Ministry for Primary Industries
Manatū Ahu Matua

Risk Profile: *Toxoplasma Gondii* in red meat and meat products

MPI Technical Paper No: 2015/08

Prepared as part of a New Zealand Food Safety Authority contract for scientific services

ISBN No: 978-0-908334-11-7 (online)
ISSN No: 2253-3923 (online)

January 2008

New Zealand Government

Growing and Protecting New Zealand

Client Report
FW06106

RISK PROFILE: *TOXOPLASMA GONDII* IN RED MEAT AND MEAT PRODUCTS

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ACKNOWLEDGMENTS

The authors would like to thank Murray Wilkins, AgVax; and Sue McAllister, University of Otago for helpful discussions.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

SUMMARY

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles include elements of a qualitative risk assessment, as well as providing information relevant to risk management. Risk profiling may result in a range of activities e.g. immediate risk management action, a decision to conduct a quantitative risk assessment, or a programme to gather more data. Risk Profiles also provide information for ranking of food safety issues.

This Risk Profile concerns *Toxoplasma gondii* (*T. gondii*) in red meat and meat products.

The definitive host for *T. gondii* is the *Felidae* family i.e. the organism reproduces sexually only in cats. Most cats in New Zealand have antibodies to *T. gondii* that indicates exposure to the parasite at some stage (Thompson, 1999).

It is estimated that approximately one third of humanity has been exposed to *T. gondii*, and that 16 – 40% of the UK and USA population has been infected. Higher rates are estimated in Central and South American and continental Europe.

The reason that toxoplasmosis is not more widely recognised is that the infection is usually asymptomatic in immunocompetent humans. Clinical symptoms more frequently manifest in at risk groups with immature and developing immune systems such as the foetus or neonate, or those with immuno-suppressed or immuno-compromised systems such as organ transplant recipients, cancer patients and those with HIV/AIDS. Congenital toxoplasmosis may occur if a previously unexposed pregnant woman becomes infected, with serious outcomes occurring in a proportion of babies at birth or later in life. Toxoplasmic encephalitis may occur in AIDS patients.

Toxoplasmosis was a notifiable disease in New Zealand between 1987 and 1996. Hospital discharge records and notification data during this period do not match and this has been attributed to under-reporting. Information gathered from discharge records of publicly funded hospitals in New Zealand between 2000 and 2006 show 84 cases of toxoplasmosis (non-congenital) and 7 cases of congenital toxoplasmosis, including 3 fatalities. There is very little information on which to comment on the prevalence of clinical consequences of toxoplasmosis infection in New Zealand.

The available data on seropositivity of the whole New Zealand population suggest that infection with *Toxoplasma* is as prevalent in New Zealand as other developed countries. Studies of seroconversion in pregnant women in New Zealand suggest a higher prevalence of congenital toxoplasmosis than has actually been observed.

There are three recognised modes of transmission, direct ingestion of the oocyst originating from cat faeces contaminating the environment (e.g. soil, water), consumption of the cyst in animal flesh, and congenital transmission. This Risk Profile addresses the postnatally acquired form of the disease transmitted through cysts in red meat and meat products. Pig meat is particularly associated with *T. gondii* infection.

Due to the inadvertent nature of infection, it is extremely difficult to ascertain modes of transmission and, in particular, the extent of infection via the consumption of cysts in red meat. The United States Department of Agriculture estimate that 50% of *T. gondii* infections in that country are related to consumption of raw or undercooked meat, making toxoplasmosis the third leading cause of deaths caused by infectious foodborne agents in the USA.

In a survey of consumer meat handling conducted recently by ESR, a question on cooking preferences for meat found that 1% preferred rare pork, 5% preferred their lamb rare and 19% liked a rare steak. Consumption of raw meat in New Zealand is very infrequent, according to the survey.

New Zealand has had no reported outbreaks of toxoplasmosis, and there have also been no case control studies, cohort studies or other risk factor studies. No data could be found on the prevalence of cysts in red meat or meat products in New Zealand.

While information on seropositivity in New Zealand livestock is limited, it is likely that New Zealanders domestically produced red meat does contain *Toxoplasma*. Imported red meat is less likely to contribute to exposure given that only small amounts of beef and sheep meat are imported, and pigmeat is required to be frozen. Ameliorating factors for any exposure are that animal seropositivity appears to overestimate infectivity, and *Toxoplasma* exposure will be controlled through cooking and freezing.

Despite the focus on cats as the primary host, ownership of cats as pets has not always been identified as a risk factor in case-control studies of pregnant women. These studies have identified the consumption of undercooked meats as a risk factor, although the most recent study, from England, did not.

The current shortage of information on the prevalence of infection in New Zealand livestock, or the prevalence of contamination in red meat, precludes a definitive risk assessment of this food/hazard combination. As noted by the European Food Safety Authority (EFSA), considerable method development would be required if efficient monitoring programmes for livestock or food were to be implemented.

Education programmes for vulnerable groups in relation to toxoplasmosis, as recommended by EFSA, are in place in New Zealand.

Data gaps

- Prevalence of *Toxoplasma* infection in livestock in New Zealand;
- Prevalence of contamination of red meat and red meat products with *T. gondii* in New Zealand;
- Accurate data concerning toxoplasmosis cases in at risk groups;
- Lack of data regarding prevalence of infection in domestic cats (the definitive host) in New Zealand; and,
- Information on awareness by at risk groups in relation to the transmission risks of *T. gondii*, this information would be useful in targeting risk communication messages.

1 INTRODUCTION

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. The place of a risk profile in the risk management process is described in "Food Administration in New Zealand: A Risk Management Framework for Food Safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000). Figure 1 outlines the risk management process.

Figure 1: Risk Management Framework

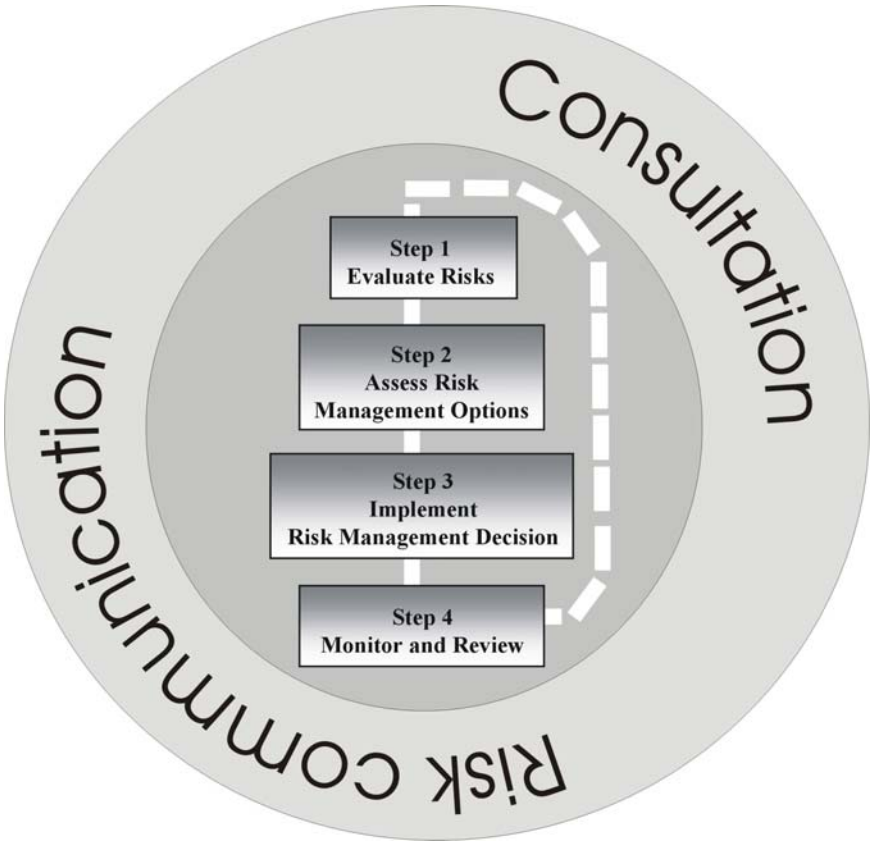


Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

1. Risk evaluation

- identification of the food safety issue
- **establishment of a risk profile**
- ranking of the food safety issue for risk management
- establishment of risk assessment policy
- commissioning of a risk assessment
- consideration of the results of risk assessment

2. Risk management option assessment

- identification of available risk management options
- selection of preferred risk management option
- final risk management decision

3. Implementation of the risk management decision

4. Monitoring and review.

The Risk Profile informs the overall process, and provides an input into ranking the food safety issue for risk management. Risk Profiles include elements of a qualitative risk assessment. However, in most cases a full exposure estimate will not be possible, due to data gaps, particularly regarding the level of hazard in individual foods. Consequently the risk characterisation part of a risk assessment will usually rely on surveillance data.

Risk Profiles also provide information relevant to risk management. Based on a Risk Profile, decisions are made regarding whether to conduct a quantitative risk assessment, or take action, in the form of gathering more data, or immediate risk management activity.

Toxoplasma gondii was originally chosen as a topic for risk profiling as the significance of human infections in New Zealand was identified as a knowledge gap in a review conducted in 1998 (Hasell, 1998). This report is an update of the first Risk Profile on this topic, which was written in 2002.

The sections in this Risk Profile are organised as much as possible as they would be for a conventional qualitative risk assessment, as defined by Codex (1999).

Hazard identification, including:

- A description of the organism
- A description of the food group

Hazard characterisation, including:

- A description of the adverse health effects caused by the organism.
- Dose-response information for the organism in humans, where available.

Exposure assessment, including:

- Data on the consumption of the food group by New Zealanders.
- Data on the occurrence of the hazard in the New Zealand food supply.
- Qualitative estimate of exposure to the organism (if possible).
- Overseas data relevant to dietary exposure to the organism.

Risk characterisation:

- Information on the number of cases of adverse health effects resulting from exposure to the organism with particular reference to the food (based on surveillance data)
- Qualitative estimate of risk, including categorisation of the level of risk associated with the organism in the food (categories are described in Appendix 1).

Risk management information:

- A description of the food industry sector, and relevant food safety controls.
- Information about risk management options.

Conclusions and recommendations for further action

2 HAZARD IDENTIFICATION: THE ORGANISM

The information contained in this Risk Profile is current to the date of publication. Please be aware that new information on the subject may have arisen since the document was finalised.

The following information is taken from a number of different sources but, unless otherwise referenced, is primarily derived from a data sheet prepared by ESR under a contract for the Ministry of Health in 2000-2001. The data sheets are now located on the NZFSA website and are intended for use by regional public health units. The datasheets will be updated from time to time, and new versions will be placed on this website:
http://www.nzfsa.govt.nz/science/data-sheets/index.htm

2.1 *Toxoplasma gondii*

2.1.1 The organism

Toxoplasma gondii is an obligate intracellular protozoan parasite that belongs to the phylum Apicomplexa, subclass coccidia (Montoya and Liesenfeld, 2004). The organism is classed as a Category 4 parasite, in that cysts or eggs are shed with faeces of the reservoir definitive host (in this case members of the cat family), in which the organism is able to reproduce sexually in large numbers. Excreted organisms are able to infect intermediate hosts such as warm blooded animals (including humans) and birds. The intermediate hosts are often also major food sources for humans e.g. livestock providing red meat (Goldsmid *et al*., 2003).

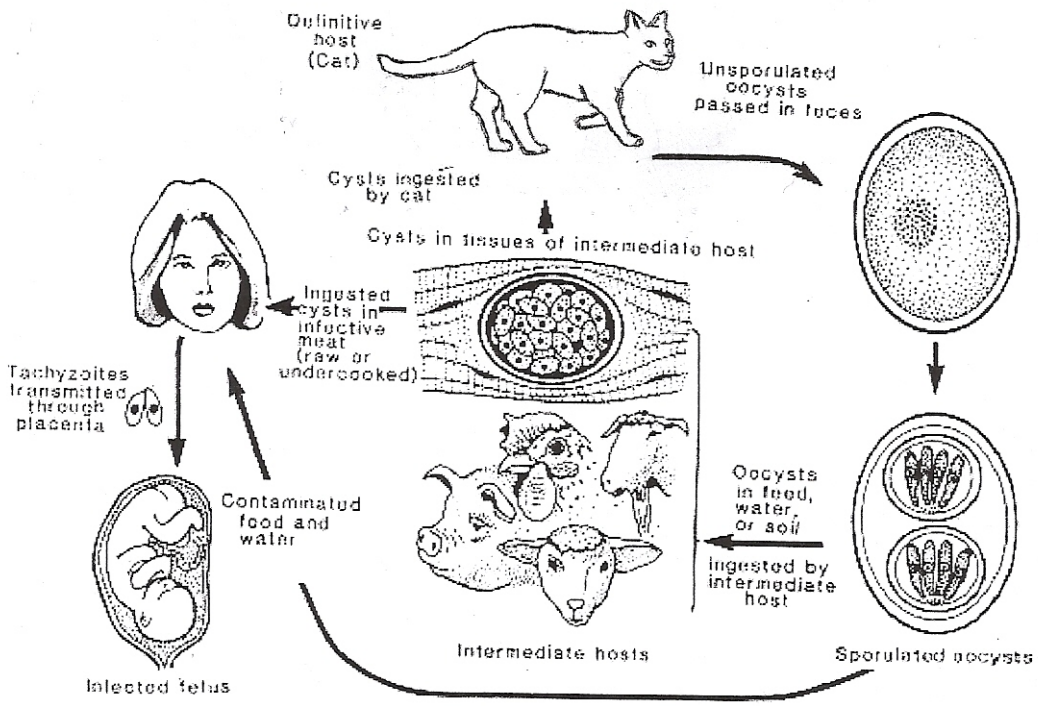
The organism has a complicated life cycle with numerous stages. The three infectious stages are tachyzoites, bradyzoites and sporozoites.

The environmentally resistant stage of *T. gondii* is called the oocyst, which are formed only in the cat family (*Felidae*). Cats can become infected and shed oocysts after eating any of the parasite's three lifestages (tachyzoite, bradyzoite or sporozoite), often in meat from intermediate hosts. The time to faecal shedding of oocysts depends on the lifestage ingested, for example 3 – 10 days for bradyzoites, 21 or more days for tachyzoites or sporozoites. The ingested bradyzoite appears better adapted to convert into oocysts in the cat's digestive system, with nearly 100% of cats shedding oocysts. However, after ingesting tachyzoites and sporozoites, fewer than 50% shed oocysts.

When mature, the oocysts are discharged into the intestine and passed out with the faeces. At this stage they are unsporulated and non-infectious. It takes one to five days (atmosphere and temperature dependent) to sporulate in the environment. Mature oocysts contain two sporocysts, each sporocyst contains four sporozoites. Sporulation is necessary for the oocyst to be infective for the next host. The oocyst can remain infective in water or moist soil for over 12 months (Heymann, 2004). Estimates of the number of oocysts shed after initial infection ranges from 300,000 to 100 million, thereby logarithmically amplifying the cycle (Frenkel, 1990). A cat will usually have just one episode of shedding oocysts in its life and 90% of cats that have undergone the intestinal cycle will not shed oocysts again after re-infection with bradyzoites or cysts (Frenkel, 1990). Dubey (1996a) reports, that under experimental conditions, cats do not shed oocysts after reinoculation with *T. gondii* tissue cysts (bradyzoites), but this immunity can wane with time.

Figure 2 depicts the life cycle of *T. gondii* from the cat as definitive host and how the organism is transmitted to humans via contaminated food and water and via undercooked meat.

Figure 2: Diagram of the life cycle of *T. gondii*



Source: Dubey and Beattie, (1988).

The tachyzoite triggers a strong inflammatory response and tissue destruction, manifesting as clinical symptoms. Under pressure from the immune response, the tachyzoite forms intracellular cysts or bradyzoites in the tissue. The cyst wall is elastic, thin and can enclose hundreds of parasites (range 2 - >1000; Dubey, 2001). Sites where the cysts are located can include visceral organs, such as lungs, liver and kidneys, although muscular and neural tissue is favoured such as the eye, brain, skeletal and cardiac muscle. Intact, the cysts cause no harm and the bradyzoites can persist inside for the life of the host. However, if released from the cysts, bradyzoites transform into tachyzoites, are disseminated to all organs, and restart the infection (Montoya and Liesenfeld, 2004).

It has been theorised that humans could acquire and ingest oocysts from the fur of an infected cat. However, even where cats are passing faeces containing thousands of oocysts, none have been detected on the fur (Dubey, 1995).

2.1.2 Growth and survival

Note: in microbiological terms “D” refers to a 90% (or decimal or 1 log cycle) reduction in the number of organisms.

Growth: The organism does not grow in foods or in other environments outside of a suitable host.

Survival: The oocysts can survive outside of susceptible hosts.

Temperature: Oocysts in faeces or suspended in water retain infectivity for up to 400 days at temperatures ranging from 4 to 37°C. When frozen at -21°C, unsporulated oocysts (as excreted into the environment) are killed within 1-7 days at this temperature, but some sporulated oocysts may survive freezing (Frenkel and Dubey, 1973).

Water Activity: Sporulated oocysts are gradually inactivated by drying. Encysted *T. gondii* may survive for 4 days in 8% NaCl.

Bradyzoites are more resistant than tachyzoites to the acidic conditions of the stomach. They have been shown to survive artificial gastric juice for at least two hours, long enough to infect humans (Smith, 1993).

2.1.3 Inactivation (CCPs and Hurdles)

T. gondii tissue cysts in meat are inactivated in three ways; cooking, freezing and irradiation.

Temperature: Heating meat throughout to reach a temperature in excess of 66°C is sufficient to kill cysts in meat (Goldsmid *et al.,* 2003). From a linear regression model, the D times for cysts (or bradyzoites) in pork were 336 seconds at 49ºC, 44 seconds at 55ºC, 6 seconds at 61ºC and 1 second at 67°C (Dubey *et al*., 1990). In two of four mutton steaks heated by microwave to 65ºC, residual infective parasites were detected, possibly due to uneven heating (Lundén and Uggla, 1992).

Freezing causes cysts in meat tissue to lose infectivity. Various reports have described this effect at temperatures between –6°C and –40°C (Kotula *et al*., 1991). These authors also conducted a more detailed study of the effect of freezing under commercial conditions on *T. gondii* tissue cysts in pork. Tissue cysts remained viable for up to 11.2 days at –6.7°C, and up to 22.4 days at –3.9 and -1°C. However, cysts were rendered noninfective at temperatures of –9.4°C or less for all time points, except in a very few instances. From the thermal death curve, a temperature of –12.4°C was obtained as the theoretical temperature at which *T. gondii* would be inactivated instantaneously.

In a recent study investigating the effects of time and temperature on the viability of *T. gondii* tissue cysts in enhanced pork loins (Hill *et al.,* 2006), meat case temperatures were measured and were found to vary between -3.2 and +7.0°C. In experimental work related to this, ingestion of infected enhanced pork loins stored at -5 and 0°C for 7 and 14 days did not result in infection and shedding of oocysts in seronegative cats. However, pork stored at 3.8 and 6.1°C prior to consumption did give rise to infection. This research suggests that storage of enhanced pork at or below 0°C is essential to inactivate tissue cysts.

The infectivity of oocysts stored in water at various temperatures has been assessed by mouse bioassay (Dubey, 1998). Infectivity was destroyed after 1 minute at 60°C or higher, while at the other extreme, there was no marked loss of infectivity at up to 25°C for 200 days. At 4°C

infectivity to mice remained after 54 months. Smith (1992) reports that treatment with boiling water for five minutes is effective in destroying oocysts.

Preservatives: The organism has been demonstrated to be rendered noninfective by curing in sodium chloride and sucrose (approximately 15% of each by weight of meat) at 4°C for 64 hours (Lundén and Uggla, 1992). However, cured pork was cited as a risk factor for infection in a case control study in Italy (Buffolano *et al.,* 1996). The authors commented that meats are sometimes cured using much lower levels of salt or sucrose.

Meat enhancement solutions (sodium chloride plus potassium/sodium lactate): Hill *et al.* (2004) reported data on the viability of *T. gondii* tissue cysts in pork loins treated with enhancement solutions. This study found that 2% sodium chloride or ≥1.4% potassium or sodium lactate, used alone or in combination with other components, was able to prevent the subsequent development of infection in cats fed infected pork loins stored for up to 45 days at 4°C.

