The major ways in which man acquires toxoplasmosis are through handling and consumption of raw meat and contact with feces (in litterpans and soil) from cats infected with Toxoplasma gondii. To prevent toxoplasmosis, certain steps should be taken. Meat should be heated throughout to 150°F (66°C) before consumption, and hands should be washed with soap and water after handling meat to remove any toxoplasma organisms contaminating the skin. Infection of indoor cats can be prevented by feeding them dry, canned, or boiled food and by restraining them from hunting. To the extent that diet cannot be controlled, an indoor cat’s litterpan should be cleaned daily, feces should be flushed down the toilet, and the pan should be disinfected with boiling water. A person should wear disposable gloves while performing these tasks. Since soil contaminated by oocysts from T. gondii in feline feces is difficult to disinfect, contaminated sand in children’s sandboxes should be disposed of, and gloves should be worn while one is working with soil that may contain feces from cats. Pregnant women should avoid cats whose source of food is unknown and should not empty litter pans, as toxoplasmosis can be transmitted to infants in utero.

The transmission of toxoplasmosis first became of interest with the discovery of neonatal infection. In the newborn baby, the disease often gives rise to disorders of the central nervous system and the eyes [1, 2]. Subsequently, the transmission of toxoplasmosis from raw or undercooked meat was suspected [3, 4], confirmed experimentally [5], and illustrated by an epidemic attributed to hamburger meat [6]. It became apparent during the last few years that a stage of Toxoplasma gondii was shed in the feces of cats [7–9]. After an oocyst was discovered in feline feces and it was shown that Toxoplasma can be transmitted like other coccidia [10, 11], the focus of interest in the epidemiology of toxoplasmosis shifted almost entirely to the role of cats since there is no evidence that other animals shed toxoplasma oocysts [10, 12].

The following background data about feline toxoplasmosis are important to this study: (1) cats shed toxoplasma oocysts after eating mice, rats, birds, or meat containing toxoplasma cysts, as well as after the ingestion of oocysts [10]. (2) The prepatent period to the shedding of oocysts is three to five days after the ingestion of mice or meat containing toxoplasma cysts, and 20–34 days after the ingestion of oocysts [11]. (3) Oocysts are shed by cats for one to two weeks during primary infection and not at all or briefly in reduced numbers after reinfection [13–16]. (4) Oocysts are noninfectious until sporulation, which requires one to five days (or longer), depending on aeration and temperature [11, 16]. (5) Under favorable circumstances, oocysts remain infectious for several months to a year or longer [8, 16].

Oocysts are the key to the epidemiology of toxoplasmosis (figure 1), since they infect all mammals and birds tested and, circumstantially, humans [12]. Transmission of Toxoplasma gondii by carnivorism also appears to be frequent, and most of the animals eaten have probably been infected with oocysts. Of lesser frequency appears to be transplacental infection, although medically important [17, 18], and transmission via chicken eggs [19].

A number of sensationalist articles in the lay
press have raised more questions and fears about cats and toxoplasmosis than they have answered. For this reason, we wish to present data on the length of time that toxoplasma oocysts are shed and survive in feces of cats under several conditions, to outline methods by which oocysts in feces can be diagnosed and disinfected, and to suggest methods by which the potential communicability of Toxoplasma by cats can be diminished. Although obtained with one strain of Toxoplasma gondii only, these data provide a preliminary guide until common variations can be investigated.

Materials and Methods

Tests for infectivity of oocysts in mice. Material to be tested for infectivity was administered to groups of two to six mice by stomach tube; when bacterial contamination could be excluded, such as after exposure to 2% sulfuric acid, the specimen was injected ip. The number of infectious oocysts was estimated either by titration in mice or indirectly by comparison of the day of death of indicator mice with a standard titration curve (table 1).

