



**RISK PROFILE:
SALMONELLA (NON TYPHOIDAL)
IN
CEREAL GRAINS**

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by

Susan Gilbert
Dr Rob Lake
Peter Cressey
Nicola King

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RISK PROFILE:
***SALMONELLA* (NON TYPHOIDAL)**
IN
CEREAL GRAINS

Dr Stephen On
Food Safety Programme Leader

Dr Rob Lake
Project Leader

Dr Andrew Hudson
Peer Reviewer

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SUMMARY

The purpose of a Risk Profile is to provide 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.

The food/hazard combination addressed by this Risk Profile is *Salmonella* (non-typhoidal) in cereal grains. The cereals considered are wheat, rice, maize, barley, rye, oats, sorghum, millet and triticale, consumed directly as cereal grains (dried and/or cooked) or as primary processed products, such as flour. In New Zealand and internationally, wheat, maize and rice are the cereals consumed in the greatest amounts. New Zealand imports all its rice (principally from Australia and Thailand) and approximately one third of its wheat (mainly from Australia).

As raw materials, cereals may be contaminated with *Salmonella* from animal or human faecal material. After harvest, rodents and birds are particularly important sources, if adequate storage is not maintained. Due to the low water activity of cereals and their milled products, growth of *Salmonella* does not occur, but the bacteria remain viable for long periods (months). The low water activity of cereal and cereal products also promotes heat resistance in *Salmonella*.

Cereals and their products are consumed by almost all New Zealanders on a daily basis. Most will be consumed in forms that have been rendered *Salmonella*-free through processing. Any residual risk will come from consumption of minimally processed foods that allow the bacteria to survive. In some cases growth may be possible following addition of hydrating ingredients such as water or milk. Examples of such foods include cereal based infant foods, uncooked breakfast cereals (e.g. muesli), or products designed to be baked at home (e.g. cookie dough). Outbreaks have been linked to these foods overseas. Ingestion of raw flour may also occur during home baking or activities with homemade play-dough.

The potential for exposure to *Salmonella* in raw flour by eating uncooked baking mixture has been demonstrated by a significant outbreak of salmonellosis linked to contaminated flour in New Zealand. The samples of flour tested in this outbreak investigation had low counts of *Salmonella*. Assuming that these samples were representative of the material causing disease and it was raw flour that was consumed (i.e. through home baking), then this outbreak points to a high risk of illness from consuming relatively few cells. However, the possibility that much higher counts of *Salmonella* were present in the actual flour that was ingested cannot be excluded (e.g. if the distribution of contamination was not homogenous).

Although there are no New Zealand data, surveys in Australia and North America have found prevalence of *Salmonella* contamination in wheat flour in the range 0.0-1.3%. There is even less information available on the prevalence of *Salmonella* on other cereal grains, but it appears likely to be similarly low. There is almost no information on the concentration of *Salmonella* on cereal grains or in milled products. However, flour samples analysed in association with the recent New Zealand outbreak contained very low concentrations of *Salmonella*. Exposure events whereby consumers are exposed to cereal grains or milled products, without a further bactericidal step, are likely to be uncommon (consumption of uncooked dough or batter, Bircher muesli consumption, infant cereal consumption).

Overall, the risk of human salmonellosis due to contaminated cereal grains must be classified as low. However, the outbreak linked to flour indicates that when cereal contamination occurs it has the potential to affect large numbers of people, even if potential exposures occur via specialised behaviours (e.g. ingestion of uncooked home baking materials) or less common foods (e.g. uncooked muesli ingredients).

Due to the fact that cereal grains have not often been considered as a cause of human salmonellosis a number of significant data gaps exist, including:

- Information on the actual routes of introduction of *Salmonella* into cereal grains;
- Data on the prevalence of *Salmonella* in cereal grains in New Zealand, either domestically produced or imported, and serotypes present;
- Data on the concentration of *Salmonella* in cereal grains, prior to and following primary processing;
- Frequency of consumption and serving sizes of potential risk foods (e.g. uncooked batter or dough, Bircher-style muesli, cereal-based weaning foods);
- Data on concentration of *Salmonella* in risk foods at consumption; and
- Dose-response for *Salmonella* from cereal grains.

However, cereal grains are likely to be infrequently contaminated with *Salmonella* and a survey to generate such data to fill these data gaps would need to test very large numbers of samples. A more effective approach to assessing the risks associated with this food/hazard combination may be to assess potential sources of *Salmonella* contamination of cereal grains and the current controls.

It is uncertain whether the outbreak where flour was identified as the vehicle was caused by contamination prior to or during harvest or at the flourmill. A number of hazard controls exist in the cereal growing and processing industries that will reduce the likelihood of *Salmonella* contamination (e.g. the New Zealand Crop Quality Assurance Scheme). However, no information is available on the effectiveness of these controls.

Risk communication regarding the consumption of uncooked flour products (e.g. cake batter, cookie dough) may be warranted, given the recent outbreak. Such communications might also address the possibility of home made play-dough /raw flour being consumed during play.

1 STATEMENT OF PURPOSE

The purpose of a Risk Profile is to provide information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles are part of the Risk Management Framework (RMF)¹ approach taken by the New Zealand Food Safety Authority (NZFSA). The Framework consists of a four step process, as shown in Figure 1.

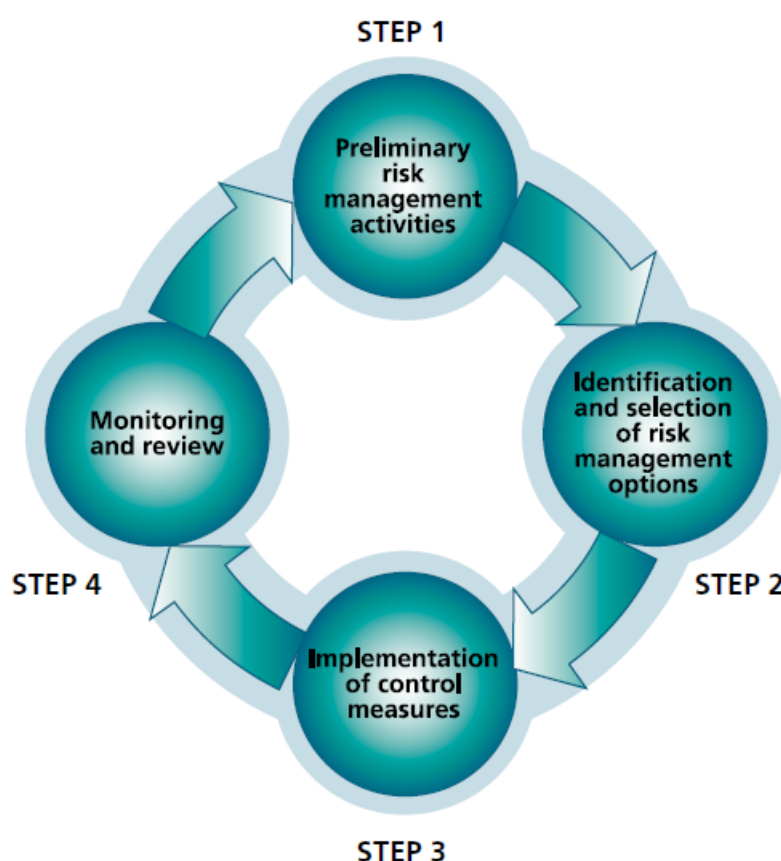


Figure 1: The four steps of the Risk Management Framework

This initial step in the RMF, Preliminary Risk Management Activities, includes a number of tasks:

- Identification of food safety issues
- Risk profiling
- Establishing broad risk management goals
- Deciding on the need for a risk assessment
- If needed, setting risk assessment policy and commissioning of the risk assessment
- Considering the results of the risk assessment
- Ranking and prioritisation of the food safety issue for risk management action.

¹ <http://www.nzfsa.govt.nz/about-us/risk-management-framework/index.htm>

Risk profiling may be used directly by risk managers to guide identification and selection of risk management options, for example where:

- Rapid action is needed
- There is sufficient scientific information for action
- Embarking on a risk assessment is impractical.

1.1 Food/ Hazard Combination and Risk Management Questions

The food/hazard combination addressed by this Risk Profile is *Salmonella* (non-typhoidal) in cereal grains and their products.

NZFSA has recognised non-typhoidal *Salmonella* as one of the three most important foodborne pathogens in New Zealand. The organisation is taking a strategic approach to *Salmonella* Risk Management, with the ultimate aim of achieving a 30% reduction in foodborne salmonellosis after five years¹. Underpinning this strategy are a range of preliminary risk evaluation activities, including risk profiling to better understand the risk of *Salmonella* attributable to a range of food types.

NZFSA have commissioned this Risk Profile in order to address the following specific risk management questions:

- What are the different contamination pathways for *Salmonella* on to cereal grains (domestic and imported) during primary production and what possible controls may be applied?
- What is the applicability of the New Zealand Crop Assurance Scheme to *Salmonella* control in cereal grains?
- What is the public health risk of salmonellosis associated with the consumption of cereal grains in New Zealand?

¹ NZFSA *Salmonella* Risk Management Strategy 2009-12, <http://www.nzfsa.govt.nz/foodborne-illness/salmonella/strategy/salmonella-risk-management-strategy-2009-2012.pdf>

2 HAZARD AND FOOD

2.1 *Salmonella*

This group of bacteria is comprised of two species: *Salmonella enterica*, which is divided into 6 subspecies (*enterica*, *salamae*, *arizonae*, *diarizonae*, *houtanae* and *indica*), and *Salmonella bongori* (Jay *et al.*, 1997). Most pathogenic isolates from humans and other mammals belong to *Salmonella enterica* subspecies *enterica*. Other *Salmonella enterica* subspecies and *Salmonella bongori* are more common in cold blooded animals and the environment, and are of lower pathogenicity to humans and livestock (Jay *et al.*, 1997).

Salmonella typing is primarily performed using serological identification of somatic (O), flagella (H), and capsular (K) antigens. There are more than 2,400 different *Salmonella* serotypes.

Salmonella enterica serotypes are normally denoted in a shortened form that includes a non-italicised serotype name, e.g. *Salmonella enterica* subsp. *enterica* serotype Enteritidis becomes *Salmonella* Enteritidis. In older publications this may be represented as a full species name i.e. *Salmonella enteritidis*.

Further subtyping may be performed using susceptibility to bacteriophages. These types are denoted as phage type (PT) or definitive phage type (DT) numbers. These two terms are interchangeable and both are used in the literature.

Salmonella Typhi and *Salmonella* Paratyphi are serotypes which cause a serious enteric fever and are particularly well adapted to invasion and survival in human tissue. They have a particular antigen makeup and differing ecology to other serotypes of *Salmonella*. *Salmonella* cholerae-suis (SCS) is a typhi-like serotype that infects pigs. SCS is only found in a few countries, excluding New Zealand, and has a distinct pathogenic profile. This Risk Profile does not include these human and porcine typhoidal serotypes.

Information on the behaviour of *Salmonella* in foods is given in Appendix 1.

2.2 Sources of *Salmonella*

Human: Person to person transmission of *Salmonella* is well recognised, and secondary transmission of *Salmonella* in outbreaks has been demonstrated (Loewenstein, 1975). Carriage in faeces in convalescent cases can be quite substantial with numbers approximating 10^6 - 10^7 salmonellae/g persisting up to 10 days after initial diagnosis. Reduction in numbers with time is variable; most people will have counts of less than 100 salmonellae/g after 35 to 40 days, but a count of 6×10^3 /g has been recorded in one patient 48 days post-illness (Pether and Scott, 1982).

Animal: *Salmonella* can be found in mammals, fish, reptiles, amphibians, insects and birds. Most *Salmonella* colonisations in animals produce no clinical signs. Some serotypes are largely confined to particular animal reservoirs causing both systemic and enteric disease, for example *S. cholerae-suis* is host restricted to pigs (Allison *et al.*, 1969) while other serotypes (for example *S. Typhimurium*) are frequently associated with intestinal infections in a wide range of phylogenetically unrelated species (Paulin *et al.*, 2002). Both plant and animal product-based animal feed ingredients may be contaminated with salmonellae.