Subsequent research by Hill *et al.* (2006) investigated how quickly the successful enhancement solutions used in their previous study were able to inactivate *T. gondii* tissue cysts in pork loins. Storage of pork loins at 4°C for 8 hours post-enhancement prior to consumption was found to be sufficient time for inactivation to occur.

Radiation: Exposure of tachyzoites to 70 J m^{-2} ultraviolet light renders the organism non-infectious.

A minimal effective dose (MED) of Co^{60} at 0.6kGy was calculated to inactivate cyst infectivity (Song *et al*., 1993). Dubey and Thayer (1994) found a lower dose of 0.4kGy was sufficient and that temperature during irradiation had no marked effect. Because of the variability in the number of cysts present in pork and variation in carcass sizes, a 1 kGy dose is recommended for complete disinfection (Smith, 1992).

Pressure: High pressure processing (HHP) of ground pork containing viable *T. gondii* cysts was studied at various pressure and time combinations (Lindsay *et al.,* 2006). Results of mouse bioassay found that none of the cysts exposed to 300 or 400 Mpa remained infective; while cysts processed at 200, 100 or no pressure (control) remained infective regardless of treatment time.

2.1.4 Sources

Human: There have been no reports of direct person-to-person spread nor transmission during breastfeeding. Transplacental transmission from mother to baby can occur if the mother is infected for the first time during, or just before, pregnancy (Montoya and Liesenfeld, 2004). Transmission by organ transplantation can also occur. A large number of blood donors are potentially seropositive, however cases of transfusion-transmitted toxoplasmosis have only been reported in acute leukaemia cases undergoing chemotherapy (Smith, 1997).

The sub-clinical human infection can be chronic and become activated if the immune system becomes weakened.

Animal: Cats are the definitive host and warm-blooded mammals and birds are intermediate hosts. Contamination of the environment by oocysts shed in faeces by cats is a possible route of transmission. Although the risk from handling cat litter has been proposed, ownership of a cat has not been found to be a risk factor in several studies (AFSSA, 2005).

Transmission directly through contact with intermediate host animals does not appear to be important in the epidemiology of human infections.

Animals such as flies, earth worms and cockroaches that have come into contact with infected faeces may harbour the organism and mechanically spread the oocysts (Dubey, 1996a).

Food: Along with poor hand hygiene, the consumption of poorly cooked meat and badly washed raw vegetables are the principal food related risk factors for human infection (AFSSA, 2005). Especially important are vegetables originating from gardens where there may be cats (Smith, 1993).

Meat from bovines is less frequently contaminated by cysts than pig, sheep and goat meats. Unpasteurised goat's milk has also been implicated in some outbreaks (Smith, 1993; Sacks *et al*., 1982; Skinner *et al*., 1990). Meat from birds, including poultry, may contain *T. gondii* cysts (AFSSA, 2005). Other meats that may contain cysts are rabbit, horse, and game (e.g. deer), and occasionally, cured meats (ham).

Environment: Consumption of water contaminated by oocysts shed by cats can result in infection, and several outbreaks have been linked to drinking insufficiently treated water. Oocysts can survive in soil for several months (Frenkel, 1990). Soils may be important intermediates in the transmission from cats to humans via buried faeces.

Transmission Routes: There are five transmission routes identified (Leroy, 2005).
i) The ingestion of raw or inadequately cooked infected meat, or ingestion of uncooked foods that have been in contact with such meat

ii) Ingestion from oocysts through direct contact with faeces passed by an infected cat or indirect contact such as contaminated soil such as when gardening, or eating unwashed vegetables, or drinking water that has been in contact with contaminated cat faeces.

iii) Transplacental infection during a primary infection of the mother.

iv) Organ transplantation or blood transmission (seropositive donor to seronegative recipient).

v) Accidental inoculation by laboratory personnel, for example, the use of contaminated needles or work with infected animals.

3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: Red Meat and Meat Products

As the organism does not grow outside a live host, the characteristics of the food group are less relevant in terms of contributing to risk. Specific edible meats found to contain cysts include (Smith, 1991):

- Beef and veal;
- Small game animals (including rabbits);
- Fowl (chicken and pigeons);
- Horse meat;
- Deer and elk (wapiti) meat;
- Mutton, lamb and goat meat; and
- Pork.

Cysts have also been reported in wallabies (MAF, 1974; 1980; 2000), little blue penguins (Mason *et al*., 1991), and possums (Eymann *et al*., 2006), although these animals are not considered livestock.

Beef and veal are generally considered as less likely to be contaminated; clinical disease is rare in cattle and the organism is rapidly eliminated from tissues (Smith, 1991).

The meats listed above all fall into the risk profile category of red meat, apart from chicken and pigeons. While raw meats have been most commonly implicated in cases of toxoplasmosis, cured meats such as ham have also been shown to occasionally contain cysts (Warnekulasuriya *et al.*, 1998).

Although visual examination or serological testing can be used in the field or slaughterhouses to prevent other parasites (*Taenia solium* and *Trichinella spiralis*) from entering the food supply, these are not options for *T. gondii* due to the length of time and complexity of the extraction procedures and assays (Gamble, 1997). Consequently control mechanisms must focus on destruction of cysts via cooking or other mechanisms.

The effect of heating and freezing on inactivation of *T. gondii* cysts has been reviewed in Section 2.1.3.

Freezing to -12.4°C has been suggested as the theoretical temperature for instant inactivation of *T. gondii* cysts in meat tissue (Kotula *et al*., 1991). In New Zealand, meat cuts presented for retail sale (butchers, supermarkets) will not usually have been frozen. However, wholesale meat used in the production of sausages, meat pies, etc. is likely to have undergone freezing at some stage (Graeme Keeley, Technical Manager, PPCS, personal communication). Only a small proportion of meat for domestic consumption will have been frozen prior to sale (Clyde Daly, AgResearch, personal communication). The current chilling regime used for "Quality Mark" meat production requires a temperature of 7°C or less prior to shipping, with 4°C achieved by retail sale. This means that if the cysts are present in the meat at slaughter, they may still be present in retail chilled meats.

3.2 The Food Supply in New Zealand

There are 17,000 commercial sheep and beef cattle farms in New Zealand, most of which are owned and operated by farming families. Livestock numbers for New Zealand in 2005 are shown in Table 1. A high proportion of sheep and beef production is exported.

Table 1: Livestock numbers, production and export for New Zealand

Livestock type	Number of animals (000 head) For year 2005	Meat production in year to September 2005 (000 tonnes carcass weight)
Sheep	39,928	105 mutton 438 lamb
Beef	4,431	23 veal 629 beef
Pigs	389	50
Deer	1,705	N/a
Goats	142	N/a

N/a: Not available
Source: MAF sonzaf website accessed 17.07.07 MAF (2007)

One major technological advancement has been the extension of shelf life for chilled meat, which means that each year a greater proportion of meat is being exported as chilled cuts rather than frozen carcasses. In 2006, 15% of all lamb exports were exported chilled (MIA, 2006).

As the figures indicate, the vast majority of meat production in New Zealand is exported.

New Zealand has a relatively small pork industry, which focuses on the domestic market. Since 1995, pigmeat production has been relatively static averaging 49,000 tonnes per year, although it appears that pigmeat consumption has been slowly increasing (New Zealand Pork Industry Board, 2001).

3.2.1 Imported food

Approximately 4,500 tonnes of meat preparations were imported during the year to September 2005, with almost 80% coming from Australia.

New Zealand imports relatively small amounts of beef and sheep meat according to data from Statistics New Zealand. For the year to September 2005 approximately 3,200 tonnes of beef carcasses and cuts and 3,100 tonnes of sheepmeat (all types) were imported from Australia.

Imports of pork have increased steadily to now make up approximately 38% of total supply (31,864 tonnes in 2005), mostly sourced from Australia and Canada. New Zealand maintains

an Import Health Standard for the importation of pork, including a requirement that the pork is frozen to −18°C (Anon, 2006). This means that in theory imported pork should be *T. gondii* free; see http://www.pork.co.nz/profile.asp). Currently due to Import Health requirements, Canadian pigmeat is imported uncooked, held in transitional facilities, cooked and then released onto the New Zealand market so is unlikely to harbour *T. gondi*.

These data, when compared to the production and export figures above, suggest that approximately 5% of New Zealand's beef and sheep meat for domestic consumption derives from Australia, while approximately 38% of pigmeat for domestic consumption is imported, principally from Australia and Canada.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

When *T. gondii* oocysts/tissue cysts are ingested by humans or other animals, digestive juices break down the cyst to release the bradyzoites, which transform to tachyzoites in the small intestine and cause infection. The organism then penetrates the intestinal wall and migrates throughout the body where it can invade a variety of tissues (a vacuole around the organism protects against the host's defence mechanism). Within the tissues, the parasite multiplies asexually, forming tissue cysts (Murrell, 1995; Hill and Dubey, 2003; Montoya and Liesenfeld, 2004).

4.1 Symptoms

Incubation: A foodborne outbreak in Australia reported an incubation period of 3 – 25 days, with a mean of 11 days (Robson *et al.*, 1995). In another outbreak associated with cats (oocysts), the incubation period was 5 – 20 days (Heymann, 2004).

Symptoms: In immunocompetent humans, the infection is common although under-reported because 80-90% of infections are asymptomatic. Clinical toxoplasmosis is rare. In about 10-20% of cases, a viral-like febrile illness occurs with lymphadenopathy (swollen lymph nodes) (Mead *et al.*, 1999). This is usually mild and self-limiting and individuals seldom seek medical attention (Smith, 1997).

Clinical toxoplasmosis typically affects individuals with developing or impaired immune systems such as the developing foetus, the elderly, medically immunosuppressed patients, and those who are immunocompromised by disease (e.g. AIDS) (Smith, 1997). The central nervous system is the site most typically affected by infection, with a wide variety of symptoms (Montoya and Liesenfeld, 2004).

It has been reported that pregnant women are significantly (2.2 times) at higher risk of seroconversion for toxoplasmosis than non-pregnant women (7.7 times higher if the woman is an adolescent) (Avelino et al., 2003). This has been ascribed to alterations in the immune system necessary for the tolerance of the foetus and/or hormonal imbalances (Daunter, 1992).

If a previously unexposed pregnant woman becomes infected, the parasite can be transmitted across the placenta to cause congenital toxoplasmosis, the most commonly cited health concern. The mother may remain asymptomatic (Goldsmid *et al*., 2003). In general, the older the mother, the more likely they are to be seropositive which means that those entering motherhood early may have more risk of initial infection while carrying their child. Women who are seropositive for *T. gondii* prior to pregnancy, but who are healthy and immunocompetent, do not transmit the parasite to their foetuses.

Transplacental infection occurs when rapidly dividing tachyzoite cells circulate in the bloodstream and cross the placenta to infect the foetus (Heymann, 2004). While the infection may not damage the foetus, it is capable of causing death or serious neurological damage in up to 5% of infections (Gilbert *et al.*, 2006). Hydrocephalus, intracranial calcification and retinochoroiditis are the most common manifestations of tissue damage due to congenital toxoplasmosis (Gras *et al*., 2001).

The probability of transmitting the parasite increases with the trimester of pregnancy; rates of

transmission are more than 60% in the last trimester. However, frequency of transmission and severity of disease are inversely related (Montoya and Liesenfeld, 2004). Three to four percent of infected neonates die, while the remainder will suffer from various forms of long term disease (mental retardation, blindness and epilepsy) (Smith, 1997). It has been estimated that in congenital infections of babies, 8-10% have brain and eye lesions while 10-13% become visually impaired. Nearly all those born with subclinical disease will develop symptoms later on.

Although the immune systems of elderly people (>65 years) undergo changes that should make them more susceptible to toxoplasmosis, there is no evidence that the parasite becomes reactivated in healthy seropositive elderly individuals (Smith, 1997).

In immuno-compromised people (those suffering from AIDS or undergoing immunosuppressive therapy) disease seems to result from the activation of a previously subclinical infection. Reactivation most often involves the central nervous system and symptoms can include meningoencephalitis, maculopapular rash, generalised skeletal muscle involvement, cerebritis, chorioretinitis, pneumonia and myocarditis (Heymann, 2004). It has been estimated that up to 30% of AIDS patients in Europe and 10% in the USA will die from toxoplasmosis, the majority via toxoplasmic encephalitis (Luft and Remington, 1992). Although any organ may be involved, infection of the brain is that most often reported, a persistent severe and bilateral headache progressing to confusion, lethargy, ataxia and coma. The predominant lesion is necrotised tissue in the thalamus (Renold *et al.,* 1992).

Condition: Toxoplasmosis.

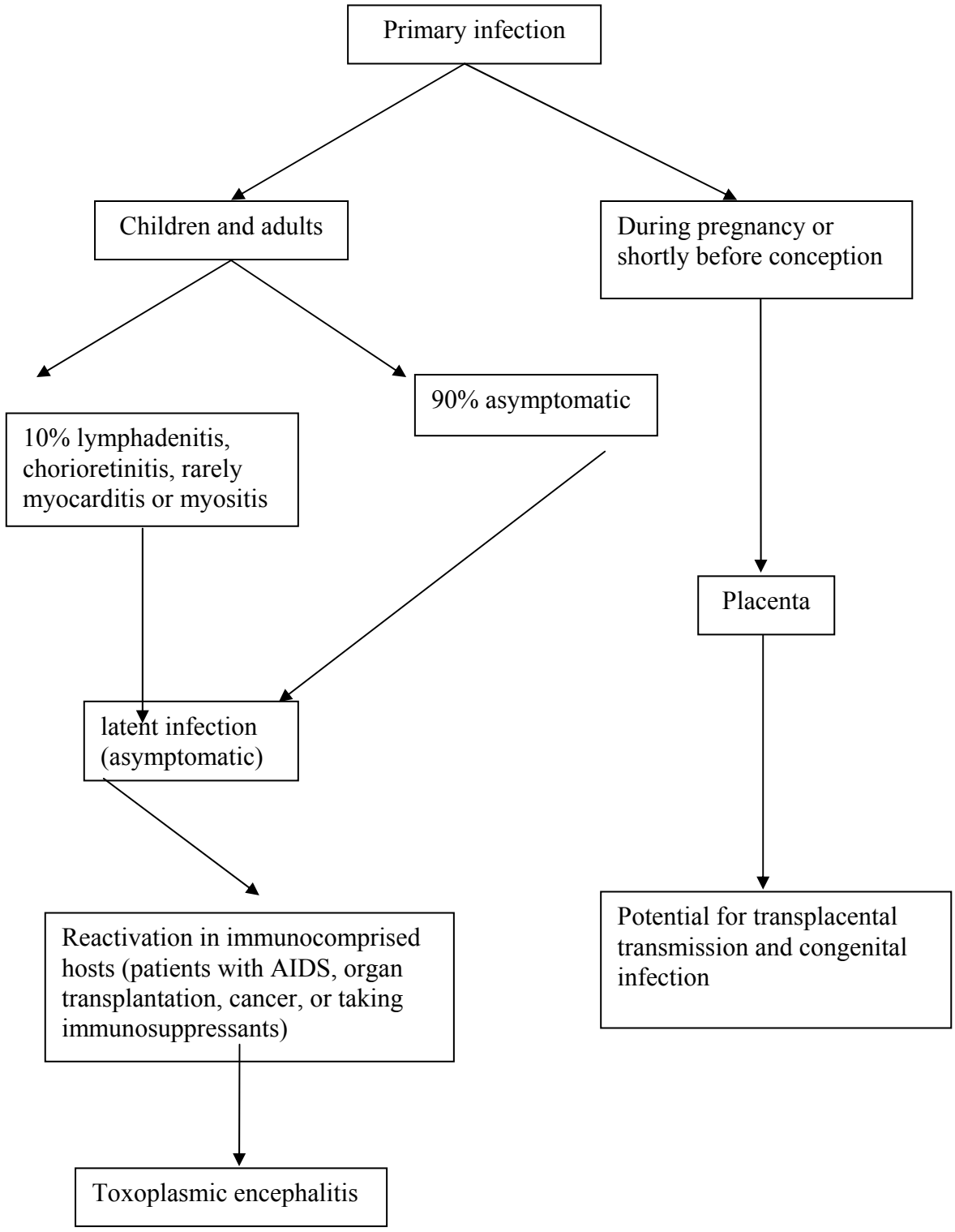
Toxins: T. gondii does not produce a toxin.

Long Term Effects: In healthy people infection rarely leads to death. Vulnerable populations are most likely to experience long term effects after infection. Examples include visual impairment and brain damage. The role of toxoplasmosis in inducing Guillain-Barré syndrome is probably infrequent (Smith, 1993).

Treatment: Currently there is no human vaccine for the disease. Treatment is not routinely administered for a healthy immunocompetent host. In immuno-compromised patients such as patients with symptomatic toxoplasmosis, prophylactic treatment with pyrimethamine can be given in combination with sulfadiazine and folinic acid (to avoid bone marrow depression). Clindamycin has been additionally used to treat ocular toxoplasmosis (Heymann, 2004). However these drugs can be problematic when used with pregnant women. Spiramycin is commonly used to prevent placental infection (Heymann, 2004).

The potential health outcomes are summarised in Figure 3.

Figure 3: Health outcomes for *T. gondii* infection



Source: Montoya and Liesenfeld, 2004

4.2 Types Causing Disease

T. gondii has a low genetic diversity with only three genetic types I, II and III classified on the basis of antigens, isoenzymes, and restriction fragment length polymorphism (Hill and Dubey, 2003). Most strains isolated from patients with AIDS are Type II. Type I and II strains have been recorded in patients with congenital disease, whereas strains isolated from animals are mostly Type III (Montoya and Liesenfeld, 2004; Howe and Sibley, 1995; Nowakowska *et al.,* 2006).

The differential occurrence of these types is not absolute however. For example, studies of *T. gondii* isolates from free range asymptomatic chickens in various South American countries have found all three genetic types (Dubey *et al*., 2002b; Dubey *et al*., 2006).

4.3 Dose Response

No information has been found regarding the dose response relationship for *Toxoplasma gondii* in humans. For ethical reasons, establishing a dose infection relationship based on experimental trials and healthy human volunteers is not foreseeable. In animal studies, the feeding of graded doses of oocysts to pigs (an intermediate host) found that one sporulated oocyst was infective, the results were 13/14 pigs fed 10 oocysts, 13/14 pigs fed 1 oocyst and 4/14 pigs fed < 1 oocyst were infected. Higher isolations were observed in the tongue as opposed to the brain and heart (Dubey *et al*., 1996). In another study, ten bradyzoites were not infective to cats, but higher numbers (100 and 1000) caused shedding of infective oocysts (Dubey, 1996b; Dubey, 2001).

4.4 High Risk Groups in the New Zealand Population

The following sections provide information on groups in the New Zealand population for whom infection by *Toxoplasma* could have serious consequences.

4.4.1 Pregnant women

Live births data for the year to September 2007 were 62,360 (http://www.stats.govt.nz/ accessed January 2008).

4.4.2 Immune compromised

HIV: In 2005, 218 people in New Zealand were newly diagnosed with HIV, the highest since records began in 1985. Since 1985, a total of 2474 cases of HIV have been recorded in New Zealand (ESR, 2006).

AIDS: There were 49 notifications of AIDS cases in 2005 and 8 deaths. Since 1983, there have been 891 AIDS notifications in New Zealand. The number of deaths due to AIDS peaked at 66 in 1992. In the last four years, there have been approximately 10 deaths each year (ESR, 2006).

Cancer: The most recently available statistics on the incidence of cancer and cancer mortality in New Zealand are from 2002 (website accessed 15.08.06 http://www.nzhis.govt.nz/stats/tables/cancer2002.xls and publications/cancer.html).

In that year, 17,943 new cases of cancer were registered, made up of 9,399 (52.4%) males (348.2 cases per 100,000) and 8,544 (47.6%) females (287.0 cases per 100,000). During the same period, mortality due to cancer was 7,800 (120.9 cases per 100,000) made up of 4125 males (142.8 per 100,000) and 3675 females (104.9 per 100,000). It is uncertain what proportion of the New Zealand population is suffering from cancer at any particular time.

Recipients of organ or tissue donations: The New Zealand Organ Donation website gives the following numbers for transplants performed in 2005; kidney (deceased donor) 47; kidney (living donor) 46, liver (deceased donor) 24, liver (living donor) 4, heart 13, lungs 8, pancreas 2 (http://www.donor.co.nz accessed October 2006). It appears likely that the total New Zealand population of surviving major organ transplant recipients is less than 2000 people (0.05% of the total population).

5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply

5.1.1 Test methods

Most of the information regarding the prevalence of *Toxoplasma gondii* is derived from studies that measure antibodies against the organism present in the blood of humans and animals. A review carried out by Remington *et al*., (1995) has examined the range of antibody detection methods including the Sabin-Feldman dye test, indirect fluorescent antibody test (IFAT), complement fixation test (CFT), the modified agglutination test (MAT) and enzyme-linked immunosorbent assay (ELISA).

To detect *Toxoplasma* in meat or tissue samples, extracts are used in mouse-based assays to demonstrate infectivity. The mice are inoculated orally or subcutaneously with the extracts, and then the mouse sera can be examined for antibodies, and/or tissues can be examined microscopically. Assays have also been used with cats and, because of the large numbers of oocysts they shed following infection, they are more sensitive than mice for detecting infective cysts (Dubey, 2001). However, the mouse bioassay is more commonly used.

Results from such mouse assays have not consistently demonstrated that material extracted from the tissue of seropositive animals is infective. In a Canadian study, although seroprevalence of *Toxoplasma* antibodies in pigs was between 3.5 and 13.2%, none of the extracts tested showed any infectivity in the mouse bioassay (Gajadhar *et al.,* 1998). This suggests that serological data from mouse assays may not accurately assess the risk from improperly cooked pork products.

Direct detection in foods is difficult, one reason being that the methodology requires the concentration of the organism from muscle tissue prior to detection. Due to the low numbers of oocysts present in environmental samples (e.g. water), detection methods are largely based on filtration similar to those used for other parasites (Fayer *et al*., 2001).

The presence of *Toxoplasma* DNA in samples (including commercial meats) has more recently been demonstrated using the polymerase chain reaction (PCR) (Aspinall *et al.*, 2002). PCR detects the presence of *T. gondii* DNA but not necessarily the presence of viable cysts.

5.1.2 *Toxoplasma* prevalence in livestock animals in New Zealand

There are no data on the prevalence of *Toxoplasma* in red meat in New Zealand. Some reports provide indirect data on the prevalence of infection in livestock and other animals, including pets (see Section 7.3.1).

Pigs: A review of parasites of New Zealand pigs stated that the prevalence of *Toxoplasma* in New Zealand pigs is unknown, although there have been occasional reports of clinical toxoplasmosis in farmed pigs (Fairley, 1996).

Sheep: The "Veterinary Handbook" prepared for the New Zealand Veterinary Association states that the incidence of toxoplasmosis in young ewes is "up to 30% with placentitis and abortion or perinatal deaths. Up to 90% of ewes may have serum antibodies by 2 years. Disease rare in other species." (Manktelow, 1984). Reports in Surveillance (MAF, 2001; 2002) stated that most sheep abortions in New Zealand are caused by *T. gondii* infection or *Campylobacter fetus.* Infection with *T. gondii* was noticeably more prevalent as the lambing season approached.

A live *Toxoplasma* vaccine for sheep has been developed by AgVax at Wallaceville and is registered and in use in New Zealand. The vaccine uses the s48 strain of the tachyzoites which have lost their lifecycle persistence. After vaccination, the s48 tachyzoites multiply in the young sheep for approximately seven days. Bradyzoites and subsequent tissue cysts do not develop and the parasite dies, leaving the sheep immune and in the long term, the meat is safe to consume. The vaccine is a preventive measure in order to reduce the risk of abortion caused by *Toxoplasma* infection during subsequent pregnancy (Murray Wilkins, AgVax, personal communication). More information on the vaccine can be found at the following website address;
http://www.agvax.com/animal_health/sheep/toxovax/technical_manual/toxoplasmosis_the_infection.htm. The vaccine is most commonly used for the vaccination of ewe hoggets or rising two-tooth ewes joining the flock.

In 2004, 61 abortions in sheep due to *T. gondii* were reported (MAF Biosecurity Authority, 2005).

Goats: Thompson (2001) reported *T. gondii* as the most common cause of infectious abortion in goats although the author notes that caprine abortions associated with *Neospora* and *Sacrocystis* species could be easily misdiagnosed as toxoplasmosis. Opel *et al.* (1991) reported on the seropositivity of New Zealand domesticated clinically normal goats from 17 farms. Positive samples were found from the sera of 7% of kids (n=88), 23% of yearlings (n=65) and 37% of adults (n=145). This increase of positive titres with age was statistically significant, as was a higher frequency of positives in dairy compared to fibre breeds. There were no data on feral goats, although the author proposed that the infection rate in dairy goats could reflect intensive husbandry practices. In addition, hay feed might be contaminated by cats, which implies greater prevalence in domesticated rather than feral populations.

Cattle: A review of infectious diseases in cattle in New Zealand stated that infection with *T. gondii* is probably common in New Zealand but the clinical disease is rarely recorded (Vermunt and Parkinson, 2000).

Deer: The prevalence of *T. gondii* infection in feral deer is not known. In 1997, MAF assembled a farmed deer serum bank of 1,150 specimens, of which a random sub-sample of 417 were tested for toxoplasmosis antibodies. The overall seroprevalence was 219/417 (52.5%) (using reciprocal titres of ≥ 8 as positive). Seroprevalence increased with age from 15.4% in < 1 year-olds to 86.6% in deer 8 years old and above. The prevalence is consistent with overseas deer surveys (Reichel *et al.*, 1999).

5.2 Food Consumption: Red Meat and Meat Products

Red meat consumption has declined over the last 20 years, mainly due to mutton and lamb, see Table 2. A major shift in consumption patterns has taken place with major and smaller gains by the poultry and pork industries respectively.

Table 2: New Zealand domestic meat consumption per capita 1985, 1995, 1996, 1999 to 2006 (kg/person/year)

Year	Mutton and Lamb	Beef and Veal	Pig meat	Total Red meat	Poultry	Total Meat
1985	27.3	36.5	14.2	78.0	15.0	93.0
1995	23.2	34.6	15.7	73.5	26.2	100.1
1996	20.6	37.8	16.1	74.5	25.1	99.8
1999	14.3	31.2	17.1	62.6	26.8	89.5
2001[1]	16.6	27.1	16.5	60.2	31.0	91.3
2001/02[2] (Sept. end)	16.1	27.6	17.3	61.0	34.1	95.1
2002/03[3] (March end)	15.3	29.4	17.9	62.6	35.9	98.5
2005[4] (June end)	13.6	32.4	19.6	65.6	37.1	102.7

From New Zealand Meat and Wool Board's Economic Service (MWBES) Annual Review of the Sheep and Beef Industry, 1999-2000.
[1] Data from website; http://www.beef.org.nz/statistics/slides.asp
[2] Compendium of New Zealand Farm production statistics, April 2003.
[3] PIANZ, December 2003
[4] New Zealand Meat and Wool Board's Economic Service (MWBES) Meat Consumption and Expenditure June Qtr 2006

The meat consumption figures for New Zealand in Table 2 are similar to estimates made for the Australian population (Baghurst, 1999). An international comparison of meat consumption as calculated for 2003 is given in Table 3.

Table 3: International comparison of meat consumption, 2003 (kg/person/year)

Country	Bovine meat	Sheep and goat meat	Pigmeat	Poultry meat
Argentina	54.7	1.5	5.1	19.4
Australia	45.1	14.4	21.1	35.6
Canada	34.3	1.0	27.4	36.3
New Zealand	26.4	24.8	20.7	35.2
UK	20.9	5.9	26.0	30.0
USA	41.9	0.5	30.1	50.2

Source: http://faostat.fao.org/

The figures given in Table 2 represent the meat available for consumption in New Zealand. Information on amounts of meat reported to be actually consumed by individuals can be

abstracted from the 1997 National Nutrition Survey (NNS) (Russell *et al.*, 1999). FSANZ have carried out an analysis of this dataset (ANZFA, 2001), including application of a set of standard recipes, to allow composite foods to be reduced to their component parts. Table 4 gives the estimates for New Zealand domestic meat consumption derived by ANZFA and compares those levels of consumption to the estimates based on meat available for consumption in Table 2.

Table 4: Mean estimates of New Zealand domestic meat consumption in 1997 and estimates of meat available for consumption, 2004

Meat type	**Estimated consumption (1997)***	**Amount available for consumption (2004)#**	
unit	**g/person over 15 yrs/day**	**kg per person per year**	**g per person per day**
Beef and veal	87.9	29.93	82.0
Sheep and Lamb	13.7	23.54	64.5
Pig meat	32.3	7.86	21.5
Deer meat	0.9	3.28	8.8
Total red meat	**134.9g**	**64.6kg**	**181g**

* from ANZFA analysis of 1997 National Nutrition Survey data (ANZFA, 2001)

\# recalculated from Table 2 and 2003 import data, (population at June 2004; 4,009,200) source: http://www.stats.govt.nz/

The difference between these two estimates of consumption will reflect wastage (meat available for consumption, but not consumed) and under-reporting in the National Nutrition Survey (NNS). Through use of standard recipes the FSANZ analysis of the most up to date (1997) NNS data will include all meat consumed, including meat that is consumed as a component of a processed food such as meat pies or luncheon meat (ANZFA, 2001).

The analysis of the 1997 NNS data concluded that 77.7% of the population consumed red meat (cattle, sheep or pig meat) during any 24 hour period. The mean daily consumption was 172.5 g. The median daily consumption, for consumers only, was 124.1 g/day. The 97.5th percentile daily consumption, for consumers only, was 616 g/day.

Table 5 represents an analysis of dietary records from the 1997 National Nutrition Survey and shows a breakdown of total red meat and red meat product consumption on the basis of number of servings and on the basis of consumption weight. Beef is the predominant red meat consumed in New Zealand with over 50% of the total red meat consumed by weight. Pig meat contributes around 15% and sheep meat approximately 9%.

Table 5: Types of red meat and meat products consumed, by servings and by weight

Meat type	Percentage of total red meat consumed (by servings)	Percentage of total red meat consumed (by weight)
Beef (including veal)		
Corned beef	6.3	5.0
Beef mince and beef mince recipes (patties, hamburgers, etc)	14.7	24.1
Beef cuts (steak, roast, schnitzel, etc)	20.2	26.2
Sheep meat (Lamb, hoggett and mutton)		
Hoggett/mutton cuts	4.1	3.6
Lamb cuts	6.0	5.2
Lamb mince and lamb mince recipes	0.1	0.1
Pig meat (including ham and bacon)		
Pig meat cuts	6.8	8.3
Pig meat mince	0.1	0.1
Bacon	7.3	2.9
Ham	11.5	4.3
Mixed meat products		
Sausages, saveloys, frankfurters and hotdogs	13.6	15.1
Other meats		
Venison	0.4	0.5
Other	8.9	4.6

5.3 Qualitative Estimate of Exposure

It is not possible to make a qualitative estimate of exposure to *T. gondii* from red meat in New Zealand. Although there is some evidence to suggest that a proportion of the farmed animals in New Zealand have been infected with the organism, it is not possible to translate this seropositivity information into data regarding infectiveness. While estimates for the number of servings of red meat consumed and the median serving size can be made, there is insufficient information to estimate the frequency of contamination or the likely level of contamination at retail.

In addition, the infectivity of any *Toxoplasma* in imported pork is likely to be low, given the requirement for freezing at -18˚C, which should destroy the organism.

5.3.1 Number of servings and serving size

The estimation of total number of servings of red meat consumed on a per annum basis involves a number of assumptions:

- That the sample set employed for the NNS is typical of the total population;
- That the results of the 24 hour dietary recalls are typical of the full 365 day period of one year; and,

- That the consumption of red meat by the population less than 15 years of age will not be significantly different to that for the survey population (the NNS only surveyed people 15 years and older).

Analysis of dietary records from the 4636 people interviewed for the 24 hour dietary recall survey carried out as part of the 1997 NNS (Russell *et al.*, 1999) identified approximately 5,800 records of food items consumed (servings) which were likely to have contained red meat, red meat products, ready-to-eat (RTE) red meat products or red meat extracts. On this basis red meat and red meat products should be considered to be a very commonly consumed group of foods, with only milk (dairy products) and cereal grains being consumed with similar frequency.

T. gondii principally constitutes a risk to the foetus due to consumption by the mother leading to *in utero* exposure. To assess the number of servings of red meat for the New Zealand population which may result in foetal exposure, the number of servings for the total population may be multiplied by the annual live births rate (for New Zealand; 58,250 to June 2006 calender year) as a percentage of the June 2006 total estimated population (4,140,300) gives a birth rate of 14.1 per 1,000 per annum. Identical to the USA rate of 14.1 births per 1,000 in 2003, (see website http://mchb.hrsa.gov/whusa_05/pages/0425lb.htm accessed 24 August 2006).

For the population as a whole, due to the large number of foods which may contain red meat as a minor component (e.g. meat-based soups) serving sizes will vary over a very wide range. The FSANZ analysis of the 1997 NNS data (ANZFA, 2001), which includes the use of standard recipes to reduce composite foods to their component parts, calculated a median total daily meat intake for consumers of 124.1 g/day. Based on the estimate of 5,800 servings from the survey and 3,599 (of 4636 people surveyed) consumers, it can be calculated that, on average, this median intake will represent 1.6 servings of red meat. This calculation gives a median serving size of 77.6 g, a mean serving size of 107.8 g and a 97.5th percentile serving size of 385 g.

5.3.2 Frequency of contamination

No information is available on the frequency of contamination of red meat and red meat products with *T. gondii* in New Zealand.

5.3.3 Predicted contamination level at retail

No information is available on which to base a prediction of contamination of red meat and red meat products at retail with *T. gondii.*

5.3.4 Growth rate during storage and most likely storage time

The organism does not grow in foods or in other environments outside of a suitable host.

T. gondii is destroyed by freezing at temperatures of –12.5°C or lower. Discussion with meat industry representatives suggest that meat reaching the consumer as meat cuts is unlikely to have been frozen, while processed meat products (e.g. sausages) are highly likely to have been manufactured from frozen meat. Approximately 60% of the 5800 servings identified

from the 1997 National Nutrition Survey as being likely to contain red meat would be classified as manufactured meat products. It is likely that all of these servings would have undergone a sufficiently rigorous freezing regime to destroy the organism.

5.3.5 Heat treatment

T. gondii is susceptible to heat inactivation, and this occurs within a few seconds at temperatures above 60°C (see section 2.1.3). Red meat servings cooked medium to well done are unlikely to contain active organisms. Thomson and Lake (1995) carried out a survey of meat consumption and cooking practices amongst 902 respondents in the Hawke's Bay and Christchurch. Approximately four percent of respondents reported consuming servings of meat that were cooked to a 'rare' condition.

In a recent ESR postal survey of meat handling in the home (Gilbert *et al*., 2005), 1000 randomly chosen people across New Zealand were asked their preferences when cooking meat and poultry. Approximately one third replied to the survey. Table 6 below displays the results.

Table 6: Data for meat and poultry cooking preferences in New Zealand

Meat type	Raw (%)	Rare (%)	Medium (%)	Well done (%)	Very well done (%)	Total*	Don't eat
Roast lamb	0	16 (5)	130 (43)	138 (46)	16 (5)	300	17
Roast pork	0	2 (1)	53 (19)	191 (67)	38 (13)	284	32
Steak (pan fried)	2 (1)	58 (19)	152 (50)	78 (26)	13 (4)	303	11
Chicken (roast)	0	0	35 (27)	48 (38)	45 (35)	128	8
Sausages (pan fried)	0	0	40 (14)	217 (75)	31 (11)	288	27
Minced beef/hamburgers	0	0	53 (18)	209 (70)	35 (12)	297	18
Chicken livers	0	1 (1)	17 (22)	41 (53)	18 (23)	77	219

* the total column of respondents does not include the "don't eat" column therefore only those expressing a preference are used in percentage calculations.

As expected, pan-fried steak was the meat that people were most likely to prefer rare or raw.

5.3.6 Exposure summary

While red meat and meat products constitute a very commonly consumed food, the lack of any data on the prevalence of the organism in foods hampers any attempt to assess likely exposure. The organism if present in red meat is likely to face either one of two significant hurdles: freezing for processed meat products or cooking for fresh meat cuts.

5.4 Overseas Context

5.4.1 Seroprevalence in animals

The prevalence of antibodies for *Toxoplasma* in animals overseas derived from serological tests has been summarised in Table 7.

Table 7: Reported prevalence of *T. gondii* antibodies in overseas animals

Country	Number tested	Animal tested	Percentage seropositive	Reference
Australia (Tasmania)	160	Lambs	16.9	Munday, 1975
	145	Other sheep	61.7	Munday, 1975
	173	Vealers	2.3	Munday, 1975
	114	Other cattle	0	Munday, 1975
	30	Cracker pigs	22.3	Munday, 1975
	139	Other pigs	7.2	Munday, 1975
Australia	1,159	Sheep	7.4-9.2	O'Donoghue *et al.*, 1987
Australia	151	Tasmanian pademelons	3.3	Johnson *et al.*, 1988
	85	Bennett's Wallabies	17.7	Johnson *et al.*, 1988
Brazil	439	Goats	28.9	Pita Gondim *et al.*, 1999
	240	Sheep	18.7	
	194	Cattle	1.0	
	104	Water buffaloes	3.8	
Canada	2,800	Pigs	8.6	Gajadhar *et al.*, 1998
France	164	Lambs	22.0	Dumètre *et al*., 2006
	93	Ewes	65.6	
Iran	290	Cattle	0	Sharif *et al*., 2006
	400	Goats	30	
	588	Sheep	35	
Mexico	490	Cattle	11.9	Garcia-Vazquez *et al.*, 1993
	1203	Pigs	8.9	
	707	Goats	3.2	
Norway	NS	Sheep	18.0	Kapperud *et al.*, 1996
	NS	Cattle	5.1	Kapperud *et al.*, 1996
	NS	Pigs	2.5	Kapperud *et al.*, 1996
Serbia	611	Cattle	76.3	Klun *et al*., 2006
	511	Ewes	84.5	
	605	Pigs	28.9	
Spain (Grand Canary Island)	1052	Goats	63.3	Rodrìguez-Ponce *et al.*, 1995
USA	3,707	Pigs	32 (range <1	Smith, 1991

Country	Number tested	Animal tested	Percentage seropositive	Reference
			to 69)	
USA	5,936	Sheep	37	Smith, 1991
	2,449	Goats	23	Smith, 1991
USA	382	Black tailed deer	20	Smith, 1991
	30	White tailed deer	3	Smith, 1991
	93	Bison	3.1	Smith, 1991
USA	1264	Pigs	0.9*	Dubey *et al*., 1997
USA (Illinois)	4252 2617 391 188 18 128 95 61 1243	Pigs – finishing Pigs – sows Cats Raccoons Skunks Opossums Rats White footed mice House mice	2.3 15.1 68.3 67.0 38.9 29 6.3 4.9 2.1	Dubey *et al.,* 1995
USA	1897	Domestic pigs	47.4	Gamble *et al.,* 1999
USA	3479	Pigs	19.5	Patton *et al*., 1997
Worldwide	73,717	Pigs	22 (range 0 to 97)	Smith, 1991
Several surveys	5,862	Sheep	21	Smith, 1991
Several surveys	2,795	Goats	25	Smith, 1991
Several surveys	2,747	Horses	14.7	Smith, 1991

NS = Not Stated

* The low seroprevalence in these results is attributed to the lack of cats on the remote island where these feral pigs were tested, the presence of only one domestic cat was known on the island.

5.4.2 Prevalence in animal tissue

Pork has been regarded as the major vehicle for transmission of *T. gondii* infections in humans. Data has shown however that with the shift from small outdoor farms to modern intensive indoor rearing systems in Europe and North America, the prevalence in young market-weight pigs has been declining and pork consumption is unlikely to still be a major risk factor for toxoplasmosis (Dubey 1996c; Tenter *et al*., 2000). A small isolated farm in Massachusetts where modern methods were not used appears to support this view as 93% (51/55) of the 6 month old pigs harboured viable *T. gondii* cysts (Dubey *et al.,* 2002a). Prevalences in fattening pigs in several EU countries was reported to be <1% (Tenter *et al.,* 2000). In Zimbabwe, seroprevalence was lowest in commercial farms practicing good hygiene (19.75%, n=238) and highest in backyard scavenging pigs (35.71%, n=70). The authors concluded that modern intensive husbandry systems were important in reducing *T. gondii* infection in domestic pigs (Hove *et al*., 2005).

Data reported in reviews of analyses for *T. gondii* in animal muscle tissue are summarised in Table 8. Dubey (2000) has suggested that a higher risk of *T. gondii* in women who ate raw

sausages, salami and other cured meats could be explained by the higher prevalence of the organism in older animals that are more commonly used for such products.

Table 8: Reported prevalence of *T gondii* cysts in overseas animal tissue

Country	No. tested	Meat tested	% positive	Assay Method	Reference
Brazil	149	Fresh pork sausage	8.7	Mouse bioassay	Dias *et al.*, 2005
Canada	2,800	Pig heart and diaphragm	0	Mouse bioassay	Gajadhar *et al.*, 1998
England	67	Ready-to-eat cured meats	1.5	PCR*/Tissue culture	Warnekulasuriya *et al.*, 1998
UK	71	Retail meat; consisting of 57 pork 9 lamb 4 beef 1 beef/pork	38 33 67 25 100	PCR*	Aspinall *et al.*, 2002
USA	50	Pig diaphragm	24	Mouse bioassay	Jacobs *et al.*, 1960
	60	Beef cattle diaphragm	1.7	Mouse bioassay	Jacobs *et al.*, 1960
	86	Sheep diaphragm	9.3	Mouse bioassay	Jacobs *et al.*, 1960
	55	Pigs -6 month old - hearts and tongues (isolated small farms)	92.7	Cat bioassay	Dubey *et al.*, 2002a
	2094 2094 2094	Beef Chicken Pork	0 0 0.33	Cat bioassay followed by further cat and mice bioassay	Dubey *et al.*, 2005
NS	4,302	Cattle	5	NS	Smith, 1991
NS	7,313	Pig (most samples were not of edible tissue)	10	NS	Smith, 1991

NS = Not Stated

*PCR methodology detects presence of *T. gondii* DNA, not the presence of viable cysts.

In the Dubey *et al*. (2002a) study, the samples were obtained from 55 pigs destined for human consumption. Five hundred grams of heart and tongue material were fed to 55 *T. gondii*-free cats. Fifty-one cats (92.7%) shed 25 – 810 million *T. gondii* oocysts in their faeces. In a surprising finding, further analysis found that two of the infected cats had eaten apparently seronegative pig tissue (tests carried out were the Sabin-Feldman dye test, modified agglutination test and the Western blot test). This suggests that these tests are not entirely effective. The authors concluded that all meat should be cooked to industry guidelines before human consumption.

In conclusion, the data presented in Tables 7 and 8 tend to suggest that prevalences obtained when testing for cysts are lower than those obtained by serological tests. However, testing by PCR (Aspinall *et al.*, 2002) has shown, in one study at least, prevalences similar to those found by serology. This suggests that the method used can influence the results and it is not possible to say which is the "true" measure.

6 RISK CHARACTERISATION

Analysis of sera for infection determines the presence of IgG or IgM classes of antibodies specific for a *Toxoplasma* antigen. The presence of IgG antibodies (which persist in the body for long periods) indicates previous infection. IgM antibodies are a more recently available test, and this class of antibody rises early in infection. For infections with most other pathogens, IgM disappears in the weeks following primary infection. However, screening for toxoplasmosis is unusually difficult because of the persistence of the IgM antibody response. After primary *T. gondii* infection, IgM antibody frequently remains positive for many months or even years and may falsely indicate a recent infection (Morris and Croxson, 2004).

Seroconversion in pregnant women refers to cases where IgG and IgM are absent prior to conception, but appear in blood samples following delivery, thus demonstrating infection during pregnancy.

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence and outbreaks

An early investigation of toxoplasmosis in New Zealand was undertaken by Manning and Reid (1956). Sera from blood donors and student nurses (age range 16 – > 56 years) were collected and tested for antibodies using the Sabin-Feldman dye test – see Table 9. This test uses live virulent tachyzoites of *T. gondii* as the antigen to a series of dilutions of the test serum thus measuring the total amount of antibody in the test serum capable. The results show an increase in the percentage positive with age. Titre levels were also determined and were found to be higher in the younger age groups reflecting the more recent infection status. Sera from 9-year-old children from one school were also tested. Results are shown in Table 10. The number of positive results was considered unexpectedly high by the authors.

Table 9: Dye test results (student nurses and blood donors)

Age group	Sex	No. tested (total)	No. positive (%)	Overall percentage positive
16-25	M F	25 45	7 (28) 12 (27)	27%
26-35	M F	35 30	16 (46) 14 (47)	47%
36-45	M F	31 23	15 (48) 15 (65)	56%
46-55	M F	24 25	14 (58) 13 (52)	55%
Over 56	M F	9 11	6 (67) 7 (64)	65%
Total	**M** **F**	**124** **134**	**58 (47)** **61 (46)**	**46%**

Adapted from Manning and Reid, (1956)

Table 10: Dye test results (nine year old school children)

Sex	No tested	No positive (%)	No. positive at reciprocal titre 16 32 64 128 256 512
Male	22	9 (41)	- 1 - 3 3 2
Female	33	9 (27)	- - 2 2 1 4
Total	**55**	**18 (33)**	**- 1 2 5 4 6**

Adapted from Manning and Reid, 1956.

In respect to congenital toxoplasmosis, the incidence is unknown and New Zealand currently has no routine antenatal serological screening for *T. gondii* infection.

A study of the incidence of toxoplasmosis in pregnancy in New Zealand reported that New Zealand Health Information Service discharge data for ten years (probably 1989 to 1999) indicated only 27 cases of toxoplasmosis in women of child bearing age (Moor *et al.*, 2000).

Toxoplasmosis was a notifiable disease in New Zealand from 1987 to 1996. Only one case of congenital toxoplasmosis was notified during this period (in 1990). This low notification rate has been ascribed to under-reporting, as exemplified by National Health Institute data that indicate, in 1988, nine diagnoses of clinical and serological toxoplasmosis were made in babies (Moor *et al.*, 2000).

The International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) is a coding of diseases signs, symptoms etc developed by the World Health Organisation (WHO). The following ICD-10 codes relate to toxoplasmosis:

B 58 Toxoplasmosis (excludes congenital toxoplasmosis)
- B58.0 Toxoplasma oculopathy
- B58.1 Toxoplasma hepatitis
- B58.2 Toxoplasma meningoencepthalitis
- B58.3 Pulmonary toxoplasmosis
- B58.8 Toxoplasmosis with other organ involvement (myocarditis, myositis)
- B58.9 Toxoplasmosis unspecified

P 37 Other congenital infectious and parasitic diseases
- P37.1 Congenital toxoplasmosis due to hydrocephalus

Table 11 shows the number of cases discharged from public hospitals from 2000 to 2006 with a primary diagnosis of toxoplasmosis and a breakdown of the different clinical manifestations in terms of ICD 10 code (Chris Lewis, New Zealand Health Information Service, personal communication, 2007). Readmissions of the same patient have been excluded.

Table 11: Public hospital discharge records for other acquired toxoplasmosis manifestations by year, gender and age range

	Year	Male	Female	Age range
B58.0 Toxoplasma oculopathy	2000	4	0	7-57
	2001	1	0	44
	2002	0	4	22-71
	2003	3	0	24-62
	2004	2	1	3-51
	2005	4	4	18-64
	2006	1	2	11-43
Total: 26		**15**	**11**	
B58.2 Toxoplasma meningoencepthalitis	2003	1	0	38
	2004	1	0	56
	2006	0	2	36-55
Total: 4		**2**	**2**	
B58.3 Pulmonary toxoplasmosis	2001	0	1	42
	2004	0	1	35
	2005	1	1	23-52
Total: 4		**1**	**3**	
B58.8 Toxoplasmosis with other organ involvement (myocarditis, myositis)	2000	2	1	33-86
	2003	2	0	43-55
	2004	2	0	36-67
	2005	3	3	22- 44
Total: 13		**9**	**4**	
B58.9 Toxoplasmosis unspecified	2000	4	2	36 – 48
	2002	3	2	27 – 57
	2003	3	2	11 – 40
	2004	3	1	21 – 41
	2005	5	3	21 – 54
	2006	6	3	1 – 50
Total: 37		**24**	**13**	
Total for B58: 84		**51**	**33**	

H32.0 (chorioretinitis) was a secondary diagnosis in 11 of the cases with a primary diagnosis of toxoplasma oculopathy r recorded from 2000 to 2006. H22.0 (iridocyclitis) was secondary in 5 of the cases.

Toxoplasmotic meningoencephalitis was listed as a contributory factor in two HIV-associated deaths, one in each of the years 2001 and 2003.

In terms of congenital diagnoses records, as a primary diagnosis of P37.1, there were 3 fatalities recorded, all females in the years 2000, 2001 and 2003. Two were neonates while one was 16 years old. For hospital discharges, there were 3 cases recorded in 2000, and 1 each in years 2001, 2002, 2003 and 2006, all neonates (Chris Lewis, NZHIS, personal communication, 2007).

Metcalfe *et al.* (1981) reported on IgG levels to *Toxoplasma* in populations in Auckland, Hamilton, Taranaki, Wellington and Napier/Hastings in the period of 1976 to 1978. Overall 45% of females and 48% of males had antibody titres that suggested prior infection (reciprocal antibody titres of 64 or greater).

A survey of 566 pregnant women in Hamilton (Cursons *et al.*, 1982) reported the prevalence of antibodies to *T. gondii* as shown in Table 12.

Table 12: Reported prevalence of *Toxoplasma gondii* antibodies in pregnant women in Hamilton

Age Group and number (n=566)	Percentage of sample population	Positive for antibody (%)	Seroconversion rate estimated (% per annum)
15-20, n=123	21.7	58.5	-
21-25, n=203	35.9	57.1	-
26-30, n=168	29.7	60.0	0.6
31-35, n=54	9.5	68.5	1.7
>35, n=18	3.2	56.0	-

Based on these and earlier data, a seroconversion rate of 0.62% per annum has been calculated, which was used as the basis for a further calculation to predict that 164 maternal infections could be expected annually resulting in 66 foetuses becoming infected (Moor *et al.*, 2000). Actual diagnoses of *Toxoplasma* infection in pregnant women are much lower; the same study reported that although 12% of live births occur in the Wellington region, only 10 cases of toxoplasmosis in pregnancy were diagnosed in Wellington from 1989 to 1997. It was suggested that this discrepancy was "due in part to the relatively asymptomatic nature of *Toxoplasma* infection and in part to a lack of awareness amongst many of the caregivers".

These analyses do not include any information to indicate whether the infection was acquired from food (undercooked meat) or other sources, particularly cats or soil in which cats have defecated.

A study involving pregnant women submitting their first routine set of antenatal blood tests in Auckland in 2000 tested 500 serum samples across five age groups, 100 samples per age group (Morris and Croxson, 2004). Results are shown in Table 13. Testing was conducted for both IgG and IgM antibodies.

Table 13: Seropositivity in blood samples from pregnant women Auckland, n=500

Age group (years)	IgG positive (%)	IgM positive (%)
<20	36	4
21 – 25	24	5
26 – 30	29	0
31 – 35	34	2
>36	40	1
All ages	**33**	**2.4**

The authors used these data, along with the numbers of births to women in these age ranges in the Auckland region, to estimate that up to 2.2% of women could have initial antenatal serology consistent with recent infection. Infections during the first trimester have a 10% chance of being transmitted to the foetus, suggesting that 2 cases of first trimester congenital toxoplasmosis could be occurring per 1000 pregnancies in Auckland. However, this level of clinical disease is not currently being detected. Due to the potentially unnecessary anxiety that antenatal screening might cause, and the uncertain benefit of medical interventions, the authors concluded that antenatal screening for *T. gondii* is not currently justified in New Zealand and that risk communication is the preferred option.

6.1.2 Clinical consequences of toxoplasmosis infection and people with AIDS

Toxoplasmosis is a potentially important infection for people with AIDS. Opportunistic infection was a concurrent main clinical indicator for 570 of the 891 people notified with AIDS from 1983 to 2005 in New Zealand (either living or dead). A specific infection is not always reported, but toxoplasmosis or *T gondii* encephalopathy was reported for 13 of these 570 cases (Sue McAllister, Otago University, pers. comm., October 2006).

6.1.3 Case control studies and risk factors

No case control studies or risk factor studies have been conducted in New Zealand.

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

It has been suggested that nearly one third of humanity has been exposed to *T. gondii* (Hill *et al*., 2005; Dubey and Beattie, 1988). In the UK and USA, 16 – 40% of people are considered to be infected, while in Central and South Amercia and continental Europe infection spans 50 – 80% of the population (Dubey and Beattie, 1988; Smith, 1997). Joynson (1992) estimated the overall rate of toxoplasmosis in the UK as between 23 and 33% with regional differences. With age, seropositivity increases by 0.5-1% until 50% of people are infected by the time they reach 60 years old.

Sukthana (2006) collated the seropositivity rates in Europe, the Americas and Southeast Asia. Additional surveys found have been added to this table and are presented in Table 14. It is difficult to comment on these rates due to the differences in ages, tests and range of results. However, in general, Western Europe rates are below 50% (except for France). Scandinavian countries and the USA have low seropositivity rates at generally below 30%, while South American countries and African countries report the highest rates (generally above 50%).

In the India survey (Joshi *et al.,* 1998), the dietary habits (vegetarians and non-vegetations) were also examined with respect to seropositivity rates. No statistical differences were found.

The seropositivity rates in the African studies are all over 43% except for Niger (18%). It appears that the prevalence of toxoplasmosis was higher in humid coastal regions as opposed to the drier inland desert areas.

Worldwide, the number of cases of congenital toxoplasmosis have been estimated at between 140,900 and 1,127,200, based on an estimated rate of 0.1 to 0.8% of 140.9 million live births in 1992 (Roberts *et al.*, 1994).

Table 14: Seropositivity rates overseas in humans

Countries	Year	Seropositivity (%)	References
Western Europe			
Austria	1998	43	Moese and Vander-Moese, 1998
Belgium	1990	50	Luyasu *et al*., 1997
France	-	up to 75	Tenter *et al*., 2000
Germany	2004	26-54	Gross, 2004
Italy	-	18-60	Tenter *et al.,* 2000
Netherlands	1996	40.5	Kortbeek *et al.,* 2004
Spain	2004	28.6	Munoz Batet *et al*., 2004
Switzerland	1990-91	46	Jacquier *et al*., 1995
UK	-	23-33	Joynson, 1992
Scandinavia			
Denmark	1999	27.8	Lebech *et al.,* 1999
Finland	1995	20.3	Lappalainen *et al.,* 1995
Norway	1992-94	10.9 (preg. women)	Jenum *et al*., 1998
	-	56.6 (women)	Smith, 1991
	-	21.8 (military rec.)	Smith, 1991
Sweden	2001	14.0-29.4	Evengård *et al.,* 2001
Central & Eastern Europe			
Croatia	-	36.4	Tonkic *et al*., 2002
Poland	-	46.4-58.5	Sroka, 2001
Slovenia	1996-99	34 (preg. women)	Logar *et al.,* 2002
The Americas			
USA	-	9.9 (military rec.)	Smith *et al*., 1996
	2004	16-40	Dubey, 2004
	1999-2000	15.8 (aged 12-49)	Jones *et al*., 2003a
	1988-1994	22.5 (age > 12)	Jones *et al.,* 2001
	1988-1994	15 (women 15-44)	
	1988-1994	25.4 (age ≥ 20) (males -26.2) (females - 24.7)	Kruszon-Moran & McQuillan, 2005
Central America			
Costa Rica	1996	76	Arias *et al*., 1996

Countries	Year	Seropositivity (%)	References
Cuba	1987	29.7	Machin Sanchez *et al.,* 1993
Mexico	-	35	Tenter *et al*., 2000
Panama	1988	90 (at age 60)	Sousa *et al.,* 1988
South America			
Venezuela	-	63	Diaz-Suarez *et al*., 2003
Argentina	-	72	Tenter *et al*., 2000
Brazil	-	59	Tenter *et al.,* 2000
West Indies	1991	57	Prabhakar *et al.,* 1991
	-	45 (1-19 yrs)	Rawlins and Prabhakar, 1989
Asia			
Indonesia	-	58[a]	Konishi Houki *et al.,* 2000
Malaysia	-	44.8	Nissapatorn & Abdullah, 2004
India	-	1.5 – 21	Mohan *et al.,* 2002
	-	17.2	Joshi *et al*., 1998
China	-	0.7	Smith, 1991
Japan	-	2.9 (Age 20-29) 40 (Age 70-90) 29 (males) 16 (women)	Smith, 1991
Africa			
Nigeria (Ibadan)	-	78 (preg. women)	Jones *et al*., 2001
Nigeria (S. Delta)	-	83	Jones *et al*., 2001
Somalia	-	44	Jones *et al*., 2001
Niger	-	18	Julvez *et al*., 1996
Egypt		43	el-Nawawy *et al*., 1996

a male:female ratio = 63:52

6.2.1.1 USA

It has been estimated that 0.6% of the US population experiences an acute toxoplasmosis infection each year, representing approximately 1,500,000 infections per year (Mead *et al.*, 1999). The USDA estimate that 50% of *T. gondii* infections are caused by raw or undercooked meat consumption (Buzby and Roberts, 1996), and a community-based Maryland study comparing meat eaters with non-meat eaters supports this assumption (Roghmann *et al.,* 1999). This means that an estimated 750 deaths per annum are due to toxoplasmosis and, if one half or 375 deaths are due to raw or undercooked meat, then toxoplasmosis ranks as the third leading cause of foodborne deaths in the USA (Mead *et al*., 1999).

Jones *et al.* (2003a) and Lopez *et al.* (2000) have estimated a range of between 400 and 4,000 cases of congenital toxoplasmosis each year in the USA.

Toxoplasmic encephalitis occurs in approximately 7% of USA AIDS patients, although this proportion declined from 1992 to 1997 (Schwartzman, 2001). A worldwide proportion of 10% has been estimated for the number of AIDS patients that die due to toxoplasmosis, while the equivalent figure in Europe has been estimated as 30% (Luft and Remington, 1992 cited in Hill and Dubey, 2003).

6.2.1.2 Europe

The Eurotoxo project, launched in 2002, is a European consensus initiative that has worked to provide a consensus on the current state-of-science knowledge on toxoplasmosis. The aim is to construct a research agenda from current knowledge so that policy decisions can be made that best prevent congenital toxoplasmosis and its consequences (See website: European Toxo Prevention Project http://eurotoxo.isped.u-bordeaux2.fr/WWW_PUBLIC/US-EUROTOXO-PublicAccess-Frame.htm).

In 2004, the European Food Safety Authority collected data from 25 member states and Norway covering 11 zoonotic agents, including *Toxoplasma* (EFSA, 2005). Eighteen States reported a total of 1,736 human cases of toxoplasmosis in 2004, 45 of which were registered as congenital infections. Data for the years 2000 – 2003 were of a similar order. The overall EU incidence was 0.6 cases per 100,000 population. As some countries only report certain clinical aspects of the disease or do not report the illness at all, this rate is considered an under-estimation. For further information on the characteristics of the European surveillance systems, refer to the Eurotoxo survey on European surveillance systems (Bénard and Salmi, 2005).

The 2005 Zoonoses Report for the United Kingdom (DEFRA, 2006) reported that between 1998 and 2005 there was an average of 117 toxoplasmosis cases reported per annum directly from National Health Service laboratories (total UK population in 2006 was 60.5 million). There were also an additional 300-500 cases identified each year through the *Toxoplasma* Reference Unit which captures the milder cases affecting the general population and also the more severe, life-threatening symptoms affecting HIV/AIDS patients, organ transplant recipients and unborn children.

The 2004-2005 British Paediatric Surveillance Unit Annual Report (BPSU, 2005; Gilbert *et al.,* 2006) included symptomatic toxoplasmosis in childhood to inform the debate on neonatal screening. The research in the UK (Gilbert *et al*., 2006) studied 38 cases of toxoplasmosis infected children (<16 years) between the years 2002 and 2004. Key points made were that confirmed symptomatic cases were reported more often by opthalmologists (19 cases) than by paediatricians (4) and the majority (22/38: 58%) were due to congenital infection, leaving 16/38 (42%) as post-natally acquired toxoplasmosis – all 16 had retinochoroiditis symptoms and no other complications. All 16 children post-natally infected, presented after 4 years of age, 14 of the 16 (88%) presenting at 10 or more years of age. In total, 31 of the 38 cases had ocular toxoplasmosis, with almost half of these cases infected post-natally.

6.2.1.3 Australia

Toxoplasmosis is not a notifiable disease in Australia and there are few data on the incidence of the disease. However, extrapolation of data from overseas (principally the USA – Mead *et al.,* 1999) has been used to estimate the extent of foodborne toxoplasmosis in Australia (Australian Government, 2005).

Total cases (95% Credible Interval (CrI))	% Foodborne (95% CrI)	Foodborne illness (95% CrI)
17,100 (9,000 – 26,000)	35 (0-71)	5,900 (0-13,900)

This represents the majority of the estimated 6,000 cases of "other" (i.e. non-gastrointestinal) foodborne illness in Australia.

Most other information concerning toxoplasmosis in Australia relates to the congenital form of the disease. The annual number of cases of congenital toxoplasmosis in Australia has been estimated as 534 (0.2% of 0.3 million live births) (Roberts *et al.*, 1994). An early paper (Sfameni *et al*., 1986) examined the case for antenatal screening for *Toxoplasma* amongst other infections such as rubella and cytomegalovirus (CMV). The sera of 3,463 pregnant women attending antenatal clinics in Melbourne were tested during a 12 month period from March 1983. Prevalence of *Toxoplasma* antibody rose from 40.2% in women aged 16-20 to 54.8% in those aged 36-40. Over the 20 year span, the increase was 7.3/1000 women years (5.5/1000 during a 9 month period) – a similar rate to the observed seroconversion rate.

A survey for congenital toxoplasmosis in Western Australia (Walpole *et al*., 1991) involved approximately a quarter of infants born in the State. Sera from 10,207 pregnant women and cord blood specimens from 18,908 infants were tested (7523 of these were linkable to maternal sera). Fourteen infected mothers were identified (specific IgM antibodies present), 11 from maternal sera and 3 from the cord blood (0.23 per 1000 births to non-immune mothers). Maternal immunity was indicated in 35% (3544/10207) at the first ante-natal visit. Subsequent tests on the eleven cases indicated no serological or clinical evidence of congenital toxoplasmosis in the offspring of these mothers. The rate of maternal infection in susceptible pregnancies was calculated to be 1.6 per 1,000. The maternal-foetal transmission rate was estimated at $\leq$ 24%.

There were no clinical signs of congenital infection in the three infants associated with the positive cord blood but long-term checks would be required. Thirteen of the 14 infected mothers were interviewed, only three identified a known risk factor. The authors concluded that an ante natal toxoplasmosis screening programme was not justifiable in Western Australia.

6.2.1.4 Worldwide data for seroconversion during pregnancy

Worldwide, data for seroconversion during pregnancy from a number of studies are summarised in Table 15. Kemmeren *et al*. (2006) calculated from a number of studies in the Netherlands, a prenatal infection rate from mother to foetus of 23 per 10,000 births.

Table 15: Seroconversion rates during pregnancy for *T. gondii* infection

Country	Number tested	Seroconversion (%)	Reference
Australia (Western)	10207 women 18,908 cord blood	1.6 per 1000	Walpole *et al*., 1991

Denmark	NS	0.21	Evengård *et al.*, 1999
England	1621	0.023	Zadik and Siddons, 1995
England	13328	3-16 infected foetuses per 100,000 pregnancies	Allain *et al.*, 1998
Finland	NS	0.24	Evengård *et al.*, 1999
France	7605	0.1	Smith, 1991
France	951	Native French 2.3 Immigrant 1.6 (probability of infection)	Smith, 1991
Norway	NS	0.17	Evengård *et al.*, 1999
Sweden	3094	0.13	Evengård *et al.*, 1999
USA	NS	0.38-0.75 (estimate)	Roberts and Frenkel, 1990
World	NS	0.1-0.8 (estimate)	Murrell, 1995
NS	95929	0.5	Smith, 1991

NS = Not Stated

6.2.2 Contribution to outbreaks and incidents

Sukthana (2006) has collated information on the percentage of *Toxoplasma* infections associated with meat in Europe (Table 16). This information suggests that pork is less important than other red meat types as a transmission vehicle for tissue cysts.

Table 16: Percentage of human toxoplasmosis associated with meat types consumed

Country	Percentage			
	Beef	**Pork**	**Lamb**	**Salami**
Belgium[1]	6	2	10	10
Denmark[1]	27	2	8	4
Italy[1]	12.5	3	0.5	12.5
Norway[1,2]	19	3	21	3
Switzerland[1]	8	13	10	5

1 Cook *et al.*, 2000, 2 Kapperud *et al.*, 1996
Source: Sukthana (2006).

Reported outbreaks of foodborne toxoplasmosis associated with rare meat are summarised in Table 17.

Table 17: Summary of information on outbreaks of infection with *T. gondii* associated with raw or rare red meat

Country	Number of cases	Implicated Meat	Reference
Australia	5	Raw lamb	Smith, 1993
Australia	12	Undercooked kangaroo	Robson *et al.*, 1995

Brazil	95	Rare meat (hamburger?)	Smith, 1993
Canada	4	Raw seal and/or caribou	McDonald *et al.*, 1990
England	3	Raw lamb	Smith, 1993
England	1	Raw or rare steak	Smith, 1993
Korea	3	Raw pig liver and spleen*	Choi *et al.*, 1997
Korea	5	Raw pig liver*	Choi *et al.*, 1997
USA	2	Rare meat	Smith, 1993
USA	1	Rare meat	Smith, 1993
USA	3	Rare/raw venison	Sacks *et al.*, 1983
USA	6	Rare lamb	Smith, 1993
USA	4	Raw beef	Smith, 1993
USA	5	Rare meat (hamburger)	Smith, 1993

* Although offal, these entries are included in the table for information.

A search of the Pubmed database (21.11.06) did not locate reports of any further outbreaks associated with meat.

An outbreak of toxoplasmosis associated with a municipal drinking water facility has been reported in Victoria, Canada (Bowie *et al.*, 1997). Domestic cats and cougars living in the area of the surface water reservoir were shown to be shedding oocysts (Aramini *et al*., 1999). The water supply was unfiltered, and although disinfection using chloramination was used, this method is unproven against *T. gondii*.

An outbreak between November 2001 and January 2002 in Brazil was linked to contaminated water and commercial ice cream prepared with the water (de Moura *et al.,* 2006). An unfiltered municipal treated water supply was epidemiologically linked to and confirmed by PCR as the source of the outbreak. A litter of kittens delivered and living on top of the reservoir tank fitted the timeframe of contamination. Cracks in the roof of the reservoir allowed rainwater penetration and possibly infected cat faeces through into the reservoir. In addition, chlorination levels were inadequate to destroy the oocysts. Confirmation of *T. gondii* in the kittens could not be made as the kittens could not be caught.

6.2.3 Overseas studies

A key cohort study overseas is part of the Eurotoxo project. The study includes 1200 toxoplasma-infected women, spread over 15 centres in 7 countries in Europe and Brazil. The women in the study were recruited between 1997 and 1999, enabling a long term study of the children born subsequently. In particular, risks of transmission, signs of damage and effect of prenatal treatments have been looked at. The project has already resulted in a number of publications, these are listed on a website link to the European Multicentre Study on Congenital Toxoplasmosis;
http://www.ucl.ac.uk/paediatric-epidemiology/EMSCOT/emscot.html.

A systemic review of risk factors for *T. gondii* infection in pregnant women is also part of the Eurotoxo project. This review identified four case-control studies, the findings are summarised below, as well as a recently published English study. The Eurotoxo authors concluded that case-control studies are not the best evidence on which to investigate risk factors, especially using personal retrospection that introduces recall bias. From these studies, risk factors vary according to local food customs, food hygiene and lifestyles. The

authors thought that a "transversal" designed study was of greater benefit (i.e. prospective, information on both risk and disease infection collected simultaneously). Only one such study was found by the review: conducted in Serbia-Montenegro conducted between 1988 and 1991 (Bobic *et al*., 1998). The study of 1157 reproductive age females (15years-49 years) in Belgrade found that undercooked meat consumption and exposure to soil were significant, while ownership of cats had no influence.

A case control study in France investigated risk factors for *Toxoplasma* infection in pregnancy (Baril *et al.*, 1999). A total of 80 pregnant women who seroconverted to *Toxoplasma* were matched with 80 pregnant women who had repeatedly negative tests. The risk factors determined from a multi-variate analysis were:

- Consumption of undercooked beef OR=5.5 (95% CI 1.1-27);
- Having a pet cat OR=4.5 (95% CI 1.0 – 19.9);
- Frequent consumption of raw vegetables outside the home OR 3.1 (95% CI 1.2 – 7.7).

Undercooked meat was defined in this study as rare if the centre of the meat was still raw and medium if the centre was still pink.

Insufficient numbers of study participants reported consumption of undercooked pork to determine an odds ratio. Poor handwashing (OR=9.9, 95% CI: 0.8 – 125), and consumption of undercooked lamb (OR=3.1, 95% CI 0.85 – 14) had elevated but not statistically significant odds ratios in the multi-variate model. Receipt of documentary advice on prevention was associated with a lower risk of infection.

A study in Naples (Buffolano *et al.*, 1996) was conducted with pregnant women who were interviewed with regard to risk factors and tested for their levels of serum anti-*Toxoplasma* IgG and IgM antibodies. The levels of these were used to determine the time of infection (IgM positive and IgG positive = recently infected, IgG negative = susceptible and IgM negative, IgG positive = previously infected). Complete results were available for 3518 women, of whom 42 (1.2%) were recently infected, 1380 (39%) were previously infected and 2096 (60%) were susceptible.

Compared to the susceptible group, recent infection (i.e. IgM positivity) was associated with:

- Eating cured pork (more than once a week or once a month) OR 2.9 (95% CI 1.6-5.5)
- Eating raw meat (more than once a week or once a month) OR 2.6 (95% CI 1.4-4.7)
- Gardening (once a week or once a month) OR 2.0 (95% CI 1.1-3.7)

Parity, rural/urban residence, living on a farm, or owning a cat were not significant. This was the first report identifying cured pork as a significant risk factor for recent *Toxoplasma* infection. The authors recommended further studies to confirm this association and to determine viability of tissue cysts in cured pork products. There was also a dose dependent response between frequency of consumption of cured pork (weekly or monthly) and raw meat and the odds ratio.

A prospective case control study using similar criteria to that of the Naples study for defining cases was carried out in Norway from 1992 to 1994 (Kapperud *et al.*, 1996). From 37,000

pregnant women, a total of 63 women with recent primary infection and 128 seronegative controls were matched. The independently associated risk factors were:

- Eating raw or undercooked minced meat products OR 4.1 (95% CI 1.5-11.2)
- Eating unwashed raw vegetables or fruits OR 2.4 (95% CI 1.1-5.6)
- Eating raw or undercooked mutton OR 11.4 (95% CI 2.1-63.1)
- Eating raw or undercooked pork OR 3.4 (95% CI 1.1-10.4)
- Cleaning cat litter box OR 5.5 (95% CI 1.3-22.7)
- Washing the kitchen knives infrequently after preparation of raw meat, prior to handling another food item OR 7.3 (95% CI 1.1-50.2)

More recently a multi-centre study in Europe again used seroconversion to define cases (n = 252), which were compared with controls (n = 852) (Cook *et al.*, 2000). The following factors were the most strongly predictive of acute infection in pregnant women;

		p value
• Eating raw/undercooked beef	OR 1.73 (95% CI 1.1-7.2)	0.01
• Eating raw/undercooked lamb	OR 3.13 (95% CI 1.4-7.2)	0.007
• Eating "other"* meat	OR 4.12 (95% CI 1.6-10.9)	0.004
• Contact with soil	OR 1.81 (95% CI 1.2-2.7)	0.005
• Travel outside of Europe/USA or Canada	OR 2.33 (95% CI 1.3-4.1)	0.003

* specifically venison, horse, rabbit, whale and game birds

Weaker associations ($p>0.05$) were observed for tasting meat during meal preparation, eating salami, drinking unpasteurised milk and working with animals. Contact with cats, kittens, cat faeces or cats who hunt for food were not identified as statistically significant risk factors, possibly because the excretion of oocysts after infection is limited to only a few weeks. Between 30 and 63% of the infections could be attributed to meat consumption (including cured meats), and 6 to 17% to contact with soil.

A case control study in Jordan compared seropositive (n = 132) and seronegative (n = 148) pregnant women (Jumaian, 2005). Consumption of undercooked meat ($p<0.0001$) and contact with soil ($p<0.022$) were significant risk factors, whereas ownership of a cat was not.

6.2.4 Risk assessments overseas

A probabilistic model that describes incidence of the disease has been developed but only a conference abstract has been located (Cassin *et al.,* 1996). In discussion of this model (Lammerding and Paoli, 1997), three key factors are reported as being simulated: cat ownership, consumption of implicated products, and the age distribution of pregnancy. Note that the majority of case-control studies already discussed, do not cite cat ownership as a risk factor.

Two models to estimate the incidence of toxoplasmosis in the UK have been published. A model to estimate time and age-specific incidence was based on 3,785 blood samples collected from patients aged 2 – 100 in South Yorkshire, England between 1969 and 1990 (Ades and Nokes, 1993). The results showed that incidence appeared to have fallen sixfold between 1915 and 1970, remaining stable in the following 20 years up to 1990. The

incidence was higher in children than in adults, and in women throughout the childbearing period was estimated as 0.07 or less per 100 susceptible persons.

A recent study (Nash *et al.,* 2005) established the relative importance of risk factors for an antenatal population in England. *Toxoplasma* immune status was determined by immunoassay, and a questionnaire investigated dietary and environmental factors of 1897 women. Seroprevalence was 172/1897 (9.1%) confirming that the prevalence of infection has continued to decline since the 1960s (Ades and Nokes, 1993; Joynson, 1992; Walker *et al.,* 1992). A significantly higher seroprevalence was associated with;

- Rural location of childhood home;
- Childhood home in Europe (excl. UK);
- Feeding a dog raw meat;
- Increased age.

Living with a cat or kitten as a child resulted in a non-significant higher prevalence.

In contrast to mainland European studies, there were weak associations with diet. Table 18 displays the red meat consumption and raw meat handling factors investigated in the study:

Table 18: Relative importance of risk factors for an English antenatal population

Question	Response	Cases sero +ve	Cases sero -ve	RR	95% CI	*P* value
Eaten beef, pork or lamb in the past 10 years?	Yes	166	1641	1.91	0.72-5.01	
	No	4	79			0.245
How was beef eaten?	Not eaten	9	131			0.346
	Rare	14	96	2.12	0.88-5.1	
	Medium	36	332	1.58	0.74-3.36	
	Well done	111	1163	1.39	0.69-2.8	
How was pork eaten?	Not eaten	12	172			0.043
	Rare	2	3	9.56	1.46-62.76	
	Medium	6	25	3.44	1.19-9.98	
	Well done	150	1522	1.41	0.77-2.59	
How was lamb eaten?	Not eaten	22	233			0.624
	Rare	1	6	1.77	0.20-15.33	
	Medium	18	134	1.42	0.74-2.75	
	Well done	129	1349	1.01	0.63-1.63	
Often fed cat/kitten raw meat	No	143	1540		1.10-2.35	
	Yes	28	177	1.61		0.856
Often fed dog raw meat	No	138	1528			
	Yes	34	189	1.84	1.30-2.61	0.650
Smoking while handling raw meat	No	167	1683			0.188
	Yes	5	25	1.85	0.82-4.16	

RR = Relative risk, CI = Confidence interval

There were weak associations with eating undercooked or medium-cooked pork that were not significant after logistic regression. There was a significant association with feeding a dog raw meat but no association between feeding the same to a cat or kitten. There was no association with handling raw meat in the kitchen but there was a non-significant association between smoking and handling of raw meat.

The consumption frequency of meats was also investigated and associations were apparent with beef tongue (P=0.092), lamb chops (P=0.085), beefburgers (P=0.018) and cured pork products (P<0.001) but after multi-variate logistic regression, the results were not significant.

A single multi-variate analysis was also carried out on the preference for 18 fresh and frozen food purchases prior to their pregnancies. For 15/18 products where the preference was frozen, the women had a lower relative risk for seropositivity than a preference for fresh food. Frozen purchases with a higher relative risk were beef tongue, pork roast and lamb roast. There was no attempt to determine whether a valid statistical difference occurred between fresh versus frozen products because no valid statistical method could be found by the authors.

6.3 Estimate of Risk for New Zealand

The reported seroprevalence of antibodies to *Toxoplasma* infection in New Zealanders varies from 25 - 65% in male and female adults in the 1950s, 56 - 68% in pregnant women in a study in the early 1980s, and 33% in pregnant women in Auckland in 2000. These percentages are in keeping with reports for seropositivìty in Europe and the USA (Table 14). In the Netherlands the seropositivity in 1996 was estimated to be 40.5% (Kemmeren *et al.*, 2006). Estimates of seroconversion in pregnant women for New Zealand are similar to those for overseas (Table 15).

Data on the prevalence of infection of animals in New Zealand are scant, and no surveys of meats have been found. Data on the prevalence of infection in overseas animals are quite variable and this precludes the development of a proper exposure assessment. Further research on the prevalence of infection of meat animals (particularly pigs) in New Zealand, together with a survey to determine the presence of contamination (preferably with some estimate of infectivity) in meat for retail sale, would be required for a proper risk assessment.

The multicentre case control study from Europe found that between 30 and 63% of *T. gondii* infections could be attributed to meat consumption (including cured meats) (Cook *et al.*, 2000). Although pork is often cited as a high-risk meat, it is clear from the outbreak and case control studies that other meats are also associated with transmission. Eating contaminated food has been estimated to cause 50% of *T. gondii* infections in the USA (Mead *et al.*, 1999).

Although data on the prevalence of antibodies in the blood of animals may over-estimate infectivity, it seems likely that New Zealanders are exposed to infective cysts via undercooked meat. However, there is no information currently available to link cases of *Toxoplasma* infection with foodborne transmission, or to assess its importance relative to other transmission routes.

6.4 Risk Categorisation

The rationale for categorisation of food/hazard combinations is presented in Appendix 1.

The proportion of severe outcomes of *Toxoplasma* infection in immunocompetent people is very low. However, the proportion of severe outcomes resulting from congenital toxoplasmosis is significant at 5%, and so this infection has been assigned the highest severity category. It is not possible to assign a category based on incidence, as there is insufficient information on which to base such an assessment for New Zealand. However, current reporting systems suggest that severe disease caused by *T. gondii* is very uncommon in New Zealand.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
Toxoplasma gondii in red meat and meat products	1 (>5% serious outcomes for congenital toxoplasmosis)	Rare but robust information is lacking		

7 RISK MANAGEMENT INFORMATION

When considering preventative measures for acquired toxoplasmosis, the culture and beliefs of the population should be taken into account rather than giving orthodox health education (Sukthana, 2006). This advice comes from the different transmission routes attributed worldwide and whether direct ingestion of cysts via raw or undercooked meat is likely to be important, or whether transmission stems from ingestion of oocysts from cat excreta in food or soil. The subject of this Risk Profile is the direct ingestion of the cyst from red meat animals.

7.1 Relevant Food Controls

Prevention or minimisation of infection with *T. gondii*, at least in pigs, appears to be possible through the use of confinement systems for production. These include bird and cat-proofed buildings, feed storage in enclosed silos, use of pelleted and heat-treated feed, not keeping cats on finishing sites and regular rodent control programmes. Such measures have been shown to almost eliminate seroprevalence in pigs in North Carolina (Davies *et al.*, 1998).

Recommendations for the prevention of toxoplasmosis in the United States have been published (Lopez *et al.*, 2000). The recommendations were for the control of foodborne pathogens in general, rather than being specific for *Toxoplasma*:

- To cook meat to a safe temperature (internal temperature of 145°F (62.7°C) for beef, lamb and veal roasts and steaks, 160°F (71.1°C) for pork, ground meat and wild game, 180°F (82.2°C) for poultry); (note that cooking temperature for poultry has since been revised down to 165°F (73.8°C));
- Peel or thoroughly wash fruits and vegetables before eating;
- Clean cooking surfaces and utensils after they have contacted raw meat, poultry, seafood or unwashed fruits or vegetables.

Smith (1991) has summarised the following advice from a number of authors to prevent *T. gondii* infection in the home environment:

- All meats should be cooked to at least 70°C, and meats not tasted during preparation/cooking;
- Hands, cutting boards, kitchen-tops, utensils should be thoroughly washed after raw/undercooked meat contact. Do not touch mucous membranes (nose, eyes, mouth) until hands are washed;
- Thoroughly wash fruits and vegetables;
- Protect food from cockroaches, flies or other insects;
- Feed pets only dry, canned or thoroughly cooked foods;
- Freeze meat to at least –12.5°C.