Collection of toxoplasma oocysts. Seronegative cats were infected when fed mice chronically infected with the M-7741 strain of Toxoplasma gondii. Feces were collected daily, the presence of oocysts was determined, generally after flotation in a 40% (w/v) solution of sucrose sp gr, 1.15, and sporulation was permitted to occur in 2% sulfuric acid while the feces were aerated by agitation on a shaker. In several experiments, untreated fecal material was used as indicated, without concentration or preservative. The morphology of the toxoplasma oocysts and of the two common coccidia of cats, Isospora felis and Isospora rivolta, is shown in figure 2. At the time of shedding, an oocyst is generally one cytoplasmic mass; after sporulation, there are two sporocysts, each containing four sporozoites.

Drying. For studies of ambient conditions, relative humidity was determined from daily readings of dry and wet bulb thermometers. To study the effects of different degrees of humidity, several
Table 1. Relationship between dose and mortality in mice fed different dilutions of oocysts of *Toxoplasma gondii* (M-7741 strain).

<table>
<thead>
<tr>
<th>Dilution of inoculum</th>
<th>Minimal infectious dose</th>
<th>Days until death</th>
<th>Survivors/total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean ± Range</td>
<td></td>
</tr>
<tr>
<td>Original</td>
<td>$10^5$</td>
<td>5.3 ± 0.87</td>
<td>5–6</td>
</tr>
<tr>
<td>1:10</td>
<td>$10^4$</td>
<td>7.6 ± 1.8</td>
<td>5–9</td>
</tr>
<tr>
<td>1:100</td>
<td>$10^3$</td>
<td>8.1 ± 1.8</td>
<td>5–10</td>
</tr>
<tr>
<td>1:1000</td>
<td>$10^2$</td>
<td>11.8 ± 0.90</td>
<td>11–13</td>
</tr>
<tr>
<td>1:10,000</td>
<td>$10^1$</td>
<td>(26.7) ± 17.0</td>
<td>7–60</td>
</tr>
<tr>
<td>1:100,000,000</td>
<td>$10^0$</td>
<td>(47.5) ± 18.5</td>
<td>14–60</td>
</tr>
</tbody>
</table>

**NOTE.** For the computation of a mean survival time, survivors with infection were considered to have died after 30 days, and survivors without infection after 60 days, at the termination of the experiment.

* One survivor was infected.

Mixtures of sulfuric acid and water were prepared according to a table of values that took the room temperature into account [20]. Petri dishes containing fresh feces from cats were placed on a pedestal in tightly sealed jars. In the jar containing pure sulfuric acid, the progress of drying was observed visually and by daily weighing of the fecal specimens. In other experiments, a thin aqueous paste of feces was applied to filter paper and allowed to dry at room temperature in 30%–46% relative humidity.

**Chemicals.** The effects of mild (2% iodine and 2.3% NaI) and strong (7% iodine and 5% KI) tincture of iodine (USP) were tested on feces thinly spread on filter paper. Also, 5% and concentrated solutions of ammonium hydroxide (1.4% and 28% ammonia) were tested on filter paper impregnated with oocysts in feline feces. Concentrated household ammonia was added to cover moist feline feces in bulk in capped bottles.

Other methods were described in detail elsewhere [11, 12, 16, 21, 22].

**Results**

**Number of oocysts, duration of shedding, and appearance of antibody.** Oocysts began to appear in feces on the fourth day after cats ate infected mice, and between 1,000 and one million were shed per cat on the fifth day. Peak production took place between the fifth and the eighth days, and oocyst shedding ceased about day 14. Antibody was first detected on day 9 in two out of three kittens receiving the largest inocula. On day 12, when oocyst production had almost ceased, antibody was present in all three kittens of this group, but in none of the four kittens receiving the smaller doses. By day 25, three out of four of these kittens, however, had developed antibody. Figure 3 illustrates these findings.

**Infectivity of oocysts: mortality vs. sero-conversion.** Tenfold dilutions of a suspension of sporulated oocysts were fed to groups of six mice to determine the relationships between dose and mortality, days until death, or survival (table 1). Most of the mice died from the infection, but the time until death varied with the dose. Conversely, in tests of survival of oocysts, the mean day of death per group can be used to determine the number of viable oocysts. All surviving mice were examined for antibody and for cysts; it was found that with this moderately murine-virulent strain, only two mice survived the infection.

