
stage of infection, this form of the parasite invades and replicates within cells; 2) bradyzoite—during latent infections, this form of the parasite is present in tissue cysts; and 3) sporozoite—this form of the parasite is found in oocysts, which are environmentally resistant. *T. gondii* tachyzoites can invade and multiply in most cell types and are found in all organs in acute infection, especially in muscle (including heart), liver, spleen, lymph nodes, and the central nervous system. The tachyzoite is the form of the organism responsible for congenital infection. Cell invasion results in death of parasitized cells and an acute inflammatory reaction. Placental lesions are usually microscopic in humans, but macroscopic necrosis has been reported in animals (5).

Members of the family Felidae (including domestic and feral cats) are the definitive hosts of *T. gondii*. During acute infections, cats excrete unsporulated (i.e., noninfectious) oocysts in their feces; after several days to several weeks, depending on environmental conditions, the oocysts sporulate and become infectious. Under favorable conditions (i.e., in warm, moist soil), oocysts can remain infectious for approximately 1 year. They do not survive well in arid, cold climates and can be destroyed by heating (6–10).

Toxoplasmosis can be transmitted to humans by three principal routes. First, humans can eat raw or inadequately cooked infected meat (especially pork, mutton, and wild game meat (11)) or eat uncooked foods that have come in contact with infected meat. Second, humans can inadvertently ingest oocysts that cats have passed in their feces, either in a cat litter box or in soil (e.g., soil from gardening or unwashed fruits or vegetables). Third, a woman can transmit the infection to her unborn fetus transplacentally. In adults, the incubation period ranges from 10 to 23 days from ingestion of undercooked meat, and from 5 to 20 days from ingestion of oocysts from cat feces.

A report conducted by the Economic Research Service of the United States Department of Agriculture concluded that one half of the toxoplasmosis cases in the U.S. are caused by eating contaminated meat. The estimated economic burden of these infections is $7.7 billion each year, primarily from congenital toxoplasmosis (12). Other data are available to assist in estimating the portion of the disease burden of toxoplasmosis attributable to consumption of infected meat (i.e., raw or undercooked meat or cross-contamination from raw or undercooked meat). A recent study compared results from a cross-sectional seroprevalence study of Seventh Day Adventists, a religious group that follows a diet containing no meat, with serologic results from a control group of volunteers who were not Seventh Day Ad-

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**Fig. 1.** Life cycle of *Toxoplasma gondii*. 
treated, congenital toxoplasmosis can be associated with rare exception (15), do not transmit the infection to their fetuses. Women infected with T. gondii during pregnancy can transmit the infection across the placenta to their fetuses. The risk of congenital disease is lowest (10–25%) when acute maternal infection occurs during the first trimester and highest (60–90%) when acute maternal infection occurs during the third trimester (10, 16, 17). However, the severity of disease is worse if infection is acquired in the first trimester (10, 18). The overall risk of congenital infection from acute T. gondii infection during pregnancy is 20% to 50%.

The classic triad of signs suggestive of congenital toxoplasmosis includes chorioretinitis, intracranial calcifications, and hydrocephalus. Most infants infected in utero are born with no obvious signs of toxoplasmosis on routine examination, but up to 80% develop learning and visual disabilities later in life if they are followed into adulthood (19, 20). If untreated, congenital toxoplasmosis can be associated with severe and even fatal disease (21).

In adults, the severity of T. gondii infection is correlated with the immune status of the infected person. Toxoplasmosis in immunocompetent adolescents or adults is generally mild or inapparent. Mild infections can result in lymphadenopathy, fever, fatigue, and malaise, all of which usually resolve within weeks to months without specific treatment. However, infection in immunocompromised persons can be severe. Immunosuppression caused by AIDS or therapies for malignancies, transplants, or lymphoproliferative disorders can result in reactivation of preexisting latent T. gondii infections. Reactivation most often involves the central nervous system, and symptoms may include those of meningoencephalitis or a mass lesion. Women with reactivated T. gondii infection can transmit the organism congenitally (22, 23).

Recent epidemiologic studies have identified the following risk factors for T. gondii infection: owning cats (24), seropositive cats in farming areas (25), cleaning the cat litter box (26), eating raw or undercooked pork, mutton, lamb, beef, or mince meat products (24, 26, 27), gardening (25), eating raw or unwashed vegetables or fruits, eating raw vegetables outside the home (24), contact with soil (27), washing kitchen knives infrequently (26), having poor hand hygiene (24), and travel outside of Europe, U.S., or Canada (27). Protective factors for T. gondii infection include a meat-free diet (13), living at high altitudes and in arid climates (28, 29), and living in climates with frequent freezing and thawing (30).

