DRAFT MICROBIOLOGICAL RISK ASSESSMENT

LISTERIA MONOCYTOGENES

IN

COLD-SMOKED SALMON

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Acknowledgements

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MICROBIOLOGICAL RISK ASSESSMENT OF LISTERIA MONOCYTOGENES IN COLD-SMOKED SALMON

OBJECTIVE

The objective of this risk assessment is to identify and characterise the risk to public health in Australia and New Zealand arising from the contamination of cold-smoked salmon with *L. monocytogenes*. This risk assessment builds on the risk assessment ‘Finfish seafoods’ conducted during the Review of Microbiological Standards (ANZFA 1999) as part of the establishment of the joint Food Standards Code for Australia and New Zealand.

BACKGROUND

Regulations in Australia and New Zealand

Standard 1.6.1 – Microbiological limits for foods, of the joint Australia New Zealand Food Standards Code (ANZFSC), applies limits for *L. monocytogenes* in some foods. These limits apply to foods sampled at any point during their stated shelf life.

For processed ready-to-eat finfish, such as smoked salmon, a level of up to 100 colony forming units (cfu) is permitted in one of the five samples, with the other four samples being “not detected in 25g”. This decision arose due to the absence of a listericidal step during processing of smoked fish. Therefore, there is the possibility of occasional low level contamination occurring, as *L.monocytogenes* is frequently present in the processing environment. However, scientific opinion indicates that low numbers of *L. monocytogenes* are unlikely to cause illness in consumers other than possibly the most severely immunocompromised. However, as *L. monocytogenes* can grow in the fish during refrigerated storage, the application of the microbiological limits until the end of the products shelf life would mean that processors may need to impose on their processes a “not detected” process outcome. This would ensure that any growth of bacteria from levels below the detection during the shelf life of the product would not exceed 100/g.

Standard 1.6.1 of the ANZFSC will replace the existing microbiological criteria in late 2002. The existing criteria in Australia are contained in the Food Standards Code (FSC), which requires a “zero tolerance” (not detected in 25 g) for smoked fish products. This regulation applies only at the end of the production process or at the wholesale retail stage. There are no limits for product in the marketplace, thus low levels of *L.monocytogenes* at the wholesale stage (below detection level) could potentially increase to detectable during the shelf life of the product.
In New Zealand, while there are currently no mandatory limits for smoked salmon stipulated within the *Food Regulations 1984 (NZFR)*, there are the “General microbiological reference criteria for *L. monocytogenes*” (Ministry of Health 1995). These microbiological reference criteria require that all ready-to-eat foods (including smoked salmon), and those that undergo a listericidal step, meet a zero tolerance (absence in five samples of 25g). These criteria apply until the end of the stated shelf life of the food. These criteria are not part of New Zealand law, but are to be used where no standard exists in law for monitoring purposes, or as supplements to existing standards where public health concerns dictate.

The New Zealand (Mandatory) Food Standard 1997 does however prohibit the import or sale of smoked or smoke flavoured vacuum packed fish containing “pathogenic bacteria”. This constitutes a zero tolerance for the presence of *L. monocytogenes*.

In summary, the *FSC* currently imposes a zero tolerance for smoked salmon at the end of processing but no limits for product in the marketplace. However it should be noted there are guidelines for the recall of packaged ready to eat food contaminated with *L. monocytogenes*, for use by Australian State and Territories enforcement agencies that give a “zero tolerance” for foods in which *L. monocytogenes* can grow and a maximum of 100 colony forming units /gram (cfu/g) for those in which it cannot. This would mean that packaged smoked salmon in the marketplace would be expected to have zero *L. monocytogenes* since the bacteria can grow. In New Zealand, the Ministry of Health’s guidelines and the Food Standard 1997 set a zero tolerance for smoked salmon at all times. In the ANZFSC, which is expected to become mandatory in December 2002, the standard allows a level of 100 cfu/g in one of five samples of smoked salmon, with the other four samples to meet a requirement of absent in 25g, all samples taken from a batch throughout its shelf life.

**Published risk assessments on *Listeria monocytogenes* in cooked crustacea**

The Food and Agriculture Organization of the United Nations (FAO) and the World Health Organisation (WHO) convened an Expert Consultation on Risk Assessment of Microbiological Hazards in Foods in July 2000, with an aim of developing an international strategy and identified mechanisms required to support risk assessment of microbiological hazards in foods (FAO/WHO 2000). Although the risk assessment is still being finalised (FAO/WHO 2001) it has been drawn upon in the development of this risk assessment.

The United States Food and Drug Administration has recently completed draft risk assessments for a variety of selected ready-to-eat foods (US FDA 2001). The draft report concluded that smoked seafood had a high predicted relative risk of causing listeriosis to individual consumers on a per serving basis. The draft risk assessment prepared by the US FDA is a detailed summary and analysis of data available, and hence has been drawn upon heavily in the development of this risk assessment.
Methodology

A microbiological risk assessment is defined by the Codex Alimentarius (Codex Alimentarius Commission 1999) as:

“A key element [in microbiological risk analysis] in assuring that sound science is used to establish standards, guidelines and other recommendations for food safety to enhance consumer protection and facilitate international trade.”

Risk assessment is divided into four steps:

1. Hazard identification: the identification of biological, chemical and physical agents capable of causing adverse health effects and which may be present in a particular food or group of foods.
2. Hazard characterisation: the qualitative and/or quantitative evaluation of the nature of the adverse health effects associated with the hazard. For the purpose of a microbiological risk assessment, the concerns relate to microorganisms and/or their toxins.
3. Exposure assessment: the qualitative and/or quantitative evaluation of the likely intake of biological, chemical, and physical agents via food as well as exposures from other sources if relevant.
4. Risk characterisation: the process of determining the qualitative and/or quantitative estimation, including attendant uncertainties, of the probability of occurrence and severity of known or potential adverse health effects in a given population based on hazard identification, hazard characterisation and exposure assessment.

A microbiological risk assessment can be qualitative, quantitative or a combination of both (semi-quantitative). A qualitative risk assessment is used when insufficient information is available to estimate the risk numerically; a quantitative risk assessment provides numerical expressions of risk and an indication of the attendant uncertainties. This microbiological risk assessment is qualitative, drawing on local (Australian and New Zealand) data, where available.

RISK ASSESSMENT

General considerations

During the review of the microbiological criteria for foods, stakeholders identified the seafood groups processed finfish and cooked crustacea as being of particular concern with respect to the standards for the presence of L. monocytogenes.

In order to allow a focussed scientific assessment of the adequacy of the current microbiological criteria for foods in these categories, ANZFA sought to identify specific foods of relevance to Australian and New Zealand consumers, within the broader categories of cooked crustacea and processed finfish. This risk assessment document is one of two risk assessments addressing these concerns. This risk assessment focuses on processed ready-to-eat finfish, and in particular, the following commodities:
- Cold-smoked salmon
  - produced in Australia and New Zealand
  - imported into Australia and New Zealand (predominantly from Denmark)

An analysis of the amount of each commodity produced or imported, as well as the amount of each commodity consumed in Australia and New Zealand, determined which food commodities within the broader categories were of greatest significance. Data on the amount of production, importation and consumption of each commodity within the category ‘processed ready-to-eat finfish’ was gathered from a variety of sources (Walsh pers. comm. 2001; Bremer pers. comm. 2001; Steere pers. comm. 2001; ABARE 2001; AQIS pers. comm. 2001; Ministry of Health pers. comm. 2001; McLennan et al 1997; McLennan et al 1998\textsuperscript{a}; McLennan et al 1998\textsuperscript{b}; McLennan et al 1999; Russell et al 1999).

These commodities are thus the focus of this microbiological risk assessment.

1. **HAZARD IDENTIFICATION**

1.1 *Listeria monocytogenes*

There are seven species of Listeria, of which only one, *L. monocytogenes*, is consistently associated with foodborne illness (ICMSF 1996). *L. monocytogenes* has four serogroups [1/2, 2, 4 and 7] (Bannerman 1995) and serovars 4b, 1/2a and 1/2b account for most cases of human listeriosis (ICMSF 1996).

Nyfeldt first reported human listeriosis in 1929 (Kampelmacher 1989). In 1953, Potel linked foodborne listeriosis with animals; isolating *L. monocytogenes* from a cow with *Listeria* mastitis and stillborn twins from a woman who had ingested untreated milk from the infected animal (McCarthy 1990). Since then, and particularly over the last twenty years, *L. monocytogenes* has been recognised as a significant cause of foodborne illness (Sutherland et al 1997).

1.2 Incidence of Listeriosis

1.2.1 Sporadic cases and outbreaks

Listeriosis occurs both as isolated sporadic cases and as outbreaks. Since 1980, a number of large food-associated outbreaks have been documented. However, the majority of foodborne disease caused by *L. monocytogenes* occurs sporadically, and is not linked to a specific food or another case of listeriosis (Sutherland et al 1997). Nevertheless, most infections are believed to be foodborne (Farber et al 1991; Schuchat et al 1992).

