RISK PROFILE:
SALMONELLA (NON TYPHOIDAL)
IN AND ON EGGS

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

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RISK PROFILE:
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*Risk Profile: Salmonella (Non Typhoidal) in and on eggs* May 2004
SUMMARY

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

The rates of reported salmonellosis in New Zealand have fluctuated considerably since 1985, within the range of 35-65 per 100,000. No clear upward or downward trend is apparent in recent years. The rate of reported salmonellosis in New Zealand is similar to other developed countries (including Australia) although the United States is consistently lower. This may be due to differences in reporting systems.

The serotype information on human isolates of Salmonella reveals a dynamic situation. Two serotypes that have emerged since 1997 have been S. Typhimurium DT160 and S. Brandenburg. In two case-control studies of salmonellosis, egg consumption was not identified as an elevated risk factor. Although eggs and egg dishes have been suspected in approximately 1% of reported outbreaks of salmonellosis since 1997, in only one small outbreak (2 cases), has some supporting laboratory data been obtained.

Eggs are a commonly consumed food in New Zealand, as elsewhere in Western countries. The exposure from this food/hazard combination is likely to be low, for the following reasons:

- the important pathogenic serotype S. Enteritidis PT4 is not established in the New Zealand egg supply;
- two retail egg surveys (South Island in 1994 and Auckland in 2001) have shown an absence of internal contamination of eggs by salmonellae; and
- the South Island survey also showed an absence of external contamination of eggs by salmonellae.

The level of external contamination on eggs requires further investigation however, as the Auckland Survey in 2001 found significant levels of contamination (14% of samples). The serotypes isolated were not the most common serotypes from human cases in New Zealand although they did occur in up to 3% of cases in 2002.

Any survey should examine both internal and external contamination of free range, caged and barn produced eggs. If the high level of external contamination found in Auckland is confirmed, then risk management measures, such as cleaning regimes, will need further investigation.

New Zealand is fortunate in having a poultry industry and egg supply in which types of Salmonella that have caused major problems overseas (S. Enteritidis PT4 and S. Typhimurium DT104) are not endemic. Import controls on poultry are partially designed to maintain this status. New Zealand cases of human illness caused by these types of bacteria appear to be infections principally acquired overseas.
1 INTRODUCTION

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

Figure 1: Risk Management Framework

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In more detail, the four step process is:

1. **Risk evaluation**

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

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

3. Implementation of the risk management decision

4. Monitoring and review.

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

The Risk Profiles also provide information relevant to risk management. Based on a Risk Profile, decisions are made regarding whether to conduct a quantitative risk assessment, or take action, in the form of gathering more data, or immediate risk management activity. This Risk Profile concerns non-typhoidal *Salmonella* spp. in and on eggs.

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

*Hazard identification, including:*

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

*Hazard characterisation, including:*

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

*Exposure assessment, including:*

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

*Risk characterisation:*

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

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

Conclusions and recommendations for further action.
HAZARD IDENTIFICATION: THE ORGANISM

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

2.1 Salmonella

2.1.1 The organism

This group of organisms is comprised of two species: *Salmonella enterica*, which is divided into 6 subspecies, and *Salmonella bongori* (Jay et al., 1997). Most isolates from humans and warm-blooded animals belong to subspecies I: *Salmonella enterica* subspecies *enterica*. Other *Salmonella enterica* subspecies and *Salmonella bongori* occur more commonly from cold-blooded animals and the environment, and are of lower pathogenicity.

*Salmonella* typing is performed using serological identification of both the somatic (O), and flagella (H) antigens.

*Salmonella enterica* serotypes are normally denoted in a shortened form that includes a non-italicised serotype name, e.g. *Salmonella enterica* subsp *enterica* serovar 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 a differing ecology to other serotypes of *Salmonella*. They are not included in this Risk Profile.

2.1.2 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 and the data are still not universally accepted and doubts surrounding the experimentation noted (International Commission on Microbiological Specifications for Foods, 1996).

pH: Minimum 3.8, optimum 7-7.5, maximum 9.5. The minimum pH is influenced by other factors such as temperature, the acid present, and the presence of nitrite etc. For example, at 10°C the minimum pH allowing growth was 4.4-4.8 (13 isolates tested), while at 30°C it was 3.8-4.0.

Atmosphere: Can grow in the presence or absence of air. Growth under nitrogen is only slightly less than that under air. Grows at 8-11°C in the presence of 20-50% CO₂. Growth at low temperatures is retarded in the presence of 80% CO₂ compared to air.

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

*Salmonella* is known to survive well in foods, on surfaces and in the environment. A correlation has been shown between the ability of isolates to survive heat, acid and hydrogen peroxide and their ability to survive on surfaces (for instance, *S. Enteritidis* (Humphrey *et al*., 1995)).

**Temperature**: *Salmonella* can survive for long periods under refrigeration. Survival for >10 weeks in butter held at temperatures between –23 and +25°C has been noted. *Salmonellae* can survive for 28 days on the surfaces of vegetables under refrigeration. Some foods, including meat appear to be protective of *Salmonella* during freezing (International Commission on Microbiological Specifications for Foods, 1996).

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

2.1.3 **Inactivation (CCPs and Hurdles)**

**Temperature**: Death can occur during the freezing process, but those that survive remain viable during frozen storage. Freezing does not ensure the inactivation of salmonellae in foods. Note that in microbiological terms “D” refers to a 90% (or decimal or 1 log cycle) reduction in the number of organisms.

D times: 60°C usually 2-6 min; 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 important as a food pathogen (Doyle and Mazzotta, 2000). D times in intact eggs have been reported as 4.5 and 6.0 minutes at 58 and 57°C respectively (Schuman *et al*., 1997).

N.B. D times for *Salmonella* can depend on the type of food involved. Extremely high D times have been reported for experiments with milk chocolate. Values reported were up to 1050 min at 70°C, 222 min at 80°C and 78 min at 90°C. This also applies to other low water content foods.

**pH**: Inactivation at sub-optimal pH depends on many factors including the type of acid present and the temperature. Reducing pH reduces heat resistance.

**Water activity**: Decline in numbers is greatest at water activities just below that allowing growth. Lower \(aw\) values appear to have a protective effect.

**Preservatives**: Salmonellae are not unusually resistant to preservatives commonly used in foods. Preservative action can be enhanced by the use of several factors such as reduced pH and temperature in combination, see above pH comments.

**Radiation**: D value around 0.5 kGy, up to 0.8.
2.1.4 Sources

Human: Faeces of infected people contain large numbers of the organism and shedding may continue for up to 3 months. The median period for shedding is 5 weeks. A small proportion (<1%) of cases become chronic carriers.

Animal: Some serotypes are confined to particular animal reservoirs, but many are capable of crossing between species to cause disease in humans, by direct contact and via food. Most Salmonella infections in animals are asymptomatic. Poultry and pigs are regarded as major reservoirs of the organism although in New Zealand, carriage rates in poultry have dropped considerably throughout the 1990s. Animal feeds made from animal products may be contaminated by salmonellae. Salmonellae can also be found in fish, terrapins, frogs and birds.

Food: Meat or other products derived from infected animals can be important vehicles of salmonellosis. Other animal products, e.g. eggs, unpasteurised or re-contaminated pasteurised milk and dairy products, can also act as vehicles.

Environment: Salmonellae shed in faeces can contaminate pasture, soil and water. The bacteria can survive for months in soil. Contamination in the environment by salmonellae from animal sources can act as a source of infection of other animals.

Transmission Routes: May be transmitted to humans via contaminated food or water, animal contact, or from a contaminated environment. A simple overview is a cycle of events involving feedstuffs, animals, foodstuffs, and then people.

2.2 Salmonella Serotypes in New Zealand

The non-typhoidal Salmonella are divided into approximately 2000 serotypes. Most of these are capable of causing disease in humans, although a few have restricted host ranges.

The ESR Enteric Reference Laboratory at the Kenepuru Science Centre provides Salmonella typing services for New Zealand. In addition to isolates from human cases sent by clinical laboratories, the laboratory also provides typing for isolates from animals and foods submitted by various sources. Isolates derived from poultry originate directly from poultry producers, commercial diagnostic laboratories and from Veterinary Pathology Laboratories. Summaries of the isolates submitted by Veterinary Pathology Laboratories are published in the MAF Biosecurity Authority journal Surveillance. All isolates are reported by the Enteric Reference Laboratory as annual and quarterly tables on the ESR website www.esr.cri.nz under publications.

Detailed information on serotypes from poultry and human isolates in New Zealand is given in Sections 5 and 6 respectively.

2.2.1 S. Enteritidis Phage Type 4 and S. Typhimurium Definitive Type 104

Two serotypes that have caused major problems overseas are S. Enteritidis Phage Type 4 (PT4) and the antibiotic resistant S. Typhimurium Definitive Phage Type 104 (DT104).
S. Enteritidis PT4 is a phage type that has the ability to infect egg contents by transovarian transmission. To date, these phage types have not been detected in New Zealand’s broiler or egg laying flocks.

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 hen 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).

The number of reported cases of salmonellosis in the United Kingdom during the 1990s was relatively constant at around 30,000 cases per year (IFST, 1997). The most common Salmonella involved was S. Enteritidis, followed by S. Typhimurium. The antibiotic resistant S. Typhimurium strain DT104 made up an increasing proportion of the S. Typhimurium isolates from 1991 to 1996. This presented difficulties in treatment, and a relatively high mortality rate (3%) occurred amongst cases. The most common food sources were comminuted meats, especially sausages and burgers.
3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: Eggs

Most eggs for human consumption are derived from hens, but eggs from other birds such as ostriches, ducks, and quail are also consumed. Eggs are generally marketed and consumed as shell eggs. For commercial use, and in food service operations, eggs are broken from their shells, and may then be mixed whole or separated into whites and yolks. Further processing includes pasteurisation, drying, and possibly mixing with other ingredients. Eggs are made up of:

- The cuticle, a largely proteinaceous coating on the exterior of the shell;
- The shell, which is mostly calcium carbonate;
- The outer coarse membrane;
- The inner fine membrane;
- The outer thin white;
- The thick white;
- The inner thin white;
- The chaliziferous layer which anchor the yolk; and
- The yolk

The cuticle makes the shell resistant to the entry of water through the shell’s pores, and consequently also provides protection against bacterial contamination. Cuticle damage, for example by abrasive cleaning, increases the risk of microbial contamination.

The egg’s contents are moist (approximately 64% water) and contain the nutrients required for bacterial growth. Barriers to prevent bacterial contamination of the contents include the physical barrier of the cuticle, shell and associated membranes, and antimicrobial components present in the egg contents (International Commission on Microbiological Specifications for Foods, 1998). Cracks allow penetration by bacteria through physical barriers, and faecal contamination of the shell surface provides a challenge likely to result in greater and earlier penetration. When eggs are immersed in a bacterial suspension of a lower temperature than the internal egg temperature, a pressure gradient is set up and bacteria are drawn in through the shell (Moats, 1978).

The egg white contains a number of antibacterial factors including lysozyme and conalbumin, and is also of a high pH (9.1-9.6) which is unfavourable for the growth of most bacteria. Lysozyme degrades the bacterial cell wall and is most effective against Gram positive organisms while conalbumin sequesters metal ions needed by bacteria for growth.

The yolk is as rapidly perishable as milk, yet with the protection of the shell and antimicrobial components of the egg white, the contents of an egg can remain edible for months, even when stored at room temperature (ICMSF, 1998).
### 3.2 Contamination of Eggs by *Salmonella*

#### 3.2.1 Contamination of egg contents

*Salmonella* can contaminate eggs internally by two routes:
- **transovarian** - a vertical transmission; and
- **trans-shell** - a horizontal transmission

Vertical transmission is considered by FAO/WHO (2002:Interpretative Summary;p4) to be the major route of *Salmonella* contamination and is more difficult to control, while horizontal transmission, usually derived from faecal contamination on the eggshell can be effectively reduced by cleaning and disinfection of the environment (FAO/WHO, 2002). In the New Zealand context however, this statement regarding vertical transmission does not apply because the particular types of *S.* Enteritidis that infect egg contents by transovarian transmission have not been detected in New Zealand poultry flocks.

Currently, therefore, incidents of salmonellosis in New Zealand where eggs are implicated are likely to be caused by initial external contamination of the egg rather than transovarian contamination of the egg contents. Transovarian transmission is described briefly below for informative purposes.

**Transovarian (vertical) transmission**: This unusual ability by certain *Salmonella* serovars to colonise and infect hen ovaries or oviduct tissues may result in transfer of salmonellae to the yolk or albumen, prior to formation of the shell or shell membranes. The emergence of *S.* Enteritidis since the late 1970s as the leading cause of human salmonellosis in many countries (not New Zealand) is attributed to this unusual ability. There have been suggestions that *S.* Enteritidis is better able to achieve invasion of such tissues than other species (Humphrey, 1994). However, other *Salmonella* species are also occasionally found in reproductive tissues (e.g. *S.* Typhimurium and *S.* Heidelberg (Poppe *et al.*, 1998)), and this point is not yet clear (Advisory Committee on Microbiological Safety of Food, 2001).

Host specific poultry pathogens; *S.* Gallinarum and *S.* Pullorum, can also be isolated from hen reproductive tissues. These serovars are not believed to be present in the New Zealand poultry flock. MAF reports annually to the World Organisation for Animal Health (OIE) on the status of *S.* Pullorum, which has not been isolated in New Zealand since 1985 (*S.* Gallinarum is not reportable to OIE).

**Trans-shell (horizontal) transmission**: In fowl, the intestinal, urinary and reproductive tracts share a common orifice so surface contamination (usually faecal) of the egg when laid can occur. This may result in egg contents becoming contaminated via microorganisms penetrating through pores or cracks in the shell. The shell has between 7000 to 17,000 pore canals. They are more numerous and larger at the blunt (larger) end. During the first 24 hours after lay, carbon dioxide is lost through the shell, and this results in the pH of the albumen rising to approximately 9.0, thus acting as an antimicrobial defence mechanism.

#### 3.2.1.1 Contamination of eggs contents through the shell

A result of the importance of *S.* Enteritidis in causing infections overseas is that there has been considerable research into this serotype. The information in this section regarding
trans-shell transmission pertaining to S. Enteritidis can be applied in a generic manner to all non-typhoidal salmonellae with the exception of S. Senftenberg, which is less heat sensitive than other serotypes.

The mechanisms for contamination and the properties of the egg that reduce contamination of the contents have been reviewed (Mayes and Takeballi, 1983). Some published reports have suggested a relationship between eggshell quality and bacterial penetration. Measures of eggshell quality include conductance (a measure of porosity), and shell strength and thickness. The latter is positively correlated with specific gravity. The ICMSF state that the number of pores per egg increase with an ageing flock which gives a lower specific gravity for eggs from older hens (ICMSF, 1998). While decreasing specific gravity of eggs has been associated in some reports with an increase in ability for Salmonella to penetrate shells, an examination of eggs from flocks of various ages found no correlation with specific gravity (Berrang et al. 1998). The same study found that generally eggs from older birds were more prone to invasion when immersed in a suspension of the pathogen, but this was not statistically significant. Consequently, factors other than shell quality were considered to be important for the penetration of Salmonella through the shell. Shell strength is influenced by two factors:

- the hen's diet, particularly its calcium, phosphorus, manganese and vitamin D intake;
- the egg size, which increases as the hen ages while the mass of shell material that covers it stays fixed. Hence the shell is thinner on larger eggs (see: http://newton.ex.ac.uk/teaching/CDHW/egg/index.html).

Penetration of salmonellae applied to the outside of eggs in chicken faeces has been shown when the eggs were incubated at 25°C for three days (Schoeni et al., 1995). A lesser degree of penetration was demonstrated at 4°C. Greater penetration was observed when conditions simulating those encountered in the hatchery were replicated. The variability in penetration by salmonellae of individual eggs has been shown (Stokes et al., 1956). This latter study noted the penetration of eggs when stored at 1°C but only when a high number of cells were used as an inoculum. When penetration did occur, growth did not unless the eggs were transferred to a higher temperature.

The age of the egg has been shown to influence the degree of penetration by S. Enteritidis and S. Typhimurium (Miyamoto et al., 1998), with eggs 0.25-3h old being more susceptible to penetration than older eggs. Penetration could be reduced by cooling eggs prior to exposure to the pathogen. The susceptibility of eggs immediately after laying (1-3 minutes old) has also been shown for bacteria in general (Sparks and Board, 1985).

Growth of Salmonella Enteritidis is rapid in egg yolk at 25°C and slows, as would be expected, with decreasing temperature, with minimal growth occurring at 10°C (Gast and Holt, 2000; Humphrey et al., 1989), while growth in the albumen is slower (Baron et al., 1997). High numbers (around 10^8/ml) were reached in yolk after 2 days incubation at 25°C, whereas no growth occurred in albumen at 10°C. Salmonella Typhimurium has been shown to grow in whole and blended eggs at 12°C, but not at 7°C when incubated for 24h (Baker et al., 1983). At 37°C, S. Enteritidis gradually declined in egg albumen, but persisted in the yolk (Baker, 1990).
Different isolates have been shown to reach different maximum numbers in liquid whole egg incubated at 37°C (Gast and Holt, 1995). Twelve isolates were tested and the final numbers attained varied by 3.5 log_{10} units, with some reaching numbers in excess of 7.5 log_{10}/g. This effect was largely abolished by the addition of iron and so presumably reflects differences among the isolates in their ability to sequester iron.

Contamination of eggs at 25°C where S. Enteritidis was introduced onto the inner membrane of the air cell was determined to be greater when the air cell was uppermost than when it was downwards (Clay and Board, 1991). No growth was detected with incubation at 4°C. The growth of S. Enteritidis in the yolk of eggs which had their albumens inoculated did not occur in eggs less than three weeks old and stored at 20°C (Humphrey and Whitehead, 1993). However, growth has been reported in eggs incubated at 8°C for two of three isolates after only a few days (Baker, 1990) (although most yolks remained negative and the contamination did not seem to increase with incubation period). Growth after this time appeared to be associated with a change in the membrane permitting invasion of the yolk or leakage of nutrients from the yolk into the albumen. Growth of the organism was minimal in albumen separated from the yolk.

The survival of S. Enteritidis on the shell and membrane at 20°C has been shown to be dependent on relative humidity, with increased survival at higher RH values (Himathongkham et al., 1999).