Owning a cat has not been a consistent risk factor for T. gondii infection. Owning a cat was not shown to be a risk factor for T. gondii infection in two studies of pregnant women (27, 31) and in a study of HIV-infected persons (32). Risk for T. gondii infection does not occur from merely owning a cat, but rather from being exposed to cat feces from a cat shedding oocysts. Cats generally only shed oocysts for several weeks during their lives if they become infected with T. gondii. Cats kept indoors that do not hunt prey or are not fed raw meat are not likely to acquire T. gondii infection and, therefore, pose little risk. Unless cats are sick with diarrhea, little or no feces will stick to their perianal area (11). Usually, adult cats are not diarrheal during the period in which they are shedding oocysts (11). Because of their grooming habits, fecal matter has not been found on the fur of clinically normal cats (11). In addition, in a study of cats induced to shed oocysts, no oocysts could be found on the cats’ fur after they shed oocysts (33). Therefore, the possibility of transmission to human beings via touching cats is thought to be minimal or nonexistent (11).

Neighborhood or feral cats that defecate in gardens or sandboxes may pose the greatest risk for toxoplasmosis for some persons, whether or not they own a cat.

It should also be noted that because cats may not develop antibodies to T. gondii during the oocyst-shedding period, serologic examination of cats does not provide useful information regarding the ability of a particular cat to transmit toxoplasmosis (11). A cat that has a positive serologic test for T. gondii probably has shed oocysts previously and, therefore, may pose less of a risk than a serologically negative cat, but cats can shed oocysts more that once, thus, serologic testing of cats is not really helpful in this instance either.

Outbreaks of toxoplasmosis in humans have been attributed to ingestion of raw or undercooked ground beef, lamb, pork, venison (34–39), unpasteurized goat’s milk (40), contaminated, unfiltered drinking
High *T. gondii* seroprevalence is found in countries such as France where undercooked meat is commonly eaten (45, 46), and in tropical areas of Latin America or sub-Saharan Africa where cats are abundant and the climate favors survival of oocysts (47–54). Studies in Scandinavian countries (55–57) and England (58) show lower seroprevalence, more similar to that in the U.S.

**DIAGNOSIS OF TOXOPLASMOsis IN PREGNANCY**

Acute toxoplasmosis is diagnosed rarely by detecting the parasite in body fluids, tissue, or secretions; the most common method of diagnosis is based on antibody detection. The presence of elevated levels of *Toxoplasma*-specific IgG antibodies indicates infection has occurred at some point, but does not distinguish between an infection acquired recently and one acquired in the distant past. In acute infection, IgG and IgM antibodies generally rise within 1 to 2 weeks of infection (59). Determining when *T. gondii* infection occurred in a pregnant woman is important because infection before conception poses little risk for transmission of infection to the fetus; however, infection after conception does pose such risk. Detection of *Toxoplasma*-specific IgM antibodies has been used as an aid in determining the time of infection, but IgM antibodies have been reported to persist for up to 18 months postinfection (6). A negative IgM with a positive IgG result indicates infection at least 1 year previously. A positive IgM result may indicate more recent infection or may be a false-positive reaction. A flow diagram for *T. gondii* testing and guide to interpretation of *T. gondii* tests are presented in Figure 2 and Table 1.

In the United States, commercial test kits for *Toxoplasma*-specific IgG and IgM antibodies are readily available. Some commercial IgM tests have had problems with specificity, resulting in unacceptably high rates of false-positive test results. In 1996, the Food and Drug Administration (FDA) and Centers for Disease Control (CDC) conducted extensive evaluations of the six most commonly used commercial IgM kits in the U.S. to determine the extent of the problem with the specificity of these kits. Sensitivity and specificity rates for these six kits ranged from 93.3% to 100.0% and from 77.5% to 99.1%, respectively (60).

As a result of these findings, in 1997 FDA distributed an advisory to physicians in the U.S. highlighting these test limitations. The agency provided a guide for interpreting test results (Table 1) and issued a recommendation to laboratory personnel and physicians advising them to be aware of the specificity problems associated with some commercial test kits before making decisions about the clinical management of their patients.

IgM-positive results should be confirmed by a *Toxoplasma* reference laboratory (60). A toxoplasmosis reference laboratory may be able to help narrow down the time of infection with tests such as the IgG avidity test (61, 62). IgG avidity, or the strength with which IgG binds to *T. gondii*, usually shifts from low avidity to high avidity at about 5 months after infection and can be used to rule out primary *T. gondii* infection in early pregnancy in approximately three quarters of women with positive IgM serum tests (61). Although commonly used in Europe, the IgG avidity test is available currently in only two labora-

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**Table 1. Guide to interpretation of *Toxoplasma*-specific IgM antibody test results**

<table>
<thead>
<tr>
<th>IgG Positive</th>
<th>IgM Negative:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infected for more than 1 year</td>
<td></td>
</tr>
<tr>
<td>1. Infection within last 2 years</td>
<td></td>
</tr>
<tr>
<td>2. False positive IgM</td>
<td></td>
</tr>
</tbody>
</table>

**Test serum for presence of *Toxoplasma*-specific IgG antibodies**

**IgG Negative: Not infected**
- Retest in 3 weeks if acute infection suspected

**To determine approximate time of infection, test serum for presence of *Toxoplasma*-specific IgM antibodies**

**IgG Positive IgM Negative:**
- Infected for more than 1 year

**Obtain second sample 2 weeks after first sample. Send both samples to a *Toxoplasma* reference laboratory for confirmation of IgG and IgM results and, if necessary, avidity, AC/HS, IgA, and IgE testing to more accurately determine the time of primary infection before any intervention.**

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**Fig. 2. Algorithm for the serodiagnosis of toxoplasmosis in people older than 1 year of age. Adapted from Wilson M, McAuley JM. *Toxoplasma*. In: Murray PR, Baron EJ, Pfaller MA et al., eds. Manual of Clinical Microbiology, 7th Ed. Washington DC: ASM Press, 1999, pp 1347–1382.**

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TABLE 1  Guide to general interpretation of Toxoplasma gondii serology results obtained with commercial assays

<table>
<thead>
<tr>
<th>Results</th>
<th>Report/Interpretation for humans (except infants)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG IgM</td>
<td></td>
</tr>
<tr>
<td>Negative Negative</td>
<td>No serological evidence of infection with Toxoplasma.</td>
</tr>
<tr>
<td>Negative Equivocal</td>
<td>Possible early acute infection or false-positive IgM reaction. Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same, the patient is probably not infected with Toxoplasma.</td>
</tr>
<tr>
<td>Negative Positive</td>
<td>Possible acute infection or false-positive IgM result. Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same, the IgM reaction is probably a false-positive.</td>
</tr>
<tr>
<td>Equivocal Negative</td>
<td>Indeterminate: obtain a new specimen for testing or retest this specimen for IgG in a different assay.</td>
</tr>
<tr>
<td>Equivocal Equivocal</td>
<td>Indeterminate: obtain a new specimen for both IgG and IgM testing.</td>
</tr>
<tr>
<td>Equivocal Positive</td>
<td>Possible acute infection with Toxoplasma. Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same or if the IgG becomes positive, both specimens should be sent to a reference laboratory with experience in the diagnosis of toxoplasmosis for further testing.</td>
</tr>
<tr>
<td>Positive Negative</td>
<td>Infected with Toxoplasma for &gt;1 year.</td>
</tr>
<tr>
<td>Positive Equivocal</td>
<td>Infected with Toxoplasma for probably &gt;1 year, or false-positive IgM reaction. Obtain a new specimen for IgM testing. If results with the second specimen remain the same, both specimens should be sent to a reference laboratory with experience in the diagnosis of toxoplasmosis for further testing.</td>
</tr>
<tr>
<td>Positive Positive</td>
<td>Possible recent infection within the last 12 months, or false-positive IgM reaction. Send the specimen to a reference laboratory with experience in the diagnosis of toxoplasmosis for further testing.</td>
</tr>
</tbody>
</table>

Editors' Note: Toxoplasma Serology Laboratory, Research Institute, Palo Alto Medical Foundation, Ames Building, 795 El Camino Real, Palo Alto, CA 94301; phone: 650-853-4848, and MRL Reference Laboratories, 5785 Corporate Ave., Cypress, CA 90630; phone: 800-445-0185.