1.2.2 International

The estimated incidence of listeriosis in European countries is four to eight cases per million of the general population per year. In France, the estimated incidence of listeriosis is sixteen cases per million general population per year (Bille 1990). The United States estimates that approximately 8.8 people per million general population become seriously ill with listeriosis each year, with a fatality rate of 20%. Of all the foodborne pathogens, *L. monocytogenes* resulted in the highest hospitalisation rate in the United States (US FDA 2001).
While the incidence rate is low compared to other foodborne illnesses, such as *Salmonella*, the mortality rate is much higher, ranging between 5 and 33%, and averaging 22% (Rocourt *et al* 1992). In general, the incidence of listeriosis appears to be decreasing in most countries. Transitory increases in cases appear to be linked to specific outbreaks (Codex 2001).

### 1.2.3 Australia and New Zealand

The number of reported cases of invasive listeriosis in Australia from 1991 to 2000 is approximately fifty-six cases per year (Communicable Diseases Network Australia 2001), which equates to an estimated incidence of invasive listeriosis in Australia of three cases per million of the general population per year (Sutherland *et al* 1997). In Australia, the exact mortality rate is not known, although the data available would suggest a rate of approximately 23% (see Appendix 1).

The annual average number of reported cases of invasive listeriosis in New Zealand since 1990 is seventeen. The estimated incidence of invasive listeriosis in New Zealand is five cases per million of the general population per year (Anon 1996-2001). The fatality rate in New Zealand since 1995 is approximately 17%.

An overview of listeriosis in Australia and in New Zealand can be found in Appendices 1 and 2 respectively. Australia and New Zealand rates of listeriosis are comparable to international rates of listeriosis.

### 1.2.4 Epidemiological links between seafood and listeriosis

Outbreaks of listeriosis are relatively uncommon compared to many foodborne infections. Some of the outbreaks that have been investigated have been linked to the consumption of seafood. Outbreaks recorded outside of Australia and New Zealand include:

- two cases in healthy adults, with a potential involvement of imitation crabmeat (artificially flavoured Alaska pollock) (Farber *et al* 2000);
- nine cases from cold-smoked rainbow trout, resulting in two deaths (Ericsson *et al* 1997); and
- nine cases of mild listerial gastroenteritis in pregnant women, with investigators suggesting that cooked shrimp was the most likely vehicle of infection (Riedo *et al* 1994).

Cases linked to seafood locally include:

- one case (stillborn twins) from smoked mussels produced and consumed in New Zealand (Brett *et al* 1998);
- contaminated shellfish or raw fish in New Zealand was loosely linked to 22 cases, including five deaths; the link was on the basis of recall of food consumption, not microbiological testing (Lennon *et al* 1984);
- reference is made in a publication to a possible link between smoked salmon and two miscarriages in 1993 in Australia (Arnold *et al* 1995), however it is noted that the food consumed was not available for testing;
• an identical strain (PGFE typing) of *L. monocytogenes* was isolated from a case and from
smoked salmon (Tan *et al* 1995);
• four cases of listerial gastroenteritis from smoked mussels processed in New Zealand, re-
labelled with incorrect use-by dates in Tasmania, and consumed in Tasmania (Mitchell 1991;

The US FDA risk assessment (2001) looked at all documented outbreaks internationally,
including those listed above, and ranked fish products third behind meat and dairy products in
terms of responsibility for outbreaks for which the food linkage has been identified.

1.3  **Association of *Listeria monocytogenes* with seafood**

Knowledge of the frequency of contamination and the level of *L. monocytogenes* in the selected
foods categories, in the raw commodity, during processing, and in the retail sector, is essential
for undertaking a comprehensive and accurate assessment of the risks posed by *L.
monocytogenes*. In particular, this information is needed for seafood available to the Australian
and New Zealand consumers so that the relevant risk can be assessed. In the absence of
appropriate data, it may be necessary to make some judgement based on a theoretical risk.

1.3.1  **Recalls of seafood due to the presence of *Listeria monocytogenes* (Australia and New
Zealand)**

Analysis of Australian recall data\(^1\) shows that there were fifty-six recalls due to the presence of *L.
monocytogenes* between 1990 and May 2001 and that nineteen of these (34%) were due to
contamination of seafood products (see Fig 1). The majority of recalls were in the category
‘smoked seafood’. This category includes smoked salmon, as well as other types of smoked
seafood.

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\(^1\) A recall is defined as action taken to remove from sale, distribution and consumption foods which may pose a safety
hazard to consumers (ANZFA 2001). Recalls can occur at wholesale, retail or consumer level.
The levels of contamination of the recalled products were only reported for two cold-smoked salmon samples. One sample yielded 4 cfu/100g, and the other sample yielded <0.03/g (most probable number [MPN]).

No recalls have occurred in New Zealand for the presence of *L. monocytogenes* in seafood.

### 1.3.2 Seafood imports associated with *L. monocytogenes* (Australia and New Zealand)

In both countries, imported foods are required to comply with the same regulations as food produced domestically (eg. *Food Standards Code* or *Food Regulations 1984*). Sampling and testing foods imported into Australia is the responsibility of the Australian Quarantine and Inspection Service (AQIS). In New Zealand, this is the responsibility of the Ministry of Health.

In Australia, smoked salmon is considered a high risk product, which means that 100% of smoked salmon is referred to AQIS for inspection. However, a performance based system is applied to ‘risk listed’ foods such as smoked salmon, and thus there are three different inspection rates:

1. Initially, the first five shipments of a particular food first arriving from a particular producer are inspected (100%), and after five consecutively cleared shipments, inspection intensity drops to the next level.
2. One in four shipments are then inspected, with the other three being automatically released (25%); after twenty cleared inspections, inspection intensity drops to the next level.
3. One in twenty shipments are then inspected (5%).

Results from tests on smoked salmon imported into Australia from 1995 to 1999 show that 10.6% of samples tested for *L. monocytogenes* failed (presence in 25g) (see Table 1).

<table>
<thead>
<tr>
<th>Year</th>
<th>Total tested</th>
<th>Total failed</th>
<th>% Total failed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995</td>
<td>30</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1996</td>
<td>37</td>
<td>2</td>
<td>5.4</td>
</tr>
<tr>
<td>1997</td>
<td>75</td>
<td>8</td>
<td>10.6</td>
</tr>
<tr>
<td>1998</td>
<td>92</td>
<td>18</td>
<td>19.5</td>
</tr>
<tr>
<td>1999</td>
<td>85</td>
<td>6</td>
<td>7.0</td>
</tr>
<tr>
<td>Total</td>
<td>319</td>
<td>34</td>
<td>10.6</td>
</tr>
</tbody>
</table>

(Sourced from data from AQIS – Imported Food Program)

The results from New Zealand from July 1994 to June 2001 show that all seventy samples of smoked fish (including smoked salmon) imported into New Zealand that have been tested were found to contain *L. monocytogenes* (ESR pers. comm. 2001). It is not known what proportion of these samples were ready-to-eat smoked salmon.

### 1.3.5 Incidence of *Listeria* spp. in cold-smoked salmon – published

There have been a number of published studies on the occurrence of *L. monocytogenes* in smoked fish, including cold-smoked salmon (see Table 2). These studies generally show that contamination at the retail level is not uncommon, with up to 40% of samples containing *Listeria* in several surveys, although frequencies between 10 and 20% are most common. A low rate of contamination was reported in 1995 for Australian cold smoked salmon (Garland 1995). Fletcher however reported that a small survey of 10/13 cold smoked salmon samples in New Zealand contained *Listeria*, although all positive samples were tested at the end of their shelf life (Fletcher *et al* 1994). In a study of processing facilities where *Listeria* management was an issue the contamination rate was high at 78% (Eklund *et al* 1995).

