REVIEW

Congenital toxoplasmosis in the United Kingdom: to screen or not to screen?

R E Gilbert, C S Peckham

The United Kingdom National Screening Committee recently reviewed the evidence for prenatal and neonatal screening for toxoplasma infection and concluded that there was insufficient evidence to recommend screening in the United Kingdom. This issue will need to be revisited as new information or treatments become available. In this paper, the extent to which the research evidence on toxoplasma infection meets the criteria that need to be fulfilled for the introduction of screening in the United Kingdom is reviewed.

Toxoplasma gondii occurs worldwide and is one of the most common parasitic infections in humans. Infection is acquired by ingestion of viable tissue cysts in undercooked meat, or of oocysts excreted by cats and contaminating soil or water. When primary infection occurs during pregnancy, Toxoplasma gondii can be transmitted from the mother through the placenta to her fetus. In a few cases, congenital infection causes neurological or visual impairment, or death. Infection in the mother or congenital infection in the child are usually asymptomatic and can only be detected by serological screening for toxoplasma specific antibodies. The principal screening options are prenatal serological screening followed by maternal antibiotic treatment to prevent fetal infection or fetal damage and neonatal screening, based on filter paper blood spots, followed by treatment of infected infants to reduce the risk of damage by the parasite to the eyes and brain.

There is no consensus on the most appropriate screening or treatment strategy. Denmark, Sweden, and Massachusetts offer neonatal screening, whereas prenatal screening, which operates in France, Switzerland, and parts of Italy, involves monthly retesting of susceptible pregnant women compared with 3 monthly retesting elsewhere. Treatment also varies. For example, in Austria, infected women are treated with a pyrimethamine-sulphonamide combination whereas most other centres use spiramycin, which has fewer adverse effects, as first line treatment. Infected women are usually treated throughout pregnancy, but short courses of treatment have been used in The Netherlands and Norway. Finally, almost all infected children are treated but the duration varies from 3 months in Denmark to 2 years in parts of France. These differences reflect the lack of reliable evidence for the effectiveness of treatment during pregnancy or infancy.

PRENATAL SCREENING
Susceptibility to toxoplasma infection in pregnancy

The proportion of pregnant women who are susceptible to toxoplasma infection is currently 80% to 90% in northern Europe (United Kingdom, Norway, Sweden), 50% in France, and 20% in parts of Brazil. The incidence of maternal infection estimated from population based cohort studies ranges from 1 to 8/1000 susceptible pregnancies, with the highest reported rates in France (table 1). The prevalence of IgG antibodies in the childbearing age group indicating previous toxoplasma infection, has fallen over the past 3 decades in many European countries leaving more women susceptible to infection during pregnancy. The birth prevalence of congenital toxoplasmosis ranges from <1/10 000 live births in Sweden and Massachusetts, 3/10 000 in Brazil, to an estimated 10/10 000 live births in France. Contemporary data on the incidence of maternal infection and birth prevalence of congenital toxoplasmosis are not available for the United Kingdom. However, figures extrapolated from Norway, Sweden, and Massachusetts, which have similarities in seroprevalence, culture, and climate, are consistent with the expected decline from previous incidences in the United Kingdom.

Risk of mother to child transmission of infection, clinical manifestations, and impairment

The risk of mother to child transmission of infection rises steeply with the gestational age at maternal seroconversion. About 5% of women who seroconvert at 12 weeks of gestation and 80% of those who seroconvert just before delivery transmit infection to their infant (fig 1). Conversely, the risk of clinical signs (lesions in the brain or retina) in an infected child decreases from about 60% for women who seroconvert at 12 weeks gestation to about 5% for those who seroconvert just before delivery (fig 2 A). Given the relation between the risks of infection and clinical signs, the risk of giving birth to a child with clinical signs is greatest (10%) for women who seroconvert between 24 and 30 weeks of pregnancy and lowest (about 5%) for women who seroconvert before 12 weeks or at term (fig 2 B).