In France, a screening program was implemented in 1976 to detect and treat T. gondii infection during pregnancy. The goal of this program is to institute preventive measures for seronegative women and to ensure early diagnosis and treatment of infection acquired during pregnancy. Since the beginning of the program, premarital and prenatal medical examinations for T. gondii antibodies have been performed. Premarital examinations are conducted to distinguish previously infected women from women who have not been previously infected. When an uninfected woman becomes pregnant, testing is conducted at her first prenatal examination during her first trimester and at six additional examinations conducted monthly during her second and third trimesters. In addition, women are educated about prevention methods during pregnancy (71). If these screening tests detect evidence of acute infection during pregnancy, treatment for the woman is initiated with spiramycin in an effort to prevent transmission to the fetus. If infection in the fetus is confirmed through fetal blood sampling and amniocentesis, pyrimethamine and sulfadiazine or sulfadoxine is added to the regimen (72, 73) because spiramycin does not generally cross the placenta.

Although the proportion of the population included in the French program has been incomplete, it has been associated with a decline in the incidence of congenital infection, as well as a decline in severe disease detected at birth. The proportion of the decline specifically attributable to the program or to the general decline in Europe in rates of seropositivity is difficult to determine because no unscreened group of women exists for comparison.

Austria implemented a toxoplasmosis-screening program in 1975. Nearly all women who become pregnant are serologically screened early in pregnancy and, if the results are found to be negative initially, are tested again during the second and third
trimesters. Women with *T. gondii* infections are treated as soon as infection is detected. Although seropositivity rates among pregnant Austrian women have declined from approximately 50.0% during the late 1970s to 36.7% during the early 1990s, the incidence of congenital *T. gondii* infection has declined even more, from 50 to 70 cases per 10,000 births before the program to 1 per 10,000 births during the early 1990s (74). As with the French program, the lack of an unscreened comparison group precludes determining the proportion of the decline attributable to the screening program, and the lack of cost figures precludes cost-effectiveness analyses.

The European Research Network on Congenital Toxoplasmosis was established in 1993 and has sponsored several studies regarding public health interventions for congenital toxoplasmosis. Most recently, a multicenter study was conducted to evaluate the effectiveness of toxoplasmosis treatment administered during pregnancy in preventing transmission of maternal infection to the fetus. Pregnant women who visited one of five European university medical centers for prenatal care were screened for *T. gondii* antibodies at their first prenatal visit. Women who were seronegative were retested at least once every trimester in two centers and monthly in the other centers, until the birth of the infant. For women who seroconverted during pregnancy, prenatal antibiotic treatment was started, and their infants were followed for 1 year after birth. Treatment regimens consisted of spiramycin or a combination of pyrimethamine and sulfadiazine. If prenatal infection was confirmed with amniocentesis or cordocentesis, women were treated with pyrimethamine and sulfadiazine or sulfadoxine. Of the women who screened positive and did not receive prenatal therapy, transmission from mother to infant occurred in 72% of the mother-infant pairs; of women who received prenatal therapy, transmission occurred in 39% of the mother-infant pairs. However, this difference was not statistically significant when the time of gestation that *T. gondii* infection occurred was accounted for in the treated and untreated groups. Nevertheless, 20% of the untreated mothers gave birth to infants with severe sequelae, whereas only 3.5% of the treated mothers gave birth to infants with severe sequelae. Furthermore, the earlier antibiotics were administered after infection, the less likely sequelae were detected in the infant (75).

From January 1988 through June 1989, a cost-benefit analysis of *T. gondii* screening during pregnancy was conducted in a prospective study in Finland. The study compared costs of screening alternatives for primary infections during pregnancy with the costs of no screening. With screening, the annual costs of congenital toxoplasmosis were U.S.$95 per pregnancy; without screening, annual costs were U.S.$128 per pregnancy. Furthermore, screening along with health education was more beneficial than health education alone (76). The study findings suggest screening is cost beneficial in countries with a low incidence of congenital toxoplasmosis, such as Finland. The findings of other studies suggest screening programs can also be beneficial in areas with high incidences of congenital toxoplasmosis (72, 77, 78).