There are three reports that have enumerated the number of *L. monocytogenes* in smoked fish. One study from Denmark showed contamination of between 8 and 100 MPN/g in cold-smoked salmon at the end of its stated shelf life (Dalgaard *et al* 1998). A study in the USA of cold-smoked salmon manufacturers with *Listeria* contamination showed levels of *L. monocytogenes* at 0.3 – 34.3/g (Eklund *et al* 1995). Another Danish study looked at the levels of *L. monocytogenes* in cold-smoked salmon stored at 5±1°C for a period of 14-20 days and 21-50 days (Jørgensen *et al* 1998). This study shows that high levels of *L. monocytogenes* can survive in cold-smoked salmon during its shelf life (see Table 3).
<table>
<thead>
<tr>
<th>Sample</th>
<th>Origin of sample</th>
<th>Point of sampling</th>
<th>No. of samples</th>
<th>Samples positive for <em>L. monocytogenes</em></th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoked salmon</td>
<td>Australia</td>
<td>Retail</td>
<td>59</td>
<td>11 [18%]</td>
<td>Arnold <em>et al</em> 1995</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Italy</td>
<td>Wholesale</td>
<td>165</td>
<td>31 [18%]</td>
<td>Cortesi <em>et al</em> 1997</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Canada</td>
<td>Retail and manufacturers</td>
<td>12</td>
<td>0 [0%]</td>
<td>Dillon <em>et al</em> 1992</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>USA</td>
<td>Manufacturers with ‘Listeria problems’</td>
<td>61</td>
<td>48 [78%]</td>
<td>Eklund <em>et al</em> 1995</td>
</tr>
<tr>
<td>Smoked fin fish</td>
<td>Not stated</td>
<td>Not stated</td>
<td>1210</td>
<td>164 [13%]</td>
<td>Elliott <em>et al</em> 2000</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Imported and domestic (Canada)</td>
<td>Retail</td>
<td>32</td>
<td>10 [31%]</td>
<td>Farber 1991</td>
</tr>
<tr>
<td>Packaged cold-smoked salmon</td>
<td>New Zealand</td>
<td>Retail (all positive samples were past the “use-by” or “best before” date)</td>
<td>13</td>
<td>10 [76%]</td>
<td>Fletcher <em>et al</em> 1994</td>
</tr>
<tr>
<td>Cold-smoked fish</td>
<td>UK</td>
<td>Retail and manufacturer</td>
<td>58</td>
<td>2 [3%]</td>
<td>Fuchs <em>et al</em> 1994</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Australia</td>
<td>Wholesale</td>
<td>285</td>
<td>1 [&lt;1%]</td>
<td>Garland 1995</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Iceland</td>
<td>Retail and manufacturer</td>
<td>8</td>
<td>0 [0%]</td>
<td>Hartemink <em>et al</em> 1991</td>
</tr>
<tr>
<td>Cold-smoked fish</td>
<td>Domestic and imported (USA)</td>
<td>Retail and manufacturer</td>
<td>291</td>
<td>51 [17%]</td>
<td>Heinitz <em>et al</em> 1998</td>
</tr>
<tr>
<td>Finfish</td>
<td>New Zealand</td>
<td>Retail</td>
<td>25</td>
<td>8 [32%]</td>
<td>Hudson <em>et al</em> 1992</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Imported</td>
<td>Retail</td>
<td>388</td>
<td>39 [10%]</td>
<td>Jemmi 1993</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Denmark</td>
<td>Retail</td>
<td>380</td>
<td>142 [37%]</td>
<td>Jørgensen <em>et al</em> 1998</td>
</tr>
<tr>
<td>Packaged gravad or smoked salmon / rainbow trout</td>
<td>Sweden</td>
<td>ns</td>
<td>344</td>
<td>49 [14%]</td>
<td>Lindqvist et al 2000</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Cold-smoked salmon</td>
<td>Sweden</td>
<td>Retail</td>
<td>13</td>
<td>2 [15%]</td>
<td>Loncarevic et al 1996</td>
</tr>
<tr>
<td>Vacuum-packed cold-smoked salmon</td>
<td>Norway</td>
<td>Manufacturer</td>
<td>65</td>
<td>7 [10%]</td>
<td>Rørvik et al 1995</td>
</tr>
<tr>
<td>Vacuum-packed ready-to-eat smoked salmon</td>
<td>Norway</td>
<td>Retail</td>
<td>33</td>
<td>3 [9%]</td>
<td>Rørvik et al 1997</td>
</tr>
<tr>
<td>Vacuum-packaged sliced cold-smoked salmon</td>
<td>Denmark</td>
<td>Manufacturer</td>
<td>1028</td>
<td>110 [10%]</td>
<td>Rørvik et al 1997</td>
</tr>
</tbody>
</table>

**Table 3 – Levels of L. monocytogenes in cold-smoked salmon stored at 5±1°C**

<table>
<thead>
<tr>
<th>Storage time (days)</th>
<th>Number of samples</th>
<th>Number of positive samples</th>
<th>Number of positive samples</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;10/g*</td>
<td>10-100/g</td>
</tr>
<tr>
<td>14-20</td>
<td>115</td>
<td>11</td>
<td>23</td>
</tr>
<tr>
<td>21-50</td>
<td>75</td>
<td>17</td>
<td>11</td>
</tr>
</tbody>
</table>

*MPN/g (Jørgensen et al 1998)

### 1.3.6 Incidence of Listeria spp. in cold-smoked salmon in Australia and New Zealand – unpublished

A targeted call for data provided ANZFA with very limited information on the incidence of *L. monocytogenes* in cold-smoked salmon sold or produced in Australia and New Zealand. Data was provided for thirty-eight samples (see Table 4). From this data it is evident that some smoked salmon in the marketplace may not comply with an “absent in 25g” limit.
Table 4 - Incidence of *L. monocytogenes* in cold-smoked salmon in Australia and New Zealand

<table>
<thead>
<tr>
<th>Description</th>
<th>Point of sample</th>
<th>Source</th>
<th>Reason for sampling</th>
<th>Number of samples</th>
<th>Number positive for <em>L. monocytogenes</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoked salmon</td>
<td>Retail</td>
<td>Tasmania</td>
<td>Regulatory surveillance</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Retail</td>
<td>Norway</td>
<td>Regulatory surveillance</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Salmon</td>
<td>Border</td>
<td>Imported [source unknown]</td>
<td>Regulatory surveillance</td>
<td>5</td>
<td>1 sample contained 3 MPN/g</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Retail</td>
<td>Source unknown</td>
<td>Regulatory surveillance</td>
<td>3</td>
<td>2 (enumeration not performed)</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Unknown</td>
<td>Tasmanian</td>
<td>Regulatory surveillance</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Unknown</td>
<td>New Zealand</td>
<td>Regulatory surveillance</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Vacuum-packaged smoked salmon</td>
<td>Unknown</td>
<td>South Australia</td>
<td>Regulatory surveillance</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>Unknown</td>
<td>Unknown</td>
<td>Regulatory surveillance</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

1.3.7 Summary of incidence data

Smoked fish including cold-smoked salmon is frequently contaminated with *L. monocytogenes*. This is evident for both imported and locally produced product. There is however limited data as to the counts present in this product.

2. HAZARD CHARACTERISATION

2.1 Listeriosis

2.1.1 The Disease and Population Susceptibility

There are two main clinical forms of infection with *L. monocytogenes* – listerial gastroenteritis, where usually only mild, flu-like symptoms are reported, and the classic invasive listeriosis, where the bacteria penetrate the gastrointestinal tract and invade normally sterile sites within the body. The latter form can be very severe, and in some cases, life-threatening. Invasive listeriosis is an opportunistic infection and a relatively rare disease, with a wide range of symptoms including meningoencephalitis and septicaemia. Mild or asymptomatic infections in pregnant women may lead to infection of the foetus (Sutherland *et al* 1997).
The incubation period prior to individuals becoming symptomatic with listeriosis can be long (up to 3 months) but most commonly in the region of several days, and for the gastrointestinal form less than 24 hours (Sutherland et al 1997).

It is estimated that about 2 to 6 percent of the healthy population harbours *L. monocytogenes* in their intestinal tract, which suggests that people are frequently exposed to *L. monocytogenes* (Rocourt et al 1997; Farber et al 1991). This may also suggest that most people have tolerance to infection by *L. monocytogenes*, and given the relatively low number of reported cases, exposure rarely leads to serious illness (US FDA 2001; Marth 1988; Hitchins 1996).

However, a number of risk groups for listeriosis have been identified, including pregnant women and their foetuses, neonates, elderly, and the immuno-compromised (e.g. HIV/AIDS patients and renal transplant patients). Healthy individuals may become infected, but only very rarely (Sutherland et al 1997). There is thus a relatively identifiable subsection of the population who are highly susceptible to infection.

This risk assessment is concerned primarily with risk associated with invasive listeriosis and takes into consideration the existence of sub-groups within the population with regards to susceptibility to infection.

### 2.1.2 Infective dose and risk quantification

Epidemiological evidence from investigations where the vehicle of infection has been identified indicates that less than 100 cfu/g of *L. monocytogenes* is unlikely to cause illness in the general population (Dalton et al 1997; Junttila et al 1989; Misrachi et al 1991; Miettinen et al 1999; Aureli et al 2000; Berrang et al 1988; Ericsson et al 1997; Goulet et al 1998). There is one study that suggests that the level of *L. monocytogenes* required to cause illness in susceptible groups may be lower (Maijala et al 2001). A number of countries, including Australia\(^\text{ii}\), accept the presence of counts of less than 100/g for some foods as being low risk. However, this will usually apply only to those foods in which the bacteria do not grow readily during normal storage conditions and/or that do not have a listericidal step in their preparation.

Factors affecting the likelihood of illness developing in an individual consumer may include their immune status, the type of food consumed, the virulence and infectivity of the pathogen, the concentration of the pathogen in the food, and the number of repetitive challenges (NACMF 1991). Thus, even when an outbreak occurs not all persons consuming the contaminated foods will develop an infection.