Abbreviations: EMSCOT, European multicentre study on congenital toxoplasmosis; IgG, immunoglobulin G; IgM, immunoglobulin M

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Over 95% of children with congenital toxoplasmosis seem to be developmentally normal. Stillbirth or postnatal death occurs in about 1% of infected babies and a further 2% have severe neurological impairment. The prevalence of less severe developmental impairment, which might not be detected until school age, is not known due to the lack of long-term follow up studies of children identified by screening. Clinical signs in the brain or eyes are found in about one in six infected infants. Intracranial calcifications are usually detectable at birth and affect 9% of infected children and about 2% of infected children have ventricular dilatation detected on cranial ultrasound. Eye lesions (retinochoroiditis), caused by reactivation of latent (bradyzoite) cysts in the retina and the associated inflammatory reaction, can appear at any time during childhood, or even adult life. Cohort studies based on children who were treated prenatally and postnatally reported that 10% of children have retinochoroiditis detected during infancy, and 23% have at least one lesion by 7 years. Of these, up to half have some degree of unilateral impairment.

Detection of maternal infection

Prenatal screening for toxoplasma infection involves monthly or 3 monthly testing of women with undetectable antibodies to T. gondii at their first prenatal test to detect seroconversion. To detect infection occurring early in pregnancy, women who are immunoglobulin G (IgG) and IgM positive at their first prenatal test, undergo further tests for high or rising IgG titre, low IgG avidity, IgA antibodies, or a combination of these. None of these tests for recent infection reliably determine the timing of infection and most women identified will have acquired infection before conception and are not at risk of fetal infection. How often maternal infections are missed has not been assessed.

Table 1 Incidence of maternal infection* and birth prevalence of congenital toxoplasmosis†

<table>
<thead>
<tr>
<th>Study, country, and recruitment period</th>
<th>Pregnant women immune n (%)</th>
<th>Incidence (95% CI)/1000 susceptible pregnancies</th>
<th>Birth prevalence (95% CI)/10000 live births</th>
</tr>
</thead>
<tbody>
<tr>
<td>Netherlands 1987–88</td>
<td>28049 (45)</td>
<td>5.4 (4.1 to 7.1)</td>
<td>4.3 (2.2 to 7.5)</td>
</tr>
<tr>
<td>Finland 1988–89</td>
<td>16733 (20)</td>
<td>3.4 (2.3 to 4.9)</td>
<td>2.4 (0.7 to 6.1)</td>
</tr>
<tr>
<td>Denmark 1992–96</td>
<td>89873 (28)</td>
<td>2.9 (2.4 to 3.4)</td>
<td>3.0 (1.98 to 4.37)</td>
</tr>
<tr>
<td>France 1995</td>
<td>12919 (54)</td>
<td>8.1 (5.7 to 11.7)</td>
<td>101</td>
</tr>
<tr>
<td>Sweden 1997–99</td>
<td>40978 (14)</td>
<td>0.5 (0.26 to 0.89)</td>
<td>0.73 (0.15 to 2.14)</td>
</tr>
<tr>
<td>Norway 1992–93</td>
<td>33740 (11)</td>
<td>0.82 (0.48 to 1.32)</td>
<td>3.3 (1.63 to 5.83)</td>
</tr>
<tr>
<td>Massachusetts 1986–94</td>
<td>635000 (10)</td>
<td>0.8 (0.6 to 1.1)</td>
<td>3.3 (1.63 to 5.83)</td>
</tr>
<tr>
<td>Poland 1998–99</td>
<td>27516 (59)</td>
<td>4.7 (2.5 to 8.1)</td>
<td></td>
</tr>
</tbody>
</table>

*For each susceptible 9 month pregnancy based on seroconverting women; †includes any live born infant with congenital toxoplasmosis, whether symptomatic or not; ‡estimated assuming transmission risk of 29% and mean interval between negative and positive test of 26 months.

Figure 1 Risk of congenital infection by duration of gestation at maternal seroconversion based on a cohort of 354 seroconverting women reported by Dunn et al.

Figure 2 Risk of developing clinical signs (not necessarily symptomatic) before age 3 years according to gestational age at maternal seroconversion: (a) in the presence of congenital infection; (b) when infection status of the fetus is not known (dotted lines are 95% CI given model in Dunn et al.).
Diagnosis of fetal infection

Once maternal infection has been confirmed, treatment is prescribed and the woman referred for amniocentesis after 14 weeks of gestation for fetal diagnosis. Results of polymerase chain reaction (PCR) analysis of amniotic fluid are used to determine subsequent treatment. This is highly specific but a negative PCR result (sensitivity 64% (95% confidence interval 53% to 75%)) does not rule out fetal infection. Fetal diagnosis is required if termination is being considered for congenital toxoplasmosis. However, termination is rare unless there is ultrasound evidence of intracranial lesions indicating an increased risk of impairment.26 As these signs have rarely been reported before 22 weeks of gestation, late termination is required.26

