infectious organisms, especially if the animals have diarrhea.

A recent study detected *Salmonella* in milk filters on 3% of dairy farms in Southwestern Ontario. Human *Salmonella* infection, of a serotype matching the milk filter isolate, was detected in 24% of the families with infected milk filters (unpublished observations).

Despite public health efforts, a very high proportion of farm families still drink raw milk. Their knowledge of the risks may not be as extensive as we have thought. In recent interviews with 43 dairy farmers and their wives, only 37 (86%) had heard about salmonellosis in animals and only 36 (84%) had heard about salmonellosis in humans (unpublished observations). Of these, 24 were aware that people might get the disease from improperly cooked poultry, 21 from raw milk, 19 from animals, 15 from water, 11 from food poisoning, 11 from other people, and 10 from raw eggs. If the population isn't fully informed about *Salmonella* and its transmission, we can expect even less knowledge about *Campylobacter* and *Yersinia* infections.

Despite public health efforts, a very high proportion of farm families still drink raw milk.

Even farm families who have heard of these infections may not feel that they are at significant risk from raw milk. While it appears that regular users may develop partial immunity to enteric organisms over time, they remain vulnerable to heavy infections or to new strains. When *Salmonella muenster* first affected Ontario dairy herds, many dairy farm families developed illness. Also many rural people do not appreciate the vulnerability of the very young, the elderly, or the debilitated to these infections, either through direct exposure or through secondary spread.

Preventing Zoonoses in Farm Families

In our health unit, we estimate that approximately two-thirds of the zoonoses occurring in farm families are preventable. For this reason we suggest that investigations by public health inspectors of individual cases of *Salmonella*, *Campylobacter*, and *Yersinia* infections are rewarding in rural residents. The focus should be on health education in a number of basic areas:

1. *raw milk* — provide information on the risks; advise people never to use raw milk themselves or to provide it to anyone else; give instructions for home pasteurization.
2. *personal hygiene* — emphasize handwashing after being in the barn or handling animals, poultry or eggs, and always before eating or smoking; emphasize good hygiene for children and the risks to toddlers in barns and hen houses.
3. *wells* — test water supplies regularly and correct deficiencies.
4. *farm visits* — advise groups organizing farm visits never to serve raw milk, make sure the farm water supply is safe, and ensure handwashing after the visitors have been in the barn or around animals.

Veterinarians are often the first to identify problems on a farm, and as such, have a unique opportunity to offer advice on the prevention of human disease through emphasis on personal hygiene and avoidance of raw milk consumption.

TABLE III
Source of *Salmonella* Infections by Residence within Perth County, 1979-85

Source of Infection	Place of Residence Rural	Town/Village	City
Raw milk consumption	42%	0%	3%
Farm animal contact	19	15	3
Food poisoning/handling	9	46	43
Person to person transmission	4	13	13
Contaminated water	4	0	0
Travel	3	3	3
Other	1	0	1
Unknown	18	23	35

TABLE IV
Source of *Campylobacter* Infections by Residence within Perth County, 1979-85

Source of Infection	Place of Residence Rural	Town/Village	City
Raw milk consumption	39%	21%	15%
Farm animal contact	14	4	5
Contaminated water	10	0	2
Person to person transmission	9	14	7
Pets/other animals	4	14	17
Food poisoning/handling	1	7	10
Travel	1	0	5
Other	3	0	2
Unknown	17	39	37

Toxoplasmosis as a Significant Disease in Man and Animals with Special Reference to Preventive Measures by the Farm Community

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In man, *disease* caused by *Toxoplasma gondii* occurs occasionally, with variable significance; in the majority of infections, illness is brief and is likely to be misdiagnosed as "the flu". On the other hand, *infection* of man by *Toxoplasma gondii* is very common, with the prevalence rising to and levelling at approximately 50%, by 25 years of age (1). Apparently, about half of the population unwittingly makes it through life without ever being infected.