Food: Red and white meats, meat products, milk, cheese and eggs are considered the major food sources of human salmonellosis, although a wide variety of other foods have been associated with outbreaks (Jay *et al.*, 2003). Other foods that have been contaminated by *Salmonella* include seafood (shellfish, salmon), nuts and nut products (desiccated coconut, peanut butter), cereal and cereal products (barley, cereal powder), spices (white and black pepper, paprika), oilseeds and oilseed products (cottonseed, soybean sauce, sesame seeds), vegetables (watercress, tomatoes, lettuce, potato and other salads, bean sprouts), fruit and fruit products (watermelon, melon, cider) and other miscellaneous products (chocolate, cocoa powder, dried yeast, candy). The *S. Enteritidis* types that are capable of transovarian transmission into eggs are not endemic in New Zealand so this food type is likely to be of lower risk here (Lake *et al.*, 2004a). Tahini, a product made from crushed sesame seeds, has been contaminated with *Salmonella* and caused a number of outbreaks worldwide, including New Zealand and Australia (Unicomb *et al.*, 2005).

Environment: Salmonellae in sewage effluents or animal faeces can contaminate pasture, soil and water. They can remain viable for months in soil. The organism may also be dispersed in dust and aerosols generated during the handling and processing of animals. Contamination in the environment can be spread by rodents or wild bird populations and act as a source of infection for other animals.

Transmission routes: Salmonellae may be transmitted to humans via person to person transmission, contaminated food or water, animal contact or from a contaminated environment. The faecal-oral route is the most common.

2.3 The Food Supply in New Zealand: Cereal Grains

The cereals addressed by this Risk Profile are: wheat, rice, maize, barley, rye, oats, sorghum, millet and triticale. Regionally specific cereals, such as fonio (*Digitaria* spp.) and teff (*Eragrostis tef*), and the pseudocereals buckwheat (*Fagopyrum esculentum*) and quinoa (*Chenopodium quinoa*) are not included in this risk profile.

The main cereals grown for human consumption are wheat (either hexaploid bread wheat, *Triticum aestivum*, or tetraploid durum or pasta wheat, *Triticum durum*), rice (*Oryza sativa*) and maize (*Zea mays*). New Zealanders also consume lesser amounts of barley (*Hordeum vulgares*), rye (*Secale cereale*) and oats (*Avena sativa*). Other minor cereals (by consumption) are sorghum (*Sorghum bicolor*), millet (varieties of small-grained grasses) and triticale (a hybrid of tetraploid wheat and rye).

All the cereal grains included in the current risk profile have similar proximate composition, with low moisture content (8-14%), moderate protein content (7-17%) and low, but variable fat content (0.7-7%). All cereals are characterized by a high content of complex carbohydrates (starch; 68-80%). Removal of the outer grain layers through processes such as flour milling, rice polishing or barley pearling, will tend to decrease the protein and fat content of the cereal and increase the proportion of starch present.

The relevant forms of these cereals are those consumed directly as cereal grains (dried and/or cooked) or as direct mill products (flour, meal, rolled oats). Fermented products (e.g. beer) are excluded because fermentation and other processing (e.g. filtration) would be expected to

eliminate *Salmonella*. Further processed cereal products, such as bread, pasta, noodles, pastries and other baked goods are also excluded.

Supplemental information on cereals is given in Appendix 1.

2.3.1 Domestic cereal grain production

Rice, rye, sorghum and millet are not grown as commercial crops in New Zealand.

The New Zealand cereal industry is relatively small and focussed in the Canterbury District. Cereal production in 2009 was wheat (408,400 tonnes), oats (41,600 tonnes), barley (449,800 tonnes) and maize (257,000 tonnes)¹.

The majority of oats, barley and maize produced in New Zealand are used for animal feed (Armstrong *et al.*, 2004; Booker, 2009; Crop & Food Research, 2008). Significant amounts of barley (25% of production) are malted for use in beer production (Crop & Food Research, 2008).

Until the deregulation of the wheat and flour milling industries in 1987, New Zealand aimed to be self-sufficient with respect to the production and processing of milling grade wheat (Ali, 1994). From 1962 the central instrument of the regulated market was the New Zealand Wheat Board. Deregulation led to a decline in wheat growing in New Zealand and significant consolidation in the wheat and flour processing industries, particularly in the flour milling industry. There were 18 flour mills in New Zealand in 1983 (Ali, 1994). In 2010, there are six mills producing flour for human consumption and these are operated by three companies (Weston Milling, Champion Flour Mills, Milligans Food Group)².

2.3.2 Imported cereal grains

Importations of cereals and cereal products into New Zealand during the 2009 year are shown in Table 1¹. Only products with import volumes greater than 1,000 tonnes are shown. Wheat for milling contributes the greatest volume of imported cereals.

Table 1: Cereal grains and cereal grain products imported into New Zealand during the 2009 year, by HS10 descriptor

HS10 Descriptor*	Tonnes	Major countries of origin
Cereals; meslin and wheat other than durum	257,185	Australia (>99%)
Wheat or meslin flour	13,207	Australia (85%), Thailand (8%)
Cereal groats and meal; of wheat	1,540	Australia (99%)
Cereal grains; rolled or flaked; of oats	3,973	Australia (97%)
Cereal flour; of maize (corn)	1,826	USA (52%), Australia (44%)
Cereal groats and meal; of maize (corn)	1,066	Australia (90%)

¹ Source: Statistics NZ, website www.stats.govt.nz, accessed 5 March 2010

² Source: New Zealand Flour Millers Association website <http://www.flourinfo.co.nz/default.asp?contentID=509>, accessed 22 March 2010

HS10 Descriptor*	Tonnes	Major countries of origin
Cereals; husked (brown) rice	1,121	Australia (69%), Thailand (18%)
Cereals; rice, semi-milled or wholly milled, whether or not polished or glazed	38,828	Thailand (43%), Australia (16%), USA (16%)
Cereals; rice, broken	2,034	Thailand (53%), USA (14%), Pakistan (11%), India (10%)
Cereal flour; of rice	2,117	Thailand (56%), Australia (16%)
Cereals; grain sorghum	38,940	Australia (>99%)

HS = Harmonised System

* Only HS codes with import volumes of greater than 1,000 tonnes have been included

2.3.3 Sources of contamination of cereal grains by *Salmonella*

While much of the material in following sections refers predominantly to wheat, observations are likely to be applicable to all cereal grains. The structure of the cereal plant, the growing process, the proximate composition of the grains and primary processing are sufficiently similar for all cereals to make sources of contamination and the behaviour of the organism in the food similar for all cereal grains.

When correctly handled and stored, *Salmonella* cannot grow on dry cereal grain but, if present, can remain viable. Contamination can occur:

- In the field (pre-harvest);
- In transport containers previously used for animals or their products;
- On site in the mill, from machinery and environment; and
- From human workers in the production chain.

2.3.3.1 Preharvest

Because salmonellae are primarily transported in human and animal faecal matter, contamination of field crops will be low unless there is considerable exposure to animal or human activity. Cereal crops have the potential to become contaminated through direct deposition of *Salmonella*-containing animal faeces or through deposition of soil or dust previously contaminated with animal faecal material. Spray irrigation of farm effluent is also practiced in New Zealand and this provides a potential route for contamination of adjacent cereal crops by animal faecal material. However, the edible grain of cereal crops is enclosed within an outer casing (husk, glume, etc.) until harvest, that may protect the grain against direct deposition.

Experiments have demonstrated that *Salmonella* is able to colonise and spread through the inside of a plant. Surface root rhizospheres of barley plants were inoculated with two strains of *S. Typhimurium* (10^8 CFU/ml). After two weeks both *Salmonella* strains were present inside the roots at over 10^6 CFU/g fresh root weight and both strains were repeatedly found in the intercellular space of the root cortex (Kutter *et al.*, 2006). However, this study did not determine whether *Salmonella* could be detected in grain from inoculated plants.

Salmonellae are able to survive in soils for extended periods following animal defecation or application of human or animal waste to land (Thomason *et al.*, 1975; Zibilske and Weaver, 1978). Persistence of salmonellae in acid soils is facilitated by their ability to adapt to low pH environments (Foster, 1995). There is also some evidence that salmonellae may survive in soils in a viable but non-culturable state (Turpin *et al.*, 1993).

2.3.3.2 Post-harvest

Transport vessels, containers and storage areas

In addition to contamination in the field, contamination could also occur at a number of steps during transport and storage. These risks include;

- Unclean ship/vehicle/container holds, due to previous cargoes, water leakages, condensation, birds and vermin;
- Loading of vessels' holds in the open;
- Access by birds and vermin to storage facilities, contaminating handling equipment, conveyor belts, condensation, water leakages; and
- Personnel.

There is a wide range of animals that may come in contact with stored cereal grains on the farm or during storage at the mill. *Salmonella* has been isolated from a number of animals common in the farm environment, including mice (Henzler and Opitz, 1992; Singer *et al.*, 1992; Weigel *et al.*, 2007; Whyte *et al.*, 2003), rats (Kinde *et al.*, 2005; Schnurrenberger *et al.*, 1968), wild birds (Craven *et al.*, 2000; Davies and Wray, 1996; Pangloli *et al.*, 2008; Pennycott *et al.*, 2006; Weigel *et al.*, 2007), insects (Hazeleger *et al.*, 2008; Kinde *et al.*, 2005; Pangloli *et al.*, 2008; Weigel *et al.*, 2007) and larger mammals (e.g. cats, raccoons, opossums) (Schnurrenberger *et al.*, 1968; Weigel *et al.*, 2007).

Mill environment

Depending on the cereal and intended end product, milling can involve removal of debris and extraneous material, spraying with water and tempering (conditioning) to adjust water levels, removal of the bran, removal of the germ and grinding into flour, meal or grits (ICMSF, 1996).

Screening and tempering

Grains are screened before tempering in order to remove extraneous material ('screenings' e.g. stones, husks, weed seeds). Tempering or conditioning is a vital step in the process because dry grains are too brittle for milling. Water is added (by careful measurement) usually in a fine spray, increasing the moisture content (for wheat from 8-12% to 14-15% (a_w 0.68-0.70)). The grain is rested to allow even penetration (usually overnight, sometimes 24-36 hours depending on wheat type and initial moisture content). This makes separation of the grain constituents easier by toughening the outer bran layer to prevent it from disintegrating during milling, softening the endosperm ready for grinding, and making the germ pliable so the rollers readily flatten and release it from the kernel (Berghofer *et al.*, 2003; Boyacioglu and Sunter, 2004; Estrada-Girón *et al.*, 2005). Maize can be ground without the tempering step (ICMSF, 1996).

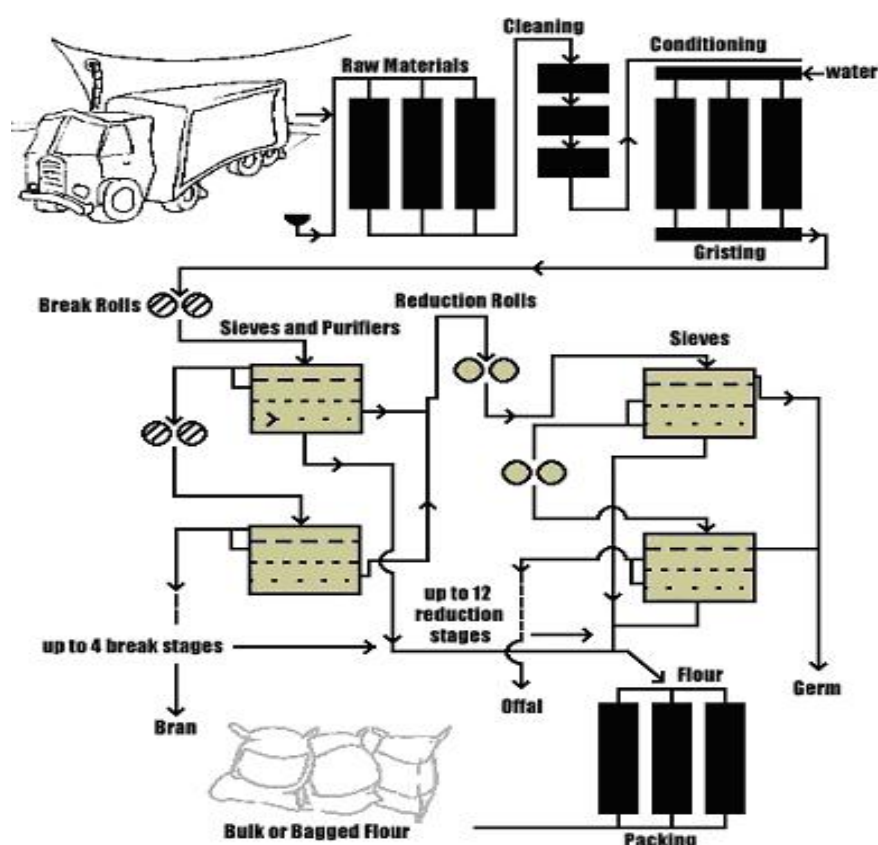
Screening reduces microbial loading so that sound clean grains, properly screened, contain few micro-organisms (ICMSF, 1996). However, conditioning often increases microbial counts

(Berghofer *et al.*, 2000). It was suggested that this was due to contamination from bins and augurs used for handling the grain, rather than from growth during conditioning.