Treatment during pregnancy

In most centres, infected women are initially treated with spiramycin to reduce the risks of mother to child transmission.7 After a positive fetal diagnosis, pyrimethamine-sulphonamide combination (sulphadiazine or sulphadoxine) is prescribed. In some centres, women infected in late pregnancy and at high risk of fetal infection are treated with pyrimethamine-sulphonamide without undergoing amniocentesis. Adverse effects of spiramycin are rare and not serious. However, pyrimethamine-sulphonamide is associated with dose related bone marrow suppression and severe allergic reactions, and may be teratogenic.27

No randomised controlled trials have reported the effectiveness of prenatal treatment.26–28 Earlier studies showing benefits of treatment used historical controls and did not take account of gestation at seroconversion.29 However, more recent analyses of retrospective cohort studies took into account the strong confounding effect of gestation at maternal infection. Two studies assessed the effect of timing and type of treatment on mother to child transmission and found no evidence for an effect of prenatal treatment (table 2).13 14 Also, an ecological comparison of cohorts in Vienna, Denmark, and The Netherlands with women in Lyon, where monthly retesting of susceptible women allowed early identification and treatment, showed no evidence that intensive management was beneficial (table 3).7

Less information is available on the effect of prenatal treatment on intracranial and ocular lesions in infected children. Three recent analyses of cohort studies, which took account of confounding due to gestation at maternal infection, reported conflicting results (tables 2 and 3).7–13 In the Lyon cohort, the risk of clinical manifestations was similar in treated and untreated mother-child pairs.7 The ecological comparison

### Table 2  Results from cohort studies on the effect of prenatal treatment

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Mother to child transmission</th>
<th>Clinical manifestations by 3 years</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lyon cohort 1987 to 1995:</td>
<td>(n=554 seroconverting women)</td>
<td>(n=181 infected children)</td>
<td></td>
</tr>
<tr>
<td>Timing of treatment after seroconversion</td>
<td>&lt;4 weeks: 1.0</td>
<td>&lt;4 weeks: 1.0</td>
<td>No significant effect</td>
</tr>
<tr>
<td></td>
<td>4–7 weeks: 1.29 (0.61 to 2.73)</td>
<td>&gt;7 weeks: 1.40 (0.60 to 3.31)</td>
<td></td>
</tr>
<tr>
<td>Type of treatment</td>
<td>Pyrimethamine-S 0.91 (0.45 to 1.84)</td>
<td>Spiramycin alone 1.0 (0.45 to 2.04)</td>
<td>No significant effect</td>
</tr>
<tr>
<td></td>
<td>None 1.06 (0.37 to 3.03)</td>
<td>Pyrimethamine-S 0.93 (0.35 to 5.16)</td>
<td></td>
</tr>
</tbody>
</table>

Combined analysis of cohorts from Brussels, Lille, Reims, Oslo, and Helsinki: (n=144 seroconverting women) (n=64 infected children)

| Timing of treatment after maternal infection | p=0.3 | p=0.02 | Significant reduction in clinical manifestations |
| Timing of antibiotic | p=0.05 | p<0.05 | No significant effect |
| Treatment v none | p=0.7 | 0.3 (0.10 to 0.86) | Significant reduction in clinical manifestations |

Pyrimethamine=S=combined treatment of pyrimethamine and sulphadiazine.

### Table 3  Ecological comparison of mother to child transmission and clinical manifestations according to centre

<table>
<thead>
<tr>
<th>Centre (retesting interval)</th>
<th>Infected women (% treated prenatally)</th>
<th>Children with congenital toxoplasmosis (with clinical manifestations)</th>
<th>Adjusted* relative risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mother to child transmission</td>
<td>Clinical manifestations by age 3 years</td>
</tr>
<tr>
<td>Lyon</td>
<td>549 (94)</td>
<td>156* (42)*</td>
<td>1.00</td>
</tr>
<tr>
<td>Austria</td>
<td>131 (90)</td>
<td>34* (3)</td>
<td>1.24 (0.88 to 1.59)</td>
</tr>
<tr>
<td>Denmark</td>
<td>123 (9)</td>
<td>26 (5)</td>
<td>0.59 (0.41 to 0.81)</td>
</tr>
<tr>
<td>The Netherlands</td>
<td>51 (50)</td>
<td>12 (4)</td>
<td>0.65 (0.37 to 1.01)</td>
</tr>
</tbody>
</table>

*Adjusted for gestation at seroconversion according to Lyon cohort; 11–13 refers to terminations for congenital toxoplasmosis.