Numbers of organisms present initially may be very small, and they are unlikely to grow until such time as they can penetrate the vitelline membrane and contaminate the yolk. Depending on temperature, replication can then be rapid and high numbers attained.

### 3.2.2 Egg shell contamination

A range of *Salmonella* serotypes have been isolated from egg shells. This may be the result of infection of the lower reproductive tract or faecal contamination from hens with gastrointestinal infection with *Salmonella*. Further shell contamination may occur from the environment into which the eggs are laid.

The survival of S. Enteritidis on the shell surface is relatively short (a few days) when applied as an aqueous suspension (Baker, 1990). Survival was better at 7°C than it was at room temperature.

### 3.3 Egg Production with Respect to Salmonella

This section has been split into three parts; the layer hen, the egg, and pasteurisation/cooking controls.

#### 3.3.1 The layer hen (on-farm)

Poultry can become colonised by pathogens via drinking water, feed, or pecking in contaminated soil or litter (ICMSF, 1998). Bird management and the differences between caged, barn and free range systems are detailed in the Egg Producers Federation’s Technical Annex:(C41-48), brief comments on birds, feed, water and environmental factors are as follows:
**Birds:** Acquiring birds from *Salmonella*-free parent flocks is an important means to reduce infection. Competitive exclusion is used effectively in some countries to reduce the likelihood of establishment of salmonellae in very young chicks. This involves the provision of a culture preparation made from the intestinal flora of healthy birds to assist in the early establishment of a favourable intestinal flora. Alternatively, a *Salmonella* vaccine may be used to prevent infection of poultry with the organism. More information on vaccines can be found in section 7.1.3.

Layer hens should be raised under conditions that minimise stress. It has been reported (Nascimento and Solomon 1991) that stress can cause oviduct damage leading to ultrastructural defects in the eggshell, increasing the susceptibility to bacterial invasion.

**Feed:** Contaminated feed is often a significant source of salmonellae on the farm, attributed to animal derived ingredients such as rendered animal by-products, and fishmeal. Provision of *Salmonella*-free feed is regarded as an essential factor. For broiler feed, this is often done by processing feed into pellets, with the associated heating which is effective in eliminating *Salmonella*, provided recontamination is prevented. Layer feed is mostly (unpelleted) mash, which does not receive adequate heat treatment to kill pathogens. The addition of organic acids is often used to reduce contamination in mash, which has the advantage of protecting feed against recontamination during storage and distribution. Where feed can become wet (free range), it should be provided in suitable daily quantities only.

**Water:** Open troughs can be a source of contaminated water through litter, feed, faeces etc. Untreated water can potentially transmit infection. Chlorination of drinking water has been cited as a control measure (ICMSF, 1998).

**Environmental:** Cages, litter and nesting materials should be clean. Where possible they should also be kept free from faeces. Pathogen transmission can be reduced by a variety of on farm measures such as cleaning and disinfecting houses between successive flocks. Vermin control is also important in preventing spread of pathogens. Rodents, insects, birds and domestic pets have been suggested as potential sources of *Salmonella*.

### 3.3.2 The egg: collection, handling and washing

Egg collection and grading is discussed in more detail in the Egg Producers Federation’s Technical Annex:(C48-57), some comments on collection and washing are as follows;

Egg collection on small farms is often by hand and should be at least daily, as often as every four hours is ideal. Usually caged hen eggs in larger establishments have semi-automated collection (eggs roll by gravity from cage to collection trough). This type of system has been reported to produce lower contamination rates than eggs laid into nests and the shorter the time between the egg being laid and collected, the lower the contamination of the shell, even under unfavourable conditions (ICMSF, 1998). The eggs are then transferred usually by hand to paper or polystyrene trays to be candled and graded. Eggs are stored blunt end up, this prevents the yolk from drifting towards the inner membrane, bypassing the protective barriers in the egg white and possible contamination from any microorganisms that have penetrated to the membrane.
Where permitted, the eggs may be transported to a washing station. Factors affecting microbial penetration and spoilage when washing eggs are discussed by (Stadelman, 1994). A brief listing of these, together with washing recommendations by (ICMSF, 1998), can be found in the Technical Annex, (Egg Producers Federation of New Zealand, 2002, C-3:50).

Egg handling can involve washing eggs in sanitisers. However there is some evidence that washing increases contamination in both whole and cracked eggs (Vadehra et al., 1969), an effect which is most pronounced when the eggs were subsequently stored at 23°C rather than 10°C. The inclusion of chicken manure in the wash water increased the number of contaminated eggs, but the same effect was observed when sterile chicken manure was added. Cracked eggs showed a greater propensity toward contamination than did whole eggs. Also the nature of chemicals used for eggs washing has been reported to influence the egg’s subsequent susceptibility to bacterial penetration, possibly due to their effect on the cuticle (Wang and Slavik, 1998).

The pH of the washwater has been shown to be important for the survival of S. Enteritidis and the ability of the organism to cross contaminate from inoculated to uninoculated shell egg surfaces (Catalano and Knabel, 1994). Washwater at pH 11 and 37.7°C was determined to prevent cross contamination and reduce the level of contamination on eggs occurring prior to washing. Slow chilling of the eggs to 7.2°C over 2-3 days permitted greater survival on the shell, and penetration into eggs washed at pH 9.

Egg washing has been reviewed (Moats, 1978). Factors favouring bacterial invasion (related to spoilage in this review) include washing in water colder than the eggs, washing in water with high bacterial counts, washing in water containing appreciable soluble iron, and washing in machines with surfaces contaminated by bacteria.

3.3.3 Pasteurisation and cooking controls for *Salmonella* in eggs

Effective pasteurisation of intact shell eggs inoculated with around $10^8$ S. Enteritidis has been described at 58°C and 57°C although the quality of the albumen was affected. However, immersion-pasteurised eggs were still suitable for numerous culinary uses (Schuman et al., 1997). Other investigations of whole egg pasteurisation found a 3 log reduction when eggs were heated in a circulating water bath operating at 57°C for 25 minutes and a 5 log reduction when eggs were exposed to dry heat for 180 minutes at 55°C. A combination of the two, with the dry heat stage reduced to 60 minutes produced a 7 log reduction (Hou et al., 1996). This process is claimed to give a 6 log margin of safety when considering levels of S. Enteritidis normally found in eggs.

D values for a six isolate mixture of salmonellae in liquid egg yolk were 0.57 minutes at 61.1°C, 0.20 minutes at 63.3°C, and <0.20 minutes at 64.4°C (Palumbo et al., 1995). However, the addition of NaCl or sucrose increased the D time (increased thermal resistance). For example the D time at 63.3°C was 11.5 minutes when the yolk contained 10% NaCl. When $a_w$ lowering solutes were added the thermal death curve showed distinct shoulders and tailing, i.e. was not log linear. The log reductions calculated for various egg products pasteurised to standards were as below:
### Product

<table>
<thead>
<tr>
<th>Salmonella</th>
<th>Temp (°C)</th>
<th>Time (min)</th>
<th>Log_{10} reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Egg yolk</td>
<td>61.1</td>
<td>3.5</td>
<td>6.14</td>
</tr>
<tr>
<td>Egg yolk + 10% sucrose</td>
<td>63.3</td>
<td>3.5</td>
<td>4.86</td>
</tr>
<tr>
<td>Egg yolk + 10% NaCl</td>
<td>63.3</td>
<td>3.5</td>
<td>0.30</td>
</tr>
<tr>
<td>Egg yolk + 5% NaCl + 5% sucrose</td>
<td>63.3</td>
<td>3.5</td>
<td>0.43</td>
</tr>
<tr>
<td>Egg yolk + 20% NaCl</td>
<td>64.4</td>
<td>3.5</td>
<td>0.76</td>
</tr>
</tbody>
</table>

In experiments using 9 strains of S. Enteritidis, the D time at 60 minutes varied from 0.69 to 0.31 minutes (Baker, 1990). The mean D time was 0.42 minutes with a standard deviation of 0.1 minute.

Similar experiments have been carried out with liquid egg whites (Palumbo et al., 1996). The pasteurisation of egg white is difficult because of the lack of functionality of the food on heating i.e albumen is denatured in a few minutes at or above 60 °C. Homogenised whole egg and yolk are reasonably stable at this temperature (ICMSF, 1998). Pasteurisation standards in the USA allow for heating regimes where hydrogen peroxide is added to the egg white, with residual peroxides being removed by the addition of catalase, the addition of hydrogen peroxide increases the heat sensitivity of salmonellae. D times for a six isolate mix of salmonellae were 3.87 minutes at 51.5°C and 1.60 minutes at 53.5°C in the presence of 0.875% H_{2}O_{2} in egg white of pH 8.8. In the absence of hydrogen peroxide, D times were 2.74 minutes at 55.5°C, 1.44 minutes at 56.6°C and 0.78 minutes at 57.7°C. The log_{10} reduction of S. Senftenberg in pasteurisation processes meeting the US standards during plate pasteurisation varied from 3.64 to 1.80. pH was also an important factor with the log_{10} reduction at 56.6°C for three minutes being 3.60 at pH 7.8 and 1.08 at pH 9.3. This is important as the pH of egg white rises from around 8.2 to 8.9-9.1 with time. It was concluded that the current time and temperature combinations do not provide a 99.99% reduction in salmonellae in commercial egg white. Pasteurisation controls in New Zealand are discussed in section 7.1.4.

D times for a number of Salmonella isolates heated in liquid egg white and yolk are presented below (Chantarapanont et al., 2000):

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Phage type</th>
<th>D Value (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>in liquid albumen at 52°C</td>
<td>in liquid yolk at 56°C</td>
</tr>
<tr>
<td>Enteritidis</td>
<td>4</td>
<td>5.18</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3.82</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>4.49</td>
</tr>
<tr>
<td></td>
<td>13a</td>
<td>4.57</td>
</tr>
<tr>
<td></td>
<td>34</td>
<td>3.91</td>
</tr>
<tr>
<td></td>
<td>34</td>
<td>3.76</td>
</tr>
<tr>
<td>Senftenberg</td>
<td>13.43</td>
<td>19.96</td>
</tr>
</tbody>
</table>

The medium in which the inoculum was grown has been shown to influence the D value of salmonellae in liquid egg (Muriana et al., 1996).
Experiments with eggs cooked to simulate whole, boiled, fried and scrambled eggs have shown that salmonellae in the yolks can survive while the yolk is still liquid (Humphrey et al., 1989). Whole eggs inoculated with around 10^7 salmonellae/g still contained viable organisms in the yolk after 4 minutes boiling (the temperature of the yolk reached around 56°C). Eggs containing the same inoculum fried “sunny side up” still contained countable numbers of organisms, while those cooked “over easy” could still yield the inoculum after enrichment. Different approaches to cooking scrambled eggs gave different inactivations of the inoculum, depending, unsurprisingly, on the final temperature reached. Microwave cooking could be as efficient as cooking in a pan. At times normally used for boiling eggs, the inoculum size was not correlated with overall survival as long as the yolk was still liquid after cooking.

For S. Typhimurium inocula of around 10^4-10^5 cells/g were only destroyed when the yolk of a boiled egg reached 75.4°C (Baker et al., 1983). Poaching and the cooking of omelettes were found to destroy the organism, whereas eggs fried “sunny side up” were not free of the inoculum.

In an investigation into hard-cooking methods, eggs inoculated with 10^7-10^8 salmonellae inoculated into the yolk were cooked according to two methods (Chantarapanont et al., 2000):

- the American Egg Board method which is to place eggs into water at 23°C, heat to 100°C, remove from the heat and hold for 15 minutes; and,
- placing eggs in water at 100°C, then holding for 15 minutes at that temperature.

As might have been expected, inactivation was slower in eggs at an initial temperature of 10°C than 21°C, and slower in large eggs than medium eggs. *Salmonella* could be recovered from eggs cooked by the first method for up to 9 minutes, whereas after 9 minutes at the second method, *Salmonella* could still be recovered from extra large eggs. It was generally recommended that regardless of the method used to hard cook eggs, sufficient time in boiling water should be given to completely solidify the yolk.

In further studies some consideration of time and temperature has been made. The results are summarised below:

<table>
<thead>
<tr>
<th>Cooking method</th>
<th>Inoculum (cfu/ml)</th>
<th>Time needed for complete kill</th>
<th>Final temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scrambling</td>
<td>4.2 x 10^5</td>
<td>2 min.</td>
<td>74</td>
</tr>
<tr>
<td>Poaching</td>
<td>3.2 x 10^4</td>
<td>5 min.</td>
<td>75</td>
</tr>
<tr>
<td>Boiling</td>
<td>5.9 x 10^4</td>
<td>7 min.</td>
<td>75</td>
</tr>
<tr>
<td>Frying:</td>
<td>2.7 x 10^5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Covered</td>
<td></td>
<td>4 min.</td>
<td>70</td>
</tr>
<tr>
<td>Sunnyside up</td>
<td></td>
<td>7 min.</td>
<td>64</td>
</tr>
<tr>
<td>Turned over</td>
<td></td>
<td>3 + 2 min.</td>
<td>61</td>
</tr>
</tbody>
</table>
When hands were used to crack eggs inoculated with S. Enteritidis up to 25% of fingers tested were positive for *Salmonella*, and even after washing 1.8% of fingers remained positive (Humphrey *et al.*, 1994). When batter mixes were prepared with an electric mixer, *Salmonella* was dispersed up to 40cm from the mixing bowl without any obvious associated splashing of the mix. Most importantly S. Enteritidis PT4 survived for 24 h on a soiled formica surface even when present at low initial numbers. Cross contamination was almost instantaneous when ready-to-eat food was placed onto egg droplets containing this organism (Bradford *et al.*, 1997). Somewhat longer exposure was required for transfer when the egg droplets were allowed to dry.

### 3.4 The Food Supply in New Zealand

The poultry industry in New Zealand is divided into two main sectors:

- poultry meat production including livestock breeding; and,
- table egg production.

These are linked via commercial hatcheries and large feedmills. Differences occur in breeds of hen, i.e. broiler hens are mainly Ross or Cobb and layers are usually Hyline. The hatcheries for meat and egg production are also physically separated. The hatcheries and feedmills are generally owned by the vertically integrated poultry processing and breeding companies (PIANZ, 1999). There are some exceptions such as a number of smaller layer farms that produce their own feed.

The majority of egg production is from caged hens, with a small percentage produced by barn raised or free range hens.

Annual retail sales of poultry meat, eggs and compounded stockfeed, now account for $1200 million. The majority is domestically consumed, with small quantities of live chicks, hatching eggs, poultry meat and table eggs exported to Papua-New Guinea and the Pacific Islands.

At 31 December 2003, New Zealand had approximately 2.9 million commercial layer hens for egg production, producing approximately 72 million dozen eggs for sale. In 2003, New Zealand had 170 commercial egg producers, with the largest 20 producers accounting for 50% of annual production. (PIANZ, pers.comm., 2004).

Over 85% of eggs (61 million dozen) are sold as table eggs within New Zealand, the remaining 15% going to the catering and bakery industries. Since deregulation in the late 1980s, there has been a decline in the number of commercial egg producers (Egg Producers Federation of New Zealand, 2002, Technical Annex C-3).

### 3.4.1 Imported food

Small amounts of egg derived products are imported into New Zealand. This is mostly dried egg contents. Import statistics for the year ending March 2003 indicated approximately 69 tonnes of dried egg contents were imported, mainly from Canada.
4  HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

*Salmonella* possess systems that enable them to adhere to small intestinal epithelial cells, provided they survive the low pH of the stomach, and other defence mechanisms (Jay *et al.*, 1997). After entering the cell as part of a vesicle (endosome), non-typhoidal salmonellae multiply and release endotoxin. The invading bacterial cells often cause only a limited, localised intestinal event with no systemic involvement, resulting in damage to the mucous membrane of the small intestine and colon. Both invasion and enterotoxin production are required to cause diarrhoea. A small proportion of cases may experience septicemia or longer-term illness, such as reactive arthritis.

In contrast, *Salmonella* Typhi enter the gastrointestinal tract, invade the local lymphatic tissue and pass via the blood stream to various organs (Jay *et al.*, 1997). The discussion below pertains only to non-typhoidal *Salmonella* infections.

4.1 Illness and Symptoms

**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. Endotoxins are produced in the intestines after infection.

**People Affected:** The young, old, and immunocompromised are particularly at risk. In addition people of less privileged socioeconomic groups and those living in higher population densities are more at risk.

**Long Term Effects:** Septicaemia and subsequent non-intestinal infections can occur. Reactive arthritis or Reiter’s syndrome may occur 3-4 weeks after gastrointestinal symptoms. Approximately 2% of a population exposed to a triggering infection will develop reactive arthritis, which may last for up to a year or longer (Smith, 1994).

**Treatment:** The infection is usually self-limiting although fluid replacement may be required. Antibiotic treatment seems to be either ineffective or results in relapse or prolonged faecal shedding. Certain groups, e.g. new born children, may benefit from antibiotic treatment.

4.2 Dose Response

Besides the number of bacterial cells, the probability that a given dose will cause disease depends on factors such as age and health status of the person, and the food(s) consumed with the cells.

Doses generally recognised to cause disease at high attack rates are in the range of $10^5$ to $10^6$ cells. Low attack rates have been observed in one outbreak where 4-45 cells were consumed,
another where the dose was 6 cells/65g of ice cream (Vought and Tatini, 1998) and another where the number of cells eaten was estimated at 0.7 to 6.1 in six different cases (D’Aoust, 1985). Different serotypes may have different dose responses.

However, these observations simplify a situation whereby there is no threshold dose for infection. The FAO/WHO *Salmonella* in broilers and chicks risk assessment produced a dose response curve based on outbreak data involving principally the *S. Enteritidis* serotype (Anonymous, 2001:p86). It was not possible to get a statistically significant single best fit curve for all the outbreak data, but broadly a Beta-Poisson curve fit was used to generate approximate bounds for the dose response. The graph shows, at the median of the dose response curve, that an ingestion of $10^{10}$ cells results in a probability of around 0.9 of illness, while the ingestion of $10^5$ cells results in a probability of around 0.02. This kind of curve explains that low levels of cells are capable of causing disease, and how outbreaks where the food has been widely consumed but only a small proportion of consumers have become ill can occur.

