RISK PROFILE:
YERSINIA ENTEROCOLITICA IN PORK

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by

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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, ranking of a particular food safety issue.

This Risk Profile concerns *Yersinia enterocolitica* in pork, as pigs are the only animals consumed by people that regularly harbour pathogenic serotypes of this organism. Rates of yersiniosis are relatively high in New Zealand compared to Australia and other countries. Pork has been implicated in a proportion of cases, through outbreak investigations and through a case-control study. However, it should be noted that case-control studies have identified other more important risk factors for yersiniosis, such as unreticulated sewage, water from a home supply and handling farm animals. In addition the number of yersiniosis outbreaks is small and so the information from them is not strong.

There are two pork related food consumption patterns that emerge from the literature as important in yersiniosis. Firstly there is the consumption of raw pork in Belgium, and the fact that raw pork is used to feed young children in that country (Tauxe *et al.,* 1987). Secondly there is the association between chitterlings preparation and yersiniosis in African American children in the USA. Neither seems likely to be important in New Zealand.

Effective cooking and pasteurisation will eliminate *Y. enterocolitica* from foods. However there is still the potential for cross contamination from uncooked foods (especially meats) to other foods which are then not cooked before consumption. The identification of food from a sandwich bar as a statistically significant exposure in the case control study suggests that this might be a route for transmission of yersiniosis.

There are indications from outbreaks and the case control study that pork is involved in the transmission of a proportion of the yersiniosis cases in New Zealand, and this proportion is apparently small. This risk assessment would be considerably strengthened by information on the prevalence of pathogenic *Y. enterocolitica* in pigs at slaughter and in retail pork products. However, there are some methodological hurdles to be overcome before survey work can be undertaken.

Some data gaps exist that could aid the assessment or risks:

- Prevalence and numbers of pathogenic *Y. enterocolitica* contamination on pig carcasses after slaughter;
- Prevalence and numbers of pathogenic *Y. enterocolitica* contamination on retail pork products;
- Equivalent prevalence and enumeration information on pathogenic *Y. enterocolitica* in other farmed animal species, to provide context to pig/pork data;
- Dose response data.

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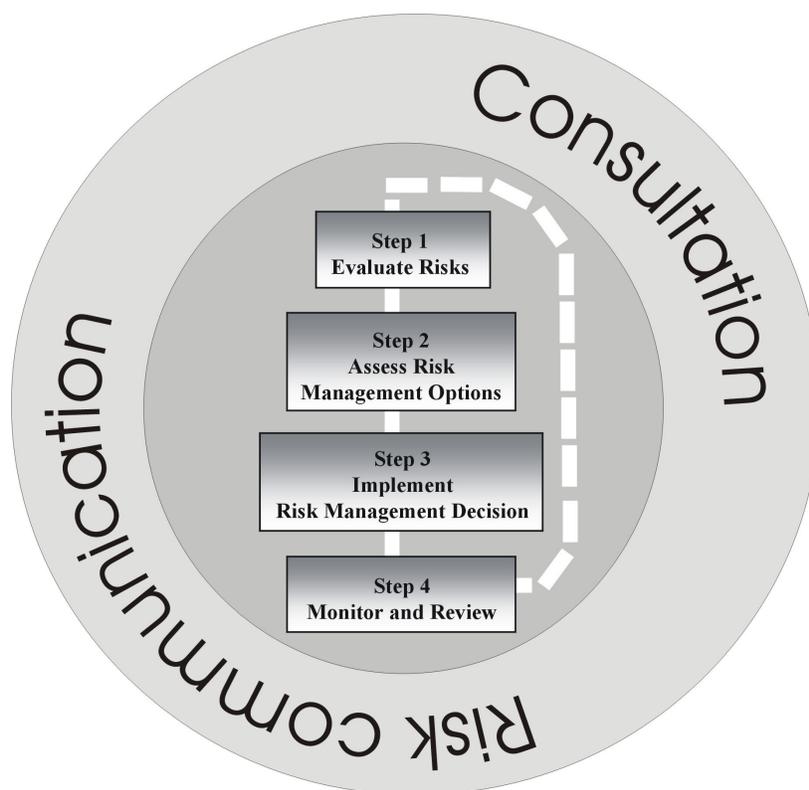
Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

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.

The 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.

This Risk Profile concerns *Yersinia enterocolitica* in pork. The risk for transmission of this bacterium in pork is based on the fact that pigs are the only animals consumed by people which regularly harbour pathogenic serotypes (Kapperud, 1991).

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 following information is taken from data sheets prepared by ESR under a contract for the Ministry of Health. The data sheets are intended for use by regional public health units.

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

2.1 *Yersinia enterocolitica*

2.1.1 <u>The organism/toxin</u>

Yersinia enterocolitica is one of the three species of *Yersinia* considered to be pathogenic to humans and animals. The others are *Yersinia pseudotuberculosis* which causes inflammation of the lymph nodes, and *Yersinia pestis*, which was responsible for the bubonic plague. The latter two species are not associated with foodborne transmission.

A number of other non-pathogenic *Yersinia* species can be recovered from foods, and are readily distinguished from *Y. enterocolitica* on the basis of biochemical tests.

The organism can grow in the presence or absence of oxygen. It is able to grow at refrigeration temperatures, however, only a proportion of isolates are pathogenic. It is not regarded as a good competitor with other bacteria.

2.1.2 <u>Growth and survival</u>

Growth:

<u>Temperature</u>: Range 0-44°C, optimum 28-29°C

<u>pH</u>: The minimum pH for growth is in the range 4.1-5.1 depending on the temperature and the acidulant (Adams *et al*., 1991). Maximum pH around 10.0, optimum 7.2-7.4.

<u>Atmosphere</u>: Growth is retarded under vacuum packaging, 100% N_2, and CO_2/N_2 gas mixes, but the effect is more pronounced at refrigeration temperatures. For example growth on beef mince under 20% CO_2:80% N_2 was much the same as under air at 15°C, but was completely inhibited at 1°C.

<u>Water activity</u>: Can grow in up to 5% NaCl (a_w=0.945).

Survival:

<u>Temperature</u>: Survived (numbers increased and then declined) for 64 weeks in spring water stored at 4°C. Readily withstands freezing e.g. storage in milk for 30 days at –20°C had a negligible effect on survival.

<u>pH:</u> At a given pH below that allowing growth, survival is greater at lower temperatures (Little *et al*., 1992).

Water Activity: Survived well in soil maintained at its original water content, but reduced in number significantly when soil was allowed to air dry.

2.1.3   Inactivation (CCPs and Hurdles)

Temperature: D time at $55^{o}C$ = approx. 2 min, D time at $60^{o}C$ = approx. 0.5 min, D time at $65^{o}C$ = approx. 2 sec. Pasteurisation is an effective heat treatment.

pH: At any given pH lower than that allowing growth the bactericidal activity of different acidulants was in the order acetic acid >lactic acid > citric acid > sulphuric acid. However, at $4^{o}C$ D values are generally measured in days.

Water activity: 5-7% NaCl inhibits growth.

Preservatives: Growth is retarded by potassium sorbate up to 5000 ppm at pH 6.5 in a dose-dependent manner. At pH 5.5 concentrations above 1000 ppm virtually eliminate growth or cause inactivation depending on dose.

Sodium nitrite at a concentration of 150 ppm retarded growth on bologna. Sodium nitrate was less inhibitory than sodium nitrite and potassium nitrate when tested in pork mince.

Inhibited by some spice extracts including cloves, allspice, sage, cinnamon, rosemary and oregano when present at 4.1-4.7%.

Lactate and ALTA 2341 (shelf life extender) lengthened lag times in poultry but effectiveness decreased as temperature increased.

Radiation: D values (kGy) approx. 0.1-0.2 at $25^{o}C$, 0.4 at $-30^{o}C$. More sensitive to ultraviolet radiation than *E. coli* and commercial UV water treatment units producing 30 mWs/cm^2 are considered adequate.

2.1.4   Sources

Human: Person-to-person transmission can occur. Transmission within hospitals has been documented.

Animal: Mostly associated with pigs, especially the tongue and tonsil area as well as the intestines and faeces (Nesbakken *et al*., 2003). Serotype O:3 is common in pigs in New Zealand (Wright *et al.*, 1995). May also be carried by companion animals. Has been isolated from rats and insects. A New Zealand study failed to detect the organism in 100 ovine and 100 bovine carcass swabs. *Yersinia* is a significant pathogen of deer, but in New Zealand the species responsible is *Y. pseudotuberculosis* (Gill, 1996).

Transmission from animals to humans is suspected.

Food: Associated with pork and pork products. Has been isolated from dairy products, fruit, vegetables, tofu, pastries and sandwiches. A New Zealand study detected *Y. enterocolitica* in 3.4% of 203 foods tested, but these included non-pathogenic types (Hudson *et al.*, 1992). The proportion of cases that are foodborne have been estimated at between 65 and 90%.

Environment: Waterborne transmission has resulted in disease, sometimes via contaminated food. Can be isolated from drinking and surface waters, as well as sewage sludge.

Transmission Routes: Ingestion of contaminated food is the primary route.

2.2 Serotypes Causing Disease

It is now well recognised that there are two major subclasses of *Y. enterocolitica*, environmental strains and pathogenic strains. A major distinction is that pathogenic strains require the presence of a plasmid, which encodes for some of the virulence determinants. A number of phenotypic methods exist which indicate the presence of the plasmid (e.g. Congo Red binding) and schemes have been developed that use the serotype and biotypes of isolates to assign pathogenicity.

The diversity of *Y. enterocolitica* strains has allowed the establishment of several biogroups, based on the behaviour of the strains in biochemical tests (Bottone, 1997). Virulence is better summarised in terms of these biotypes, with biotypes 1B (serotypes O:8, O:4, O13a,13b, O18, O:20 O:21), 2 (O:9, O:5,27), 3 (O:1,2,3, O:5,27), 4 (O:3) and 5 (O:2,3) recognised as being virulent (Bottone, 1997).