Milling and sifting

Milling and sifting separates the grain into bran (hulls), germ and endosperm. The objective is to maximise white flour production with as little contaminating bran or germ as possible. Bran is unwanted due to its texture and colour while germ is high in fat reducing the shelf life of the flour (New Zealand Flour Millers Association, 2009). The grain is passed between five or six break rolls which have a shearing action. After each break roll the sheared particles are passed through sieves, progressing from the coarsest to the finest. The germ is flattened and separated out after the third break and the larger particles on the top sieve after the fifth or sixth break are primarily bran. Separated endosperm is passed through a series of reduction rolls that are smooth and have a pulverizing effect, and in between these the pulverized endosperm is further sifted to separate flour from coarser particles that need further reduction. The fine endosperm flour is called millstream while the screened out mixture of bran, germ and some endosperm is called mill feed. Large mills may have 30-40 millstreams with varying characteristics to produce different grades of flour (Estrada-Girón *et al.*, 2005). A diagram of the process is provided in Figure 2.

Figure 2: The process for milling wheat



Kind permission for replication from NZ Flour Millers Association (2009)

As the wheat grain layers are separated, surface-adhering contaminants are concentrated into the bran and germ. Consequently, the inner endosperm contains lower microbial counts and the crushed refined flour is the “cleanest” end product (Berghofer *et al.*, 2003). Berghofer *et al*

described how the microbial quality of incoming wheat strongly influenced the final microbial quality of end products, but some higher microbial results midstream in the milling process indicated equipment contamination (Berghofer *et al.*, 2003). If flour is contaminated, the dilution effect in the bulk product is believed to reduce its concentration.

Storage

Moisture

The critical target moisture content for flour and maize meal is 12% (or less), at which point it is stable because microbial growth (including that of spoilage fungi) is not supported at this level (Hesseltine and Graves, 1966). Water can potentially enter the flour product from a number of sources, including condensates on equipment, high atmospheric humidity and improper cleaning procedures. The very nature of the grinding and sifting generates considerable heat that contributes to the condensation on the equipment.

Insects

Several researchers have reported the carriage of *Salmonella* by insects, and contamination can occur at any point from in the field to the domestic kitchen.

Transmission of *S. Montevideo* from contaminated to clean wheat by rice weevil has been demonstrated (Husted *et al.*, 1969). Contamination of clean wheat by the weevils was greater after the weevils had been exposed to wheat contaminated by *S. Montevideo* for 14-21 days. The weevils retained the pathogen internally and externally for at least one week.

Similarly, *S. Montevideo* was transferred from contaminated wheat to clean wheat by granary weevils, rice weevils, saw-toothed flour beetles, red flour beetles, lesser grain borers, cadelle and the flat grain beetles (Crumrine *et al.*, 1971). There was no subsequent carriage to second and third samples of clean wheat, which suggests that the number of *Salmonella* cells carried by the insects may have been low. In addition, the progeny of insects developed in contaminated wheat had reduced abilities to transmit *S. Montevideo* from contaminated to clean wheat, except for the progeny of rice weevils, saw-toothed grain beetles and red flour beetles.

2.3.3.3 General controls

A number of measures have been suggested to minimise microbial contamination of grain and grain products:

- Chlorinated water used for conditioning;
- Regular cleaning of milling plant and equipment to prevent accumulation of material (prevents moulds and insect harbourage). Waterless cleaning of all dry product areas;
- Programme of bird, rodent and insect control;
- Avoiding condensation in the plant, particularly above areas where water can fall into product or on surfaces that come into contact with product;
- Immediate removal of dead insects after fumigation of areas such as boots and elevators (ICMSF, 1996).

2.3.4 Behaviour of *Salmonella* in cereal grains

2.3.4.1 Unprocessed cereal grains

Cereal grains may carry the viable cells of many pathogens if the grain is exposed to animal or human contamination, e.g. animal contact in the field, transport vehicles also used to carry animals or animal products, or contamination by insects, mice, rats and birds (ICMSF, 2005).

Most cereal grains are allowed to dry in the field before harvesting, with moisture content of less than 14% usually achieved. The exception is rice, which is harvested at 20-24% moisture and then dried to below 14% moisture before storage (ICMSF, 2005). On occasions, weather conditions will require other cereal grains to be harvested at moisture contents greater than 14%, requiring mechanical drying before storage. The low moisture content of dry cereal grains prevents microbial growth. Typical moisture contents for unprocessed cereal grains are: wheat (hard) 13%, maize 13.8%, rice (brown) 12%, oats (rolled) 8.3% and rye 11% (ICMSF, 2005). A cereal moisture content of 15% approximates to a water activity of 0.70 (ICMSF, 2005). No bacteria and few fungi will grow at water activities below 0.70 (ICMSF, 2005).

Under the low water activity conditions of dry cereal grains salmonellae can remain inactive but viable, with numbers only decreasing slowly over time (ICMSF, 2005). This is consistent with the general behaviour of the organism in low water activity foods. Survival for periods of greater than a year has been reported in some such foods (ICMSF, 1996).

As water availability increases, *Salmonella* on unprocessed cereal grains appear to decline rather than grow. A study of *S. Montevideo* on wheat stored at various constant relative humidities (RH) has demonstrated that survival declined with increasing RH (Crumrine and Foltz, 1969). Samples of Ottawa red winter wheat were inoculated at 10^6 CFU/g and held in chambers (25°C, RH range from 7 to 98%) over a 28-week sampling period. After 28 weeks at RH between 7 and 22% the counts of *S. Montevideo* reduced to 10^4 CFU/g, while in samples stored at 33-62% RH, counts declined to $10^2 - 10^4$ CFU/g, and at 75% RH or higher almost no salmonellae were recovered from the samples. This is consistent with finding in animal feeds with high cereal content, where studies with *S. Montevideo* and *S. Heidelberg* in poultry feed demonstrated more rapid die off at higher water activities ($a_w = 0.75$) than lower water activities ($a_w = 0.43$ or 0.52) (Juven *et al.*, 1984).

2.3.4.2 Flour or meal

All cereal grains may be milled to produce flour or meal. Depending on the milling process the product may contain the entire grain contents (wholemeal) or predominantly the starchy endosperm (white flour). The milled product is amenable to uptake of water and shaping; characteristics that are important for production of simple foods (gruels, grits and porridges) or processed foods (see next section). The milling process varies with the type of cereal grain and the intended end use (ICMSF, 2005).

Salmonella present on cereal grains will be distributed throughout the flour during milling. *Salmonella* can survive in flour and are remarkably heat resistant. This has been attributed to the low water activity.

The survival of *S. Weltevreden* inoculated into autoclaved plain wheat flour and heated in hot air has been reported (Archer *et al.*, 1998). Samples of flour (a_w 0.2-0.6) were inoculated and

equilibrated at a variety of relative humidities (6% to 35%) prior to heating. Dry heating was conducted at temperatures from 57°C to 77°C. Death curves were biphasic with an initial rapid decline ($>1 \log_{10}$ CFU/g) coinciding with rapid decreases in water activity. Irrespective of the initial a_w value, all samples showed a decrease to $a_w < 0.2$ during the initial 5 to 10 minutes of heating as most of the water evaporated. A linear survival curve (from which D-values were calculated) followed, indicating slower inactivation¹. D-values ranged from 29 to 875 minutes in this second phase. The z values ranged from 15.2 to 53.9°C, compared to a typical value of 5.7°C for *Salmonella* in a moist environment².

The time needed to reduce populations of eight *Salmonella* serotypes inoculated into corn flour (15% moisture) by $2 \log_{10}$ CFU/g (10^5 /g to 10^3 /g) during heating at 49°C has been reported (VanCauwenberge *et al.*, 1981). The time ranged from 0.8 to 6.7 hours, depending on the serotype. *S. Thompson* (4h) and *S. Tennessee* (6.7h) were the most heat resistant, and *S. Newington* (0.8h) and *S. Typhimurium* (1.0h) were the most sensitive. The reduction of *S. Senftenberg* in corn flour at 10% moisture held at 49°C was also determined. At 15% moisture, the $2 \log_{10}$ CFU/g reduction took 2.2 hours while at 10% moisture it took 5.8 hours. The reduction time was much longer (over 80 hours) in equivalent controls held at ambient temperature (25°C).

2.3.4.3 Processed foods

The majority of processed foods produced from cereal flour involve the addition of water and other ingredients to form a dough or batter that is then baked (e.g. bread, biscuits, cakes, pastries), dried (e.g. pasta) or extruded (e.g. snack foods). While survival under the low moisture conditions of harvested grain and milled flour has the potential to increase the heat resistance of *Salmonella*, the thermal processing applied to produce most finished cereal products are likely to destroy any organisms present. For example, bread and baked goods are typically cooked at temperatures of 175°C or greater, while products such as porridges/gruels or pasta are boiled, which involves high water activity and temperatures of 100°C. The rest of this section will focus on cereal foods that have the potential to be consumed in minimally processed forms, without any further bactericidal treatment.

The manufacture of dried breakfast cereal products (flakes, puffs or extrusions) involves the addition of water to process the cereals, followed by heat treatment then addition of vitamins, sweeteners, colourants, etc. *Salmonellae* may be present in the final dry product, though the low water activity prevents growth (ICMSF, 2005). The behaviour of *S. Enteritidis* on commercial breakfast cereals (corn flakes, brown rice flakes and wheat bran flakes), with and without milk added, has been investigated (Ui *et al.*, 2009). Dry flakes were inoculated ($6 \log_{10}$ CFU/g) and stored 56 days at ambient temperature. Numbers of the pathogen rapidly declined and after 14 days *S. Enteritidis* were only detectable using enrichment methods. Despite being reduced to low numbers, the remaining salmonellae grew after the addition of milk, reaching approximately $4 \log_{10}$ CFU/g after 5 hours. People usually consume these products straight after adding milk so if present, growth of *Salmonella* will not normally be an issue. The exception to these comments is muesli, particularly Bircher muesli. Bircher muesli may contain minimally processed cereal grains (rolled oats) and may be soaked in water or milk overnight before consumption.

¹ In microbiological terms “D” refers to a 90% (a decimal or $1 \log_{10}$ cycle) reduction in the number of organisms.

² In microbiological terms “z” refers to the temperature required to reduce the number of organisms by 90% (a decimal or $1 \log_{10}$ cycle).

It has been claimed that cereal-based infant foods may contain dormant *Salmonella* cells, in addition to the risk of cross contamination during preparation (Abushelaibi *et al.*, 2003). In New Zealand, these foods typically contain rice flour, oatmeal, maize flour, wheat flour or barley meal and come ready-to-feed or are prepared in the home to a porridge-like consistency. They are generally fed after addition of milk or water and without heating, to infants recently weaned (typically 6-12 months of age). Two outbreaks have occurred where contaminated infant foods were implicated (Rushdy *et al.*, 1998; Silverstolpe *et al.*, 1961). A mixture of four *Salmonella* strains were inoculated into infant cereals (rice, oatmeal and mixed) that had been hydrated with water, milk or apple juice (Abushelaibi *et al.*, 2003). A variety of incubation temperatures and times were used to simulate home preparations. *Salmonella* did not grow or decline in any hydrated cereal stored at 4°C for 24 hours. At 15°C and 25°C, numbers increased in samples hydrated with water or milk. However, cereals hydrated with the more acidic apple juice were less supportive of *Salmonella* growth, particularly rice cereal. The authors concluded that hydrated infant cereals should be consumed immediately after preparation or held at 4°C for less than 8 hours to reduce potential risks from *Salmonella*.

The water activity of raw microwaveable and conventional popcorn kernels is between 0.86 and 0.89. As popcorn kernels heat up, water inside expands and pressure builds against the hard outer surface. The kernel then pops itself inside out. Anaya *et al.* assessed the viability of *Salmonella* Typhimurium DT 4,12:i:1,2 in artificially-contaminated popcorn cooked conventionally in a pan (final internal temperature 110°C, 4 minutes total) and in a microwave oven (130°C, 3 minutes total) (Anaya *et al.*, 2008). Raw *Salmonella*-free popcorn was inoculated (1×10^3 - 8×10^6 CFU/g) with a *S. Typhimurium* strain previously isolated from raw microwave popcorn and cooked. After microwave oven treatment *Salmonella* were only recovered from samples inoculated at 2×10^6 CFU/g or greater. After conventional cooking, viable cells were recovered from samples inoculated at 9×10^4 CFU/g or greater. The results demonstrated that microwave cooking achieves a greater reduction of salmonellae than conventional cooking which may be because additional ingredients in the microwaveable popcorn interact to produce higher local temperatures. There are no reported cases of salmonellosis due to consumption of popcorn and this research indicates that only a high level of raw product contamination would pose a risk to consumers.

2.4 Exposure Assessment

2.4.1 *Salmonella* in cereal grains

No data could be located regarding the prevalence of *Salmonella* in domestically produced or imported cereal grains in New Zealand (under normal conditions). There are some data available collated from outbreak investigations (Section 3.3.4).

Table 8, Appendix 1 summarises available information from overseas on the prevalence of *Salmonella* in cereal grains and flours. The only large scale surveys are for milled wheat, oats, maize and durum wheat from the USA and Australia. The studies carried out in the USA were of milled product, while the Australian study including incoming wheat, intermediate milling fractions and finished flour. In milled wheat samples (n = 412 - 4,358) from both countries the prevalence of *Salmonella* was 0.1 – 1.3%, while the only survey to examine the other grains (sample numbers range from n = 180 for milled durum wheat to n = 1,772 for milled maize) did not find any *Salmonella*.

2.4.2 Serotypes of *Salmonella* in cereal grains

The ESR Enteric Reference Laboratory (ERL) undertakes typing of *Salmonella* isolates from human and non-human sources for all of New Zealand. Summaries are published on the website: http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php. Isolates from non-human sources may be described as coming from 'Food', but are not further categorised to allow identification of isolates coming from cereals or cereal products.

2.4.3 Food consumption: cereal grains

Cereals (principally rice, maize and wheat) form the basis of the human diet in most societies. In New Zealand, wheat is the primary cereal consumed, mainly in the form of bread. Food Balance Sheets (FBS) for New Zealand for the latest available year (2005) show that from a total cereal consumption of 93.7 kg/capita/year, 84% of cereal consumption is wheat, 9% rice, 2% maize, 2% oats and small amounts of barley and rye¹.

Data from the 1997 National Nutrition survey are summarised in Table 2. The Australia New Zealand Food Authority (now Food Standards Australia New Zealand) estimated consumption of raw agricultural commodity equivalents, based on analysis of the ingredients of complex foods and back calculation to source materials.

Table 2: Cereal grain consumption by New Zealand adults (ANZFA, 2001)

Cereal	Percent consuming in 24-hour period (%)	Average daily consumption, all (g/day)*	Average consumption, consumers only (g/day)*	97.5 th percentile consumption, consumers only (g/day)
Cereal grain fractions	98.3	127.3	129.5	370.1
Wheat flour	98.0	106.6	108.7	347.3
Rice, polished	20.4	10.2	50.0	213.8
Maize flour	23.0	3.2	14.1	68.2
Cereal brans, processed	13.6	0.9	6.7	49.9
Rye, wholemeal	23.5	2.3	9.9	27.1
Oats	22.5	5.9	26.1	99.3
Millet	2.1	0.1	6.0	27.9

'All' refers to the overall set of respondents, including people who did not report consuming cereals in the previous 24-hours. 'Consumers' refers only to those who reported consumption of cereals in the previous 24-hours.

These data confirm that cereal grains, principally wheat, are consumed by the majority of the population on any given day. The source for these data did not allow identification of the specific cereal grain products covered by the current Risk Profile.

¹ <http://faostat.fao.org>

2.4.4 Evaluation of exposure

2.4.4.1 *Number of servings and serving sizes*

Analysis of 24-hour dietary recall records from the 1997 National Nutrition Survey (NNS; 4,636 adults 15+ years old) (Russell *et al.*, 1999) and the 2002 Children's National Nutrition Survey (CNS; 3,275 children 5-14 years) (Ministry of Health, 2003) revealed 17,529 servings in the NNS where one or more cereals would have been a major ingredient (greater than 20% by weight) and 14,490 servings in the CNS. Cereals will be included in many more food servings as a minor ingredient.

Using a total New Zealand population of 4,341,427 (Statistics New Zealand population clock, accessed 27 November 2009), assuming the same proportion of adults (15+ years; 78.5%) and children (<15 years; 21.5%) as at the last census, and by necessity making the assumption that the diets of children less than 5 years old are not substantially different to diets of children aged 5-14 years:

$$\begin{aligned}\text{Annual number of servings (total population)} &= 4,341,427 \times ((0.215 \times 14,490/3,275) + \\ &\quad (0.785 \times 17,529/4636)) \times 365 \\ &= 6.2 \times 10^9 \text{ servings}\end{aligned}$$

This represents a very high number of servings, as would be expected from a diet staple such as cereal grains. It should be noted that this figure represents the total number of cereal servings. Directly consumed cereal grains and their primary processed products are likely to make up a very small proportion of these servings. However, the data sets used did not allow identification of foods such as Bircher muesli or practices such as eating raw cake batter.

2.4.4.2 *Frequency of contamination*

Contamination of cereal grain products consumed in a raw state is unknown for New Zealand.

An Australian study of nine flour mills showed that *Salmonella* was detected in only 2 from 412 milled wheat samples (the authors stopped testing for *Salmonella* part-way through the study due to the low numbers) (Berghofer *et al.*, 2003). These samples were both milling samples; *Salmonella* was not detected in the incoming wheat or end product, so contamination may have occurred in the mill. Given that New Zealand imports most of its wheat from Australia, this study indicates that contamination of imported raw wheat is likely to be infrequent.

New Zealand imports most of its rice from Thailand, USA and Australia. No information was found on *Salmonella* contamination of uncooked rice grains. An Australian survey of fried rice from retail (takeaway) outlets did not detect *Salmonella* in any of 63 samples (Millard, 2006). However, this study was mainly concerned with *Bacillus cereus* contamination and the cooking involved with fried rice production means that detection of *Salmonella* would have been unlikely.

2.4.4.3 *Predicted contamination level at retail*

Unknown.

2.4.4.4 *Growth rate during storage and most likely storage time*

Due to the low water activity of stored grain and flour, growth of *Salmonella* spp. does not occur. Most likely storage times can vary in domestic settings. The New Zealand Flour Millers Association (2009) recommends that flour is bought in quantities that will last a maximum of two to three months. This is particularly applicable to wholemeal flour because it contains wheat germ that has a high fat content and can go rancid over time. Where flour is to be stored for extended periods it can be frozen.

2.4.4.5 *Heat treatment*

Almost all milled cereal grains are baked, fried or otherwise cooked (or fermented) before consumption in a manner that will eliminate any *Salmonella*. Cereal grain products that may receive a lesser heat treatment, or none at all, include cereal based infant foods, uncooked breakfast cereals (e.g. muesli), or products designed to be baked at home (e.g. cookie dough). Ingestion of raw flour may occur during home baking or homemade play-dough.

2.4.4.6 *Exposure summary*

The information presented above indicates that cereal grain products are commonly eaten, mostly in forms that have undergone a heat inactivation process. There are very few cereal grain products consumed in forms which may result in *Salmonella* exposure. The probability of contamination by *Salmonella* of raw cereals in New Zealand is unknown.

3 EVALUATION OF ADVERSE HEALTH EFFECTS

3.1 Disease characteristics

Incubation: 6-48 hours (usually 12-36 hours).

Symptoms: Diarrhoea, abdominal pain, vomiting, nausea and fever lasting 1-7 days. Hospitalisation rate estimated at 22.1%, case fatality rate 0.8%.

Condition: Salmonellosis.

Toxins: Toxins are not produced in foods.

People Affected: The young, old, and immunocompromised are particularly at risk along with lower socioeconomic groups and those living in higher population densities.

Long Term Effects: Septicaemia and subsequent extra-intestinal infections can occur. Reactive arthritis may occur 3-4 weeks after gastrointestinal symptoms. Approximately 2% of a population exposed to a triggering infection will develop reactive arthritis. The disease usually resolves within six months, but may persist for more than a year in some cases (Hannu *et al.*, 2006).

Treatment: The infection is usually self-limiting, uncomplicated gastroenteritis although fluid replacement may be required, especially in the elderly or young children. Less than 2% of clinical cases require antibiotic treatment. The site of infection and the immunity status of the case determine treatment choices.

Supplemental information on adverse health effects is given in Appendix 2.

3.2 Dose-response

The dose required to cause disease varies and is multi-factorial. Low attack rates have been observed in one outbreak where 4-45 cells were consumed, and another where the dose was 6 cells in 65 g of food (Anonymous, 1996). The outbreak was due to *S. Enteritidis* contamination of ice cream. Different serotypes may have different dose responses, with doses generally recognised to cause disease at high attack rates in the range of 10^5 to 10^7 cells.

The most commonly used dose-response model was produced by the joint risk assessments of *Salmonella* in eggs and broiler chickens by FAO/WHO (FAO/WHO, 2002). Results from a number of human feeding trials of *Salmonella* serotypes have been analysed to develop dose-response models (most recently by Oscar (2004) using a three phase linear model). These feeding trials have a number of deficiencies, particularly at low doses, as described in the FAO/WHO report. Consequently the FAO/WHO model augmented the data with information from outbreak reports. These reports were screened and a final 20 outbreaks were used in the database (12 Enteritidis, 3 Typhimurium, Heidelberg, Cubana, Infantis, Newport and Oranienburg). Several vehicles of transmission were implicated including meat, eggs, dairy products and water. A beta-Poisson model was used to develop the mathematical relationship, and a maximum likelihood technique used to generate the curve best fitting the data. The graph shows that for the ingestion of 10^{10} cells there was in a probability of around 0.9 of illness, while the ingestion of 10^1 cells resulted in a probability of around 0.02. Thus the

probability of illness from exposure to small doses is low. For outbreaks where food contains only low numbers of organisms but has been widely consumed, a small proportion of consumers are likely to become ill.

It has been repeatedly reported that the probability of disease following ingestion of small numbers of cells is higher when the implicated food has a high fat or protein content. For example, chocolate or peanut butter may protect cells from gastric juices so permitting a lower dose than usual to cause infection. Experimentation has also shown this to be the case for high fat foods (minced beef) and high protein foods (egg white). It was concluded that the pH of the microenvironment of the organism in the food matrix is crucial for determining resistance to stomach acids (Waterman and Small, 1998).

An outbreak of *S. Typhimurium* not used in the FAO/WHO model involved consumption of roast pork. The dose causing disease was calculated to be 2.6×10^5 MPN/g. The outbreak occurred in a home for mentally disabled students in Kanagawa, Japan. The roast pork stored at the caterer's facility was found to contain 4.3×10^4 MPN/g. From 140 people, 105 exhibited food poisoning symptoms, an attack rate of 75% (the FAO/WHO model predicts a probability of illness between 0.5-0.75) (Murase *et al.*, 2000).

3.3 New Zealand Outbreak Information and Human Health Surveillance

Salmonellosis is a notifiable disease in New Zealand. The number of cases and incidence of notified (non-typhoidal) salmonellosis since 2003 is shown in Table 3.

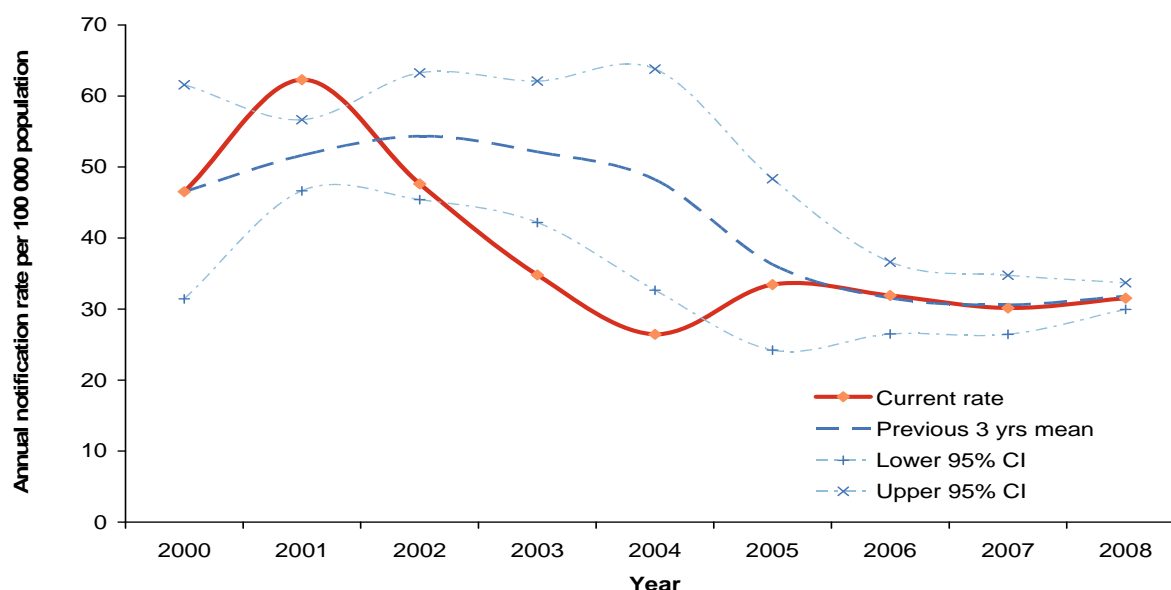
Table 3: Incidence data for salmonellosis in New Zealand

Year	Number of cases	Incidence (cases/100,000)
2003	1,401	34.8
2004	1,080	26.4
2005	1,383	33.7
2006	1,335	31.9
2007	1,274	30.1
2008	1,346	31.5
2009	1,129	26.2

Number of cases data taken from (ESR, 2010), Population data for June each year taken from (http://www.stats.govt.nz/methods_and_services/access-data/tables/national-pop-estimates.aspx). Due to population adjustments by Statistics New Zealand rates may differ slightly from older Annual Surveillance Summary reports.

