RISK PROFILE:
CLOSTRIDIUM BOTULINUM
IN
READY-TO-EAT SMOKED SEAFOOD
IN
SEALED PACKAGING

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

by

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April 2006
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Risk Profile: Clostridium botulinum in RTE Smoked Seafood in Sealed Packaging
ACKNOWLEDGEMENTS

We would like to thank:

- Leone Basher, Biosecurity MAF, New Zealand;
- Graham Fletcher, Crop and Food; and
- Dorota Broda, Jackie Boerema and Gale Brightwell, AgResearch; for their advice and assistance in providing information,
- Maurice Wilson ESR MASC for his guidance and review of the document, and
- Cathy Webb of the New Zealand Seafood Industry Council for review comments on the document.
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SUMMARY

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. Risk Profiles include elements of a qualitative risk assessment, as well as providing information relevant to risk management. Risk profiling may result in a range of activities e.g. immediate risk management action, a decision to conduct a quantitative risk assessment, or a programme to gather more data. Risk Profiles also provide information for ranking of food safety issues. This risk assessment is focused on the food/hazard combination of Clostridium botulinum in smoked seafood in sealed packaging.

Of the eight types of C. botulinum toxin, the type most commonly associated with marine environments (and therefore seafood) is toxin type E. C. botulinum type E is considered an aquatic organism.

Seafood is defined for this Risk Profile as including both finfish and shellfish/crustacea. Ready-to-eat products are those which have undergone processing to include one or more pathogen reduction steps to render the products safe for consumption without further heating or cooking by the consumer. Normal cooking will destroy vegetative cells and toxins; consequently the focus is on product prepared without a cooking step; such as smoked products, and which are stored under conditions where the bacterium may grow and produce toxin e.g. low oxygen or modified atmosphere packaging.

Freezing or aerobic packaging will prevent C. botulinum growth in seafood. In product that is packaged under reduced oxygen conditions, a combination of hurdles to growth may be employed, including low temperature storage and reduced water activity through salting.

Smoking is not regarded as a primary control, although cold-smoking allows the survival of competing microflora which will inhibit growth and/or cause spoilage, and hot smoking times and temperatures will destroy vegetative cells, as well as Group II spores. Smoking will also contribute to water activity reduction.

Although data are limited, there are none showing that C. botulinum type E is present in the New Zealand aquatic environment.

An environmental study in 1992 looking for the presence of C. botulinum in New Zealand pond and waterway sediments found only Types C and D (which do not affect humans) in 11 of 20 sites in the Auckland area. Samples from other urban North Island sediments were negative.

During 1999-2000, Crop and Food Research collected 498 sediments samples from around the New Zealand coastline. The samples were enriched and tested for botulinum toxin by mouse bioassay. No positive samples were found. These samples have since been archived at –85°C and are undergoing further testing. This will involve anaerobic enrichment to grow any C. botulinum spores, followed by extraction of DNA from the enriched samples and PCR analysis to identify any toxin-producing genes.
The available data all support a very low risk for New Zealanders from *Clostridium botulinum* in vacuum packed smoked seafood. This food type has a low consumption, and studies have found no evidence for *C. botulinum* with the potential to affect humans in the New Zealand aquatic environment. The amount of imported product is low, and controls relevant to *C. botulinum* control have been specified for such imports.

The current lack of evidence of *C. botulinum* type E in the New Zealand aquatic environment means that New Zealand smoked seafood manufacturers do not use the higher salt concentrations used overseas to control *C. botulinum*. A survey of local and imported smoked salmon found salt levels in domestic product below those recommended in the scientific literature for *C. botulinum* control in non-frozen finfish. This means that the presence or absence of type E *C. botulinum* in the New Zealand aquatic environment is an important point. The more extensive testing planned by Crop and Food Research on the marine sediment samples will contribute valuable evidence on this point.

The risk management options for *C. botulinum* in smoked seafood are well understood. The question is whether these controls need to be implemented given New Zealand’s environmental status for *C. botulinum*. Better information on actual processing conditions by New Zealand manufacturers would assist in understanding the hurdles currently being achieved in local product.

Conducting surveys of seafood for *C. botulinum* is unlikely to be useful given that contamination is almost certainly absent. The data gaps identified in this Risk Profile are:

- Status of controls for *C. botulinum* in cold and hot smoked vacuum packed seafood in New Zealand; this includes information on salt levels, storage temperatures and shelf lives.
- Processing conditions, product types and ingredients used by New Zealand manufacturers of smoked seafood.
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

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

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

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

3. **Implementation of the risk management decision**

4. **Monitoring and review.**

The Risk Profile informs the overall process, and provides an input into ranking the food safety issue for risk management. Risk Profiles include elements of a qualitative risk assessment. However, in most cases a full exposure estimate will not be possible, due to data gaps, particularly regarding the level of hazard in individual foods. Consequently the risk characterisation part of a risk assessment will usually rely on surveillance data. The Risk Profiles also provide information relevant to risk management. Based on a Risk Profile, decisions are made regarding whether to conduct a quantitative risk assessment, or take action, in the form of gathering more data, or immediate risk management activity.

This Risk Profile concerns *Clostridium botulinum* in ready-to-eat smoked seafood in sealed packaging. Certain ready-to-eat smoked seafoods are prepared by processes that will not destroy any *Clostridium botulinum* spores present, and outgrowth and neurotoxin production are possible when the seafood is packed under conditions of low oxygen.

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

**Hazard identification, including:**

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

**Hazard characterisation, including:**

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

**Exposure assessment, including:**

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

- Information on the number of cases of adverse health effects resulting from exposure to the organism with particular reference to the food (based on surveillance data).
- Qualitative estimate of risk, including categorisation of the level of risk associated with the organism in the food.

**Risk management information:**

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

**Conclusions and recommendations for further action**
2 HAZARD IDENTIFICATION: THE ORGANISM

The following information is adapted from a data sheet prepared by ESR under a contract for the Ministry of Health (and now kept on the NZFSA website). The data sheet is intended for use by Regional Public Health Units.

2.1 Clostridium botulinum

2.1.1 The organism/toxin

C. botulinum bacteria are straight to slightly curved, Gram positive rods. They form oval, subterminal heat-resistant endospores which can distend the cell (Szabo and Gibson, 2003). The organism can produce the most potent biological neurotoxins known, which if ingested, result in ‘botulism’. The amount of type A toxin required to cause death in humans varies between 0.1 and 1.0 mg (Szabo and Gibson, 2003). A lower lethal dose is estimated at 1ng/kg body weight by the European Commission, (2002).

There are eight recognised antigenically distinct toxins, designated as types A, B, Cα, Cβ, D, E, F and G (Austin, 2001). All are neurotoxins except Cβ which is an ADP-ribosylating enzyme. C. botulinum type G has been renamed C. argentinense. Most isolates are single toxin producers although some can produce multiple toxins. Four of the types; A, B, E and F cause botulism in humans, although incidents involving Type F toxin are rare. Type C botulism in humans is very rare. Types C, D and E cause botulism in mammals, birds and fish (WHO, 2002).

The toxin type most commonly associated with marine environments (and therefore seafood) is toxin type E. C. botulinum type E is thus considered an aquatic organism (Huss, 1980).

It is important to note the difference between intoxication and infection when discussing botulism. Ingestion of the pre-formed toxin (foodborne) or inadvertent injection are intoxications, whereas infections occur in infant botulism, adult infectious botulism and the wound form of illness because the disease results from ingestion or wound infection by spores. Where spores germinate, the vegetative cells produce the toxin in vivo. This Risk Profile concerns the foodborne intoxication form of the disease.

The mouse bioassay is the accepted procedure worldwide for detection of the neurotoxin and is primarily used to test patient stools. There is currently no diagnostic test to detect toxin at the nerve endings (Midura, 1996).

Groups and types:

The species is sub-divided on the organism’s proteolytic ability. Four physiological Groups; I, II, III and IV are currently recognised. Most human botulism outbreaks are caused by Groups I (proteolytic) and II (non-proteolytic).

Group I includes isolates producing toxin types A, B and F, and their proteolytic activity generally causes spoilage of food and offensive odours that render the food unacceptable.
Group II includes isolates producing toxin types B, E and F, and proteolytic spoilage does not occur which means that foods can appear unaltered with no unpleasant accompanying odours. Marine environments are most commonly associated with *C. botulinum* producing toxin type E. Illness in animals is usually caused by Group III *C. botulinum* strains. There are no records of disease in humans or animals from Group IV *C. botulinum* (Szabo and Gibson, 2003).

2.1.2 Growth and survival

**Growth:**

**Temperature:** These data are for growth under optimum laboratory conditions. Minimum growth temperatures will be higher where pH and *a w* values are lower or where preservatives are added to the food.

<table>
<thead>
<tr>
<th>Group</th>
<th>Minimum</th>
<th>Optimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>10.0°C</td>
<td>35 – 40°C</td>
<td>45 – 50°C</td>
</tr>
<tr>
<td>II</td>
<td>3.0°C</td>
<td>25 – 30°C</td>
<td>40 – 45°C</td>
</tr>
</tbody>
</table>

1 The ICMSF (1996) and Szabo and Gibson (2003) cite 3.3°C as the minimum growth temperature for Group II. Work by Graham et al. (1997) found growth and detected toxin production at 3°C in 5 weeks. In a review by Peck (2006), growth was also reported at 3°C after 7 weeks, at 3.1°C after 6 weeks and at 3.2°C and 3.3°C after 5 weeks.


For the proteolytic Group I strains, growth below 10°C has not been reported and at 15°C it is slow.

Group II’s ability to grow at refrigeration temperatures means they are psychrotrophic organisms.

At the minimum temperatures, toxin production may take several weeks of to occur (Szabo and Gibson, 2003). In the scientific literature, measures of growth are often reported as ‘time to detectable toxin’.

**pH:** All strains of *C. botulinum* grow and produce toxin down to pH 5.2 (when other conditions are optimal). Group I grow slowly down to pH 4.6. Group II strains grow slowly down to pH 5.0. At this point, cells generally undergo sporulation. In some circumstances, germination and growth can take place below an initial pH of 4.6 (Szabo and Gibson, 2003). These generally involve another organism, such as a mould or bacterium being present, which raises the pH levels. There are occasional recorded instances of toxin production at lower pH values, e.g. potatoes adjusted to pH 4.83, and an outbreak linked to canned tomato juice where initially the food had a pH less than or equal to 4.6 (Szabo and Gibson, 2003).

**Atmosphere:** *Clostridium botulinum* is considered to be an anaerobe, but it can grow in air if the redox potential (*Eh* - the overall balance of oxidizing and reducing agents present) is low. For example, a foodborne outbreak of botulism occurred when *C. botulinum* spores were...
trapped between the skin and the foil in baked potatoes (Angulo et al., 1998). It has been suggested that the metabolic activity of high numbers of spores germinating lowers the $E_h$ to allow clostridial bacterial growth (Szabo and Gibson, 2003).

The presence of up to 20% oxygen in packaged foods has been shown not to prevent growth of the bacterium. Carbon dioxide concentrations above 75% have retarded growth of *C. botulinum*, but toxin production by the Group II organisms in fish (under temperature abuse conditions) still occurs under 100% carbon dioxide. Modified atmosphere packaging together with refrigeration did not prevent growth and toxin production by Group II organisms in cooked turkey.

**Water activity:** *C. botulinum* can grow at water activity ($a_w$) values of 0.9353 (NaCl =10%) for Group I and 0.9707 (NaCl=5%) for Group II. Interactions with additional factors such as pH can influence the NaCl concentrations required to permit growth. Growth of Group I *C. botulinum* occurred at pH 5.2 in the presence of 3.95% NaCl, while at 4.67% NaCl, growth occurred at pH 5.6 (Szabo and Gibson, 2003). Toxins can be produced at $a_w$ values which permit growth.

Group I *C. botulinum* could not grow at 3.95% NaCl at a pH of 5.0. At 4.67% NaCl, growth was inhibited at pH 5.4 (Szabo and Gibson, 2003).

**Growth enhancing micro-organisms:** The growth of acid-tolerant moulds such as *Cladosporium* spp., and *Penicillum* spp. can result in pH increases in foods that permit the growth of *C. botulinum* (Austin, 2001).

**Survival:**

**Temperature:** Reductions in numbers of vegetative cells under frozen storage are poorly understood. All types of spores and toxins are resistant to freezing. Studies have demonstrated that frozen storage does not reduce the activity of preformed botulinum neurotoxin in food (ICMSF, 1996).

**Water activity:** Spores can survive drying. Botulinum toxin type A can be preserved and used in medicine by drying with human serum albumin (stabiliser) at pH 7.3, although a substantial loss in toxicity occurs (up to 50 - 90%) (Schantz and Johnson, 1992).

2.1.3 **Inactivation, Critical Control Points and hurdles**

Note that in microbiological terms “D” refers to a 90% (or decimal or 1 log cycle) reduction in the number of organisms. Details of thermal inactivation of *C. botulinum* have largely been derived from work for the food canning industry.

**Temperature:** At temperatures at or below 3°C, germination and growth of both Group I and Group II *C. botulinum* will not occur. This can be used as a control point for storing products under deep chill conditions without any additional controlling factors (Betts, 1996). It is unlikely however that such super-chilled temperatures can be maintained throughout the distribution chain, particularly for products destined for domestic use (Peck, 2006).
All vegetative cell types of *C. botulinum* are readily killed at pasteurisation temperatures. Destruction of spores requires higher times and temperatures. For spores of Group I strains, the ‘botulinum cook’ was developed. This is a well known food processing procedure for low-acid (pH>4.6) canned foods based on the destruction of Group I heat resistant spores. The ‘12D’ process, as it is also known, equivalent to exposure to moist heat at 121°C for 3 minutes, is sufficient to reduce the population of spores by a factor of $10^{12}$. At 100°C, it takes 25 minutes to inactivate 90% of spores.

Group II spores are less resistant to heat than Group I. At 100°C, 90% of spores are destroyed in less than 0.1 minute (ACMSF, 2005).

Recent papers have challenged the log linear nature of spore inactivation which is the conventional model for bacterial inactivation by heat, and suggest a Weibull model distribution curve to represent the relationship more accurately (Van Boekel, 2002). Application of this model to *C. botulinum* spores has been reported (Peleg and Cole, 2000) but the implications for commercial sterility requirements are not resolved.

All neurotoxins can be inactivated at time/temperature combinations ranging from 65°C for 1.5 hours to 85°C for 1 minute. Alternative time/temperature combinations of 80°C for 30 minutes to 100°C for 10 minutes have been suggested (Slovis and Jones, 1998).

**pH:** Since a pH of 4.6 is considered the demarcation point at or beneath which Group I *C. botulinum* is not able to germinate and grow, high acid canned foods (pH< 4.6) are not required to undergo the full ‘botulinum cook’. Similarly, the low pH formed by fermentation in certain meat products prevents growth. Neurotoxins are inactivated at pH 11 and above.

**Water activity:** Growth of Group I *C. botulinum* strains is inhibited when NaCl in water exceeds 10% ($a_w=0.9353$). Growth of *C. botulinum* type E is inhibited for 13 weeks at 10°C by the presence of 5% NaCl (Graham *et al.*, 1997). At temperatures less than this, the concentration of NaCl required to inhibit growth at a given pH reduces such that at 5°C, 3.5% NaCl results in a similar inhibition.

**Stability in water:** In tap water, the toxicity of toxin types A and B are reduced by 80% at room temperature for 1 to 3 days, compared to 2 to 4 days for type E toxin (Szabo and Gibson, 2003).

**Preservatives:** Preservatives such as nitrates, sorbic acid, parabens, phenolic antioxidants, polyphosphates and ascorbates inhibit *C. botulinum* growth. Interactions between reduced water activity, pH and temperature are usually used in combination with preservatives to achieve several microbial hurdles. Curing salts, particularly sodium nitrite, are used in meat preservation specifically to control *C. botulinum*. Smoking of meat and meat products has little effect on spores although adding liquid or generated smoke reduces the inhibitory level of NaCl in aqueous phase from 4.6 to 2.8% for type A spores and from 3.7% to less than 2% for type E spores (Szabo and Gibson, 2003). Nitrates and nitrates are not permitted as a preservative in preserved fish by the Australia New Zealand Food Standards Code.

**Competitive micro-organisms:** The acid produced by lactic acid bacteria such as *Lactobacillus*, *Pediococcus* and *Streptococcus* spp. is inhibitory to growth of *C. botulinum* because they reduce the pH and some produce bacteriocins (research cited in Austin, 2001).
An inhibitory action was also observed against type E *C. botulinum* by naturally occurring *Bacillus* spp, the food medium was cooked surimi nuggets (Lyver et al., 1998).

Radiation: All spores are relatively resistant. Resistance is greater below about –80°C than at ambient temperature. To inactivate *C. botulinum* spores in foods and neutral buffers, at a temperature below 10°C, the D values for group I strains vary between 2.0 and 4.5 kGy. Group II, type E spores require marginally less radiation at 1.0 to 2.0 kGy (Szabo and Gibson, 2003). Toxins, like all proteinaceous toxins, are not inactivated by the level of irradiation used by the food processing industry (ICMSF, 1996).

Disinfectants: The sporicidal nature of chlorine and iodophor compounds and their common use in the food industry as disinfectants is potentially useful, although optimum effectiveness is determined by the type of compound, concentration, exposure time, temperature and presence of organic matter. Chlorine is more effective at low pH (3.5) than at neutral or high pH. Group I spores are more resistant than those from Group II organisms.

Ozone and chlorine dioxide will inactivate spores, as will ethylene oxide, although the mechanism is unknown. Hydrogen peroxide levels up to 35% are required for spore inactivation (usually together with a raised temperature combination) (Szabo and Gibson, 2003).

Pressure: Under high pressure, spores are reported to be very pressure resistant in contrast to vegetative cells. However under low pressure, spore inactivation can be more rapid and complete. This is explained by the process taking place in two stages. First, the pressure causes the spores to germinate, then further pressure (or high temperature) inactivates the germinated cell. Throughout the whole pressure range, there is a strong synergy with heat. The inactivation of *C. botulinum* is extremely important in an expanding range of pressure-pasteurised products including those with high pH and high water activity (Peck et al., 2004).

To summarise, the canning industry have developed procedures which destroy *C. botulinum* spores. Where a ‘botulinum cook’ is not practical, hurdles are used, mainly in the form of temperature controls (to inhibit germination and growth), lowered pH (acidification) and reduced water activity through manipulation of salt levels.

2.1.4 Sources

**Human:** *C. botulinum* is not a normal part of the healthy human intestinal flora.

**Animal:** The disease in animals is usually the intoxication form although spores of *C. botulinum* have been found in the intestinal tracts of fishes, birds and mammals and in decomposing carrion. Many predator and scavenger vertebrates feeding from carrion are assumed to have developed immunity by selection. Animals particularly affected are cattle and birds, and to a lesser extent, horses, sheep, pigs and zoo animals. The main types involved in animal cases are toxins C and D, and to a lesser extent, toxin types F and G. An outbreak of *C. botulinum* type C in wild waterfowl in 1971 demonstrated that the genus occurred in New Zealand. In 1986, the first confirmed case of botulism in a dog in New Zealand (intoxication - type C) occurred in Hamilton (Wallace and McDowell, 1986).
Food: Many surveys have identified *C. botulinum* spores in food, especially fish, meats, honey and vegetables/mushrooms.

Environment: *C. botulinum* is found worldwide. Types A, B and F spores are distributed widely in soils and sediments. However, there is a recognised divide in North America. Soil west of the Mississippi river in the USA is predominantly contaminated with type A spores while soil east of the river is predominantly type B. Sediments from the Great Lakes region contain type E only while wetlands soil from Saskatchewan, Canada yield type C spores. The ICMSF (1996) reports that type B spores are more prevalent in European soils. Despite its ubiquitous nature, the level of spore contamination is often low or very low.

Smith (1978) reported that type A strains were isolated from neutral to alkaline soils (pH 7.5 average) while type B strains were from slightly more acidic soils (average pH 6.25). The reasons for this are unknown.

In New Zealand, types C and D (which are not linked to human botulism) have been isolated in lake and waterway sediments in the Auckland area (Gill and Penney, 1982).

Transmission Routes: Person-to-person transmission of botulism does not occur (WHO, 2002). Transmission routes for foodborne botulism are predominately through home-preserved foods, particularly traditional native foods.

2.2 *Clostridium botulinum* Isolation and Typing

Direct plating of food or faeces onto suitable agars rarely yields *C. botulinum* because of the presence of other competitive organisms. Heat treatments to isolate spores followed by subculturing from enrichments are the preferred method. Incubation is generally 5 to 10 days at 26 to 35°C. Identification of neurotoxin types after enrichment is carried out by mouse bioassay protection tests (takes 48 hours) or ELISA (enzyme-linked immunosorbent assays), both techniques are detailed in Szabo and Gibson, (2003). A table of the various detection methods (mouse lethality, ELISA, endopeptidase, various PCR methods, fluoroimmunoassays, fibre optic-based biosensor, evanescent wave immunosensor) can be found along with type of neurotoxin detectable and associated bibliographic references in European Commission (2002).

Molecular techniques have determined the gene sequence of the bacterium. Comparative analysis of the 16S and 23S rRNA gene sequence have identified sequences which are characteristic of each physiological group (I, II, III and IV) and confirm how close genetically *C. botulinum* is to non-neurotoxin producing clostridia (Szabo and Gibson, 2003).

The fact that the organism can be a natural inhabitant of unpolluted waters means that testing for faecal indicators is of little use. Samples may be positive for presence of *C. botulinum* yet free of faecal indicators (Hudson, 1997).

Testing for *C. botulinum* is technically difficult and can create potential biohazard problems in laboratories. In some instances, such as testing for compliance with import food criteria, salt concentration and aerobic plate counts (APC) (to determine temperature abuse) are used as alternate markers for control.
2.3 Spore Formation and Destruction

Spores represent a metabolically dormant form of the organism derived from vegetative cells. Spore formation is generally induced by restriction in availability in one or more nutrients, or else a slowing of growth of cells. It also appears that spore production comprises part of the population of a growing culture (Setlow and Johnson, 1997).

Spores are more resistant to environmental challenges and control measures than vegetative cells. Such challenges include freezing, drying, pressure, radiation, ultraviolet light, chemicals, and heat. Reactivation of spores can be initiated by low pH, a number of chemicals (especially nutrients), and most commonly, sublethal heat (Setlow and Johnson, 1997).
3 HAZARD IDENTIFICATION: THE FOOD

3.1 Relevant Characteristics of the Food: RTE Smoked Seafood in Sealed Packaging

Seafood is defined for this Risk Profile as including both finfish and shellfish/crustacea. Ready–to-eat products are those which have undergone processing to include one or more pathogen reduction steps to render the products safe for consumption without further heating or cooking by the consumer. Normal cooking will destroy vegetative cells and toxins; consequently the focus is on product prepared without a cooking step; such as smoked products, and which are stored under conditions where the bacterium may grow e.g. low oxygen or modified atmosphere packaging.

Smoking was originally used as a preservation technique, caused by the reduction of water activity in combination with the effect of prior brining or salting. The anti-bacterial qualities in the smoke also served to reduce the number of organisms present (Cutting and Spencer, 1968). With current food preservative technologies, commercial smoking operations are used to impart characteristic organoleptic qualities rather than its original purpose.

Placing smoked seafood into sealed packaging such as vacuum packaging reduces the amount of oxygen present therefore prolonging shelf life by inhibiting the growth of aerobic spoilage bacteria (USFDA, 2001). However, the creation of a low redox potential combined with a process which does not destroy botulinum spores has the potential, if not properly controlled, to allow the germination of spores and subsequent neurotoxin production.

The flesh of fish is naturally low in carbohydrate, and post mortem acidification is restricted in the tissues. The ultimate muscle pH is 6.2 – 6.5, compared with mammalian muscle at approximately 5.5. With no carbohydrate present, any microbes present use nitrogenous materials for nutrients, producing off-flavours and odours more quickly than mammalian muscle (Adams and Moss, 2000).

The type of packaging is important, the purpose of sealed packaging is to inhibit aerobic spoilage microbes thereby increasing shelf-life. Sealed packaging can be defined in terms of its oxygen transmission rate. Packaging that provides an oxygen transmission rate of 10,000 cc/m²/24 hrs (for example 1.5 mm polyethylene) would not be considered as sealed as it could provide sufficient exchange of oxygen to allow aerobic spoilage organisms to grow (and therefore spoilage will take place before any neurotoxin is produced) (USFDA, 2001). An oxygen-impermeable package can be defined as having an oxygen transmission rate less than 100 cc/m²/24 hrs, for example 2 mm polyester (USFDA, 2001).

For the purposes of this Risk Profile, vacuum-packaged (VP) and Modified Atmosphere Packaging (MAP) will be considered as sealed packaging.

The Code of Practice for Fish and Fishery Products developed by Codex includes a draft standard for ready-to-eat smoked finfish (Codex, 2005b). The proposed draft standard was prepared by Denmark and is currently at Step 3 of the Procedure.

The draft proposes some process definitions, for example salting and smoking. Salting of the fish is defined as dry salting, brining by immersion, or brining by injection.
The draft definition of smoking as described by Codex is as follows:

- Hot smoking: fish are treated with smoke generated from burning or smouldering wood or any other plant material at a temperature that will cause complete coagulation of the fish flesh;
- Cold smoking: as above but without visible coagulation of the fish flesh;
- Liquid smoking: fish are treated with liquid smoke, regenerated from smoke condensates [in a smoking chamber under the same time and temperature conditions as hot or cold smoking].

The draft standard also states that the formation of *C. botulinum* can be controlled through an application of science based options involving packaging type, storage temperature, and the use of salt in the water phase.

### 3.2 The Food Supply in New Zealand: RTE Smoked Seafood in Sealed Packaging

The New Zealand fishing industry is the fourth largest export earner behind dairy, meat and forestry. In 2003, the industry exported $1.2 billion worth of seafood, around 2% of the total global seafood trade. New Zealand’s Exclusive Economic Zone (EEZ) is the fourth largest coastal fishing zone in the world at 2.2 million square miles (Seafood Industry Council, [http://www.seafood.co.nz/business/](http://www.seafood.co.nz/business/) accessed 12.04.06). The zone extends 200 nautical miles out from the coastline. Only 130 species of fish are commercially caught, of which 43 species are commercially significant. Predominantly these are the deepwater species such as hoki, hake, ling, orange roughy, oreo dories, squid, and silver warehou as well as spiny red rock lobster, paua (abalone), greenshell mussels, and snapper. A fisheries quota management system was implemented in 1986 to control the marine catches in order to sustain yields (Ministry of Fisheries, 1999). Improving storage techniques to lengthen shelf life is important to New Zealand because of the distance of markets when exporting.

The Seafood Industry Council was created in 1996 to represent all sectors of the fishing industry. A number of more specialist industry organisations exist, these can be accessed form the Seafood Industry Council website; [http://www.seafood.co.nz](http://www.seafood.co.nz) under the industry organisations link.

#### 3.2.1 Imported food

For the year ending September 2005 New Zealand imported 30.9 tonnes of smoked fish (23.3 tonne from Chile, 3.6 tonne from Philippines, 2.5 tonne from UK) and 5.5 tonnes of smoked salmon from Norway - this equates to about 0.02 g/person/day (Peter Cressey, ESR, personal communication, April 2006).

Under the tariff codes for smoked fish vacuum-packed imports ([http://www.nzfsa.govt.nz/imported-food/high-risk/13fishsmokedvacuumpackednf.htm](http://www.nzfsa.govt.nz/imported-food/high-risk/13fishsmokedvacuumpackednf.htm)), smoked fish include pacific, Atlantic and Danube salmon, herrings, blue cod, eel fillets, whole eels, hoki, kahawai, mackerel, mullet, red cod, snapper, tarakihi, trevally and others.

Any imported smoked seafood must meet salt concentration requirements and microbiological criteria under the Imported Food Standards, Management Rule 13: Fish - smoked (vacuum-packed) see [http://www.nzfsa.govt.nz/imported-food/high-]
As stated in Section 2.2, salt concentration and APC counts are used as alternate markers. The salt content must be more than 3.4% in the aqueous phase and the criterion for APC/g at 35°C; n=5, c=2, m=10⁴, M=10⁵. New Zealand imposes these stricter requirements on imported product (as opposed to domestic product) because *C. botulinum* has been found in overseas aquatic environments and is a potential contaminant.

### 3.3 Processing and the Control of *Clostridium botulinum* in Vacuum Packed Seafood

The meat and internal organs of healthy freshly caught fish are virtually sterile, although the skin, gills and alimentary tract can all carry substantive bacterial loads. Fish skin counts have been reported between $10^2$ to $10^7$ cfu cm⁻², and from $10^3$ to $10^9$ cfu g⁻¹ in gills and intestines. Spoilage is usually microbial although oily fish are susceptible to oxidative rancidity (Adams and Moss, 2000). Obligately anaerobic bacteria are uncommon on the surface of fish but can occur in significant numbers in the intestine (ICMSF, 1998). Because molluscs are commonly associated with inshore environments, their microflora can reflect terrestrial influences (ICMSF, 1998).

*C. botulinum* contamination is most likely to derive from marine sediments, and is usually only prevalent in low numbers in fish. Studies have found type E and non-proteolytic strains of types B and F in the intestines and occasionally the skin of marine fish (Hobbs, 1976, cited in ICMSF, 1998).

*C. botulinum* may also occur in farmed fish sediments where the organism may enter the environment via the fish food and proliferate in any dead fish present (Bell and Kyriakides, 2000). Finfish are not immune from neurotoxin and have themselves died of botulism due to the consumption of other dead finfish, this has been described in farmed juvenile Coho salmon (Ecklund *et al.*, 1982a).

The evisceration stage is the single most important step to contain *C. botulinum* contamination, but even with the most hygienic methods employed, it is not possible to eliminate its presence completely from the raw material (Bell and Kyriakides, 2000).

A Code of Practice for Fish and Fishery Products (Codex, 2005a) is currently being developed and is in draft form at Step 3 (of 8) of the procedure (therefore the Standard is still open to discussion). Appendix V (Codex, 2005b) summarises the control and prevention of *C. botulinum* toxin formation, categorised according to storage temperature and packaging, including reduced oxygen packaging. This is reproduced in Table 1 below. From this information, it is clear that control of *C. botulinum* is dependent on the use of multiple hurdles including storage temperature, packaging, and salt concentration. The draft Codex Code recognises that as new technologies are developed, new controls may be forthcoming such as modified atmosphere with high oxygen content.

A UK guideline which uses these hurdle concepts is described in the Campden and Chorleywood Food Research Association publication; “A Code of Practice for the manufacture of vacuum and modified atmosphere packaged chilled foods with particular regard to botulism” (Betts, 1996). The following definitions summarise the shelf-life and storage-life categories used (modified from Betts, 1996);
• Short shelf life (10 days or less @ chill temperatures >3°C to 8°C). Temperature is the sole controlling factor in this category.

(A draft document published January 2004 (Food Standards Agency, 2004) and debated by ACMSF, (2004) is considering whether the short shelf life category should be split; i.e. those products stored between 3°C and 5°C to have a maximum shelf-life of 10 days and those products stored >5°C to 8°C to reduce the maximum shelf life to 5 days).

• Long shelf-life (greater than 10 days @ chill temperatures >3°C to 8°C), the products must meet one or more of the specific controlling factors detailed below;

> Minimum heat treatment of 90°C for 10 minutes of equivalent,
> pH of 5 or less throughout the food,
> salt level of 3.5% (aqueous) throughout the food,
> a_w of 0.97 or less throughout a food,
> any combination of heat and preservative factors which has been shown to prevent growth of toxin production by C. botulinum.

• Entire storage life spent below or at 3°C, there will be no growth of Group I or II C. botulinum and therefore it is not appropriate to determine length of storage life; the growth of other organisms or quality issues will determine shelf-life.

Studies of the growth of C. botulinum type E in vacuum packed seafood are as follows;

A study of processed crawfish in Louisiana, USA, did not find this bacterium in uninoculated samples (Lyon and Reddmann, 2000). Cooked crawfish were inoculated with $10^3$ C. botulinum type E spores per g, vacuum packaged in both a high barrier film and an air-permeable bag, and stored at 4°C and 10°C for 30 days. C. botulinum type E toxin was not detected in any of the inoculated samples until day 30, when the vacuum packed samples stored at both 4°C and 10°C were positive.

Shelf life and the potential for toxin production by C. botulinum type E in retail type packages of fresh aquacultured salmon fillets stored under a variety of atmospheres, packaging and temperatures were investigated in a study by the US FDA (Reddy et al., 1997). Samples were inoculated with 100 spores per g. At each of the storage temperatures (4, 8 and 16°C) toxin was detected about the same time, or after, the sample was declared spoiled. At 4°C, under vacuum packaging, toxin was not detected until after 66 days.

Neither of these studies involved smoked or salted seafood. Inoculation was with spores rather than vegetative cells.

Growth of C. botulinum type E was monitored in vacuum-packed cold-smoked rainbow trout containing 3.2% (w/v) NaCl and compared to the growth predicted by Food MicroModel and Pathogen Modeling Program (Hyytiä et al., 1999). Toxic samples were detected after 3 and 4 weeks storage at 8 and 4°C respectively, but there was no increase in the numbers of the organism present. There was a small fall followed by a small rise in numbers at 4 °C when the inoculum was between 1 and 10 cfu/g. The models did not predict the behaviour of the organism well.
Time to toxin production in raw fish at the same and similar temperatures appears to be rather more rapid, with a concomitant increase in the numbers of the organism present (Hyytiä et al., 1999; Kimura et al., 2001).

An extensive seafood challenge study with non-proteolytic *C. botulinum* in MAP has been carried out and the results are presented in Szabo and Gibson (2003). In summary, all the studies bar one demonstrate that as storage temperatures rise, the margin of safety decreases between sensory spoilage and the growth and subsequent neurotoxin formation of *C. botulinum*. The exception were fresh cod fillets that became toxic before sensory rejection, even though the product was stored at 4°C in 100% carbon dioxide. The key message from these studies is the strict control of storage below 4°C or less to prevent toxin development prior to the product spoiling (Szabo and Gibson, 2003).
### Table 1: Draft Codex proposals for control and prevention of *C. botulinum* toxin formation in fish and fishery products

<table>
<thead>
<tr>
<th>Storage temperature</th>
<th>Packaging</th>
<th>Water phase salt*</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>0° to 3°C</td>
<td>Any</td>
<td>No minimum water phase salt is needed</td>
<td>Temperature monitoring required on each package</td>
</tr>
<tr>
<td>&gt;3°C to 5°C</td>
<td>Aerobically packaged</td>
<td>No minimum water phase salt is needed</td>
<td>Storage temperature is for the control of pathogens generally and for quality. In air-packaged products, aerobic spoilage organisms provide sensory signs of spoilage before the formation of toxin by <em>C. botulinum</em>. Nonetheless, where there is a reasonable possibility of severe time/temperature abuse, the country where the product is being consumed might choose a water phase salt barrier of at least 3% to 3.5% as a precautionary measure. For that reason, the country where the product is consumed may still require water phase salt as a barrier to growth of non-proteolytic strains of <em>C. botulinum</em> if there are concerns about the ability of transporters, retailers or consumers to maintain time/temperature control.</td>
</tr>
<tr>
<td>Frozen (≤-18°C)</td>
<td>Reduced Oxygen (including vacuum packaging and modified atmosphere packaging)</td>
<td>No minimum water phase salt is needed for safety</td>
<td><em>C. botulinum</em> toxin cannot form when product is frozen. Because toxin production can occur after thawing, labelling information about the need to keep frozen, to thaw under refrigeration, and to use the product immediately after thawing is important.</td>
</tr>
<tr>
<td>&gt;3°C to 5°C</td>
<td>Reduced Oxygen (including vacuum packaging and modified atmosphere packaging)</td>
<td>Water phase salt at minimum level of between 3% &amp; 3.5% may be selected by the country where the product is to be consumed.</td>
<td>Water phase salt at minimum level of between 3% &amp; 3.5% in combination with chilling will significantly delay (or prevent) toxin formation</td>
</tr>
<tr>
<td>&gt;5°C to 10°C</td>
<td>Reduced oxygen</td>
<td>5% water phase salt</td>
<td>Non-proteolytic <em>C. botulinum</em> are controlled under these conditions.</td>
</tr>
</tbody>
</table>

* As an alternative to water phase salt, time/temperature controls alone may be used (at or below 3°C). (Source: Codex, 2005b – Draft proposals at Step 3).
3.3.1 Salting

Salting is used in combination with smoking. As well as providing flavour, the salt in combination with the drying nature of smoking, results in a firmer texture. Traditional products preserved by salting alone have a high salt content (20% or more). A lower salt content requires chilling or freezing to preserve the final product (MAF, 1995). The main preservative effect of salt is in the reduction of water activity in the product.

3.3.2 Smoking

Cold smoking is carried out in smoking chambers at ambient temperatures between 25 – 30°C for several hours (Bell and Kyriakides, 2000). The fish is not cooked but two distinct processes take place; drying and the penetration of smoke constituents (Cutting and Spencer, 1968). The treatment of cold-smoked products is such that natural spoilage (and therefore competitive) bacteria are not destroyed. These bacteria would cause spoilage before toxin production could occur and so acid-forming bacteria complement the hurdle approach.

Hot smoking involves placing the seafood in special kilns, operating at around 90 – 95°C for several hours during which time the internal temperature of the products reaches 65 – 75°C for approximately 30 minutes. The behaviour of Group II C. botulinum spores, and the time/temperature combinations required in the hot-smoking process to achieve destruction of these spores are described in Table 2 (ICMSF, 1996).

Table 2: D values for C. botulinum Group II spores (minutes); effect of temperature on heat resistance

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Type B</th>
<th>Type E</th>
<th>Type F</th>
</tr>
</thead>
<tbody>
<tr>
<td>77°C</td>
<td>-</td>
<td>2.4 – 4.1</td>
<td>0.2 – 9.5</td>
</tr>
<tr>
<td>80°C</td>
<td>-</td>
<td>0.55 – 2.15</td>
<td>-</td>
</tr>
<tr>
<td>82.2°C</td>
<td>1.5 – 3.2* (100**)</td>
<td>0.49 – 1.2</td>
<td>0.25 – 42.4</td>
</tr>
</tbody>
</table>

*D value (minutes). Note: D values vary with the medium in which the spores are heated (e.g. buffer or food), the pH and aw, and with the manner in which spores were produced, and depending on strain.

**10 µg/ml lysozyme in recovery medium

Non-proteolytic Group II spores, particularly type E are relatively heat sensitive and should normally be killed by adequate hot smoking (Hudson, 1997).

In the field, five commercial hot-smoking processes used by Finnish fish-smoking companies were studied, in particular the thermal inactivation of type E spores. The fish media used were rainbow trout and whitefish media at 75°C to 93°C (Lindström et al., 2003). The reduction in the number of non-proteolytic spores was less than 10^3. The importance of moist heat was demonstrated by enhancing spore inactivation. Current European guidelines recommend a 10^6 pathogen reduction factor (90°C for 10 minutes or equivalent).

The hot smoking process will not destroy Group I proteolytic spores; only a botulinum cook equivalent will do this. However, because the minimum temperature for growth of Group I C. botulinum is 10°C, the chill chain provides a control. At temperatures above 10°C, the proteolytic nature of the organism would cause spoilage (including pack inflation and
blowing) and this will avoid product consumption (Hudson, 1997). The salt level required to inhibit growth of Group I spores (10%) would be to an unacceptable for organoleptic reasons and therefore refrigeration is an important control step for these types of spores.

3.3.3 Sodium nitrite and potassium nitrate

Sodium nitrite has been used in combination with sodium chloride for decades in processed meats as an inhibitor of *C. botulinum*, however its use in fishery products is restricted in many European countries and here in New Zealand and Australia. (Although use of sodium nitrite is permitted in the USA – see section 7.1.5). Nitrate alone has little inhibitory effect but where nitrate-reducing bacteria are present, can act as a reservoir for nitrite. Nitrite levels decrease during storage, dependent on temperature and product formulation.

Hyytia *et al.*, (1997) have studied the depletion rates for sodium nitrite and potassium nitrate in vacuum-packed cold-smoked rainbow trout over an eight-week period stored at 4 and 8°C. They also assessed the toxigenesis of non-proteolytic *C. botulinum* over a six-week period at the same temperatures in the presence of each curing salt. The findings were that sensorial shelf-life of products were considerably improved by both curing salts compared to the control using NaCl only. Aerobic plate counts at 4°C were significantly lower in the nitrite and nitrate treated samples then those controls solely using NaCl, the differences at 8°C were smaller. Nitrite depletion was more rapid at 8°C while nitrate depletion was not significantly affected by temperature. Overall the concentrations of nitrite and nitrate used did not completely inhibit toxigenesis of non-proteolytic *C. botulinum* over the six-week storage although numbers of toxic samples were considerably reduced using nitrite and nitrate in the curing process.
4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

In humans, there are five recognised clinical forms of botulism (WHO, 1999):

- **Foodborne**: food contaminated with preformed botulinum neurotoxin, the toxins associate with non-toxic proteins and the resulting toxin is then protected through the gastric acid conditions of the stomach,
- **Infant**: ingested spores that survive the acidity of the stomach and germinate, colonise and produce neurotoxin *in vivo* in the intestinal tracts of infants below 12 months of age,
- **Adult infectious**: affects adults with altered gastro-intestinal anatomy (e.g. abdominal surgery) and microflora, e.g. conditions such as Crohn’s disease. Similar disease mechanism to infants, ingestion of spores and colonisation of the intestines producing toxæmia,
- **Wound**: where spores grow and produce neurotoxin in the wound, associated with intravenous and subcutaneous drug use, and
- **Inadvertent botulism**: suspected where patients have a history of ‘botox’ injections. The toxin is administered into large muscle groups for a systemic effect or as a suicide attempt. A marked clinical weakness is observed along with electro-physiologic abnormalities.

This Risk Profile concerns foodborne botulism. Thorough literature reviews on food-associated botulism have been carried out by Hauschild and Dodds (1993) and Lund and Peck (2000).

All toxins share the same pathogenic mechanism, essentially because they are based on the same structure. However, type A has the greatest affinity for nerve tissue (Midura, 1996).

4.1 Symptoms

*Incubation*: The disease is one of ingestion of pre-formed neurotoxin, and the time period from ingestion of the toxin and a response which can range from a few hours to several days. Typically symptoms are noticeable between 12 to 36 hours.

*Symptoms*: Initially nausea, vomiting and sometimes diarrhoea. Nausea and vomiting occur more often in type B and E intoxication rather than in type A, whereas muscle weakness is not often associated with type E (Austin, 2001). The early onset of nausea and vomiting in type E cases is credited with subsequently lower mortality (ICMSF, 1996). Neurological symptoms follow usually in the cranial nerve areas, symptoms include blurred or double vision, dilated pupils, photophobia, loss of mouth and throat functions. The symptoms descend through the body and include ptosis, paralysis of motor nerves, fatigue, lack of muscle co-ordination and respiratory impairment. Where the disease is fatal, the main cause is respiratory failure and/or obstructed air passages. Constipation may be present after the onset of neurological symptoms and abdominal pain may be present throughout (Austin, 2003; Szabo and Gibson, 2003; WHO, 1999). Misdiagnosis is common and is particularly confused with Guillain-Barre syndrome (Shapiro *et al.*, 1998).

*Condition*: Foodborne botulism, usually confirmed by toxin detection in the patient’s serum (Szabo and Gibson, 2003).
Toxins: Foodborne intoxication is predominately the ingestion of the pre-formed toxin which is absorbed through the gastric and upper intestinal mucosa. The toxin enters the blood and subsequently affects the peripheral nervous system (Szabo and Gibson, 2003). Once the toxin reaches the bloodstream, it circulates the body for between 8 – 10 days (Gelli et al., 2002).

At Risk Groups: All members of the population are susceptible. There are no particular at risk groups identified.

Long Term Effects: Before the effect of anti-sera in 1960, the fatality rate was high. Austin (2001) cites the Chinese situation where the fatality rates were around 50% before anti-serum treatment, with a sharp decline to 8% with the advent of the treatment. The fatality rate quoted in Szabo and Gibson (2003) is currently 5 – 15%. Improved medical care mainly through prompt antitoxin administration and respiratory support are credited with the declining mortality rate (WHO, 1999).

Treatment: There are no drugs reported in the literature that can reverse the effects exerted by botulinal neurotoxins (Szabo and Gibson, 2003). The objective in the early stages of the illness is to inactivate and remove the neurotoxin. Injections with anti-serum neutralise circulating toxin can be effective if administered early. Supplies of bivalent antitoxin (type A and B) are held in Auckland hospital pharmacy department for New Zealand. Removal of residual toxin is performed by gastric lavage or treatment with emetics if food exposure was recent, or cathartics or enema procedures (WHO, 1999). Subsequent treatment focuses on counteracting respiratory muscle and diaphragm failure by mechanical ventilation (Austin, 2001).

4.2 Dose Response

Botulinal neurotoxin is among the most toxic of naturally occurring substances known. The minimum toxic dose of botulinal neurotoxin is uncertain in humans. Estimates have placed the amount of type A toxin needed to cause death between 0.1 and 1.0µg. These figures were extrapolated from mouse models (ICMSF, 1996). In monkeys and mice, LD$_{50}$ of neurotoxin was measured at approximately 0.4ng/kg. The dose was introduced intravenously (Gill, 1982).
5  EXPOSURE ASSESSMENT

5.1  The Hazard in the New Zealand Food Supply: *Clostridium botulinum* in RTE Smoked Seafood in Sealed Packaging

5.1.1  *Clostridium botulinum* in RTE smoked seafood in sealed packaging

There have been no New Zealand surveys for *C. botulinum* or its toxin in ready-to-eat smoked seafood in sealed packaging or in any type of seafood.

A survey of the microbiological quality of vacuum packed smoked salmon (Nortje et al., 2001) aimed to determine whether locally produced vacuum packaged fish met import standards and to establish the level of equivalence in imported product. It should be noted that this report did not address the question of whether *C. botulinum* type E was in the New Zealand environment.

The survey focussed on end of the shelf-life tests for total aerobic and anaerobic counts, the presence of presumptive coliforms, faecal coliforms, *Escherichia coli*, coagulase-positive staphylococci, *Salmonella* spp. and *Listeria monocytogenes*. The samples consisted of hot and cold-smoked salmon from 4 local manufacturers and one set of samples from imported cold-smoked salmon to serve as an international control. All samples were stored at 4°C until ± 2 days of their ‘best before/use by’ dates.

Aerobic plate counts were higher for cold smoked product (adjusted mean 7.44 log$_{10}$ organisms/g) than hot smoked product (adjusted mean 5.00 log$_{10}$ organisms/g). Salt (in water phase) concentrations were determined in 4 hot-smoked and 5 cold-smoked samples produced by two New Zealand manufacturers. The results ranged from 0.8% to 2.4%. The mean for the hot-smoked salmon was 1.6% while the mean for the cold-smoked salmon was 1.7%. The report concludes that it is doubtful whether current processes are operating at constant temperatures below 3°C and with salt concentrations “consistently below 3.8%, the products might be at risk regarding *Clostridium botulinum*”. The low salt concentrations found in the Nortje et al. (2001) survey were consistent with a Northland Health survey of smoked fish (Graham and Patterson, 1995). The salt content in 14 samples ranged from 0.8 to 4.1%.

In the outbreak of foodborne botulism type A associated with home-preserved tiroi (made from mussels and watercress) a “very suspicious organism” was later cultured from the remaining food consumed by the cases. However, it could not be confirmed as *C. botulinum* (Flacks, 1985).

5.1.2  *Clostridium botulinum* in the New Zealand aquatic environment

*C. botulinum* Type E is most relevant to this Profile, being found in aquatic environments. Although data are limited, there are none showing that this *C. botulinum* type is present in the New Zealand aquatic environment.

An outbreak of type C botulism in wild waterfowl in 1971 demonstrated that the organism was in New Zealand. This led to an environmental study looking for the presence of *C.
*botulinum* in New Zealand pond and waterway sediments (Gill and Penney, 1982). The samples were incubated and the media tested for toxin. Types C and D were detected in 11 of 20 sites in the Auckland area. Samples from other urban North Island sediments were negative. The authors concluded that although the survey in Auckland was limited, the failure to detect toxins of other types indicates their rarity in New Zealand.

During 1999-2000, Crop and Food Research collected 498 sediments samples from around the New Zealand coastline. The samples were enriched and tested for botulinum biotoxin by mouse bioassay. No positive samples were found. These samples have since been archived at −85°C and are undergoing further testing. This will involve anaerobic enrichment to grow any *C. botulinum* spores, followed by extraction of DNA from the enriched samples and PCR analysis to identify any toxin-producing genes (Graham Fletcher, Crop and Food, personal communication, April 2006).

### 5.1.3 *Clostridium botulinum* in the New Zealand terrestrial environment

The only other research examining *C. botulinum* in the New Zealand environment is on samples from land or animals. Between 1996 and 1998, AgResearch obtained more than 250 isolates of *C. botulinum* from vacuum-packed chilled meats and meat plant environments in South Island, including the hides, faeces and tonsils of slaughtered animals, soil, vegetation and mud. The isolates were all non-proteolytic (Group II) types B, E or F. The typing assignment was based on restriction fragment length polymorphism (RFLP) analysis and DNA sequencing of the 16S rRNA genes. However, none of the isolates carried botulinal neurotoxin genes (Broda *et al.*, 1998).

While the 16S rDNA genes of New Zealand *C. botulinum* isolates share 100% similarity with toxigenic non-proteolytic *C. botulinum* types B, E or F, genomes of some of these isolates hybridise at <60% with that of the reference strains (i.e. has <60% similarity). This means that some New Zealand isolates are true non-toxigenic *C. botulinum* while others need to be classified as different species (Dorata Broda, November 2005, unpublished data). The scientists involved have argued that such non-toxigenic isolates should be renamed to reflect their lack of risk (Broda *et al.*, 2001).

However, in experiments between 2000 and 2002, AgResearch scientists detected fragments of the botulinal neurotoxin genes by PCR in DNA isolated from samples taken from farm environments (without isolation of bacteria). The DNA sequences of the fragments identified them as neurotoxin types B, E or F sequences (Dorata Broda, AgResearch, November 2005, personal communication).

The identification of fragments of B, E and F toxin genes in these samples raises the possibility that Group I (proteolytic) toxin B or F producing *C. botulinum* are present, despite not being identified amongst the isolates obtained in the earlier experiments. Alternatively, the carriage of these toxin genes by *Clostridium* species other than *C. botulinum* (e.g. *C. baratti, C. butyricum*) and/or by another yet un-cultured species may also be possible.
5.2 Food Consumption: RTE Smoked Seafood in Sealed Packaging

Seafood consumption in New Zealand has been summarised in Food Consumption Data for risk assessments (Cressey et al., 2006) based on the National Nutrition Survey. Estimates for the adult population (15+ years) consuming finfish were 18% daily (834/4636), for crustaceans 0.9% (43/4636) and shellfish 2.4% (112/4636). However, only a small proportion of this will be smoked product.

Based on the available data, smoked seafood is rarely consumed by New Zealanders. From the National Nutrition Survey of adults (>15 years) only 30 servings have been identified that can unequivocally be classed as smoked fish, representing 28 consumers (0.6% of study population). The most common type of smoked fish was smoked salmon (17 servings), followed by smoked fish, type not specified (5 servings), smoked hoki (3 servings) and smoked cod and eel (2 servings each). The mean serving size was 71.0 g. All servings, with the exception of two servings of lox (smoked salmon) on bagels, include reference to a cooking method in the food descriptor.

Therefore with 0.6% of respondents consuming an average of 71.0 g serving of smoked seafood, then the population average intake is approximately 0.4 g/person/day, however some caution should be attached to this figure due to relatively small sample numbers (Peter Cressey, personal communication, April, 2006).

Two shellfish servings (two different consumers) refer to smoked mussels - 0.04% of study population. Serving sizes 43.5 and 122 g, with no mention of cooking. No servings of crustacea include reference to smoking.

From the Children’s Nutrition Survey (Ministry of Health, 2003) only three servings specifically mention smoked fish, representing three different consumers (0.09% of study population), serving sizes 118, 22 and 44 g. All mention a cooking step in the food descriptor. There were no mentions of smoked shellfish or crustacea.

In Australia, data from the 1995 National Nutrition Survey also does not distinguish between the two smoked types although the consumption of hot-smoked seafood is believed to be small in comparison to cold-smoked (FSANZ, 2005). In Risk Profiling carried out by Sumner (2002), it was calculated that there were 25 million servings available in Australia, each of 100g, of smoked seafood. This equates to 25% of Australians eating smoked seafoods a few times a year, again the distinction between hot and cold-smoked seafood is not made.

5.3 Qualitative Estimate of Exposure

There are few data on consumption of this food type by New Zealanders, and none on contamination levels. Heat treatment is by definition excluded. Consequently a qualitative estimate of exposure is not useful.
5.4 **Clostridium botulinum** in RTE Smoked Seafood Overseas

5.4.1 Presence of *Clostridium botulinum* in marine environments overseas

Spores of *C. botulinum* type E are widely distributed in the shores and bottom deposits of lakes and coastal waters and occur in intestinal tracts of fish and aquatic animals overseas (ICMSF, 1996). Types A and B are generally found in land environments although they are occasionally found in water.

A summary of the environmental distribution data of *C. botulinum* collected overseas can be found in Hauschild (1989) and Bell and Kyriakides (2000), see Table 3. The results demonstrate that although *C. botulinum* is present in many aquatic environments, their numbers and type vary greatly with the location.

**Table 3: Summary of *C. botulinum* toxin types in overseas aquatic sediments**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of samples positive /No. of samples (%)</th>
<th>Toxin types (% of those identified)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>North America</strong></td>
<td></td>
<td>A  B  C/D  E  F</td>
<td></td>
</tr>
<tr>
<td>East coast</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newfoundland</td>
<td>6/62 (9.7)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Nova Scotia</td>
<td>31/151 (20.5)</td>
<td>0  7  61  32  0</td>
<td></td>
</tr>
<tr>
<td>Gulf of St Lawrence</td>
<td>71/390 (18.2)</td>
<td>0  0  0  100  0</td>
<td></td>
</tr>
<tr>
<td>Gulf of Maine</td>
<td>2/315 (0.6)</td>
<td>0  0  0  100  0</td>
<td></td>
</tr>
<tr>
<td>New York to Florida</td>
<td>12/335 (3.6)</td>
<td>8  17  42  33  0</td>
<td></td>
</tr>
<tr>
<td>West coast</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northwest Alaska</td>
<td>17/23 (73.9)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Alaska</td>
<td>32/66 (48.5)</td>
<td>0  0  0  100  0</td>
<td></td>
</tr>
<tr>
<td>British Columbia</td>
<td>16/257 (6.2)</td>
<td>8  0  0  92  0</td>
<td></td>
</tr>
<tr>
<td>Washington</td>
<td>142/199 (71.4)</td>
<td>1  3  0  96  0</td>
<td></td>
</tr>
<tr>
<td>Oregon</td>
<td>32/92 (34.8)</td>
<td>31  3  0  56  10</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>California (nrth 36°)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Florida to Texas</td>
<td>24/400 (6.0)</td>
<td>12  4  38  46  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Gulf Coast</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alabama</td>
<td>3/74 (4.1)</td>
<td>0  0  0  67  33  0</td>
<td></td>
</tr>
<tr>
<td>Denmark coast</td>
<td>297/445 (66.7)</td>
<td>0  0  0  100  &gt;90  0</td>
<td>Hauschild (1989); Huss (1980)</td>
</tr>
<tr>
<td>Greenland coast</td>
<td>39/105 (37.1)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Faroe Islands coast</td>
<td>1/110 (0.9)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Finland trout farm sediment</td>
<td>?/? (68.0)</td>
<td>0  0  0  100  0</td>
<td>Hielm et al., (1998)</td>
</tr>
</tbody>
</table>

**Europe**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of samples positive /No. of samples (%)</th>
<th>Toxin types (% of those identified)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artic fishing grounds</td>
<td>0/26</td>
<td></td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Baltic sea</td>
<td>7/7 (100)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Denmark coast</td>
<td>297/445 (66.7)</td>
<td>0  0  0  100  &gt;90  0</td>
<td>Hauschild (1989); Huss (1980)</td>
</tr>
<tr>
<td>Greenland coast</td>
<td>39/105 (37.1)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Faroe Islands coast</td>
<td>1/110 (0.9)</td>
<td>0  0  0  100  0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Finland trout farm sediment</td>
<td>?/? (68.0)</td>
<td>0  0  0  100  0</td>
<td>Hielm et al., (1998)</td>
</tr>
<tr>
<td>Location</td>
<td>No. of samples positive /No. of samples (%)</td>
<td>Toxin types (% of those identified)</td>
<td>Reference</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>-------------------------------------------</td>
<td>------------------------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Iceland coast</td>
<td>0/20</td>
<td>A - - B - - C/D - - E - - F -</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Ireland</td>
<td>??/ (18.0)</td>
<td>0 100 0 0 0</td>
<td>Smith et al., (1978)</td>
</tr>
<tr>
<td>Norway coast</td>
<td>9/30 (30.0)</td>
<td>0 0 0 100 0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Scandinavia coast</td>
<td>??/ (79.0)</td>
<td>0 0 0 100 0</td>
<td>Cann et al., (1965)</td>
</tr>
<tr>
<td>Skagerrak, Kattegat Sound</td>
<td>57/114 (50.0) 106/106 (100)</td>
<td>0 0 0 100 0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Asia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bangladesh coast</td>
<td>2/20 (10.0)</td>
<td>0 0 100 0 0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Hokkaido coast</td>
<td>5/50 (10.0)</td>
<td>0 0 0 100 0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Indonesia coast (west)</td>
<td>11/592 (1.9) 10/122 (8.2)</td>
<td>18 36 36 46 0 10 20</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Iran (Caspian Sea)</td>
<td>23/135 (17.0)</td>
<td>0 8 0 92 0</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Java coast</td>
<td>6/46 (13.0)</td>
<td>0 25 63 0 12</td>
<td>Hauschild (1989)</td>
</tr>
<tr>
<td>Thailand (Gulf of Siam)</td>
<td>12/762 (1.6)</td>
<td>0 0 83 17 0</td>
<td>Hauschild (1989)</td>
</tr>
</tbody>
</table>

*C. botulinum* has rarely been detected in marine environments in Australia. Studies suggest that type E is particularly rare. In 1957, Ohye and Scott (cited in Szabo and Gibson, 2003) studied 22 marine muds from Tasmania and NSW. Two (9.1%) of the samples were positive for type B neurotoxin.

Between 1965 and 1970, Christian (cited in Szabo and Gibson, 2003) examined 528 muds, cultivated soils, fish intestines and potato washings from NSW, Tasmania and Queensland. Specifically looking for *C. botulinum* type E, this study did not detect any toxin type in any the samples.

A survey by Gibson *et al.*, (1994) examined 368 samples from ship ballast waters (306 samples), harbour sediments (61 samples), port sediments (17 samples) and estuarine sediments not exposed to commercial shipping (19 samples). This was to determine whether ships that collect ballast water in areas of the world where *C. botulinum* is prevalent, could be carriage vessels for spores that subsequently survive and are then deposited in Australian waters. All samples were negative except for *C. botulinum* type C which was detected in one ballast sample from a ship docked in Queensland.

### 5.4.2 Presence of *Clostridium botulinum* in seafood overseas

Data on the prevalence of *C. botulinum* in seafood overseas have been summarised in Table 4.
Table 4: Summary of *C. botulinum* prevalence and toxin types in overseas seafood

<table>
<thead>
<tr>
<th>Origin</th>
<th>Product</th>
<th>Sample size (g)</th>
<th>% positive samples</th>
<th>MPN per kg</th>
<th>Type(s) identified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Great Lakes, USA</td>
<td>Eviscerated whitefish chubs</td>
<td>10</td>
<td>12</td>
<td>14</td>
<td>E,C</td>
</tr>
<tr>
<td>Atlantic Ocean</td>
<td>Vacuum-packed frozen flounder</td>
<td>1.5</td>
<td>10</td>
<td>70</td>
<td>E^1</td>
</tr>
<tr>
<td></td>
<td>Haddock fillets</td>
<td>-</td>
<td>24</td>
<td>170</td>
<td>E^2</td>
</tr>
<tr>
<td>California</td>
<td>Dressed rockfish</td>
<td>10</td>
<td>100</td>
<td>2400</td>
<td>A,E^1</td>
</tr>
<tr>
<td>Alaska</td>
<td>Salmon</td>
<td>24-36</td>
<td>100</td>
<td>190</td>
<td>A^1</td>
</tr>
<tr>
<td>Alaska</td>
<td></td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>E^2</td>
</tr>
<tr>
<td>Oregon</td>
<td></td>
<td>-</td>
<td>6</td>
<td>-</td>
<td>E^2</td>
</tr>
<tr>
<td>Washington</td>
<td></td>
<td>-</td>
<td>8</td>
<td>-</td>
<td>E^2</td>
</tr>
<tr>
<td>Viking Bank, USA</td>
<td>Vacuum-packed fish</td>
<td>-</td>
<td>42</td>
<td>63</td>
<td>E^1</td>
</tr>
<tr>
<td>Pacific (NW)</td>
<td>Smoked fish (28 processors)</td>
<td>-</td>
<td>5</td>
<td>9</td>
<td>E</td>
</tr>
<tr>
<td>Denmark</td>
<td>Smoked salmon</td>
<td>20</td>
<td>2</td>
<td>&lt;1</td>
<td>B^1</td>
</tr>
<tr>
<td>Caspian Sea</td>
<td>Salted carp</td>
<td>2</td>
<td>63</td>
<td>490</td>
<td>E^1</td>
</tr>
<tr>
<td></td>
<td>Smoked fish</td>
<td>-</td>
<td>0</td>
<td>&lt;68 (sic)</td>
<td>E^2</td>
</tr>
<tr>
<td>Osaka, Japan</td>
<td>Fish and seafood</td>
<td>30</td>
<td>8</td>
<td>3</td>
<td>C,D^1</td>
</tr>
<tr>
<td>Finland (PCR)</td>
<td>438 Raw fish samples (intestines, surface and whole)</td>
<td>-</td>
<td>10 – 40 dependent on fish species</td>
<td>-</td>
<td>E^3</td>
</tr>
<tr>
<td></td>
<td>214 Vacuum-packed retail product**</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>123 air-packed retail product</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>England</td>
<td>Vacuum packed fish</td>
<td>-</td>
<td>0</td>
<td>-</td>
<td>-^2</td>
</tr>
<tr>
<td>England</td>
<td>-</td>
<td>-</td>
<td>&lt;1</td>
<td>-</td>
<td>E</td>
</tr>
<tr>
<td>North Sea</td>
<td>-</td>
<td>-</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Norwegian Sea</td>
<td>-</td>
<td>-</td>
<td>44</td>
<td>-</td>
<td>E</td>
</tr>
<tr>
<td>Indonesia</td>
<td>Fish</td>
<td>-</td>
<td>3</td>
<td>6</td>
<td>A,B,C,D,F^2</td>
</tr>
</tbody>
</table>

(Austin, 2001^1; Dodds, 1993^2; Hyytiä *et al.*, 1998^3)

The high contamination prevalence of *C. botulinum* spores in Baltic Sea sediments (up to 100%) appears to reflected in the various fish species caught in these regions. For example, in the Finland study cited above, contamination levels of vacuum-packed smoked fishery products were as follows;
<table>
<thead>
<tr>
<th></th>
<th>Positive samples</th>
<th>MPN Kg(^{-1}) (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold-smoked rainbow trout</td>
<td>2/64 (3%)</td>
<td>160 (40-290)</td>
</tr>
<tr>
<td>Hot-smoked rainbow trout or salmon</td>
<td>2/50 (4%)</td>
<td>30 (30-40)</td>
</tr>
<tr>
<td>Hot-smoked whitefish</td>
<td>5/50 (10%)</td>
<td>40 (30-60)</td>
</tr>
</tbody>
</table>

The authors concluded that type E posed a serious health risk for those consuming fishery products from the Baltic Sea region (Hyytiä et al., 1998).

In France, a survey was conducted for the presence of *C. botulinum* types A, B and E in raw food materials destined for refrigerated processed foods of extended durability (REPFEDs) (Carlin et al., 2004). 102 samples of fish and shellfish were collected, PCR testing for botulinal toxin genes found 8 were positive; (3 x A, 2 x B, 3 x A and B). Concentration of spores were 2 – 3 /kg. There were no samples positive for type E.

In relation to Australia, Szabo and Gibson (2003) conclude that the area of vacuum packaged seafood and *C. botulinum* has not been studied extensively and there is little public information available on the occurrence of *C. botulinum* in Australian waters, seafood or in seafood processing environments.
6 RISK CHARACTERISATION

6.1 Adverse Health Effects in New Zealand

6.1.1 Incidence

There have been no notifications of human botulism in New Zealand through the surveillance system since records began in 1987.

To date, there has been one published report (Flacks, 1985) involving two patients (sisters) with foodborne botulism in Rotorua, New Zealand. The incident occurred in February 1984 before surveillance records began and both cases were linked to the consumption of home-preserved Tiroi made from watercress and boiled mussels. The husband of one of the sisters also ate the food but developed no symptoms and a third person who ate the food developed only diarrhoea. Botulism was confirmed in the sisters and trivalent ABE antitoxin given intramuscularly. Blood from one of the patients contained type A botulinum toxin. It appears that the boiling of the mussels rather than steaming may have destroyed inoculating fermentative organisms that would have otherwise rendered the food safe (Hudson et al., 2001). A “very suspicious organism” was cultured later from the food but could not be confirmed as \textit{C. botulinum}. The author states that earlier cases of botulism may have gone unrecognised in New Zealand because of the reticence in diagnosing a previously unrecorded disease.

There have not been any releases of botulinal anti-toxins to treat cases of botulism since the Rotorua cases in 1985 (Pam Raynel, Vaccine Supplies, ESR – personal communication, August 2006).

In the ESR 2004 Annual Report (2005), hospital discharge data were cited for one case of botulism in 1989, two cases in 1994 and one case in 1995. Subsequent investigations (conducted for this Risk Profile) with the Ministry of Health and relevant District Health Boards revealed that these records were miscoded.

6.2 Adverse Health Effects Overseas

6.2.1 Incidence

Reported cases of botulism in developed countries are rare, as shown in Table 5. The number of foodborne botulism cases during the period 1988 to 1998 in Europe has been compiled by Eurosaurveillance (1999) where most cases occurred in Germany and Italy.
Table 5: Comparison of foodborne botulism incidence between countries

<table>
<thead>
<tr>
<th>Country</th>
<th>Period</th>
<th>Rate</th>
<th>No. of cases</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canada*</td>
<td>1995</td>
<td></td>
<td>15</td>
<td>Health Canada, 2006</td>
</tr>
<tr>
<td></td>
<td>1996</td>
<td></td>
<td>18</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1997</td>
<td></td>
<td>19</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1998</td>
<td></td>
<td>7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1999</td>
<td></td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td></td>
<td>4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2001</td>
<td></td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2002</td>
<td></td>
<td>10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2003</td>
<td></td>
<td>6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2004</td>
<td></td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Denmark</td>
<td>1984 - 89</td>
<td>-</td>
<td>16 cases (11 outbreaks, 12 fatalities) 100% E</td>
<td>Gelli et al., 2002</td>
</tr>
<tr>
<td>England and Wales</td>
<td>1999</td>
<td></td>
<td>0</td>
<td>WHO (2000)</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td></td>
<td>28</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1978-1989</td>
<td></td>
<td>304 cases (in 175 outbreaks, 2 fatalities) 2% E</td>
<td>Gelli et al., 2002</td>
</tr>
<tr>
<td>Germany*</td>
<td>1999</td>
<td>0.023</td>
<td>19</td>
<td>WHO (2000)</td>
</tr>
<tr>
<td></td>
<td>2000</td>
<td>0.013</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>USA</td>
<td>1899-1995</td>
<td>-</td>
<td>2,444 cases in 1026 outbreaks, 1,040 fatalities</td>
<td>Gelli et al., 2002</td>
</tr>
<tr>
<td></td>
<td>1971-1989</td>
<td>-</td>
<td>597 cases (in 272 outbreaks, 11 fatalities) 17% type E</td>
<td>Gelli et al., 2002</td>
</tr>
<tr>
<td></td>
<td>2003</td>
<td></td>
<td>Approx. 1 in 1,800,000</td>
<td>CDC, 2005</td>
</tr>
</tbody>
</table>

*Botulism not distinguished by clinical form therefore could include infant, wound etc.

Food-borne botulism cases recorded in 38 countries between 1951 and 1989 have been reviewed (WHO, 1999). This shows that 72% of outbreaks and 48% of cases were reported from Poland where home-preserving is common. The toxin type was determined in 2622 outbreaks during this period, the findings were 34% type A, 52% type B, and 12% type E. The rarity of type F foodborne botulism was confirmed, only two incidents during this period were assigned to this toxin type.

In Canada, Alaska, Scandinavia and Northern Japan, food-borne botulism outbreaks involving fish, particularly traditional native dishes, have been reported. Most of these outbreaks have been linked to type E neurotoxin (Austin, 2001). Specific reported foodborne botulism cases associated with seafood are summarised in Table 6.
Table 6: Reported foodborne botulism cases associated with seafood overseas

<table>
<thead>
<tr>
<th>Country</th>
<th>Period</th>
<th>Usual toxin type</th>
<th>No. of cases</th>
<th>Food type</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Egypt</td>
<td>1991</td>
<td>E</td>
<td>91 (hospital)</td>
<td>Uneviscerated salted mullet fish</td>
<td>Weber et al., 1993</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>18 fatal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Finland</td>
<td>2006</td>
<td>E</td>
<td>1</td>
<td>Vacuum-packed smoked whitefish</td>
<td>Lindström et al., 2006</td>
</tr>
<tr>
<td>Germany (imported fish from Finland)</td>
<td>1997</td>
<td>E</td>
<td>2</td>
<td>Hot-smoked Vacuum-packed whitefish</td>
<td>Korkeala et al., 1998</td>
</tr>
<tr>
<td>Japan</td>
<td>1951 - 1987</td>
<td>E</td>
<td>479</td>
<td>Fish or fish products</td>
<td>Hauschild, 1993</td>
</tr>
<tr>
<td>Russia</td>
<td>2004</td>
<td>?</td>
<td>35 (2 fatal)</td>
<td>Fish</td>
<td>ProMED-mail, 2005</td>
</tr>
<tr>
<td></td>
<td>2005</td>
<td>?</td>
<td>6</td>
<td>Salted fish</td>
<td></td>
</tr>
<tr>
<td>USA</td>
<td>1987</td>
<td>E</td>
<td>8 (1 fatal)</td>
<td>Uneviscerated salt-cured, air-dried fish</td>
<td>Bell and Kyriakides, 2000</td>
</tr>
<tr>
<td>USA</td>
<td>1985</td>
<td>E</td>
<td>2 (both fatal)</td>
<td>Uneviscerated salt-cured, air-dried fish</td>
<td>Badhey et al., 1986</td>
</tr>
</tbody>
</table>

The Australian Surveillance body, OzFoodnet, has not reported any cases of foodborne *C. botulinum* since its inception in 2000. One single Australian case of foodborne botulism (associated with home preserved asparagus) was reported in 1992 (Paterson et al., 1992), which was the first recorded episode there since 1981. During the period 1942 – 83 there were five reported outbreaks of botulism in Australia (Hauschild, 1993). One of the outbreaks involving two cases was linked to the consumption of Australian canned tuna (Murrell, 1979).

6.2.2 Contributions to outbreaks and incidents

The importance of home food preservation as a risk factor for outbreaks of botulism is shown by the data shown in Table 7 (Hauschild, 1993, cited in EFSA, 2004). Meats are the most common food type, with fish and fruits/vegetables following.
Table 7: Food vehicles and preparation methods associated with outbreaks of botulism

<table>
<thead>
<tr>
<th>Country</th>
<th>Number of outbreaks with food identified</th>
<th>Fish (%)</th>
<th>Meats (%)</th>
<th>Fruit/vegetables (%)</th>
<th>Other* (%)</th>
<th>Home-prepared (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argentina</td>
<td>14</td>
<td>21</td>
<td>29</td>
<td>36</td>
<td>14</td>
<td>79</td>
</tr>
<tr>
<td>Belgium</td>
<td>8</td>
<td>12</td>
<td>75</td>
<td>0</td>
<td>62</td>
<td>38</td>
</tr>
<tr>
<td>Canada</td>
<td>75</td>
<td>20</td>
<td>72</td>
<td>8</td>
<td>0</td>
<td>96</td>
</tr>
<tr>
<td>Czechoslovakia</td>
<td>14</td>
<td>7</td>
<td>72</td>
<td>14</td>
<td>7</td>
<td>100</td>
</tr>
<tr>
<td>Germany</td>
<td>- East</td>
<td>31</td>
<td>26</td>
<td>52</td>
<td>19</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>- West</td>
<td>55</td>
<td>13</td>
<td>78</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Poland</td>
<td>1500</td>
<td>12</td>
<td>83</td>
<td>5</td>
<td>0</td>
<td>75</td>
</tr>
<tr>
<td>USA</td>
<td>222</td>
<td>17</td>
<td>16</td>
<td>59</td>
<td>9</td>
<td>92</td>
</tr>
</tbody>
</table>

* includes mixed vehicles

In the USA, among the 294 outbreaks during the period 1973 to 1997, 47 (16%) outbreaks were due to fish (Gelli et al., 2002).

6.2.3 Case control studies

The largest documented outbreak of type E foodborne botulism (Weber et al., 1993) in Cairo, Egypt led to a case control study being undertaken. 45 patients were interviewed with an investigation subsequently conducted among five families of hospitalised patients. Illness was associated with eating unviscerated salted mullet fish, (95% CI = 6.6, p < 0.001).

6.2.4 Risk assessment and other activity overseas

Food Standards Australia New Zealand (FSANZ) has prepared a Primary Production and Processing Standard for Seafood (Proposal P265). In this document risk assessments from Australia on C. botulinum in smoked seafood were discussed (FSANZ, 2005:p.247).

The Food Standards Australia New Zealand Assessment Report included risk assessments on C. botulinum in hot and cold smoked finfish products. The FSANZ conclusion was that foodborne botulism was a severe illness, the likelihood of occurrence was “unlikely”. The assessment stated that for ready-to-eat cold smoked fish products C. botulinum spores and vegetative cells are likely to survive the cold-smoking process, but growth is unlikely except in the case of vacuum or modified atmosphere packed products. Even in the case of such products, salt concentration (5% or more) are likely to inhibit growth and toxin production.

For hot smoked products, the assessment stated that spores will survive the smoking process, but again growth is unlikely except in the reduced oxygen packed products. Even in the case of such products, salt concentrations (3.5% or more), processing to an internal temperature of greater than 63°C for at least 30 minutes, and proper storage at less than 5°C will inhibit growth and toxin production.
The “medium” relative risk ranking assigned for *C. botulinum* in both these products reflected the current risk management including industry inherence to good manufacturing practice, good hygiene practice and appropriate product formulation (e.g. pH, levels of salt, preservatives) (FSANZ, 2005). As *C. botulinum* mostly occurred in the gut of fish, the risk was mostly from fish that were not eviscerated before smoking. The hazard identification section (p 294-6) also referred to surveys of Australian marine samples which failed to find *C. botulinum* Type E.

The Food Standards Australia New Zealand review cited specific risk assessments by Ross and Sanderson (2000) and Sumner (2001). Ross and Sanderson (2000) were commissioned by Safe Food Production, New South Wales (NSW) to carry out a Risk Assessment of selected seafoods in NSW. They considered the risk to New South Wales consumers from *C. botulinum* in vacuum-packed ready-to-eat fish products was ‘relatively low’, and would only result from gross temperature abuse.

Sumner (2001) compiled a Seafood Food Safety Risk Assessment for Seafood Services Australia Ltd and concluded that the risk of *C. botulinum* in smoked vacuum-packed fish was “negligible”. This was based on the low levels of spores likely to be in products available in the Australian marketplace and the typical salt levels in these products.

For Australia, a semiquantitative seafood safety risk assessment (Sumner and Ross, 2002) assessed a range of hazard/product combinations. Scores for *C. botulinum* in canned fish and vacuum packed smoked fish were 25 and 28 respectively. Risk ratings occurred on a scale of 0-100 (0 =no risk, 100 = everybody eating a meal containing a lethal dose of the hazard every day). A “low” risk equated to a score of <25, “medium” to 26-40 and “high” >40. Because the scale is logarithmic, an increment of 6 in the ranking relates to a 10-fold increase in risk. The *C. botulinum* risks therefore fell into the lowest ranked category.

6.3 Qualitative Estimate of Risk

The available data all support a very low risk for *C. botulinum* in vacuum packed smoked seafood. This food type has a low consumption, and studies have found no evidence for *C. botulinum* with the potential to affect humans in the New Zealand aquatic environment. The amount of imported product is low, and controls relevant to *C. botulinum* control have been specified for such imports.

6.4 Risk Categorisation

Botulism was included in the severity rating in the risk categorisation system in the Appendices to other Risk Profiles. The proportion of severe outcomes (hospitalisation, long term sequelae, and death) resulting from foodborne botulism is approximately 80% for hospitalisation, with a 5 – 15% mortality rate, placing this disease in the highest severity category.

Given the rarity of mortality linked to foodborne botulism in New Zealand in the last twenty years, obviously the incidence category would be the lowest.
7  RISK MANAGEMENT INFORMATION

7.1  Relevant Food Controls

In the USA, the USFDA recommendation is that the hot-smoking process destroys *C. botulinum* type E by ensuring a core temperature of 62.5°C for 30 minutes. In addition, for vacuum packaged or modified atmosphere packaged product, a high salt content of at least 3.5% in aqueous phase, or else 3.0% aqueous salt and 100 – 200mg/kg sodium nitrite are required (FDA, 1995).

These processes produce product that has been described as generally unacceptable to New Zealand consumers. The majority of New Zealand processors do not use high smoking temperatures to control *C. botulinum* because the assumption is made that *Clostridium botulinum* type E spores are unlikely to be present, and that by storing product below 3°C, the hazard is negligible. This view is reinforced by the lack of notified foodborne type E botulism cases in products made by New Zealand processors (Fletcher *et al.*, 2003).

These comments were made in guidelines for the safe preparation of hot-smoked seafood in New Zealand (Fletcher *et al.*, 2003). The guidelines were produced in consultation with the seafood industry and are intended to provide seafood processing companies with practical advice.

The guidelines describe the reasons why hot-smoked products may pose a risk and lists *C. botulinum* as one of the three most important food-borne illnesses associated with the hot-smoked products. Hot-smoking time and temperature combinations are given for smoked mussels, salmon, cod and others and are aimed at achieving a 12 D-value listericidal process rather than destruction of *C. botulinum* Group II spores. A pellicle (skin) forms with heat which can prevent smoke penetration through the product therefore it is recommended that smoke is applied at the beginning of the thermal process.

The hot smoking times and temperatures in this report are less than required by the US FDA; for example at 63°C hot smoking times of 12-26 minutes are recommended for the various products.

In addition, a factsheet produced by Crop and Food (Bremer *et al.*, 2003) discusses *C. botulinum* in seafood. Type E is acknowledged as being of most concern to MAP packaged seafood producers but again asserts that the bacterium has not been isolated in the New Zealand marine or terrestrial environment so is difficult to clearly define its risk. The authors conclude that the absence of associated botulism incidences must indicate a very low risk however strict regimes should be adopted to limited potential contamination and ensure products are adequately chilled. The trend towards the use of less preservatives and milder heat to produce more “natural” foods is noted and super-chilled temperatures below 2.8°C are suggested to ensure food safety. Where super-chilled temperatures can not be guaranteed over the shelf-life of the product the authors suggest that additional preservative measures such as salting, smoking, hot processing or the addition of preservatives such as nitrite must be used. [Note: the use of nitrites and nitrates are not permitted under the Australia New Zealand Food Standards Code for semi-preserved fish, see sections 3.3.3 and 7.1.2].
A Guide to HACCP and seafoods has been published by MAF (available at the following website address),

A number of HACCP plan examples are worked through under Appendix II, and Plan II.4 concerns hot smoked mussel meat. Three initial Food Safety Objectives are stipulated for the product;

1. To achieve a *Listeria monocytogenes* free product,
2. To ensure that temperature controls are less than 3.3°C in order to prevent *Clostridium botulinum* toxin production taking place, and
3. To minimise the presence of shell pieces.

An important statement is made regarding *Clostridium botulinum* in part 7 of the HACCP plan. The footnote states that

“There is currently no published evidence of *Clostridium botulinum* type E being found in New Zealand seafood. Other relevant *Clostridium botulinum* types are not considered reasonably likely to occur.” (MAF, 1997).

It should be noted that these guidelines were written in June 1997. More recent publications for example Graham *et al* (1997), detected growth of non-proteolytic *C. botulinum* and toxin production at 3°C.

Background information to the HACCP plan for hot smoked mussel meat includes a short paragraph on *Clostridium botulinum*. It states that non-proteolytic strains of *C. botulinum* are most commonly associated with the marine environment however this toxin type has not been found in New Zealand waters. The statement points out that the two food-borne botulism cases in New Zealand were toxin type A (associated with home-preserved mussels and watercress in tiroi) and the study by Gill and Penney found *C. botulinum* types that are not toxic to humans.

7.1.1 **Animal Products Act 1999**

Risk Management Programmes (RMPs) are part of the emerging food assurance system in New Zealand. They form part of the Animal Products Act (APA) 1999. These will eventually be aligned with the Food Safety Programmes (FSPs) required by the Food Act 1981.


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, with further detail being provided in specifications.
By 1 July 2006, all animal product primary processing businesses, except those exempt under the Act or under the Animal Products (Exemptions and Inclusions) Order 2000, were required to have a risk management programme. A risk management programme is a documented programme to identify and manage biological, chemical and physical hazards. The programme is to be based on the principles of Hazard Analysis and Critical Control Point (HACCP): identifying the hazards, the systems of control, and demonstrating that the controls are effective. Risk management programmes are to be designed by individual businesses for the animal materials used, the processes performed and the product range produced.

7.1.2 Control during or after processing

The interplay between salt, temperature and smoke and their inhibitory effects in smoked seafood is complex. The balance between these hurdles must be sufficient to prevent growth of *C. botulinum* type B, E and F. These include;

- Thorough and hygienic evisceration of fish before processing,
- Control over the amount of salt in the finished product in combination with other hurdles such as heat damage and competitive bacteria to prevent growth of non-proteolytic types especially type E, and
- Restricting the amount of time food is exposed to temperatures favourable for growth and toxin formation during processing and storage, (USFDA, 2001).

In addition, raw and ready-to-eat food processing areas must be kept strictly separate and handling procedures designed so that cross contamination does not occur.

Preservatives which are inhibitory to *C. botulinum* such as nitrite are permitted in the USA, however in the Australia New Zealand Food Standards Code, sodium and potassium nitrite and nitrate salts are not on the list of permitted additives.

The Australia New Zealand Food Standard can be found under 1.3.1, Schedule 1, under part 9.3, semi-preserved fish and fish products.

If a product has the required NaCl concentration and an APC indicating that product has not been temperature abused, this could be taken as an indication that the product is safe (Hudson, 1997).

In New Zealand the Microbiological Reference Criteria (Ministry of Health, 1995) for ready-to-eat foods (with some components not cooked during manufacture, which best describes smoked seafood), the APC criteria is \( n = 5, c = 2, m = 10^5 \) and \( M = 5 \times 10^5 \). For a *C. perfringens* count (for sulphite reducing anaerobes), the criteria gives \( m = 10^2 \) and \( M = 10^3 \) which Hudson (1997) proposes as possibly valid when applied to vacuum packaged smoked fish but needs further analysis.
7.1.3 Consumers

Continuation of refrigerated storage for vacuum packed smoked seafood in the domestic kitchen will be an important control point. Although toxins of *C. botulinum* are relatively sensitive to heat (inactivated by 80°C for 10 minutes or equivalent), consumers are unlikely to heat foods of this type before consumption.

7.1.4 Environment

The absence of *C. botulinum* in the marine environment is very important to the New Zealand seafood industry. To help maintain this, controls have been put into place by the Ministry of Fisheries (Import Health Standard - Biosecurity Act 1993) to prevent ballast waters, tank sediments and the cleaning of hulls from overseas vessels from contaminating our waters. Due to the predatory nature of the North Pacific Starfish on New Zealand’s indigenous shellfish population, under no circumstances are ballast waters from Tasmania or Port Philip Bay, Victoria, Australia to be discharged into New Zealand waters.

The Ministry of Fisheries biosecurity measures to protect against ballast water and tank sediments can be found at the following website address; [http://www.fish.govt.nz/sustainability/biosecurity/ballastwater.html#](http://www.fish.govt.nz/sustainability/biosecurity/ballastwater.html#)

7.1.5 Risk management options/studies overseas

A list of international and USA regulations and guidelines for seafood can be found on the following website address: [http://seafood.ucdavis.edu/guidelines/international.htm](http://seafood.ucdavis.edu/guidelines/international.htm) and [http://seafood.ucdavis.edu/guidelines/usguidelin.htm](http://seafood.ucdavis.edu/guidelines/usguidelin.htm).

**Australia**: A Proposal for a Primary Production and Processing Standard for Seafood (P265) was first raised by Food Standards Australia New Zealand in December 2002. The final Standard (FSANZ, 2005) will apply to Australia only and not to New Zealand (Standard 4.2.1). The Standard comes into effect on 26 May 2006. The proposal is for a set of national standards to protect public health and safety by implementing a set of basic safety provisions for medium and low risk categories, and specific measures to manage higher risk products (particularly oysters and bivalve molluscs). As described in Section 6.2.4, *C. botulinum* in cold or hot smoked fish products falls into the medium risk category.

A list of the Codes of Practice for Safe Seafood Production in Australia has also been compiled in Attachment 8 (FSANZ, 2005).


**USA**: The USFDA (2001) require that the smoker temperature in cold-smoked fish in reduced oxygen packaging must not exceed 32.2°C (90°F). For hot-smoked fish in the same packaging;

- The internal temperature of the fish must be maintained at or above 62.8°C throughout the fish for at least 30 minutes
Not less than 3.5% water phase salt in the loin muscle, or, where permitted, the combination of 3.0% water phase salt in the loin muscle and 100 – 200 ppm nitrite,

The product must not be exposed to temperatures above 10°C for more than 12 h nor to temperatures above 21.1°C for more than 4 h, excluding time above 60°C,

The product must not be exposed to storage temperatures above 10°C, which may be assured by;

- A maximum cooler temperature of 10°C, and/or
- The presence of sufficient cooling media (e.g. adequate ice to completely surround the product,

The product must not be exposed during transportation to temperatures above 10°C, which may be assured by

- A maximum refrigerated container temperature of 10°C throughout transit, or
- The presence of sufficient cooling media (e.g. adequate ice to completely surround the product) upon receipt.

7.2 Economic Costs

No estimates for the cost of botulism for New Zealand are available. Overseas studies indicate the high cost of this severe illness.

Infant botulism hospitalisation has been reported in up to 80% of cases with the average stay approximately 4 to 5 weeks but differs with toxin type. Type A cases have a mean hospitalisation time of 5.4 weeks because it is more severe, whereas type B are 3.8 weeks (Midura, 1996). In California, for the year 1990, mean hospital costs exceeded SUS 80,000 per case. Overall, the most protracted illness in 1988, where the patient was hospitalized for 10 months, cost more than SUS 635,000 (Midura, 1996), equivalent to SUS 890,000 at 1993 prices (Arnon, 1995).

7.3 Other Transmission Routes

7.3.1 Other transmission routes: food

Type E foodborne botulism is associated with fish. Foodborne botulism types A and B have been associated mainly with meat and poultry products, although foods as diverse as fresh-cut packaged vegetables, cooked vegetables, fresh pasta, melons, sautéed onions, shredded cabbage, home-preserved asparagus, bottled chopped garlic, peyote, baked potato, fried lotus rhizome in mustard, home canned bamboo shoots have also been identified (EFSA, 2004). Many surveys overseas have identified *C. botulinum* spores in food, especially meats, honey and vegetables/mushrooms. A collation of these surveys can be found in Table 8.
### Table 8: Prevalence of *C. botulinum* spores in food surveys worldwide

<table>
<thead>
<tr>
<th>Product</th>
<th>Origin</th>
<th>Sample size (g)</th>
<th>% positive samples</th>
<th>MPN per kg</th>
<th>Type(s) identified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raw meat</td>
<td>North America</td>
<td>3</td>
<td>&lt;1</td>
<td>0.1</td>
<td>C</td>
</tr>
<tr>
<td>Cured meat</td>
<td>Canada</td>
<td>75</td>
<td>2</td>
<td>0.2</td>
<td>A</td>
</tr>
<tr>
<td>Raw pork</td>
<td>U.K.</td>
<td>30</td>
<td>0-14</td>
<td>&lt;0.1-5</td>
<td>A,B,C</td>
</tr>
<tr>
<td>Cooked, vacuum-packed potatoes</td>
<td>The Netherlands</td>
<td>-</td>
<td>0</td>
<td>0.63</td>
<td>-</td>
</tr>
<tr>
<td>Mushrooms</td>
<td>Canada</td>
<td>-</td>
<td>-</td>
<td>2100</td>
<td>B</td>
</tr>
<tr>
<td>Random samples*</td>
<td>USA</td>
<td>30</td>
<td>1</td>
<td>0.4</td>
<td>A,B</td>
</tr>
</tbody>
</table>

Source: (Austin, 2001)

#### 7.3.2 Other transmission routes: environment

*C. botulinum* occurs commonly in soil, dust and aquatic environments worldwide (EFSA, 2004). This may result in the ingestion of spores through ingestion of these materials directly, or by contamination of foods by these materials. An example is contaminated honey causing infant botulism.
8 CONCLUSIONS

8.1 Description of Risks to Consumers

8.1.1 Risks associated with smoked seafood in sealed packaging

As stated in Section 6.3 the available data all support a very low risk of botulism for consumers of New Zealander vacuum packed smoked seafood. This food type has a low consumption, and studies have found no evidence for *C. botulinum* with the potential to affect humans in the New Zealand aquatic environment. The amount of imported product is low, and controls relevant to *C. botulinum* control have been specified for such imports.

The controls required for *C. botulinum* in seafood are well understood, and have been described in a draft Codex standard. They involve primarily a combination of storage temperature and salt concentrations to reduce water activity. Smoking is not regarded as a primary control, although cold-smoking allows the survival of competing microflora which will inhibit growth and/or cause spoilage, and hot smoking times and temperatures will destroy vegetative cells, as well as Group II spores. Smoking will also contribute to water activity reduction.

There is no current requirement for New Zealand smoked seafood manufacturers to meet overseas requirements in respect of salt concentration to control *C. botulinum*. A survey of locally produced smoked salmon found salt levels below those recommended in the scientific literature for *C. botulinum* control in non-frozen finfish.

New Zealand smoked seafood producers do not use salt as a critical control point (Graham Fletcher, Crop and Food Ltd, personal communication, April 2006).

This means that the presence or absence of type E *C. botulinum* in the New Zealand aquatic environment is an important point. The more extensive testing planned by Crop and Food Research on the marine sediment samples will contribute valuable evidence.

8.1.2 Risks associated with other foods

Cases of notified foodborne botulism in New Zealand are extremely rare making it difficult to comment on the risks associated with other foods. The only documented outbreak occurred just over twenty years ago and was associated with tiroi and type A neurotoxin. The absence of notified cases suggests that either the spores are not present in the foodstuffs consumed in New Zealand or that the controls in place during food processing are sufficient to prevent botulinal neurotoxin formation.

Anecdotal observation suggests that a wider variety of preserved foods under anaerobic conditions are being imported, and given the predominance of home-preserved foods in outbreaks overseas, this may be an emerging risk of botulism for New Zealanders.
8.1.3 **Quantitative risk assessment**

At this stage a quantitative risk assessment on this food:hazard combination would not be useful.

8.2 **Commentary on Risk Management Options**

The risk management options for *C. botulinum* in smoked seafood are well understood. The question is whether these controls need to be implemented, given New Zealand’s environmental status for *C. botulinum*. Better information on actual processing conditions by New Zealand manufacturers would assist in understanding the hurdles currently being achieved in local product.

Despite evidence from surveys that have not found *C. botulinum* in Australian marine samples, Food Standards Australia New Zealand appear to rely on hot-smoking temperatures, storage temperatures and salt levels as described by the US FDA and Codex to control this organism (FSANZ, 2005).

8.3 **Data gaps**

Conducting surveys of seafood for *C. botulinum* is unlikely to be useful given that contamination is almost certainly absent. The data gaps identified in this Risk Profile are:

- Status of controls for *C. botulinum* in cold and hot smoked vacuum packed seafood in New Zealand; this includes information on salt levels, storage temperatures and shelf lives.
- Processing conditions, product types and ingredients used by New Zealand manufacturers of smoked seafood.
9 REFERENCES


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