In the United States and Europe around 90% of yersiniosis cases are caused by serogroup O:3 (Thisted-Lambertz *et al.*, 1996; Weynants *et al.*, 1996). In Europe the second most frequent pathogenic serovar is O:9. In New Zealand biotype 4 (serotype O:3) isolates account for over 90% of cases of yersiniosis (Fenwick and McCarthy, 1995; Wright *et al.*, 1995).

Genetic typing has shown an association between biotype 4 isolates from clinical sources and isolates from animals (pigs) (Dolina and Peduzzi, 1993).

2.3 Methodological Problems

Isolation of *Y. enterocolitica* from foods is arguably one of the least satisfactory tests that exist for foodborne pathogens. Once isolated, a presumptive *Y. enterocolitica* colony must be confirmed as belonging to a pathogenic biotype and serotype (Barton *et al.*, 1997). The total time from sample receipt to confirmation of a pathogenic *Y. enterocolitica* is therefore very long, making the test of little utility in outbreak investigation. In addition, some methods are selective against some serotypes of *Y. enterocolitica*. For example, De Zutter *et al.* (1994) found that irgasan ticarcillin chlorate broth (ITC) behaved poorly in the enrichment of serotype O:9 from pork. It is therefore difficult to assemble any enrichment/plating system that is suitable for all serotypes. These difficulties mean that there is a paucity of reliable data regarding the prevalence of this organism on foods.

Work has been carried out in New Zealand on methods for the detection of *Y. enterocolitica* in foods. A PCR method has been developed which is capable of detecting the presence of pathogenic *Y. enterocolitica* in food enrichments. However, as yet, no plating medium has been found to be sufficiently discriminatory or sensitive to allow the recovery of these organisms by culture. This may be due, in whole or in part, to similar difficulties with the enrichment medium.

3   HAZARD IDENTIFICATION: THE FOOD

3.1   Relevant Characteristics of the Food: Pork

Meat has a high water and protein content, and contains only a low amount of fermentable carbohydrates. Essentially it is an excellent medium for microbial growth, although the pH of good quality meat (5.4-5.5, 6 in some muscles) is lower than the optimum growth pH for most spoilage and pathogenic micro-organisms.

It is well established that *Y. enterocolitica* is able to grow on a range of pork products, even at refrigeration temperatures. Shenoy and Murano (1996) demonstrated growth of heat injured cells in minced pork at 4 and 25°C when samples were stored under air, vacuum or modified atmosphere (50% CO_2:50% N_2). The different storage atmospheres had no effect on the growth of the organism. However, there is evidence that serotype O:3 may be outcompeted by competing non-pathogenic microbiota (Fukushima and Gomyoda, 1986) at 6 and 25°C. The organism was able to survive in these experiments. This has also been shown for serotype O:8 (Schiemann and Olson, 1984). In the Shenoy and Murano paper (1996) the pork samples were heat treated to injure the *Y. enterocolitica* present, and this will have also reduced the competing microbiota. Growth of *Y. enterocolitica* in pork is therefore likely to depend on some function of the ratio of pathogen to competing non-pathogenic organisms and the storage temperature (*Y. enterocolitica* may have a competitive advantage at lower temperatures).

Y. enterocolitica was unable to grow, but could survive in raw sausage type materials (pork spread, knackwurst, and cervelat made without the addition of starter cultures) when the meats were inoculated (10^5cfu/g) prior to stuffing, smoking and cold storage (3-5°C) or curing (13-16°C).   Surviving bacteria could be detected for up to 30 days under cold storage, and 15 days under curing conditions. Kinetics of inactivation were approximately log-linear with rapid die offs at the end of the period of survival (Kleemann and Bergann, 1996). The authors recommended that manufacturers adhere to stringent hygiene rules of Good Manufacturing Practice.

Growth is likely in cooked foods (since the competing non-pathogenic microbiota will be reduced) stored under refrigeration (which may select for the pathogen). Hanna *et al.* (1977) observed that, at 25°C, growth on cooked pork was somewhat faster than on raw pork. This difference in growth rates could be attributed to the level of competing non-pathogenic microbiota present. Growth also occurred at 7°C on raw pork.

In processed meats, potassium nitrate, sodium nitrate and sodium nitrite are all bactericidal towards *Y. enterocolitica* when added to pork mince (de Giusti and de Vito, 1992). No organisms survived after 48h exposure of 10^4 cfu/g to 150 ppm KNO_3, 150 ppm $NaNO_3$ and 100 ppm $NaNO_2$.

3.2   The Food Supply in New Zealand

The New Zealand pig industry is relatively small and focussed on the domestic market. A total of 708,000 pigs were slaughtered during the year to September 2002, an increase of 2% on the previous year, producing 45,200 production tonnes of pigmeat (MAF, 2002). The increase in slaughter numbers followed six years of steady decreases.

Pig production is based on approximately 750 specialist, mainly family–owned pig farms and two large corporate pig producing units. These corporate entities are both associated with bacon processing companies (http://www.maf.govt.nz/mafnet/sectors/meat/pork.html). Canterbury and Waikato are the main production areas.

The interests of the New Zealand pig industry are coordinated through the New Zealand Pork Industry Board (NZPIB), a producer-funded body governed by the Pork Industry Board Act 1997. Funding for the NZPIB is from a levy on all pigs at the time of slaughter. Assurance of food safety of processed pork products is also a major objective of the Pork Processors Association (PPA), an incorporated society of which the majority of New Zealand's significant pork processors are members.

NZPIB initiated and the PPA funded the further development of Module 7 (PQIP 07) in the PQIP Code of Practice. This module provides a basis for the design, implementation and operation of a Food Safety Programme. It has been accepted by the New Zealand Food Safety Authority as providing such a basis for processed pork products, excluding uncooked comminuted fermented meats, although this latter category will be included in the near future.

3.2.1   Imported food

Pork imports have increased in recent years (MAF, 2002) and New Zealand is being viewed as a growth market by pork exporting countries (FAS, 2001). Approximately 33% of pork for domestic consumption is imported, principally from Canada (47% by product weight; MAF, 2002), followed by Australia (36%). Imported volumes of pork increased by 27% from the 2000/01 year to the 2001/02 year.

MAF Biosecurity requires that imported pork is cooked and frozen, or else frozen and imported into a transitional facility where it is cooked. There are several Import Health Standards which contain requirements for the importation of processed pork products (see: (http://www.maf.govt.nz/biosecurity/imports/animals/standards/index.htm).

The standards cover:
- Processed pork products from Canada, Denmark, Mexico, USA which are required to have been subjected to specified heat treatments or pH modification,
- Pork products from Australia, Sweden. Edible offals must be frozen to -18ºC during transport,
- Unprocessed pork products from Canada, Denmark, the Mexican state of Sonora, USA which are required to receive specified heat treatments or pH modification after arrival,
- Specified pork products from Italy. These include prosciutto di Parma (Parma ham) or other ham that has undergone an equivalent 12 month curing process and cooked pork products.

While it is probable that the required cooking regimes will render some imported product free of *Y. enterocolitica*, product that is not required to be cooked (e.g. that from Porcine Reproductive and Respiratory Syndrome-free countries such as Australia) and imported product that might have been recontaminated after cooking may still be contaminated with *Y. enterocolitica*.

4   HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

Ingested cells of pathogenic *Y. enterocolitica* which survive passage through the stomach acid adhere to the mucosal cells of the Peyer's patches (gut-associated lymphoid tissue).   The adhered cells are taken up by the epithelial cell, from which they are released into the lamina propria where they invade phagocytic cells and multiply extracellularly producing a local inflammatory response.   Damage to the absorptive epithelial cells results in malabsorption and fluid loss characterised by diarrhoea. A heat stable enterotoxin is produced, but its role in pathogenesis, if any, is unclear (Adams and Moss, 2000)

4.1   Symptoms

Incubation: Approximately 7 days, range 1-11 days.

Symptoms: In younger children (< 5 years) the symptoms of *Y. enterocolitica* infection are predominantly those of enterocolitis (vomiting, diarrhoea, low-grade fever and less frequently abdominal pain) (Natkin and Beavis, 1999; Ehara *et al*., 2000). In contrast, older children are more likely to experience abdominal pain as the prominent symptom (Ehara *et al*., 2000). Adults usually present with nonspecific abdominal pain and diarrhoea (Natkin and Beavis, 1999).   Bacteraemia and sepsis may occur in high risk individuals, such as those with diabetes, liver disease, immunosuppression etc.

Abdominal pain in the lower right quadrant can lead to unnecessary appendectomies being performed. This was illustrated in an outbreak in the USA, which was attributed to contaminated milk. Of 11 patients, three had appendicitis-like symptoms and two had appendectomies performed (Shorter *et al.,* 1998).

Condition: Yersiniosis. Hospitalisation rate estimates vary from 0.5-24%, case fatality rate estimates 0-0.5%.

Toxins: Toxins are not produced in foods by this organism.

People Affected: There is a bimodal distribution by age with peaks in those aged 4 or less, and those in the 20-34 year age group. Males are affected more frequently than females.

Long Term Effects: Complications of *Y. enterocolitica* infection may include reactive arthritis, septicaemia, lymphadenitis, disturbed liver function, and erythema nodosum. In a study of 261 Dutch patients these complications occurred generally in older patients (Stolk-Engelaar and Hoogkamp-Korstanje, 1996).   Of the 261 patients with gastrointestinal yersiniosis, uncomplicated enteritis was diagnosed in 169 patients, complicated enteritis in 37, appendicular syndrome in 33, ileitis in 8 and colitis in 14. Four patients died of generalised peritonitis, and other complications included reactive arthritis, septicaemia, lymphadenitis, disturbed liver function, and erythema nodosum.

In this study there was an additional group of patients (n=142) who had complicated yersiniosis such as arthritis and erythema nodosum without gastrointestinal symptoms.

A study in the UK found *Y. enterocolitica* as frequently in the faeces of controls as in cases reflecting the organism's ability to produce asymptomatic infections. No difference between the types of *Y. enterocolitica* in cases and controls was identified (Tompkins *et al.,* 1999).

Reactive arthritis (synonymous with Reiter's syndrome) may sometimes follow infection. People who are HLA (human lymphocyte antigen)-B27 positive are particularly at risk (Natkin and Beavis, 1999). The illness normally appears one to three weeks after infection and continues for a few weeks or months.

In a study of foodborne illness in the United States, Mead *et al.* (1999) assumed 90% foodborne transmission, a hospitalisation rate of 24.2% and a case fatality rate of 0.05%.

Treatment: Antibiotics have not been shown to reduce the severity or duration of the gastrointestinal illness in children less than six years of age, but may prevent complications of *Y. enterocolitica* enteritis in older children. In compromised patients at risk of bacteraemia, antibiotic treatment is recommended (Bottone, 1999).

4.2 Dose-response

Very little is known about the dose response relationship for *Y. enterocolitica*. Some mouse oral challenge studies showed clearly that a plasmid is required for virulence, but only one challenge dose ($2x10^9$ /ml) in water was given to mice which had been deprived of water for 24 h (Lee *et al.,* 1981). Clinical signs of infection were either diarrhoea or death. Varying doses were given by intraperitoneal injection but this does not reflect oral challenge.

Szita *et al.* (1973) reported on the consumption by one of the authors of 3.5×10^9 organisms. The infection caused enterocolitis with fever and lasted 4 weeks. The organism was reported to be isolated "almost in pure culture" from (presumably) the faeces during this period. Since only one dose was used in one subject, and the dose administered was very high, the experiment adds little to the elucidation of the dose response relationship.

It might be speculated that since cross contamination during chitterlings preparation is a known route of infection the dose required to elicit disease in children, at least, might be much less than 10^9.

It is emerging that for bacterial foodborne pathogens the concept of an infectious dose is no longer valid. Dose response models provide an estimate of the probability of disease at a given dose, and such models are reasonably well developed but not for *Y. enterocolitica*. However, the concept that there is some "cut off" dose, other than 0, that is required to cause disease seems unlikely to represent reality. It is likely though that for some organisms there will be a range of doses over which infection and disease is very unlikely.

5   EXPOSURE ASSESSMENT

5.1   The Hazard in the New Zealand Food Supply: *Yersinia enterocolitica* in Pork

5.1.1   *Yersinia enterocolitica* in pigs

Few data on the prevalence of *Y. enterocolitica* in New Zealand pigs are available in the published literature. A study of *Y. enterocolitica* as a cause of human gastroenteritis in New Zealand (Fenwick and McCarthy, 1995) included preliminary results from a Massey University survey of farm animals. This indicated that that pigs were the only domestic animals to carry *Y. enterocolitica* serotype O:3, whereas serotypes O:9 and O:5,27 were recovered from a wider range of animal species, including cattle, deer, goats, sheep, cats and dogs.

A report on yersiniosis as an emerging problem in New Zealand (Wright et al., 1995) claimed that serotype O:3 biotype 4 was present in New Zealand pigs at a relatively high prevalence rate, as it is in other pig producing countries.

A report on method development for *Y. enterocolitica* detection (Hussein *et al*., 2001) reported that 10 subject pigs were free of *Y. enterocolitica* prior to experimental inoculation. However, the husbandry of the animals was not reported and they are likely to have been housed in special units and so are unlikely to be representative of commercially reared animals.

5.1.2   *Yersinia enterocolitica* in other farmed animals

During 1984-85 rectal contents of 330 cull cows and 66 lambs were sampled and tested for *Yersinia* species (Bullians, 1987). The prevalence of *Yersinia* spp. in cows was 0.6% and in lambs was 28%. Thirteen of the isolates from lambs were *Y. enterocolitica*, of which four were biotype 5 and the remainder were untypable, but were within the group of biotypes 1 to 4. Three isolates of *Y. enterocolitica* were serotyped, with two being serotype O:3 and the other being untypable.

A follow-up survey was carried out in 1985-86 involving 281 lambs and 220 cattle (youngest obtainable bulls). The prevalence of total *Yersinia* species was 37% in lambs and 16% in cattle. Of 61 *Y. enterocolitica* isolates from lambs 33 were biotype 4, serotype O:3. From young bull cattle two out of eight *Y. enterocolitica* isolates were biotype 4, serotype O:3 (Judi Lee, NZFSA, personal communication). A further study of 140 lambs (14 lambs from each of 10 lines), the prevalence of *Yersinia* species within a line ranged from 0 to 85%, with the prevalence of *Y. enterocolitica* biotype 4, serotype O:3 within a line ranging from 0 to 80% (Judi Lee, NZFSA, personal communication).

These results conflict with the findings of Fenwick and McCarthy (1983) and indicate that pigs are not the only domestic animal to carry *Y. enterocolitica* serotype O:3.

5.1.3 *Yersinia enterocolitica* in pork

Very little information is available. A small survey of ready-to-eat pork products detected *Y. enterocolitica* in two of 34 samples, but the types detected were not pathogenic (Hudson *et al.*, 1992, Table 4).

5.1.4 Conclusion

There is obviously a data gap with regard to contamination of pork in New Zealand by *Y. enterocolitica*. This data gap is unlikely to be filled until suitable analytical methods can be established (see section 2.3). Furthermore, the results presented in Section 5.1.2 suggest that there is disagreement on the prevalence of *Y. enterocolitica* in species other than pigs and more up-to-date comparative data are required.

5.2   Food Consumption: Pork

Pork consumption in New Zealand has increased steadily during the period 1985-1999 (Table 1).

Table 1:   New Zealand domestic meat consumption per capita 1985, 1995, 1996 & 1999 (kg/person/year)

Year	Sheep and Lamb	Beef and Veal	Pork	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 pork consumption figures for New Zealand in Table 1 are similar to estimates made for the Australian population (Baghurst, 1999). The Australian consumption levels for 1996-97 were 17.9 kg pork/person/year.

Reference to Food Balance Sheets maintained by FAO (http://apps.fao.org) suggests that pork consumption in New Zealand is moderate by international standards, with per capita consumption levels in European countries commonly in excess of 30 kg/person/year. FAO calculates an average for 'Developed' countries of 29 kg/person/year, while 'Developing' countries have an average pork consumption of just over 11 kg/person/year.

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). FSANZ have recently 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 2 gives the estimates for meat consumption derived by FSANZ and compares those levels of consumption to the estimates based on meat available for consumption (Table 1).

Table 2: **Mean estimates of 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
Pork	32.3	44.1
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 38.0% of the population consumed pork during any 24 hour period with a mean consumption, for all respondents, of 32.3 g/person/day. The mean daily consumption, for consumers only, was 85.1 g/day. The median daily consumption, for consumers only, was 47.6 g/day. The 97.5[th] percentile daily consumption, for consumers only, was 374 g/day.

Of the pork consumed, approximately 30%, by weight, is in the form of cuts of meat (steaks, chops, roasts) and pork mince, 15% as ham, 10% as bacon, and 2% as salami. The balance of the pork consumed will be as an ingredient in foods such as sausages, pizzas and pies.

5.3   Qualitative Estimate of Exposure

5.3.1   Number of servings and serving sizes

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

- That the sample set employed for the NNS are 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,
- That the consumption of pork 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).

The FSANZ analysis of the data from the 1997 National Nutrition Survey (ANZFA, 2001) identified 1760 respondents (38.0% who reported consuming pork in the previous 24 hour period). Assuming that consumers would generally only consume one serving of pork per day and assuming a New Zealand total population of 3,737,490 (Census 2001) the total number of servings per annum would be:

Annual number of servings (total population) $=$ 3,737,490 x 0.38 x 365
$=$ 5.2×10^8 servings per annum

This represents a high number of servings, however in many cases the size of the serving would be expected to be quite small, as for deli meats.

The mean daily consumption for consumers only identified by ANZFA was 85.1 g/day, while the median and 97.5^{th} percentiles were 47.6 and 374 g/day respectively. If it assumed that consumers will generally only eat one serving of pork per day then these figures will equate to mean, median and 97.5^{th} percentile serving sizes.

To put these figures in perspective, the number of servings of pork calculated for the New Zealand population are greater than the number calculated for poultry, while the median serving size is approximately half that for poultry.

5.3.2   Frequency of contamination

The proportion of pork samples contaminated with *Y. enterocolitica* in New Zealand is not known and the overseas data are very variable, possibly reflecting different methodologies used. However, the proportion of retail pork samples that might be contaminated with pathogenic types appears to be lower than found in carcasses (see Tables 3 and 4 below) and so the frequency of contamination by pathogenic types is very much reduced, by perhaps around 90%, over the crude isolation rate.

5.3.3   Predicted contamination level at retail

The information to make comment on this does not really exist. This represents a significant data gap.

5.3.4   Growth rate during storage and most likely storage time

Since *Y. enterocolitica* can grow at refrigeration temperatures growth during storage can only be slowed. On raw foods growth may be largely inhibited by the normal microflora of the food, although some selection for *Y. enterocolitica* might occur at low temperatures. Growth on cooked foods is likely, and shelf life times applied to vacuum packed pork products such as ham may make growth significant.

5.3.5 Heat treatment

Y. enterocolitica is not unusually heat resistant and normal cooking practices should be enough to inactivate it. Post cooking contamination of ready-to-eat foods may be a significant factor in transmission.

5.3.6 Exposure summary

Not enough is known about this organism to comment in an informed manner on this. *Y. enterocolitica* may be reasonably common on ready-to-eat pork products and opportunities exist for it to reach high numbers. However, the proportion of pathogenic types on ready-to-eat foods might be quite small.

5.4 Overseas Context

5.4.1 *Yersinia enterocolitica* in pigs

Table 3 summarises data from the literature on the prevalence of *Y. enterocolitica* in pigs at or prior to slaughter.

Table 3:   Prevalence of *Y. enterocolitica* in pigs at or prior to slaughter

Country	Samples tested	Number (%) positive	Sero/Biotype information	Reference
Canada	Slaughter pig faeces	36/200 (18.0)	7 type 1, 3 type 3, 26 type 4	Mafu *et al.,* 1989
Canada	Slaughter pig diaphragms	5/200 (2.5)	All type 4	Mafu *et al.,* 1989
Canada	Finishing pig herds	Interval (% +ve) Frequency (%) 0 32.7 0-10 15.0 10-20 22.4 20-30 7.5 30-40 6.5 40-50 3.7 50-60 4.7 60-70 2.8 70-80 2.8 80-90 1.9 Overall prevalence: 20.9% ±2.1%	85.5% O:3 9.1% O:5 0.4% O:8 3.3% O:9 1.8% NT	Letellier *et al.,* 1999
Denmark	Pig throat swabs	84/282 (30.0)	O:3	Pedersen, 1979
Denmark	Pig rectal samples	360/1458 (24.7)	O:3 (all)	Andersen, 1988
Denmark	Pig caecal contents	48/293 (16.4)	O:3 (all)	Anonymous, 1999
Finland	Pig faeces	26/147 (17.7) animals 5/14 (35.7) herds	All O:3	Asplund *et al.,* 1990
Finland	Pig tonsils:herds with low condemnation records	41/131 (31.3) animals 8/13 (61.5) herds	All O:3	Asplund *et al.,* 1990
Finland	Pig tonsils:herds with high condemnation records	134/350 (38.3) animals 26/35 (74.3) herds	All O:3	Asplund *et al.,* 1990

Country	Samples tested	Number (%) positive	Sero/Biotype information	Reference
Finland	Pork tongues	47/51 (92) by PCR 40/57 (78) by culture	49/51 (96%) biotype 4 2 (4%) biotype 1A	Frederiksson-Ahomaa *et al.,* 1999
Finland	Pig carcass	17/80 (21.3) by PCR 5/80 (6.3) conventional	All tested biotype 4	Frederiksson-Ahomaa *et al.,* 2000
Finland	Pig ear	4/17 (23.5) by PCR 2/17 (11.8) conventional	All tested biotype 4	Frederiksson-Ahomaa *et al.,* 2000
Finland	Pig liver	5/13 (38.5) by PCR 4/13 (30.8) conventional	All tested biotype 4	Frederiksson-Ahomaa *et al.,* 2000
Finland	Pig kidney	11/13 (84.6) by PCR 9/13 (69.2) conventional	All tested biotype 4	Frederiksson-Ahomaa *et al.,* 2000
Finland	Pig heart	5/8 (62.5) by PCR 4/8 (50.0) conventional	All tested biotype 4	Frederiksson-Ahomaa *et al.,* 2000
Finland	Pork tongues	82/99 (83) by PCR 79/99 (80) conventional	97.8% biotype 4, serotype O:3 2.2% biotype 1A, serotype NT	Fredriksson-Ahomaa *et al.,* 2001
Finland	Pork heart	4/8 (50) by PCR 1/8 (13) conventional	100% biotype 4, serotype O:3	Fredriksson-Ahomaa *et al.,* 2001
Finland	Pork kidney	5/8 (63) by PCR 4/8 (50) conventional	100% biotype 4, serotype O:3	Fredriksson-Ahomaa *et al.,* 2001
Norway	Pig oral cavity	25/30 (83.3)	O:3	Nesbakken, 1988
Norway	Pig carcasses, post evisceration	7/60 (11.7)	O:3	Nesbakken *et al.,* 1994
Norway	Lymphoid tissues	23/72 (31.9)	All isolates serotype O:3	Nesbakken *et al.,* 2003
Norway	Intestinal tract	14/120 (11.7)	All isolates serotype O:3	Nesbakken *et al.,* 2003
Norway	Carcass surface	3/96 (3.1)	All isolates serotype O:3	Nesbakken *et al.,* 2003
Sweden	Pig carcasses, post evisceration	5/60 (8.3)	O:3	Nesbakken *et al.,* 1994
Trinidad	Pig rectal samples	23/141 (16.1)	All isolates biotype 4	Adesiyun and Krishnan, 1995
Trinidad	Pig tongue swabs	9/141 (6.4)	All isolates biotype 4	Adesiyun and Krishnan, 1995
Trinidad	Pig tonsil swabs	11/150 (7.3)	All isolates biotype 4	Adesiyun and Krishnan, 1995
Trinidad	Carcasses contaminated	30 141 (21.3)	All isolates biotype 4	Adesiyun and Krishnan, 1995
USA	Pork carcasses after singeing and polishing	0/270 (0)	NC	Saide-Albornoz *et al.,* 1995
USA	Pork carcasses after final rinse	1/270 (0.04)	NC	Saide-Albornoz *et al.,* 1995
USA	Pork carcasses after 24h chilling	0/270 (0)	NC	Saide-Albornoz *et al.,* 1995
USA	Slaughter pigs	0/932 (0)	NC	Miller *et al.,* 1997
USA	Pigs at slaughter	29/103 (28.2) lots with at least 1 +ve pig	89.7% O:5 3.7% O:3	Funk *et al.,* 1998
USA	Pork tongues	5/15 (33.3) conventional 10/15 (66.7) by Taqman PCR	ND	Vishnubhatla *et al.,* 2001

NT=Not typable

The data in Table 3 indicate that contamination of pig carcasses is common at slaughter. The sero/biotypes identified are quite often those of pathogens. Contaminants originate from the faeces and pig's head, especially the nasopharynx and contamination of other parts of the carcass appears to result from inadequate control of these sources.

5.4.2 *Yersinia enterocolitica* in pork

The prevalence of *Y. enterocolitica* in pork products offered for sale at retail is shown in Table 4, as is information of the sero/biotypes that have been identified. The data show that pork products are frequently contaminated with *Y. enterocolitica*. However, the proportion of pathogenic sero/biotypes has changed with respect to the distribution in the animal at slaughter. Biotype 1A which is not a human pathogen is common in retail samples. Therefore the effective exposure to pathogenic *Y. enterocolitica* is very much reduced in retail products.

An exception is the outbreak associated with chitterlings (large intestines of pigs) (Lee *et al.,* 1990). In this outbreak chitterlings were prepared from raw material in homes, and the infection spread from caregivers to children. This is not surprising given the association of pathogenic serotypes with pigs at slaughter and with faeces. From the data obtained, the intestine of the pig could reasonably be expected to contain pathogenic types of *Y. enterocolitica*.

In cooked products the prevalence is in the order of 1-2%, and again the types present are usually not pathogenic. One study did find high contamination rates in ready-to-eat foods, but none of the isolates was pathogenic.

A word of caution is due here. The isolation of *Y. enterocolitica* from foods is notoriously difficult and there is no one wholly satisfactory method for all serotypes. It is likely, therefore, that the prevalences listed here are underestimates of the true values.

Table 4: **Prevalence of *Y. enterocolitica* in raw pork and raw or cooked pork products**

Country	Samples tested	Number (%) positive	Sero/Biotype information	Reference
New Zealand	**Ready-to-eat pork products**	**2/34 (6.3)**	**Serotype O:8 biotype 1A, other isolate not serotypes O:3, O:5, O:8 or O:9**	**Hudson *et al.,* 1992**
Argentina	Cooked ham	1/100 (1)	Biotype 2, serotype O:9	Velázquez *et al.,* 1993
Argentina	Salami	2/150 (1.3)	Biotype 1, serotype O:5 and biotype 2 serotype O:9	Velázquez *et al.,* 1993
Argentina	Porcine cheese (pork head, beef jaws and tongue, cooked)	2/100 (2)	Both biotype 2, serotype O:9	Velázquez *et al.,* 1993
Argentina	Mortadella	0/100 (0)	-	Velázquez *et al.,* 1993
Canada	Processed pork products	5/69 (7.2)	2 x O:17, 1 O:13,7, 2 x NT	Schiemann, 1980
Canada	Raw pork products	63/128 (49.2)	Of typable isolates; 41.3% O:3, 37.9% O:5, 3.4% O:8, 3.4% O:16, 3.4% O:17, 3.4% O:21, 6.9% O:13,7	Schiemann, 1980

Country	Samples tested	Number (%) positive	Sero/Biotype information	Reference
Canada	Pork	4 / 10 (50)	All 1A	Toora *et al.,* 1994
Denmark	Retail pork	10/306 (3.2)	ND	Anonymous, 1999
Finland	Pork and minced pork	0/104 (0)	-	Asplund *et al.,* 1990
Ireland	Pork	14/20 (70)	54.8% 1A, 22.6% 2, 3.2% 4, 19.4% NT	Logue *et al.,* 1996
Ireland	Bacon (open)	4/10 (40)	88% 1A, 12% NT	Logue *et al.,* 1996
Ireland	Bacon (vac packed)	3/10 (30)	80% 1A, 20% NT	Logue *et al.,* 1996
Ireland	Cooked ham (open)	8/20 (40)	84.2% 1A,15.8% NT	Logue *et al.,* 1996
Ireland	Cooked ham (MAP)	5/20 (25)	78.9% 1A,15.8% 4, 5.2% NT	Logue *et al.,* 1996
Ireland	Roast pork (open)	12/20 (60)	88.2% 1A, 11.8% NT	Logue *et al.,* 1996
Japan	Retail pork	37/1278 (2.9)	35.1% O:3 biotype 4 5.4% O:3 biotype 3 40.5% O:3 biotype 3 variant, 18.9% O5:27 biotype 3	Fukushima *et al.,* 1997
Mexico	Raw pork	21/40 (53)	121/166 type 1A 15/166 type 2 4/166 type 3 7/166 type 4 19 not identified	Ramirez *et al.,* 2000
Mexico	Cooked pork tongues	9/40 (23)	48/75 type 1A 7/75 type 2 1/75 type 3 2/75 type 4 17 not identified	Ramirez *et al.,* 2000
Mexico	Cooked pork sausage	2/40 (5)	58/87 type 1A 9/87 type 2 2/87 type 3 5/87 type 4 13 not identified	Ramirez *et al.,* 2000
Netherlands	Minced pork	50/200 (25.0)	3 O:3, 1 O:9, 46 other	de Boer and Nouws, 1991
USA	Chitterlings-outbreak associated	8/11 (72.7%)	All (100%) samples contained serotype O:3, three (37.5%) also contained O:1,2,3 or O:5,27	Lee *et al.,* 1990
USA	Ground pork	32/100 (32)-conventional 47/100 (47) by TaqMan PCR	ND	Vishnubhatla *et al.,* 2001
USA	Ground pork or sausage from processors	4/120 (3.3)	ND	Duffy *et al.,* 2001
USA	Whole pork muscle, store packed	19/96 (19.8)	ND	Duffy *et al.,* 2001
USA	Whole muscle, pumped or brined pork	5/96 (5.2)	ND	Duffy *et al.,* 2001
USA	Store ground pork and/or pork sausage	11/96 (11.5)	ND	Duffy *et al.,* 2001
USA	Prepacked ground pork and/or pork sausage	1/96 (1.0)	ND	Duffy *et al.,* 2001
USA	Boneless pork loins prior to packaging	0/270 (0)	ND	Duffy *et al.,* 2001

Country	Samples tested	Number (%) positive	Sero/Biotype information	Reference
USA	Boneless pork loins after 36 days storage at 2°C	12/270 (4.4)	ND	Duffy *et al.,* 2001

NT=non typable   MAP = Modified atmosphere packaging

Table 5 summarises the limited quantitative data on levels of *Y. enterocolitica* in pork products.

Table 5:   Quantitative data for *Y. enterocolitica* in pork products

Samples tested	No. (%) positive	Counts (/g)		Reference
Vienna sausage	1/51(2.0)	260		Nortjé *et al.,* 1999
Salami	0/51 (0)	-		Nortjé *et al.,* 1999
Ham	2/51 (3.9)	260, 2730		Nortjé *et al.,* 1999
Pork	See table above but these data for *Yersinia* spp. only	$<0.7 \log_{10}$ 0.7-$1.7 \log_{10}$ 1.7-$2.7 \log_{10}$ 2.7-$3.3 \log_{10}$	8 (40%) 4 (20%) 4 (20%) 4 (20%)	Logue *et al.,* 1996
Bacon (open)	See table above but these data for *Yersinia* spp. only	Negative $<0.7 \log_{10}$ 0.7-$1.7 \log_{10}$	5 (50%) 2 (20%) 3 (30%)	Logue *et al.,* 1996
Bacon (vac packed)	See table above but these data for *Yersinia* spp. only	Negative $<0.7 \log_{10}$	6 (60%) 4 (40%)	Logue *et al.,* 1996
Cooked ham (open)	See table above but these data for *Yersinia* spp. only	Negative $<0.7 \log_{10}$ 1.7-$2.7 \log_{10}$ 2.7-$3.3 \log_{10}$	10 (50%) 7 (35%) 1 (5%) 2 (10%)	Logue *et al.,* 1996
Cooked ham (MAP)	See table above but these data for *Yersinia* spp. only	Negative $<0.7 \log_{10}$ 0.7-$1.7 \log_{10}$	13 (65%) 4 (20%) 3 (15%)	Logue *et al.,* 1996
Roast pork	See table above but these data for *Yersinia* spp. only	Negative $<0.7 \log 10$ 1.7-$2.7 \log 10$ 2.7-$3.3 \log 10$ 3.3-$4.0 \log 10$ 4-$5.7 \log 10$	7 (35%) 8 (40%) 1 (5%) 1 (5%) 2 (10%) 1 (5%)	Logue *et al.,* 1996

MAP = Modified atmosphere packaging

With reference to Table 5 it seems that in most cases *Y. enterocolitica* is found in foods at levels less than 10^3 cells/g ($3.0 \log_{10}$/g). However the data available come from only two studies, and one reports numbers of *Yersinia* spp. only. Therefore quantitative data for pathogenic *Y. enterocolitica* in retail pork products represents a significant data gap.

6   RISK CHARACTERISATION

6.1   Adverse Health Effects in New Zealand

6.1.1   Incidence

Yersiniosis was made a notifiable disease in New Zealand in 1996, with the laboratory confirmation requiring the isolation of *Y. enterocolitica* or *Y. pseudotuberculosis* from blood or faeces. *Y. pseudotuberculosis* typically causes mesenteric adenitis and septicaemia while *Y. enterocolitica* causes enteric disease.

The incidence data for yersiniosis in New Zealand for 1997-2002 are given in Table 6. This information is also shown graphically in Figure 2.

Table 6:   Incidence data for yersiniosis in New Zealand

Year	Rate per 100,000 (number of cases)	Reference
1997	13.3 (480)	ESR, 1998
1998	15.1 (547)	Perks *et al.*, 1999
1999	13.9 (503)	Kieft *et al.,* 2000
2000	11.0 (397)	Lopez *et al.,* 2001a
2001	11.5 (429)	Sneyd *et al.,* 2002
2002	12.7 (476)	Sneyd and Baker, 2003

Figure 2:   Yersiniosis notifications in New Zealand by month, June 1997 – December 2002

Yersiniosis notifications by month, 1997 - 2002
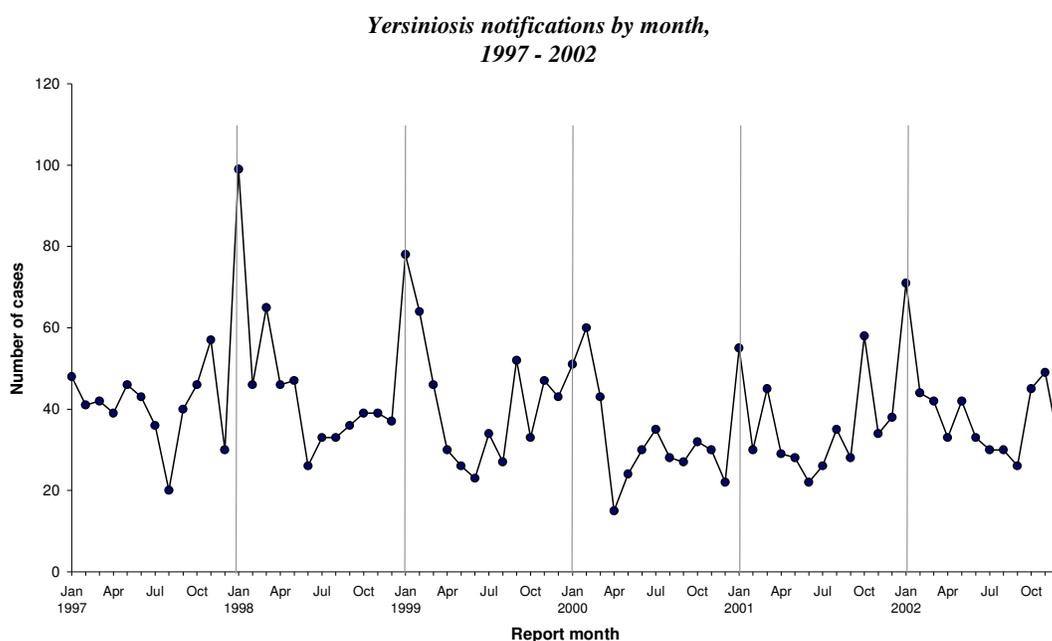
The proportion of infection with *Y. pseudotuberculosis* amongst these notified cases is low. From 1997 to May 2003 there were approximately 3,000 yersiniosis notifications and of these a species of *Yersinia* was recorded for approximately 250 cases. Only 24 were recorded as *Y. pseudotuberculosis* while approximately 220 were *Y. enterocolitica*.

The most commonly encountered strain amongst human cases in New Zealand is serogroup O:3, followed by O:9. The age distribution of notifications shows two peaks; 1-4 years and 20-29 years (Begg and Bennett, 1997).

Two further studies have provided information on yersiniosis rates in New Zealand. In 1995 a one year study in the Eastern Bay of Plenty analysed all faecal samples obtained from people who presented with gastrointestinal symptoms to General Practitioners or hospitals (Wright, 1996). The analysis for a variety of gastrointestinal pathogens identified 21 cases for whom the faecal samples contained *Yersinia*. This represented an incidence of 87 per 100,000 in the target population, which was still regarded as an underestimate of the community rate as not all cases of gastrointestinal infection seek medical assistance.

An earlier study, conducted from 1988 to 1993 (Fenwick and McCarthy, 1995) analysed 231,128 faecal samples submitted to the Diagnostic Laboratory from patients with symptoms of gastrointestinal infection in Auckland. The samples were all cultured for enteric pathogens and *Yersinia* isolates were submitted for further typing. A total of 1469 samples, representing 941 cases, were positive for strains of *Yersinia*, representing an isolation rate of 0.6%. The majority of the isolates (918) were identified as *Y. enterocolitica*. The most common type amongst these isolates was Biotype 4, serotype 0:3. No clear seasonal pattern emerged, but the age distribution showed two peaks, amongst 0-4 and 25-29 year olds.

Overall *Yersinia* was detected about 40% as often as *Salmonella* and about 10% as often as *Campylobacter* in this study. This is a relatively higher proportion of *Yersinia* cases than that determined from national notification data (about 20% of the incidence of *Salmonella* and about 5% of the incidence of *Campylobacter*; Lopez *et al.*, 2001a).

An asymptomatic control group was not included with either of these studies, although an English study found similar isolation rates for *Y. enterocolitica* in cases and controls (Tompkins *et al.,* 1999).

Culture for *Y. enterocolitica* is part of the routine screening for pathogens conducted by most clinical and public health laboratories in New Zealand (Carolyn Nicol, Enteric Reference Laboratory, ESR, pers. comm.). Although there are indications that some under-reporting of yersiniosis is occurring in New Zealand (Mitchell *et al.*, 2001), it is also possible that some of the *Y. enterocolitica* isolations found in the Eastern Bay of Plenty and Auckland were of non-pathogenic species, and that there is a background carriage rate of this bacterium for the New Zealand population as a whole.

6.1.2   Outbreaks

As shown in Table 7, *Yersinia* is identified as the causal agent in very few reported outbreaks in New Zealand each year.

Table 7:   Reported outbreak data for *Yersinia enterocolitica* in New Zealand

Year	Outbreaks*	Cases**	Reference
1997	2/97 (2.1%)	5/1209 (0.4%)	ESR, 1998
1998	2/207 (1.0%)	9/1552 (0.6%)	Naing *et al.*, 1999
1999	0/352 (0%)	0/2302 (0%)	Perks *et al.*, 2000
2000	0/273 (0%)	0/1903 (0%)	Lopez *et al.*, 2001b
2001	3/369 (0.85%)	10/2095 (0.4%)	ESR, 2002
2002	3/337 (0.9%)	10/2890 (0.3%)	Boxall and Ortega, 2003

* Totals are for outbreaks of enteric disease only
** Includes both suspected and confirmed cases

The data reported for these outbreaks indicated that consumption of pork or ham was suspected as the source of infection in four outbreaks, and a further outbreak was associated with backyard slaughter of pigs and was possibly due to zoonotic exposure. Animal contact was noted in two other outbreaks, although in one of these cases the implicated animal species (ducks) is not a recognized source of pathogenic *Y. enterocolitica*. For three of the remaining outbreaks person to person transmission was mentioned, while no possible vehicle was identified in the remaining outbreak. Person-to-person spread was also suggested in two outbreaks as a factor contributing to the progression of the outbreak, following an index infection from food or zoonotic sources.

6.1.3   Clinical consequences of *Yersinia* infection

Table 8 summarises information on the clinical consequences of yersiniosis in New Zealand since 1997.

Table 8:   Summary of clinical consequences of yersiniosis in New Zealand

Period	Hospitalised*	Fatalities	Reference
1997	20/371 (5.4%)	0	ESR, 1998
1998	29/452 (6.4%)	2/547 (0.4%)	Perks *et al.*, 1999
1999	26/386 (6.7%)	0	Kieft *et al.*, 2000
2000	35/301 (11.6%)	0	Lopez *et al.*, 2001
2001	17/279 (6.1%)	0	Sneyd *et al.*, 2002
2002	31/476 (6.5%)	0	Sneyd and Baker, 2003

*Percentages are determined on the basis of cases for which information was available

The two fatalities recorded in 1998 were of a 24 year old adult male and a 10-month-old infant. Exposure to animal products was listed as a risk factor in the case of the adult male. Food was not implicated as the source of infection in either case, although the case reports provide very few details.

6.1.4 Case control studies and risk factors

One case-control study of *Y. enterocolitica* infections has been conducted in New Zealand (Satterthwaite *et al*., 1999). It included 186 cases and 379 controls located in Auckland between April 1995 and June 1996. Exposure factors covered included environmental (animal exposure, childcare, water and sewerage), food (range of meats as well as shellfish and fruit and vegetables), and food preparation practices.

Of the potential food vehicles only pork (OR = 1.34, CI = 1.03-1.75) had a significantly higher rate of consumption amongst cases than controls. However, this risk factor was the second lowest significant factor in terms of population attributable risk. The other statistically significant risk factors were: eating food from a sandwich bar (OR 1.18), and more than two people living in a household (OR 2.20) (this suggests that person to person transmission is important). "Protective" factors included looking after a child (OR 0.51), having a town sewerage connection (OR 0.34), connection to a town water supply (OR 0.20), eating raw fruit and vegetables (OR 0.98), eating bacon (OR 0.75) or smallgoods (OR 0.73). While handling animals at work (OR 2.18) or at home (OR 1.32) occurred at higher rates amongst cases than controls, the association was not statistically significant.

The population attributable risk (PAR) was calculated for each factor in this study. It found that the most significant exposure was unreticulated sewage (PAR 0.593). The food related PAR values were: pork (0.147), vegetables and fruit (0.219), food from a sandwich bar (0.203).

Unpublished results from the one year study of gastrointestinal disease in the Eastern Bay of Plenty (Wright, 1996) were cited in a review of yersiniosis in New Zealand written for the Ministry of Health (Begg and Bennett, 1997). Consumption of water from a home supply was reported to cause a statistically significant three fold increase in the risk for intestinal *Y. enterocolitica* infection. The same study also identified the handling of cattle (relative risk = 4.88; p = 0.008) and sheep (relative risk = 14.80; p = 0.001) as associated with an increased risk of infection. In a published report on the project (Wright *et al.*, 1995) contact with farm animals was cited as a risk factor (relative risk 3.9; 95% CI 1.95-7.68) but contact specifically with pigs was not. These risk factors were identified in a predominantly rural region of New Zealand, and different factors may be important in urban populations.

New Zealand has experienced problems in the recent past with deaths following blood transfusions because the blood was contaminated with *Y. enterocolitica* (Theakston *et al.,* 1997). These authors estimated a fatality rate on 1 in 104,000 transfusions, which is 80 times the equivalent figure for the USA. The problem arises when asymptomatic donors give blood containing small numbers of *Y. enterocolitica* cells. Storage of blood, an excellent microbiological growth medium, under refrigeration enables the cells to grow to high numbers. When this blood is used in a transfusion the high numbers present can cause the patient to die. Whether the high fatality rate in New Zealand is a result of better reporting, different practices or a higher rate of infection, is not known.

6.1.5   Animal contact

Transmission from animals to humans is suspected, as shown by an investigation of parallel trends in yersiniosis in cattle and humans in central France. Although most cases were believed to have been caused by foodborne transmission, a proportion of cases were cattle breeders or had contact with animals (Gourdon *et al*., 1999).

A survey of 381 *Y. enterocolitica* isolates obtained from (mostly) clinical cases in New Zealand animal diagnostic laboratories between 1988 and 1996 provided data on their biotype and serotype (Fenwick, 1997). Yersiniae were isolated from alpacas, cattle, sheep, goats, deer, dogs, cats, pigs, horses, primates and birds. Strains of *Yersinia* potentially pathogenic for people made up 34% of the total. This paper commented that cattle are likely to be the major reservoir of serotype O:9, biotype 2 *Y. enterocolitica* in New Zealand.   Dogs may also act as a secondary reservoir of pathogenic *Y. enterocolitica*.

In summarising notifications for the 2002 year Sneyd and Baker (2003) noted that of 200 yersiniosis notifications where information on contact with farm animals was provided, 59 (29.5%) reported contact. A total of 91 cases were identified as having had contact with farm animals, wild animals or pets.

As mentioned above, in the Auckland case control study (Satterthwaite *et al*., 1999) handling animals at home or at work gave elevated, but not statistically significant odds ratios. The Eastern Bay of Plenty study (Wright *et al*., 1995) contact with farm animals was a significant risk factor for yersiniosis (relative risk 3.9; 95% CI 1.95-7.68) but contact specifically with pigs was not.

6.2   Adverse Health Effects Overseas

6.2.1   Incidence

Table 9 summarises incidence data for yersiniosis from a range of overseas sources.

Table 9:   Incidence data for yersiniosis overseas

Country	Incidence (cases/100,000)	Year	Reference
New Zealand	**12.7**	**2002**	**Sneyd and Baker, 2003**
Australia	1.7	1998	Thomson *et al.,* 1999
Australia	0.6	2000	Lin *et al*., 2002
Belgium	12	1984	Tauxe *et al.,* 1987
Czech Republic	1.9	1993-1999	National Institute of Public Health, Czech Republic, 2000
Denmark	29	1985	Nielsen and Wegner, 1997
Denmark	15	1995	Nielsen and Wegner, 1997
Denmark	6.4	1999	Dansk Zoonosecenter, 2000
Denmark	5.3	2001	Dansk Zoonosecenter, 2002
Denmark	4.5	2002	Dansk Zoonosecenter, 2002

Country	Incidence (cases/100,000)	Year	Reference
Finland	11.3-17.5	1995-1999	Fredriksson-Ahomaa *et al.,* 2001
UK	0.7 (0-4 year group) 0.5 (> 65 year group) approx 0.3 (other age groups)	1996	PHLS, 1997
USA (selected states and counties)	1.0	1996	Wallace *et al.,* 2000
USA (selected states and counties)	0.9	1997	Wallace *et al.,* 2000
USA (selected states and counties)	1.0	1998	CDC, 2000

The steady decline in the incidence of yersiniosis in Australia and a lack of outbreaks led to the removal of this disease from the national notifiable diseases list in January 2001 (Lin *et al.*, 2002).

New Zealand's rate of reported yersiniosis is well above most comparable countries overseas. This comment assumes, of course, that the efficiency of reporting is similar in each country.

6.2.2   Contributions to outbreaks and incidents

Outbreaks of yersiniosis are uncommon events (see Table 10) and those that have been recorded generally have not involved pork. Most of the published pork-related outbreaks have been caused by the preparation of chitterlings (see above/below) and were not related to consumption of the food itself but more due to cross contamination during their preparation. An outbreak in Hungary affected eight people of 18 who had consumed parts of the same "pork cheese", a product in which small pieces of boiled chitterlings are stuffed into a pig's stomach prior to a further cooking step. The same biotype and phage type was isolated from some of the people involved and from leftover food (Marjai *et al.,* 1987). The symptoms observed reflected other observations that children typically suffer from enteritis whereas adults tend to suffer from extra intestinal infections.

Table 10:   Contribution of *Y. enterocolitica* to foodborne disease

Country	Incidents	Outbreaks	Year(s)	Reference
Canada	0%	0%	1979	Todd, 1987
Canada	0.12% incidents, 1.9% cases	ND	1975	Todd, 1992
Canada	0%	ND	1976	Todd, 1992
Canada	0%	ND	1977	Todd, 1992
Canada	0%	ND	1978	Todd, 1992
Canada	0.13% incidents, 0.01% cases	ND	1980	Todd, 1992
Canada	0%	ND	1981	Todd, 1992
Canada	0.20% incidents,	ND	1982	Todd, 1992

Country	Incidents	Outbreaks	Year(s)	Reference
	0.08% cases			
Canada	0%	ND	1983	Todd, 1992
Canada	0%	ND	1984	Todd, 1992
Sweden	<1% incidents, <1% cases	<1% outbreaks, <1% cases	1992-1997	Lindqvist *et al.,* 2000
UK	ND	None recorded	1992-1994	Djuretic *et al.,* 1996
UK	ND	None recorded	1995-1996	Evans *et al.,* 1998
USA	ND	<1% outbreaks, <1% cases	1973-1987	Bean and Griffin, 1990
USA	ND	None recorded	1988-1992	Bean *et al.,* 1996
USA	ND	0.1% outbreaks, <0.1% cases, 3.4% fatalities	1993-1997	Olsen *et al.,* 2000

ND   No Data supplied [*]Both outbreaks involved pork as the vehicle

Chitterlings are raw pork intestines that are eaten as part of the Winter festivities (Thanksgiving through to New Year) in African American families in the USA. Their preparation is laborious and time consuming although the food is cooked for extensive periods prior to consumption. Chitterlings appear frequently in the scientific literature regarding yersiniosis.

An outbreak of yersiniosis attributed to chitterlings consumption was reported among 15 children from African American families in the 1988-1989 winter holidays (CDC, 1990). It was thought that the children's infection was a result of cross contamination from the chitterlings to the children via the preparer of the food.

Abdel-Haq *et al.* (2000) reported on an investigation of pathogens in the faeces of children attending a hospital in Michigan. Pathogens were isolated from 10.6% of 10,570 samples tested, and of these positive cultures 142 (12.6%) were *Y. enterocolitica*. All but one of the patients was African American and 85% were one year old or younger. Most of the patients (84%) presented in November, December or January. Seven of 78 patients (9%) who had blood cultures tested were suffering from *Yersinia* septicaemia. Twenty five of 30 cases about whom information was obtained reported exposure to chitterlings. It was concluded that chitterlings preparation by parents is a likely risk factor in the demographic in question. The same pattern of disease has been shown in other studies (e.g. Lee *et al.,* 1990; Lee *et al.,* 1991).

A case has also been linked to a child suffering from sickle cell anaemia and who had been exposed to chitterlings (Stoddard *et al.,* 1994).

Other than the chitterlings outbreaks, other outbreaks of yersiniosis have not generally been linked to the consumption of pork or pork products.

6.2.3   Case control studies

Table 11 gives results of overseas case-control studies considering risk/protective factors associated with yersiniosis. As with the New Zealand study, the consumption of pork and pork products has been identified as a risk factor for yersiniosis.

Table 11:   Case control studies. Relevant risk/protective factors

Country	Risk factors	Odds Ratios/P values	Reference
Belgium	Eating raw pork (risk)	OR=12 (CI 2-36.8), P=0.00002	Tauxe *et al.,* 1987
Canada	Eating salami (risk)	OR=9.0 (CI=1.25-395), P=0.027.	Harb *et al.,* 2000
Norway	Eating pork items (risk)	P=0.02	Ostroff *et al.,* 1994; Kapperud *et al.,* 1995
Norway	Eating sausages (risk)	P=0.03	Ostroff *et al.,* 1994; Kapperud *et al.,* 1995

The Canadian study (Harb *et al.*, 2000) was a retrospective case-control study initiated after an outbreak of *E. coli* O157:H7 in salami. Several yersiniosis cases had also reported exposure to dry fermented meat products. Only 19 of 47 cases were able to be included, and although the association with salami was statistically significant, *Y. enterocolitica* was not isolated from salami products collected from cases for the *E. coli* O157:H7 investigation.

The study in Belgium (Tauxe *et al*., 1987) was initiated because that country was reported to have the highest *Y. enterocolitica* isolation rate in the world. A total of 40 cases were matched with controls. Illness was not associated with eating a number of foods, including cooked meat (including game and pork tongue). However, eating any raw pork was strongly associated with illness. Raw pork seems to be a common foodstuff in Belgium, but the paper suggests that it is not commonly consumed in France or the UK. This is despite the selling of Belgian pork contaminated with *Y. enterocolitica* in France near the Belgian border.

The Norwegian report concerned a prospective case control study that matched 67 cases with 132 controls (Ostroff *et al.*, 1994). As well as the association between consuming pork items and sausage in the two weeks prior to illness, patients were also more likely to have a preference for eating meat prepared raw or rare, and for drinking untreated water. Changes to slaughtering practices were amongst the suggestions for control measures (see Section 7.2.1).

6.2.4   Risk assessments and other activity overseas

No risk assessments for *Y. enterocolitica* conducted overseas have been located. A paper on risk management in poultry slaughter (Nielsen and Wegener, 1997) claimed that pork or pork products were the major source of human yersiniosis in Denmark, and a decline in yersiniosis incidence was ascribed to improved hygiene at slaughter (see Section 7.2).

6.2.5   Secondary transmission

The data on secondary transmission are contradictory. For example, a Japanese study comments "Secondary infection with *Yersinia* among close family members was frequent" (Fukushima *et al.,* 1985) to "there was little clinical or laboratory evidence of secondary spread to family members who did not eat tofu" (Tacket *et al.,* 1985). However, given that *Y. enterocolitica* is excreted at 10^4-10^9 cells/g in the faeces of patients (Fukushima *et al.,* 1985), that a nosocomial outbreak was probably caused by person-to-person spread (Ratnam *et al.,* 1982) and the observation that chitterlings preparers can infect children (see above) then there does seem reasonable likelihood that secondary transmission is important, as it is in similar diseases where the organism is present in faeces at high numbers (e.g. salmonellosis). Szita *et al.* (1973) reported a number of incidents that appeared to reflect person-person transmission, while a prospective Canadian case-control study reported spread of the disease to family members in 47% of cases (Kapperud and Slome, 1991).

6.3   Qualitative Estimate of Risk

Data on the prevalence of *Y. enterocolitica* in pork in New Zealand are limited, and the contamination rate found in a single study (6%) may be an underestimation, due to the known difficulty of isolating the organism from food (Hudson *et al.*, 1992).

Approximately 30% of the pork supply in New Zealand is imported, with Canada being the major source. Literature information suggests that prevalence of *Y. enterocolitica* may be as high as 50% in Canadian pork, although the information available is reasonably old (Schieman, 1980; Toora *et al.*, 1994). The controls on imported product described in the relevant MAF standards (see Section 3.2.1) should reduce risk from this source, although pork from some countries (e.g. Australia) may enter New Zealand without an additional pathogenic reduction treatment and pork from other sources may still be recontaminated after treatment.

Rates of yersiniosis are relatively high in New Zealand compared to Australia and other countries. Pork has been implicated in a proportion of cases, through outbreak investigations and through a case-control study. However, it should be noted that the case-control study identified other more important risk factors for yersiniosis, such as unreticulated sewage (Satterthwaite *et al.,* 1999), and water from a home supply and handling farm animals were identified as risk factors in another study (Begg and Bennett, 1997). In addition the number of yersiniosis outbreaks is small and so the information from them is not strong.

Overall it is difficult to assess the risk from *Y. enterocolitica* in pork. While it is likely that a proportion of cases are due to transmission in pork, the absence of information on the prevalence of *Y. enterocolitica* in pigs or retail pork products limits the ability to make an assessment.

6.4 Risk Categorisation

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

Based on notification rates to July 2001 (and an assumption that 65% of yersiniosis cases are foodborne (Lake *et al.*, 2000)) the estimated incidence is 38 per 100,000 (Appendix 1). It is uncertain what percentage of these cases may be caused by pork or pork products. However, the population attributable risk analysis in the case control study (Satterthwaite *et al.*, 1999) suggested that pork was a factor in approximately 15% of all cases of yersiniosis (15% of a total estimated rate of 59 per 100,000 is approximately 9 per 100,000). This would place the rate of yersiniosis in New Zealand due to consumption of pork and pork products in incidence Category 3 (Appendix 1; 1-10 cases/100,000).

It should be noted that the lack of information on the rate of contamination of pork and pork products in New Zealand with pathogenic strains of *Y. enterocolitica* means it is not possible to make an estimate of dietary exposure to *Y. enterocolitica* to support this estimated rate of yersiniosis.

Lake *et al.* (2000) identified 16 hospitalised cases of yersiniosis out of a total number of estimated cases of 2920 (0.5%) after correction of incidence for under-reporting. No more serious outcomes were identified, associated with cases of yersiniosis. On this basis yersiniosis would fall on the boundary between the least severe and moderately severe categories (Appendix 1). Information from the notifiable disease database (Episurv) suggests a higher rate of hospitalization and identified two deaths due to yersiniosis during the period 1997-2002. These data suggest that foodborne yersiniosis should probably be assigned to the moderate severity category (Category 2: Severe outcomes 0.5-5%).

It could be argued that *Y. enterocolitica* in pork should actually be assigned to a higher category for incidence. There are indications that yersiniosis is an under-recognised disease in New Zealand. The notified rate of yersiniosis in New Zealand for the 12 month period to the end of October 2001 was 11.3 cases/100,000 of population (Lopez *et al.*, 2001). The study of Wright (1996) suggests that the actual incidence of yersiniosis may be 6-8 times greater (70-90 cases/100,000).

6.5 Summary

Food/hazard combination	Severity	Incidence	Trade importance	Other considerations
Yersinia enterocolitica in pork	2 (0.5-5% serious outcomes)	3 (1-10 per 100,000)		

7   RISK MANAGEMENT INFORMATION

7.1   Relevant Food Controls

7.1.1 Meat processing

Production and processing of animal material and animal products In New Zealand is regulated under the Animal Products Act 1999 in order to:

- manage associated risks; and
- facilitate overseas market access.

The Animal Products Act requires all animal products traded and used to be "fit for intended purpose". This means they must meet New Zealand animal product standards. This means they must meet New Zealand animal product standards. The New Zealand animal product standards are contained in Part 1 of the Animal Product Regulations 2000.

The Animal Products Act (except for Part 2) and the transitional Act commenced on 1 November 1999. Part 2 of the Animal Products Act commenced on 20 November 2000. Part 2 provides the requirements for risk management programmes that apply anywhere in the value chain from production, through processing, to the market.

The risk management system comprises the following main types of controls:

- risk management programmes;
- regulated control schemes; and
- controls relating to the export of animal material and animal products.

All animal product primary processing businesses, except those exempt under the Act or under the Animal Products (Exemptions and Inclusions) Order 2000, must have a risk management programme.

A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

The application of HACCP based food safety plans is being promoted by the New Zealand Food Safety Authority (NZFSA). The United States (New Zealand's largest beef market) requires that HACCP plans are in place in processing plants, and countries in the European Union also require a partial application of HACCP principles. In addition to the National Microbiological Database that has been established by NZFSA, a separate voluntary testing regime is in place for STEC, principally for exports to the United States.

7.1.2   Pork Quality Improvement Process (PQIP)

The New Zealand Pork Industry Board has led the development of a total quality management programme, the Pork Quality Improvement Process (PQIP), based on HACCP principles (see: http://www.pork.co.nz/pqip/default.asp).

PQIP Standards 03 (Standard for pig killing plants for pork and bacon weight pigs) and 05 (Standard for the New Zealand Fresh Pork Processing Industry) relate to animal processing. In 1997 the Pork Industry Board also produced a video demonstrating recommended hygiene practices for pig slaughter (Begg and Bennett, 1997). As discussed in Section 7.2 below, the level of *Y. enterocolitica* contamination can be significantly influenced by slaughter practices.

The pork industry has also supported development of Module 7 (PQIP 07) in the PQIP Code of Practice. This module provides a basis for the design, implementation and operation of a Food Safety Programme. It has been accepted by the New Zealand Food Safety Authority as providing such a basis for all processed pork products, including uncooked comminuted fermented meats.

7.2   Relevant Food Controls: Overseas

7.2.1   Scandinavia

Nielsen and Wegener (1997) reported that for Denmark "Pork or pork products are assumed to be the major source of human yersiniosis..".   Denmark has implemented a strategy for controlling salmonellosis, and as part of this has focused on critical control points in the pork slaughter line. Two changes to practice have been implemented. Firstly a plastic bag is placed over the anus during processing to prevent the spill of faecal material onto the carcass. Survey work showed that when bags were not used 10% of carcasses were contaminated with *Y. enterocolitica* O:3, but when bags were used the contamination rate was reduced to 0.8%. Secondly, changes have been made so that the pork tongue is removed from the head at a late stage in processing, so minimising any cross contamination that may originate from that source. These changes have occurred at the same time as a significant reduction of disease in that country. In 1985 the incidence was 29 cases/100,000, and in 1995 it was 15 case/100,000. Projections were made that the rate would be 9 in 1996. Later data for 1999 shows a rate of 6.4/100,000 (Anonymous, 1999) and rates have further declined since (see Section 6.1.2).

Other studies have shown that pig carcass contamination with *Y. enterocolitica* can be significantly influenced by slaughterhouse practices. Andersen (1988) took swab samples from the medial hind limb and the split sternum of pigs post slaughter and found that the percentage of carcasses positive for *Y. enterocolitica* was heavily dependent on the method of evisceration. Manual evisceration resulted in the highest rates of contamination (26.3% positive on medial hind limb and 12.9% positive on split sternum). Mechanical evisceration ('bung cutter') resulted in a substantial reduction in contamination rates (13.4% on medial hind leg and 7.4% on split sternum), while a combination of mechanical evisceration and enclosing the rectum in a plastic bag resulted in even lower levels of contamination (3.0% on medial hind limb and 5.6% on split sternum). These results were confirmed by Nesbakken *et al.* (1994) in studies in Norwegian and Swedish slaughterhouses.

A substantial reduction in the incidence of yersiniosis (25%) in Norway occurred after introduction of this technique of covering the rectum with a plastic bag in approximately 90% of Norway's slaughterhouses (Nesbakken and Skjerve, 1996). There is no indication that this technique is employed in New Zealand.

Borch *et al.* (1996) carried out a HACCP analysis of the complete pig slaughter process and identified evisceration, excision of the tongue, pharynx and tonsils, meat inspection and deboning of the head as critical control points with respect to *Y. enterocolitica* contamination. The compulsory veterinary inspection by incision of the submaxillary and mesenteric lymph nodes have been identified as sources of cross contamination by Nesbakken *et al.* (2003).

7.2.2   New Zealand practices in relation to control points

In New Zealand all evisceration of pigs during processing is performed manually, and plastic bags over the rectum are not used. The head of the animal is not removed until late in the process, and keeping the tonsils intact has been identified as a critical control point in minimising bacterial contamination.

In New Zealand the first processing step is to put the carcass into a tub of warm water to soften the hair, followed by a dehairing tumbler. The next stage involves removing the remainder of the hair by singeing. This practice has been shown to also have a positive effect in controlling bacteria, especially around the animal anus, in a study conducted by the Pork Industry Board (Frances Clement, pers. comm, 2004).

7.3   Economic Costs

The annual economic cost to New Zealand of cases of yersiniosis caused by foodborne transmission has been estimated as $2,037,000, which represents 3.7% of the estimated total cost of foodborne infectious intestinal disease (Scott *et al.*, 2000). The number of cases and outcomes used for this estimate were based on an average of notification and hospitalisation data from 1991 to 1998 (Lake *et al.*, 2000). This estimate was based on several assumptions, the most important of which was that 65% of all cases of yersiniosis were caused by foodborne transmission. The estimated value includes direct and indirect medical costs, the value of productive days lost, and the statistical value of mortality, but not the value of lost quality of life.

In this study (Lake *et al.*, 2000) the ratio of reported:unreported cases of 5:1 was derived from the mid-range of the same ratios for campylobacteriosis and salmonellosis found in a prospective UK study of infectious intestinal disease (Wheeler *et al.*, 1999). The results of the study in the Eastern Bay of Plenty (Wright, 1996) (see Section 6.1.1) suggest that this ratio is too conservative.

This estimate covers all potential food vehicles. No data are available on the proportion of transmission due to pork and pork products alone.

8   CONCLUSIONS

8.1   Description of Risks to New Zealand Consumers

8.1.1   Risks associated with pork

The rate of notified cases of yersiniosis in New Zealand is markedly higher than that in Australia, the USA, and the UK. Although there may be differences in reporting systems, the ten fold difference is unlikely to be due to that alone. There are studies that suggest that yersiniosis is an under-recognised disease in New Zealand, although the magnitude of the currently notified rate is sufficient cause for further investigating potential sources of infection..

There is evidence to indicate that a minor proportion of *Y. enterocolitica* intestinal infections are caused by transmission in pork, but the information available does not suggest that this vehicle is the cause of the difference in incidence of infections between New Zealand and, for example, Australia.

There are two pork related food consumption patterns that emerge from the literature as important in yersiniosis. Firstly there is the consumption of raw pork in Belgium, and the fact that raw pork is used to feed young children in that country (Tauxe *et al.,* 1987). Secondly there is the association between chitterlings preparation and yersiniosis in African American children in the USA. Neither seems likely to be important in New Zealand.

Effective cooking and pasteurisation will eliminate *Y. enterocolitica* from foods. However there is still the potential for cross contamination from uncooked foods (especially meats) to other foods which are then not cooked before consumption. The identification of food from a sandwich bar as a statistically significant exposure in the case control study suggests that this might be a route for transmission of yersiniosis.

There are indications from outbreaks and the case control study that pork is involved in the transmission of a proportion of the yersiniosis cases in New Zealand, and this proportion is apparently small. This risk assessment would be considerably strengthened by information on the prevalence of pathogenic *Y. enterocolitica* in pigs at slaughter and in retail pork products. However, there are some methodological hurdles to be overcome before survey work can be undertaken.

If it is assumed that *Y. enterocolitica* infections are associated with pork consumption, then religious groups such as Jews and Muslims who strictly adhere to pork-exclusive food laws should demonstrate a much lower incidence than the pork-eating portion of the population. Data testing this theory have not been located.

8.1.2   Risks associated with other foods

In North America outbreaks of yersiniosis have been associated with raw milk, pasteurised milk, chocolate milk, reconstituted powdered milk, bean curd and bean sprouts (Tauxe *et al.*, 1987; Satterthwaite *et al.*, 1999).

It is possible that some as yet unidentified, food preparation or consumption practice occurs in New Zealand that could explain the higher rate of yersiniosis here compared to other countries. The high documented prevalence of pathogenic strains of *Y. enterocolitica* in New Zealand lambs and to a lesser extent cattle (Bullians, 1987) suggests that the potential of these species to contribute to exposure through animal contact and/or meat consumption in New Zealand needs to be further investigated.

Untreated water, unreticulated sewage and handling farm animals have been identified as other risk factors in studies in New Zealand.

8.1.3   Quantitative risk assessment

There is insufficient information available at present to undertake a quantitative risk assessment for this food/hazard combination. Better information showing the potential for transmission of *Y. enterocolitica* in retail pork products would be needed before undertaking a full risk assessment.

8.2   Commentary on Risk Management Options

The slaughter process modifications made in Denmark and Norway appear to have effectively reduced both contamination rates and the incidence of human cases of yersiniosis. These practices are not standard practice in New Zealand, but the demonstration of significant *Y. enterocolitica* contamination on carcasses would be needed to support introduction of such measures.

8.3   Data gaps

The data gaps identified in this Risk Profile are:

- Prevalence and numbers of pathogenic *Y. enterocolitica* contamination on pig carcasses after slaughter;
- Prevalence and numbers of pathogenic *Y. enterocolitica* contamination on retail pork products;
- Equivalent prevalence and enumeration information on pathogenic *Y. enterocolitica* in other farmed animal species, to provide context to pig/pork data;
- Dose response data.

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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