Retesting and treatment protocols:

Lyon: Monthly retesting. Spiramycin first line treatment for maternal infection. Pyrimethamine-sulphadiazine given for fetal infection diagnosed or maternal infection in third trimester. Treatment continued until delivery.

Austria: Retesting 3 monthly. Pyrimethamine-sulphadiazine first line treatment for maternal infection. Spiramycin given if fetal diagnosis negative. Treatment continued until delivery.

Denmark: Retrospective testing of first stored prenatal sample in mothers of IgG positive neonates based on Guthrie card testing. No treatment.

The Netherlands: Retesting at 4–8 week intervals. Spiramycin-sulphonamide given for 3 weeks for maternal infection, repeated once after 2 weeks without treatment. No fetal diagnosis.
found no clear evidence that intensive treatment protocols were beneficial\(^7\); a lower risk of clinical manifestations in Austria was based on three patients and may be a chance finding. However, there was a significant reduction in clinical manifestations found in the analysis of cohorts in Brussels, Reims, Lille, Oslo, and Helsinki. This could be partly explained by referral to fetal medicine centres of more severely affected untreated pregnancies detected late in pregnancy.\(^8\) None of the studies showed that pyrimethamine-sulphadiazine was more effective than spiramycin. This casts doubt on the value of prenatal diagnosis to determine the type of prenatal treatment. No published studies have examined the effect of prenatal treatment on functional impairment in later childhood. However, a prospective cohort study, The European multicentre study on congenital toxoplasmosis (EMSCOT), is currently examining this outcome.

**Timing of prenatal treatment**

The rationale for monthly retesting of susceptible women in France is that the earlier treatment is given, the greater the effect on mother to child transmission. However, there is no evidence for an effect of earlier treatment on mother to child transmission (tables 2 and 3)\(^9\)\(^10\); nor is there direct evidence in humans for a latent phase between maternal and fetal infection when treatment might have an effect. Animal studies show that mother to fetus transmission of infection occurs during maternal parasitaemia, which ceases as the maternal antibody response develops.\(^11\) If the same were true in humans, treatment after serological detection of maternal infection would be given too late to prevent transmission. The strong association between the gestation at maternal seroconversion and the risk of fetal infection (fetuses exposed to an infected mother for longest have the lowest risk of infection) supports the hypothesis that, in most cases, mother to child transmission occurs soon after maternal infection. Evidence is also lacking for a latent phase between fetal infection and tissue damage, when treatment might reduce clinical signs and symptoms. Studies in animals show that once infection has occurred, transformation from the free tachyzoite to the bradyzoite cyst form, which is not susceptible to antibiotics,\(^12\) occurs within days of infection in immune competent people.\(^13\) These findings suggest the possibility that prenatal treatment may be given too late to have an effect on damage to fetal tissues.

**Postnatal diagnosis of congenital toxoplasmosis**

Overall about 75% of children born to infected women do not acquire congenital toxoplasmosis.\(^1\) Consequently, it is important to determine congenital infection status as soon as possible to limit treatment to infected children. The reference standard for congenital toxoplasmosis is based on persistence or disappearance of IgG antibodies at 12 months.\(^2\) Early positive diagnosis can be based on the PCR analysis of amniotic fluid, or on neonatal IgM or IgA, as these tests are highly specific. However, the sensitivity of IgM or IgA in early infancy is 85% (95% CI 71% to 99%) for children born to untreated women\(^3\) and lower (73% (95% CI 62 to 82%)) in infants of women treated prenatally. Decisions about postnatal treatment must take account of the pretest probability of congenital toxoplasmosis. For example, children born to women who seroconverted before 20 weeks have a low risk of infection (<20%): if postnatal IgM or IgA is negative such children are unlikely to be infected.

**Postnatal treatment for congenital toxoplasmosis**

Postnatal treatment usually consists of an alternating regimen of spiramycin and pyrimethamine-sulphonamide. The duration of treatment varies from 3 months to 24 months in some French centres, although in one Dutch study, infected children received no treatment.\(^7\) Treatment aims to reduce the risk of new eye lesions and neurological impairment. Evidence for the effectiveness of postnatal treatment is limited to randomised controlled trials of treatment in HIV infected children at risk of disseminated toxoplasmosis.\(^14\)\(^15\) However, in healthy immunocompetent infants with congenital toxoplasmosis, the parasite is likely to be in the form of latent bradyzoite cysts that are not penetrated by antibiotics.\(^16\)\(^17\) An important unanswered question is whether there is persistence or re-emergence of the free tachyzoite in immune competent infants and fetuses that would benefit from prolonged antibiotic treatment.

**Implementation issues**

Implementation of prenatal screening would require systems to ensure repeated blood sampling of susceptible pregnant women and laboratory facilities for serological and PCR testing. Such systems would need to be established in the United Kingdom. Quality control of laboratory testing would be important as there is evidence of marked variation in the performance of serological and PCR testing.\(^18\)\(^19\) Also, clinical services would need to be able to deal with the many women with false positive test results. Using a single screening test with 99% specificity, each time 1000 uninfected women are tested there will be 10 false positive results. False positive test results can be reduced by performing several tests on the same sample and on repeat samples, but at additional cost. A further problem is how to manage the many women identified by tests for recent infection (rising IgG titre or low IgG avidity), most of whom were infected before conception. In some centres, such women account for 80% of all treated women.

The potential harms and benefits of prenatal screening are summarised in table 4. There are several ways in which adverse effects can be reduced. Firstly, fewer repeated tests of susceptible women would lead to fewer false positive results and reduce costs, and, as there is no clear evidence that early treatment is beneficial, may not adversely affect fetal outcome. Secondly, tests of recent infection in women before 20 weeks of gestation mainly identify women infected before conception or at very low risk of adverse fetal outcome. Avoidance of these tests would reduce harm due to unnecessary investigations, treatment, and termination.\(^20\) Thirdly, as there is no evidence that pyrimethamine-sulphonamide is more effective than spiramycin, use of such combination treatment could be stopped, thereby avoiding serious side effects. Fourthly, amniocentesis for fetal diagnosis could be discontinued, as there is no evidence that results lead to a beneficial change in treatment. However, fetal diagnosis is required if termination of pregnancy is being considered. Finally, adverse effects of postnatal treatment could be reduced by limiting antibiotic treatment to children at high risk of congenital toxoplasmosis (based on positive neonatal IgM/IgA results) and by limiting the duration of treatment.

**NEONATAL SCREENING**

Neonatal screening identifies neonates with congenital toxoplasmosis, most of whom are asymptomatic and would not be detected by routine paediatric surveillance. The main potential benefits are treatment to prevent the emergence of new eye lesions after birth, to reduce the risk of neurological impairment, and to allow early identification of children with visual or neurological impairment to offer remedial treatment. Neonatal screening is based on the detection of specific IgM (and in some centres IgA)\(^21\) in neonatal filter paper blood spots. Detection of IgM has been reported to identify about 85% of infected children\(^22\)\(^23\) with a positive predictive value of about 50%\(^24\) (in settings where birth prevalence ranges from 1 to 5/10 000 live births).\(^25\) However, neonatal screening misses a few severely affected children who are IgM negative at birth\(^26\) and were probably infected in early pregnancy. Some of these would be detected due to clinical manifestations.
Treatment regimens vary from 3 months of continuous pyrimethamine-sulphonamide in Denmark, to 12 months in Massachusetts. There have been no comparative studies of the effect of postnatal treatment in children identified by screening and adverse effects, particularly due to bone marrow suppression, have been reported in 10% to 50% of children. Spiramycin has been associated with a prolonged Q-T interval on electrocardiograms in preterm infants and cardiac arrest in two infants.

**COSTS**

Cost effectiveness studies of prenatal screening have generally failed to take account of the cost of false positive tests reflected in these crude net cost estimates and in the past, have been hampered by the lack of information on treatment effectiveness. Reports from France suggest that the cost for prenatal serological testing alone amounts to £50 million for 780,000 births a year in France (P Thulliez, personal communication) and in the United Kingdom would involve substantial additional start up costs, as well as clinical care and counselling costs. Assuming that the costs of serological testing are similar in the United Kingdom (about 700,000 births a year) and that 70 children with congenital toxoplasmosis are born each year, even if screening were 100% effective (far beyond the upper confidence intervals shown in table 2), the cost would be £174,000 for each infected child prevented, most of whom would be asymptomatic. This compares with costs of around £30,000 for each affected live birth prevented by prenatal screening for Down's syndrome. By contrast, neonatal screening can be added into established Guthrie card based screening and is estimated to cost £1.85 million to £3.5 million for a population of 700,000 live births (E Petersen, personal communication). Under the highly optimistic assumption that postnatal treatment prevents eye lesions in half the infected children born each year (35 children benefit), the maximum cost would be £100,000 per affected child prevented. In practice, the benefits of postnatal treatment may be far less.