The notification rate per 100,000 population for cases of salmonellosis in New Zealand from 2000 – 2008 is shown in Figure 3. The rate has been stable since 2005 at approximately 30 per 100,000.

Figure 3: Incidence of notified salmonellosis in New Zealand 2000 - 2008



Reproduced from (Williman *et al.*, 2009)

The incidence of salmonellosis is characterised by a late summer peak and a winter trough.

Highest rates are often reported from the lower South Island; in 2008 the highest rates were from South Canterbury (37 cases, 66.9/100,000) and Otago (129 cases, 68.9/100,000).

Reported rates are similar for males (33.6/100,000 in 2008) and females (28.6 per 100,000 in 2008). Age specific rates are highest for the <1 year age group (135.8/100,000 in 2008), and 1 to 4 year olds (108.9/100,000 in 2008).

3.3.1 Clinical outcomes: Salmonellosis in New Zealand

Hospitalisation and fatality rates for notified cases of salmonellosis in New Zealand are given in Table 4. These outcomes are not always reported for each case, so percentages are expressed in terms of the number of cases for which outcomes are known. The hospitalisation rate and number of deaths has been stable over many years.

Table 4: Outcome data for salmonellosis in New Zealand, 2003-2008

Year	Hospitalised cases	Fatalities	Reference
2003	167/1118 (14.9%)	0/1401	(ESR, 2004b)
2004	109/871 (12.5%)	0/1080	(ESR, 2005b)
2005	142/1134 (12.5%)	1/1383 (0.07%)	(ESR, 2006b)
2006	148/1111 (13.3%)	1/1335 (0.07%)	(ESR, 2007b)
2007	110/833 (13.2%)	1/1274 (0.07%)	(ESR, 2008a)
2008	123/896 (13.7%)	1/1346 (0.07%)	(ESR, 2009a)

Chronic sequelae of *Salmonella* infections include reactive arthritis. A study carried out in the south of New Zealand found evidence of preceding *Salmonella* infection in two of 60 (3.3%);

95th percentile confidence interval 0.4-11.5%) cases of reactive arthritis (Highton and Priest, 1996). While no relevant New Zealand information is available, an English study isolated *Salmonella* from 0.2-0.4% of faecal samples from general population cohorts (Food Standards Agency, 2000)

3.3.2 Serotypes causing disease in New Zealand

The principal serotypes of *Salmonella* identified from notified cases in New Zealand for the period 2005-2008 are *S. Typhimurium* (approximately 50% to all identified isolates, with the most frequent definitive phage type being DT160), and *S. Enteritidis* (approximately 10%) (Williman *et al.*, 2009).

Table 5 shows the trend for the number of human *Salmonella* isolates for selected serotypes or phage types during the period 2005-2008.

Table 5: Selected *Salmonella* serotypes and subtypes of laboratory-confirmed human isolates, 2005 – 2008

Subtype	2005	2006	2007	2008
<i>S. Typhimurium</i>	757	733	596	729
DT160	248	260	152	135
DT42	27	28	15	93
DT101	67	71	43	72
DT1	114	72	91	72
DT156	75	87	73	67
DT74	28	42	29	21
Other or unknown	198	173	193	269
<i>S. Enteritidis</i>	151	107	151	124
PT9a	73	53	60	45
PT1b	9	9	18	19
PT26	9	7	17	10
Other or unknown	60	38	56	50
<i>S. Infantis</i>	67	58	86	86
<i>S. Chester</i>	0	1	37	64
<i>S. Mbandaka</i>	8	22	14	39
<i>S. Saintpaul</i>	65	35	25	35
<i>S. Brandenburg</i>	68	55	47	33
<i>S. Virchow</i>	16	13	34	14
Other or unknown serotypes	274	319	277	215
Total	1 406	1 343	1 267	1 339

Reproduced from Williman *et al.* (2009)

3.3.3 Outbreaks

The number of reported outbreaks of salmonellosis in recent years in New Zealand is given in Table 6 (figures exclude *S. Typhi* and *S. Paratyphi*). The number of cases reported as outbreaks is approximately 10% of those reported as sporadic cases. As a proportion of enteric outbreaks or cases, salmonellosis makes a small contribution; the outbreak data are dominated by reported outbreaks of norovirus.

Table 6: Reported outbreak data for salmonellosis in New Zealand 2003-2008

Year	Salmonellosis outbreaks/ total enteric outbreaks	Cases/Total Enteric Cases*	Reference
2003	23/315 (7.3%)	59/2649 (2.2%)	(ESR, 2004a)
2004	5/313 (1.6%)	74/3971 (1.9%)	(ESR, 2005a)
2005	26/338 (7.7%)	120/2343 (5.1%)	(ESR, 2006a)
2006	22/481 (4.6%)	74/6162 (1.2%)	(ESR, 2007a)
2007	8/477 (1.7%)	141/7821 (1.8%)	(ESR, 2008b)
2008	15/428 (3.5%)	163/6295 (2.6%)	(ESR, 2009b)

* Includes both suspected and confirmed cases

A search of the Episurv outbreak database was carried out to identify outbreaks of salmonellosis where foods that included cereal grain products had been identified as suspected. The time-frame analysed was 2000-2009, during which time there were 190 domestically acquired outbreaks of salmonellosis reported. Of these, foodborne transmission was suspected in 108 outbreaks, and at least one suspected food was reported in 75 of these. Of these 75, there were 33 outbreaks where one or more of the foods implicated contained cereals or cereal products, usually as part of a mixed food (Table 11, Appendix 2). In the majority of outbreaks, other risk factors were also present. In most of these outbreaks the cereals were in a cooked form (e.g. bread, cooked rice), so it is unlikely that the salmonellosis was caused by the cereal portion of the food. There were only two outbreaks where *Salmonella* was isolated from the foods (shaded in table). Information from one of these outbreaks (2005) was incomplete and it was not clear which of the foods was *Salmonella*-positive. A large outbreak in 2008 was caused by contaminated flour milled in a single New Zealand mill and distributed under several brands (see section 3.3.4).

Given the wide variety of cereal grain-containing products and frequent consumption, it is not surprising that such foods are often listed amongst the suspected vehicles for an outbreak. Also, many of the implicated foods contain non-cereal ingredient that could be the source of any *Salmonella* present. These data do not necessarily indicate that cereal products are important vehicles for salmonellosis outbreaks in New Zealand.

3.3.4 Case control studies and risk factors

3.3.4.1 Outbreak of *Salmonella* in flour

A case-control study was conducted for an outbreak in 2008-09 suspected of being linked to flour (Lisa McCallum, ESR, personal communication). Between 13th October 2008 and 28th January 2009, a total of 75 cases of salmonellosis caused by *S. Typhimurium* phage type 42 were identified. Of these, 67 isolates were the same strain based on molecular analysis; 16/67 cases were aged 4 years or younger and 76% of cases were female. Twelve cases were hospitalised and there were no fatalities. The majority of the cases resided in Canterbury (22/75) and Otago (17/75).

For the case-control study, 33 cases and 66 controls were interviewed. The cases had an odds ratio (OR) of 3.6 compared to controls for eating, licking or tasting uncooked baking mixture ($p=0.001$; 95% C.I. 1.2-10.7). Examination of the individual baking ingredients found that after adjusting for eggs, flour had an odds ratio (OR) of 5.7 (95% CI 1.1 to 29.1, $p=0.035$).

After adjusting for flour, eggs had an OR of 0.8 (95% CI 0.2 to 3.4, $p=0.762$). An elevated significant OR was also found for a specific supermarket and brand of flour.

Flour samples were collected and tested for *Salmonella* from open packets in the homes of cases (4/26 positive), unopened packets that had been on sale in retail outlets prior to withdrawal (2/41 positive) and retrieved/withdrawn flour (3/23 batches of flour positive). Contamination levels were estimated for 3 of the positive samples. *Salmonella* counts ranged from 1 per 300g to 1 per 50g.

The same outbreak strain had been previously isolated from poultry feed produced by an animal feed mill from a by-product of flour milling called “broll”. Broll is the husk of the wheat kernel removed during milling of flour. The broll had been produced by the same flour mill that produced the contaminated flour, during the same time period. Environmental swabs were taken at the implicated flour mill as part of the flour outbreak investigation, but the outbreak strain was not isolated.

The flour company that produced the flour has two mills located in North and South Islands. Only the flour from the South Island mill was found to be contaminated which is consistent with the majority of cases being from the South Island. The South Island flour mill receives wheat from more than 400 New Zealand growers as well as imported wheat. Testing of withdrawn flour was undertaken to narrow down the search for a particular wheat source. Although further positive batches of flour were identified, the source of the contaminated wheat could not be established.

3.3.4.2 Other case-control studies concerning *Salmonella* in New Zealand

Two general case-control studies have been carried out concerning salmonellosis in New Zealand (NZFSA, 2002; Thornley *et al.*, 2002; Thornley *et al.*, 2003) and are summarised in Appendix 2. Neither of these studies addressed risk factors that might be relevant to cereals or cereal based foods.

3.4 Adverse Health Effects Overseas

The incidence of notified cases of salmonellosis in New Zealand is similar to rates in other developed countries, particularly Canada and Australia (see Appendix 2, Table 10). In contrast to New Zealand, in the EU the dominant serotype is *S. Enteritidis* (see Appendix 2, Table 11).

3.5 Health Burden of Infection with *Salmonella* spp.

An estimate of the burden of foodborne disease for New Zealand (Cressey and Lake, 2007) includes an estimate for foodborne salmonellosis of 111 disability adjusted life years (DALYs). This represents 60.7% of the total 186 DALYs for salmonellosis, with the percentage foodborne being derived from an expert consultation process. This placed foodborne salmonellosis fourth on the list for foodborne disease burden (after campylobacteriosis, norovirus infection, and perinatal listeriosis).

The burden of disease to the health system and society in general has also been considered, through a cost of illness estimate, based on the same incidence data (Cressey and Lake, 2008). This estimated the total cost for salmonellosis as \$4.8 million, with foodborne infections costing \$2.8 million.

A recent New Zealand study, using molecular sub-typing data and Bayesian techniques ('modified Hald model') estimated the attributable food source for human salmonellosis cases in New Zealand in 2003 (Mullner *et al.*, 2009). However, cereals and cereal products were not considered in this study.

In the USA, foodborne salmonellosis was estimated to cost the economy \$US2.3 billion annually (1998 \$US) (Dickson *et al.*, 2002). A more recent estimate of the cost of foodborne salmonellosis in the USA, including quality-of-life costs (death, pain, suffering and functional disability), arrived at a much higher cost estimate, \$US 14.6 billion (Scharff, 2010).

European estimates of the cost of salmonellosis are more in line with New Zealand estimates (given population differences), with Kemmeren *et al.* estimating the cost of salmonellosis in the Netherlands to be 8.8 million Euros in 2004 (Kemmeren *et al.*, 2006).

3.6 Adverse Health Effects Summary

The incidence of salmonellosis in New Zealand is comparable with the incidence in other developed countries. There has only been one recorded *Salmonella* outbreak in New Zealand where a cereal grain or cereal grain product was the confirmed vehicle for transmission.

4 EVALUATION OF RISK

4.1 Existing Risk Assessments

No existing risk assessments for *Salmonella* spp. in cereal grains were located.