Dose response models have been developed for individual *Salmonella enterica* serotypes. For example, models have been compared with data for *S. Typhosa* (Holcomb et al., 1999) and one has been produced for *S. Meleagridis* (Teunis et al., 1999). The former study indicated that at ingestion of $10^2$ or less the probability of disease is very low, a dose of $10^8$ gives a probability of around 0.8, and exposures above $10^{11}$ are needed to obtain probabilities approaching 1. The curve for the *S. Meleagridis* model is much steeper, with probabilities of disease approaching 0 at doses $<10^4$, and approaching 1 at doses exceeding $10^9$. These dose response curves are therefore somewhat shifted to a higher dose being required to cause the same probability of disease when compared to the FAO/WHO model based on outbreak data.

It has been observed that foods with high fat content, like chocolate, peanut butter and cheese may protect cells from gastric juices so permitting a lower dose than usual to cause infection (D’Aoust, 1985). Experimentation has 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 is crucial in determining its resistance to stomach acids (Waterman and Small, 1998).
5 EXPOSURE ASSESSMENT

5.1 The Hazard in the New Zealand Food Supply: *Salmonella* in Poultry

5.1.1 *Salmonella* in poultry (broilers)

The National Microbiological Database, now administered by the NZFSA, collates information derived from microbiological monitoring of the effects of slaughter and dressing processes. Procedures for poultry (broilers only) were introduced in June 2001 (see: [http://www.nzfsa.govt.nz/meatdoc/programmes/nmd/poultry/index.htm](http://www.nzfsa.govt.nz/meatdoc/programmes/nmd/poultry/index.htm)). Participation by the industry is voluntary but is strongly supported by the Poultry Industry Standards Council. Current participation covers approximately 96% of poultry production in New Zealand (Roger Cook, NZFSA, personal communication). Samples are taken from the production line after chilling and draining/dripping, but before manual grading, mixing in bins, bagging or further processing. Data supplied by PIANZ indicates that the *Salmonella* prevalence levels are: 2001 2.1% (data from third and fourth quarters only), 2002 1.0%, 2003 2.0%. These levels are low by international standards.

However, these data apply to broilers only. No information on the *Salmonella* status of layer hens in New Zealand has been located. It is likely that the *Salmonella* status of layer flocks is different for the following reasons (Sharon Wagener, NZFSA, pers. comm.):

<table>
<thead>
<tr>
<th>Factor</th>
<th>Layer Hens</th>
<th>Broilers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheds</td>
<td>usually kept in multi-age sheds, sheds rarely emptied</td>
<td>sheds depopulated, sanitized and fumigated between runs</td>
</tr>
<tr>
<td>Lifespan</td>
<td>kept for longer, possibly greater exposure to pathogens</td>
<td>usually slaughtered at less than 6 weeks, less opportunity for infection</td>
</tr>
<tr>
<td>Environment</td>
<td>majority are caged (small percentage barn raised/free range)</td>
<td>barn raised</td>
</tr>
<tr>
<td>Feed</td>
<td>Mash (unlikely to be heat treated, often organic acids added)</td>
<td>Heat treated pellets</td>
</tr>
<tr>
<td>Biosecurity</td>
<td>standards of biosecurity at layer farms and broiler farms can vary significantly across the industry.</td>
<td></td>
</tr>
</tbody>
</table>

5.1.2 *Salmonella* in and on eggs in New Zealand

A survey carried out by ESR in 1994 examined eggs sampled from Otago, Southland and Canterbury. No *Salmonella* were detected on the shells of 341 samples of 6 eggs (2046 eggs in total) or in the contents of 339 samples of 6 eggs (2037 eggs in total). The same survey noted that overall, 64 of 4090 (1.5%) eggs examined were contaminated with visible faecal material. Most of these (62%) were collected directly from the producer rather than retail sources (Johnson, 1995). There was no distinction made in this survey between free range, barn produced and caged bird eggs.

A case control study was conducted in Auckland in 2001 to investigate a potential outbreak of *S. Typhimurium* DT160 (Thornley *et al.*, 2002). An additional part of the investigation was analysis of retail samples of eggs for *Salmonella* contamination, as raw egg consumption had been suggested as a possible source of the outbreak. Thirty seven lots of eggs,
comprising 93 individual samples were tested for the internal and external presence of *Salmonella*. Six different brands of eggs were tested. Four were free range produced eggs and two were caged hen produced. Unfortunately cracked eggs were not included in the study. Of the samples, each comprising a minimum of 6 eggs, none were internally contaminated. The two brands of eggs from caged birds yielded *Salmonella* on their exterior. Eight samples yielded *S*. Thompson, one *S*. Infantis and 11 *Salmonella* species 6,7:k: -. The latter isolates were probably unconfirmed *S*. Thompson. In total, 13 samples (14.0%) yielded *Salmonella*, as more than one isolate was tested from each positive sample. Several samples were tested for some of the 37 batches and, in terms of lots, 7 of 37 (18.9%) contained *Salmonella* as a surface contaminant. *S*. Typhimurium DT160 was not found on the eggs (Maurice Wilson, ESR Mount Albert Science Centre, pers. comm.).

5.1.3 Conclusion

The relatively low prevalence of *Salmonella* contamination of broiler chickens in New Zealand probably may not reflect the *Salmonella* status of the egg laying hen population, on which no information is available.

The information regarding the absence of internal contamination in eggs is consistent in both existing surveys. However, the more recent survey in Auckland indicates a relatively high prevalence of external contamination (14%), by comparison with international data (see Section 5.4.1). The serotypes of *Salmonella* found on the surface of eggs in this survey are not the most common serotypes isolated from human cases of salmonellosis in New Zealand, but do occur (see Section 6.1.4).

5.2 Food Consumption: Eggs

The GEMS/Food regional diets give a range of egg consumptions from 3.7g/person/day (African diet) to 37.6 g/person/day (European diet) (see: [http://www.who.int/foodsafety/publications/chem/regional_diets/en/](http://www.who.int/foodsafety/publications/chem/regional_diets/en/)).

New Zealand is generally considered to align most closely to the European diet.

New Zealand Food Balance Sheet for 2001 gives a per capita consumption of eggs of 9.8kg/person/year (26.8g/person/day) (see: [http://apps.fao.org/](http://apps.fao.org/)). This figure is the same as that derived from 2002 egg production figures, by assuming that all eggs sold in New Zealand are consumed (55,964,430 dozen at a weight of 58.5 g/egg) (see: [http://www.maf.govt.nz/statistics/primaryindustries/livestock/poultry/eggssold.xls](http://www.maf.govt.nz/statistics/primaryindustries/livestock/poultry/eggssold.xls)).

A FSANZ assessment (ANZFA, 2001) of the data from the 1997 National Nutrition Survey (Russell et al., 1999) concluded that 80.3% of respondents reported consuming eggs in the previous 24 hour period. The FSANZ analysis estimated the mean daily consumption of eggs for all respondents as 23.2 g/person/day. The simulated diets used for the 1997/98 New Zealand Total Diet used lower estimates of egg consumption (21.8 g/person/day for adult males and 13.9 g/person/day for adult females) (Brinsdon et al., 1999).

Australian statistics indicate much lower levels of egg consumption in that country, with 16.7% of the population 19 years and over consuming eggs or egg-based dishes in any 24...
hour period. The average consumption of eggs was 13.7 g/person/day (Australian Bureau of Statistics, 1999).

The UK National Food Survey (http://statistics.defra.gov.uk/esg/publications/nfs/default.asp) for 2000 gives an estimate of average household consumption of 1.75 eggs/person/week or 14.6 g/person/day at an egg weight of 58.5 g.

The situation in the USA is similar to New Zealand with more than 80% of the population aged one year or over consuming eggs each day. Average intake of eggs for adult over 19 years is in the range 0.27-0.29 g/kg body weight/day or 19-20 g/day for a 70 kg adult (EPA, 2003). Egg consumption in the US has been declining steadily since 1970 (Putnam and Allshouse, 1999), this appears to be partially, due to publicity of the cholesterol content of eggs (Yen et al., 1996).

5.3 Qualitative Estimate of Exposure

5.3.1 Number of servings and serving sizes

From the National Nutrition Survey, 3722 individual dietary records were deemed to represent consumption of a serving of poultry eggs. Using a total survey population of 4636 and a total New Zealand population 4,000,000:

Annual number of servings (total population)  = 3722 x 4,000,000/4636 x 365  
= 1.17 x 10^9 servings

This represents a high number of servings, as would be expected from a commonly consumed food such as eggs.

5.3.2 Frequency of contamination

Data for New Zealand from two surveys (1994 South Island survey-2056 eggs & 2001 Auckland survey-558 eggs) indicate that contents of shell eggs are rarely if ever contaminated by salmonellae. The data for the exterior of shells from the two existing surveys are contradictory, although the most recent survey indicates a significant level of contamination (14%).

5.3.3 Predicted contamination level at retail

Given that eggs are not refrigerated during retail display in New Zealand, on any occasion where the yolk of an egg became contaminated by salmonellae, they would be likely to grow to very high numbers rapidly. However, there is currently no evidence that eggs are contaminated internally in New Zealand.
5.3.4 Growth rate during storage and most likely storage time

The FAO/WHO study (2002) has examined the effect of shelf life and temperature in storage and its effect on *Salmonella* growth. The predictive model can be found at http://www.fao.org/DOCREP/005/Y4392E/y4392e0j.htm#bm19.3.4.

Bacteria contaminating the outside of shells are unlikely to grow as, although nutrients may be present, moisture levels would be too low.

A survey of Auckland consumer knowledge of food safety issues (Bloomfield and Neal, 1997) found that 75.7% of respondents believed that fresh eggs should be stored in the refrigerator, while 20.7% thought the cupboard shelf was suitable, and 3.6% were unsure. This finding was similar to the results from earlier studies that addressed the same issue. Refrigerated storage was identified as appropriate by 71% of respondents in a Canterbury survey (Hodges, 1993) and 56% of respondents in a Wellington postal survey (Kerslake, 1995). The authors of the Auckland study considered that refrigerated storage should be recommended, and this was one area of food safety knowledge that could be improved.

Current packaging instructions on packages of retail eggs in New Zealand are for storage at 7°-15°C, as recommended by the Egg Producers Federation of New Zealand Code of Practice.

5.3.5 Heat treatment

A telephone survey of 1260 people in the USA determined that 53% of respondents ate raw eggs (Klontz *et al.*, 1995). Examples of raw egg-containing foods were; cookie batter, home made ice cream, home made egg nog, Caesar salad, frosting, homemade shakes, home made hollandaise sauce and home made mayonnaise.

An analysis of four surveys on egg consumption in the USA has been published (Lin *et al.*, 1997). In one of these surveys, 27% of egg-containing dishes were described as undercooked, with each person consuming undercooked eggs 20 times a year. The meals involved were eggs fried over easy and sunny side up 49%, scrambled eggs 29%, poached eggs 13%, soft-boiled eggs 7% and hard boiled eggs 2%.

Heat treatment of eggs and egg-containing foods is therefore highly variable, but a significant proportion of eggs are likely to be eaten raw or undercooked (in the context of eggs this would mean cooked to a point where the yolk is still liquid).

5.3.6 Exposure summary

Eggs are a widely consumed food, and there will be significant exposure to raw or undercooked eggs i.e. eggs cooked in a manner unlikely to eliminate *Salmonella* in the egg contents. The probability of an egg being internally contaminated with *Salmonella* is very low, based on existing information.

There appears to be a higher potential for egg surfaces to be contaminated with *Salmonella*. This offers the potential for contamination of foods through cross contamination during...
handling. Growth of *Salmonella* may occur if food preparation is followed by unsuitable storage.

### 5.4 Overseas Context

The importance of egg contamination by *S. Enteritidis* overseas means that a lot of the more recent data concern that serotype only, and are not particularly relevant to New Zealand. Therefore some of the references used below are quite old, but have been included as relevant to New Zealand, particularly where the report provides data on serotypes other than *S. Enteritidis*.

#### 5.4.1 *Salmonella* in whole and liquid eggs

Information on the prevalence and numbers of salmonellae in eggs and egg products is presented in the tables below. Some caution needs to be used when considering the data in terms of internal and external contamination of whole eggs. Data have been presented to demonstrate that sterilisation of the shell prior to examination of the contents may be ineffective (Himathongkham *et al.*, 1999). Cross contamination from shell to contents may therefore occur in some cases where the shell was intended to be sterilised.
Table 1: Prevalence of *Salmonella* on whole egg shell surfaces

<table>
<thead>
<tr>
<th>Country</th>
<th>Samples tested</th>
<th>Number (%) positive</th>
<th>Serotype information (% of isolates)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argentina</td>
<td>122 shells</td>
<td>0</td>
<td>NA</td>
<td>Favier et al. 2001</td>
</tr>
<tr>
<td>Australia</td>
<td>336 cracked eggs 511 soiled eggs</td>
<td>0 2 (0.3)</td>
<td><em>S</em>. Anatum (50%) <em>S</em>. Singapore (50%)</td>
<td></td>
</tr>
<tr>
<td>Canada</td>
<td>94 composites of “grade crack” eggs</td>
<td>2 (2.1)</td>
<td>NS</td>
<td>D'Aoust et al. 1980</td>
</tr>
<tr>
<td>Spain</td>
<td>372 whole eggs associated with outbreaks 998 whole eggs not associated with outbreaks</td>
<td>5 (1.3), 3 from shells, one from the contents and one from a cracked egg. 6 (0.6) 1 from contents, 5 from shells and 1 were both tested simultaneously</td>
<td><em>S</em>. Enteritidis (60%), <em>S</em>. Typhimurium PT 96 (40%) <em>S</em>. Enteritidis (100%)</td>
<td>Perales and Audicana 1989</td>
</tr>
<tr>
<td>USA (Hawaii)</td>
<td>106 dozen whole eggs</td>
<td>10 (9.4). Seven of these were from one processor found to have faulty washing equipment.</td>
<td>In order of frequency of occurrence; <em>S</em>. Braenderup (20%), <em>S</em>. Oranienburg (40%), <em>S</em>. Mbandaka (10%), <em>S</em>. Cerro (10%), <em>S</em>. Ohio (10%), <em>S</em>. Havana (10%), <em>S</em>. Montevideo (10%), <em>S</em>. Livingstone (10%). (More than one serotype was isolated from some samples)</td>
<td>Ching-Lee et al. 1991</td>
</tr>
<tr>
<td>USA</td>
<td>222 Clean egg shells 232 Dirty egg shells 123 Washed dirty egg shells 85 duck egg shells 16 guinea hen egg shells 18 turkey egg shells</td>
<td>3 (1.3) 11 (4.7) 6 (4.8) 4 (4.7) 1 (6.3) 0</td>
<td>NS NS NS NS</td>
<td>Solowey et al. 1946</td>
</tr>
<tr>
<td>USA</td>
<td>90 eggshells prior to processing 90 eggshells after processing and packing</td>
<td>7 (7.8) 1 (1.1)</td>
<td><em>S</em>. Heidelberg (77.3%) and <em>S</em>. Montevideo (22.7%) All <em>S</em>. Heidelberg</td>
<td>Jones et al. 1995</td>
</tr>
</tbody>
</table>
### Table 2: Prevalence of *Salmonella* in whole and liquid eggs

<table>
<thead>
<tr>
<th>Country</th>
<th>Samples tested</th>
<th>Number (%) positive</th>
<th>Serotype information (% <em>Salmonella</em> isolates)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>1400 unwashed farm eggs</td>
<td>3 (0.21)</td>
<td><em>S.</em> Typhimurium (100%)</td>
<td>Baker <em>et al.</em> 1980</td>
</tr>
<tr>
<td>USA</td>
<td>100 dozen</td>
<td>1 dozen (1)</td>
<td><em>S.</em> Heidelberg (100%)</td>
<td><a href="http://www.sma.org/smj/96sept8.htm">www.sma.org/smj/96sept8.htm</a></td>
</tr>
</tbody>
</table>