### 2.2 Growth conditions for *Listeria monocytogenes*

Growth of *L. monocytogenes* in foods will be influenced by a variety of factors, including the nature and concentration of essential nutrients, pH, temperature, water activity, the presence of food additives that could enhance or inhibit growth and of other microbial flora (Lovett et al 1990; Doyle 1988). The limits and optima for key factors are summarised in Table 5.

---

\(^\text{ii}\) Refer to Recall Guidelines for Packaged Ready-to-eat foods found to contain *Listeria monocytogenes* at point of sale, www.anzfa.gov.au/recallssafety/listeria/index.cfm
Table 5 – Growth conditions for *L. monocytogenes*

<table>
<thead>
<tr>
<th></th>
<th>Minimum</th>
<th>Optimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature (°C)</td>
<td>-1.5</td>
<td>37</td>
<td>45</td>
</tr>
<tr>
<td>pH</td>
<td>4.39</td>
<td>7.0</td>
<td>9.4</td>
</tr>
<tr>
<td>Water activity (aw)</td>
<td>0.90</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

(ANZFA unpublished)

Under conditions outside of the growth range, the bacteria may survive, so that growth will recommence once suitable conditions reoccur. There may be considerable interaction between the factors controlling growth rates, especially at the limits of the ranges for each factor. The conditions under which the bacteria were growing before being introduced into the food may also influence subsequent growth by causing an extended lag phase before significant growth commences (ICMSF 1996).

Traditional packaging methods do not affect the growth of *L. monocytogenes*, although newer methods may be effective in reducing the growth of *L. monocytogenes*. Thus, packaging may not influence the growth of *Listeria* but will provide essential protection against contamination during storage and transport.

*L. monocytogenes* can grow at salt concentrations as high as 13-14%, and survive in concentrations of 30% (Fuchs *et al* 1992; Farber *et al* 1992). Salt alone is thus unlikely to be a control for the growth of *L. monocytogenes* in processed seafood.

*L. monocytogenes* is able to survive for long periods in processing plants, household refrigerators and freezers (US FDA 1999; Salamina 1996).

In frozen food, the bacteria are preserved or moderately inactivated (Lou *et al* 1999). As temperatures are elevated above the minimum for growth, the growth rate will increase. The characteristics of the food substrate and any additional factors such as the presence of acids added during processing, will influence both the lag phase before growth occurs and generation times. Once growth has commenced after the lag phase, the bacteria will multiply exponentially until various factors slow down growth. Eventually the growth rate will equal the death rate and a maximum population will be achieved. The level reached appears to be temperature dependent and is higher at higher temperatures of refrigerated storage. Thus, a food stored at 5°C will have a lower final count of *L. monocytogenes* than the same food stored at 8°C (Duffes *et al* 1999).

*L. monocytogenes* is capable of survival and growth under a wide range of conditions, particularly where other pathogenic microorganisms are unlikely to grow. Therefore, risk management strategies for *L. monocytogenes* in foods will differ from risk management strategies for other pathogenic microorganisms.

2.3 Growth potential for *Listeria monocytogenes* in cold-smoked salmon

Cold-smoked salmon has an approximate pH of 6, and a water activity range of 0.983 – 0.964. The salt content of cold-smoked salmon is 3-6% (Ross *et al* 2000). These conditions support the growth of *L. monocytogenes*. The process used to cold-smoke salmon is not listericidal.
Therefore, storage temperatures and shelf life for cold-smoked can have a significant impact on bacterial numbers if contamination occurs.

There have been several studies on the growth rate of *L. monocytogenes* in cold-smoked salmon. In addition, studies have been conducted on other smoked finfish. These studies provide an average growth rate in smoked fish of 0.155 logs/day at 5°C. This is a moderate growth rate compared to most other ready to eat foods (US FDA 2001).

### 2.4 Sources and routes of contamination of seafood with *Listeria*

*L. monocytogenes* is widely present in the environment, including soil, sewage, plant matter, animal feed, dust and water (Sutherland *et al* 1997; Fenlon 1999). *Listeria* species have been isolated from polluted waters and rivers and coastal areas with a high content of organic material. However, its presence in clean “open” water has not been established (Ben Embarek 1994). Higher ambient water temperatures (>20°C) may inhibit the growth of *Listeria* or mask its presence (Ben Embarek 1994; Motes 1991).

Salmon intended for smoking may be harvested from the seas, rivers or ponds and lakes. Aquaculture may take place in open or closed bodies of water. Fish produced using aquaculture conditions could potentially be more vulnerable to contamination than free-range catches, however it is considered that contamination prior to harvest is not a primary concern (FAO 1999).

The steps in the processing of cold-smoked salmon are shown in Flow Chart 1 in Appendix 4. None of the processing steps is listericidal, although some processors may introduce treatments that can reduce contamination levels. Many of the processing steps offer opportunities for the introduction of *L. monocytogenes* and for its subsequent growth. Attempting to quantify the impact of these factors on possible counts on the final product is very complicated. Some steps will be of greater significance than others in determining the final counts. Studies tracking *L. monocytogenes* during processing such as that by Rørvik *et al* (1995) reproduced in Table 6 illustrate the variability of contamination and the multiple sources that may exist.

#### Table 6 – Incidence of *L. monocytogenes* during cold-smoked salmon processing in Norway

<table>
<thead>
<tr>
<th>Sample</th>
<th>Origin of sample</th>
<th>Point of sampling</th>
<th>Number of samples</th>
<th>Samples positive for <em>L. monocytogenes</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Vacuum-packed cold-smoked salmon</td>
<td>Norway</td>
<td>Post-slaughter</td>
<td>50</td>
<td>0 [0%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Before filleting</td>
<td>24</td>
<td>4 [16%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After filleting</td>
<td>5</td>
<td>2 [40%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After salting</td>
<td>13</td>
<td>4 [30%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After smoking</td>
<td>8</td>
<td>0 [0%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After trimming</td>
<td>13</td>
<td>5 [38%]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After slicing</td>
<td>8</td>
<td>1 [12%]</td>
</tr>
</tbody>
</table>
The main sources of contamination are thus from post-harvest contamination. The level of contamination post-harvesting is variable.

3. EXPOSURE ASSESSMENT

3.1 Post-cooking conditions

*L. monocytogenes* can grow during refrigerated storage of food (Harrison et al 1991). A higher storage temperature will facilitate increased growth and may influence the maximum concentration of bacteria reached (see section 2.2). The numbers of *L. monocytogenes* present at the point of consumption will depend on when the contamination occurred and at what level and the time and temperature of subsequent storage.

3.1.1 Storage time

In New Zealand, an average shelf-life stated by manufacturers for smoked salmon is usually six weeks from packaging. In Australia, the stated shelf-life varies between four to six weeks depending on the processor.

While a longer shelf life may provide more time for *Listeria* if present to grow, as there is a maximum level for bacterial numbers, if this is reached early in the storage of the smoked salmon, the length of subsequent storage may have limited effects on contamination levels.

Generally, imported smoked salmon is frozen, thereby slowing the growth rate of *L. monocytogenes*, if present.

3.1.2 Storage temperatures

The majority of smoked salmon purchased at the retail level is chilled, but not frozen. Temperatures during chilled storage are controlled by various regulations. For example, there are requirements for storing chilled seafood specified in Standard 3.2.2 – *Food Safety Practices and General Requirements*, within the ANZFSC. This Standard requires chilled ‘potentially hazardous food’ to be stored at 5°C or below. However, the Standard allows food businesses to store chilled ‘potentially hazardous food’ at higher temperatures if the business can demonstrate that this would not affect the safety of the food. ‘Potentially hazardous food’ is food that has to be kept at certain temperatures to minimise the growth of any pathogenic microorganisms that may be present in the food or to prevent the formation of toxins in the food. Seafood, with the exception of live seafood, would normally be considered ‘potentially hazardous’.

As the growth and levels of *L. monocytogenes* are influenced by temperatures during storage, the better the cold chain is maintained the less growth will occur. Temperature control of food stored in domestic refrigerators in Australia is generally poor. In a recent survey, 36% of Australian domestic refrigerators (n=171) had their fresh-food compartments above 5°C for greater than 50% of the time (Jay *et al* 1998). Comparable information is not available for New Zealand.
3.1.3 Packaging

Smoked salmon sold at the retail level is usually vacuum packaged. Smoked salmon used in catering is often sold in bulk, vacuum packaged lots.

Packaging methods such as controlled atmospheric packaging, modified atmosphere packaging and vacuum packing, do not have an effect on the ability of *L. monocytogenes* to grow (Grau *et al* 1992; Dillon *et al* 1994) although more recent research suggests that some reductions in growth rates can be achieved by manipulating the packaging environment.