4.2 Estimate of Risk for New Zealand

4.2.1 Risk associated with cereal grains

As raw materials, cereal grains may be contaminated with *Salmonella* from animal or human faecal material. After harvest, rodents and birds are particularly important sources, if adequate storage is not maintained. Due to the low water activity of cereal grains and their milled products, growth of *Salmonella* does not occur, but the bacteria remain viable for long periods (months). The low water activity of cereal grains and cereal grain products also promotes heat resistance in *Salmonella*.

Cereal grains and their products are consumed by almost all New Zealanders on a daily basis. Most will be consumed in forms that have been rendered *Salmonella*-free through processing. Any residual risk will come from consumption of minimally processed foods that allow the bacteria to survive. In some cases growth may be possible following addition of hydrating ingredients such as water or milk. Examples of such foods include cereal-based infant foods, uncooked breakfast cereals (e.g. muesli), or products designed to be baked at home (e.g. cookie dough). Outbreaks have been linked to these foods overseas. Ingestion of raw flour may also occur during home baking or activities with homemade play-dough.

The potential for exposure to *Salmonella* in raw flour by eating uncooked baking mixture has been demonstrated by a significant outbreak of salmonellosis linked to contaminated flour in New Zealand. The samples of flour tested in this outbreak investigation had low counts of *Salmonella*. Assuming that these samples were representative of the material causing disease and it was raw flour that was consumed (i.e. through home baking), then this outbreak points to a high risk of illness from consuming relatively few cells. However, the possibility that much higher counts of *Salmonella* were present in the actual flour that was ingested cannot be excluded (e.g. if the distribution of contamination was not homogenous).

Although there are no New Zealand data, surveys in Australia and North America have found prevalence of *Salmonella* contamination in wheat flour in the range 0.0-1.3%. There is even less information available on the prevalence of *Salmonella* on other cereal grains, but it appears likely to be similarly low. There is almost no information on the concentration of *Salmonella* on cereal grains or in milled products. However, flour samples analysed in association with the recent New Zealand outbreak contained very low concentrations of *Salmonella*. Exposure events whereby consumers are exposed to cereal grains or milled products, without a further bactericidal step, are likely to be uncommon (consumption of uncooked dough or batter, Bircher muesli consumption, infant cereal consumption).

Overall, the risk of human salmonellosis due to contaminated cereal grains must be classified as low. However, the outbreak linked to flour indicates that when cereal contamination occurs it has the potential to affect large numbers of people, even if potential exposures occur via specialised behaviours (e.g. ingestion of uncooked home baking materials) or less common foods (e.g. uncooked muesli ingredients).

4.2.2 Risks associated with other foods

Three case-control studies have assessed risk factors for human salmonellosis in New Zealand. A case control study of sporadic salmonellosis cases reported elevated odds ratios for consumption of pork steak (OR = 9.0) and hot dogs (OR = 2.8) (Baker *et al.*, 2003). A national case-control study of emergent *S. Brandenburg* found a slightly elevated odds ratio for consumption of sheep or lamb (OR = 1.2), but all other foods included in the questionnaire (imported food, uncooked vegetables, unpeeled fruit, pies, whole chicken, bacon, small goods, eggs, dairy products were protective (OR < 1) (Baker *et al.*, 2007). A case-control study of an outbreak of *S. Typhimurium* DT160 identified elevated risk associated with consumption of fast food (Thornley *et al.*, 2003).

A recent New Zealand study, using molecular sub-typing data and Bayesian techniques ('modified Hald model') estimated the attributable food source for human salmonellosis cases in New Zealand in 2003 (Mullner *et al.*, 2009). An estimated 60.2% (Bayesian credible interval 47-74%) of food sourced human salmonellosis was attributed to transmission by pork, followed by poultry (21%) and beef and veal (12%). The authors urged caution in interpreting these results since molecular sub-typing data for pork were sparse and more biased than data for other food animal species (Mullner *et al.*, 2009).

A recent assessment of outbreaks and all cases of notified human salmonellosis from 2000 to 2009 concluded that, while foodborne transmission appeared to be important, it was not possible to quantitatively attribute the burden of salmonellosis to specific foods (Adlam *et al.*, 2010).

Transmission in poultry currently represents a minor component of salmonellosis etiology in New Zealand (Lake *et al.*, 2004b). Recent surveys of eggs have found *Salmonella* on the exterior of 1.8% (95th percentile confidence interval 0.8-3.3%) of samples, and no evidence of contamination internally (Wilson, 2007).

4.3 Data gaps

Due to the fact that cereal grains have not often been considered as a cause of human salmonellosis a number of significant data gaps exist, including:

- Information on the actual routes of introduction of *Salmonella* into cereal grains;
- Data on the prevalence of *Salmonella* in cereal grains in New Zealand, either domestically produced or imported, and serotypes present;
- Data on the concentration of *Salmonella* in cereal grains, prior to and following primary processing;
- Frequency of consumption and serving sizes of potential risk foods (e.g. uncooked batter or dough, Bircher-style muesli, cereal-based weaning foods);
- Data on concentration of *Salmonella* in risk foods at consumption; and
- Dose-response for *Salmonella* from cereal grains.

However, cereal grains are likely to be infrequently contaminated with *Salmonella* and a survey to generate such data to fill these data gaps would need to test very large numbers of samples. A more effective approach to assessing the risks associated with this food/hazard combination may be to assess potential sources of *Salmonella* contamination of cereal grains and the current controls.

A project recent initiated by NZFSA (A review of the use of water and natural fertilisers during the growing, harvest and packing of horticultural produce) may provide useful information on the potential of irrigation water and natural fertilisers to introduce *Salmonella* into cereal grains.

5 AVAILABILITY OF CONTROL MEASURES

5.1 Risk Management Strategy

In March 2009 NZFSA released their *Salmonella* Risk Management Strategy 2009-2012¹. The Strategy aims to achieve a 30% reduction in the reported annual incidence of foodborne salmonellosis after five years. The strategy focuses on non-typhoid *Salmonella* and begins with a primary focus on intelligence gathering from a wide range of food sectors.

The objectives of the *Salmonella* risk management strategy are to:

- Quantify the proportion of foodborne cases attributable to:
 - specific foods
 - animal feeds
 - domestically produced versus imported foods
 - multi-resistant and virulent *Salmonella* genotypes associated with foods
- Identify sources of *Salmonella* contamination of specific foods and animal feeds
- Determine the relative value of different interventions throughout the food chain in reducing the risk of salmonellosis
- Make prioritised risk management decisions on appropriate *Salmonella* control measures across the food chain, and according to data availability
- Design and implement an effective monitoring and review programme to support strategic goals.

5.2 Current Risk Management Measures

5.2.1 Relevant food controls

5.2.1.1 *Import Health Standards*

The MAF Biosecurity website contains a list of cereal grains for which import health standards exist when importing from all countries.² There are generally three categories:

- Non-viable grain: Grain that has been heat-treated and is accompanied by a phytosanitary certificate;
- Viable grain; and
- Grain that is to be devitalised (by either heat or irradiation): Application of heat treatment must be upon arrival in New Zealand. Grains must be heat treated at 85°C (core temperature) and 40% relative humidity for a minimum of 15 continuous hours or at a temperature/time regime verified to be effective in devitalising seed. Treatments must be carried out in a MAF facility or under MAF supervision.

Individual import health standards are available for grains of oat (*Avena* spp.), barley (*Hordeum* spp.), millet (*Panicum* spp.), rye/ryecorn (*Secale cereale*), sorghum (*Sorghum bicolor*), triticale (*Triticosecale*), wheat (*Triticum* spp.) and maize/popcorn/sweetcorn (*Zea mays*). None of these standards address *Salmonella* and they are primarily to prevent entry of

¹ <http://www.nzfsa.govt.nz/foodborne-illness/salmonella/strategy/salmonella-risk-management-strategy-2009-012.pdf>

² <http://www.biosecurity.govt.nz/imports/plants/standards/bnz-gcftp-phr.htm>

weed seeds, invertebrate pests and microorganisms of plant health significance. However, the specified heat treatment would probably be sufficient to destroy any *Salmonella* present on the grain.

5.2.1.2 Cereal industry controls in New Zealand

The Arable Food Industry Council (AFIC) is the main organisation that represents the cereal grain industry in New Zealand. An AFIC taskforce produced the New Zealand Crop Quality Assurance Scheme (NZCQAS)¹ which is based on HACCP principles, but does not specifically address *Salmonella* risks. The manual does, however cover:

- Fertiliser application, stating that it should maintain soil fertility, minimise leaching and minimise the risk of food safety hazards;
- Pre-harvest storage and equipment preparation;
- Grain drying;
- On-farm grain storage; and
- Transportation of grain.

The attention to equipment and storage cleaning included in the scheme is likely to have benefits for *Salmonella* control. However, the lack of any pre- or post-implementation information on *Salmonella* prevalence in cereal grains in New Zealand means that it is not possible to comment on the scheme's effectiveness for *Salmonella* control.

5.2.2 Relevant environmental controls

5.2.2.1 Resource consents

Spray irrigation of farm land with effluent has the potential to spread animal faecal material to adjacent cereal crops. Consents can be viewed on some authority websites. Relevant resource consents granted in the Canterbury region were reviewed and found to contain controls to prevent spray-drift contamination of adjoining properties, including:

- The discharge shall be managed to ensure that aerosols and spray-drift arising from the application of effluent onto land are contained within the boundary of the property on which this consent is exercised;
- The discharge shall not cause an odour which is offensive or objectionable beyond the boundary of the property on which this consent is exercised; and
- There shall be no discharge: a) within 20 metres of any surface water body; b) within 30 metres of any bore; and c) such that contaminants are likely to run-off and enter any surface water body.

No specific information was found on the effectiveness of these measures in terms of control of *Salmonella* contamination of cereal grains. However, two general comments can be made, based on the scientific literature:

- Spraying of effluent (wet dissemination) results in desiccation of bacteria in the effluent and decreased survival. By comparison, aerosolisation of dust (dry dissemination) results in partial rehydration of bacteria and improved survival (Tang, 2009).

¹ links can be found on some of the member body websites for example United Wheatgrowers; <http://www.uwg.co.nz/quality/history.cfm>

- The structure of the cereal plant will tend to protect the grain from direct deposition of bacteria and a study of crops following direct application of sewage effluent did not detect faecal indicator organisms on edible grains (wheat and rice), while faecal indicator organisms were detected on fodder crops (clover, sorghum) and vegetables (cabbage, gourd) (Minhas *et al.*, 2006).

The resource consent provisions summarised above would, at least, mean that any contamination of cereal crops would be considerably less than that due to direct spray irrigation with effluent. The lowered survival of bacteria during wet dissemination and the physical protection provided to the cereal grain by the cereal head morphology suggest that contamination of cereal grains via this route is likely to be minimal.

5.3 Options for Risk Management

It is uncertain whether the outbreak where flour was identified as the vehicle (see section 3.3.4.1) was caused by contamination prior to or during harvest or at the flourmill. A number of hazard controls exist in the cereal growing and processing industries that will reduce the likelihood of *Salmonella* contamination (e.g. the New Zealand Crop Quality Assurance Scheme). However, no information is available on the effectiveness of these controls.

Risk communication regarding the consumption of uncooked flour products (e.g. cake batter, cookie dough) may be warranted, given the recent outbreak. Such communications might also address the possibility of home made play-dough /raw flour being consumed during play.

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7 APPENDIX 1: HAZARD AND FOOD

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

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

7.1 Salmonella

7.1.1 Growth and survival

Growth:

Temperature: Minimum 7°C, growth greatly reduced at <15°C. Maximum 49.5°C. Optimum 35-37°C. Some evidence for growth at temperatures <7°C exists, but this is serotype specific, the data are still not universally accepted and doubts surrounding the experimentation exist.

pH: Minimum 3.8, optimum, 7-7.5, maximum 9.5. The minimum pH is influenced by other factors such as temperature, acid present, and the presence of nitrite etc.

Atmosphere: Can grow in the presence or absence of air as a facultative anaerobe. The growth rate on beef muscle stored at 20°C under nitrogen is only slightly less than that obtained when stored under air (Grau, 1983). At high concentrations of CO₂ (50-60%), growth is strongly inhibited on beef steak and minced beef at 10-11°C, but at 20°C there is little inhibition (Luiten *et al.*, 1982; Silliker and Wolfe, 1980).

Water activity: Minimum 0.94, optimum 0.99, maximum >0.99.

Survival:

Salmonella are known to survive well in foods and on surfaces. Particularly in foods with low water activity e.g. flour.