**Note:**
- NT = Non typable
- ND = No Data
- NS = Not Stated
- NA = Not Applicable

---

**Table 2:** Prevalence of *Salmonella* in whole and liquid eggs

<table>
<thead>
<tr>
<th>Country</th>
<th>Samples tested</th>
<th>Number (%) positive</th>
<th>Serotype information (% <em>Salmonella</em> isolates)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albania</td>
<td>79 lots of 10</td>
<td>1 (1.3) isolated from the shell</td>
<td><em>Salmonella</em> group C</td>
<td>Telo <em>et al.</em> 1999</td>
</tr>
<tr>
<td>Australia</td>
<td>Bulk unpasteurised liquid egg</td>
<td>NS (21.8)</td>
<td><em>S.</em> Typhimurium (69.3%), <em>S.</em> Anatum (7.8%), <em>S.</em> Singapore (4.6%), <em>S.</em> Hessarek var 27 (3.7%), <em>S.</em> Oranienburg (3.7%), <em>S.</em> Chester (2.8%), <em>S.</em> Adelaide (1.8%), <em>S.</em> Havana (1.8%), <em>S.</em> Bovis-morbificans (0.9%), <em>S.</em> Bredeny (0.9%), <em>S.</em> Give (0.9%), <em>S.</em> Kottbus (0.9%), <em>S.</em> Pullorum (0.9%), <em>S.</em> Senftenberg (0.9%), <em>S.</em> Taxony (0.9%), <em>S.</em> Typhimurium (56.9%), <em>S.</em> Singapore (8.5%), <em>S.</em> Saint-paul (5.6%), <em>S.</em> Anatum (5.2%), <em>S.</em> Oranienburg (4.0%), <em>S.</em> Adelaide (2.4%), <em>S.</em> Derby (2.0%), <em>S.</em> Tennessee (2.0%), <em>S.</em> Bredeny (1.2%), <em>S.</em> Havana (1.2%), <em>S.</em> Give (0.9%), <em>S.</em> Kottbus (0.9%), <em>S.</em> Pullorum (0.9%), <em>S.</em> Senftenberg (0.9%), <em>S.</em> Taxony (0.9%)</td>
<td>Peel 1976</td>
</tr>
<tr>
<td></td>
<td>Export 524</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Local 622</td>
<td>NS (24.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Country</td>
<td>Samples tested</td>
<td>Number (%) positive</td>
<td>Serotype information (% Salmonella isolates)</td>
<td>Reference</td>
</tr>
<tr>
<td>---------</td>
<td>----------------</td>
<td>---------------------</td>
<td>-----------------------------------------------</td>
<td>-----------</td>
</tr>
<tr>
<td></td>
<td>Bulk pasteurised liquid egg</td>
<td></td>
<td>S. Ondestepoort (1.2%), S. Senftenberg (1.2%), S. Birkenhead (0.8%), S. Give (0.8%), S. Hessarek var 27 (0.8%), S. Kottbus (0.8%), S. Newbrunswick (0.8%), S. Newington (0.8%), S. Newport (0.8%), S. Potsdam (0.8%), S. Rubislaw (0.8%), Salmonella untypable (0.8%)</td>
<td>D'Aoust et al. 1980</td>
</tr>
<tr>
<td></td>
<td>Export 5,088</td>
<td>NS (0.04)</td>
<td>S. Typhimurium (100%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Local 560</td>
<td>NS (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Whole egg contents 847</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canada</td>
<td>(whole eggs-half dozens)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Layer hatching: Surplus 126 Early 126 Culled* 126</td>
<td>2 (1.6) 1 (0.8) 9 (7.1)</td>
<td>S. Typhimurium PT 66 and PT 3 S. Heidelberg PT8 S. Typhimurium PT 66 and PT 193, S. Heidelberg PT8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Broiler hatching: Surplus 42 Early 42 Culled 42</td>
<td>0 0 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Layer table: Regular 168 Early 84</td>
<td>0 1 (1.2)</td>
<td>S. Agona (100%)</td>
<td></td>
</tr>
<tr>
<td>Canada</td>
<td>Whole “Grade Crack” eggs (cracked but not leaking). Five egg</td>
<td></td>
<td>S. Infantis (41.5%), S. Montevideo (15.4%), S. Schwarzengrund (10.0%), S. Bareilly (10.0%), S. Oranienburg (10.0%), S.</td>
<td></td>
</tr>
<tr>
<td>Country</td>
<td>Samples tested</td>
<td>Number (%) positive</td>
<td>Serotype information (% <em>Salmonella</em> isolates)</td>
<td>Reference</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------------</td>
<td>---------------------</td>
<td>----------------------------------------------------------------------------------------------------------------</td>
<td>----------------------------</td>
</tr>
<tr>
<td>England</td>
<td>Bulk liquid egg from 11 farms.</td>
<td>39 (13.0)</td>
<td>Heidelberg (5.4%), S. Cerro (5.4%), S. Typhimurium (5.4%), S. Alachua (5.4%), S. London (5.4%)</td>
<td>Chapman <em>et al.</em> 1988</td>
</tr>
<tr>
<td>England</td>
<td>(whole eggs-half dozens)</td>
<td>5 (55.5)</td>
<td>S. Typhimurium PT 141. S. Typhimurium PT 104 and S. Virchov</td>
<td>de Louvois 1993</td>
</tr>
<tr>
<td>England</td>
<td>(whole eggs-half dozens)</td>
<td>65 (0.9)</td>
<td>S. Enteritidis PT4 (52.2%), other S. Enteritidis (24.4%), S. Infantis (1.1%), S. Livingstone (12.2%), S. Typhimurium (10.0%), Other (4.4%) S. Enteritidis PT4 (11.3%), other S. Enteritidis (1.9%), S. Infantis (39.4%), S. Livingstone (22.5%), S. Braenderup (5.6%), S. Typhimurium (6.3%), Other (11.9%)</td>
<td>Suzuki <em>et al.</em> 1981</td>
</tr>
<tr>
<td>Japan:</td>
<td>(Unpasteurised, frozen liquid)</td>
<td>7 (11.7)</td>
<td>S. Cerro (72.8%), S. Braenderup (14.8%), S. Thompson (6.1%), S. Infantis (3.9%), S. Mbandaka (1.7%), S. Senftenberg (0.7%)</td>
<td>Wilson <em>et al.</em> 1998</td>
</tr>
<tr>
<td>Plant A</td>
<td>60</td>
<td>7 (11.7)</td>
<td>S. Enteritidis PT4 (22.2%), S. Enteritidis PT1 (11.1%) (the internal contaminant), S. Infantis (22.2%), S. Mbandaka (11.1%), S. Monetvideo (11.1%), S. Typhimurium DT104 (11.1%), S. Kentucky (11.1%)</td>
<td></td>
</tr>
<tr>
<td>Plant B</td>
<td>44</td>
<td>37 (84.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plant C</td>
<td>19</td>
<td>3 (15.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plant D</td>
<td>30</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northern Ireland</td>
<td>2,090 half dozen</td>
<td>9 (0.4) total comprising 1 (0.05) internal 8 (0.38)</td>
<td>S. Enteritidis PT4 (22.2%), S. Enteritidis PT1 (11.1%) (the internal contaminant), S. Infantis (22.2%), S. Mbandaka (11.1%), S. Monetvideo (11.1%), S. Typhimurium DT104 (11.1%), S. Kentucky (11.1%)</td>
<td>Advisory committee on the microbiological safety of food 2001</td>
</tr>
<tr>
<td>UK, imported eggs</td>
<td>1,433 half dozen whole eggs</td>
<td>29 (2.0)</td>
<td>S. Enteritidis PT21 (34.4%), S. Enteritidis PT6 (10.3%), S. Enteritidis PT11 (10.3%), S. Enteritidis PT4 (6.9%), S. Taksony (17.2%), S. Livingstone (6.9%), S. Braenderup (6.9%),</td>
<td></td>
</tr>
</tbody>
</table>

*Risk Profile: Salmonella (Non Typhoidal) in and on eggs*
<table>
<thead>
<tr>
<th>Country</th>
<th>Samples tested</th>
<th>Number (%) positive</th>
<th>Serotype information (% <em>Salmonella</em> isolates)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>UK, domestic eggs (see also Section 5.4.2)</td>
<td>13,970 half dozen whole eggs</td>
<td>138 (1.0%)</td>
<td>S. Enteritidis PT4 (59.4%), S. Enteritidis PT7 (8.0%), S. Enteritidis PT8 (4.3%), S. Enteritidis PT6 (1.4%), other S. Enteritidis PT (13.0%), S. Typhimurium DT 104 (3.6%) other S. Typhimurium (0.7%), S. Mbandaka (2.9%), S. Livingstone (3.6%), S. Kimuenza (1.4%), S. Indiana (1.4%), S. Virchow (1.4%), S. Infantis (3.4%) S. Braenderup (0.7%), other (1.4%)</td>
<td>Advisory committee on the microbiological safety of food 2001</td>
</tr>
<tr>
<td>UK, from “high street retail outlets”</td>
<td>7,730 half dozen samples</td>
<td>17 (0.2%) of which 9 (0.1%) were from the egg surface and 8 (0.1%) from the contents.</td>
<td>S. Enteritidis (94.1%) of which 76.5% were PT4. N.B. 4 samples exceeded $10^4$ <em>Salmonella</em>/ml egg contents after 5 weeks storage at 21°C. Three were S. Enteritidis PT4, one S. Enteritidis PT1A</td>
<td>de Louvois 1994</td>
</tr>
<tr>
<td>USA:</td>
<td>(Unpasteurised liquid)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>USDA data winter/spring</td>
<td>40</td>
<td>4 (6.7)</td>
<td>NS</td>
<td>Garibaldi <em>et al.</em> 1969</td>
</tr>
<tr>
<td>USDA data summer/autumn</td>
<td>100</td>
<td>54 (54.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patterson and Brant data, summer</td>
<td>29</td>
<td>15 (51.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bergquist and Klusmeyer data</td>
<td>80</td>
<td>19 (23.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Winter/spring</td>
<td>18</td>
<td>10 (55.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Summer/autumn</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>USA</td>
<td>37 shell membranes, clean shells</td>
<td>0</td>
<td>NS (can’t be separated from information surface contaminants).</td>
<td>Solowey <em>et al.</em> 1946</td>
</tr>
<tr>
<td></td>
<td>33 shell membranes</td>
<td>3 (9.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Risk Profile: Salmonella (Non Typhoidal) in and on eggs*
<table>
<thead>
<tr>
<th>Country</th>
<th>Samples tested</th>
<th>Number (%) positive</th>
<th>Serotype information (% <em>Salmonella</em> isolates)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>dirty shells</td>
<td>39 shell membranes</td>
<td>6 (15.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>washed dirty shells</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>58 whole liquid from eggs with clean shells</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>55 whole liquid from eggs with dirty shells</td>
<td>2 (3.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>30 whole liquid from eggs with washed dirty shells</td>
<td>1 (3.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 yolk from clean eggs</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 yolk from dirty eggs</td>
<td></td>
<td>1 (12.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 yolk from washed dirty eggs</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 white from clean eggs</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 white from dirty eggs</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 white from washed dirty eggs</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 duck egg shell membrane</td>
<td></td>
<td>1 (5.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 guinea-hen egg shell membrane</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 turkey egg shell membranes</td>
<td></td>
<td>1 (25.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 goose egg shell membranes</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22 whole duck egg contents</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 whole guinea-hen egg contents</td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Risk Profile: *Salmonella* (Non Typhoidal) in and on eggs

May 2004
Country | Samples tested | Number (%) positive | Serotype information (% *Salmonella* isolates) | Reference
--- | --- | --- | --- | ---
USA | 7 whole turkey egg contents 8 whole goose eggs contents | 0 | S. Montevideo (38.5%), S. Anatum (61.5%) S. Pullorum (100%) | Cantor and McFarlane 1948
USA (Arkansas) | 2132 shell scrapings 2584 egg contents | 13 (0.6) 30 (1.2) | | www.sma.org/smj/96sept8.htm
USA (Hawaii) | 100 dozen whole egg contents | 0 | NA | Ching-Lee et al. 1991
USA | 106 dozen whole eggs | 0 | NA | Jones et al. 1995
USA | 180 | 0 | NA | Jones et al. 1995

* Culled refers to eggs removed for defects such as cracks and intended for processing to pasteurised egg

Table 3: Quantitative data for *Salmonella* in egg products

<table>
<thead>
<tr>
<th>Samples tested</th>
<th>Counts</th>
<th>Reference</th>
</tr>
</thead>
</table>
| Frozen unpasteurised whole eggs | Plant A: 36-91 MPN/g 2 (3.3%), 130-230 1 (1.7%), 330-490 1 (1.7%), 1,100-2,400 3 (5%)  
Plant B: 30 MPN/g 4 (9.1%), 36-91 6 (13.6%), 130-230 5 (11.4%), 330-490 6 (13.6%), 530-790 5 (11.4%), 1,100-2,400 2 (4.5%), 14,000-54,000 9 (20.5%)  
Plant C: 30 MPN/g 1 (5.3%), 130-230 1 (5.3%), 1,200-2,400 1 (5.3%)  
Plant D: None positive | Suzuki et al. 1981 |
| Unpasteurised liquid egg samples | For 100 positive samples <1 MPN/g 86 (86.0%), 1.4 to 2.9 10 (10.0%), 5.3 1(1.0%), 24 1 (1.0%), 110 (1.0%) | |
No difference in the rate of contamination was detected between eggs produced in English battery farms and those that were free range (de Louvois, 1993). This was still the case in the latest 2003 UK retail egg survey, see section 5.4.2.2. Faecal contamination was detected on 12% of the *Salmonella* positive eggs and on 4% of the uncontaminated eggs. A significant association between contamination by *Salmonella* and the presence of faecal matter (as well as with cracking) has been shown for Canadian eggs (Poppe *et al*., 1998), but not for contaminated eggs in Northern Ireland (Wilson *et al*., 1998).

Some evidence for a seasonal variation in *Salmonella* contamination of eggs is apparent. For example, in Canada 31% of samples were positive in August, and only 6% in December (D'Aoust *et al*., 1980).

5.4.2 Recent information from the UK:

In March 2004 the results of two surveys of eggs for *Salmonella* in the UK were released.

5.4.2.1 Catering premises

In 2003, a joint study was carried out by (UK) Local Authorities Coordinators of Regulatory Services (LACORS) and Health Protection Agency (HPA) for England & Wales. The study examined eggs from catering premises throughout the UK in relation to use and contamination with *Salmonella* spp.. During 2002 and 2003, foods made with eggs from catering premises predominated in reports of national and localised food borne outbreaks of *Salmonella* Enteritidis. Eggs imported into the UK from Spain were implicated in *S*. Enteritidis outbreaks between 2002 and 2004. Current advice from the UK Food Standards Agency is that eggs from Spain should be heat-treated.

A total of 34,116 (5,686 pooled samples of six) eggs were collected from 2,104 catering premises in April and May 2003. The majority (88%) of eggs sampled were UK produced. *Salmonella* spp. were isolated from 17 (0.3%) pools of eggs. Of these, 15 were *S*. Enteritidis. One each of *S*. Livingstone and *S*. Typhimurium Definitive Type 7 were also isolated (see: [http://www.lacors.com](http://www.lacors.com)).

The report noted that while *Salmonella* spp. were not found in the 12% of non-UK eggs in this study, sporadic introduction of highly contaminated eggs from abroad may occur.

5.4.2.2 Retail Premises

A Department of Health funded retail survey of UK produced eggs sampled in England was undertaken between May 1995 and April 1996. *Salmonella* spp. were detected in 0.99% of the 13970 samples of 6 eggs (an estimated contamination rate per individual egg of 1 in every 100 boxes of 6 eggs). There was no significant change in *Salmonella* contamination of UK produced eggs since a previous survey in 1991.

A survey of retail eggs in Northern Ireland between April 1996 and October 1997 found *Salmonella* in 0.43% (9 samples) of 2090 samples of 6 eggs (including one sample (0.05%) which was internally contaminated by *S*. Enteritidis PT1(Wilson *et al* 1998).

The most recent study was carried out by the UK Food Standards Agency of A-grade shell eggs on retail sale between March and July 2003 (see: [http://www.foodstandards.gov.uk/news/newsarchive/2004/mar/salmonellaeggnews](http://www.foodstandards.gov.uk/news/newsarchive/2004/mar/salmonellaeggnews)). A total
of 4753 samples (mostly boxes) of six eggs were purchased from a cross-section of retail outlets across the UK. The shell and contents were tested for Salmonella contamination. Overall 9 samples (0.34%) were Salmonella contaminated (seven from England, two from Wales, estimated contamination rate of approximately 1 in every 290 boxes of 6 eggs). All Salmonella positive samples were from egg shells. The report concludes that the small number of positive samples does not point to systemic contamination from infected flocks but rather random contamination from the production environment. There was no statistically significant difference between the prevalence of Salmonella contamination in samples;

- Purchased in England, Scotland, Wales or Northern Ireland;
- From different egg production types (e.g. free range, barn, caged birds etc);
- Derived from non-Lion code eggs and Lion code eggs (84% of the samples had the Lion code mark on the egg boxes); and
- From eggs stored chilled or at ambient temperature.

However, there was a significantly higher prevalence of Salmonella from eggs purchased from medium sized retailers than in large retail outlets.

On an England only basis, the results compared with the 1995/96 retail survey show a 3-fold reduction. The prevalence of S. Enteritidis PT4 has also declined sharply; 0.11% in the 2003 retail survey compared to 0.58% in the 1995/96 retail survey. In addition, it was significant that all the positive results in 2003 were for shell contamination, whereas in 1995/1996 the positive results were a mixture of internal and external contamination.

The reduction in Salmonella contamination of eggs was attributed to measures introduced by the UK egg industry to control Salmonella. The most important of these measures is probably the vaccination of laying hens against S. Enteritidis under the British Egg Industry Council Lion Code scheme. Approximately 80% of all laying hens in the UK are vaccinated.
6 RISK CHARACTERISATION

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

The number of notified cases and rates per 100,000 population of salmonellosis in New Zealand are shown in Table 4. The incidence data is also shown graphically (by year) in Figure 2, while the number of cases (by month) are shown in Figure 3.

Table 4: Incidence data for salmonellosis in New Zealand, 1985-2003

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of cases</th>
<th>Incidence (cases/100,000)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985</td>
<td>1234</td>
<td>38.9</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1986</td>
<td>1335</td>
<td>40.4</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1987</td>
<td>1140</td>
<td>34.5</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1988</td>
<td>1128</td>
<td>34.1</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1989</td>
<td>1860</td>
<td>56.2</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1990</td>
<td>1619</td>
<td>50.0</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1991</td>
<td>1244</td>
<td>36.9</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1992</td>
<td>1239</td>
<td>36.7</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1993</td>
<td>1340</td>
<td>39.7</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1994</td>
<td>1522</td>
<td>45.1</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1995</td>
<td>1334</td>
<td>39.5</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1996</td>
<td>1140</td>
<td>31.5</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1997</td>
<td>1169</td>
<td>35.3</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1998</td>
<td>2069</td>
<td>57.2</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>1999</td>
<td>2079</td>
<td>57.5</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2000</td>
<td>1802</td>
<td>48.1</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2001</td>
<td>2417</td>
<td>64.7</td>
<td>Sneyd et al., 2002</td>
</tr>
<tr>
<td>2002</td>
<td>1870</td>
<td>50.0</td>
<td>Sneyd and Baker, 2003</td>
</tr>
<tr>
<td>2003</td>
<td>1404</td>
<td>37.6</td>
<td>ESR, 2003*</td>
</tr>
</tbody>
</table>

* Provisional data from ESR Monthly Surveillance Report from December 2003
Figure 2: Salmonellosis notification rates by year 1980 – 2002

Salmonellosis notifications by year, 1980 - 2002

Reproduced from Sneyd and Baker (2003)

Figure 3: Salmonellosis notifications by month January 1996 – December 2002

Salmonellosis notifications by month
January 1996 - December 2002

Reproduced from Sneyd and Baker (2003)
The frequency of salmonellosis is characterised by a late summer peak and a winter trough. Two changes to this cyclic pattern have occurred since 1998:

- A spring peak occurred in 1998 and each subsequent year, corresponding to the emergence of S. Brandenburg as an important cause of human salmonellosis in New Zealand.
- The winter trough has become less pronounced due to the increasing numbers of STM 160 cases since July 2000 (Anonymous, 2001b).

Based on results from a prospective GP and community based study of infectious intestinal disease in the UK, a ratio of unreported to reported cases of salmonellosis of 3.2:1 was used to generate an estimated total of 9218 cases per year in New Zealand using 1998 data (Lake et al., 2000).

### 6.1.2 Clinical consequences of *Salmonella* infection

Hospitalisation and fatality rates for notified cases of salmonellosis in New Zealand are given in Table 5. 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.