**Development of toxoplasma infectivity in feline feces and its persistence at room temperature.** To simulate conditions of a litterbox, a bolus of feline feces containing numerous nonsporulated oocysts was placed in a paper cup and kept in a room heated to 23 C–28 C with a relative humidity of 22%–44% in Kansas in midwinter. Small portions were fed to mice daily. By the second or third day, the feces in the cup appeared dry, and on the third day they became infectious to mice. Infectivity persisted until the 14th day. Table 2 shows results of this study.

**Effects of humidity on development and survival of oocyst infectivity.** Freshly deposited feline feces containing numerous unsporulated oocysts were placed in a series of humidity gradients (table 3). A small amount of feces was removed on days 1, 2, 4, 6, 8, 11, 14, 16, 18, 23, 25, 28, 30, and 32 and tested for infectivity in
Toxoplasmosis in Cats and Man

Figure 3. Correlation of time and magnitude of production of oocysts from and antibody to *Toxoplasma gondii* in seven cats (semilog chart). Note that antibody appears in most cats after oocyst production has declined or ceased. Data on oocysts are combined from cats with identical inocula, but individual antibody titers are given. Negative determinations are connected with antibody titers by broken lines. (Redrawn from [22].)

INOCULA

• 10 INFECTION MOUSE BRAINS
• 10^{-4}DILUTION
• 10^{-6}DILUTION

YIELD PER CAT

• 15,586,000
• 2,377,000
• 314,500

![Graph showing correlation](image)

---

...period. We observed that oocysts survived for over a year in vials with 2% sulfuric acid at 4°C.

Effects of drying, tincture of iodine, and ammonia on toxoplasma oocysts in a thin layer of cat feces. In order to simulate surface contamination with feces from cats containing *Toxoplasma* under conditions where drying can occur, 10^5 washed, sporulated oocysts were mixed with cat feces, diluted with water, and applied to filter paper. Drying could be determined from the time the filter paper became stiff, which was designated time zero. Two papers were dried only, and four papers were first dried and then covered completely by one of four candidate disinfectants (figure 4). The infectivity remaining was estimated by comparison with mean survival times in table 1. Strong ammonia (28%) killed all the oocysts in 10 min, and strong tincture of iodine, in 30 min. Mild tincture of iodine and dilute ammonia (1.4%) were not much more effective...

Figure 2. A comparison of the more common oocysts found in cat feces, together with a human hair (h) (female, scalp) and red blood cells, for comparison of size. The largest oocysts, those of *Isospora elis* (l), measure about 40 x 30 μ and are about a half to a third the diameter of the human hair. The intermediate oocysts, those of *Isospora rivolta* (r), measure about 25 x 20 μ and are still easily visible with low power, although much smaller than the width of a hair. *Toxoplasma gondii* (t) oocysts are the smallest, measuring about 12 x 10 μ unsporulated and 12 x 11 μ sporulated. They are barely visible under the low power (100 x). As shown under 400x magnification, they can be compared with about two diameters of a human erythrocyte (slightly crenated). For comparison with the red blood cells, the oocysts have been suspended in isotonic saline.

mice. The untreated feces were infectious by the second or fourth day. However, oocysts recovered by flotation and sporulated in 2% sulfuric acid reached infectivity on day 1. Over concentrated sulfuric acid and in the presence of 19% relative humidity, feces were still somewhat moist on the sixth day; they appeared dry on the eighth day when *Toxoplasma* was isolated, but infectivity was lost on the 11th day. In 37% and 58% relative humidity, infectivity persisted for 11 days, and in 80% relative humidity, for 18 days. In an atmosphere saturated with water, oocysts were infectious during the entire 32-day observation...
Table 2. Development and persistence of infectivity of *Toxoplasma gondii* oocysts in feces of cats. Portions originally containing about 1,000 oocysts were fed to two mice at daily intervals.