Temperature: *Salmonella* can survive well in foods for long periods at low refrigeration temperatures. In frozen foods, although *Salmonella* numbers are considerably reduced, some survive for long periods. Some foods, including meat, ice-cream and butter, appear to be protective of *Salmonella* during freezing and frozen storage. Rapid freezing promotes survival with lower frozen storage temperatures and less fluctuation giving greater survival (Jay *et al.*, 2003).

Frozen storage temperatures near 0°C result in greater death or injury to bacterial cells. In minced chicken breast (pH 5.8), 60-83% of *Salmonella* cells survived storage at -20°C for 126 days, whereas at -2°C and -5°C only 1.3% to 5.8% of cells respectively were still viable after 5 days.

pH: *Salmonella* appear to be significantly less tolerant of low pH (pH 2.5; hydrochloric acid)

than *Shigella* spp. or *Escherichia coli*. These last two organisms possess additional acid survival systems that are not present in salmonellae (Gorden and Small, 1993; Lin *et al.*, 1995).

Water Activity: Survival in dry environments is a characteristic of these organisms. For example, they can survive in bitter chocolate (a_w 0.3-0.5) for months. Exposure to low a_w environments can greatly increase the heat resistance of these organisms.

7.1.2 Inactivation

Note that in microbiological terms “D” refers to a 90% (a decimal or 1 log₁₀ cycle) reduction in the number of organisms.

Temperature: Inactivation is greater during the freezing process rather than subsequent frozen storage, but those cells that survive remain viable. Freezing does not ensure the inactivation of salmonellae in foods.

D times: at 60°C usually 2-6 min; at 70°C usually 1 min or less. Some rare serotypes (e.g. *S. Senftenberg*) are significantly more heat resistant than the others, but this organism is not considered to be important as a food pathogen (Doyle and Mazzotta, 2000).

D times for *Salmonella* can depend on the type of food involved. Long D times have been reported for experiments with *Salmonella* Typhimurium in milk chocolate. Values reported were up to 1050 min at 70°C, 222 min at 80°C and 78 min at 90°C.

pH: Low pH values and the nature of the acidulant determines the rate of death. Temperature is also a factor.

In the studies by Alford and Palumbo, the authors demonstrated how decreasing temperature increases the inhibitory effects of pH and NaCl. In broth, at 10°C, growth of 22/23 strains were inhibited by pH 5 and 2% NaCl (Alford and Palumbo, 1969). At pH 5.8, (more representative of meat), 5% NaCl at 10°C was required to inhibit growth. Increasing the salt concentration slightly decreased survival time at 10°C.

Water activity: At a_w levels below those allowing growth, salmonellae die slowly. The rate of death decreases as the a_w is lowered and also decreases as the temperature is reduced (Troller and Christian, 1978).

Radiation: The effect of gamma or beta radiation on *Salmonella* DT104 in ground pork has been researched (Rajkowski *et al.*, 2006). A mixture of six strains was used to inoculate three ground pork products (of varying fat content). The amount of beta radiation to achieve a 90% reduction was around 0.43 kGy regardless of fat content.

Disinfectants: Sanitisers appear to have some effectiveness against *Salmonella* during pork primary processing (Childers *et al.*, 1977). Sanitising the transport vehicle and lairage with chlorine or quaternary ammonium compounds was not effective in reducing *Salmonella* contamination of the carcass. However, altering the procedures during evisceration had a significant effect. For example, the wearing of plastic gloves and disinfecting the knife in 82°C water before each carcass reduced contamination of the carcass by 50%. Dipping the knife into 500ppm chlorine solution (pH 6) or in 25-ppm iodine solution reduced

contamination by 75%.

7.2 The Food Supply: Cereal Grains

7.2.1 Cereal grain production

Cereals are grasses, typically of the monocotyledonous families *Poaceae* or *Gramineae* and include wheat, oats, rye, sorghum, barley, millet, rice, maize and triticale (a hybrid of wheat and rye). All are annual plants¹. Cereal grains are the edible starchy fruit of these cereals. The bulk of the cereal grain is made up of the endocarp or endosperm, composed mainly of carbohydrate and protein. The germ is the reproductive embryo of the fruit and is high in vitamins B and E, minerals and antioxidants. The bran layer is the protective outer shell that is partly waterproof and contains fibre, B vitamins and minerals. Bran is composed of a hard outer layer (pericarp) and a softer underlying layer (aleurone).

Wheat can be further categorised into several species, these include²:

- Bread, modern cultivated hexaploid cultivars (*Triticum aestivum*)
- Durum, hard tetraploid wheat used to make semolina and pasta (*T. durum*)
- Spelt, an ancient hexaploid wheat species (*T. spelta*)
- Einkorn, a diploid wheat. The name can refer to a wild (*T. boeoticum*) or a domesticated (*T. monococcum*) species
- Emmer, a tetraploid wheat. The name can refer to a wild (*T. dicoccoides*) or a domesticated (*T. dicoccum*) form.

Maize (corn), wheat and rice account for 87% of the world's grain harvest. People primarily consume rice as intact grains (usually with the bran removed), whereas wheat, barley, oats and rye are commonly consumed in a processed form. In the production of refined grains, such as white flour, the bran and germ are removed. In general, high protein wheat ("hard" wheat) is better for breadmaking and low protein wheat ("soft" wheat) for cakes and biscuits. Pasta requires very high protein flour, such as durum wheat varieties. Durum wheat is not grown in New Zealand (New Zealand Flour Millers Association, 2009).

Pasta dough is produced primarily from wheat flour and has 30% moisture content. When dried (at approximately 40°C), the moisture content lowers to 10-12% (ICMSF, 1996). *Salmonella* spp. can survive and grow in pasta dough during manufacture, particularly if there are 'wet spots' due to uneven mixing. *Salmonella* spp. will not grow in dry pasta, but may survive for long periods (up to a year) in dry pasta stored at room temperature (Rayman *et al.*, 1979). Noodles are predominately produced from rice flour and egg (or egg yolk) can be added to the dough to make egg noodles.

7.3 Prevalence of *Salmonella* in Cereals Grains Overseas

Overseas prevalence data for *Salmonella* in cereal grains and their products from individual countries is collated in Table 7.

¹ Accessed at: <http://en.wikipedia.org/wiki/Cereal>. Accessed: 19 March 2010

² Accessed at: <http://en.wikipedia.org/wiki/Wheat>. Accessed: 19 March 2010

Table 7: Prevalence of *Salmonella* spp. in cereal grains overseas

Country	Year	Samples tested	Number (%) positive	Reference
Australasia				
Australia - NSW - Queensland - Victoria - WA	1997-1998 and 1998-1999 wheat seasons	Milling process and end product from 9 wheat flour mills – total 650 samples of which 412 tested for <i>Salmonella</i>	2/412 (0.5%) The point in the milling process at which <i>Salmonella</i> positive samples were detected was not specified, but was not incoming wheat or finished flour	(Berghofer <i>et al.</i> , 2003)
Europe				
Spain	2008	Raw popcorn (number not stated)	8-13%	(Anaya <i>et al.</i> , 2008) (citing unpublished data)
Turkey	2006	Wheat, moisture range 12.3 to 14.2%	0/142 (not detected)	(Aydin <i>et al.</i> , 2009)
North America				
USA	1977	Rye flour Brown rice Other cereals included in this survey but no <i>Salmonella</i> detected: Rice flour, Barley flour, Wheat flour, Millet flour, Oat flour, Barley, Oats, Rye, Wheat, Coarse bran, Corn meal, Wheat germ	1/3 <i>S. Molade</i> (33%) 1/3 <i>S. Anatum</i> (33%)	(Andrews <i>et al.</i> , 1979)
USA	1977	1 bottle x 25g sample wheat bran tablets	0 (not detected)	(Thomason <i>et al.</i> , 1977)
USA	1984-1991	Wheat flour	4/1,170 (0.34%)	(Sperber, 2003)

Country	Year	Samples tested	Number (%) positive	Reference
USA	1989	Wheat flour samples consisting of: - 681 hard red winter - 1,355 soft red winter - 188 spring - 816 durum	40/3,040 (1.3%)*	(Richter <i>et al.</i> , 1993)
USA	2007	Milled cereal grains consisting of: - 4,358 wheat - 1,772 maize - 714 oats - 286 whole wheat - 180 durum wheat	6/7,310 (0.08%) - All were wheat (0.14% of wheat samples)	(Sperber <i>et al.</i> , 2007)

* Highest frequency occurring in the autumn and winter months, lowest frequency in summer, see Table 12.

Richter *et al* examined seasonal differences in the prevalence of *Salmonella* in wheat (Table 8). Soft red winter wheat had the highest percentage of positive samples (2.29%) and durum wheat the lowest (0.25%). The highest frequency of salmonellae contamination occurred (for all wheat types) in the winter season (2.98% positive) and lowest in the summer months (0.25% positive) (Richter *et al.*, 1993).

Table 8: Percentage and number of wheat samples positive for *Salmonella*, by season

Wheat type	Winter		Spring		Summer		Autumn		Total	
	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>
Hard red winter	0.74	136	1.79	223	0	181	0	141	0.73	681
Spring	0	32	1.33	75	1.79	56	0	25	1.06	188
Soft red winter	6.13	310	1.42	422	0.30	338	1.75	285	2.29	1,355
Durum	0	194	0.90	222	0	220	0	180	0.25	816
Total	2.98	672	1.38	942	0.25	795	0.79	631	1.32	3,040

8 APPENDIX 2: EVALUATION OF ADVERSE HEALTH EFFECTS

Salmonellae possess virulence determinants that enable them to adhere to small intestinal epithelial cells, provided they survive the low pH of the stomach and other innate immune host defence mechanisms (Jay *et al.*, 2003). After entering epithelial cells, pathogenic salmonellae may multiply within a protective vacuole. Disruption of cellular tight junctions, leading to paracellular passage of ions, water and immune cells together with induction of host inflammatory cells is likely to contribute to the production of diarrhoea (Haraga *et al.*, 2008).

Two serotypes that have caused major problems overseas are *S. Enteritidis* which is capable of transovarian transmission into eggs (especially phage type 4 (PT4)) and the antibiotic resistant *S. Typhimurium* definitive phage type 104 (DT104).

S. Enteritidis PT4 became the most prevalent *Salmonella* causing human infection in the United Kingdom during the 1980s and 1990s. This was, in part, due to the fact that chicken eggs can be infected with *S. Enteritidis* PT4 internally or externally by the time they are laid, or can subsequently become contaminated after lay (Advisory Committee on the Microbiological Safety of Food, 1993). Similar problems occurred in the USA, but involved a wider range of phage types.

New Zealand does not appear to have a reservoir of the phage types associated with transovarian egg contamination. The notified human cases of salmonellosis infected with *S. Enteritidis* PT4 have usually recently travelled overseas.

Antibiotic resistant *S. Typhimurium* DT104 is infrequently isolated from humans in New Zealand (39 isolates since 1992, including a small 3 case outbreak in 1997). Of the 39 human isolates 37 were multi-resistant. During the period since 1997 this serotype has only been isolated on 7 occasions from non-human sources (4 bovine, 1 environmental, 1 poultry feed and 1 poultry environment) (Wilson *et al.*, 2000). Three of the non-human isolates have been multi-resistant strains (Carolyn Nicol, ERL, personal communication).

8.1 New Zealand Outbreaks Where a Cereal Grain-containing Product was Listed as a Suspected Food

Relevant outbreaks are summarised in Table 9.