#### Table 5: Outcome data for salmonellosis in New Zealand

<table>
<thead>
<tr>
<th>Year</th>
<th>Hospitalised cases</th>
<th>Fatalities</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997</td>
<td>117/974 (12.0%)</td>
<td>2/1169 (0.17%)</td>
<td>ESR, 1998</td>
</tr>
<tr>
<td>1998</td>
<td>161/1816 (8.9%)</td>
<td>2/2069 (0.1%)</td>
<td>Perks et al., 1999</td>
</tr>
<tr>
<td>1999</td>
<td>192/1797 (10.7%)</td>
<td>1/2079 (0.05%)</td>
<td>Kieft et al., 2000</td>
</tr>
<tr>
<td>2000</td>
<td>215/1554 (13.8%)</td>
<td>7/1802 (0.4%)</td>
<td>Lopez et al., 2001</td>
</tr>
<tr>
<td>2001</td>
<td>279/1934 (14.4%)</td>
<td>2/2417 (0.1%)</td>
<td>Sneyd et al., 2002</td>
</tr>
<tr>
<td>2002</td>
<td>206/1473 (14.0%)</td>
<td>1/1870 (0.05%)</td>
<td>Sneyd and Baker, 2003</td>
</tr>
</tbody>
</table>

Evans et al. (1998) reported a case fatality rate of 0.3% in 1995 and 0.8% in 1996 in England and Wales.

Chronic sequelae of *Salmonella* infections include rheumatoid arthritis, which occurs in 0.2 to 2.4% of cases involved in outbreaks caused by S. Typhimurium (Lindsay, 1997). In New Zealand, the high number of cases of infection with another triggering organism, *Campylobacter*, means that very few cases of reactive arthritis recorded by the New Zealand Health Information Service are attributed to *Salmonella* infections (Lake et al., 2000).

### 6.1.3 Case control studies and risk factors

The first case-control study of salmonellosis cases in New Zealand concerned S. Typhimurium DT160 (Thornley et al., 2002; Thornley et al., 2003). This was prompted by a marked increase in the number of S. Typhimurium DT160 human isolates during 2001, such that this serotype is now the most common amongst human isolates of *Salmonella* in New Zealand. 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. An investigation of a common source outbreak in Auckland in February 2001 identified an association between S. Typhimurium
DT160 infection and consumption of potato salad with egg mayonnaise (Callaghan and Simmons, 2001). In addition to telephone interviews of cases (119) and controls (235), sampling and analyses were conducted of roof-collected rainwater supplies from the homes of cases, and egg brands consumed by cases (see Section 5.1.2). For the twelve cases of infection which had roof-collected water supplies, S. Typhimurium DT160 was found in samples of water from the homes of six cases. Other Salmonella serotypes were isolated from the surface of egg samples, but not S. Typhimurium DT160.

The strongest finding was that there was an association between infection with S. Typhimurium DT160 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. Consumption of takeaway food had a weakly positive association with infection (mOR = 1.7, CI: 1.04-2.8), but consumption of whole chicken was less common amongst cases than controls (mOR = 0.4, CI: 0.2-0.6). Contact with another individual with diarrhoea and vomiting was also associated with S. Typhimurium DT160 infection (mOR = 3.1, CI: 1.7-5.7). Population attributable ratios (PAR) were calculated and the largest PAR% was demonstrated for consumption of takeaway food (26.1%). However, no single type of takeaway outlet was significantly associated with illness. These results suggest that consumption of poultry is not a risk factor for transmission of S. Typhimurium DT160 in New Zealand at present.

In this study, egg consumption was examined in detail (a variety of egg dishes as well as derived foods such as mayonnaise and custard). None of the egg related risk factors had a statistically significant odds ratio.

Another case-control study was initiated 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). 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 3 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 that represents a risk to farmers and others who have direct occupational contact with infected sheep.

Consumption of eggs was one of the risk factors included in the survey of cases and controls. Consumption of eggs in general was protective for salmonellosis (OR 0.45, 95% CI 0.26-0.74). The odds ratios for more detailed analyses (cooked eggs, raw eggs, homemade food with raw eggs, homemade food with cooked eggs) were not significant.

6.1.4 Outbreaks

The number of reported outbreaks of salmonellosis in recent years in New Zealand is given in Table 6.

<table>
<thead>
<tr>
<th>Year</th>
<th>Outbreaks*</th>
<th>Cases**</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997</td>
<td>14/97 (14.4%)</td>
<td>152/1209 (12.6%)</td>
<td>ESR, 1998</td>
</tr>
<tr>
<td>1998</td>
<td>31/207 (15.0%)</td>
<td>223/1552 (14.4%)</td>
<td>Naing <em>et al.</em>, 1999</td>
</tr>
<tr>
<td>1999</td>
<td>43/352 (12.2%)</td>
<td>275/2302 (11.9%)</td>
<td>Perks <em>et al.</em>, 2000</td>
</tr>
<tr>
<td>2000</td>
<td>30/273 (11.0%)</td>
<td>312/1903 (16.4%)</td>
<td>Lopez <em>et al.</em>, 2001</td>
</tr>
<tr>
<td>2001</td>
<td>37/369 (10.0%)</td>
<td>214/2095 (10.2%)</td>
<td>Sneyd <em>et al.</em>, 2002</td>
</tr>
<tr>
<td>2002</td>
<td>36/337 (10.7%)</td>
<td>253/2890 (8.8%)</td>
<td>Boxall and Ortega, 2003</td>
</tr>
</tbody>
</table>

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

The low numbers of outbreaks in 1997 is due to the fact that the electronic reporting system was introduced in mid-1997, rather than indicating that a lower number of outbreaks occurred during that year.

The EpiSurv outbreak module was searched for outbreaks of salmonellosis in which egg or egg dishes were a suspected vehicle over the past six years. The results are given in Table 7. One of these thirteen outbreaks involved chocolate mousse as the suspected vehicle of infection although it was not categorically stated that the dish contained egg. In several of the other outbreaks, eggs or egg dishes were only one of several potential food vehicles.

Out of the thirteen outbreaks with egg or egg dishes as a suspected vehicle of infection, only one outbreak had laboratory confirmation in that *Salmonella* was also isolated from the suspect food, although there are caveats to this finding;

The outbreak (ref. No. AK2001175) involved two cases infected with *Salmonella Typhimurium* DT160. The same *Salmonella Typhimurium* phage type was isolated from the suspect vehicle of infection; a raw egg mayonnaise, some of which had been left over from the meal shared by the two cases. The cases did not have contact before the common meal. No Critical Control Point failure was identified in the preparation of the home made raw mayonnaise and raw egg was the only high risk ingredient identified in the mayonnaise.
The same brand of eggs as used in the mayonnaise was sampled (shell rinse and contents) three weeks later (a different batch to that used to prepare the food). *Salmonella* Typhimurium DT160 was not isolated from the 4 x half dozen egg samples. It was unclear whether the eggs originated from free range or caged hen production systems. The period between the meal and receiving the sample for analysis (12 days) raises the potential for the egg mayonnaise itself to have been contaminated by the cases who contracted the infection from a different source.

### Table 7: Reported data for salmonellosis outbreaks where eggs are a suspected vehicle in New Zealand

<table>
<thead>
<tr>
<th>Year</th>
<th>Outbreaks</th>
<th>Cases*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1998</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>1999</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>2000</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2001</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>2002</td>
<td>5</td>
<td>22</td>
</tr>
<tr>
<td>2003</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* *Includes both suspected and confirmed cases*

#### 6.1.5 Serotypes of *Salmonella* isolates from human cases in New Zealand

The following overview of human isolates of *Salmonella* in New Zealand is for the year 2003 and was published in Table 17 of the ESR Annual Report (2003) “Notifiable and Other Diseases in New Zealand”. Selected *Salmonella* serotypes and subtypes were laboratory confirmed in a total of 1562 cases of salmonellosis for 2003. (Note the total excludes *S. Paratyphi* and *S. Typhi*). There has been a decrease of approximately 1000 cases on the previous year 2002 (2559). In decreasing order of prevalence, the serotypes are;

- *S. Typhimurium* (STM) 953 cases (61%),
- *S. Enteritidis* 137 cases (9%),
- *S. Infantis* 89 (6%),
- *S. Brandenburg* 55 (4%).

Of the *S. Typhimurium* isolates;

- DT 160 334 (21% of the total 1562 cases),
- DT 1 110 (7%),
- DT 156 95 (6%),
- DT 135 68 (4%),
- DT156 95 (6%),
- DT101 66(4%),
- Other or unknown 280 (18%).

Of the *S. Enteritidis* isolates;

- PT9a 65 (4%)
- PT4 22 (1%)
- Other or unknown 50 (3%).
Figure 4 shows the trend for the number of human *Salmonella* isolates for each of five common isolates during the period 1998-2003. ‘Other’ in this Figure refers to the sum of all typed isolates other than the five serotypes specified (‘other’ will therefore include *S*. Enteritidis and *S*. Infantis).

**Figure 4: Trend in occurrence of *Salmonella* serotypes amongst human isolates, 1998-2003**

(STM = *Salmonella* Typhimurium)

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6.1.6 *S*. Enteritidis Phage Type 4 & *S*. Typhimurium Definitive Type 104

Up to 1990, sporadic isolates of *S*. Enteritidis were isolated from various sources. However since 1990, this serotype has appeared more frequently amongst isolates from both humans and animals (Carman and Gardner, 1997).

*S*. Enteritidis has been recovered from cattle, sheep, goats, deer, dogs, a hedgehog, and once from poultry feed in New Zealand. It has also been isolated from environmental sources and imported products e.g. prawns and spices. It was recovered initially from two dogs in the Waikato between 1967 and 1972. Phage typing of New Zealand animal isolates started in 1986, most have been Phage type 9a. However one bovine isolate in each year 1988, 1991 and 1992 were Phage type 4. *S*. Enteritidis PT4 has not been isolated from poultry products or eggs to date in New Zealand. Phage type 4 is important as it is considerably more pathogenic for poultry than other strains (Carman and Gardner, 1997).

The majority of human salmonellosis infections with *S*. Enteritidis in this country appear to be associated with overseas travel, especially to South East Asia (*ESR LabLink* 1998, 5-1 and 1999, 6-1). Forty of the 71 isolates in 1999, and 31 of 50 isolates in 2000, were from
returning overseas travellers. Unfortunately the reference laboratory does not receive travel history for all isolates.

*S. Enteritidis* PT4 is currently the second most common *S. Enteritidis* type isolated from humans in New Zealand (41 of 172 (24%) in 2002) (*ESR Lablink* 2003; 10:4). Of these cases, 16 (39%) have been notified as overseas travelers.

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 multiresistant (Carolyn Nicol, Enteric Reference Laboratory, pers. comm.). During the period since 1997 this strain 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 multiresistant strains (Carolyn Nicol, Enteric Reference Laboratory, pers. comm.). In 2002 there was one isolate of *S. Typhimurium* DT104 from a human case, and four from non-human sources (three from canine sources and one from a feline source) (*ESR Lablink* 2003; 10:4).

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Table 8 shows the reported incidence of salmonellosis in several countries. New Zealand might expect to have a lower than average incidence because we do not have in the country, *Salmonella* types that can contaminate the insides of eggs, leading to disease when raw egg is used in foods such as ice cream and Caesar salad. However, the New Zealand incidence rate of 37.6 per 100,000 in 2003 appears to be comparable with other countries with a few exceptions.
Table 8: Reported incidence data for notified cases of salmonellosis overseas

<table>
<thead>
<tr>
<th>Country</th>
<th>Incidence (cases/100,000)</th>
<th>Year</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>31.6</td>
<td>1996</td>
<td>Lin et al., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>38.1</td>
<td>1997</td>
<td>Lin et al., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>40.7</td>
<td>1998</td>
<td>Lin et al., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>37.7</td>
<td>1999</td>
<td>Lin et al., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>32.1</td>
<td>2000</td>
<td>Lin et al., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>36.2</td>
<td>2001</td>
<td>Australia’s notifiable disease status 2002 Annual Report NNDSS</td>
</tr>
<tr>
<td>Australia</td>
<td>39.5</td>
<td>2002</td>
<td>Australia’s notifiable disease status 2002 Annual Report NNDSS</td>
</tr>
<tr>
<td>Denmark</td>
<td>61.5</td>
<td>1999</td>
<td><a href="http://130.226.165.6/annualreport1999/index.html">http://130.226.165.6/annualreport1999/index.html</a></td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>14.5</td>
<td>1996</td>
<td>Wallace et al., 2000</td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>13.7</td>
<td>1997</td>
<td>Wallace et al., 2000</td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>12.3</td>
<td>1998</td>
<td>Centers for Disease Control and Prevention, 2000a</td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>13.6</td>
<td>1999</td>
<td>Centers for Disease Control and Prevention, 2001</td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>12.0</td>
<td>2000</td>
<td>Centers for Disease Control and Prevention, 2001</td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>15.1</td>
<td>2001</td>
<td><a href="http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5115a3.htm">http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5115a3.htm</a></td>
</tr>
<tr>
<td>USA (selected sites)</td>
<td>16.10</td>
<td>2002</td>
<td><a href="http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5215a4.htm">http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5215a4.htm</a></td>
</tr>
</tbody>
</table>
Ireland has seen a decline in human salmonellosis rates since 1998. The Food Safety Authority of Ireland attribute this decline to a national *Salmonella* monitoring and control programme, the Bord Bia egg quality assurance scheme, educational campaigns targeting consumers and caterers, and increased official agency inspections.

The USA incidence data for 2001 and 2002 is not comparable to the earlier data in Table 8, as the earlier figures were based on surveillance of five states, while the FoodNet network has now been expanded to include data from eight states. The incidence of salmonellosis in the USA was reported to have decreased by 15% during the period 1996-2001. The predominant *Salmonella* serotypes in the USA in 2001 were *S. Typhimurium*, *S. Enteritidis*, *S. Newport*, *S. Heidelberg* and *S. Javiana* (CDC, 2002).

### 6.2.2 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 9. The lowest proportion of outbreaks caused by *Salmonella* (3.7%) was in Taiwan (Chiou *et al.*, 1991). Foodborne illness in Taiwan is dominated by outbreaks of infection with *Vibrio parahaemolyticus*, probably due to high consumption of seafood.

In contrast, in Italy, where 1379 (91%) of outbreaks of known aetiology were attributed to *Salmonella*, 726 (77%) of these outbreaks where a vehicle was confirmed or suspected involved foods containing eggs. Most egg containing foods involved were tiramisu and egg pasta (53% of outbreaks of salmonellosis where a food vehicle was confirmed or suspected). The high consumption in Italy of these local foods may help to explain the high proportion of salmonellosis outbreaks among outbreaks in general in this country. Of the isolates obtained in association with egg-containing food outbreaks, 91.9% were *S. Enteritidis*. The rest comprised *S. Typhimurium* (6.4%), *S. Panama* (1.0%), *S. Cholera-suis* (0.3%) and *S. Ohio* (0.3%).
Table 9: Proportion of foodborne disease attributed to infection with *Salmonella* overseas

<table>
<thead>
<tr>
<th>Country</th>
<th>Incidents</th>
<th>Outbreaks</th>
<th>Year(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada</td>
<td>31.6% of incidents of known cause (76.2% were of unknown cause)</td>
<td>47.5% of outbreaks of known cause (81.8% were of unknown cause)</td>
<td>1979</td>
<td>Todd 1987</td>
</tr>
<tr>
<td>Canada</td>
<td>27.4% of incidents of known cause (69.2% were of unknown cause)</td>
<td>40.8% of outbreaks of known cause (75.5% were of unknown cause)</td>
<td>1980</td>
<td>Todd 1987</td>
</tr>
<tr>
<td>Canada</td>
<td>25.7% of incidents of known cause (78.0% were of unknown cause)</td>
<td>NS</td>
<td>1975-1984 (mean)</td>
<td>Todd 1992</td>
</tr>
<tr>
<td>England and Wales</td>
<td>NS</td>
<td>38.5% of outbreaks of known cause (17.0% were of unknown cause)</td>
<td>1992-1994</td>
<td>Djuretic et al. 1996</td>
</tr>
<tr>
<td>England and Wales</td>
<td>NS</td>
<td>19.4% outbreaks of known cause, 16.0% of outbreak cases (25.9% were of unknown cause)</td>
<td>1995</td>
<td>Evans et al. 1998</td>
</tr>
<tr>
<td>England and Wales</td>
<td>NS</td>
<td>19.8% outbreaks of known cause, 14.3% of outbreak cases (22.2% were of unknown cause)</td>
<td>1996</td>
<td>Evans et al. 1998</td>
</tr>
<tr>
<td>Italy</td>
<td>NS</td>
<td>91% of outbreaks of known cause (11.0% were of unknown cause)</td>
<td>1991-1994</td>
<td>Scuderi et al. 1996</td>
</tr>
<tr>
<td>Japan</td>
<td>NS</td>
<td>17.2% of cases of known cause, 23.8% of outbreak cases (16.2% were of unknown cause)</td>
<td>1981-1995</td>
<td>Lee et al. 2001</td>
</tr>
<tr>
<td>Korea</td>
<td>NS</td>
<td>28.3% of outbreaks of known cause, 31.2% of outbreak cases (26.6% were of unknown cause)</td>
<td>1981-1995</td>
<td>Lee et al. 2001</td>
</tr>
<tr>
<td>Netherlands</td>
<td>14.2% of incidents with known cause (91.7% were of unknown cause)</td>
<td>15.5% of outbreaks of known cause (90.4% were of unknown cause)</td>
<td>1991-1994</td>
<td>Simone et al. 1997</td>
</tr>
<tr>
<td>Sweden</td>
<td>17.6% of incidents of known cause, 14.5% incident cases (66% incidents were of unknown cause)</td>
<td>17.8% of outbreaks of known cause, 14.5% of outbreak cases (61% of outbreaks were of unknown cause)</td>
<td>1992-1997</td>
<td>Lindqvist et al. 2000</td>
</tr>
<tr>
<td>Taiwan</td>
<td>NS</td>
<td>3.7% of outbreaks of known cause</td>
<td>1981-1989</td>
<td>Chiou et al. 1991</td>
</tr>
</tbody>
</table>

*Risk Profile: Salmonella (Non Typhoidal) in and on eggs*
<table>
<thead>
<tr>
<th>Country</th>
<th>Incidents</th>
<th>Outbreaks</th>
<th>Year(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>NS</td>
<td>28% of outbreaks of known cause, 45% of outbreak cases</td>
<td>1973-1987</td>
<td>Bean and Griffin 1990</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>35.1% of outbreaks of known cause, 36.8% of outbreak cases (59.4% were of unknown cause)</td>
<td>1988</td>
<td>Bean <em>et al.</em> 1996</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>41.2% of outbreaks of known cause, 56.2% of outbreak cases (56.2% were of unknown cause)</td>
<td>1989</td>
<td>Bean <em>et al.</em> 1996</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>57.4% of outbreaks of known cause, 63.2% of outbreak cases (55.5% were of unknown cause)</td>
<td>1990</td>
<td>Bean <em>et al.</em> 1996</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>57.0% of outbreaks of known cause, 62.3% of outbreak cases (59.5% were of unknown cause)</td>
<td>1991</td>
<td>Bean <em>et al.</em> 1996</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>54.8% of outbreaks of known cause, 56.3% of outbreak cases (64.1% were of unknown cause)</td>
<td>1992</td>
<td>Bean <em>et al.</em> 1996</td>
</tr>
<tr>
<td>USA</td>
<td>NS</td>
<td>13.3% of outbreaks of known cause, 37.9% of outbreak cases, and 46.4% of outbreak deaths (68.1% of outbreaks were of unknown cause)</td>
<td>1993-1997</td>
<td>Olsen <em>et al.</em> 2000</td>
</tr>
</tbody>
</table>

*ND = No Data supplied  *Both outbreaks involved pork as the vehicle
*NS = Not Stated*
Table 10 gives some examples of salmonellosis outbreaks associated with eggs that have been reported in the literature. Some of the outbreaks have involved hundreds of people. Given the number of outbreaks implicating eggs as the vehicle, the table below can only ever be a sample of those that have occurred. To illustrate this point, 123 outbreaks are listed for the USA between 1990 and 1998 on one website (www.cspinet.org/foodsafety/egg_safety_app.html).