Although the findings of some European studies suggest *T. gondii* screening programs of women of childbearing age can prevent cases of congenital toxoplasmosis, several concerns limit support for such programs in the U.S. Some researchers have argued that because the prevalence of congenital toxoplasmosis is so rare in the United States, acute early pregnancy toxoplasmosis is far too uncommon to warrant routine screening (79). The American College of Obstetricians and Gynecologists (ACOG) (80) does not recommend routine screening. Screening may lead to equivocal or false-positive test results (60, 77), which could lead to inappropriate treatment. There have been no randomized trials using spiramycin or other medications to assess the effect of treatment on congenital transmission. Authors conducting a review of studies performed from 1966 through 1997 concluded that it is unclear whether antenatal treatment in women with toxoplasmosis reduces congenital transmission (81). In a multicenter evaluation of 144 pregnant women with acute toxoplasmosis in Europe, prenatal antibiotic therapy had no impact on the maternal-fetal transmission rate. However, prenatal treatment reduced the rate of severe sequelae among infected infants from 20% to 3.5% (75). In their review of the literature, authors from Norway, a country with a low incidence of toxoplasmosis similar to the U.S., concluded that sufficient scientific evidence is not yet available to propose screening for toxoplasmosis in pregnant women (82). In addition, the Royal College of Obstetricians and Gynecologists in the United Kingdom has recommended that prenatal screening for toxoplasmosis should not be introduced in the U.K. (83). Finally, researchers from the U.S. used decision analysis to evaluate clinical outcomes with three alternatives: 1) no testing for congenital toxoplasmosis, 2) targeted screening in cases of incidental abnormalities noted on ultrasound, and 3) universal serologic screening of pregnant women followed
by amniocentesis to diagnose fetal infection in cases of maternal seroconversion. Universal screening reduced the total number of cases of congenital toxoplasmosis compared with no testing or targeted screening. However, compared with no testing, universal screening with medical treatment resulted in 18.5 additional pregnancy losses for each case of toxoplasmosis avoided (84). Risks associated with amniocentesis were an important factor (0.36% fetal death rate). No cost assessment was done in this analysis. There is still a need for cost-effectiveness studies to enable comparison of the benefits of expanded testing in the U.S., the costs of such testing, and the unintended adverse consequences that might accompany such testing.

Side effects of treatments must be considered when treating pregnant women. Spiramycin is a macrolide antibiotic that can cause gastrointestinal and dermatologic reactions in the mother. The Medical Letter lists spiramycin as the drug that should be used for toxoplasmosis for prophylactic use during pregnancy, however, it is an investigational drug in the U.S. and can only be obtained through the manufacturer (85). Although sulfonamides have been associated with kernicterus in the newborn when given in late pregnancy, this complication was not noted to occur in a review of screening programs by French authors (86). Pyrimethamine is not generally recommended for pregnant women because it is a folic acid antagonist (pregnancy category C). Folate serves as a coenzyme in the synthesis of DNA, RNA, myelin, and lipids. A lack of folic acid in pregnancy has been associated with neural tube defects early in pregnancy before the neural tube has closed. Screening programs do not generally recommend pyrimethamine and sulfadiazine treatment before 18 weeks’ gestation (10). Pyrimethamine has also been associated with heart and kidney malformations in infants and may increase the risk of central nervous system cancer in childhood (87, 88). Pyrimethamine and sulfadiazine also carry a risk of bone marrow suppression in both the mother and the infant (10). A supplement of folic acid should be given with pyrimethamine to compensate for the reduction of folic acid caused by pyrimethamine. Unfortunately, because there are few cases available for study, it has not been possible to fully assess the risks of side effects due to treatment for toxoplasmosis during pregnancy.

Much of the above discussion raises the issue of when targeted screening should be done for toxoplasmosis in the U.S. In their decision analysis, Bader et al. (84) determined that screening high risk women (defined as a five-fold increase in risk or a rate of maternal toxoplasmosis as high as 1 in 100 pregnancies) would still lead to at least 2.9 fetal losses per case of toxoplasmosis avoided. Therefore, they concluded that screening of cat owners would probably not be appropriate. In a large well-designed study of acute T. gondii infection in pregnant women from the European Research Network on congenital toxoplasmosis, none of the risk factors for T. gondii infection increased the risk by as much as five-fold (27). Nevertheless, in this study, the highest risks were associated with eating undercooked lamb or other less conventional meats that included wild game meats and with travel outside Europe, U.S., or Canada.

It would seem prudent to screen pregnant women for acute toxoplasmosis if there were suspicious ultrasound findings. Such findings include hydrocephalus, intracranial calcifications, microcephaly, fetal growth restriction, ascites, and hepatosplenomegaly. In addition, HIV-infected women should be screened for toxoplasmosis (80).

**PREVENTION OF TOXOPLASMOsis IN PREGNANT WOMEN**

Recommendations for prevention of toxoplasmosis in pregnant women were discussed at a conference at the Centers for Disease Control and Prevention and published (71). These recommendations were as follows:

- To prevent toxoplasmosis and other food-borne illnesses, food should be cooked to safe temperatures. A food thermometer should be used to measure the internal temperature of cooked meat to ensure that meat is cooked all the way through. Beef, lamb, and veal roasts and steaks should be cooked to at least 145°F; and pork, ground meat, and wild game should be cooked to 160°F before eating. Whole poultry should be cooked to 180°F in the thigh to ensure that the meat is cooked thoroughly.
- Fruits and vegetables should be peeled or washed thoroughly before eating.
- Cutting boards, dishes, counters, utensils, and hands should always be washed with hot soapy water after they have contacted raw meat, poultry, seafood, or unwashed fruits or vegetables.
- Pregnant women should wear gloves when gardening and during any contact with soil or sand because cat waste might be in soil or sand. After gardening or contact with soil or sand, hands should be washed thoroughly.
- Pregnant women should avoid changing cat litter if possible. If no one else is available to change the cat litter, pregnant women should use...
gloves, then wash hands thoroughly. The litter box should be changed daily because T. gondii oocysts require several days to become infectious. Pregnant women should be encouraged to keep their cats inside and not adopt or handle stray cats. Cats should be fed only canned or dried commercial food or well-cooked table food, not raw or undercooked meats.

- Health education for women of childbearing age should include information about meat-related and soil-borne toxoplasmosis prevention. Healthcare providers should educate pregnant women at their first prenatal visit about food hygiene and prevention of exposure to cat feces.

- Healthcare providers who care for pregnant women should be educated about two potential problems associated with the T. gondii serology tests. First, no assay exists that can determine precisely when the initial T. gondii infection occurred. Second, in populations with a low incidence of T. gondii infection, such as in the U.S., a substantial proportion of the positive IgM test results will probably be false-positive.

- The government and the meat industry should continue their efforts to reduce T. gondii in meat.

One approach to preventing toxoplasmosis focuses on educating women of childbearing age about minimizing their risk for infection with T. gondii. Educational interventions assume that increased knowledge results in awareness, which consequently results in changes in risky behavior and declines in infection rates. Messages emphasize the importance of avoiding eating raw or undercooked meat, handling raw meat safely, and washing hands after gardening or changing the cat litter box.

A study conducted as part of prenatal classes at Canadian public health agencies evaluated the effect of a 10-minute teaching session on three behaviors: practices associated with cleaning the cat litter box and limiting the cat’s diet to cooked food; safe food-handling practices; and hand washing after exposure to cat feces, garden soil, or raw meats. Among women in the classes, behavior improved regarding the behaviors associated with cats; however, behavior regarding food-handling practices remained unchanged. In addition, only professional women showed improvement in their hand washing practices (89).

During 1979 to 1990, a Belgian study assessed the effectiveness of educational sessions held in hospital settings. Baseline data were collected during 1979 to1982, when no education measures were in effect. During 1983 to 1990, education sessions were provided to pregnant women. The intervention was associated with a 63% decrease in seroconversion rates (90). Although it is possible that other factors such as a reduction of T. gondii infection in meat could be responsible for the reduction in seroconversion rates, this study suggests that educational intervention is beneficial in preventing acute toxoplasmosis among pregnant women.

Educational programs are a potentially powerful intervention because of their low cost, and because pregnant women are motivated to protect the health of their babies. However, the impact of educational programs is difficult to evaluate because of the limited number of comparative studies conducted with rigorous scientific methodology and of sufficient size to enable calculation of the effectiveness of the intervention compared with its cost.

AREAS FOR FUTURE RESEARCH

There is a need for more complete and accurate population-based data regarding the incidence of toxoplasmosis and the number of cases by mode of transmission. Development of techniques that would enable tracing the source of individual infections to food-borne, cat-borne, or soil-borne transmission would help in defining the best approaches for preventive education. Efforts are needed to develop more accurate screening diagnostic tests and improved confirmatory tests. Research is needed also to further determine the therapeutic value, costs, and benefits of prenatal toxoplasmosis screening. Genetic research is underway to determine if specific strains are more infectious or pathogenic to humans. It is already known that at least three clonal lineages of T. gondii exist (91). It also may be possible with genetic research to determine the source and spread of T. gondii infections in animals and humans. Finally, more research needs to be done on the most effective means of primary prevention of toxoplasmosis through education.

References

3. Kimball AC, Kean BH, Fuchs F. Congenital toxoplasmosis: a