Methods of Infection and Possible Consequences

Depending on lifetime eating habits, sanitation, and chance, there are several

methods by which man is known to become infected.

a) *prenatal transmission*

The probabilities and consequences of human congenital infection have been described (2). With seroconversion during pregnancy, approximately 40% of babies become infected. Of prenatally infected infants, about 60% are asymptomatic. Of symptomatic babies, approximately 44% experience generalized disease accompanied by one or more of splenomegaly, jaundice, fever, anemia, hepatomegaly, lymphadenopathy, pneumonia, rash, retinochoroiditis, hypothermia, and convulsions. In the remaining symptomatic babies, neurologic manifestations predominate, with a combination of retinochoroiditis, abnormal spinal fluid, anemia, convulsions, intracranial calcification, internal hydrocephalus, fever, splenomegaly, lymphadenopathy, hepatomegaly, and microcephaly. In both of the symptomatic groups, the mortality rate is about 12%.

In man, disease *caused by* Toxoplasma gondii *occurs occasionally, with variable significance; in the majority of infections, illness is brief and is likely to be misdiagnosed as "the flu".*

b) *ingestion of viable cysts in meat*

Bradyzoites, released by digestion of cysts in meat, become rapidly-dividing tachyzoites which invade many body tissues (acute stage of toxoplasmosis) until their transformation into cysts of dormant bradyzoites, by factors of acquired resistance. A dramatic episode, illustrating the clinical consequences of such an event, has been described (3). Five medical students ate rare hamburger at the same place, on the same night. The illness of acute toxoplasmosis was apparent in all, after eight to 13 days; fever, headache, myalgia, lymphadenopathy and splenomegaly, accompanied by an extremely high and rising titer in the Sabin-Feldman dye test. None died, and all were essentially normal within approximately two weeks, although myalgia persisted in some for another month. Such a syndrome, in any individual case, is quite likely to be misdiagnosed as "the flu", in the absence of serology. Subsequent infections are unlikely to result ever again in the clinical signs of acute toxoplasmosis, due to acquired resistance to tachyzoite multiplication and invasion. This resistance is likely to be maintained for life by the presence of microscopic cysts of dormant but viable bradyzoites. Human death occurs occasionally as a result of acute toxoplasmosis and is sometimes attributed to "widespread focal myocarditis" (4).

c) *ingestion of infective oocysts from the feces of cats*

Until the late 1960's there was no adequate explanation to account for the presence of microscopically visible cysts of *Toxoplasma gondii* in tissue sections of the plant-eating animals, such as cattle and sheep. It was then demonstrated (5, 6) that only felids (7), were capable of shedding oocysts of *Toxoplasma gondii* in their feces. This provided a means by which herbivorous animals could become infected, namely by the ingestion of feed contaminated with feline feces. Uncontrolled populations of unfed cats are tolerated on almost all farms for rodent control, and all classically use the granary as a litter box. Dozens, if not hundreds, of such fecal deposits can be regularly ground and mixed with the grain and fed to farm animals. Since this discovery (5, 6), the news media and even medical literature have belabored the possibility of fecal transmission from cats directly to man, with particular reference to pregnant women. To be sure, attention is warranted to prevent the ingestion of cat feces, but there appears to be a greater risk of acquiring toxoplasmosis by eating undercooked ("pink") meat. As such, the culprit cat is much more likely to be found in the barn than in the living room.

Even so, there are many qualifications as to the shedding of oocysts in the feces of any cats. On farms, *Toxoplasma gondii* is likely maintained by cannibalism in a high proportion of the mouse population. Unfed cats on farms are most likely infected by eating viable cysts in mice. Three to five days after a cat's FIRST infection by cysts, oocysts are shed in the feces, often in large numbers, but only for approximately two weeks (8). Subsequent infections are considered not to be accompanied by the fecal shedding of any (or many) oocysts, with the exception of second infections in cats in which the primary infection occurred under 13 weeks of age (9). Barn cats, feeding largely on mice, are very likely to be infected repeatedly and be immune to shedding oocysts within six months. It is therefore probable that the feces of younger barn cats in the feed, are largely, if not exclusively, responsible for infection in meat-producing livestock. Vaccination of cats, to prevent the shedding of oocysts, has been investigated and efficacy has been demonstrated (10), but a vaccine is not yet commercially available.

Attention is warranted to prevent the ingestion of cat feces, but there appears to be a greater risk of acquiring toxoplasmosis by eating undercooked ("pink") meat.

d) *ingestion of viable* Toxoplasma gondii *in the milk of goats*

There is strong circumstantial evidence that the consumption of unpasteurized milk of goats was the source of clinical toxoplasmosis in man on two occasions (11, 12). In another similar report (13), *Toxoplasma gondii* was isolated, by inoculation of mice, from the milk of one of the goats in question.

e) *being recipient of infected organ transplants*

Two heart transplant recipients developed significant toxoplasmosis shortly after surgery (14). Neither recipient had evidence of exposure to *Toxoplasma gondii* before transplantation and both donors had serological evidence of recently-acquired infection at the time of transplantation. One patient died on postoperative day 30, with severe toxoplasmic myocarditis and multiple foci of necrotizing toxoplasmic encephalitis. The second patient survived, but *Toxoplasma gondii* was isolated from material aspirated from one of three brain abscesses, considered responsible for postoperative "confusion". The donor hearts were

considered to be the most likely source, and combined with the immunocompromised state of organ recipients which occurs due to the necessity of antirejection drugs, the authors strongly recommended using transplant organs only from seronegative donors.

f) *being recipient of blood which contains* Toxoplasma gondii
Four patients with acute leukemia developed disseminated toxoplasmosis following leukocyte transfusions (15). Serological data obtained about the donors retrospectively, revealed elevated antitoxoplasma titers. *Toxoplasma gondii* was found in several tissues at postmortem of three transfusion recipients who died. The authors noted that the parasite has been recovered from the buffy coat of patients with toxoplasmosis and that the organisms are known to retain their viability after suspension in citrated blood and storage at 5°C for up to 50 days. They cautioned that seropositive donors should be avoided, particularly when giving leukocyte-rich blood products to patients receiving cancer chemotherapy or immunosuppressive agents.

Interpretation of Serology and Future Infection

In man and animals, a negative titer to *Toxoplasma gondii* means that infection has not occurred up to the time of testing. Seronegative status of humans could be considered desirable, except that these individuals are vulnerable to illness following their first infection. Additionally, infected cats are seronegative during the brief period when oocysts are shed in their feces (16).

In man and animals, a positive titer means that infection by *Toxoplasma gondii* has occurred at least once. If the titer is low and the individual is clinically normal, infection likely occurred some months or years earlier and the parasite is present within microscopic cysts as dormant but viable bradyzoites. If the titer is high and the individual is clinically normal, there has likely been a recent exposure (perhaps days or weeks) for at least the second time, i.e. an anamnestic response. If the titer is high and the individual is ill, it is likely that the acute stage of toxoplasmosis (after a first infection, within the last few weeks) is being experienced.

It has become increasingly apparent that seropositive humans are not necessarily immune to clinical toxoplasmosis, depending on future medical events. For most, the cysts of dormant bradyzoites in their tissues will confer a lifetime of resistance. For some, however, there is a risk of re-activation of dormant cysts to the acute disseminated stage. Such an event could follow the decline in body defences brought about by the use of corticosteroids, antirejection drugs, chemotherapy for cancer, or the development of AIDS. As far as is known, seropositive women will not transmit toxoplasmosis to future offspring (17).

Clinical Toxoplasmosis in Animals

Acute toxoplasmosis causing death is occasionally reported at postmortem in cats (18), dogs (19), and swine (20). Cattle (21) and horses (22) can be infected and can host viable cysts of bradyzoites in edible tissues, but clinical disease is considered to be unusual. The most significant problem due to toxoplasma infection in veterinary practice is abortion in sheep (23, 24) and goats (25, 26).

The focus of prevention is directed towards that admirable hunter, the barn cat.

To complicate matters further, *Toxoplasma gondii* may be spread venereally in the semen of rams (27) and bucks (28). Ewes, having aborted once due to toxoplasmosis, are unlikely to do so again (29, 30) and are likely worth keeping. Does however, may transmit prenatal infection a second time (31), and consideration might be given to culling after a first congenital infection is confirmed. The problem with culling such does is finding seronegative replacement stock, when seropositive goats are so common; for example, 23% in Montana (32) to 55% in Ontario (33).

Prevention of Toxoplasmosis on Farms

The following suggestions are made regarding toxoplasmosis on the farm, with particular reference to premises with sheep or goats.

1. Confirm the etiology of all abortions, by laboratory submission of the placenta, fetus, and serum samples (at the time of abortion) from the dam. The placenta is particularly important to submit, even if it has to be retrieved "from the barnyard and hosed down".
2. Maintain an "adult only" population of barn cats by spaying females and driving new arrivals away. Adult cats are likely to have acquired resistance and are thus unlikely to shed oocysts in their feces.
3. Remove placentas and aborted material from access by cats (or rodents), as a potential source of further infection (34).
4. Cats and man should never drink unpasteurized milk of goats.
5. Cats and man should never eat undercooked mutton, chevon, pork, beef, or poultry.
6. All feces from cats should be carefully removed from grain and hay, which are fed to meat-producing animals. Cats should be denied access to stored grain, e.g. by fencing off the granary with chicken wire.
7. Cats in commerical feed mills should be managed in a similar manner, with the same preventive care regarding infection and access to stored feed.
8. Consider keeping ewes that have aborted due to confirmed toxoplasmosis, unless there are other reasons to cull.
9. Consider culling does that have aborted due to confirmed toxoplasmosis, as they may do so again.
10. Submit serum from sires of offspring aborted due to confirmed toxoplasmosis. Strongly consider culling seropositive bucks, as prolonged excretion of *Toxoplasma gondii* has been demonstrated in the semen of goats (28).

Conclusion

Further information regarding other preventive measures, with particular reference to direct transmission from the feces of cats to man has been reviewed recently (35).

Attention is drawn herein to the potential seriousness of toxoplasmosis in man and to the most probable source of infection being via meat produced on the farm. The focus of prevention is directed towards that admirable hunter, the barn cat. *Toxoplasma gondii* will continue to exist via cannibalism in mice. As such, it will always be "waiting" to gain access into its definitive host, the susceptible cat. Veterinarians can help to minimize the occurrence of

toxoplasma cysts in meat, by educating the farm community about preventive measures.

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Q Fever — Human Disease in Ontario

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Although Q fever is not a new disease in Canada, interest in this zoonotic infection has been renewed in the past few years with the recognition of greater numbers of cases in Ontario, Quebec, and the Maritime provinces. In Atlantic Canada, it is an important and relatively common cause of atypical pneumonia. In Ontario, 30-45 cases of acute Q fever have been reported in each of the past three years. This is probably an underestimate of the true incidence of disease in the province as most cases are either unrecognized or unreported.

Epidemiology

Q fever is caused by *Coxiella burnetii*, a small, pleomorphic bacterium that is taxonomically the sole member of the genus *Coxiella* of the Family *Rickettsiaceae*. It is an obligate intracellular parasite that replicates in the cytoplasm of eukaryotic cells (1). The organism may infect a wide variety of wild and domestic animals, but cattle, goats, and sheep appear to play a major role in transmission of infection to humans. Q fever is known to occur nearly worldwide, but the greatest incidence of disease occurs in rural areas where livestock are kept. *Coxiella burnetii* is shed in the urine, feces, milk, and products of conception of infected animals. Gravid ewes shed particularly large numbers of organisms, especially at parturition; the placenta may contain as many as 10^9 organisms per gram of tissue. The organism may survive in the environment for prolonged periods of time, probably because of spore formation. Infection is usually acquired by inhalation of infectious aerosols, although ingestion of contaminated unpasteurized milk has also been considered to be a possible route of infection.

Q fever may occur sporadically or in epidemics. Most sporadic cases occur in