**Table 10: Examples of outbreaks of salmonellosis from eggs overseas**

<table>
<thead>
<tr>
<th>Country</th>
<th>Number involved</th>
<th>Implicated Food and serotype</th>
<th>Year(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>53</td>
<td>Mock ice cream containing raw eggs. <em>S.</em> Typhimurium PT 135</td>
<td>2000</td>
<td>Sarna <em>et al</em>., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>16</td>
<td>Egg sandwiches or cross contamination from shell eggs. <em>S.</em> Typhimurium PT 135a</td>
<td>2002</td>
<td>McCall <em>et al</em>., 2003</td>
</tr>
<tr>
<td>Australia</td>
<td>11</td>
<td>Tiramisu containing raw eggs. <em>S.</em> Typhimurium PT 135a</td>
<td>2001</td>
<td>Hall, 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>18</td>
<td>Raw eggs used in rice pudding and potato topped pie. <em>S.</em> Typhimurium PT 135</td>
<td>2001</td>
<td>Tribe <em>et al</em>., 2002</td>
</tr>
<tr>
<td>Australia</td>
<td>17</td>
<td>Raw eggs used in mayonnaise and Caesar salad. <em>S.</em> Potsdam</td>
<td>2002</td>
<td>Unicomb <em>et al</em>., 2003</td>
</tr>
<tr>
<td>France</td>
<td>At least 12</td>
<td>Egg mayonnaise <em>Salmonella</em> Typhimurium</td>
<td>NS</td>
<td>Carraminana <em>et al</em>., 1997</td>
</tr>
<tr>
<td>Spain</td>
<td>&gt;18</td>
<td>Cracked eggs held at room temperature overnight prior to omelette preparation. <em>S.</em> Enteritidis PT1</td>
<td>1996</td>
<td>Furtado <em>et al</em>., 1997</td>
</tr>
<tr>
<td>UK</td>
<td>186</td>
<td>Egg and cress sandwiches, egg and mayonnaise sandwiches, mayonnaise from one chain store. <em>S.</em> Bareilly</td>
<td>2003</td>
<td>Cowden <em>et al</em>., 2003</td>
</tr>
</tbody>
</table>
| USA | 91 | Breakfast eggs, *S.* Heidelberg | 1985 | Centers for Disease Control and

**Risk Profile: Salmonella (Non Typhoidal)**

*in and on eggs* 45

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<table>
<thead>
<tr>
<th>Country</th>
<th>Number involved</th>
<th>Implicated Food and serotype</th>
<th>Year(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>28</td>
<td>Macaroni cheese containing egg, <em>S. Enteritidis</em> PT8</td>
<td>1991</td>
<td>Luby and Jones, 1993</td>
</tr>
<tr>
<td>USA</td>
<td>56</td>
<td>Hollandaise sauce, <em>S. Enteritidis</em> PT8</td>
<td>1994</td>
<td>Centers for Disease Control and Prevention, 1996</td>
</tr>
<tr>
<td>USA</td>
<td>13</td>
<td>Cheesecake containing lightly cooked egg white and yolk, <em>S. Enteritidis</em> PT4</td>
<td>1997</td>
<td>Centers for Disease Control and Prevention, 2000</td>
</tr>
<tr>
<td>USA</td>
<td>43</td>
<td>Lasagna containing eggs, <em>S. Enteritidis</em> PT 8</td>
<td>1997</td>
<td>Centers for Disease Control and Prevention, 2000</td>
</tr>
</tbody>
</table>

NS = Not Stated

In a description of *S. Enteritidis* infections in the USA it was noted that the proportion of *Salmonella* isolates of this serotype rose from 5% in 1976 to 26% in 1994. In the period of 1985-1994, 582 outbreaks of disease were attributed to this serotype, with 24,058 cases, 2290 hospitalisations and 70 deaths (Centers for Disease Control and Prevention, 1996). From 1994 to 1998 there were another 197 outbreaks of *S. Enteritidis* infections in the USA (Centers for Disease Control and Prevention, 2000). Many of these would have been caused by transovarian transmission to shell eggs which were eaten raw or undercooked.

In Ireland, eggs were identified as the vehicle of infection in 13% of outbreaks in 1998 and 1999 (Fitzgerald *et al.*, 2001). The serotypes identified were *S. Enteritidis* PT5a (1), *S. Enteritidis* PT4 (5), *S. Typhimurium* (1), *S. Enteritidis* (1) and unknown (1). Data from the Netherlands showed an increase from 3.9% to 34.4% between 1987 and 1991 of the proportion of human *Salmonella* isolates that were identified as *S. Enteritidis* (van de Giessen *et al.*, 1994).

### 6.2.3 Case control studies

Case control studies investigating the causes of infection with *Salmonella* in a number of countries are summarised in Table 11.
Table 11: Case control studies associating egg consumption with salmonellosis

<table>
<thead>
<tr>
<th>Country</th>
<th>Risk/protective factors</th>
<th>Odds Ratios/P values</th>
<th>Reference</th>
</tr>
</thead>
</table>
| England | Ingestion of products containing raw eggs  
S. Enteritidis  
S. Typhimurium | OR undefined but significant.  
No association. | Banatvala et al. 1999 |
| France | For sporadic S. Enteritidis infections in children ≤ 5:  
Consumption of raw eggs or undercooked egg-containing foods  
Storing eggs more than 2 weeks after purchase (a higher OR was noted in summer months) | 2.4 (CI 1.2-4.8)  
3.8 (CI 1.4-10.2) | Delarocque-astagneau et al. 1998 |
| USA* (Minnesota) | Consuming undercooked eggs or egg-containing foods in the 3 days prior to illness.  
S. Enteritidis  
S. Typhimurium | 5.2 (1.9-14.2)  
2.4 (1.1-5.5) | Hedberg et al. 1993 |

* This study also showed a correlation between the extent to which eggs were cooked and illness. The egg attributable risk for S. Enteritidis infections was 0.8/100,000 and for S. Typhimurium was 0.5/100,000.

A different type of case control study has been undertaken in South East Wales to examine the kitchen practices that lead to sporadic Salmonella food poisoning (Parry et al., 2002). Food handling practices in homes with a case of salmonellosis were compared to households without a case. With respect to egg related factors, it was found that consumption of raw eggs was a risk factor and handling free range eggs was a protective factor (believed to be a surrogate for different lifestyle). Since the study was conducted in Wales, most cases (76 of 93 cases where data were available) were caused by S. Enteritidis. Other serotypes involved were S. Typhimurium DT104 (10), PT 104b (1), 12 (1), 49 (1) and untypable (1). The remaining isolates were S. Infantis, S. Oyonnax and S. Virchow. No handling practice was identified as a risk/protective factor.

6.2.4 Risk assessments


The risk assessment had several objectives;

“1. To develop a resource document of all currently available information relevant to risk assessment of Salmonella in eggs and broiler chickens and also to identify the current gaps in the data that need to be filled in order to more completely address this issue.
2. To develop an example risk assessment framework and model for worldwide application.
3. To use this risk assessment work to consider the efficacy of some risk management interventions for addressing the problems associated with *Salmonella* in eggs and broiler chickens.”

The emphasis of the egg component of this risk assessment is on *S. Enteritidis*, and therefore it is not directly applicable to New Zealand.

A QRA specific for *S. Enteritidis* in pasteurised liquid egg used to make mayonnaise in the home has been produced (Whiting and Buchanan, 1997). Results are presented for four scenarios, some of which resulted in acceptable results, but in one, 32% of simulations resulted in a probability of infection exceeding $10^{-2}$. The model uses a selection of input point estimates and so does not attempt to predict real life risk, but rather is used more to emphasise the importance of CCPs such as correct pasteurisation and storage temperatures.

A risk assessment has been published concerning the use of cracked eggs in Canada (Todd, 1996). One output from the risk assessment is that cracked eggs were at least three, and up to 93, times more likely than intact eggs to cause outbreaks of salmonellosis. It was estimated that cracked eggs could be a vehicle for up to 10,500 cases of salmonellosis per annum. The risk of salmonellosis from the consumption of cracked shell eggs was 1 in 3800.

An analysis of consumer storage and use of eggs in Belgium has been made (Grijspeerdt and Herman, 1999). This used data on storage times and temperatures and categorisation of the kinds of dishes prepared into low, medium and high risk to produce a “hazard index” histogram. 98.2% of the outcomes of 10,000 iterations of a Monte Carlo simulation fell into the “low hazard” category. It was concluded that most respondents did not show hazardous behaviour.

An overview of a risk assessment is given in the most recent UK report of *S. Enteritidis* in eggs (Advisory committee on the microbiological safety of food, 2001). It was concluded that sufficient quantitative data are not available to allow the production of a “reliable quantification of risks”.

A comprehensive risk assessment, again focused on *S. Enteritidis* has been produced by the USDA FSIS (*Salmonella* Enteritidis risk assessment team, 1998). The objectives were to;

- establish the unmitigated risk of foodborne disease from *S. Enteritidis*;
- identify and evaluate potential risk reduction strategies;
- identify data needs; and,
- prioritise future data collection efforts.

Again, because of the focus on *S. Enteritidis* and the use of US consumption data the model is not directly relevant to New Zealand. However the concepts used could form the basis of a New Zealand model. It has been produced using @Risk software.
6.2.5 Secondary transmission

Secondary transmission of *Salmonella* in outbreaks is a recognised phenomenon. Carriage in faeces post illness can be quite substantial, with numbers approximating $10^6-10^7$/g persisting up to 10 days post illness. Reduction in numbers with time is variable but a count of $6 \times 10^3$/g has been recorded in one patient 48 days post illness (Pether and Scott, 1982).

6.3 Qualitative Estimate of Risk

Eggs are a commonly consumed food in New Zealand, as elsewhere. The exposure from this food/hazard combination is likely to be low, for the following reasons:

- the important pathogenic serotype *S*. Enteritidis PT4 is not established in the New Zealand egg supply;
- two surveys (of limited sample size) have shown an absence of internal contamination of eggs by salmonellae in New Zealand; and,
- one of these surveys also showed an absence of external contamination of eggs by salmonellae in New Zealand; the other survey found a significant level of external contamination.

Internal contamination of eggs represents the greatest opportunity for pathogen growth and therefore higher risk. Growth on the exterior of eggs is unlikely, and any risk derives from the potential for cross contamination during egg handling.

Egg consumption was not identified as an elevated risk factor in two case-control studies of salmonellosis. Although eggs and egg dishes have been suspected in approximately 1% of reported outbreaks of salmonellosis since 1997, in only one small outbreak (2 cases), has some supporting laboratory data been obtained.

The serotypes found on the exterior of eggs in the 2001 Auckland survey are not the most common serotypes from human cases in New Zealand, but they did occur in up to 3% of cases in 2002.

Overall therefore, the risk to New Zealanders from salmonellae in eggs appears to be low. The level of external contamination on eggs requires further investigation.

6.4 Risk Categorisation

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

The proportion of severe outcomes (hospitalisation, long term sequelae, and death) resulting from salmonellosis has been estimated as approximately 1% based on New Zealand Health Information Service data (Lake *et al.*, 2000) although reported fatalities amongst notified cases is closer to 0.1%. The proportion of outbreak cases that are hospitalised is approximately 10% (Table 5) but these cases may not represent the outcome distribution in sporadic cases as well. Therefore, salmonellosis is assigned to the middle severity category 2 (i.e. severe outcomes represent between 0.5 and 5% of all cases).
The current rate of foodborne salmonellosis, both reported and unreported, is likely to be lower than the estimated rate for 1998 of 230 per 100,000 (Lake et al., 2000), given that the reported rate of salmonellosis for 2003 is lower than that for 1998 (Table 6). This estimate was based on several assumptions, the most important of which was that 92% of all cases of salmonellosis were caused by foodborne transmission. This estimate was based on estimates published for the USA by the USDA Economic Research Service (87-96%) (Buzby et al., 1996) and the Centres for Disease Control (95%) (Mead et al., 1999).

Eggs will be the vehicle in only a small proportion of these foodborne salmonellosis cases. The mode of transmission reported for outbreaks in New Zealand suggest that eggs may be involved in a small proportion (approximately 1%) of salmonellosis outbreaks reported from 1997 to 2003. This suggests that Salmonella transmission in eggs should be assigned to Category 1 in terms of incidence (i.e. between 1 and 10 per 100,000), albeit the lower end of this range.

### 6.5 Summary

<table>
<thead>
<tr>
<th>Food/hazard combination</th>
<th>Severity</th>
<th>Incidence</th>
<th>Trade importance</th>
<th>Other considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Salmonella</em> in/on eggs</td>
<td>2 (0.5 – 5 % serious outcomes)</td>
<td>1 (1-10 per 100,000)</td>
<td>Low, due to small export market</td>
<td>New Zealand poultry is free of two serotypes which have caused major problems in poultry and eggs overseas. This also has implications for imports of poultry and eggs into New Zealand.</td>
</tr>
</tbody>
</table>
7 RISK MANAGEMENT INFORMATION

7.1 Relevant Food Controls

7.1.1 The Animal Products Act

The Animal Products Act 1999 reforms the New Zealand law that regulates the production and processing of animal material and animal products to:

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

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


The risk management system potentially applies anywhere in the value chain from production, through processing to the market. The risk management system comprises the following main types of controls:

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

The Animal Products Amendment Act 2002 introduced staggered dates for different industries to comply with the Animal Products Act 1999. Egg producers that pay a commodity levy (i.e those that purchase 100 or more hens in a year) must have a RMP in place by 1 July 2004. All other egg producers must have a RMP by 1 July 2005.

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

The Animal Products Act 1999 requires egg producers to have a risk management programme (RMP) to control hazards and other risk factors so that shell eggs are fit for their intended purpose. The RMP must cover their primary processing operation (from the laying farm to packing of shell eggs). The Egg Producers Federation of New Zealand (http://www.epfnz.org.nz/) in conjunction with the New Zealand Food Safety Authority, have developed a Code of Practice to support the development of RMPs by producers.

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Risk Profile: Salmonella (Non Typhoidal) in and on eggs
7.1.2 Egg Producers Federation of New Zealand Inc Code of Practice

As mentioned above, a Code of Practice (CoP) for New Zealand egg producers is being developed as a cooperative effort between the NZFSA and the Egg Producers Federation. Draft 7 of the Code of Practice was released for comment on 1 August 2002, and is available at: www.nzfsa.govt.nz/animalproducts/publications.

The scope of the Code is the primary processing of shell eggs and other egg products that are intended for human or animal consumption. It does not cover secondary processing of eggs (e.g. pulping and pasteurisation), bird welfare, or environmental issues.

The Code includes a valuable Technical Annex as Appendix C which covers hazard identification and contextual information, as well as a summary of the overseas situation regarding hazards in eggs. The Annex covers many of the same topics as this Risk Profile.

The following material is taken from the CoP, and is relevant to egg cleanliness and Salmonella control.

7.1.3 Poultry industry on-farm controls to prevent layer flock contamination by Salmonella in New Zealand

On the layer farm, flocks are subject to a whole flock health scheme as described in the Animal Products (Specifications for Products Intended for Human Consumption) Notice 2002, Clause 106, see: http://www.nzfsa.govt.nz/animalproducts/legislation/notices/animal-intend-oct-02.pdf. This is a general requirement that layer flocks “are subject to and comply with a whole flock health scheme designed to ensure that hazards associated with eggs which are likely to affect human health are identified and managed in an appropriate manner”.

Birds on rearing and layer farms are vaccinated with an attenuated S. Typhimurium vaccine (MeganVac) under veterinary supervision. The vaccine is protective against S. Typhimurium but also reduces infection with S. Enteritidis; (see: http://www.usaha.org/reports/reports00/r00sal.html). Vaccination takes place at three stages; at day old in the hatchery, at two-six (2-6) weeks and between thirteen - sixteen (13-16) weeks of age. The procedures used are described in Appendix H of the Egg Producers CoP.

The flock health scheme includes Salmonella surveillance:

“Salmonella surveillance is to be done during rearing and laying phases: A foam or gauze drag swab per shed from a representative sample of cages or rearing area shall be taken once the birds reach 6 weeks of age, and again at 12-16 weeks of age (i.e. one environmental swab at each rearing ‘stage’).

If Salmonella-positive samples are returned from these tests, the Salmonella shall be serotyped, and a thorough cleaning programme is to be undertaken, as per the documented response procedure. This procedure is likely to include a thorough cleaning and sanitisation at depopulation, and retesting of the cleaned shed to achieve Salmonella-negative status prior to repopulation.
If a *Salmonella* Enteritidis PT4 is returned at any time the egg producer shall notify NZFSA and the Egg Producers Federation, and shall recall eggs from affected flocks. Eggs from affected flocks shall not be offered for sale. The affected flocks shall be quarantined and, if confirmatory tests are returned, immediate depopulation should follow.