**IMPLICATIONS FOR CURRENT PRACTICE AND FURTHER RESEARCH**

At the present time, there is insufficient evidence to justify the introduction of prenatal screening or neonatal screening in the United Kingdom. This issue needs to be revisited when further research findings become available. Reliable information is needed on the risk of neurological or visual impairment in infected children and whether the risk or severity of impairment is reduced by prenatal or postnatal treatment. The lack of evidence for a beneficial effect of prenatal treatment is based on cohort studies and the confidence intervals include both beneficial and harmful effects. More robust studies are required and some investigators have called for a randomised placebo controlled trial to determine the effect of treatment on transmission and on symptoms and signs in infected children. However, it may be ethically unacceptable to identify infected women and then randomise them to placebo. An alternative approach, randomisation to screening versus no screening, would need to enrol about 2.5 million women in the United Kingdom and about 250,000 in France. Randomised controlled trials of postnatal treatment are more feasible and are required before introduction of neonatal screening. Trials involving new antibiotics with activity against toxoplasma bradyzoite cysts would be particularly important. In the meantime, results from a prospective cohort study (EMSCOT, http://www.ich.ucl.ac.uk/ich/html/academicunits/paed_епid/emscot.html accessed 26/07/02) expected in the next year, will provide information on the effect of treatment on transmission, clinical signs, and development at 3 years of age.

**Primary prevention**

Further research into primary prevention is required to examine the effectiveness of health information strategies. Despite
Overall conclusion

- There is insufficient evidence to justify the introduction of prenatal screening or neonatal screening in the United Kingdom. This issue needs to be revisited when further research findings become available.

Prenatal screening

- Congenital toxoplasmosis is estimated to affect about 1/10 000 live births in the United Kingdom. Less than 5% of infected children have severe neurological impairment detectable in infancy and an estimated 20–30% develop intracranial or ocular lesions by 3 years of age. The effect of congenital toxoplasmosis on developmental and visual impairment in later childhood is unknown.

- There is a lack of evidence for a latent phase when treatment might prevent transmission of the parasite to the fetus or fetal organ damage. Cohort studies provide no clear evidence for a beneficial effect of prenatal treatment on mother to fetus transmission or clinical signs in the infected child.

- The effect of prenatal treatment on functional impairment in later childhood is unknown.

- Serological screening would be labour intensive, and would require substantial service investment and additional antenatal clinic visits. A large proportion of women identified by screening would have false positive test results or have been infected before pregnancy.

Neonatal screening

- Neonatal screening is technically feasible and would not result in an excessive burden of false positive results.

- No comparative studies have evaluated whether postnatal treatment has any effect on clinical manifestations in infected children. Adverse effects of treatment are common.

- Randomised controlled trials are required to determine the effectiveness of antibiotic treatment during infancy on clinical signs and developmental function.

Primary prevention

- Information about how to avoid toxoplasmosis in pregnancy may be the most cost effective approach to preventing congenital toxoplasmosis. The feasibility and effects of water filtration and veterinary public health strategies to reduce infection in the food chain and in humans should be explored.

Conclusions

A consensus that women should be provided with information about how to avoid toxoplasmosis infection before or early in pregnancy, health information seems to have limited effect. A French study found that only 17% of women who knew that pregnancy may be the most cost effective approach to preventing congenital toxoplasmosis. The feasibility and effects of water filtration and veterinary public health strategies to reduce infection in the food chain and in humans should be explored.

particularly in developing countries, infected drinking water (http://www.plant.uoguelph.ca/saferfood/archives/fsnet/2002/1-2002/fsnet_january_15.htm#TOXOPLASMOsis - BR accessed 26/07/02) Research is required to determine the feasibility, effects, and cost effectiveness of water filtration and other interventions to reduce toxoplasma infection in the food chain and in humans. If a societal perspective of the potential costs and benefits is taken, primary prevention of toxoplasma infection in the whole population may be the most rational option.

ACKNOWLEDGEMENTS

We are grateful to the members of the working party on toxoplasma infection in pregnancy who gave thoughtful input into the report for the Antenatal Screening Sub-committee of the National Screening Committee. The report can be viewed at http://www.nell.nhs.uk/screening/antenatal_pps/toxoplasmosis.html (accessed 26/07/02) forms the basis for this article although the views expressed are those of the authors alone. Tom Newman provided helpful comments on the manuscript.

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