<table>
<thead>
<tr>
<th>Days after feces were discharged</th>
<th>Result of inoculation*</th>
<th>Toxoplasma identified on smears†</th>
<th>Antibody development</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>S,S</td>
<td>ND†</td>
<td>—</td>
</tr>
<tr>
<td>1</td>
<td>S,S</td>
<td>ND</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>S,S</td>
<td>ND</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>12,14†</td>
<td>+</td>
<td>ND</td>
</tr>
<tr>
<td>4</td>
<td>5,11†</td>
<td>+</td>
<td>ND</td>
</tr>
<tr>
<td>5</td>
<td>S,S</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>16,S†</td>
<td>+</td>
<td>ND</td>
</tr>
<tr>
<td>14</td>
<td>16,27†</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>18</td>
<td>S,S</td>
<td>ND†</td>
<td>—</td>
</tr>
<tr>
<td>22</td>
<td>S,S</td>
<td>ND</td>
<td>—</td>
</tr>
</tbody>
</table>

*Note.* Feces were stored at a temperature of 23–28°C; relative humidity was 22–44%.

* S = survival of inoculated mice, number = days until death.

† In the acute infection trophozoites were looked for. The brains of mice with chronic infection were searched for cysts.

† ND = not done.

in killing oocysts than was drying alone. Although no end point was reached, even after seven days at a relative humidity of 30%–46%, only half of the mice that received these dried fecal samples died from toxoplasmosis.

Effects of household ammonia on *toxoplasma oocysts in moist feces.* Deposited cat feces were kept moist for three days to allow most of the oocysts to sporulate. Two- to three-gram portions of moist feces were placed in bottles and about 6 ml of household ammonia was added to some of the bottles, which were then capped. After indicated intervals, the ammonia was decanted and water was added to dilute the reagent; bottles were then centrifuged and the contents washed twice to remove the odor of ammonia. The washed sediment was fed to four mice, and the results were compared with feeding of fecal material not exposed to ammonia (table 4). Whereas the untreated feces showed undiminished infectivity after 24 hr, treatment for 10 min with household ammonia lowered the number of oocysts tenfold, treatment for 1 hr lowered the number 500-fold, and treatment for 3 hr killed all oocysts.

Effects of boiling water on *toxoplasma oocysts in moist feline feces.* Cat feces, kept moist for three days to allow oocysts to sporulate, were placed on two aluminum pans; to one, cat litter was added. One liter of boiling water was poured into each pan. The initial temperature in the pan without litter

---

Figure 4. Correlation of survival of oocysts from *Toxoplasma gondii* in two experiments and the killing effects of iodine and ammonia (log-log chart).
Table 4. Effect of household ammonia on infectivity of *Toxoplasma gondii* oocysts in cat feces as tested in mice.

<table>
<thead>
<tr>
<th>Exposure time</th>
<th>Household ammonia</th>
<th>Survivors/inoculated (days to death)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Water</td>
<td>0/4 (7.5)</td>
</tr>
<tr>
<td>10 min</td>
<td>0/4 (7.5)</td>
<td>0/4 (11.8)</td>
</tr>
<tr>
<td>60 min</td>
<td>0/4 (7.5)</td>
<td>0/4 (15.8)</td>
</tr>
<tr>
<td>3 hr</td>
<td>0/4 (7.0)</td>
<td>4/4</td>
</tr>
<tr>
<td>6 hr</td>
<td>0/4 (7.8)</td>
<td>4/4</td>
</tr>
<tr>
<td>24 hr</td>
<td>0/4 (7.5)</td>
<td>4/4</td>
</tr>
</tbody>
</table>

* Purex, Lakewood, California, with 5.5% ammonia.
† Armour-Dial, Chicago, Illinois, with 4.6% ammonia.

Discussion

We will first discuss some of the biologic data relating to shedding of oocysts by cats and follow this with practical recommendations as to management of the problems presented.

*Shedding of oocysts by cats.* After primary infection, weanling kittens may shed hundreds of thousands to several million toxoplasma oocysts (figure 3), which are infectious to many animals [10, 12]. However, kittens are usually infected by cysts. For example, Wallace noted that the prevalence of antibody in kittens was low until about six months of age, which suggests that they became infected mainly when they started to hunt [23]. On the other hand, infection with the feline coccidia, *I. felis* and *I. rivolta*, is commonly acquired before weaning, between four and six weeks of age.