All testing for *Salmonella* shall be undertaken by a laboratory accredited to nationally or internationally recognised standards, such as ISO or IANZ.” (Chapter 3:58).

Other control measures include;
- Management of *Salmonella* in water;
- Testing for *Salmonella* in feed ingredients and finished feed with controls applied when positive results are returned (e.g. heat treatment or the addition of control agents);
- Biosecurity measures;
- Rejection of very dirty eggs;
- 100% of all floor eggs separated from other eggs;
- 100% visibly cracked eggs separated, those with broken membranes to be dumped; and
- Storage and transportation of Grade A and commercial eggs not higher than 15°C.

*Salmonella* controls are also mentioned under pest control (Chapter 3:30-34), internal environs (Chapter 3:35-39), external environment (Chapter 3:40-41), personal hygiene and biosecurity (Chapter 3:42-46).

7.1.4 Poultry industry processing controls to control egg contamination by *Salmonella* in New Zealand

The Code describes four grades of eggs:

A grade shell eggs = eggs without visible cracks or internal defects so are suitable for retail sale for human consumption.

Commercial eggs = eggs without visible cracks, but may have size/shape abnormalities or other minor defects that do not compromise egg safety or wholesomeness – not for retail sale in shell but still suitable for human consumption. These eggs are normally sold for catering or other similar uses.

Cracked eggs = eggs that can be sent for further processing (Pasteurisation or equivalent) or for animal consumption.

Reject eggs = eggs unsuitable for human or animal consumption.

*Salmonella* guidelines for different egg grades are provided in the example RMP (reproduced from Chapter 4 of the CoP):
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A Grade Eggs: <em>Salmonella</em> not detected in 25g from a weekly composite sample of A grade shell eggs.</td>
<td>• Dirty eggs and visibly cracked eggs are separated from these eggs.</td>
<td>• Increase test frequency. Divert eggs from known positive flocks to further processing with a bactericidal control point.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Commercial Eggs: <em>Salmonella</em> level – not yet defined.</td>
<td>• Storage and transportation temperature not higher than 15°C.</td>
<td>• Rework eggs that are still on site to meet requirements.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Visibly cracked eggs are separated from these eggs.</td>
<td>• Review refrigeration systems.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• No dry cleaning of eggs.</td>
<td>• Notify Laying Farm of issues that may relate to them.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• All dirty eggs and floor eggs to be washed in accordance with the ICMSF guidelines on pageC-51 of Appendix C: Technical Annex.</td>
<td>• Review packhouse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Storage and transportation temperature not higher than 15°C.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hazard or other risk factor</td>
<td>Aim of RMP</td>
<td>Example Product outcomes</td>
<td>Key Control Measures</td>
<td>Response if outcome not met</td>
<td></td>
</tr>
<tr>
<td>----------------------------</td>
<td>------------</td>
<td>--------------------------</td>
<td>----------------------</td>
<td>-----------------------------</td>
<td></td>
</tr>
<tr>
<td>Cracked Eggs: <em>Salmonella</em> level – not yet defined.</td>
<td></td>
<td>• All eggs that do not have an intact membrane are separated from these eggs.</td>
<td></td>
<td>RMP.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• All eggs with cracks visible on candling but intact membranes are labelled for further processing or animal consumption.</td>
<td></td>
<td>• Retrain staff.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Storage and transportation temperature not higher than 6°C.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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The CoP also includes information on “best before” dates and storage recommendations:

“Labelling must comply with NZ Food Regulations 1984. The shelf life (Best before date) for A grade and Commercial eggs are (maximum) 35 days from date of lay at or below 15 °C. Dates must be fixed to the packaging, including trays at grading, with refrigeration guidelines on the packaging. Cracked eggs have max of 14 days with refrigeration at or below 6°C”.

Annex C-52 of the Egg Producers Code refers to a New Zealand supermarket CoP which requires eggs to be held at 15°C, for a shorter shelf life of 30 days.

The Code provides instructions about separation of egg types:

“4. The following eggs must be rejected at the farm and not delivered to the packhouse:
   • Very dirty eggs (soiled area over the size of 20 cent coin), and
   • Visibly cracked eggs.

5. The following categories of eggs must be collected, kept and delivered in separate containers:
   • Good eggs
   • Floor eggs or slightly soiled eggs
   • Eggs that are unusual shapes or sizes or have minor defects.”

The draft implies that washing of Grade A eggs is unnecessary as dirty eggs (an area not over the size of a 20 cent coin) and floor eggs are removed from this grade to become commercial eggs. The commercial eggs requirements are:

   • No dry cleaning of eggs.
   • All dirty eggs and floor eggs to be washed in accordance with the International Commission on Microbiological Specifications for Foods guidelines on page C-51 of Appendix C: Technical Annex.
   • Under Critical Control Point 4 (Chapter 4:49), all eggs with a total soiling area greater than a defined surface area to be washed.

The defined surface area is clarified by the footnote;

   “To be set by egg producer. To define the surface area use an actual size e.g. 1 square cm, or refer to something of known size, e.g. 5 or 10 cent coin (as appropriate).”

At the farm level, very dirty or cracked eggs are rejected. During processing, cleaning instructions for the remaining eggs include:

Step 6: Dirty, cracked, or broken eggs where detected shall be removed from the collection system prior to grading. In automated systems where eggs are directly conveyed to the grader, a pre-candling station may be required to remove dirty, cracked, or broken eggs.

Step 7: Appropriate egg washing / drying and oiling procedures in accordance with the ICMSF recommendations given on page C-50 of Appendix C: Technical Annex shall be documented. Dirty eggs may be cleaned by dry buffing provided the egg shell cuticle is not damaged. Manual wet cleaning or wiping of eggs is not permissible.” (Chapter 4:51)
Note that the last instructions regarding dry cleaning are different to those concerning Grade A eggs above.

Where mention has been made of secondary processing such as pasteurisation for cracked eggs, the controls in place are as follows;

Pasteurisation standards for egg and egg products are given in Standard 2.2.2 of the Australia New Zealand Food Standards Code. The Standard states that egg products must be pasteurised or undergo an equivalent treatment to meet the microbiological standard 1.6.1. Australia has a further processing Standard (1.6.2) regarding the processing of egg products. (In New Zealand this is regulated under Animal Products Act 1999).

A New Zealand pasteurised egg manufacturer has advised that when exporting, the standard of pasteurisation used depends on the customer country requirements. For the New Zealand domestic market, the Australian standard (1.6.2) is used (Tecklok Wong, ESR, pers. comm.).

### 7.1.5 Control measures overseas

Control measures have, in the main, been implemented to control S. Enteritidis transovarian transmission. However, many of the controls will also minimize potential problems from other serotypes which may be introduced into the egg.

Other Country’s requirements and Code of Practice/Control systems are set out in pages C61 to C66 of the Technical Annex to the NZ CoP draft 7.

The Technical Annex to the NZ CoP describes the rationale for egg storage conditions(C-52). The age of the yolk is the principal factor in the growth of S. Enteritidis (Humphrey and Whitehead, 1993). Growth rates are more rapid in eggs 21 days or older. The yolk membrane becomes more permeable during storage (especially after 3 weeks). When eggs are stored above 20°C, S. Enteritidis can multiply. The 1993 report from the UK ACMSF recommended that eggs be maintained at temperatures below 20°C and consumed within 21 days.

The USDA FSIS Risk Assessment estimated that a 12% reduction in risk could be achieved if eggs were immediately cooled after lay to an internal temperature of 45°F (7.2°C) (Technical Annex C-53). However, rapid cooling may also increase the risk of shell cracking (see Section 7.2).

The Australian Code of Practice recommends that eggs are stored “below 20°C at the farm, during transport and at the retail outlet, in conditions which avoid surface condensation or contamination” (AEIA, 2001, referenced in Technical Annex C-54)
7.1.6 Controls in the USA

In 1990 the USDA initiated a programme of compulsory testing for *S.* Enteritidis in breeding flocks that produce egg-laying chickens. Tracebacks to farms were also carried out when eggs were implicated in outbreaks of disease and follow up investigations carried out where possible. If *S.* Enteritidis was detected eggs were diverted to be pasteurised. Funding was discontinued in 1995, but the FDA has continued the programme (Centers for Disease Control and Prevention 1996). Similar work to control *S.* Enteritidis in flocks has been carried out in Pennsylvania in the Pennsylvania Egg Quality Assurance Programme (PEQAP).

On December 10 1999, the President’s Council on Food Safety released a document, Egg Safety From Production to Consumption: An Action Plan to Eliminate *Salmonella* Enteritidis Illnesses Due to Eggs (http://www.foodsafety.gov/~fsg/ceggs.html). It is estimated that one out of every 20,000 eggs produced in the US is contaminated with *S.* Enteritidis.

The Action Plan goal was to reduce foodborne related illnesses associated with *S.* Enteritidis in eggs by 50% by 2005 and to eliminate egg-associated *S.* Enteritidis illnesses by 2010.

The following strategies were identified in the action plan;

**Objective 1**: Reduce the number of SE-containing eggs marketed to the consumer

**Objective 2**: Reduce exposure of consumers to SE-containing foods

**Objective 3**: Expand and upgrade surveillance systems for human SE infection

**Objective 4**: Expand and upgrade surveillance systems for poultry SE infection

**Objective 5**: Accelerate SE outbreak detection and initiation of investigations

**Objective 6**: Improve coordination and communication related to outbreaks

**Objective 7**: Ensure information is available to make science-based decisions

**Objective 8**: Develop and distribute science-based educational materials

The Food and Drug Administration issued refrigeration and safe handling labels regulations which came into effect in June and September 2001 respectively. The rationale behind these regulations can be found at http://vm.cfsan.fda.gov/~lrd/fr99706a.html.

This means that eggs upon delivery at retail establishments including caterers, must be placed promptly under refrigeration at or below 45°F (7.2°C). In addition, the shell egg carton must bear the following required statement;

“SAFE HANDLING INSTRUCTIONS: To prevent illness from bacteria: keep eggs refrigerated, cook eggs until yolks are firm, and cook foods containing eggs thoroughly”.

At present, there is no US federal law requiring an expiration date to be marked on cartons.

The American Egg Board (http://www.aeb.org) and United Egg Producers have developed a voluntary Quality Control Campaign that includes *Salmonella* controls. The controls are described on the website as:

“Egg industry programs start by keeping breeder flocks free of *Salmonella*. Ongoing research is dedicated to discovering how *Se* gets into flocks and how it might be blocked. The industry also uses strict quality-control practices and sanitation procedures all through production,
processing and preparation. This includes testing chicks to be sure they’re free of Salmonella, bio-security (such as washing and sanitizing not only the eggs, but facilities, too) and other measures. To block $Se$ from multiplying in the egg in the rare event it’s present, eggs are held at cool temperatures following packing and throughout transportation. Important, too, are industry education programs which encourage food preparers to use safe food-handling practices.”

7.1.7 Control of Salmonella Enteritidis PT4 in the United Kingdom

The UK experienced more than a 170% increase in reported human salmonellosis cases between 1981 and 1991. The number of laboratory confirmed cases of human salmonellosis in the UK, particularly $S$. Enteritidis PT4, has shown a steady decline from 1998 onwards (ACMSF 2001). This has continued since 1998, with cases of infection with PT4 now at their lowest level since the late 1980s (Cogan and Humphrey 2003). The measures to achieve this decline included advice from the Advisory Committee on Microbiological Safety of Food (ACMSF 1993) report on Salmonella in eggs. The Chief Medical Officer made the following recommendations;

- No-one should eat raw eggs;
- Vulnerable groups should eat only eggs that have been cooked until both the white and yolk are solid;
- Eggs should be used within 3 weeks of lay and “use-by” dates should be provided on egg packs and possibly on eggs;
- Eggs should be kept at a constant temperature during storage, transport and retailing and should never exceed 20°C;
- Once purchased, eggs should be stored in a refrigerator;
- Pasteurised eggs should be substituted in raw or lightly cooked dishes; and
- A CoP to cover the handling and storage of eggs from farm to retail.

Egg assurance schemes such as the “Lion mark” have been credited in part, with the reduction in human Salmonella Enteritidis PT4 infections since 1996. The British Egg Industry Council (BEIC) represents 11 major associations of the industry and is recognised by the UK government as the official representing body of the UK egg industry. The BEIC has developed a Code of Practice for “Lion Quality” eggs. A registered trademark, the Lion quality mark can only be used by subscribers to the BEIC on eggs produced in accordance with UK/EU law and the Lion Code of Practice. The CoP was updated in 1996 and 1998. The Code includes;

- Vaccination of all pullets destined for Lion Quality egg producing flocks, against $S$. enteritidis PT4. (It should be noted that some producers not under the BEIC Lion mark scheme also vaccinate against Salmonella. It is estimated that at least 80% of all UK laying hens are vaccinated against $S$. Enteritidis);
- 21 day best before date printed on the shells as well as the egg pack of all Lion Quality eggs. Since 1 January 2000 the red Lion mark has been stamped on all individual eggs. (Under European Union legislation, the maximum “best before” date for eggs is 28 days after point of lay.);
- Complete traceability of Lion Quality eggs through a passport system; and
- Increased independent monitoring of the Code.

(See: British Egg Information Service BEIS website; http://www.britegg.co.uk/beis/beis2nf.htm).
The UK FSA has issued advice on the use of raw and partially cooked eggs by catering premises (see: http://www.food.gov.uk/multimedia/pdfs/eggleaflet.pdf). This guidance has been promoted during routine inspections by enforcement officers. The advice includes refrigeration of eggs in storage, avoid cross-contamination from eggs to ready to eat foods and ensure eggs are cooked thoroughly.

In a letter to “Science” several scientists at the UK Public Health Laboratory Service discussed the epidemic of *Salmonella* Enteritidis PT4 (Ward et al., 2000). Epidemiological investigations indicated that poultry breeding lines infected with PT4 were introduced into the United Kingdom around 1982-3, probably originating in elite flocks in continental Europe. The reasons for the decline in reported cases of salmonellosis since 1997 were described as multifactorial, including:

- the fact that several codes of practice for the control of salmonellae in chickens had been in operation in the UK since 1993;
- there had been many improvements in the poultry industry in infection control and hygiene at breeding sites; and
- in 1994 vaccination against *Salmonella* Enteritidis started in breeder flocks, and in 1998 in layer flocks.

In the UK legislative control measures for eggs were introduced in March 1989 by a Zoonoses Order (Advisory Committee on the Microbiological Safety of Food, 1993). These measures were:

- an obligation on owners of poultry flocks and processors or importers of processed animal protein to undertake regular bacteriological monitoring of birds or product on their premises or in their possession; and
- required all isolations of *Salmonella* to be notified to the Ministry of Agriculture, Fisheries, and Food (MAFF).

These statutory measures were supplemented by the introduction and adoption of voluntary Codes of Practice for the control of *Salmonella* in feedingstuffs, laying flocks, breeding flocks, and broilers, and were also backed up by a programme of research.

Compulsory slaughter of laying flocks infected with *Salmonella* Enteritidis has ended in the UK, however, if *Salmonella* Enteritidis or *Salmonella* Typhimurium is confirmed in a breeding flock, no further eggs may be sent for hatching and the flock is destroyed. Codes of Practice for control of *Salmonella* in laying flocks complement the statutory control programme and codes for breeding flocks and codes for control of *Salmonella* in hatcheries (MAFF, 2000).

From European law (see section 7.1.8) Council Decision 94/371/EC is interpreted in the UK by the Eggs (Marketing Standards) Regulations 1995 and is currently the only statutory storage requirement. A Voluntary Code of Practice on the handling and storage of eggs from farm to retail level published by MAFF in the UK (Publication reference PB2828) provides a target storage temperature of below 20°C.

An extensive report on the problem of *S. Enteritidis* in eggs in the UK is available (Advisory committee on the microbiological safety of food 2001).

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Risk Profile: *Salmonella* (Non Typhoidal) in and on eggs

May 2004
7.1.8 Egg marketing measures in Europe

The European Union Council Decision 94/371/EC states that “At the producer’s premises and until sale to the consumer, eggs shall be kept dry, out of direct sunshine and shall be stored and transported at a preferably constant temperature”.

In addition, the European Commission Council Regulations require that for all establishments with more than 350 egg laying hens, all Class A eggs sold in the EU must have their shells stamped with a distinguishing code (obtained on registration with the appropriate national authorities) from 1 January 2004. Primarily this will assist in tracing eggs which are a source of outbreaks to the exact establishment they originated from.

Only two classes of eggs; A and B, can be sold from 1 January 2004. This follows the merger of Classes B and C to Class B. Class B eggs can only be sold to industry, they must be marked as such and the packing date stated to the purchaser. Eggs displayed for retail sale must have the quality, weight gradings and farming method clearly displayed on the shop shelves. From 1 July 2005, ungraded eggs sold at local public markets will need to be marked with a code identifying method of production and the establishment, irrespective of flock size. This is to deter fraud and improve traceability.

There is a pending scientific report by the European Food Safety Authority (EFSA, http://www.efsa.eu.int/) which concerns the risks of washing eggs. Until its publication, egg washing will be permitted for Class A table eggs (as long as it is stated on the packaging) for a three year period.

7.1.9 Control of Salmonella in Denmark

Control of Salmonella in eggs is part of a larger programme aimed at controlling the organism in eggs, poultry and pork (Wegener et al. 2003). All commercial layer flocks are tested every nine weeks, as part of a much more comprehensive programme, and infected flocks slaughtered. Eggs from flocks which are contaminated are pasteurised, and all shell eggs are distributed under refrigeration (<12°C) and eggs are generally stored refrigerated in private homes. Owners of infected flocks are compensated, and the result of the programme is a reduction in Salmonella positive layer flocks from around 7% to around 2%.