3.2 Consumption data for seafood

Consumption data for the Australian and New Zealand populations has been obtained from the 1995 Australian National Nutrition Survey and the 1997 New Zealand National Nutrition Survey (McLennan *et al* 1997; McLennan *et al* 1998a; McLennan *et al* 1998b; McLennan *et al* 1999; Russell *et al* 1999). Consumption data for children (2-15 years) is only available for Australia. However, as children are not within the susceptible population for listeriosis, and their consumption rate of seafood is low, the Australian data has not been presented.

The data shows that about 35% of survey respondents between the ages of 12-64 years consume seafood at least once a month. Consumers over 65 had a lower rate of about 20%. Few consumers recorded eating seafood more than once a week. A proportion of those reporting consumption of seafood, consumed smoked salmon. As some consumers are considered to be more at risk than others from infection with *L. monocytogenes*, the data for sub-groups of women of child bearing age and men and women over 65 years has been considered separately.

3.2.1 Consumption of smoked salmon in Australia and New Zealand

The survey data indicates that for Australia and New Zealand no more than 1% of the respondents in each of the 3 groups consumed smoked salmon. The consumption of smoked salmon and the amounts consumed are shown in Table 7.

3.2.2 Estimated annual number of servings of smoked salmon in Australia and New Zealand

In order to estimate the risk that contaminated food represents to the population the annual number of servings of a food needs to be determined.

To estimate the annual number of servings of smoked salmon, the following formula has been used:

\[
\text{survey consumers of smoked salmon} \times \text{total population} \times 365
\]

\[
\text{number of survey respondents}
\]

For the Australian population, the estimated annual number of servings of smoked salmon is \(1.77 \times 10^7\).
For New Zealand, the estimated annual number of servings of smoked salmon is $3.70 \times 10^6$.

Table 7: Consumption of smoked salmon by Australian and New Zealand population groups

<table>
<thead>
<tr>
<th>Consumer group</th>
<th>Australia</th>
<th>New Zealand</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percentage of consumers (number of respondents)</td>
<td>Average amount consumed (g/day)</td>
</tr>
<tr>
<td>Smoked salmon</td>
<td>15-64 years</td>
<td>0.3%(9471)</td>
</tr>
<tr>
<td>65+ years</td>
<td>0.2%(1960)</td>
<td>21.5</td>
</tr>
<tr>
<td>Female 16-44 years</td>
<td>0.3%(3178)</td>
<td>39.7</td>
</tr>
</tbody>
</table>

Consumption of smoked salmon is low and infrequent. Children do not consume seafood regularly, or in large quantities. Women of childbearing age (identified as a risk group) have an overall low consumption of smoked salmon, however some consumers in this risk group consume large amounts of seafood. The elderly are generally low and infrequent consumers of smoked salmon.

3.2.4 Limitations of consumption data

The surveys are a retrospective compilation of food intake over the past 24 hours. The Australian National Nutrition Survey interviewed 13800 people, while the New Zealand National Nutrition Survey interviewed 4636 people. The limitations of a 24-hour food recall are (Gibson 1990):

- respondent biases: over- and underestimation of amount and types of food consumed;
- interviewer biases (may occur with different interviewing styles);
- respondent memory lapses; and
- coding and computation errors.

The average amount consumed per day gives some guidance as to amounts consumed but may be influenced significantly by consumers at the extremes consuming very small or large amounts.
4. RISK CHARACTERISATION

Epidemiological evidence, both international and local, confirms that listeriosis is a rare foodborne illness. However, the consequences of an infection may be severe. Cold smoked finfish has been implicated in one outbreak of listeriosis outside of Australia and New Zealand and may have been associated with cases in Australia. The evidence for the latter is circumstantial only. It must however be kept in mind that the food sources of many outbreaks and most sporadic cases is not known, although it is agreed that most if not all cases originate from the consumption of contaminated food. Thus the absence of more cases linked to the consumption of smoked salmon does not preclude the possibility that they have occurred.

Consumers of seafood contaminated with *L. monocytogenes* will become infected and develop illness if a serving of seafood contains sufficient bacteria to cause illness. For an individual consumer the risk of becoming ill from contaminated food will depend on a number of factors such as the amount of food consumed, the level of contamination and the person’s susceptibility. Other factors such as the virulence of the specific strain ingested, the physiological state of the bacteria, gastric acidity levels and so on may also contribute to whether or not an infection results and to the severity of the infection.

This means that not all consumers of a contaminated food will necessarily become ill even when the food is heavily contaminated i.e. the attack rate is usually <100% of people consuming a suspect food. The effect of this interaction of factors is that there is a relationship between the dose consumed and the frequency of infection. This allows certain predictions to be made with regards to the risks of contracting listeriosis from a contaminated meal.

The risk to consumers from contaminated cold-smoked salmon is evident from the data available on the frequency with which *L. monocytogenes* is found in smoked salmon. The absence of a listericidal step during processing and the frequency and ease with which fish processing plants can become contaminated with this bacteria indicate the need to manage the hazard in all production facilities.

Consumption data from Australia and New Zealand suggests that frequency of seafood consumption is low and only a small percentage of this consumption is of smoked salmon. However, consumption of highly contaminated salmon could be expected to be hazardous for vulnerable subgroups.

5. CONCLUSION

The data suggests that there is potential for smoked salmon to be contaminated with *L. monocytogenes*, and that growth of *L. monocytogenes* in smoked salmon is moderate. The shelf life of smoked salmon is four to six weeks, and *L. monocytogenes* could potentially grow to levels that may pose a risk to public health and safety. Smoked salmon is a high risk food, particularly for vulnerable subgroups.
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The Epidemiology of Listeriosis in Australia

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Summary
Listeriosis is a severe bacterial disease caused by consumption of contaminated food. Case fatality rates may be as high as 30%, and may cause death in neonates or foetuses. There are little published data on listeriosis in Australia. To review the epidemiology of listeriosis, we collected data from State and Territory health departments for the years 1998–2000. We reviewed data on Australian clusters of listeriosis for the years 1995–2000, and Victorian data on materno-foetal infections between 1991–2001. Australian health departments supplied data on 184 patients with listeriosis. We identified that 43 (23%) of these notifications related to maternal-foetal pairs, arising from 37 distinct maternal-foetal infections. The mean rate of listeriosis in Australia for the three years was 3.0 cases per million persons, and the mean rate for non-pregnancy related infections was 2.4 cases per million persons. There was a mean of 4.6 materno-foetal infections per 100,000 births each year during the three-year period.

National case numbers did not change markedly from year-to-year, or from jurisdiction-to-jurisdiction. Health Departments investigated five clusters of listeriosis during the six years between 1995–2000, three of which occurred in healthcare settings. Investigators identified that two of these outbreaks related to cooked chicken products, one to sandwiches and meat salads, and another to fruit salad. Despite few outbreaks occurring, there is a need to keep a vigilant watch for epidemics that cross jurisdictional borders. In Victoria, there has been a decreasing trend in the incidence of listeriosis, in both pregnant women, and non-pregnancy associated infections. We identified problems with the routine data collections from jurisdictions, including: missing data, and variation in recording practices. To improve surveillance and control of listeriosis we recommend: rapidly sharing typing and epidemiological information at the national level, standardising reporting practices for jurisdictions, collecting enhanced data, and conducting a national case control study for risk factors.

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**Purpose of this paper**

In this paper we review the epidemiology of listeriosis in Australia and make recommendations about national surveillance for this disease. While *Listeria* may cause mild gastrointestinal illness, this review focuses only on invasive listeriosis. Data in this report relate to the entire Australian population. This paper is intended to inform the Australia New Zealand Food Authority risk assessment of listeriosis associated with seafood.

**Introduction**

*Listeria monocytogenes* has been recognized as a human pathogen since 1929.\(^1\) The organism is ubiquitous in the environment, and is found in soil, water, and decaying vegetation.\(^2\,^3\,^4\) *Listeria* sp. is an intracellular pathogen and causes serious disease in humans and animals.

During the last 15 years, investigators have shown that epidemic listeriosis is a foodborne disease.\(^5\,^6\,^7\,^8\,^9\,^10\,^11\,^12\,^13\,^14\) Similarly, recent studies have suggested sporadic cases of listeriosis are predominantly due to contaminated foods.\(^15\,^16\,^17\) Improvements in laboratory techniques for detecting and subtyping *L. monocytogenes* have also contributed to an improved understanding of human listeriosis by rapidly identifying common sources of infection, clarifying factors important for pathogenesis, and mechanisms of growth in the environment.\(^18\,^19\,^20\,^21\,^22\)

Human disease caused by *L. monocytogenes* usually occurs in certain well-defined high-risk groups, including pregnant women, neonates, and immunocompromised adults. Infections in pregnant women are usually mild and affect the foetus or neonate. Neonatal infections have a typically high mortality rate, particularly infections early in the course of pregnancy.\(^23\) In Australia, an ageing population and the widespread use of immunosuppressive medications for treatment of malignancy and management of organ transplantation has led to an expansion of the immunocompromised population at increased risk for listeriosis. Listeriosis may also occasionally occur in persons who have no predisposing underlying condition.

Unlike infection with other common foodborne pathogens such as *Salmonella*, which rarely result in fatalities, listeriosis is associated with a mortality rate of approximately 20–40%.\(^24\,^25\) Health agencies and food regulators consider *L. monocytogenes* as one of the most serious foodborne diseases, due to the high case-fatality rate and the propensity for the pathogen to cause serious epidemics associated with contaminated food. Reports of multi-drug resistant *L. monocytogenes* infections highlight the importance of learning more about this disease and developing effective prevention efforts.\(^26\)
Case-control studies and outbreak investigations have implicated ready-to-eat foods, soft cheeses, paté, deli meats, coleslaw, fruit salad, shellfish, and dairy products as risk foods for listeriosis. In 1992, the US Food and Drug Administration (FDA) adopted a “zero tolerance policy” for *L. monocytogenes*, as a result of finding the same strain in a person with listeriosis, in turkey franks consumed by the case, and the plant where the franks were processed. Following this the food industry undertook a massive clean-up effort. In subsequent years, the incidence of listeriosis in the U.S. dropped from a high of 7 cases per 1,000,000 population in 1989 to 4 cases per 1,000,000 population in 1993. Despite these changes in 1998, the United States experienced one of its largest outbreaks of listeriosis affecting 101 persons in 22 states (Pers. comm. Paul Mead, Centers for Disease Control and Prevention, March 2002). The outbreak was linked to nationally distributed hot dogs and deli meats.

**Methods**

**Study Population**

Listeriosis is currently reported from all Australian states with a combined population approximately 19 million. There are approximately 60-70 invasive infections per year, nationally (National Notifiable Diseases Surveillance System, 2000).

**Case definition**

All jurisdictions used the National Health and Medical Research Council (1994) case definition for listeriosis, except for New South Wales. The NHMRC case definition for listeriosis is:

“Isolation of *Listeria monocytogenes* from a site which is normally sterile, including fetal gastrointestinal contents.”

The New South Wales case definition includes non-invasive listeriosis, and is:

“a) A person with clinical listeriosis in whom *Listeria monocytogenes* is isolated from a normally sterile site (eg, blood, spinal fluid, joint, pleura or pericardial fluid), OR
b) A person with clinical food borne listeriosis in whose faeces *Listeria monocytogenes* is isolated.”

**Study Period**

We collected de-identified information from all States and Territories on cases of listeriosis that were notified under public health legislation with onset dates between the years 1998–2000. We asked health agencies and public health laboratories about disease clusters that had occurred during the years 1995–2000. We also collected data from Victorian Health Department reports from 1994–2001 to observe trends in materno-foetal infections (Unpublished data).
Data Elements

We asked States and Territories to provide details about demographic characteristics, clinical features, and risk factors for each case.

States and Territories provided text-based descriptions on risk factors for cases. We recoded information on risk factors for listeriosis using conditions or treatments that may impair immune status, or pregnancy status.

For analysis, we divided the dataset into pregnancy-associated and non-pregnancy associated cases. In many instances, health departments noted whether a female was pregnant or the case was an infant. Where this was not obvious, we coded any case with an age of zero years, as a neonatal infection.

Because materno-foetal pairs were not always linked in the notified data, we developed a system for linking cases. We linked an infant case to a female case of childbearing age from the same State, where the dates of onset of symptoms or dates of notification to the health department were less than one week apart.

For the purpose of analysing pregnancy-associated cases, we considered a materno-foetal pair as a single case. For calculation of rates of pregnancy-associated infections we used data on live births and foetal deaths from the National Perinatal Statistics Unit (NPSU) for 1999.33

For calculation of rates, we used mid-point population estimates for 1998, 1999, and 2000 (Australian Bureau of Statistics).

We analysed the dataset using Epi Info version 6.04d (Centers for Disease Control and Prevention).

Results

Incidence in Australia

State and Territories provided data about 184 patients with listeriosis that became ill in the years 1998–2000. We identified that 43 (23%) of these notifications related to mothers or infants, arising from 37 distinct maternal-foetal infections. The mean rate of listeriosis in Australia for the three years was 3.0 cases per million persons, and national case numbers did not change markedly from year-to-year (Table 1). Case numbers in different regions were small, and there was little variation between rates in jurisdictions. The three year mean rates varied from 1.1 cases per million persons in the ACT to 4.9 cases per million persons in Tasmania.
Table 1: Notifications of listeriosis to Australian States and Territory health departments for 1998–2000, by type of infection.

<table>
<thead>
<tr>
<th>State</th>
<th>Year</th>
<th>Listeriosis materno-foetal infections</th>
<th>Listeriosis - other</th>
<th>Listeriosis Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
<td>Rate*</td>
<td>No.</td>
</tr>
<tr>
<td>Australian Capital Territory</td>
<td>1998</td>
<td>0</td>
<td>0.0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
</tr>
<tr>
<td>New South Wales</td>
<td>1998</td>
<td>2</td>
<td>2.3</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>2</td>
<td>2.3</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>1</td>
<td>1.1</td>
<td>17</td>
</tr>
<tr>
<td>Victoria</td>
<td>1998</td>
<td>5</td>
<td>8.0</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>5</td>
<td>8.0</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>2</td>
<td>3.2</td>
<td>8</td>
</tr>
<tr>
<td>Tasmania</td>
<td>1998</td>
<td>1</td>
<td>16.4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>0</td>
<td>0.0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>0</td>
<td>0.0</td>
<td>3</td>
</tr>
<tr>
<td>South Australia</td>
<td>1998</td>
<td>1</td>
<td>5.4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>0</td>
<td>0.0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>2</td>
<td>10.8</td>
<td>3</td>
</tr>
<tr>
<td>Western Australia</td>
<td>1998</td>
<td>0</td>
<td>0.0</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>4</td>
<td>15.5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>4</td>
<td>15.5</td>
<td>6</td>
</tr>
<tr>
<td>Northern Territory</td>
<td>1998</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>1</td>
<td>27.8</td>
<td>1</td>
</tr>
<tr>
<td>Queensland</td>
<td>1998</td>
<td>0</td>
<td>0.0</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>2</td>
<td>4.1</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>5</td>
<td>10.3</td>
<td>7</td>
</tr>
<tr>
<td>Australia</td>
<td>1998</td>
<td>9</td>
<td>3.5</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td>13</td>
<td>4.7</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>15</td>
<td>5.8</td>
<td>45</td>
</tr>
<tr>
<td>Total Cases</td>
<td>37</td>
<td>135</td>
<td>172</td>
<td></td>
</tr>
</tbody>
</table>

* Rates expressed per 1,000,000 population, except for materno-foetal infections, which are expressed per 100,000 births.

States received fewer notifications during winter months, although there was little apparent seasonality to notifications (Figure 1). In September 1999, State and Territory health departments received five materno-foetal infections. The materno-foetal infections during this month were reported from multiple jurisdictions, including Western Australia (2 cases), Victoria (1 case), and New South Wales (2 cases). This may have represented a cluster occurring across Australia, although no investigation was undertaken.
Listeriosis - Maternal-foetal infections

There were 37 distinct notifications involving pregnant women and their foetuses. The median age of mothers was recorded as 30 years old, with a range of 20–38 years old (n =29). The median length of gestation was recorded as 28 weeks, with a range of 12 to 40 weeks (n = 25). Thirteen materno-foetal infections resulted in a stillbirth or neonatal death, and nine survived (Table 2). Outcome was not recorded for the remainder of cases. No deaths were recorded for mothers.


<table>
<thead>
<tr>
<th></th>
<th>1998</th>
<th>1999</th>
<th>2000</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of pregnancy associated cases</td>
<td>9</td>
<td>13</td>
<td>15</td>
<td>37</td>
</tr>
<tr>
<td>Incidence(^1)</td>
<td>3.5</td>
<td>5.1</td>
<td>5.8</td>
<td>4.8</td>
</tr>
<tr>
<td>Deaths ≤ 24 weeks gestation</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>Deaths &gt; 24 weeks gestation</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Total Deaths</td>
<td>2</td>
<td>4</td>
<td>6</td>
<td>13(^2)</td>
</tr>
<tr>
<td>Listeria associated perinatal mortality rate</td>
<td>0.8</td>
<td>1.6</td>
<td>2.3</td>
<td>1.7</td>
</tr>
</tbody>
</table>

\(^1\) Rates per 100,000 births for Australia, 1999.
\(^2\) Includes foetal death with unknown gestation period.

\(^{iv}\) Includes one set of twins where one survived and one was stillborn.
Twenty-four (64.9%) out of 37 materno-foetal infections were recorded as non-indigenous. Four cases were recorded as indigenous. Indigenous status was not recorded for one-quarter of materno-foetal infections.

There was a decreasing trend in the incidence of both materno-foetal and non-pregnancy related infections in Victoria between the years of 1991–2001 (Figure 2).

Figure 2: Notifications of materno-foetal Listeria infections in Victoria, 1991–2001 (Unpublished data).\textsuperscript{31,32}

The practices for recording notification varied between jurisdictions, with some reporting a single case—either the mother or baby—for each pregnancy-associated infection, and others reporting both cases (Table 3). There were two sets of twins recorded in the dataset.

Table 3: Notifications reported by State and Territory health departments for pregnancy-associated listeriosis infections in Australia for 1998–2000, showing which case was represented on the dataset for each jurisdiction.
Listeriosis – Other

States and Territories identified 135 notified cases that were not coded as pregnancy-related. The median age of cases was 68 years old, with a range of 1–89 years. The highest age specific rate were in 80–89 age year old males. Rates were higher in older males than for females of the same age group, despite case numbers being similar in both sexes\(^{v}\). There was no difference between the mean ages of cases in different States \((p=0.4)\). The male to female ratio of cases was 1.0:1.06, and the gender was unknown for three cases.

Indigenous status was not routinely recorded, with status unknown for 64\% (86/135) of cases. The remaining 49 cases were non-indigenous, except for five recorded as indigenous.

There were 27 (20.0\%) deaths recorded, and the outcome was unknown for 52 (38.5\%) of cases.

Figure 3: Annual mean age and sex specific rates for non-pregnancy related listeriosis infections for Australia, 1998–2000, by ten year age group.

Risk factor information was not supplied for the majority (66.7\%) of notified cases. Some jurisdictions did not report risk factor information for the majority of cases, including New South Wales (60 cases), Northern Territory (1 case), Queensland (14 cases), and Tasmania (6 cases). The most commonly reported risk factors were solid tumours, which were recorded for 26.7\% of cases (Table 4). Haematological malignancy and diabetes each accounted for 17.8\% of cases. No immunocompromising condition was noted for 15.6\% of cases. Forty-seven cases were seventy-five years or older. Older patients were not at higher risk of dying from listeriosis \((OR 0.92, 95\% CI, 0.34–2.34)\).

\(^{v}\) The population of females surviving past the age of seventy years old is almost double that of elderly males.
Table 4: Risk factors reported for non-pregnancy related listeriosis infections for Australian jurisdictions, 1998–2000 (n = 45; Nb. Percentages do not add up, as some cases reported multiple risk factors).

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>No. cases recording risk factor</th>
<th>Proportion (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid Tumour</td>
<td>12</td>
<td>26.7</td>
</tr>
<tr>
<td>Haematological malignancy</td>
<td>8</td>
<td>17.8</td>
</tr>
<tr>
<td>Diabetes</td>
<td>8</td>
<td>17.8</td>
</tr>
<tr>
<td>End stage renal failure</td>
<td>6</td>
<td>13.3</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>4</td>
<td>8.9</td>
</tr>
<tr>
<td>Organ transplant</td>
<td>4</td>
<td>8.9</td>
</tr>
<tr>
<td>Liver disease</td>
<td>3</td>
<td>6.7</td>
</tr>
<tr>
<td>Systemic autoimmune disease</td>
<td>1</td>
<td>2.2</td>
</tr>
<tr>
<td>Other immunosuppressive condition</td>
<td>3</td>
<td>6.7</td>
</tr>
<tr>
<td>No immunocompromising condition identified</td>
<td>7</td>
<td>15.6</td>
</tr>
</tbody>
</table>

Clusters of listeriosis

States and Territories reported five clusters of cases between the years of 1995–2000 (Table 5; unpublished data).³⁴, ³⁵, ³¹, ³⁶, ³⁷, ³⁸ These clusters were responsible for 23 cases and 7 deaths. Health departments identified that cooked chicken products were responsible for two of the outbreaks, while two other outbreaks were due to fruit salads (1), and sandwiches and meat salads (1). The Victorian cluster in 1995 was a community-wide outbreak of a common strain, and no vehicle was identified.³¹, ³⁶ Three of the outbreaks related to *Listeria* infections that were acquired in health care settings.
Table 5: Clusters of listeriosis in Australia, 1995–2000.

<table>
<thead>
<tr>
<th>State</th>
<th>Year</th>
<th>No. Cases</th>
<th>No. Deaths</th>
<th>Vehicle</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>South Australia</td>
<td>1996</td>
<td>5</td>
<td>1</td>
<td>Cooked chicken products</td>
<td>Majority of cases were health care facility patients; Identified common strain using PFGE</td>
</tr>
<tr>
<td>Western Australia</td>
<td>1997</td>
<td>3</td>
<td>1 materno-foetal</td>
<td>Cooked chicken products</td>
<td>Ribotyping of human, food and environmental samples.</td>
</tr>
<tr>
<td>Victoria</td>
<td>1995</td>
<td>6</td>
<td>2 materno-foetal</td>
<td>Unknown</td>
<td>Identified common strain using PFGE; same geographic area; Four materno-foetal cases; mix of ethnicity amongst cases; No vehicle</td>
</tr>
<tr>
<td>New South Wales</td>
<td>1996</td>
<td>5</td>
<td></td>
<td>Sandwiches &amp; meat salads</td>
<td>Majority of cases were health care facility patients; foods at elevated temperatures</td>
</tr>
<tr>
<td>New South Wales</td>
<td>1997–1999</td>
<td>4</td>
<td>3</td>
<td>Fruit salad</td>
<td>Majority of cases were health care facility patients; Identified common strain using PFGE</td>
</tr>
</tbody>
</table>

Discussion

The incidence of invasive listeriosis in Australia was 3.0 cases per million population, making the incidence comparable to that of other countries (1.0–11.0 cases per 1,000,000 population). The case fatality rate during the three year period was typically high, with 35% (13/37) of materno-foetal infections resulting in death of the foetus or neonate, and 20.0% (27/135) of non-pregnancy related infections resulted in death of the patient.

One of the ways in which health authorities in Australia have sought to address the issue of listeriosis has been through public awareness campaigns. The level of consumer awareness of Listeria related illness and high-risk foods has not been well documented, however, there is some evidence for the effectiveness of distribution of information pamphlets and patient education by doctors. In Victoria, since 1991 there has been a declining trend in the incidence of listeriosis, particularly materno-foetal infections. It is difficult to determine whether this decline is due to increased awareness amongst at risk groups, or decreased exposure to the organism in foods.
Australian health agencies reported five clusters in the five years between the years 1995–2000. Three of these outbreaks were in healthcare settings, which highlights the importance of ensuring food safety is maintained in hospitals. A recent review of *Listeria* infections in Israel identified that hospitalisation *per se* was a risk factor for infection. In the Australian clusters, investigators identified cooked chicken products (2), meats and sandwiches (1), and fruit salad (1) as the vehicles. Previously, Australian investigators have identified raw vegetables, pate, and imported smoked mussels as causes of outbreaks of invasive and gastrointestinal listeriosis.

Health agencies are more commonly notified about sporadic infection in Australia, which are usually not linked to a source. It is important that health departments systematically record detailed information about these sporadic cases. We found in this review that very few States could provide important information about cases of listeriosis, such as risk factors or ethnicity. Indigenous health status was recorded as unknown in 64% of cases of non-pregnancy associated infections. It is likely that jurisdictions collect this information, but do not record it on a database.

In particular, it is important that States and Territories collect information for each case regarding:

- Pregnancy status
- Clinical presentation—central nervous system infections, bacteraemia, focal infections, or gastrointestinal
- Ethnicity—country of birth or language spoken at home, and indigenous status
- Immunosuppressive status—including coded underlying conditions, and immunosuppressive therapy
- Outcome—death, spontaneous abortion, stillbirth, recovery

In Australia, the most common underlying condition for non-pregnancy associated cases were haematological and solid malignancies, which was similar to previous reviews from Australia and other countries. To learn more about the causes of these sporadic infections, Australian health agencies should consider improving routine collection of data and conducting a case control study to explore specific food-related risk factors.

Despite the fact that very few epidemics occur, there is significant potential for clusters to occur across State and Territory boundaries due to the widely distributed nature of the food supply. One way proven to be of value is the rapid case investigations and molecular typing of all *Listeria* isolates. A working example of this is the PulseNet system in the United States, which rapidly shares Pulsed Field Gel Electrophoresis patterns across States to identify and control emerging outbreaks. PulseNet is now operational in Canada, and the European Union is currently establishing a similar system for Europe. To identify emerging outbreaks, health agencies in Australia should collaborate to develop the capacity to rapidly assemble epidemiological and molecular information on listeriosis at the national level.
There are several limitations of the data that we obtained from States and Territories, which include:

- Under reporting of listeriosis by doctors and laboratories
- Differences in State-based practices for recording pregnancy associated infections
- Incomplete information from some jurisdictions
- Poor recording of risk factor information, ethnicity and outcomes
- Slight differences in case definitions and their application from jurisdiction-to-jurisdiction
- Data from NSW potentially includes gastrointestinal listeriosis in the notification dataset, although the majority would be invasive disease, due to the difficulty of isolating *Listeria* sp. from stool.

We identified significant variation in practices for recording listeriosis cases in jurisdiction’s datasets. Some States and Territories included a mother or a baby for a pregnancy-associated infection. This made it very difficult to identify how many pregnancy-associated infections occur. In Victoria, it was relatively simple to identify pregnancy-associated cases, as they were all identified and included the mother’s details, along with other risk factor information. In many of these instances, the mother may not have met the NHMRC case definition, but the recording practice allowed consistent interpretation. It is vital that States and Territories standardise these practices to allow maximum comparability and assessment of the associated morbidity and mortality.

It is important for national policy development, that Australia regularly reviews the epidemiology of listeriosis at the national level. Because so few cases occur in States and Territories each year, the Communicable Diseases Network of Australia (CDNA) should consider collecting extra epidemiological data for listeriosis using the National Notifiable Diseases Surveillance System. Information on ethnicity, and risk factors are important for development of prevention campaigns with this disease. While there has been a decrease in the incidence of materno-foetal infections there are still infections occurring amongst ethnic minority groups, which is not evident from most State and Territory databases, or the national dataset.
Recommendation

We recommend that the:

1. Communicable Diseases Network of Australia (CDNA) and Public Health Laboratory Network develop the capacity to rapidly assemble epidemiological information along with molecular typing data for *Listeria* isolates at the national level.

2. CDNA standardise practices of States and Territories reporting listeriosis cases to the National Notifiable Diseases Surveillance System. We recommend that the CDNA have two categories for notifying listeriosis:
   a. “Pregnancy-associated”—infections that are acquired in association with pregnancy and the first three months of a neonates life. States and Territories should record only the mother as the case, with details about the infant and outcomes contained in supplementary fields for each record.
   b. “Non-Pregnancy associated”—all infections that are not associated with pregnancy.

3. CDNA support the proposal to enhance data collection of risk factor information for listeriosis to aid interpretation and assist in development of prevention strategies.

4. New South Wales Health Department considers making *Listeria* isolated from faeces only notifiable in outbreak settings.

5. Department of Health and Ageing and ANZFA consider appropriate preventive strategies for the major group at risk of listeriosis—immunocompromised people. This strategy should give particular attention to the prevention of healthcare acquired infections and may involve partnerships with Divisions of General Practice, and the College of Physicians.

6. Department of Health and Ageing and ANZFA support the OzFoodNet proposal for the case control study of listeriosis cases.
Acknowledgements

We would like to thank the following people:
• State and Territory investigators who collected and provided the data in each jurisdiction.
• Agnes Tan of the Microbiological Diagnostic Unit Public Health Laboratory in Victoria, Mark Ferson of the South Eastern Sydney Area Health Service in New South Wales, and Tim Inglis of PathCentre in Western Australia, who provided information on the cluster investigations.
• Paul Roche of the Department of Health and Ageing for his comments on this report.
• Jurisdictional members of the Communicable Disease Network of Australia for their comments and approval of this report.

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Listeriosis in New Zealand

Summary written by ANZFA from information supplied in 2001 by the Communicable Disease Group at ESR

Background to notification in New Zealand

Listeriosis was first made notifiable in 1969 following an outbreak involving 13 cases. The illness is defined by the isolation of *Listeria monocytogenes* from a normally sterile site and by meeting relevant clinical criteria, such as meningitis or intrauterine death. The current case report form divides cases into two categories, perinatal (mother and foetus or neonate) and non-perinatal. Data relating to risk factors such as pregnancy and underlying illness or immunosuppressive drugs are collected. Information relating possible infections sources is also requested, as are the usual demographic factors for cases of notifiable illness.

In addition to the cases being recorded through the National Surveillance Programme, isolates of *L. monocytogenes* from patients will be sent to the ESR Reference Laboratories for typing, as will any isolates from foods possibly linked to outbreaks. Cases are usually investigated for possible linkages to contaminated food and where an outbreak is suspected, a more intensive investigation, including testing of food samples will be undertaken.

In recent years there has been interest in the role of *L. monocytogenes* as a cause of gastroenteritis. Faecal cultures may now be performed during investigations of food poisoning events. A positive case could however be recorded in the surveillance records of notifiable illness as “gastroenteritis” rather than as “listeriosis” in the absence of the classical symptoms of listeriosis and isolation from a normally sterile site.

Incidence data

The figure below shows the reported incidence of perinatal and non-perinatal cases for the past 20 years. Data for the preceding period back to 1969 when listeriosis become notifiable is very similar.
In Table 1 the data for the 11 years up to and including 2000 have been analysed to show mortality rates (Kieft et al, 2000). During that period, the incidence of cases fluctuated between 8 and 35 with an average of 17 cases per annum. Data for the preceding period is not shown but is of a similar distribution. The average rate is about 0.5 per 100,000.

**Table 1**

**Number of reported cases of invasive listeriosis and mortality outcomes from 1990 to 2000 (Kieft et al., 2000)**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Listeriosis</td>
<td>16</td>
<td>26</td>
<td>16</td>
<td>11</td>
<td>8</td>
<td>13</td>
<td>10</td>
<td>35</td>
<td>17</td>
<td>19</td>
<td>22</td>
</tr>
<tr>
<td>Mortality (perinatal)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Mortality (non-perinatal)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

NA = not available

In Table II data for the period 1993-2000 has been broken down into age groups.
Table II

Incidence (reported cases) of listeriosis by age group

<table>
<thead>
<tr>
<th></th>
<th>0-4</th>
<th>5-14</th>
<th>15-19</th>
<th>20-44</th>
<th>45-55</th>
<th>55-65</th>
<th>&gt;65</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1993</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1994</td>
<td>1</td>
<td>3</td>
<td></td>
<td>1</td>
<td>6</td>
<td></td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>1995</td>
<td>3</td>
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<td>1</td>
<td>0</td>
<td>7</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1996</td>
<td>1</td>
<td>1</td>
<td></td>
<td>5</td>
<td>3</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>4</td>
<td>1</td>
<td>6</td>
<td>3</td>
<td>4</td>
<td>15</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>1998</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td></td>
<td>4</td>
<td>8</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>1</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>7</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>7</td>
<td></td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>6</td>
<td>22</td>
<td></td>
</tr>
</tbody>
</table>

Most of the cases in the age groups under 45 are associated with perinatal cases. Detailed analysis of cases by age done by ESR shows that occurrence of non-perinatal infections in people under 50 years of age is uncommon, in most years in the order of 0.1 per 1000,000. Even in 1997 when an outbreak occurred this rate remained low. The rates increase for people >50 with the highest rate occurring in the oldest (>70 years) segment of the population. In 1997 the rates for all age groups >50 years increased dramatically with the rate for the 70-80 group at 4.9 and for the 80+ group it was 7.7 per 100,000.

Most, but not all non-perinatal cases have a risk factor such as renal transplant, cancer or diabetes and in many cases this may be combined with age as a risk factor.

Outbreaks and food linkages

The majority of cases are considered to be sporadic. However four outbreaks of listeriosis have been recorded since 1969. Outbreaks occurred in Auckland in 1969 (14 cases) and 1980 (27) and in Christchurch in 1981-1982 (18). A nationwide outbreak occurred in 1997 (17 cases) and a cluster of 2 cases in Auckland was of particular interest because of the linkage established discussed later with a food.

Linkages to seafood were postulated but not confirmed for the 1980 outbreak

The 1997 outbreak began in February and continued for several months. All the 17 cases had the same serotype (O1/2a) and DNA type but no specific food was implicated. An indistinguishable strain was isolated from cases again in 1999.

In 2000 there were 2 outbreaks of non-invasive gastrointestinal infection reported. A total of 31 cases were linked to the outbreaks which were all associated with consumption of smallgoods from a single manufacturer. All isolates from food and faeces were of the same serotype and indistinguishable by typing (Sim et al, in prep).
A cluster involving two perinatal cases in 1980 was of particular significance because it was the first time that typing of isolates from patients, food and the processing environment was used to establish linkages. The food involved was smoked mussels (Brett et al, 1998).

Estimates of economic costs to New Zealand of listeriosis

A study of the economic cost of foodborne infectious disease estimated that listeriosis contributes 1.5% of the total cost per annum of about $NZ55 million from this type of illness. The cost per case was highest for listeriosis because of the high mortality rate at $NZ55, 000. This is 100 times higher than for illnesses such as campylobacteriosis and salmonellosis (Scott et al, 2000).

References


Flow Chart 1: Cold-Smoked Salmon

1. Pre-harvest
2. Harvest
3. Storage/Transport
4. Gilling/Gutting
5. Freezing
6. Washing
7. Storage
8. Transport
9. Storage
10. Skinning/Filleting
11. Brining
12. Cold Smoking
13. Slicing
14. Distribution
15. Storage
16. Freezing/Chilling
17. Packaging
18. Retail
19. Transport home
20. At home