*Visualization of oocysts in cat feces.* Toxoplasma oocysts are the smallest of those found in cat feces and are more difficult to recognize than those of the larger, more common *I. felis* and *I. rivolta*. Figure 2 shows oocysts of all three together with human hair and erythrocytes, in case means for direct measurement are not available. When magnified 100 times, both *Isospora* are easily detected, but 400-fold magnification is necessary to see the toxoplasma oocyst. Occasionally, other coccidia are found in cat feces [24]. One type that is generally shed after sporulation, and often as free isosporan sporocysts, has recently been suggested as the coccidian equivalent of *Sarcocystis*, a parasite in muscles of sheep and cattle [25, 26].

Standard techniques of flotation at 1.15 sp gr, as described in laboratory and parasitology manuals, will concentrate the oocysts in the supernatant, or a simplified technique modified for *Toxoplasma* may be used [21]. Oocysts should be looked for in the sugar solution. If any are found, the supernatant can be diluted with water, centrifuged, and replaced with saline containing a few drops of blood to compare the size of the smallest oocyst with that of red blood cells. Such examinations of stools should be done on fresh cat feces, before the oocysts have become infectious.

For sporulation, the oocysts can be transferred to 2% potassium dichromate or 2% sulfuric acid to kill the bacteria that compete for oxygen. With a shallow supernatant and with oxygenation aided by shaking, sporulation takes from one to two days at about 25 C.

*Identification of oocysts as Toxoplasma.* Identification depends on production of stages of *Toxoplasma* with typical morphology in tissue of mice, with the development of antibody measured by dye or by fluorescent-antibody tests [27, 28], against a standard strain of *Toxoplasma*. A titration (table 1) is useful to look for trophozoites in mice that die and for cysts in the survivors from which serum can also be obtained for antibody testing. With many toxoplasma strains, most mice develop a symptomatic infection.

*Appearance of antibody in relation to shedding of oocysts by cats.* Since under natural conditions an infectious dose would probably resemble the intermediate inoculum (figure 3), we feel that appearance of antibody would usually follow subsidence of production of oocysts. Similarly, in experimental kittens that became sick and died, antibody was generally absent or low [22]. As cats usually have lower titers of antibody than dogs, mice, or humans with acute toxoplasmosis [16], serologic data are diagnostically less useful in cats. However, titers are of similar magnitude.
whether determined by dye test or by indirect fluorescent-antibody test.

Development of infectivity of oocysts in cat feces. As shown in table 2, oocysts become infectious three days after a fecal specimen is shed. As shown elsewhere, the rate of oocyst sporulation depends on oxygenation and temperature and, under optimal circumstances, can be completed within 24–28 hr [11]. Some oocysts on the surface of a fecal specimen could be infectious within one day of shedding. Therefore, contrary to suggestions in the lay press, litterpans should be changed daily. Anaerobic conditions diminish infectivity; however, even after 30 days in thioglycollate broth, infectivity developed after exposure to air for five days [11].

Persistence of infectivity in cat feces. It was known from earlier reports that Toxoplasma, after separation from cat feces by flotation, could remain infectious in water for 17 months [8]. How long can infectivity persist in the untreated fecal material? At a relative humidity that varied between 22% and 44%, it persisted for 14 days (table 2) and at 80% humidity, for 18 days (table 3). After being dried completely over concentrated sulfuric acid, oocysts remained viable for about two days (table 3), but pure oocysts were destroyed by drying in less than 24 hr [16]. Evidently the moisture-holding quality of fecal material aids their survival. However, oocysts die earlier in cat feces than in water or in 2% sulfuric acid, used as a preservative.

Practical measures to disinfect cat feces. Although strong ammonia and strong tincture of iodine can be used for disinfection, their cost, objectionable smell, and the danger involved in their use would probably preclude their use on a daily basis. Addition of boiling water or application of dry heat (over 66 C) to feces appear to be more practical solutions. Burning provides a safe method of disposal of cat feces. They could also be flushed down the toilet to remove their infectivity from the immediate living environment to a sewage plant where they are greatly diluted and die naturally [29]. However, they are not likely to be killed by either the activated sludge process or trickling filters (R. E. McKinney, personal communication).

Impractical measures for disinfection. Since drying inactivates oocysts in cat feces too slowly to be useful for disinfection, the dehydrating action of cat litter is useful but cannot be depended on for disinfection. Disposal of cat feces in the garbage is not advised, since it would expose sanitation personnel to concentrated infectious material. Surface burial and composting is not advised, and deep burial at 4–6 ft, although probably safe, is too tiresome. Freezing is not efficient in killing oocysts (authors' unpublished observation), and chemical disinfectants, such as 10% formalin, 6% sodium hypochlorite, and dilute household ammonia, are undependable means for inactivation of oocysts.

What to do when finding oocysts in cat feces. First, be sure they are not the larger I. felis or I. rivolta, which are much more common than Toxoplasma (figure 2). If they correspond to toxoplasma oocysts in size, disinfect all the feces from the cat for a period of two to three weeks, after which time another examination can be performed to see whether excretion of oocysts has stopped. A pregnant woman or one who suspects she may become pregnant should avoid such a cat and should not change the litterbox.

How to handle feces of a cat that hunts or is fed raw meat. Feces and litterpans should be disinfected and feces disposed of daily in a safe manner.

Avoiding infection of indoor cats. Feeding dried, cooked, or canned food to an indoor cat that has no opportunity to hunt mice or birds essentially eliminates the risk of its acquiring toxoplasmosis. Meat that has been frozen is usually less infectious, but freezing is not a sure way of killing toxoplasma cysts [30]. Training a cat to defecate in a litterpan that is cleaned out and disinfected daily instead of outdoors decreases the risk of the cat's picking up oocyst-containing soil and ingesting oocysts. As shown in figure 3, serologic surveillance of cats will not indicate their infectivity. A seropositive cat is, however, a safer cat to have, since when reinfected, it will shed fewer oocysts or none at all [13, 16]. Neither are periodic examinations of stool useful to pinpoint the seven- to 10-day infectious period of cats. Control of the food provides the only practical means of avoiding infection.

Stray cats. Stray and outdoor cats present a difficult problem in the prevention of toxoplasmosis, since such cats have a greater chance of getting infected and since their feces are prac-
Toxoplasmosis in Cats and Man

It is practically impossible to control. Although fecal deposits are more dispersed than indoors, the instinct for cats to bury their feces in loose soil and children's sandpiles that have higher-than-surface humidity favors the survival and transmission of toxoplasma oocysts. Such outside sources of infection are likely to persist for weeks and months. Freezing does not kill oocysts efficiently but may cause greater attrition. Extreme dryness as in the southwestern United States could be expected to decrease the survival time of toxoplasma oocysts in the soil without killing them efficiently, which perhaps is borne out by the low rate of antibody to *Toxoplasma* prevalent in these areas [31]. Wearing work gloves while handling soil potentially contaminated with oocysts and washing hands before contact with the mouth and mucous membranes, should decrease the chance of acquiring infection from such soil. Dissemination may be indirect via flies and cockroaches [40, 41].

**Sandboxes.** As cats often bury their feces in children's sandboxes, these should be covered when not in use. Since there is no satisfactory way of sterilizing sand already contaminated, it is best to replace it.

**Toxoplasmosis of cats.** We found that 45% of stray cats [16] and 47% of domestic cats in Iowa and Kansas had antibody. However, toxoplasmosis as a disease is rarely diagnosed. Although some cases might be missed, toxoplasmosis is apparently not a disease of major importance in cats, since asymptomatic infection is common. After experimental infection with cysts (M-7741 strain), by mouth, nursling kittens usually die, young weanlings commonly get sick and die, but by the time kittens are old enough to hunt, disease and death from toxoplasmosis are rare [22]. When they are present, the manifestations of disease, such as diarrhea, weight loss, fever, pneumonia, and encephalitis, are not specific enough to help greatly in making a diagnosis. Antibody may still be absent when a young kitten dies from infection. With older kittens the development of antibody, as shown by a fourfold to eightfold rise in titer over a one- to two-week period, may be taken as highly suggestive of active infection. Oocysts may be shed during the early period of disease; sometimes this is accompanied by diarrhea.

**Toxoplasma in meat.** *Toxoplasma* has been found in pork, mutton, beef [4, 32], and chicken [12, 33]. Although the percentage of the meat containing *Toxoplasma* is variable and may be small, the frequency with which these meats are consumed may result in an appreciable exposure. This applies to cats that are fed raw meat [34] and to humans who eat it [6]. Serologic surveys of butchers and slaughterhouse workers [35] also suggest that heavy contamination of skin and mucous membranes may favor transmission. *Toxoplasma* in meat is killed by heating the meat throughout to 66 C (150 F). Freezing markedly decreases the number of viable cysts in the meat but cannot be depended upon to kill all organisms [30].

**Toxoplasmosis in man.** It is not known at present what percentage of people get infected by consumption of raw and undercooked meat or from the inadvertent ingestion of oocysts. The manner of infection is likely to vary in accordance with cultural patterns concerning meat-eating, contact with cats, and contact with soil, especially in warm, moist climates that favor oocyst survival. Most infections are asymptomatic, although manifestations of disease in the form of fever, pneumonia, myocarditis, hepatitis, encephalitis, and retinochoroiditis are frequently reported [18, 28, 36]. Disease occurs most commonly in babies infected in utero. For this reason, preventive measures are more important in women of childbearing age than in any other group. Pregnant women should eat only adequately cooked meat and either leave the cleaning of the cat litterpans to someone else or wear disposable gloves. There is no evidence that airborne infection is important [12], although the creation of aerosols containing feces should be avoided.

The routine testing of pregnant women for toxoplasmosis is debatable. However, if a pregnant woman develops fever, rash, or lymphadenopathy, she should be serologically tested for toxoplasmosis, whether or not she has known contact with cats. Routine surveillance of pregnancies in the United States has a high cost-efficiency ratio, since only from 1 in 1,000 to 1 in 10,000 babies are infected, and, with the observance of the preventive measures discussed, the incidence should be reduced. It appears preferable also to avoid routinely subjecting seronegative mothers to the fear of having a malformed baby. Since diagnosis is generally too late...
for a simple abortion, and, in spite of the considerable organization required for serologic surveillances, the main benefit obtained from detecting infection would be to start chemotherapy during pregnancy rather than after delivery.

Determination of levels of IgM on all newborn infants may be more beneficial since this is a screen for many infections [37]. Infants with high titers of IgM can then be tested for IgM antibody to Toxoplasma [38]. Although this test is not as sensitive as initially hoped [39], it focuses on those potentially sick who can be observed, investigated further, and if necessary, treated [18, 28].

Cats as pets. If their diet can be controlled, cats can be kept as relatively safe pets in regard to toxoplasmosis. To the extent that their diet cannot be controlled, cats are a risk, although a small one. The pleasure that people derive from their pets must be weighed against the risk of toxoplasmosis and of other petborne diseases, such as larva migrans, hookworm and other nematodes, tapeworms, fleas, ringworm, salmonellosis, streptococcosis, leptospirosis, pasteurellosis, and occasionally rabies. Most pleasures have a price, and so does the habit of the gourmet, or faddist, of eating raw or very rare meat. Few people give up consistent pleasures for infrequent diseases, and when the dangers and risk factors are known, people must choose. However, since an infectious cat exposes not only his owner, but also neighbors, ethical questions are raised. Stray cats present a public health problem that is even more difficult to solve than that of stray dogs, since leash laws and fences provide an approach to the control of potentially infected canine feces (larva migrans). Immune cats (those with antibody) are safer pets, since after reinfection they shed oocysts only briefly and in reduced numbers or not at all. The development of a safe vaccine for cats and man would further diminish the frequent risk of toxoplasmosis. However, much preparatory work needs to be done before a vaccine can be developed.

References

Toxoplasmosis in Cats and Man