By using an analysis of salmonellae serotypes associated with particular foods in order to ascribe the proportion of salmonellosis caused by each food, the paper notes that the incidence of salmonellosis caused by egg consumption reduced from 57.7 cases/100,000 in 1997 to 15.5 in 2001. Implementation of a similar programme in New Zealand would most likely not result in a similar reduction in incidence because of the absence of serotypes involved in transovarian infection. Cost benefit analysis indicates a net benefit measured in millions of dollars for Denmark.
7.1.10 Management options for cracked eggs in Canada

Eight options for the use of cracked eggs were proposed by Health Canada (Todd, 1996). In priority order these were:

1. Cracked eggs will be pasteurized in existing egg breaking stations and new breaking stations will be constructed.
2. Cracked eggs will not be allowed to be sold except to egg breaking stations for pasteurized liquid egg. There are currently 13 egg breaking stations across Canada, and no new egg breaking stations will be built.
3. Cracked eggs may be sold directly to a food processing plant operating under Good Manufacturing Practices (GMPs), if the product in which the cracked eggs are used is guaranteed by the manufacturer to receive a heat treatment equivalent to that specified in the AAFC Processed egg regulations.
4. Cracked eggs may be sold from registered egg stations in appropriately stored and marked cartons directly to consumers, if the station is situated on the producer’s premises and is grading eggs produced solely on these premises.
5. Cracked eggs may be sold from registered egg stations in appropriately stored and marked cartons directly to consumers from registered egg stations grading eggs from other producers.
6. Cracked eggs may be sold from registered egg stations to retail stores for resale to consumers; however, no direct sales to institutions or bakeries would be permitted.
7. Sales of cracked eggs from egg stations to restaurants would be permitted by provinces requesting this.
8. Cracked eggs would be sorted by candling into those with fine cracks (hair line) and those with visible cracks, with the former being acceptable for sale to the public at the retail level and the latter being sold exclusively for pasteurized liquid egg.

Options 1 to 3 were considered to be acceptable, but 4-6 only under specified conditions. Option 8 was not considered to be feasible.

7.1.11 Control of S. Enteritidis in Sweden

The philosophy adopted in Sweden is to “deliver Salmonella-free food to the consumers”. This is achieved through ensuring that animals destined for slaughter are free from Salmonella by preventing contamination at all points of the production chain and monitoring the production chain at critical control points for Salmonella. When detected, specific actions are taken to achieve the stated objective (Wierup et al., 1995). Salmonella-infected poultry are not allowed to be imported into Sweden.

Grandparent birds are imported as day old chicks, which are certified as originating from Salmonella-free flocks, and kept in quarantine for 15 weeks. During quarantine the animals are tested four times for Salmonella, and if the pathogen is detected the animals are destroyed.

In regard to Salmonella control in layers, in the period of 1988-1989 hatcheries, breeders for layers and eggs destined to be processed to produce pasteurised egg powder were tested. Breeder flocks and hatcheries yielded no positive results, but 18 of 381 egg samples were
positive (none with S. Enteritidis). In 1990 a voluntary programme, which later became mandatory in 1994, was introduced whereby layer flocks were tested prior to slaughter. The isolation of S. Enteritidis or S. Typhimurium resulted in the destruction of the flock, whereas the isolation of other serotypes resulted in the implementation of different controls. In 1994 the scheme was changed to include testing twice during the production period.

In addition the use of heat-treated feed is being introduced for layer flocks. The level of disease caused by Salmonella in the Swedish population has risen, but only 15-20% are reported as being acquired domestically.

### 7.2 Egg Cleaning

Eggs may be cleaned by various means, but the requirement to do this varies between countries. Wet or dry cleaning of eggs is designed to decrease faecal contamination of the egg surface, but each method brings with it problems that need to be controlled. Washing eggs in water cooler than the eggs has been suggested to result in bacteria being drawn through the shell, but washing them in water warmer than the eggs causes a rise in egg temperature making subsequent cooling more difficult (Lucore et al., 1997). These authors showed that spray washing with water at 15.5°C resulted in no more internal contamination than spray washing with water at 32.2 or 48.9°C. Bacterial counts on washed eggs have been shown to be correlated with counts on washing equipment (Moats, 1981), and the major source of contamination of the washwater was judged to be the equipment and not the unwashed eggs. A recent study showed a more than 5 log_{10} reduction in salmonellae numbers on eggs cleaned using commercial equipment (Hutchison et al., 2004). The process was robust to changes in a number of processing parameters, except for the temperature of the water used, which had to be strictly controlled for the reasons given above. Despite challenges with high numbers of two salmonellae, none of the eggs tested showed contamination of the contents when washed according to the equipment manufacturer’s instructions.

Some countries mandate the refrigerated storage of eggs, but this is not the case in New Zealand.

Cool storage of eggs as practiced in USA is designed to minimize the growth of any S. Enteritidis present in the egg’s contents (Fajardo et al., 1995). However, this same publication demonstrated increased susceptibility to contamination across the shell during rapid cooling, and larger microscopic cracks, as large as 5-10 μm, in the shells of rapidly cooled eggs.

Studies in the UK examined egg packing facilities that used eggs from farms with flocks known to be infected by S. Enteritidis (Davies and Breslin, 2003). Contamination on packing facilities that had been cleaned and sanitized the previous day varied from 6.9% to 21.4% depending on the surface sampled. Sterilised eggs which were put through the packing plants were contaminated at a rate of at least 0.3%. It was concluded that contamination in the processing facility may be a significant factor in the external contamination of shell eggs.

Various coatings, such as polysaccharides, proteins and lipids, have been studied with respect of their ability to improve keeping quality of eggs by reducing weight loss and preventing...
bacterial penetration (Bhale et al., 2003). This paper reports on the use of chitosan to extend shelf life, but does not address microbial penetration of the egg.

A study of the efficacy of three commercial cleaning and sanitising compounds (sodium carbonate, sodium hypochlorite, and potassium hydroxide) used eggs contaminated externally with S. Enteritidis by immersing them in suspensions of the bacteria at varying levels (Soljour et al., 2004). None of the chemicals applied at the manufacturers recommended concentrations were effective at eliminating S. Enteritidis at $10^4$ or $10^6$ cfu/ml, but bacteria applied at $10^2$ cfu/ml were eliminated. Inactivation was more effective with solutions at pH12 than lower pH.

7.2.1  Ultraviolet (UV) surface treatments of whole eggs

A number of studies have been carried out into the efficacy of UV treatments for the control of bacteria on eggs.

UV radiation has been widely accepted as a treatment for treating water and other liquids. In 1956, a ‘centrifilmer’ was developed for sterilising biological fluids. This system, called ‘cold pasteurisation’, allowed fluid such as liquid egg to pass through UV radiation in a thin flowing film. In a 1964 study, Ijichi et al. reported that Salmonella Senftenberg 775W, (approximately ten times more heat resistant than ordinary strains of Salmonella), reacted similarly to S. Typhimurium when UV irradiated. Ijichi reported in 1966 that there was no significant difference between a heat resistant strain of Salmonella and a standard strain under the same UV irradiation conditions.

A study by Gao et al. (1997) evaluated the effectiveness of UV in reducing Salmonella on egg shells for possible egg production facility use. The intensity of UV used was calculated so that it would not penetrate through to the egg contents and so would be a safe method for the consumer. The study used four parameters;

- test materials: egg shell, plastic belt, fibre belt metal;
- UV intensity ($\mu$W/cm$^2$): 6000, 9000, 12000;
- time of UV exposure (s): 5,10,15; and,
- initial salmonellae contamination levels (CFU/cm$^2$): $10^8$, $10^6$, $10^4$.

It was evident that the smoothness of the test material surface significantly affects the effectiveness of Salmonella decontamination by UV. The following conclusions were drawn;

1) “Ultraviolet irradiation is a safe decontamination agent for eliminating Salmonella on whole eggs.
2) Within the range of probable contaminating Salmonella in the natural environment, UV effectively decontaminated Salmonella on all four test materials.
3) Regression models for each material explained the relationship among the factors in the test range. These equations may be used as prediction models for future studies.
4) Comparison tests demonstrated that Salmonella was easier to eliminate from the plastic belt than from other materials tested; fiber belt was the most difficult; and egg shell and metal were within median range.
5) Within the test ranges, effectiveness of decontamination of *Salmonella* depended more on the length of UV exposure time and initial *Salmonella* contamination level than on UV intensity (data not shown):”

Two studies carried out by Kuo *et al.* (1997) examined the appropriate time and intensity of UV exposure to give the most effective bactericidal activity in conjunction with the effect of egg rotation. The second study looked specifically at inoculated *S. Typhimurium* populations and the effect of UV treatments on both ends of the egg. Both studies concluded that UV radiation can significantly reduce aerobic micro-organisms, yeast, moulds and *S. Typhimurium* populations. It should be noted that these studies used visibly clean unwashed shell eggs.

UV radiation has also been tested for its effect in reducing *Y. enterocolitica* on eggshell surfaces (Favier *et al*., 2001). Again visibly clean eggs were used. At the highest level of natural bacterial contamination on the eggshells in this study (4.55 log CFU/egg), 50 minute UV exposure (at approx. 4,573 µW/cm²) led to no residual agar plate growth from swabs of egg surfaces.

7.2.2 Ionising radiation for shell eggs

In July 2000, the US Food and Drug Administration approved the use of ionizing radiation for the reduction of *Salmonella* in fresh shell eggs. The regulation allows a dose up to 3kGy; although doses near this level had an effect on the yolk colour and viscosity, the dose did not raise any safety concerns. Elimination of *Salmonella* by this treatment is dependent on initial contamination levels and the dose that is absorbed. At practical dose levels, salmonellae levels may be reduced by 10 – 10,000 fold. Irradiated eggs must be labeled accordingly (FDA, 2000).

As of May 2004, the use of ionizing radiation as a food treatment is not permitted in New Zealand (except for the treatment of certain herbs and spices and certain tropical fruits).

7.3 Economic Costs

The annual economic cost of foodborne salmonellosis in New Zealand has been estimated at $4,463,000 (8.1% of the total cost of foodborne illness) (Scott *et al*., 2000). The number of cases and outcomes used for this estimate were based on an average of notification and hospitalisation data from 1991 to 1998 (Lake *et al*., 2000).

This estimate was based on several assumptions, the most important of which was that 92% of all cases of salmonellosis were caused by foodborne transmission (see Section 6.4).

The emergence of *S. Brandenburg* and *S. Typhimurium* DT160, which now account for approximately 20% of typed human *Salmonella* isolates (ESR, 2001), suggests that this figure may be too high for New Zealand. The transmission routes for these serotypes have not been fully characterised, but it appears that a proportion of human cases are zoonotic in origin (Thornley *et al*., 2002). It seems likely that New Zealand has a higher level of farm-level exposure to animals compared to the USA, and so this transmission route may be of more importance here, thus reducing the proportion of foodborne illness.
The estimated dollar value includes direct and indirect medical costs, the value of productive
days lost, and the statistical value of mortality, but not the value of lost quality of life.

This estimate covers all potential food vehicles. No data are available on the proportion of
transmission due to eggs alone, but based on the qualitative estimate of risk this should be
minor.
8 CONCLUSIONS

8.1 Description of Risks to New Zealand Consumers

8.1.1 Risk associated with egg products

The rates of reported salmonellosis have fluctuated considerably since 1985, within the range of 35-65 per 100,000. No clear upward or downward trend is apparent in recent years. The rate of reported salmonellosis in New Zealand is higher than for Australia, and most of the other countries reported in Table 8 apart from the United States. This may be due to differences in reporting systems.

The serotype information on human isolates of Salmonella reveals a dynamic situation. Two serotypes that have emerged since 1997 have been S. Typhimurium DT160 and S. Brandenburg. Eggs have not been found to be associated with transmission in case-control studies of each of these serotypes.

Despite the numbers of S. Brandenburg human isolates, and the remarkable increase in S. Typhimurium DT160 human isolates, the majority of human isolates are of a wide range of other types, which may be transmitted by foods, including eggs. Eggs and egg dishes have been suspected in a small proportion (approximately 1%) of reported outbreaks of salmonellosis since 1997. However, in only one small outbreak (2 cases), has some supporting laboratory data been obtained.

The two surveys of salmonellae in retail eggs in New Zealand are consistent in indicating an absence of internal contamination. Exterior contamination by Salmonella was not found in the South Island survey (2056 eggs), but the more recent Auckland survey (558 eggs) revealed an external contamination rate that was high by international standards. Although the serotypes found on the eggs do not feature prominently amongst human isolates, they represent a small proportion of cases. It should be noted that both surveys involved a limited sample size.

There is little evidence that transmission of Salmonella via eggs is a significant transmission route occurring in New Zealand. However, the results from the Auckland survey of eggs deserve further investigation, to determine whether the prevalence is still high, and is consistent throughout the national egg supply. If confirmed, this result may reflect poor egg production practices in some sectors of the industry.

8.1.2 Risk associated with other foods

Other vehicles identified in outbreaks in New Zealand include poultry meat and other meat products (Wilson et al., 2000). The Salmonella spp. in Poultry Risk Profile is currently being updated, but data from the NMD suggest that contamination of poultry meat by salmonellae is low.

In 1999 an outbreak of S. Typhimurium PT135 occurred in Christchurch that was linked to cold cocktail sausages from a butchery (MacLeod, 2000). Takeaway foods were identified as an important risk factor in the S. Typhimurium DT160 case-control study (Thornley et al.,...
Two outbreaks related to umu functions have been reported. In one the implicated food was potato salad which had been improperly stored (Callaghan and Simmons, 2001) and in the other the implicated food was Palusami (umu cooked packs of taro in coconut milk wrapped in taro leaves) that had been privately imported from Samoa (Ng and Simmons, 2002). In 1996 an outbreak of S. Infantis was associated with cold cooked meats but it appeared that this was due to contamination that occurred within the delicatessen (Anonymous, 1996).

Cases of salmonellosis in Auckland in August 2003 were linked to *Salmonella* Montevideo found in sesame based products such as tahini paste, hummus, and halva.

In Europe, *Salmonella* contamination of poultry is frequent, but contamination has also been found in pork, beef, other meat products, raw eggs, and dairy products (Scientific Committee on Veterinary Measures Relating To Public Health, 2000).

8.1.3  **Quantitative risk assessment**

The qualitative assessment of risk for *Salmonella* spp. in eggs indicates that the risk is low and therefore devoting extensive resources to a quantitative risk assessment of this food/hazard combination is not warranted.

8.2  **Commentary on Risk Management Options**

New Zealand is fortunate in having a poultry industry and egg supply in which types of *Salmonella* that have caused major problems overseas (S. Enteritidis PT4 and S. Typhimurium DT104) are not endemic. Import controls on poultry are partially designed to maintain this status. New Zealand cases of human illness caused by these types of bacteria appear to be infections principally acquired overseas.

8.3  **Data gaps**

The most obvious data gap to be addressed is further information on the prevalence of *Salmonella* in retail eggs, and whether there are any differences between free range, caged and barn produced eggs. Any survey should examine both internal and external contamination. If the high level of external contamination found in Auckland is confirmed, then risk management measures, such as cleaning regimes, will need further investigation.
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*in and on eggs*


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APPENDIX 1: CATEGORIES FOR RISK PROFILES

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

1. Incidence

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

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

<table>
<thead>
<tr>
<th>Disease/organism</th>
<th>Food rate (/100,000 population) Calculated for 12 months to June 2001</th>
<th>Food rate (/100,000 population) Calculated for 12 months to December 1998</th>
</tr>
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<tr>
<td>Campylobacteriosis</td>
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<td>2047</td>
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<td>1.4</td>
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</table>

* not recalculated.

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

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

<table>
<thead>
<tr>
<th>Category</th>
<th>Rate range</th>
<th>Comments/examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;1000</td>
<td>Significant contributor to foodborne campylobacteriosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Major contributor to foodborne NLV</td>
</tr>
<tr>
<td>2</td>
<td>10-100</td>
<td>Major contributor to foodborne salmonellosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Significant contributor to foodborne NLV</td>
</tr>
<tr>
<td>3</td>
<td>1-10</td>
<td>Major contributor to foodborne yersiniosis, shigellosis</td>
</tr>
<tr>
<td>4</td>
<td>&lt;1</td>
<td>Major contributor to foodborne listeriosis</td>
</tr>
</tbody>
</table>

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

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

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

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

2. Severity

Severity is related to the probability of severe outcomes from infection with the hazard.

The outcomes of infectious intestinal disease are defined in the estimate of the incidence (Lake et al, 2000) as:

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

The first three categories of cases were classed as severe outcomes. Some hospitalisations will result from dehydration etc. caused by gastrointestinal disease. However, for infections with *Listeria* and STEC hospitalisation will result from more severe illness, even if recovery is achieved.

The proportion of severe outcomes resulting from infection with the hazards can be estimated from the proportion of cases hospitalised and recover, hospitalised and long term illness, and deaths (Lake et al., 2000).

<table>
<thead>
<tr>
<th>Disease/organism</th>
<th>Percentage of outcomes involving death or long term illness from foodborne cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Campylobacteriosis</td>
<td>0.3</td>
</tr>
<tr>
<td>Listeriosis</td>
<td>60.0</td>
</tr>
<tr>
<td>VTEC/STEC</td>
<td>10.4</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>1.0</td>
</tr>
<tr>
<td>Yersiniosis</td>
<td>0.4</td>
</tr>
<tr>
<td>Shigellosis</td>
<td>2.7</td>
</tr>
<tr>
<td>NLV</td>
<td>Assumed to be &lt;0.5%</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>15.4</td>
</tr>
<tr>
<td>Typhoid</td>
<td>83.3</td>
</tr>
<tr>
<td>Toxins</td>
<td>Assumed to be &lt;0.5%</td>
</tr>
</tbody>
</table>

Categories for the probability of severe outcomes are suggested as follows:
<table>
<thead>
<tr>
<th>Severity Category</th>
<th>Percentage of cases that experience severe outcomes</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;5%</td>
<td>listeriosis, STEC, hepatitis A, typhoid</td>
</tr>
<tr>
<td>2</td>
<td>0.5 – 5%</td>
<td>salmonellosis, shigellosis</td>
</tr>
<tr>
<td>3</td>
<td>&lt;0.5%</td>
<td>campylobacteriosis, yersiniosis, NLV, toxins</td>
</tr>
</tbody>
</table>

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

**Severity category 1:**

**Bacteria**

*Clostridium botulinum*

**Protozoa**

*Toxoplasma*

**Severity category 3:**

**Bacteria**

*Aeromonas/Plesiomonas*

*Arcobacter*

*E. coli* (pathogenic, other than STEC)

*Pseudomonas*

*Streptococcus*

*Vibrio parahaemolyticus*

**Viruses**

Others (e.g. rotavirus)

**Protozoa**

*Giardia*

*Cryptosporidium*

*Cyclospora*

Others (e.g. *Entamoeba*)
### Proposed Category Matrix

<table>
<thead>
<tr>
<th>Incidence</th>
<th>&gt;100</th>
<th>10-100</th>
<th>1-10</th>
<th>&lt;1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand