



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

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by

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

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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

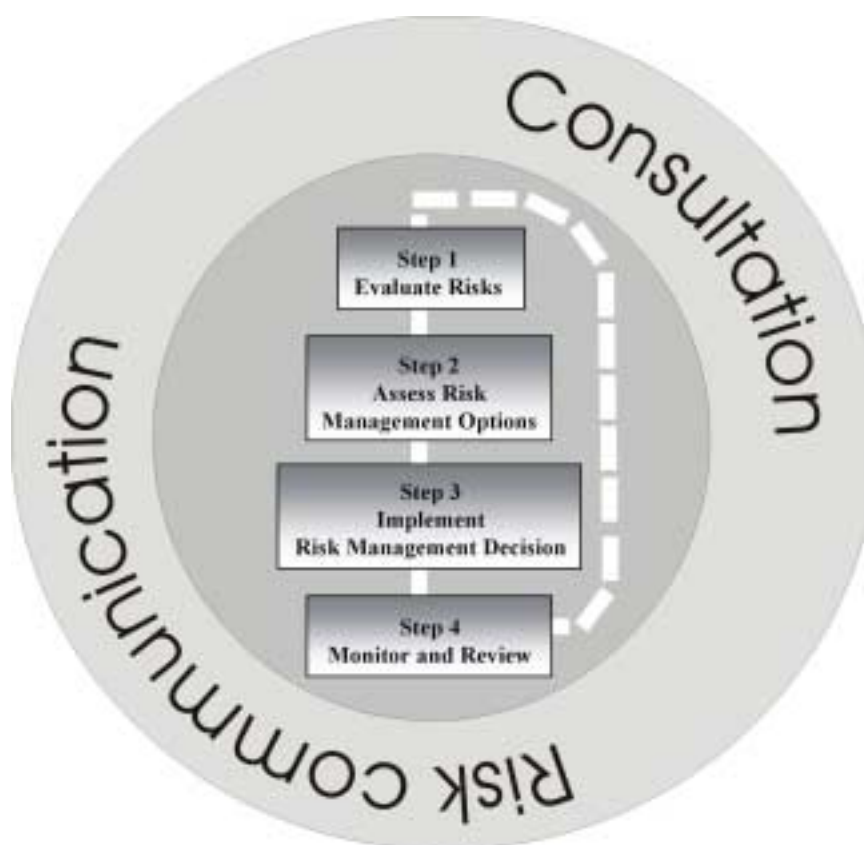


Figure reproduced from “Food Administration in New Zealand. A risk management framework for food safety” (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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 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).

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

Note: Earlier versions of this document were produced as part of a project undertaken by ESR and jointly directed by the Ministry of Health and the Ministry of Agriculture and Forestry. Responsibilities for food safety were combined into the New Zealand Food Safety Authority (NZFSA) in July 2002.

The Australia New Zealand Food Authority (ANZFA) became Food Standards Australia New Zealand (FSANZ), also in July 2002.

Information and reports published by the older organisations have been referenced to those names.

2 HAZARD IDENTIFICATION: THE ORGANISM

The following information is taken from a data sheet prepared by ESR under a contract for the Ministry of Health in 2000-2001. The data sheet is intended for use by regional public health units.

2.1 *Toxoplasma gondii*

2.1.1 The organism

This organism is an obligate intracellular protozoan parasite that is able to infect warm-blooded animals and birds. The results of infection can range from being asymptomatic in healthy adults to miscarriages with death of the foetus.

The organism has a complicated life cycle with numerous stages. Infected cats are the only species to shed oocysts in their faeces which subsequently sporulate in the environment. These can be ingested by humans or animals, and the sporozoites released enter the body. The invasive tachyzoites, which are derived from the sporozoites, then invade body tissues.

Meat animals that have been infected eventually produce cysts (which contain large numbers of bradyzoites) in muscle tissue. If eaten by humans the digestive juices break down the cyst to release the bradyzoites, which transform to tachyzoites and cause infection.

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

2.1.2 Growth and survival

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

Survival: The resting stage, or oocysts, can survive outside of susceptible hosts.

Temperature: Oocysts in faeces or suspended in water retained infectivity for up to 400 days at temperatures ranging from 4 to 37°C. Sporulated oocysts are killed to some extent by freezing at -21°C, but unsporulated oocysts are killed within 1-7 days at this temperature.

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

2.1.3 Inactivation (CCPs and Hurdles)

Temperature: Cysts in pork were killed in 336 seconds at 49°C, 44 seconds at 55°C, 6 seconds at 61°C. Microwave heating to 65°C gave variable results for the inactivation of cysts in mutton.

D times for bradyzoites are reported to be; 53.5 minutes at 49°C, 5.8 minutes at 55°C, 3.8 minutes at 61°C and 3.6 minutes at 67°C.

Cysts present in pork and mutton are inactivated by freezing at temperatures of -9.4°C or lower.

Preservatives: The organism is thought to be susceptible to curing agents used in meat products.

Radiation: Exposure of tachyzoites to 70 J m^{-2} ultraviolet light renders the organism non-infectious. A dose of 1 kGy ionising radiation would ensure that pork is free of the organism.

2.1.4 Sources

Human: Person-to-person spread has not been described, but sub-clinical human infection can be chronic and become activated if the immune system becomes weakened.

Animal: The significance of beef cattle in the epidemiology of toxoplasmosis is unclear and pigs are considered to be a more significant source of infection. Piglets can be killed by toxoplasmosis, but older infected pigs are asymptomatic.

Sheep and goats can be infected by the organism, and the major clinical effect is abortion.

Other animals that can be infected include birds, horses, game animals (e.g. deer), mice and rats, marsupials and dogs. Cats are the only animals known to shed oocysts. Animals such as flies that have come into contact with infected faeces may harbour the organism.

Food: Meat containing *Toxoplasma* cysts may act as a source of human toxoplasmosis. Infected meat and milk from cattle is not considered to be important, but undercooked pig meat is considered to be significant. Sheep and goat meats are also a potential source of infection for humans. Goat milk has also been implicated as a source of disease.

Poultry could be a source of infection if it is insufficiently cooked.

Other meats that may be sources of infection include rabbit, horse, and game (e.g. deer). It has been detected in one of 67 ready-to-eat cured meat samples (ham).

Vegetables, which may be contaminated with soil, are another potential source, particularly from gardens where there may be cats. Unpasteurised goat milk has been implicated in some outbreaks (Smith, 1993).

Environment: It is possible that the consumption of infected water can result in infection, and outbreaks have been attributed to this. The source of contamination is thought to be members of the cat family. Oocysts have been isolated from soils, and soils may be important intermediates in the transmission from cats to humans via buried faeces.

Transmission Routes: Potentially by contact with soil, consumption of contaminated water, contact with cat faeces, or by consumption of contaminated meats (and possibly foods that have been in contact with contaminated soil).

3 HAZARD IDENTIFICATION: THE FOOD

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

Meat containing *Toxoplasma* cysts is regarded as the major source of infection for human toxoplasmosis (the organism is not considered to be transmitted from person to person). 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 meats which have been 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
- pork

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). However, the organism has been found in surveys of cattle tissue, and cross contamination from other meats during processing is a potential source of contamination (Smith 1991; 1992).

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

Although visual or serological testing can be utilised in the field or slaughterhouses to prevent other parasites (*Taenia solium* and *Trichinella spiralis*) from entering the food supply, this is not an option for *Toxoplasma 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.

As indicated in Section 2 above, cooking to an internal temperature of 50°C or above rapidly destroys *Toxoplasma* cysts. An investigation into alternative cooking procedures, using mostly infected mutton as the model has been conducted (Lundén and Ugglå, 1992). The study used the mouse bioassay as the end point, examining brain tissue microscopically and serum for antibodies. After freezing (-20°C for 54 hours), curing with salt and sugar, or smoking (24 hours at less than 50°C following salt injection) all samples were found to be non-infective. However, two of four samples of microwaved steak remained infective although a thermometer inserted into the meat registered 65°C or more. This was ascribed to uneven heating of the meat in a microwave oven.

Another study has shown that freezing at -12°C or below inactivates *Toxoplasma gondii* tissue cysts, although cysts could survive freezing at higher temperatures for several days (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.

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 2001 are shown in Table 1 (MAF, 2001).

Table 1: Livestock numbers for New Zealand in 2001

Main Classes of Livestock (millions) in 2001	
Total sheep	43.99
Total beef	4.98
Total dairy	4.73
Total pigs	0.37
Total deer	2.66

For the year ending September 2001 New Zealand produced 596,300 tonnes of beef and veal meat (1% of world production). Over 80% of this production was exported, representing 10% of the world trade in beef (MAF, 2001).

Approximately 80% of New Zealand’s sheep meat production is exported. The majority is frozen, but chilled meat exports now represent 12% of the total. According to the 2001 Situation and Outlook for New Zealand Agriculture and Forestry (MAF, 2001) total production for the year ending September 2001 was approximately 440,100 tonnes of lamb and 126,800 tonnes of mutton. From this production, 360,600 tonnes of lamb and 100,000 tonnes of mutton were exported.

New Zealand venison production is expected to reach 32,000 tonnes in 2001-2002. Approximately 80% of production is exported to Europe.

New Zealand has a relatively small pig industry, which focuses on the domestic market. Currently about 48,400 breeding sows are farmed, with an estimated 350,700 pigs on farms at any one time (New Zealand Pork Industry Board, 2001). Since 1995 pigmeat production has been relatively static averaging 49,000 tonnes per year (46,500 tonnes in the year to September 2001; MAF, 2001).

3.2.1 Imported food

New Zealand imports relatively small amounts of beef and sheep meat, according to data from Statistics New Zealand. For the year to September 2001 approximately 4454 tonnes of beef carcasses and cuts were imported from Australia, with less than one tonne derived from

the United States. For the same period, 3805 tonnes of sheep meat (all types) was imported, all from Australia.

Larger amounts of pigmeat are imported. For the year to September 2001 New Zealand imported 2859 tonnes of pig meat from Australia, 8746 tonnes from Canada, 782 tonnes from Denmark and 284 tonnes from the United States. All were frozen meat carcasses and cuts.

New Zealand maintains an Import Health Standard for the importation of frozen pork, including a requirement that the pork is frozen to -18 degrees Celsius.

These data, when compared to the production and export figures above, suggest that the approximately 5% of New Zealand's beef and sheep meat for domestic consumption derive from Australia, while approximately 30% of pigmeat for domestic consumption is imported, principally from Australia and Canada.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

When *Toxoplasma gondii* oocysts are ingested by humans or other animals, the sporozoite stage is released from the oocyst in the intestine. The organism then penetrates the intestinal wall and migrates throughout the body where it can invade a variety of tissues. Within these cells the parasite multiplies and forms tissue cysts (Murrell, 1995).

4.1 Symptoms

Incubation: From 3 to around 20 days.

Symptoms: In immunocompetent humans *Toxoplasma gondii* infection is common but clinical toxoplasmosis is rare. Infection produces an asymptomatic illness or, in about 15% of cases, a viral-like febrile illness with lymphadenopathy (swollen lymph nodes) (Mead *et al.*, 1999). This is usually mild and self-limiting and individuals seldom seek medical attention (Smith, 1997).

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 with disease (e.g. AIDS) (Smith, 1997). Congenital toxoplasmosis is the most commonly cited health concern.

Women who are seropositive for *Toxoplasma gondii* prior to pregnancy but who are healthy and immunocompetent do not transmit the parasite to their foetuses. However, approximately 40% of women infected during pregnancy will transfer the infection to the developing foetus.

The probability of this occurring increases with the trimester of pregnancy; 17% in the first, 24% in the second and 62% in the third. Severity of disease is more significant the earlier infection occurs.

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 which should make them more susceptible to toxoplasmosis, there is no evidence that the parasite becomes reactivated in healthy seropositive elderly individuals (Smith, 1997).

In immunocompromised 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. It has been estimated that 30% of AIDS patients who are seropositive will develop toxoplasmic encephalitis.

Condition: Toxoplasmosis.

Toxins: Toxins are not produced in foods.

Long Term Effects: In healthy people infection rarely leads to death. Visual impairment and brain damage may follow infections.

Treatment: Sulfadiazine and pyrimethamine can be used in combination, but not with pregnant women.

4.2 Dose Response

No information has been found regarding the dose response relationship for *Toxoplasma gondii*.

4.3 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.

4.3.1 Pregnant women

Live births data for the 1999 Calendar year (Statistics New Zealand):

Total = 57,423

Births were spread evenly throughout the year, but were strongly weighted towards the northern areas of New Zealand. This total compares well with the results of the 1996 Census, which reported 54,747 New Zealanders under the age of one year on Census night. Of these 51.7% were male and 48.3% female.

4.3.2 Immune compromised

AIDS: At the end of 1998, 669 people in New Zealand were notified with AIDS. At the same date 1336 people in New Zealand were found to be infected with HIV (New Zealand AIDS foundation Annual Report, <http://nzaf.org.nz/org/environmentindex.html>).

Cancer: The most recently available statistics on the incidence of cancer and cancer mortality in New Zealand are from the 1997 year. In the 1997 year, 15,873 new cases of cancer were registered (306.7 cases per 100,000 population), made up of 8,324 males (343.7 cases per 100,000) and 7,549 females (282.3 cases per 100,000). During the same period mortality due to cancer was 7,282 (193.6 cases per 100,000) made up of 3834 males (206.8 per 100,000) and 3448 females (180.7 per 100,000). Source: New Zealand Health Information Service (NZHIS).

Recipients of organ or tissue donations: The NZHIS publication “Selected morbidity data for publicly funded hospitals 1997/98” lists only two patients under the category “V42 Organ or tissue replacement by transplant” and only five patients under the category “V43 Organ or tissue replacement by other means”. A similar document covering private hospital morbidity during 1995 reported 57 corneal transplants, 21 cases of transplantation of muscle and tendon of the hand, but no major organ transplants.

However, this is an obvious underestimate as, presumably, a number of renal, heart and other transplants take place in New Zealand each year.

5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply

5.1.1 Test methodology

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. This indicates previous infection with the organism. However, extracts of tissue from animals can also be used in mouse-based assays to demonstrate infectivity. These tests examine sera from mice for antibodies after gavage or inoculation with the extract.

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 do not accurately assess the risk from improperly cooked pork products.

Direct surveys in foods are difficult, as the methodology requires the concentration of the organism from muscle tissue prior to detection. This latter step has until recently required the use of live animals.

5.1.2 Prevalence/Incidence in animals in New Zealand

A 1996 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 (Fairley, 1996).

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).

A *Toxoplasma* vaccine for sheep has been developed by AgVax at Wallaceville and is registered and in use in New Zealand. The vaccine is used on young sheep as a preventive measure in order to reduce the risk of abortion caused by *Toxoplasma* infection during subsequent pregnancy (Murray Wilkins, AgVax, personal communication). In the 2000 year, 110 abortions due to *Toxoplasma gondii* were diagnosed in sheep in New Zealand (MAF Biosecurity Authority, 2001).

Opel *et al.* (1991) reported on the seropositivity of New Zealand goats and found that 7% of kids, 23% of yearlings and 37% of adults were positive. This increase of positive titres with age was statistically significant, as was a higher frequency of positives in dairy compared to fibre goats.

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

The overall seroprevalence of farmed deer in New Zealand has been determined as 52.5% (using reciprocal titres of ≥ 8 as positive) (Reichel *et al.*, 1999).

Most cats in New Zealand have antibodies to *Toxoplasma*, indicating that they have been exposed to the parasite at some time during their lives (Thompson, 1999).

5.2 Food Consumption: Red Meat and Meat Products

Red meat consumption has declined since 1985, as shown in Table 2. A shift in the composition of this consumption has taken place with major gains by the poultry and pork industries.

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

Year	Sheep 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

From: [New Zealand Meat and Wool Board's Economic Service](#) (MWBES) Annual Review of the Sheep and Beef Industry, 1999-2000.

The meat consumption figures for New Zealand in Table 2 are similar to estimates made for the Australian population (Baghurst, 1999). The Australian consumption levels for 1996-97 were; beef 40.2 kg/person/year, sheep and lamb 17.5 kg/person/year, pigmeat 17.9 kg/person/year, and poultry 10.2 kg/person/year.

An international comparison of meat consumption as calculated for 1998 is given in Table 3.

Table 3: International comparison of meat consumption, 1998 (kg/person/year)

Country	Red Meat consumption	White meat consumption	Total meat consumption
Argentina	64.7	24.4	89.1
Australia	57.9	50.0	107.9
New Zealand	54.1	42.3	96.4
USA	45.2	77.8	123.0
Canada	32.8	64.3	97.1
UK	22.2	50.8	73.0

Source: USDA; MWBES

The figures given above 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). Food Standards Australia New Zealand (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 FSANZ and compares those levels of consumption to the estimates based on meat available for consumption.

Table 4: Mean estimates of New Zealand domestic meat consumption (total population over 15 years), 1997 and estimates of meat available for consumption, 1996 (g/person/day)

Meat type	Estimated consumption (1997)*	Amount available for consumption (1996)#
Beef and veal	87.9	103.6
Sheep and Lamb	13.7	56.4
Pigmeat	32.3	44.1
Deer meat	0.9	
Rabbit meat	0.1	
Total red meat	134.9	204.1
Poultry	35.4	68.8
Total meat	170.3	272.9

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

from Table 1, recalculated from kg/person/year to g/person/day

The difference between these two estimates of consumption will reflect wastage (meat available for consumption, but not consumed), and under-reporting in the NNS. Through use of standard recipes, the FSANZ analysis of the 1997 NNS data will include all meat consumed, including meat which 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, for consumers only, was 172.5 g/day. 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.

5.3 Qualitative Estimate of Exposure

It is not possible to make a qualitative estimate of exposure to *Toxoplasma 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.

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 National Nutrition Survey (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.

Toxoplasma 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 birth rate (for New Zealand; 57,423 in 1999 as a percentage of the 1996 total population gives a birth rate of 1.59%, compared to the US rate of 1.5%)(USDA/HHS, 2001).

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 *Toxoplasma gondii*.

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 *Toxoplasma 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.

Toxoplasma gondii is destroyed by the freezing process. 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 can be assumed that all of these servings would have undergone a sufficiently rigorous freezing regime to destroy the organism.

5.3.5 Heat treatment

Toxoplasma gondii is relatively susceptible to heat inactivation and consumption of 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.

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 incidence of antibodies for *Toxoplasma* in animals derived from serological tests has been summarised in Table 5. This information is drawn from recent reviews of *Toxoplasma* in animals and foods.

Table 5: Reported prevalence of *Toxoplasma 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 <i>et al.</i> , 1987
Australia	151	Tasmanian pademelons	3.3	Johnson <i>et al.</i> , 1988
	85	Bennett's Wallabies	17.7	Johnson <i>et al.</i> , 1988
Canada	2,800	Pigs	8.6	Gajadhar <i>et al.</i> , 1998
Norway	NS	Sheep	18.0	Kapperud <i>et al.</i> , 1996
	NS	Cattle	5.1	Kapperud <i>et al.</i> , 1996
	NS	Pigs	2.5	Kapperud <i>et al.</i> , 1996
USA	3,707	Pigs	32 (range <1 to 69)	Smith, 1991
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
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

5.4.2 Prevalence in animal tissue

Data reported in reviews of analyses for *Toxoplasma gondii* in animal muscle tissue are summarised in Table 6.

Table 6: Reported prevalence of *Toxoplasma gondii* cysts in overseas animal tissue

Country	Number tested	Meat tested	Percentage positive	Assay Method	Reference
Canada	2,800	Pig heart and diaphragm	0	Mouse bioassay	Gajadhar <i>et al.</i> , 1998
England	67	Ready-to-eat cured meats	1.5	PCR/Tissue culture	Warnekulasuriya <i>et al.</i> , 1998
USA	50	Pig diaphragm	24	Mouse bioassay	Jacobs <i>et al.</i> , 1960
	60	Beef cattle diaphragm	1.7	Mouse bioassay	Jacobs <i>et al.</i> , 1960
	86	Sheep diaphragm	9.3	Mouse bioassay	Jacobs <i>et al.</i> , 1960
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

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 antibodies rises early in infection. Seroconversion in pregnant women refers to cases where IgG and IgM antibodies 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

Toxoplasma infection is often asymptomatic and is not diagnosed. 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. However, only one case of congenital toxoplasmosis was notified during this period. This had been ascribed to under-reporting based on laboratory testing data showing nine diagnoses in 1988 alone (Moor *et al.*, 2000).

Metcalf *et al.* (1981) reported on IgG antibody 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 pregnant women in Hamilton (Cursons *et al.*, 1981) reported the results shown in Table 7.

Table 7: Reported prevalence of *Toxoplasma gondii* antibodies in pregnant women in Hamilton

Age Group	Positive for antibody (%)	Seroconversion (% per annum)
15-20	58.5	-
21-25	57.1	-
26-30	60.0	0.6
31-35	68.5	1.7
>35	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 fetuses 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.

Toxoplasmic encephalitis could also be a problem for AIDS patients in New Zealand. Although opportunistic infections account for the majority of deaths of AIDS patients in New Zealand (as in other countries), information on the specific infection is not routinely collected (Jason Eberhardt-Phillips, AIDS Epidemiology Group, Otago Medical School, personal communication).

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Worldwide cases of congenital toxoplasmosis are 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). It has been estimated that approximately 30% of adults in the United Kingdom have antibodies against *Toxoplasma gondii*, and estimates for adults in Europe are 50 to 80% (Smith, 1997).

In the USA 23% of adolescents and adults have laboratory evidence of infection with *Toxoplasma gondii* (Lopez *et al.*, 2000). It has been estimated that 0.6% of the US population experiences an acute infection each year, representing approximately 1,500,000 infections per year (Mead *et al.*, 1999). The number of cases of congenital toxoplasmosis in the USA has been estimated as between 400-4,000 annually (Lopez *et al.*, 2000).

In the USA toxoplasmosis is a leading cause of foodborne disease resulting in death. This is largely due to infections in AIDS patients. Toxoplasmic encephalitis occurs in approximately 7% of AIDS patients in the USA, although the rate declined from 1992 to 1997 (Schwartzman, 2001).

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).

Data for seroconversion during pregnancy from a number of studies are summarised in Table 8.

Table 8: Seroconversion rates for *Toxoplasma gondii* infection

Country	Number tested	Seroconversion (%)	Reference
Denmark	NS	0.21	Evengård <i>et al.</i> , 1999
England	1621	0.023	Zadik and Siddons, 1995
England	13328	3-16 infected foetuses per 100,000 pregnancies	Allain <i>et al.</i> , 1998
Finland	NS	0.24	Evengård <i>et al.</i> , 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 <i>et al.</i> , 1999
Sweden	3094	0.13	Evengård <i>et al.</i> , 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

Serological data for humans are summarised in Table 9.

Table 9: Reported prevalence of antibodies to *Toxoplasma gondii* in various populations

Country	Number tested	Who tested	Seropositive (%)	Reference
China	3,085	Public	0.7	Smith, 1991
France	7,605	Pregnant women	36.5	Smith, 1991
Japan	3,606	Public	Age 20-29 2.9 Age 70-90 40 Males 29 Women 16	Smith, 1991
Norway	35,940	Pregnant women	10.9	Jenum <i>et al.</i> , 1998
Norway	196	Women	56.6	Smith, 1991
Norway	3,412	Military Recruits	21.8	Smith, 1991
Panama	916	Public	Rural 57.5 Urban 58.6	Smith, 1991
USA	2,862	Military Recruits	9.9	Smith <i>et al.</i> , 1996
USA	369	Public	19 male 19 female	Smith, 1991
NS	95,929	Pregnant women	8.1	Smith, 1991

NB: In all studies the seropositivity increases with age as the exposure increases.

NS = Not Stated

6.2.2 Contribution to outbreaks and incidents

Reported outbreaks of foodborne toxoplasmosis are summarised in Table 10.

Table 10: Summary of information on outbreaks of infection with *Toxoplasma gondii*

Country	Number of cases	Implicated Meat	Reference
Australia	5	Raw lamb	Smith, 1993
Australia	12	Undercooked kangaroo	Robson <i>et al.</i> , 1995
Brazil	95	Rare meat (hamburger?)	Smith, 1993
Canada	4	Raw seal and/or caribou	McDonald <i>et al.</i> , 1990
England	3	Raw lamb	Smith, 1993
England	1	Raw or rare steak	Smith, 1993
Korea	3	Raw pig liver and spleen	Choi <i>et al.</i> , 1997
Korea	5	Raw pig liver	Choi <i>et al.</i> , 1997
USA	2	Rare meat	Smith, 1993
USA	1	Rare meat	Smith, 1993
USA	3	Rare/raw venison	Smith, 1993
USA	6	Rare lamb	Smith, 1993
USA	4	Raw beef	Smith, 1993
USA	5	Rare meat (hamburger)	Smith, 1993

An outbreak of toxoplasmosis associated with a municipal drinking water facility has been reported in Canada (Bowie *et al.*, 1997). The water supply was unfiltered, and although disinfection using chloramination was used, this method is unproven against *Toxoplasma gondii*.

Three case control studies have been identified.

A study in Naples (Buffolano *et al.*, 1996) was conducted with postnatal women who were interviewed with regard to risk factors and tested for their levels of serum anti-*Toxoplasma* IgG and IgM. The levels of these were used to determine the time of infection (IgM+, IgG+ recently infected, IgG - susceptible and IgM-, IgG+ as previously infected). Of the women tested 1.2% were recently infected, 39% were previously infected and 60% were susceptible. IgM positivity was associated with;

- eating cured pork OR 2.9 (95% CI 1.6-5.5)
- eating raw meat OR 2.6 (95% CI 1.4-4.7)
- gardening OR 2.0 (95% CI 1.1-3.7)

There was also a dose/dependent response between frequency of consumption of cured pork and raw meat and the odds ratio.

A study using similar criteria for defining cases was carried out in Norway from 1992 to 1994 (Kapperud *et al.*, 1996). The following independent risk factors were identified;

- 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 multicentre study in Europe again used conversion to define cases (Cook *et al.*, 2000). The following factors were the most strongly predictive of acute infection in pregnant women;

- eating raw/undercooked beef OR 1.73 (95% CI 1.1-7.2)
- eating raw/undercooked lamb OR 3.13 (95% CI 1.4-7.2)
- eating “other” meat OR 4.12 (95% CI 1.6-10.9)
- contact with soil OR 1.81 (95% CI 1.2-2.7)
- travel outside of Europe/USA or Canada OR 2.33 (95% CI 1.3-4.1)

Between 30 and 63% of the infections could be attributed to meat consumption (including cured meats), and 6 to 17% to contact with soil.

6.3 Qualitative Estimate of Risk

The multicentre case control study from Europe found that between 30 and 63% of *Toxoplasma 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 *Toxoplasma gondii* infections in the USA (Mead *et al.*, 1999).

Data on the prevalence of infection of animals in New Zealand are scanty, 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 infective cysts in meat for retail sale, would be required for a proper risk assessment.

Nevertheless it is clear that a proportion of the meat animals in New Zealand have been infected with *Toxoplasma*. Raising pigs outdoors is common in New Zealand, particularly in the South Island (New Zealand Pork Industry Board, 2001), so it seems reasonable to expect that there will be a proportion of these animals that are seropositive.

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 very high, and so this infection has been assigned the highest severity category. However, 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.

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
<i>Toxoplasma gondii</i> in red meat and meat products	1 (>5% serious outcomes for congenital toxoplasmosis)	No information		

7 RISK MANAGEMENT INFORMATION

Prevention of infection with *Toxoplasma 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 food safety 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 for beef, lamb and veal roasts and steaks, 160°F for pork, ground meat and wild game, 180°F for poultry);
- 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.

The other recommendations for preventing congenital toxoplasmosis concentrated on the education of pregnant women, avoiding exposure to cat faeces and reducing the likelihood of cat infection (Lopez *et al.*, 2000).

Some countries have instituted screening programmes of pregnant women so that treatment can be given if necessary (reviewed in Lopez *et al.*, 2000). The study of congenital toxoplasmosis in New Zealand concluded that more information was required before screening could be recommended here (Moor *et al.*, 2000). The study stated that advising women on means of avoiding exposure is as important as offering screening testing.

Screening of newborn children is another option. This would permit treatment to reduce the long-term effects of congenital toxoplasmosis. This approach appears to have promise and is being further explored in the USA (Lopez *et al.*, 2000).

Education programmes for pregnant women in Canada and Belgium have been evaluated and found to have inconclusive results (Lopez *et al.*, 2000). However, participants in the US workshop commented that education programmes were a potentially powerful intervention because of their low cost and because pregnant women were highly motivated to protect the health of their babies. Further research into the design and effectiveness of education programmes was recommended.

7.1 Economic Costs

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.

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

8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risks associated with red meat and meat products

Toxoplasmosis does not cause serious disease in immunocompetent people. However, the risk for pregnant women is considerable, given the high likelihood of serious long-term illness caused by transmission of infection to the foetus. The available data on seroconversion of pregnant women in New Zealand suggest that there may be approximately 66 babies born with congenital toxoplasmosis each year. However this estimate is not matched by cases of congenital toxoplasmosis reported to the hospital system, and even when congenital toxoplasmosis was a notifiable disease reported cases were few.

It is likely that New Zealanders are exposed to *Toxoplasma* via domestically produced red meat, given that seropositivity amongst farmed animals is widespread. 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 seropositivity appears to overestimate infectivity, and *Toxoplasma* exposure will be controlled through cooking and freezing.

It appears from overseas studies that foodborne transmission may be involved in a significant proportion of cases of congenital toxoplasmosis. However, the relative importance of food, compared with other transmission routes (cats, soil) in New Zealand is unknown.

8.1.2 Risks associated with other foods

Given that *Toxoplasma* can only grow in a live host, then animal foods are the only likely source. 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.

8.1.3 Quantitative risk assessment

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

Better data on the prevalence of *Toxoplasma* infection in animals, and investigation of the prevalence of cysts in red meat would improve the risk assessment for this food/hazard combination. However, at this stage it appears to be more important to clarify the actual incidence of congenital toxoplasmosis in New Zealand. The authors of the New Zealand study suggested improved surveillance by official notification of cases via a direct link between clinical laboratories and the ESR (Moor *et al.*, 2000). Once a means of identifying cases is established, the circumstances surrounding the infections can be investigated. The relative importance of the possible transmission routes needs to be investigated, to determine

the best risk management option; whether to institute controls on the food supply, or increase activity in screening and education.

The seroconversion study conducted in Wellington could also be extended to cover the whole country.

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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