Table 9: New Zealand outbreaks of salmonellosis with either epidemiological (suspected) links or laboratory confirmation linked with cereal grain or cereal grain product consumption 1999 – November 2009

Year	Food Implicated	Setting	Number Ill	Confirmation
2000	Chicken burritos	Restaurant/café	2P	1
2000	Country fried chicken, chicken rolls and sandwiches	Bakery	11C	1
2000	Chicken, apple pie	Home	7C,7P	1
2000	Fish and chips (batter)	Takeaway, home	5P	1
2000	Ham in filled rolls provided to bus tour	Caterers	4C,6P	1
2000	Chicken and lamb kebabs	Takeaway	11C	5
2000	Honey chicken, barbequed pork and rice	Restaurant	11C	5
2001	Chicken panini, infected person	Restaurant, home	2C,1P	1
2001	Lasagne, ducks, infected food handler	Camp	16C	2
2001	Egg and salmon sandwiches	RSA afternoon tea	11C, 10P	5
2001	Egg fu yong, curry beef, chicken fried rice	Takeaway	1C,1P	4
2002	Ham roll	Takeaway	2C	1
2002	Beef schnitzel with egg batter, home-grown vegetables possibly contaminated with animal faeces	Home	1C,2P	1
2002	Club sandwiches with mayonnaise	Cruise ship	23C	1
2002	Potato-topped savories, infected food handler	Bakery, manufacturer of bakery products	24C,1P	4
2002	Tuna sandwiches with raw egg mayonnaise, asymptomatic food handler	Workplace	6C,7P	4
2002	Various bakery goods	Bakery	7C, 4P	5
2003	Untreated roof water supply, filo pastry pie	Holiday home	2C	1
2003	Shanghai style sliced chicken, braised gluten, salty pork and winter melon soup, Shanghai style rice with vegetables in soup, deep fried pork chops	Restaurant/café	3C,2P	4
2005	Shredded chicken noodle salad, chocolate cake	Unknown	2C	1
2005	Club sandwiches	Restaurant/café	3C	1
2005	Smoked chicken lettuce and tomato sandwich	Restaurant/café	2C	1

Year	Food Implicated	Setting	Number Ill	Confirmation
2005	Middle Eastern food: chicken, hummus, flat bread, lettuce, tomato, onions, cabbage	Takeaway	25C	2
2005	Chicken sandwich, bacon and egg pie, panini, fried chicken, chicken roll	Café/bakery	9C, 4P	3
2005	Beef lasagne	Restaurant/café	2C	4
2006	Pizza	Takeaway	1C,1P	1
2006	Taro in coconut cream, BBQ lamb flaps, chop suey in coconut cream, taro and vermicelli, pork buns	Market	11C,4P	1
2006	Egg sandwiches	Restaurant/café	1C,1P	4
2007	BBQ chicken bacon pizza	Takeaway	1C,1P	1
2007	chicken kebabs, lamb kebabs or vegetarian falafels	Takeaway	10C	1
2007	Chicken, taro, chop suey, sweet and sour mince, egg fu yong	Fundraising event	11C,8P	1
2007	Savories, Chicken Nibbles, Bacon & Egg Pies & Sandwiches	Home	1C,3P	1
2008	Flour	Home	67C	3

- 1 epidemiological (suspected) links– cases had history of exposure to implicated source
- 2 epidemiological (suspected) links– case control or cohort study showed elevated risk for cases exposed to implicated source
- 3 laboratory – pathogen suspected to have caused illness identified in implicated source
- 4 environmental investigation (suspected) links – identified critical control point failures linked to implicated source
- 5 pathogen identified in food handler

8.2 Adverse Health Effects Overseas

Table 10 shows the reported incidence of salmonellosis in several countries.

Table 10: Reported incidence data for notified cases of salmonellosis overseas*

Country	Incidence (cases/100,000)	Year	Reference
Australia	43.6	2009	1
Canada	18.0	2006	2
EU total	34.3	2007	3
United Kingdom	22	2007	3
USA	15.2	2009	4

* Does not include *S. Typhi* or *S. Paratyphi*

1 National Notifiable Diseases Surveillance System (NNDSS) <http://www9.health.gov.au/cda/source/CDA-index.cfm>

2 National Enteric Surveillance Program (NESP) <http://www.nml-lnm.gc.ca/NESP-PNSME/index-eng.htm>

3 European Centre for Disease Prevention and Control (ECDC). Annual epidemiological report on communicable diseases in Europe <http://ecdc.europa.eu/en/Pages/home.aspx> (ECDC data presented here relate to the 2007 year)

4 FoodNet – Foodborne Diseases Active Surveillance Network <http://www.cdc.gov/foodnet/>

In terms of the serotypes causing disease overseas, the European Union have collated information on the ten most frequently reported serotypes in 2007 (according to The European Surveillance System “TESSy” for infectious diseases), see Table 11. TESSy represents uploaded case-based and aggregated data that have been approved by each member state and is preferred over the Enter-net method that relies directly on Reference Laboratories or epidemiologists reports.

Table 11: Ten most commonly confirmed human salmonellosis serotypes in the EU, 2007

Serotype	N	%
Enteritidis	81,472	64.5
Typhimurium	20,781	16.5
Infantis	1,310	1.0
Virchow	1,068	0.8
Newport	733	0.6
Hadar	479	0.4
Stanley	589	0.5
Derby	469	0.4
Agona	387	0.3
Kentucky	431	0.3
Other	18,562	14.7
Total	126,281	

Source; EFSA (2009)

The most frequently isolated serotype in Australia during 2007 was *S. Typhimurium* DT135 (722/8,495, or 8.5% of all phage-typed *Salmonella* notifications) followed by *S. Typhimurium* 9 (674, 7.9%), *S. Typhimurium* 44 (460, 5.4%), *S. Typhimurium* 170/180 (337, 4.0%) and *S. Saintpaul* (329, 3.9%). Note that these figures exclude Western Australia as this state ceased routine phage-typing in July 2007 (OzFoodNet, 2008).

8.2.1 Contributions to outbreaks and incidents

Salmonellosis is a significant contributor to infectious intestinal disease incidents and outbreaks in many countries as shown by the data summarised in Table 12.

It is clear from these overseas data that salmonellosis is a significant contributor to foodborne disease, and significant vehicles are poultry meat and eggs.

Table 12: Proportion of foodborne disease in other countries attributed to infection with *Salmonella*

Country	Incidents	Outbreaks	Year(s)	Reference
Australia		50/149 (34%)	2007	(OzFoodNet, 2008)
England and Wales	NS	910/1729 (53%)	1992-2003	(Hughes <i>et al.</i> , 2007)
European Union	NS	490/890 (55%) Verified 1,888/5,332 (35%) All	2008	(EFSA/ECDC, 2010)
Japan	NS	17.2% of cases of known cause, 23.8% of outbreak cases (16.2% were of unknown cause)	1981-95	(Lee <i>et al.</i> , 2001)
Korea	NS	28.3% of outbreaks of known cause, 31.2% of outbreak cases (26.6% were of unknown cause)	1981-95	(Lee <i>et al.</i> , 2001)
Netherlands	14.2% of incidents with known cause (91.7% were of unknown cause)	15.5% of outbreaks of known cause (90.4% were of unknown cause)	1991-94	(Simone <i>et al.</i> , 1997)
Sweden	17.6% of incidents of known cause, 14.5% incident cases (66% incidents were of unknown cause)	17.8% of outbreaks of known cause, 14.5% of outbreak cases (61% of outbreaks were of unknown cause)	1992-97	(Lindqvist <i>et al.</i> , 2000)
Taiwan	NS	3.7% of outbreaks of known cause (51.4% were of unknown cause)	1981-89	(Chiou <i>et al.</i> , 1991)
USA	NS	117/1,270 (9.2%) 127/1,179 (11%)	2006 2001-2005	(Ayers <i>et al.</i> , 2009)

NS = Not Stated

Table 13 gives some examples of salmonellosis outbreaks associated with cereal that have been reported in the literature.

Table 13: Examples of outbreaks of salmonellosis from consumption of cereal grain products overseas

Country	Number involved	Implicated Food and serotype	Year(s)	Reference
Europe				
England	40	Statistical association with yeast flavoured corn snack, <i>S. Manchester</i>	1989	(Joseph <i>et al.</i> , 1991)
England	8	Infant rice cereal, <i>S. Senftenberg</i>	1995	(Rushdy <i>et al.</i> , 1998)
Sweden	110	Dried infant cereal*, consisting of dried milk, flour, oatmeal, potato meal, malt diastase, sugar, salts and vitamins. <i>S. Muenchen</i>	1955	(Silverstolpe <i>et al.</i> , 1961)
North America				
USA	209	Toasted oats cereal, <i>S. Agona</i>	1998	(Anonymous, 1998)
USA	26	Cake batter ice cream, <i>S. Typhimurium</i>	2005	(Zhang <i>et al.</i> , 2007)

* The proposed source of contamination was a consignment of African barley which, during malting, contaminated a Swedish barley consignment. The Swedish consignment contaminated the mill where the oatmeal for the infant cereal was ground. The oatmeal, with no heat treatment, was mixed with the other infant cereal ingredients.

8.2.2 Case control studies

Two case-control studies of salmonellosis in New Zealand have linked increased incidence of the disease to contact with infected animals. One concerned *S. Typhimurium* DT160 (Thornley *et al.*, 2002; Thornley *et al.*, 2003) and the other *S. Brandenburg* (NZFSA, 2002).

The study of *S. Typhimurium* DT160 was prompted by a marked increase in the number of DT160 human isolates which began in May 2001. The epidemic of *S. Typhimurium* DT160 infection among humans occurred in parallel with illness due to the same pathogen in wild birds, particularly sparrows. The organism was also isolated from poultry during 2001.

In addition to telephone interviews of cases (119, median age 8 years and 57% female) and controls (235), environmental sampling was carried out on roof-collected rainwater supplies from the homes of cases, and egg brands consumed by cases. The strongest finding was that there was an association between infection with *S. Typhimurium* and direct contact with wild birds (mOR = 12.3, CI: 2.8-54.6). However, this high risk activity was associated with only a few cases. Questions regarding consumption of a number of pork products were asked, but none were statistically associated with increased risk.

The second case-control study was conducted by ESR in late January 2002 as a component of the NZFSA quantitative risk assessment of *Salmonella* in New Zealand sheep meat (NZFSA, 2002). The aim of the study was to quantify the incidence of human infection with *Salmonella* species, in particular *S. Brandenburg*, and to estimate the contribution of New Zealand sheep

meat consumption to this incidence. The results of the study have now been reported (Baker *et al.*, 2003; Baker *et al.*, 2007). The study recruited 182 cases of salmonellosis, including 43 cases of *S. Brandenburg* infection, with the same number of matched controls.

Factors occurring in the three days prior to illness (or interview) that were significantly associated with an elevated risk of salmonellosis in general were:

- Contact with bird faeces (OR 4.87, 95% CI 1.71, 17.17);
- Contact with other sick people (OR 8.73, 95% CI 2.08, 62.91);
- Consumption of pork steak (OR 5.60, 95% CI 1.11, 72.80);
- Overseas travel (OR 9.97, 95% CI 1.72, 167.46);
- Touching of pet puppies. (OR 6.79, 95% CI 1.33, 73.03); and,
- Use of a kitchen bench, table, or sink for chopping (OR 5.47, 95% CI 1.47, 31.42).

For *S. Brandenburg* infection, two exposures were associated with a significant increase in disease risk:

- Occupational contact with live or dead sheep or lambs (OR 9.97, 95% CI 1.62, 196.29); and,
- Having a household member who had occupational contact with sheep or lamb (OR 4.28, 95% CI 1.23, 21.31).

Overall the study indicated that infection with *S. Brandenburg* had not become a foodborne disease, and instead was an important zoonotic disease representing a risk to farmers and others with direct occupational contact with infected sheep.

8.2.3 Risk assessments and other activity overseas

There were no overseas risk assessments regarding *Salmonella* spp. in cereal grains located. An Australian publication includes a Risk Profile of grains and grain-based products (Sumner, 2002). The author concluded that in general, and as borne out by the epidemiology, the risk of illness from grains products is extremely low. Mycological and chemical hazards were identified and all of these are risk ranked as low. The risk of salmonellosis is not mentioned, but the report recommended surveillance of microbial hazards associated with grain products in the absence of epidemiological evidence linking these products with foodborne disease in Australia.

9 APPENDIX 3: OVERSEAS CONTROL MEASURES

Following a multi-state outbreak of salmonellosis associated with “cake batter” ice cream in the USA, the US Food and Drug Administration issued a Bulletin (USFDA, 2005). The bulletin reminds retail and food service industries that incorporating an ingredient that is intended to be cooked (e.g. dry cake mix) into a ready-to-eat food (e.g. ice cream) poses a serious food safety risk where the product is then not subjected to a process that would destroy harmful micro-organisms. In this particular outbreak, both the sweet cream base mix for the ice cream and the egg in the dry cake mix had been pasteurised. The cake mix was labeled with instructions on how to cook it. Due to the presence of *S. Typhimurium* in the cake mix flour, the way in which the ice cream was constructed resulted in unpasteurised flour contaminating the ice cream. After the products had been mixed together, there was no subsequent processes to eliminate the pathogen prior to freezing.

The dry cake mix was designed to be rehydrated, then cooked and is not considered a ready-to-eat food. Similar products such as “cookie dough” ice creams and “cake mix” milk shakes were also identified by the FDA as posing a serious food safety risk if prepared with ingredients intended to be cooked.

The FDA asked food service operators to review their menus for these types of products. In addition they also advised routine precautionary measures to prevent cross-contamination from raw products and food preparation surfaces.