7.1.1 EFSA Review

The European Food Safety Authority provided an overview of their recommendations concerning *Toxoplasma gondii* in a document published in 2007 (EFSA, 2007).

This stated:

"Consideration of risk factors and public health priorities

- The consumption of undercooked meat containing infective tissue cysts (bradyzoites) and ingestion of infective oocysts (sporozoites) shed by cats in the environment are the main risk factors.
- Water containing infective oocysts may be an infection source or transmission vehicle for humans.
- *Toxoplasma* prevalence in livestock may be expected to rise due to an increase in outdoor rearing of animals that, as a result, are more likely to come in contact with the environmental infective stage (i.e. sporulated oocysts shed by cats).

Recommended actions to be taken to improve the protection of public and/or animal health

- Intervention strategies should be enforced to reduce the access of *Toxoplasma* into the food chain. In this connection hygiene conditions in animal husbandry and food processing procedures have to be highlighted and observed.
- Education campaigns with special focus on vulnerable groups such as pregnant women and immuno-compromised persons are important so as to ensure that the risks of acquiring *Toxoplasma* infection through contaminated food or from the environment are addressed.
- Networking between the Public Health and Animal Health sectors should be improved in order to further understanding of the transmission and epidemiology of *Toxoplasma* and toxoplasmosis. In outbreaks, tracing from fork to farm should be improved in order to better identify the source of the infestation and put appropriate control measures in place.
- Case definitions of toxoplasmosis should be further developed and harmonised so as to ensure a better comparison of data.
- Sensitive, robust and reproducible *Toxoplasma* detection methods that are harmonised and standardised, which are essential for efficient monitoring, require further development.

Suggested improvements for monitoring and reporting

- Although toxoplasmosis is known to be a very common infection this is not well reflected in the available data. The collection of representative data on the disease burden in human and the role of food, including water, in Member States is crucial for any future decision regarding food safety intervention.
- Serological methods should be standardised and harmonised for the monitoring of food producing animals, and feasible methods for the routine detection of *Toxoplasma* in meat, meat products and water should be developed and validated. This requires the production and distribution of suitable reference materials.
- Once such standardised methods are available, *Toxoplasma* monitoring should start on pre-harvest sector in food-producing animals."

7.2 Economic Costs and Burden of Illness

The economic losses caused by congenital toxoplasmosis can be considerable. Estimates for the USA range from US $0.4 – 8.8 billion, and for the UK from US $1.2 – 12 million (Roberts *et al.*, 1994; Roberts and Frenkel, 1990). The ranges are due to uncertainty as to the actual number of infected babies. Medical costs are minor, while the majority of costs derive from the value of statistical lives lost from neonatal death, and income losses by people with long term physical and mental problems resulting from toxoplasmosis in infancy. The differences in cost between the USA and UK are largely due to differences in the way income losses are estimated.

As part of a priority setting exercise for foodborne pathogens in the Netherlands, an estimate of the disease burden in disability adjusted life years (DALYs) has been published for both congenital and post-natally acquired toxoplasmosis (Kemmeren *et al.,* 2006). The DALY estimate is the sum of years of life lost and years lived with disability, weighted for severity of the illness. Disability weights were derived from Dutch studies. Although the long term goal of the project was to estimate foodborne pathogens, for this estimate all illnesses were counted, with no attempt to assign attribution according to source of infection.

For congenital toxoplasmosis the incidence of disease outcomes including stillbirth, a variety of symptoms including chorioretinitis in the first year of life, and chorioretinitis later in life were estimated. The incidence of congenital toxoplasmosis was derived from an estimate of the number of women who were infected during pregnancy and the transmission rate, giving an estimate of 8.2 (90% CI 4.9 – 12.2) per 10,000 live births (although an estimate from a different study gave 23 per 10,000 live births). A variety of studies were then used to estimate the incidence of the specific outcomes above.

For post-natally acquired toxoplasmosis, the incidence was back calculated from the incidence of an outcome: ocular toxoplasmosis (chorioretinitis). Two hundred new cases of ocular toxoplasmosis are reported annually in the Netherlands. Taking the estimate of 0.3% that presented with symptoms in the Vancouver outbreak (Burnett *et al*., 1998), this equates to an estimated 67,000 cases that contract acquired toxoplasmosis each year, an incidence rate of 41 cases per year in the Netherlands.

Taking the estimate of 14% that have mild symptoms, 0.7% severe symptoms and 0.3% with chorioretinitis, Kemmeren *et al.,* then calculated the most likely number of cases and the disease burden for the year 2004. The incidence for mild symptoms was estimated to affect 9300, severe symptoms (excl. chorioretinitis) 470 and chorioretinitis 200.

The cumulative disease burden for the Netherlands was then calculated for congenital and acquired toxoplasmosis. The result was 2400 DALYs (1200 congenital DALYs per year (range 520 – 2700) and 1200 acquired DALYS per year (range 40-2700)). Overall, toxoplasmosis was the illness with the highest disease burden of those estimated, although it also had the greatest uncertainty, resulting from poor information on incidence. At 1200 DALYs, the acquired toxoplasmosis disease burden was similar to that of campylobacteriosis in the Netherlands.

No estimates of the economic cost or burden of illness to New Zealand from toxoplasmosis have been located.

7.3 Other Transmission Routes

7.3.1 *Toxoplasma* in non-livestock animals in New Zealand

Cats: T. gondii uses cats as the definitive host, and a wide range of animals as intermediates, including humans. The infection persists better as tissue cysts in intermediate hosts than in the soil as oocysts (Frenkel, 1990). Most cats in New Zealand have antibodies to *Toxoplasma*, indicating that they have been exposed to the parasite at some time during their lives. The cats most at risk are immuno-suppressed adults or kittens that are seronegative (Thompson, 1999).

Dogs: Reports of *T. gondii* infection in dogs are rare but the true prevalence may be hidden by misdiagnosis. In New Zealand, *T. gondii* was isolated from a dog in 1956 (Hartley, 1956). A retrospective New Zealand study of 15 cases of encephalomyelitis found two cases of *T. gondii* infection. Using light microscopy *Neospora* cysts were indistinguishable from *T. gondii* cysts, electron microscopy confirmed the differences (Patitucci *et al.,* 1997). Generally adult dogs are resistant while puppies are fully susceptible (Hill and Dubey, 2003).

Wallabies: Several outbreaks of toxoplasmosis have been reported in captive wallabies (MAF 1974; 1980; 2000). In 1974, eight of 20 wallabies died over a two month period at Christchurch zoo. In the most recent outbreak in 2000, three caged wallabies developed hind limb paralysis over several months. A post-mortem on one of the animals revealed numerous intact and ruptured cysts typical of *Toxoplasmosis.* No data were found on the prevalence in feral populations of wallabies. Marsupials are apparently very susceptible to the disease, while cattle and horses are among the most resistant hosts (Hill and Dubey, 2003).

7.3.2 Prevalence of *T. gondii* in cats overseas

Surveys of *T. gondii* in cats overseas have shown a prevalence range of 0% to 90%. A summary of a selection of these surveys is below.

In Tehran, Iran, serum samples from 50 stray and 50 household cats revealed 90% of the stray cats were seropositive compared to 36% of the household cats, giving an overall infection rate of 63%. There was a high positive correlation between age and rate of infection (Haddadzadeh *et al.,* 2006).

In Adelaide, South Australia, faeces from 115 stray or unwanted cats were screened for *T. gondii.* None contained detectable levels of viable oocysts. Alongside this study, 60 samples of lamb and pork chops from 15 local butcher shops were also tested. No cysts were detected other than one viable cyst in a pork chop (Rothe *et al*., 1985).

From Illinois, USA, faeces of 274 cats trapped on 47 pigs farms, together with samples of feed and soil were bioassayed in mice for the presence of oocysts (Dubey *et al*., 1995). *T. gondii* was isolated from five faecal samples (1.8%), 2 of the 491 feed samples (0.4%) and one of the 70 soil samples (1.3%). The authors concluded that the possibility that *T. gondii* could be transmitted to pigs via rodents, feed and soil was confirmed.

A range of wild mammals, cats, dogs and humans were tested in Chile, in the Juan Fernandez Archipelgo (Stutzin *et al*., 1989). From the 27 cats tested, 85.2% were seropositive.

7.3.2.1 The development of T. gondii *vaccinations for cats*

There have been a number of attempts at developing a vaccine against *T. gondii* in cats. Frenkel *et al.,* (1991) used a live mutant strain "T-263" in kittens and found that oocyst shedding was reduced by 84%. T-263 consists of live bradyzoites however and is a major disadvantage using this approach. Freyre *et al.,* (1993) further developed the experiments and found that using two oral doses of the T-263 strain induced immunity to oocyst shedding. Field trials of an oral cat vaccine using T-263 have been conducted and concluded that the vaccine was effective (Mateus-Pinilla *et al.,* 1999). A deterministic computer simulation model was then used to assess the effect of a feline vaccine on the transmission of *T. gondii* on a pig farm (Mateus-Pinilla *et al.,* 2002). A surprising result was that the initial *T. gondii* prevalence in cats had no effect on *T. gondii* prevalence in the finishing pigs. The simulation supported the findings from the field trail in that although the vaccine was effective, it had less impact than a decrease in the number of farm cats.

Omata *et al*. (1996) tested ^{60}Co-irradiated tachyzoites vaccine in cats, although only 3 of the 14 cats did not shed oocysts. A DNA vaccine (expressing ROP2 protein) has also been tested but had no effect in reducing oocyst shedding (Mishima *et al*., 2002).

A study only recently published (Garcia *et al*., in press) has evaluated a feline vaccine made from crude rhoptry proteins of *T. gondii* with Quil-A (an adjuvant). Eleven domestic short hair cats (5 – 8 months old) were divided into four groups. Group 1 (*n*=3) received 200µg of rhoptry proteins with 20 µg of Quil-A. Group 2 (*n*=3) received phosphate buffer saline (PBS) with Quil-A (20 µg) while Group 3 (*n*=3) and Group 4 (*n*=2) received only PBS. Group 3 were unvaccinated but challenged while Group 4 were the unvaccinated and unchallenged controls. The doses were administered by the intranasal route over day 0, 21 and 42 days. The total volume of inoculant was 200µL. Groups 1, 2 and 3 were exposed to 600 tissue cysts on day 51.

One of the three cats from the vaccinated group 1 shed oocysts, 4 days after challenge. All animals from groups 2 and 3 shed oocysts while the control group 4 did not shed oocysts throughout the experiment. In comparative terms, group 1 cats shed 89.3% and 90.8% less oocysts then groups 3 and 4 respectively.

7.3.3 Risk communication

The New Zealand Food Safety Authority website has two risk communication publications that give advice on toxoplasmosis;

Food safety in pregnancy:
http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/pregnancy/

Food safety when you have low immunity:
http://www.nzfsa.govt.nz/consumers/food-safety-topics/foodborne-illnesses/low-immunity/lowimmunity.pdf

The US Centres for Disease Control have published a range of material aimed at cat owners, pregnant women, people with HIV/AIDS and for general information:

http://www.cdc.gov/ncidod/dpd/parasites/toxoplasmosis/toxoplasmosis_brochure_8.2004.pdf
http://www.cdc.gov/NCIDOD/dpd/parasites/toxoplasmosis/factsht_toxoplasmosis.htm
http://www.cdc.gov/NCIDOD/dpd/parasites/toxoplasmosis/ToxoWomen.pdf
http://www.cdc.gov/hiv/pubs/brochure/oi_toxo.htm

A study of toxoplasmosis-related knowledge and practices among 403 US pregnant women (Jones *et al.,* 2003b) found knowledge to be low: 48% had seen/heard information about the disease, 7% were aware of being tested, 40% knew it was an infection while 21% believed it was caused by a poison. The highest level of knowledge was for the risk of transmission from cats; 61% responded that the organism is shed in infected cats faeces, 60% knew that changing cat litter was a risk for transmission but only 30% were aware the organism may be found in raw or undercooked meat.

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with red meat and meat products

Meat containing *Toxoplasma* cysts is regarded in Europe and the US as an important source of infection for humans (the organism is not considered to be transmitted from person to person).

Toxoplasmosis does not often cause serious disease in immunocompetent people. However, the risk to immunocompromised people and the foetuses of pregnant women is considerable. In the case of the foetus there is a high likelihood of serious long-term illness caused by transmission of infection. Estimates of economic cost and disease burden for toxoplasmosis have been significant, making the illness prominent amongst diseases which may be foodborne.

The available data on seropositivity of the whole New Zealand population suggest that infection with *Toxoplasma* is as prevalent in New Zealand as other developed countries. Predictions of the incidence of symptomatic illness in New Zealand do not appear to be borne out by actual observations. Studies of seroconversion in pregnant women in New Zealand suggest a higher prevalence of congenital toxoplasmosis than has actually been observed, and the study authors claim that these latter data argue against the need for antenatal screening. In addition, the number of cases of diagnosed acquired toxoplasmosis in New Zealand totalled 84 for the years 2000 to 2006. This is apparently lower than might be predicted from the estimated 200 cases per year of ocular toxoplasmosis (only one of the outcomes of acquired toxoplasmosis) reported for the Netherlands (population 16.5 million) (Kemmeren *et al.*, 2006).

While information on seropositivity in New Zealand livestock is limited, it is likely that New Zealanders domestically produced red meat does contain *Toxoplasma*. Imported red meat is less likely to contribute to exposure given that only small amounts of beef and sheep meat are imported, and pigmeat is required to be frozen. Ameliorating factors for any exposure are that animal seropositivity appears to overestimate infectivity, and *Toxoplasma* exposure will be controlled through cooking and freezing.

Despite the focus on cats as the primary host, ownership of cats as pets has not always been identified as a risk factor in case-control studies of pregnant women. These studies have identified the consumption of undercooked meats as a risk factor, although the most recent study, from England, did not.

The current shortage of information on the prevalence of infection in New Zealand livestock, or the prevalence of contamination in red meat, precludes a definitive risk assessment of this food/hazard combination. As noted by EFSA, considerable method development would be required if efficient monitoring programmes for livestock or food were to be implemented.

Education programmes for vulnerable groups in relation to toxoplasmosis, as recommended by EFSA, are in place in New Zealand.

8.1.2 Risks associated with other foods

In addition to red meat sources, the organism has been found in chicken and pigeons (Smith, 1991) but no outbreaks or other evidence for transmission by these foods have been found.

Other vehicles associated with outbreaks have been unpasteurised goat's milk (Skinner *et al.,* 1990; Sacks *et al*., 1982) ice-cream (linked to contaminated water) (de Moura *et al*., 2006) and municipal water supplies (de Moura *et al*., 2006, Bowie *et al.,* 1997).

Food contaminated with oocysts such as soil grown vegetables are another potential source of infection, particularly where there is a population of cats (Smith, 1993).

8.1.3 Risk Assessment Options

Prevalence data on the presence of *Toxoplasma* cysts in red meat and meat products to estimate the exposure of the New Zealand population are lacking, which precludes a quantitative risk assessment.

8.2 Commentary on Risk Management Options

Decision making regarding risk management options must follow a better assessment of the risk of foodborne transmission of this illness. Risk communication aimed at pregnant women exist.

8.3 Data Gaps

The data gaps identified in this Risk Profile are:

- Prevalence of *Toxoplasma* infection in livestock in New Zealand;
- Prevalence of contamination of red meat and red meat products with *T. gondii* in New Zealand;
- Accurate data concerning toxoplasmosis cases in "at risk" groups;
- Lack of data regarding prevalence of infection in domestic cats (the definitive host) in New Zealand; and,
- Information on awareness by at risk groups in relation to the transmission risks of *T. gondii*, this information would be useful in targeting risk communication messages.

9 REFERENCES

Ades AE, Nokes DJ. (1993) Modeling Age- and Time-specific incidence from seroprevalence: Toxoplasmosis. American Journal of Epidemiology; 137: 1022-1034

AFSSA (2005) Toxoplasmose: état des connaissances et évaluation du risue lié à l'alimentation. Rapport do groupe de travail Toxoplasma gondii de l'Afssa. December 2005. Agence Francaise de Securite Sanitoaire des Aliments. English summary. Available from: http://www.afssa.fr/ftp/afssa/34487-34488.pdf

Allain J-P, Palmer CR, Pearson G. (1998) Epidemiological study of latent and recent infection by *Toxoplasma gondii* in pregnant women from a regional population in the UK. Journal of Infection; 36: 189-196.

Anon. (2006) Import Health Standard for the importation into New Zealand of unprocessed pig meat or pig meat products for human consumption from the United States of America. Ministry of Agriculture and Forestry.

ANZFA. (2001) Raw Commodity Consumption Figures. Canberra: ANZFA.

Aramini JJ, Stephen C, Dubey JP, Engelstoft C, Schwantje H, Ribble CS. (1999) Potential contamination of drinking water with *Toxoplasma gondii* oocysts. Epidemiology and Infection; 122: 305-315.

Arias ML, Chinchilla M, Reyes L, Linder E. (1996) Seroepidemiology of toxoplasmosis in humans: possible transmission routes in Costa Rica. Revista de biología tropical; 44:377-381.

Aspinall TV, Marlee D, Hyde JE, Sims PFG. (2002) Prevalence of *Toxoplasma gondii* in commercial meat products as monitored by polymerase chain reaction – food for thought? Internal Journal of Parasitology; 32: 1193-1199.

Australian Government (2005) Foodborne illness in Australia – Annual incidence circa 2000. Australian Government Department of Health and Ageing. Canberra http://www.ozfoodnet.org.au/internet/ozfoodnet/publishing.nsf/Content/reports-1/$FILE/foodborne_report.pdf

Baghurst K. (1999) Red meat consumption in Australia: intakes, contributions to nutrient intake and associated dietary patterns. European Journal of Cancer Prevention; 8: 185-191.

Baril L, Ancelle T, Goulet V, Thulliez P, Tirard-Fleury V, Carme B. (1999) Risk factors for *Toxoplasma* infection in pregnancy: a case-control study in France. Scandinavian Journal of Infectious Disease; 31: 305-309.

Bénard A, Salmi LR. (2005) Survey on the programs implemented in Europe for the epidemiological surveillance of congenital toxoplasmosis (unpublished report) Bordeaux (France). The EUROTOXO group.

Bobic B, Jevremovic I, Marinkovic J, Sibalic D, Djurkovic-Djakovic O. (1998) Risk factors for *Toxoplasma* infection in a reproductive age female population in the area. European Journal of Epidemiology; 14:605-610.

Bowie WR, King AS, Werker DH, Isaac-Renton JL, Bell A, Eng SB, Marion SA. (1997) Outbreak of toxoplasmosis associated with municipal drinking water. Lancet; 350: 173-177.

BPSU (2005). 19th Annual Report, 2004-2005. British Paediatric Surveillance Unit. Royal College of Paediatrics and Child Health, London. http://www.hpa.org.uk/infections/topics_az/vaccination/bpsu_2005_ann_rep.pdf

Buffolano W, Gilbert RE, Holland FJ, Fratta D, Palumbo F, Ades AE. (1996) Risk factors for recent *Toxoplasma* infection in pregnant women in Naples. Epidemiology and Infection; 116: 347-351.

Burnett AJ, Shortt SG, Isaac-Renton J, King A, Werker D, Bowie WR. (1998) Multiple cases of acquired toxoplasmosis retinitis presenting in an outbreak. Ophthalmology; 105: 1032-1037.

Buzby JC, Roberts T. (1996) ERS updates U.S. foodborne disease costs for seven pathogens. Food Review;19: 20-25.

Cassin MH, Lammerding AM, Paoli GM, McColl RS. (1996) Population response and immunity to *Toxoplasma gondii* [abstract]. In: Proceedings of the Annual Conference of the Society for Risk Analysis and International Society for Exposure Analysis; Dec 8-12; New Orleans, LA. MacLean (VA): Society for Risk Analysis. p. 129.

Choi W-Y, Nam H-W, Kwak N-H, Huh W, Kim Y-R, Kang M-W, Cho S-Y, Dubey JP. (1997) Foodborne outbreaks of human toxoplasmosis. Journal of Infectious Diseases; 175: 1280-1282.

Codex. (1999) Draft principles and guidelines for the conduct of microbiological risk assessment. Report of the thirty first session of the Codex committee on food hygiene. ALINORM 99/13A. Rome: Codex Alimentarius Commission.

Cook AJC, Gilbert RE, Buffolono W, Zufferey J, Jenum PA, Foulon W, Semprini AE, Dunn DT. (2000) Sources of *Toxoplasma* infection in pregnant women: European multicentre case-control study. British Medical Journal; 321: 142-147.

Cursons RTM, Cepulis S, Naicker G, Haslam A. (1982) The prevalence of *Toxoplasma* antibodies among pregnant women in Hamilton. New Zealand Medical Journal; 95: 536-537.

Davies PR, Morrow WEM, Deen J, Gamble HR, Patton S. (1998) Seroprevalence of *Toxoplasma gondii* and *Trichinella spiralis* in finishing swine raised in different production systems in North Carolina, USA. Preventive Veterinary Medicine; 36: 67-76.

Daunter B. (1992) Immunology of pregnancy: towards a unifying hypthesis. European Journal of obstetrics, gynecology and reproductive biology; 43: 81-95.

DEFRA (2006) Zoonoses Report, United Kingdom 2005. Department for Environment, Food and Rural Affairs.

de Moura L, Bahia-Oliveira LMG, Tuboi SH, Carmo EH, Ramalho WM, Carmargo NJ, Trevisan R, Graça RMT, da Silva AJ, Moura I, Dubey JP, Garrett DO. (2006) Waterborne Toxoplasmosis, Brazil, from Field to Gene. Emerging Infectious Diseases;12:326-329

Dias RA, Navarro IT, Ruffolo BB, Bugni FM, Castro MV, Freire RL. (2005) Toxoplasma gondii in fresh pork sausage and seroprevalence in butchers from factories in Londrina, Parana State, Brazil. Revista do Instituto de Medicina Tropical de São Paulo; 47: 185-189.

Diaz-Suarez O, Estevez J, Garcia M, Cheng-Ng R, Araujo J, Garcia M. (2003). Seroepidemiology of toxoplasmosis in a Yucpa Amerindian community of Sierra de Perija, Zulia State, Venezuela. Revista médica de Chile; 131:1003-1010. Article in Spanish.

Dubey JP (1995), Duration of immunity to shedding of *Toxoplasma gondii* oocysts by cats. Journal of Parasitology; 81: 410-415.

Dubey JP (1996a), *Toxoplasma gondii*. Section IV, Parasitology. Medical Microbiology. Eds S.Baron . 4th edition. UTMB.

Dubey JP (1996b), Infectivity and pathogenicity of *Toxoplasma gondii* oocysts for cats. Journal of Parasitology; 82: 957-960.

Dubey JP, (1996c). Strategies to reduce transmission of *Toxoplasma gondii* to animals and humans. Veterinary Parasitology; 64:65-70.

Dubey JP (1998), *Toxoplasma gondii* oocyst survival under defined temperatures. Journal of Parasitology; 84:862-865.

Dubey JP (2000), Sources of *Toxoplasma gondii* infection in pregnancy. British Medical Journal; 321: 127-128.

Dubey JP (2001). Oocyst shedding by cats fed isolated bradyzoites and comparison of infectivity of bradyzoites of the VEG strain *Toxoplasma gondii* to cats and mice. Journal of Parasitology, 87(1):215-219.

Dubey JP (2004). Toxoplasmosis – a waterborne zoonosis. Veterinary Parasitology; 126: 57-72.

Dubey JP, Beattie CP. (1988) Toxoplasmosis of Animals and Man. Boca Raton Florida. CRC Press.

Dubey JP, Thayer DW. (1994) Killing of different strains of *Toxoplasma gondii* tissue cysts by irradiation under defined conditions. Journal of Parasitology; 80: 764-767.

Dubey JP, Kotula AW, Sharar A, Andrews CD, Lindsay DS. (1990) Effect of high temperature on infectivity of *Toxoplasma gondii* tissue cysts in pork. Journal of Parasitology; 76: 201-204.

Dubey JP, Weigel RM, Siegel AM, Thulliez P, Kitron UD, Mitchell MA, Mannelli A, Mateus-Pinilla NE, Shen SK, Kwok OC. (1995) Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. Journal of Parasitology; 81: 723-729.

Dubey JP, Lunney JK, Shen SK, Kwok OCH, Ashford DA, Thulliez P. (1996) Infectivity of low numbers of *Toxoplasma gondii* oocysts to pigs. Journal of Parasitology; 82: 438-443.

Dubey JP, Rollor EA, Smith K, Kwor OCH, Thuilliez P. (1997). Low seroprevalence of *Toxoplasma gondii* in feral pigs from a remote island lacking cats. Journal of Parasitology; 83: 839-841.

Dubey JP, Gamble HR, Hill D, Sreekumar C, Romand S, Thuilliez P. (2002a) High prevalence of viable *Toxoplasma gondii* infection in market weight pigs from a farm in Massachusetts. Journal of Parasitology; 88: 1234-1238.

Dubey JP, Graham DH, Blackston CR, Lehmann T, Gennari SM, Ragozo AM, Nishi SM, Shen SK, Kwok OC, Hill DE, Thulliez P. (2002b) Biological and genetic characterisation of *Toxoplasma gondii* isolates from chickens (*Gallus domesticus*) from São Paulo, Brazil: unexpected findings. Internal Journal of Parasitology;32: 99-105.

Dubey JP, Hill DE, Jones JL, Hightwoer AW, Kirkland E, Roberts JM, Marcet PL, Lehmann T, Vianna MC, Miska K, Sreekumar C, Kwok OC, Shen SK, Gamble HR. (2005) Prevalence of viable *Toxoplasma gondii* in beef, chicken, and pork from retail meat stores in the United States: risk assessment to consumers. Journal of Parasitology; 91: 1082-1093.

Dubey JP, Patitucci AN, Su C, Sundar N, Kwok OCH, Shen SK. (2006) Characterization of *Toxoplasma gondii* isolates in free-range chickens from Chile, South America. Veterinary Parasitology; 140 : 76-82.

Dumètre A, Ajzenberg D, Rozette L, Mercier A, Dardé M. (2006) *Toxoplasma gondii* infection in sheep from Haute-Vienne, France: Seroprevalence and isolate genotyping by microsatellite analysis. Veterinary Parasitology, Article in Press

EFSA. (2005) The Community Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Antimicrobial Resistance in the European Union in 2004. European Food Safety Authority, 21 December 2005.

EFSA. (2007) Review of the Community Summary Report on Trends and Sources of Zoonoses, Zoonotic agents and Antimicrobial Resistance in the European Union in 2005 - Scientific Opinion of the Scientific Panel on Biological Hazards (BIOHAZ) and Animal Health and Welfare (AHAW). The EFSA Journal; 600: 1-32. Available from: http://www.efsa.europa.eu/EFSA/Scientific_Opinion/biohaz_op_ej600_zoonoses_report_2005_en,0.pdf.

el-Nawawy A, Soliman AT, el Azzouni O, Amer el-S, Karim MA, Demian S, el Sayed M. (1996) Maternal and neonatal prevalence of toxoplasma and cytomegalovirus (CMV) antibodies and hepatitis-B antigens in an Egyptian rural area. Journal of Tropical Pediatrics; 42: 154-157.

ESR. (2006) Notifiable and other diseases in New Zealand. Annual Report 2005. Client Report FW 0621 April 2006. Population and Environmental Health Group, Porirua.

Evengård B, Lilja G, Caprau T, Malm G, Kussofsky E, Öman H, Forsgren M. (1999) A retrospective study of seroconversion against *Toxoplasma gondii* during 3,000 pregnancies in Stockholm. Scandinavian Journal of Infectious Diseases; 31: 127-129.

Evengård B, Petersson K, Engman ML, Wiklund S, Ivarsson SA, Tear-Fahnehjelm K, Forsgren M, Gilbert R, Malm G. (2001) Low incidence of *Toxoplasma* infection during pregnant women in Sweden; Epidemiology and Infection; 127: 121-127.

Eymann J, Herbert CA, Coopern DW, Dubey JP. (2006) Serological survey of *Toxoplasma gondii* and *Neospora caninum* in the common brushtail possum (*Trichosurus vulpecula*) from urban Sydney, Australia. Journal of Parasitology; 92: 267-272.

Fairley R. (1996) Infectious agents and parasites of New Zealand pigs transmissable to humans. Surveillance; 23 (1): 17-18.

Fayer R, Gamble HR, Lichtenfels JR, Bier JW. (2001). Waterborne and foodborne parasites. In: F. Pouch-Downes & K. Ito (eds.), Compendium of Methods for the Microbiological Examination of Foods, 4th edition. APHA.

Frenkel JK. (1990) Transmission of toxoplasmosis and the role of immunity in limiting transmission and illness. Journal of the American Veterinary Medical Association; 196:233-240.

Frenkel JK, Dubey JP (1973). Effects of freezing on the viability of *Toxoplasma* oocysts. Journal of Parasitology; 59: 587-588.

Frenkel JK, Pfefferkorn ER, Smith DD, Fishback JL. (1991) Prospective vaccine prepared from a new mutant of *Toxoplasma gondii* for use in cats. American Journal of Veterinary Research; 52: 759-763.

Freyre A, Choromanski L, Fishback JL, Popiel I. (1993) Immunization of cats with tissue cysts, bradyzoites and tachyzoites of the T-263 strain of *Toxoplasma gondii*. Journal of Parasitology; 79: 716-719.

Gajadhar AA, Aramani JJ, Tiffin G, Bisaillon J-R. (1998) Prevalence of *Toxoplasma gondii* in Canadian market-age pigs. Journal of Parasitology; 84: 759-763.

Gamble HR. (1997) Parasites associated with pork and pork products. Scientific and Technical Reviews of the Office International des Epizootes; 16 (2): 496-506.

Gamble HR, Brady RC, Dubey JP. (1999) Prevalence of *Toxoplasma gondii* infection in domestic pigs in the New England states. Veterinary Parasitology; 82:129-136.

Garcia JL, Navarro IT, Biazzono L, Freire RL, da Silva Guimarães Jr. J, Cryssafidis AL, Bugni FM, da Cunha IAL, Hamada FN, Dias RCF. (in press) Protective activity against

oocyst shedding in cats vaccinated with crude rhoptry proteins of the *Toxoplasma gondii* by the intranasal route. Veterinary Parasitology.

Garcia-Vazquez Z, Rosario-Crus R, Diaz-Garcia G, Hernandez-Baumgarten O (1993) Seroprevalence of *Toxoplasma gondii* infection in cattle, swine and goats in four Mexican states. Preventive Veterinary Medicine; 17;12-132.

Gilbert SE, Lake R, Whyte RJ, Bayne G. (2005) Domestic Food Practices in New Zealand; Refrigerator survey and meat handling survey. Unpublished ESR Client Report for NZFSA FW0542, Christchurch..

Gilbert R, Tan HK, Cliffe S, Guy E, Stanford M. (2006). Symptomatic toxoplasma infection due to congenital and postnatally acquired infection. Archives of Disease in Childhood; 91: 495-498.

Goldsmid J, Speare R, Bettiol S. (2003) The parasitology of food. In: Foodborne Microorganisms of Public Health Significance. 6th edition. Eds: AD Hocking. Australian Institute of Food Science and Technology Inc., NSW Branch, Food Microbiology Group; 705 – 721.

Gollub EL, Leroy V, Gilbert R, Chêne G, Wallon M. (2005) Effectiveness of health education approaches for primary prevention of congenital toxoplasmosis (unpublished report). Bordeaux (France): The Eurotoxo Group; October 2005.

Gras L, Gilbert RE, Ades AE, Dunn DT. (2001) Effect of prenatal treatment on the risk of intracranial and ocular lesions in children with congenital toxoplasmosis. International Journal of Epidemiology; 30: 1309-1313.

Gross U. (2004) Prevalence and public-health aspects of toxoplasmosis. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz; 47: 692-697.

Haddadzadeh HR, Khazraiinia P, Aslani M, Rezaeian M, Jamshidi S, Taheri M, Bahonar A. (2006) Seroprevalence of *Toxoplasmosis gondii* infection in stray and household cats in Tehran. Veterinary Parasitology; 138; 211-216.

Hartley WJ. (1956) New Zealand Veterinary Journal; 4: 115.

Hasell S. (1998) Identification and prioritization of knowledge gaps in microbiological monitoring of New Zealand foods. ESR Client Report FW9856. Christchurch: ESR.

Heymann DL. (2004) Control of Communicable Diseases Manual. 18th edition. APHA

Hill DE, Dubey JP. (2003) *Toxoplasma gondii In* Microbial Food Safety in Animal Agriculture – Current Topics. *Eds*: M. E. Torrence and R. E. Isaacson. Iowa State Press.

Hill DE, Sreekumar C, Gamble HR, Dubey JP. (2004) Effect of commonly used enhancement solutions on the viability of *Toxoplasma gondii* tissue cysts in pork loin. Journal of Food Protection; 67: 2230-2233.

Hill DE, Chirukandoth S, Dubey JP. (2005) Biology and epidemiology of *Toxoplasma gondii* in man and animals. Animal Health Research Reviews; 6: 41-61.

Hill DE, Benedetto SMC, Coss C, McCrary JL, Fournet VM, Dubey JP. (2006) Effects of time and temperature on the viability of *Toxoplasma gondii* tissue cysts in enhanced pork loin. Journal of Food Protection; 69: 1961-1965.

Hove T, Lind P, Mukaratirwa S. (2005) Seroprevalence of *Toxoplasma gondii* infection in domestic pigs reared under different management systems in Zimbabwe. Onderstepoort Journal of Veterinary Research; 72: 231-237.

Howe DK, Sibley LD. (1995) *Toxoplasma gondii* comprises three clonal lineages: correlation of parasite genotype with human disease. Journal of Infectious Disease; 172: 1561-1566

Jacobs L, Remington JS, Melton ML. (1960) A survey of meat samples from swine, cattle and sheep for the presence of encysted *Toxoplasma*. Journal of Parasitology; 46: 23-28.

Jacquier P, Hohlfeld P, Vorkauf H, Zuber P. (1995) Epidemiology of toxoplasmosis in Switzerland: national study of seroprevalence monitored in pregnant women 1990-1991. Schweiz. Med Wochenschr. Supplment; 65: 29S-38S.

Jenum PA, Strau-Pedersen B, Melby KK, Kapperud G, Whitelaw A, Eskild A, Eng J. (1998) Incidence of *Toxoplasma gondii* infection in 35,940 pregnant women in Norway and pregnancy outcome for infected women; 36: 2900-2906.

Jones JL, Kruszon-Moran D, Wilson M, McQuillan G, Navin T, McAuley JB. (2001) *Toxoplasma gondii* Infection in th United States, Seroprevalence and Risk Factors. American Journal of Epidemiology; 154: 357-365.

Jones JL, Kruszon-Moran D, Wilson M. (2003a) *Toxoplasma gondii* Infection in the United States, 1999-2000. Emerging Infectious Diseases; 9:1371-1374.

Jones JL, Ogunmodede F, Scheftel J, Kirkland E, Lopez, Schulkin J, Lynfield R. (2003b) Toxoplasmosis-related knowledge and practices among pregnant women in the United States. Infectious Diseases in Obstetrics and Gynecology; 11: 139-145.

Johnson A, Roberts H, Munday BL. (1988) Prevalence of *Toxoplasma gondii* antibody in wild macropods. Australian Veterinary Journal; 65: 199-201.

Joshi YR, Vyas S, Joshi KR. (1998) Seroprevalence of toxoplasmosis in Jodhpur, India. Journal of Communicable Disease; 30: 32-37.

Joynson DH. (1992) Epidemiology of toxoplasmosis in the UK. Scandinavian Journal of Infectious Disease Supplement; 84: 65-69.

Julvez J, Magnaval JF, Meynard D, Perie C, Baixench MT. (1996) Seroepidemiology of toxoplasmosis in Niamey, Niger. (Article in French). Med Trop (Mars); 55: 48-50.

Jumaian NF. (2005) Seroprevalence and risk factors for *Toxoplasma* infection in pregnant women in Jordan. Eastern Mediterranean Health Journal; 11: 45-51.

Kapperud G, Jenum PA, Stray-Pedersen B, Melby KK, Eskild A, Eng J. (1996) Risk factors for *Toxoplasma gondii* infection in Pregnancy. American Journal of Epidemiology; 144: 405-412.

Kemmeren JM, Mangen MJJ, van Duynhoven YTHP, Havelaar AH. (2006) Priority setting of foodborne pathogens – Disease burden and costs of selected enteric pathogens. Report 330080001/2006. Rijskinstituut voor Volksgezondheid en Milieu (RIVM). Available from: http://rivm.openrepository.com/rivm/bitstream/10029/7316/1/330080001.pdf

Klun I, Djurković-Djaković O, Katić-Radivojević, Nikolić A. (2006) Cross-sectional survey on *Toxoplasma gondii* infection in cattle, sheep and pigs in Serbia: Seroprevalence and risk factors. Veterinary Parasitology; 135: 121-131.

Konishi Houki Y, Harano K, Mibawani RS, Marsudi D, Alibasah S, Dachlan YP. (2000) High prevalence of antibody to *Toxoplasma gondii* among humans in Surabaya, Indonesia. Japanese journal of Infectious Disease; 53: 238-241.

Kortbeek LM, de Melker HE, Veldhuijzen IK, Convn-Van Spaendonck MA. (2004) Population-based *Toxoplasma* seroprevalence study in The Netherlands. Epidemiology and Infection; 132: 839-845.

Kotula AW, Dubey JP, Sharar AK, Andrews CD, Shen SK, Lindsay DS. (1991) Effect of freezing on infectivity of *Toxoplasma gondii* tissue cysts in pork. Journal of Food Protection; 54: 687-690.

Kruszon-Moran D, McQuillan GM. (2005) Seroprevalence of six infectious diseases among adults in the United States by race/ethnicity: Data from the third National Health and Nutrition Examination Survey, 1988 – 94. Centers for Disease Control and Prevention; No. 352; March 9 2005.

Lammerding AM, Paoli GM. (1997) Quantitative Risk Assessment: An emerging tool for emerging foodborne pathogens. Emerging Infectious Diseases; 3(4): 483-487.

Lappalainen M, Sintonin H, Koskiniemi M, Hedman K, Hiilesma V, Ammala P, Teramo K, Koskela P. (1995) Cost-benefit analysis of screening for toxoplasmosis during pregnancy. Scandinavian Journal of Infectious Disease; 28: 211-212.

Lebech M, Andersen O, Christensen NC, Werte J, Nielsen WE, Pietersen B, Rechnitzer C, Larsen SO, Nørgaard-Pedersen B, Petersen E. (1999) Feasibility of neonatal screening for *Toxoplasma* infection in the absence of prenatal treatment. Danish Congenital Toxoplasmosis Study Group. Lancet; 353: 1834-1837.

Leroy V, Hadjichristodoulou C. (2005) Systematic review of risk factors for *Toxoplasma gondii* infection in pregnant women. Panel 3: prevention and screening issues. Unpublished report. Bordeaux (France): The Eurotoxo Group;September 2005.

Lindsay DS, Collins MV, Holliman D, Flick GJ, Dubey JP. (2006) Effects of high pressure processing on *Toxoplasma gondii* tissue cysts in ground pork. Journal of Parasitology; 92(1): 195-196.

Logar J, Petrovec M, Novak-Antolic Z, Premru-Srsen T, Cizman M, Arnez M, Kraut A. (2002) Prevention of congenital toxoplasmosis in Slovenia by serological screening of pregnant women. Scandinavian Journal of Infectious Disease; 34: 201-204.

Lopez A, Dietz VJ, Wilson M, Navin TR, Jones JL. (2000) Preventing congenital toxoplasmosis. MMWR Recommendations and Reports; 49 (RR02): 57-75.

Luft BJ, Remington JS. (1992) Toxoplasmic encephalitis in AIDS. Clinical Infectious Disease; 15: 211-222.

Lundén A, Uggla A. (1992) Infectivity of *Toxoplasma gondii* in mutton following curing, smoking, freezing or microwave cooking. International Journal of Food Microbiology; 15: 357-363.

Luyasu V, Robert A, Lissenko D, Bertrand M, Bohy E, Wacquez M, de Bruyere M. (1997). A seroepidemiological study on toxoplasmosis. Acta Clinica Belgica; 52: 3-8.

Machin Sanchez R, Martinez Sanchez R, Fachado Carbajales A, Pividal Grana J, Bravo Gonzalez JR. (1993) The National *Toxoplasma* Survey. I. Prevalence by sex and age. Cuba, 1987. Revista Cubana de Medicina Tropical; 45: 146-151

MAF. (1974) Toxoplasmosis in Wallabies. Surveillance; 2:14

MAF. (1980) Toxoplasmosis in Wallabies. Surveillance; 4:19

MAF. (2000) Quarterly review of diagnostic cases January – March 2000., Surveillance; 27(2): 21

MAF. (2001) Quarterly review of diagnostic cases July – September 2001; Surveillance; 28: 19

MAF. (2002) Quarterly review of diagnostic cases July – September 2002; Surveillance; 29(4): 21

MAF Biosecurity Authority. (2005) Animal Health Surveillance Report – 2004. Surveillance; 32 : 9

MAF. (2007) Situation and Outlook for New Zealand Agriculture and Forestry 2006. http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2006.htm
http://www.maf.govt.nz/mafnet/rural-nz/statistics-and-forecasts/sonzaf/2006/table-stock-numbers.xls

McDonald JC, Gyorkos TW, Alberton B, MacLean JD, Richer G, Juranek D. (1990) An outbreak of toxoplasmosis in pregnant women in Northern Quebec. Journal of Infectious Diseases; 161: 769-774.

Manktelow BW. (1984) The Veterinary Handbook. Palmerston North, New Zealand: Massey University.

Manning JD, Reid JD (1956) Toxoplasmosis in New Zealand. New Zealand Medical Journal; 55: 441-447.

Mason RW, Hartley WJ, Dubey JP. (1991) Lethal toxoplasmosis in a little blue penguin (*Eudyptula minor*) from Tasmania. The Journal of Parasitology, 77:328.

Mateus-Pinilla NE, Dubey JP, Choromanski L, Weigel RM. (1999) A field trial of the effectiveness of a feline *Toxoplasma gondii* vaccine in reducing *T. gondii* exposure for swine. Journal of Parasitology; 85: 855-860.

Mateus-Pinilla NE, Hannon B, Weigel RM. (2002) A computer simulation of the prevention of transmission of *Toxoplasma gondii* on swine farms using a feline *T. gondii* vaccine. Preventive Veterinary Medicine; 55: 17-36.

Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, Griffin PM, Tauxe RV. (1999) Food-related illness and death in the United States. Emerging Infectious Disease; 5: 607-625.

Metcalfe RV, Bettelheim KA, Densham EB, Pearce J, Sillars H, Thorn C. (1981) Studies on antibody levels to *Brucella abortus*, *Toxoplasma gondii* and *Leptospira* serogroups in sera collected in five locations of the North Island of New Zealand. Zentralblatt für Mikrobiologue und Hygiene I Abteilung Originale A; 249: 543-546.

MIA (2006) Annual Report, Meat Industry Association, New Zealand http://www.mia.co.nz/docs/annual_reports/2006%20-%20Final%20MIA%20Annual%20report%202006-MIAAR06(Web).pdf

Ministry of Health/Ministry of Agriculture and Forestry. (2000) Food Administration in New Zealand: A Risk Management Framework for Food Safety. Wellington: Joint Ministry of Health and Ministry of Agriculture and Forestry Food Harmonisation Project.

Mishima M, Xuan X, Yokoyama N, Igarashi I, Fujisaki K, Nagasawa H, Mikami T. (2002) Recombinant feline herpesvirus type 1 expressing *Toxoplasma gondii* ROP2 antigen inducible protective immunity in cats. Parasitology Research; 88: 144-149.

Moese JR, Vander-Moese A. (1998) Mother-child pass in Austria and primary toxoplasmosis infections in pregnant women. Central European Journal of Public Health; 6: 261-264.

Mohan B, Dubey ML, Malla N, Kumar R. (2002) Seroepidemiological study of toxplasmosis in different sections of population of Union Territory of Chandigarh. Journal of Communicable Disease; 34: 15-22.

Moor C, Stone P, Purdie G, Weinstein P. (2000) An investigation into the incidence of toxoplasmosis in pregnancy in New Zealand. New Zealand Medical Journal; 113: 29-32.

Montoya JG, Liesenfeld O. (2004) Toxoplasmosis. The Lancet; 363: 1965-1976.

Morris A, Croxson M. (2004) Serological evidence of *Toxoplasma gondii* infection among pregnant women in Auckland. New Zealand Medical Journal; 117: 770 – 777.

Munday BL. (1975) Prevalence of toxoplasmosis in Tasmanian meat animals. Australian Veterinary Journal; 51: 315-316.

Munoz Batet C, Guardia Llobet C, Juncosa Morros T, Vinas Domenech L, Sierra Soler M, Sanfeliu Sala I, Bosch Mestres J, Dopico Ponte E, Lite Lite J, Matas Andreu L, Juste Sanchez C, Barranco Romeu M. (2004) Toxoplasmosis and pregnancy. Multicenter study of 16,362 pregnant women in Barcelona. Medicina Clinicá (Barcelona); 123: 12-16.

Murrell KD. (1995) Foodborne parasites. International Journal of Environmental Health Research; 5: 63-85.

Nash JQ, Chissel S, Jones J, Warburton F, Verlander NQ. (2005) Risk factors for toxoplasmosis in pregnant women in Kent, United Kingdom. Epidemiology and Infection; 133: 475-483.

New Zealand Pork Industry Board (2001) Profile of the New Zealand pork industry. Wellington: New Zealand Pork.

Nissapatorn V and Abdullah KL. (2004) Review on human toxoplasmosis in Malaysia: the past, present and prospective future. Southeast Asian Journal of Tropical Medicine and Public Health; 35: 24-30

Nowakowska D, Colon I, Remington JS, Grigg M, Golab E, Wilczyski J, Sibley LD. (2006) Genotyping of *Toxoplasma gondii* by multiplex PCR and peptide-based serological testing of samples from infants in Poland diagnosed with congenital toxoplasmosis. Journal of Clinical Microbiology; 44: 1382-1389.

NZHIS (2003) Selected morbidity data for publicly funded hospitals 1 July 1999 to 30 June 2000. New Zealand Health Information Service, Ministry of Health. http://www.nzhis.govt.nz/publications/morbidity99-00.pdf

NZHIS (2004) Selected morbidity data for publicly funded hospitals 1 July 2000 to 30 June 2001. New Zealand Health Information Service, Ministry of Health. http://www.nzhis.govt.nz/publications/morbidity00-01.pdf

NZHIS (2005) Selected morbidity data for publicly funded hospitals 1 July 2001 to 30 June 2002. New Zealand Health Information Service, Ministry of Health. http://www.nzhis.govt.nz/publications/morbidity01-02.pdf

NZHIS (2006) Selected morbidity data for publicly funded hospitals 1 July 2002 to 30 June 2003. New Zealand Health Information Service, Ministry of Health.

http://www.nzhis.govt.nz/publications/morbidity02-03.pdf

O'Donoghue PJ, Riley MJ, Clarke JF. (1987) Serological survey for *Toxoplasma* infections in sheep. Australian Veterinary Journal; 64: 40-45.

Omata Y, Aihara Y, Kanda M, Saito A, Igarashi I, Suzuki N. (1996) *Toxoplasma gondii:* experimental infection in cats vaccinated with ^{60}Co-irradiated tachyzoites. Veterinary Parasitology; 65: 173-183.

Opel U, Charleston WAG, Pomroy WE, Rommel M. (1991) A survey of the prevalence of *Toxoplasma* infection in goats in New Zealand and a comparison of the latex agglutination and indirect fluorescence tests. Veterinary Parasitology; 40: 181-186.

Patitucci AN, Alley MR, Jones BR, Charleston WAG. (1997) Protozoal encephalomyelitis of dogs involving Neospora caninum and Toxoplasma gondii in New Zealand. New Zealand Veterinary Journal; 45: 231-235.

Patton S, Zimmerman J, Roberts T, Faulkner C, Diderrich V, Assadi-Rad A, Davies P, Kliebenstein J. (1997) Seroprevalence of *Toxoplasma gondii* in hogs in the National Animal Health Monitoring System. Journal of Eukaryotic Microbiology; 43: 121S.

Pita Gondim LF, Barbosa HV Jr., Ribeiro Filho CHA, Saeki H. (1999) Serological survey of antibodies to *Toxoplasma gondii* in goats, sheep, cattle and water buffaloes in Bahia State, Brazil. Veterinary Parasitology; 82: 273-276.

Prabhakar P, Bailey A, Smikle MF, McCaw-Binns A, Ashley D. (1991) Seroprevalence of *Toxoplasma gondii*, rubella virus, cytomegalovirus herpes simplex virus (TORCH) and syphilis in Jamaican pregnant women. Western Indian Medical Journal; 40: 166-169.

Rawlins SC, Prabhakar P. (1989) Toxoplasmosis in young Jamaicans. Journal of Tropical Pediatrics; 35: 234-236.

Reichel M, Timbs D, Ross GP, Penrose ME. (1999) Seroprevalence of *Leptospira* and *Toxoplasma* in New Zealand farmed deer. Surveillance; 26: 5-6.

Remington JS, McLeod R, Desmonts G. (1995). Toxoplasmosis. In: Remington & Klein (eds), Infectious diseases of the fetus and newborn infant, W.B. Saunders Co., Philadelphia pp140-267.

Renold C, Sugar A, Chave JP, Perrin L, Delavell J, Pizzolato G, Burkhard P, Gabriel V, Hirschel B. (1992) *Toxoplasma* encephalitis in patients with the acquired immunodeficiency syndrome. Medicine; 71: 224-239.

Roberts T, Frenkel JK. (1990) Estimating income losses and other preventable costs caused by congenital toxoplasmosis in people in the United States. Journal of the American Veterinary Medicine Association; 196: 249-256.

Roberts T, Murrell KD, Marks S. (1994) Economic losses caused by foodborne parasitic disease. Parasitology Today; 10: 419-423.

Robson JMB, Sullivan JJ, Nicolaides NJ, Lewis BR. (1995) A probable foodborne outbreak of toxoplasmosis. Communicable Diseases Intelligence; 19: 517-522.

Rodriguez-Ponce E, Molina JM, Hernandez S. (1995) Seroprevalence of goat toxoplasmosis on Grand Canary Island (Spain). Preventative Veterinary Medicine; 24: 229-234.

Roghmann MC, Faulkner CT, Lefkowitz A, Patton S, Zimmerman J, Morris G jr. *et al.,* (1999) Decreased seroprevalence for *Toxoplasma gondii* in Seventh Day Adventists in Maryland. American Journal of Tropical Medicine and Hygiene; 60: 790-2.

Rothe J, McDonald PJ, Johnson AM. (1985) Detection of *Toxoplasma* cysts and oocysts in an urban environment in a developed country. Pathology; 17: 497-499.

Russell DG, Parnell WR, Wilson NC *et al*. (1999) NZ Food: NZ People. Key results of the 1997 National Nutrition Survey. Wellington: Ministry of Health.

Sacks JJ, Roberto RR, Brooks NF. (1982) Toxoplasmosis infection associated with raw goat's milk. Journal of the American Medical Association; 248: 1728-1732.

Sacks JJ, Delgado DG, Lobel HO, Parker RL. (1983) Toxoplasmosis infection associated with eating undercooked venison. American Journal of Epidemiology; 118: 832-838.

Schwartzman JD. (2001) Toxoplasmosis. Current Infectious Disease Reports; 3: 85-89.

Sfameni SF, Skurrie IJ, Gilbert GL. (1986) Antenatal screening for congenital infection with rubella, cytomegalovirus and toxoplasma. Australian and New Zealand Journal of Obstetrics and Gynaecology; 26: 257-260.

Sharif M, Gholami S, Ziaei H, Daryani A, Laktarashi B, Ziapour SP, Rafiei A, Vahedi V. (2006). Seroprevalence of *Toxoplasmosis gondii* in cattle, sheep and goats slaughtered for food in Mazandaran province, Iran, during 2005. The Veterinary Journal, Article in Press.

Skinner LJ, Timperley AC, Wightman D, Chatterton JM, Ho-Yen DO. (1990) Simultaneous diagnosis of toxoplasmosis in goats and goatowner's family. Scandinavian Journal of Infectious Disease; 22: 359-361.

Smith JL. (1991) Foodborne toxoplasmosis. Journal of Food Safety; 12: 17-57.

Smith JL. (1992) *Toxoplasma gondii* in meats – a matter of concern? Dairy, Food and Environmental Sanitation 12: 341-345.

Smith JL. (1993) Documented outbreaks of Toxoplasmosis: Transmission of *Toxoplasma gondii* to humans. Journal of Food Protection; 56: 630-639.

Smith JL, Wilson M, Hightower AW, Kelley PW, Struewing JP, Juranek DD, McAuley JB. (1996) Prevalence of *Toxoplasma gondii* antibodies in U.S. military recruits in 1989: Comparison with data published in 1965. Clinical Infectious Diseases; 23: 1182-1183.

Smith JL. (1997) Long-term consequences of foodborne toxoplasmosis: Effects on the unborn, the immunocompromised, the elderly, and the immunocompetent. Journal of Food Protection; 60: 1595-1611.

Song CC, Yuan XZ, Shen LY, Gan XX, Ding JZ. (1993) The effect of cobalt-60 irradiation on the infectivity of *Toxoplasma gondii*. International Journal of Parasitology; 23: 89-93.

Sousa OE, Sanez RE, Frenkel JK. (1988) Toxoplasmosis in Panama: a 10 year study. American Journal of Tropical Medicine and Hygiene; 38: 315-322.

Sroka J. (2001) Seroepidemiology of toxoplasmosis in the Lubin region. Annals of Agricultural and Environmental Medicine; 8: 25-31

Stutzin M, Contreras MC, Schenone H. (1989) Epidemiology of toxoplasmosis in Chile V. Prevalence of the infection in humans and domestic and wild animals, studied by indirect hemagglutination reaction, in the Juan Fernandez Archipelago. V Region. Boletín chileno de parasitología; 44: 37-40.

SukthanaY. (2006) Toxoplasmosis: beyond animals to humans, a review. Trends in Parasitology; 22: 137-142.

Sumner J. (2002) Food Safety Risk Profile for Primary Industries in South Australia. Final Report June 2002. http://www.foodsafetysa.com.au/files/pages/SA_PI_Risk_profile.pdf

Tenter AM, Heckeroth AR, Weiss LM. (2000) *Toxoplasma gondii* from animals to humans. International Journal of Parasitology; 30: 1217-1258.

The Press (2006) Seared and Sauced. Section C, The Press Zest, Page 1. Thursday May 11.

Thompson J. (1999) Important infectious diseases of cats in New Zealand. Surveillance; 26(2): 3-5.

Thompson K. (2001) Infectious diseases of goats in New Zealand. Surveillance; 28 (2) :3-7

Thomson BM, Lake RJ. (1995) Heterocyclic amine formation in cooked meat and implication for New Zealanders. ESR Client report for Ministry of Health/Public Health Commission FW95/21. Christchurch: ESR.

Tonkic M, Punda-Polic V, Sardelic S, Capkun V. (2002) Occurrence of *Toxoplasma gondii* antibodies in the population of Split-Dalmatia County. Lijec Vjesn; 124: 19-22

Vermunt JJ, Parkinson TJ. (2000) Infectious diseases of cattle in New Zealand. Part 1 – Calves and growing stock. Surveillance; 27 (2): 3-8.

Walker J, Nokes DJ, Jennings R. (1992) Longitudinal study of *Toxoplasma* seroprevalence in South Yorkshire. Epidemiology and Infection; 108: 99-106.

Walpole IR, Hodgen N, Bower C. (1991) Congenital toxoplasmosis: a large survey in Western Australia. Medical Journal of Australia; 154: 720-724.

Warnekulasuriya MR, Johnson JD, Holliman RE. (1998) Detection of *Toxoplasma gondii* in cured meats. International Journal of Food Microbiology; 45: 211-215.

Zadik PM, Siddons AD. (1995) Low incidence of seroconversion with *Toxoplasma* among women in Sheffield: a seroconversion study. British Journal of Obstetrics and Gynaecology; 102: 608-610.

APPENDIX 1: CATEGORIES FOR RISK PROFILES

The assignment of a category for a food/hazard combination uses two criteria: incidence and severity.

1. Incidence

The incidence is an estimate of the proportion of the foodborne disease rate due to an individual hazard, that is transmitted by a single food or food group.

The overall rate of foodborne disease caused by individual hazards can be derived from information in the published estimate of foodborne disease (Lake *et al*., 2000). This estimate has been updated to reflect more recent notifications rates for the 12 months to June 2001, but still using 1996 census figures (3,681,546 population). Rates include estimates for unreported cases who do not present to a GP.

Disease/organism	Food rate (/100,000 population) Calculated for 12 months to June 2001	Food rate (/100,000 population) Calculated for 12 months to December 1998
Campylobacteriosis	1320	2047
Listeriosis	0.4	0.4
VTEC/STEC	1.9	1.4
Salmonellosis	176	230
Yersiniosis	38	62
Shigellosis	7	7
NLV*	478	478
Toxins*	414	414
Typhoid*	0.3	0.3
Hepatitis A*	0.4	0.4

* not recalculated.

These are **total** foodborne rates, so it is probably safe to assume that in most cases the rates associated with a particular food are likely to be an order of magnitude lower. For instance, a category of ">1000" would only be assigned if it was decided that all campylobacteriosis was due to a single food/food type.

The following categories are proposed for the rates attributable to a single hazard/food (or food group) combination:

Category	Rate range	Comments/examples
1	>100	Significant contributor to foodborne campylobacteriosis Major contributor to foodborne NLV
2	10-100	Major contributor to foodborne salmonellosis Significant contributor to foodborne NLV
3	1-10	Major contributor to foodborne yersiniosis, shigellosis
4	<1	Major contributor to foodborne listeriosis

A further category, of "no evidence for foodborne disease in New Zealand" is desirable, but it was considered more appropriate to make this separate from the others. Also separate is another category, of "no information to determine level of foodborne disease in New Zealand".

The estimation of the proportion of the total foodborne disease rate contributed by a single food or food group will require information from a variety of sources including:

- exposure estimates
- results from epidemiological studies (case control risk factors)
- overseas estimates

For illnesses where the rate is <1 per 100,000 the ability to assign a proportion is unlikely to be sensible. For such illnesses it may be more useful to consider a Risk Profile across the range of all high risk foods, rather than individual foods or food groups.

2. Severity

Severity is related to the probability of severe outcomes from infection with the hazard. The outcomes of infectious intestinal disease are defined in the estimate of the incidence (Lake *et al.*, 2000) as:

- death
- hospitalised and long term illness (GBS, reactive arthritis, HUS)
- hospitalised and recover
- visit a GP but not hospitalised
- do not visit a GP

The first three categories of cases were classed as severe outcomes. Some hospitalisations will result from dehydration, etc. caused by gastrointestinal disease. However, for infections with *Listeria* and STEC hospitalisation will result from more severe illness, even if recovery is achieved. The proportion of severe outcomes resulting from infection with the hazards can be estimated from the proportion of cases hospitalised and recover, hospitalised and long term illness, and deaths (Lake *et al*., 2000).

Disease/organism	**Percentage of outcomes involving death or long term illness from foodborne cases**
Campylobacteriosis	0.3
Listeriosis	60.0
VTEC/STEC	10.4
Salmonellosis	1.0
Yersiniosis	0.4
Shigellosis	2.7
NLV	Assumed to be <0.5%
Hepatitis A	15.4
Typhoid	83.3
Toxins	Assumed to be <0.5%

Categories for the probability of severe outcomes are suggested as follows:

Severity Category	Percentage of cases that experience severe outcomes	Examples
1	>5%	listeriosis, STEC, hepatitis A, typhoid
2	0.5 – 5%	salmonellosis, shigellosis
3	<0.5%	campylobacteriosis, yersiniosis, NLV, toxins

There are a number of hazards for which the incidence of foodborne disease is uncertain. These have been assigned to the above severity categories as follows:

Severity category 1:

Bacteria

Clostridium botulinum

Protozoa

Toxoplasma

Severity category 3:

Bacteria

Aeromonas/Plesiomonas
Arcobacter
E. coli (pathogenic, other than STEC)
Pseudomonas
Streptococcus
Vibrio parahaemolyticus

Viruses

Others (e.g. rotavirus)

Protozoa

Giardia
Cryptosporidium
Cyclospora
Others (e.g. *Entamoeba*)

Proposed Category Matrix

Incidence	>100	10-100	1-10	<1
Severity 1				
Severity 2				
Severity 3				

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand