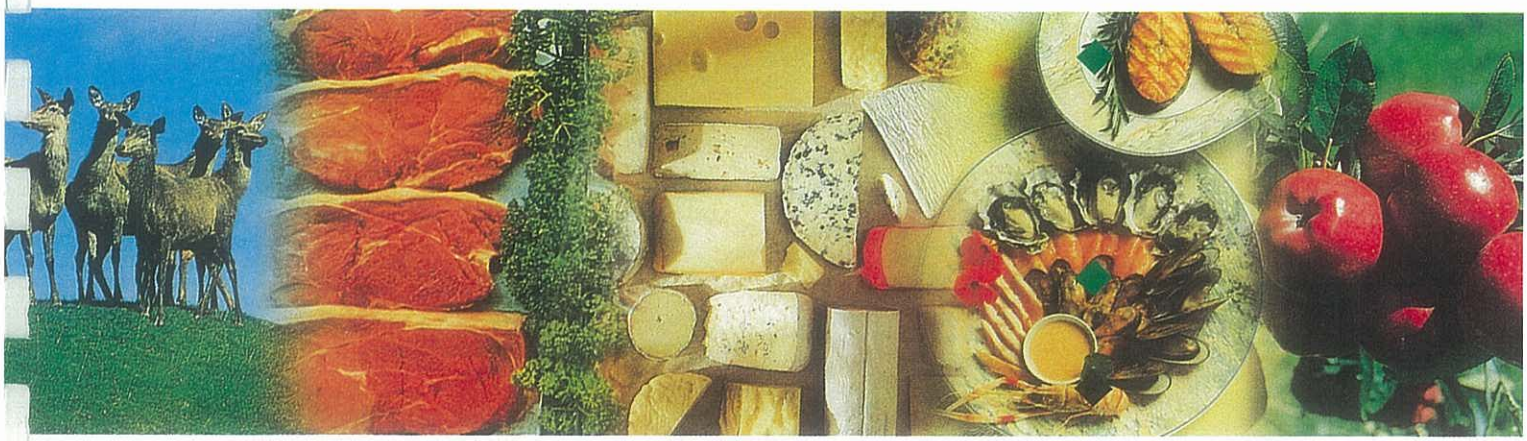
MINISTRY OF AGRICULTURE AND FISHERIES NEW ZEALAND
TE MANATU AHUWHENUA AHUMOANA AOTEAROA



THE RISK OF INTRODUCING EXOTIC DISEASES OF FISH INTO NEW ZEALAND THROUGH THE IMPORTATION OF OCEAN-CAUGHT PACIFIC SALMON FROM CANADA

MAF Regulatory Authority
(Animal Health and Welfare)

By:
Stuart C MacDiarmid
National Manager
(Agricultural Security and Animal Health)

Ref; I-CAN-135

THE RISK OF INTRODUCING EXOTIC DISEASES OF FISH INTO NEW ZEALAND THROUGH THE IMPORTATION OF OCEAN-CAUGHT PACIFIC SALMON FROM CANADA

STUART C MACDIARMID
REGULATORY AUTHORITY
SEPTEMBER 1994

TABLE OF CONTENTS

Summary 1

PART I: QUALITATIVE RISK ANALYSIS 3

Introduction 3
The commodities being assessed 4
Marker diseases 5
Salmonid diseases of concern 9

Specific diseases of salmon 17
Infectious pancreatic necrosis (IPN) 17
freedom from IPN virus 17
Infectious haematopoietic necrosis (IHN) 19
Viral haemorrhagic septicaemia (VHS) 21
Viral erythrocytic necrosis (VEN) 22
Erythrocyte inclusion body syndrome (EIBS) 23
Plasmacytoid leukaemia 24
Pancreas disease 25
Furunculosis 26
very uncommon 26
susceptibility of *A. salmonicida* to freezing 30
dose of *A. salmonicida* required to infect 33
Survival of *A. salmonicida* 33
host range of *A. salmonicida* 35
Bacterial kidney disease (BKD) 36
Enteric redmouth (ERM) 43
Salmonid rickettsial septicaemia 45
Rosette agent 46
Loma salmonae 46

Enterocytozoon salmonis 47
Edwardsiellosis 47
Proliferative kidney disease (PKD) 48
Kudoa thyrsites 49
Ceratomyxa shasta 49
Herpesvirus salmonis 50
Vibriosis 50
Hitra disease 51
Henneguya salmonicola 51
Parvicapsular disease 51

PART II: QUANTITATIVE RISK ASSESSMENT 53

Furunculosis 53
Why furunculosis? 54
"Atypical" *Aeromonas salmonicida* 54
The method 55
The major areas of perceived risk 56

Chilled, eviscerated, headless fish 58
The probability of importing contaminated, potentially infective fish 58
The evidence 59
Proportion of diseased fish per year 59
The probability of accepting a diseased fish for processing .. 60
The extent to which evisceration reduces the risk 60
The extent to which inspection and grading reduces the risk 61
The extent to which washing reduces the risk 62
Mean weight of ocean caught salmon 67
Percent remaining after evisceration and removal of the head 68
Simulation results 70

The number of fish represented/tonne of headless, eviscerated salmon 70
The number of *diseased* fish represented/tonne 71
The probability of *importing* contaminated fish, without freezing 72
Distribution of imported fish within the country 73
The evidence 75
The distribution of salmonid fish stocks within the country 76
The evidence 76
The probability that scraps will introduce infection 78
Region 1, scraps introduce infection 79
The evidence 81
Proportion disposed of as scraps 81
Proportion not incinerated 81
Proportion not buried 82
Contaminate fresh water? 86
Infect a susceptible host when present? 89
Contaminate estuarine water? 90
Infect a susceptible host when present? 90
Contaminate sea water? 90
Infect a susceptible host when present? 90
Simulation results 90
Region 2, scraps introduce infection 92
The evidence 93
Simulation results 94
Region 3, scraps introduce infection 95
The evidence 96
Simulation results 96
Cumulative risk of scraps introducing infection 98
The probability that wrapping will introduce infection 99
Region 1, wrapping introduces infection 100
The evidence 102

The probability that wrapping is contaminated 102
Proportion of wrapping not incinerated or buried 102
Contaminate fresh water? 102
Infect a susceptible host when present? 102
Contaminate estuarine water? 103
Infect susceptible host when present? 103
Contaminate sea water? 103
Infect susceptible host when present? 103
Simulation results 103
Region 2, wrapping introduces infection 104
The evidence 105
Simulation results 106
Region 3, wrapping introduces infection 107
The evidence 108
Simulation results 108
Cumulative risk of wrapping introducing infection 109
The probability that waste water will be the vehicle by which infection is introduced 110
The evidence 111
Proportion of sewage NOT treated adequately 111
Proportion of sewage discharging into fresh water 112
Region 1, waste water introduces infection 112
The evidence 114
If fish contaminated probability that kitchen waste water is contaminated? 114
Infect susceptible host when present? 115
Simulation results 119
Region 2, waste water introduces infection 120
The evidence 120
Simulation results 121
Region 3, waste water introduces infection 122
The evidence 122

Simulation results 122
Cumulative risk of waste water introducing infection 123
The combined probability of infection being introduced through chilled headless, eviscerated Pacific salmon 125

Frozen headless, eviscerated fish 127
The probability of importing contaminated, potentially infective fish 127
The evidence 128
The extent to which freezing reduces the risk 128
susceptibility of *A. salmonicida* to freezing 128
Simulation results 128
The number of fish represented/tonne of headless, eviscerated salmon 128
The number of *diseased* fish represented/tonne 128
The probability of *importing* contaminated fish, with freezing 129
Distribution of imported fish within the country 130
The distribution of salmonid fish stocks within the country 130
The probability that scraps will introduce infection 131
The evidence 131
Simulation results; cumulative risk of scraps introducing infection 131
The probability that wrapping will introduce infection 132
The evidence 133
Simulation results; cumulative risk of wrapping introducing infection 133
The probability that waste water will be the vehicle by which infection is introduced 134
The evidence 135
If fish contaminated, probability that kitchen waste water is contaminated? 135
Infect susceptible host when present? 135
Simulation results; cumulative risk of waste water introducing infection ... 135

The combined probability of infection being introduced through frozen, headless, eviscerated Pacific salmon 137

Chilled skinless fillets 139
The probability of importing contaminated fish 139
The evidence 139
The extent to which evisceration and filleting reduces the risk 139
The extent to which inspection and grading reduces the risk 140
The extent to which washing reduces the risk 140
Percent recovered as fillets 140
Simulation results 141
The number of fish represented/tonne of fillets 141
The number of *diseased* fish represented/tonne 142
The probability of *importing* contaminated, potentially infective fillets 143
The probability that scraps will introduce infection 145
The evidence 145
Proportion disposed of as scraps 145
Simulation results 146
The probability that wrapping will introduce infection 147
The probability that waste water will be the vehicle by which infection is introduced 147
The evidence 147
If fillets contaminated probability that kitchen waste water is contaminated? 147
Cumulative risk of waste water introducing infection 148
The combined probability of infection being introduced in chilled, skinless fillets 149

Frozen skinless fillets 151

The probability of *importing* contaminated fish 151
Cumulative risk of scraps introducing infection 153
Cumulative risk of wrapping introducing infection 154
Cumulative risk of waste water introducing infection 155
The combined probability of infection being introduced/tonne of fillets ... 156

Conclusions 158

Acknowledgements .....

THE RISK OF INTRODUCING EXOTIC DISEASES OF FISH INTO NEW ZEALAND THROUGH THE IMPORTATION OF OCEAN-CAUGHT PACIFIC SALMON FROM CANADA

Entia non sunt multiplicanda praeter necessitatem[1]

1. Summary

A qualitative risk analysis of the risks of introducing exotic fish diseases through importations of headless, eviscerated, wild, ocean-caught Pacific salmon from Canada demonstrated that none of 23 diseases of salmonids present in North America is likely to be introduced into New Zealand should imports be permitted.

For table fish to serve as a vehicle for the introduction of fish disease, a number of criteria must be met;

- The disease must be present in the waters of origin.
- The disease must be present in the particular fish caught (or the flesh must have become contaminated during processing).
- The pathogen must be present in the imported tissues.
- The diseased flesh must pass inspection and grading procedures.
- The pathogen in the flesh must survive storage and processing and be present at an infectious dose.
- The pathogen must be able to establish infection by the oral route or by the host being bathed in it.
- Scraps of the flesh product must find their way into a susceptible fish host in New Zealand or an infectious dose of pathogen must find its way into contact with a susceptible fish host by some other means.

Taking these factors into consideration, a qualitative risk analysis led to the conclusion that of all the exotic diseases present in North American salmonids, furunculosis, caused by the bacterium *Aeromonas salmonicida*, is the disease which would be most likely to be carried in the type of commodity under consideration. This conclusion is based, among other things, on the fact that, of all the diseases considered, none result in greater numbers of pathogen being present in the flesh of infected fish.

A quantitative risk assessment was conducted on four commodities;

---

[1] William of Occam c. 1285-1349

- Chilled, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Frozen, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Consumer packs of skinless, boneless chilled fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Consumer packs of skinless, boneless frozen fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

The quantitative risk assessment took into account what is known of the prevalence of *A. salmonicida* in wild, ocean-caught Pacific salmon, the distribution and numbers of *A. salmonicida* found in infected Pacific salmon, the effect of processing on the numbers of *A. salmonicida* in the tissues of infected fish, the survival of *A. salmonicida* in the environment, the dose of *A. salmonicida* required to infect susceptible fish (of any species), and waste management practices in New Zealand.

The risk assessment demonstrated that the risk of introducing *A. salmonicida* into New Zealand's farmed, recreational or native fish stocks is extremely remote. With the least-processed commodity (chilled, headless, eviscerated salmon) the model estimated that there is a 95% probability that there would be fewer than 1 disease introductions per 10 million tonnes imported. To put this into perspective, the analysis pointed out that the entire annual production of wild, ocean-caught Pacific salmon is no more than 100,000 tonnes.

The assessment recognised that the risks associated with other diseases would be cumulative to the risks posed by *A. salmonicida* and that any risk posed by any of the other diseases must be added to that posed by furunculosis. However, the assessment also outlined compelling reasons for considering that no disease is more likely to be introduced than *A. salmonicida* and that the cumulative risk of disease introduction is unlikely to be significantly greater than the range of risk estimates described for *A. salmonicida*.

The risk analysis concluded that the overall risk of introducing diseases of salmon through the vehicle of headless, eviscerated, wild, ocean-caught Pacific salmon, appropriately certified by the Canadian Government authorities as to origin and grade, is negligible and poses no threat to either New Zealand's wild and farmed salmonid stocks or to non-salmonid fish stocks.

PART I: QUALITATIVE RISK ANALYSIS

2. Introduction

For more than a decade Canadian trade officials have been seeking access to New Zealand for a number of commodities processed from wild, ocean-caught Pacific salmon. New Zealand has an important recreational salmonid fishery which generates significant tourist revenue. There is also a growing salmon farming industry. Because of their isolation from northern hemisphere salmon fisheries, New Zealand's salmonid stocks are considered to be free of a number of diseases which are present in fish stocks on the west coast of North America. For this reason. New Zealand has previously adopted a "zero risk" stance with respect to imports of uncooked Canadian salmon products and refused access.

The arguments against granting access have been based solely on a perceived microbiological threat. If a disease could be identified as present in Canadian fish stocks, that was considered sufficient reason to deny access. No attempt was made to actually assess whether the introduction of disease was likely. It was sufficient to postulate the possibility.

For flesh to serve as a vehicle for the introduction of fish disease, a number of criteria must be met. These criteria are;

(a) The disease must be present in the waters of origin.

(b) The disease must be present in the particular fish caught (or the flesh must have become contaminated during processing).

(c) The pathogen must be present in the imported tissues.

(d) The diseased flesh must pass inspection and grading procedures.

(e) The pathogen in the flesh must survive storage and processing and be present at an infectious dose.

(f) The pathogen must be able to establish infection by the oral route or by the host being bathed in it.

(g) Scraps of the flesh product must find their way into a susceptible fish host in New Zealand or an infectious dose of pathogen must find its way into contact with a susceptible fish host by some other means.

The likelihood of each of these criteria being met will be different for different pathogens, different types of fish and different countries of origin. However, taken together, the probability that each criterion will be met for any one disease must be considered remote.

2.1 The commodities being assessed

An earlier risk assessment[2] was concerned solely with consumer packs of frozen fillets of wild, ocean-caught Pacific salmon. However, following discussions with Canadian officials in July 1994[3], the range of commodities under consideration was broadened to include;

(a) Chilled, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

(b) Frozen, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

(c) Consumer packs of skinless, boneless chilled fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

(d) Consumer packs of skinless, boneless frozen fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

Evisceration of the wild, ocean-caught Pacific salmon intended for export to New Zealand consists of the removal from the fish of all visceral organs and tissues, gill, kidney tissue, heart and attached mesentery and vessels. This process substantially reduces the numbers of infectious micro-organisms, if any, which may be present in the fish.

There is no question that the prevalence of some diseases is greater in farmed salmon than in wild, ocean-caught salmon[4], and this has led opponents of importation to claim that even if wild, ocean-caught Pacific salmon were demonstrated to be without risk, there would still be a threat from **substitution of farmed salmon for wild salmon.** However, such claims are baseless. Farmed salmon from British Columbia are sold fresh on ice primarily to the United States; 80% of salmon is exported to the United States and 20% is consumed within Canada. Processing plants that handle farmed salmon deal solely with the farmed product. Wild-caught salmon are processed in different plants with facilities for freezing and canning. The farmed salmon is considerably **more expensive** than the ocean-caught salmon and so it is unlikely that there would be substitution of this more expensive commodity in place of the cheaper

---

[2] MacDiarmid, S C, Cotton, S. The Risk of Introducing Aeromonas salmonicida into New Zealand Salmon Fisheries Through the Vehicle of Frozen Fillets of Ocean-Caught Canadian Salmon. 46 pages. NASS Publication 93-1. Ministry of Agriculture and Fisheries, Wellington, 1993.

[3] Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994 in relation to GATT Article XXII:1.

[4] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with SC MacDiarmid, 23 December 1993.

one. However, salmon destined for export to New Zealand would be government certified as being derived from wild, ocean-caught Pacific stocks.[5]

While the presence of any disease of salmonids on Canada's west coast has been cited as sufficient reason to prohibit imports of wild, ocean-caught Pacific salmon, the disease most likely to be introduced through such imports is furunculosis, caused by the bacterium *Aeromonas salmonicida*. For this reason, *A. salmonicida* was chosen as the agent to be used in the earlier risk assessment model. The authors reasoned that if the assessment showed that there to be a negligible risk from the disease considered most likely to be introduced through the vehicle of fillets, the overall risk is also likely to be negligible. That is, unless one or more of the critical input variables for any other exotic pathogen could be shown to be significantly greater than the input variables used for *A. salmonicida*, the risk assessment on this pathogen could be taken as representative of the other risks. However, this reasoning was rejected by opponents of the proposal to permit importation of fillets of wild, ocean-caught Pacific salmon and so the present risk analysis examines an extensive range of diseases.

2.2 Marker diseases

There are no documented reports anywhere of fish diseases being introduced into any country or region through the importation of fish intended for human consumption. The only published report which supports the view that table salmon could introduce disease is a 1970 paper by Hoffman[6] who proposed that whirling disease was introduced into North America following the importation of frozen table trout from Europe. However, Hoffman was writing before the life cycle of *Myxobolus cerebralis*, the causative agent of whirling disease, had been characterised.

It is now recognised that the life cycle of *M. cerebralis* is dependent on an intermediate host, a specific oligochaete worm, and the appearances of whirling disease which Hoffman attributed to trade in table fish are far more likely, in fact, to have been due to the importation of mud and sediments associated with importations

---

[5] Officials of Department of Fisheries and Oceans, Ottawa, personal communication with SC MacDiarmid during visit to Ottawa, November 1993.

[6] Hoffman, GL. Intercontinental and transcontinental dissemination and transfaunation of fish parasites with emphasis on whirling disease (*Myxosoma cerebralis*). A Symposium on Diseases of Fishes and Shellfishes. Special Publication No. 5. Pp 69-81. American Fisheries Society, Washington D C, 1970.

of aquarium fish.[7] The epidemiology of whirling disease makes it improbable that it could be introduced in salmon intended for human consumption.[8]

The lack of risk posed by imports of eviscerated fish is reflected by the regulations governing the trade in wild-caught fish for human consumption in Europe, North America and Japan, amongst others. It is also reflected in the attitudes of organisations such as *Office International des Epizooties* (OIE), the 130 member international animal health organisation of which New Zealand is a member. Professor Tore Håstein, President of the OIE Fish Diseases Commission, expressed the opinion that if there were a significant risk of disease introduction in fillets or eviscerated fish for human consumption, the spread of important diseases would have been "tremendous" as many countries export large volumes of farmed salmonids. He cited as an example infectious salmon anaemia (ISA), a disease reported only in Norway despite that country having exported large quantities of Atlantic salmon. Professor Håstein interprets this as indicating that the risk of spreading fish diseases by means of fish intended for human consumption is negligible.[9]

Specialists with the Northern Ireland Department of Agriculture have expressed the view that the importation of table salmon, so long as they are grossly normal and thoroughly eviscerated (including removal of all the kidney and, possibly, the head), poses little risk of introducing bacterial, viral or parasitic diseases.[10]

Commodities other than eviscerated salmon may also pose a negligible disease risk. For instance at one time Australia used to import salmon heads for use as bait in crab traps[11], and yet Australia remains free of the major diseases of salmonids.

The fact that the Great Lakes fisheries, with their surrounding human population of some 40 million people, have remained free from infectious haematopoietic necrosis (IHN), a disease found on the west coast of North America, despite many tonnes of

---

7 Evelyn, TP, Pacific Biological Station, Nanaimo, Nickum, JG, US Fish and Wildlife Service, personal communication with SC MacDiarmid during meeting at Nanaimo, 25-26 July 1994.

8 Anderson, C. Epidemiological aspects of *Myxobolus cerebralis*, the agent of salmonid whirling disease. Surveillance 20(2), 19-24, 1993.

9 Professor T Håstein, President OIE Fish Diseases Commission, personal communication to Dr B D O'Neil, Chief Veterinary Officer, 11 August 1994.

10 Menzies, F, McLoughlin, Marian, Veterinary Sciences Division, Northern Ireland Department of Agriculture, personal communication with SC MacDiarmid, 26 August 1994.

11 Dr Leo Margolis, during Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994 in relation to GATT Article XXII:1. The assertion was not disputed by the Australian representatives at the meeting.

west coast salmon having been consumed in the area each year for decades, will be discussed below (Section 3.2).

It is observations such as these which has led to the concept of considering certain diseases as "marker diseases". For example, *Ceratomyxa shasta* and IHN virus, which are present only on the west coast, can be considered in relation to the volume of trade into certain markets. An example of the use of a marker virus would be infectious pancreatic necrosis (IPN). IPN is not present in British Columbia (see 3.1 below). It is endemic on the east coast of Canada but, despite years of importing farmed Atlantic salmon onto the west coast, IPN has never been detected there, despite there being a surveillance program for it on the west coast. So, the freedom of British Columbia salmon stocks from IPN is a further argument that salmon flesh is an unlikely vehicle for the introduction of disease.

Another example, for which **quantitative data** are available, is the protozoan parasite *C. shasta*. This parasite is unlikely to escape detection if present in salmon flesh because of the tissue reaction. It has a limited distribution in British Columbia compared to *Aeromonas salmonicida* and its prevalence is much lower. It is assumed that some of the spores would survive freezing and thawing but *C. shasta* has never appeared anywhere else in the world despite considerable tonnages of salmon being exported from British Columbia. (For example, between 1986 and 1991 Japan imported 117,936 tonnes of frozen, whole dressed Pacific salmon).

Quantitative data for exports into other markets free from the marker diseases IHN and ceratomyxosis are presented in Table 2.1.

Table 2.1: Canadian exports of frozen, whole or dressed, Pacific salmon to selected European countries, and the presence/absence of IHN and ceratomyxosis in each country

Country	Total exports of frozen, whole, dressed, salmon 1979-1991[1]	IHN detected (Det) or not detected (ND)	Ceratomyxosis detected (Det) or not detected (ND)
Belgium	5,364	Det[2]	ND[3]
Denmark	13,139	ND[4]	ND[3]
Finland	2,462	ND[4]	ND[3]
France	54,889	Det[2]	ND[3]
Germany	9,781	Det[2]	ND[3]
Ireland	164	ND[2]	ND[3]
Italy	12,800	Det[2]	ND[3]
Netherlands	6,818	ND[4]	ND[3]
Norway	84	ND[4]	ND[3]
Spain	617	ND[4]	ND[3]
Sweden	15,610	ND[4]	ND[3]
Switzerland	6,268	Det[4]	ND[3]
United Kingdom	9,454	ND[2]	ND[3]

1. Statistics Canada annual reports on exports by commodities.

2. Dr Alan Munro, Marine Laboratory, Aberdeen, Scotland, personal communication with officials of Department of Fisheries and Oceans.

3. J G Hnath. 1983. Ceratomyxosis. In "A Guide to Integrated Fish Health Management in the Great Lakes Basin". Eds F P Meyer, J W Warren and T G Carey. Great Lakes Fishery Commission, Special Publ. 83-2. pp. 217-222.

4. OIE 1992 World Report on Animal Diseases.

Examination of data in Table 2.1 shows that there are countries accepting very large volumes of Canadian salmon without the introduction of these diseases. In those cases where one of the marker diseases is present in the importing country, its introduction has been attributed to the importation of eggs or live fish. In no case has the

importation of dead salmon for human consumption been implicated as the vehicle by which the disease was introduced.

2.3 Salmonid diseases of concern

Critics of the earlier assessment of the disease risk posed by importation of Pacific salmon claimed that the assessment had failed to take into account diseases other than furunculosis. Although that assessment made the point that, because of the nature of the disease, furunculosis was the one most likely to enter through imported salmon flesh, because of the intramuscular location of the causative agent, the current assessment discusses a range of salmon diseases which have been raised as requiring specific discussion.

The list of particular diseases which need to be considered in any assessment of the disease risks posed by imported Pacific salmon has varied at different stages of the debate. For instance, an article published in 1990[12] listed what the writer considered to be the major diseases of salmon. These appear in Table 2.2.

[12] Anderson C D. Important diseases of salmonid fish and the risk they pose to New Zealand. Surveillance 17(2), 17-18, 1990.

Table 2.2: Important diseases of salmonid fish

Viral diseases	Infectious haematopoietic necrosis (IHN) Viral haemorrhagic septicaemia (VHS) Infectious pancreatic necrosis (IPN) Piscine erythrocytic necrosis (PEN) Herpesvirus salmonis
Bacterial diseases	Bacterial kidney disease (BKD - *Renibacterium salmoninarum* Furunculosis (*Aeromonas salmonicida*) Vibriosis (*Vibrio ordalii, V. anguillarum*)
Protozoal diseases	Whirling disease (*Myxobolus cerebralis*) Proliferative kidney disease *Ceratomyxa shasta* *Henneguya salmonicola* *Parvicapsula*
Fungal diseases	Rosette disease

However, in discussing these, the writer pointed out that *Myxobolus cerebralis* (whirling disease) is present in New Zealand and a Birna virus of the IPN group is also present here. He went on to describe how the most important disease for salmon farmers in New Zealand is vibriosis (caused by *Vibrio ordalii* and *V. anguillarum*) but this disease has little economic impact on New Zealand salmon farmers, with losses attributable to vibriosis involving usually only 5-10% of the least valuable year class on the property.

A year later, in another discussion of the disease risks posed by proposed imports of Pacific salmon from British Columbia, Anderson listed a different set of diseases as being of concern.[13] He reviewed what he considered to be the available information on the fish diseases likely to be present in marine caught Canadian salmon and placed special emphasis on the disease impact/risk of introduction of these diseases to New Zealand. Anderson stated that the amount of available information varied between diseases and that considerably more information was available for *A. salmonicida* than, for example, erythrocytic inclusion body syndrome (EIBS). In his briefing Anderson tabulated what he considered to be the most significant diseases (Table 2.3). He attempted to break down the disease risk into categories which cover the following areas of the risk assessment;

[13] From a briefing to the Chief Veterinary Officer by C D Anderson, May 1991.

Category A: Detrimental affect on farmed and wild salmonids: A category said to be a combined figure resulting from assessment of both the mortalities produced by and economic implications of, each disease.

Category B: Prevalence in salmon and fillet: Also said to have been allocated after assessment of both an estimation of disease's prevalence in ocean salmon and its likely presence in the skeletal muscle of infected salmon.

Category C: Survival: An assessment of the relative ability of the disease organism to survive both in the fillet and after release into New Zealand environmental conditions.

Category D: Ease of transmission: This related to an assessment of the relative ease of transmission of the disease agent once it had reached New Zealand waterways.

In conducting his assessment Anderson allocated arbitrary numerical scores to each category. While this was a reasonable attempt to establish the **relative** risk posed by each disease, the method was invalidated by ranking the different categories on different arbitrary numerical scales. For instance, category A was given an increased weighting owing to what the author considered its greater relative significance in relation to the other categories. It is not uncommon when people perceive risks for them to emphasise the **consequences** of the feared event while tending to overlook the actual **probability** of that event occurring. However, as the maximum scores of 10 or 20 used by Anderson are completely arbitrary, firm conclusions drawn by summing the scores allocated to each category are invalid. In Anderson's briefing the relative risk scores for each disease were those shown in Table 2.3.

Table 2.3: Evaluation of the disease impact and the risk of disease introduction with ocean caught Canadian salmon flesh imports

DISEASE	Detrimental effect on farmed and wild 20	Prevalence salmon/fillet 10	Survival 10	Ease of Transmission 10	Treatment 10	Total	Ranking
C. shasta	16	3	10	8	10	47	2b
VEN	6	7	8	10	10	41	4b
IHN	16	1	7	3	10	37	8
EIBS	6	2	4	8	10	30	9
Lymphoblastosis (microsporidium)	8	3	10	8	10	39	7
Loma species	6	7	10	8	10	41	4c
Marine anaemia	15	7	4	5	10	41	4a
VHS	18	?	4	5	10		
PKD	10	2	0*	0	9		
A. salmonicida	16	6	8	9	8	47	2a
BKD	20	9	8	7	9	53	1

Abbreviations:

C. shasta - *Ceratomyxa shasta*
VEN - Viral erythrocytic anaemia
IHN - Infectious haematopoietic necrosis
VHS - Viral haemorrhagic septicaemia
PKD - Proliferative kidney disease
BKD - Bacterial kidney disease

* consequently trade in salmon flesh poses no risk to New Zealand

However, as the maximum scores (10 or 20) are totally arbitrary, the final ranking would be different if different, equally valid but equally arbitrary, maximum scores were used. For instance, it could be argued that, as the risk assessment is about the likelihood of an exotic disease being introduced in the commodity, the figures for survival in flesh are more important and should be rated out of 20 instead of 10. However, as the briefing gave no basis for allocating **any** score, and the scores for prevalence in particular seem to have no objective basis (see below, Section 3), all that can be taken from this early attempt at risk assessment is that some diseases are more likely than others to be introduced in the flesh of wild, ocean-caught Pacific salmon.

In the same briefing document, the writer made a subjective assessment of the risks posed by another selection of salmon diseases. This assessment is reproduced in Table 2.4.

Table 2.4: Diseases which may be present in Canadian ocean-caught salmon and their assessed disease risk to New Zealand.[14]

DISEASE	RISK ASSESSMENT
Bacterial kidney disease (*Renibacterium salmoninarum*)	Extreme
Furunculosis (*Aeromonas salmonicida*)	Very significant
Ceratomyxa shasta	Very significant
Viral erythrocytic necrosis (VEN)	Significant
Marine anaemia	Significant
Loma species	Significant
lymphoblastosis	Significant
Infectious haematopoietic necrosis (IHN)	Low
Erythrocytic inclusion body syndrome (EIBS)	Low
Henneguya salmonicola	Minimal
Kudoa thyrsites	Minimal
Parvicapsular disease	Minimal
Rosette disease	Minimal
Infectious pancreatic necrosis (IPN)	Minimal
Enteric red-mouth (*Yersinia ruckeri*)	Minimal

[14] From a briefing to the Chief Veterinary Officer by C D Anderson, May 1991.

Office International des Epizooties (OIE), the 130-odd member international animal health organisation to which New Zealand belongs, categorises diseases according to their significance. No diseases of fish are classified in OIE's List A (transmissible diseases which have the potential for very serious and rapid spread, irrespective of national borders, which are of serious socio-economic or public health consequence and which are of major importance in the international trade of animals and animal products). However, there are some fish diseases classified in OIE's List B (transmissible diseases which are considered to be of socio-economic and/or public health importance within countries and which are significant in the international trade of animals and animal products). The OIE List B diseases of fish are;

viral haemorrhagic septicaemia
spring viraemia of carp
infectious haematopoietic necrosis
salmonid herpesvirosis (Type 2)
renibacteriosis (*R. salmoninarum*)
ictalurid herpesvirosis (Type 1)
epizootic haematopoietic necrosis
edwardsiellosis (*E. ictaluri*).

However, not all of these affect salmonids and many diseases of salmonids which would be of concern to New Zealand are not listed at all.

A veterinarian with expertise in fish diseases, Dr S G Fenwick[15], was asked to list the most important fish pathogens which might be introduced in imports from North America. His list included the following viruses and bacteria;

infectious pancreatic necrosis virus (IPN)
infectious haematopoietic necrosis virus (IHN)
Renibacterium salmoninarum (agent of bacterial kidney disease)
Vibrio anguillarum (agent of vibriosis)
Piscirickettsia salmonis (agent of salmonid rickettsial septicaemia)
Aeromonas salmonicida (furunculosis agent)

Fenwick went on to say that of these, *Vibrio anguillarum* is endemic in New Zealand and need not be considered in a risk assessment. However, the remainder are exotic to this country and therefore pose a threat. The introduction of any of these pathogens into New Zealand and their establishment in native fish species would have serious effects on both commercial and recreational fisheries.

---

[15] SG Fenwick, Department of Veterinary Pathology and Public Health, Massey University, Palmerston North, personal communication to SC MacDiarmid, 12 August 1994.

Another worker with a knowledge of the New Zealand salmon farming industry[16] recommended that any analysis of the potential disease risks posed by importation of Canadian salmon should include, in addition to furunculosis;

rosette agent
viral erythrocytic necrosis
marine anaemia
erythrocytic inclusion body syndrome
pancreas disease
Loma
Kudoa
lymphoblastosis.

Considering comments such as these, it became apparent that as many as 23 diseases which may affect salmon and which are present in North America need to be discussed to allay the concerns of local salmon farmers, recreational fishers and conservation groups. This expanded list of diseases, which will be considered in this risk assessment, includes;

infectious pancreatic necrosis (IPN)
infectious haemorrhagic necrosis (IHN)
viral haemorrhagic septicaemia (VHS)
viral erythrocytic necrosis (VEN)
erythrocyte inclusion body syndrome (EIBS)
pancreas disease
plasmacytoid leukaemia
Aeromonas salmonicida
bacterial kidney disease (BKD)
enteric redmouth (ERM)
salmonid rickettsial septicaemia
rosette agent
Loma salmonae
Enterocytozoon salmonis
proliferative kidney disease
edwardsiellosis
Kudoa thyrsites
Ceratomyxa shasta
Herpesvirus salmonis
vibriosis
Hitra disease (*Vibrio salmonicida*)
Henneguya salmonicola
parvicapsular disease.

---

[16] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with SC MacDiarmid, 23 December 1993.

Not every disease poses an equal threat. They differ in the likelihood of their being introduced in the commodity and they differ in the adverse impact their introduction would have in New Zealand. Each is discussed in turn below. Much of the information on these diseases was obtained by the writer on two visits to the Canadian Department of Fisheries and Oceans Pacific Biological Station, Nanaimo, British Columbia in November 1993 and July 1994. The latter visit was on the occasion of a Canada-Australia Salmon Technical Meeting held in relation to GATT Article XXII:1.

3. Specific diseases of salmon

The commodity considered in the earlier risk assessment was consumer packs of fillets of wild, ocean-caught Pacific salmon. However, following discussions in British Columbia in July 1994, the current risk assessment has been broadened to cover **fresh or frozen, headless, eviscerated, wild, ocean-caught Pacific salmon caught off the Canadian coast.** The discussion of specific diseases is based on a consideration of this commodity, rather than just fillets.

3.1 Infectious pancreatic necrosis (IPN)

IPN is present on the eastern side of Canada and the US but, despite eviscerated salmon having been traded into the western provinces and states in large volumes over many years, the fisheries of the west coast of North America remain free of this infection.

These claims of freedom from IPN virus are solidly based. As part of an on-going surveillance program for diseases of salmonids, Canadian workers from the Pacific Biological Station, Nanaimo, up to the end of 1993, have examined samples from over 64,702 wild and cultured salmon and trout for evidence of IPN, all with negative results.[17] They have also examined filter feeders from aquaculture sites for infection with IPN virus, all with negative results.[18] Results of attempted virus isolation from Pacific salmon returning to spawn are shown in Table 3.1. It must be emphasised that the data in this table on the prevalence of IPN virus, IHN virus and VHS virus in mature Pacific salmon were gathered from fish that had already returned from the sea to fresh water to spawn. The data thus also reflect infections that were acquired by the fish following their return to fresh water. The prevalences noted thus probably exaggerate the true pathogen prevalence picture for fish still at sea.

---

[17] Dr Trevor P Evelyn, Pacific Biological Station, Nanaimo, British Columbia, personal communication to SC MacDiarmid, 17 August 1994.

[18] Dr Trevor P Evelyn, Pacific Biological Station, Nanaimo, personal communication with SC MacDiarmid during a visit to Nanaimo, November 1993.

Table 3.1: Prevalence of IHN, IPN, and VHS viruses in wild, ripe or spent salmon in British Columbia from 1972 to 1993[19]

Species	No. assayed for viruses	No. fish (minimum and maximum)* positive for indicated virus		
		IHN	IPN	VHS
Pink	1,249	0 & 0	0 & 0	0 & 0
Chum	1,044	0 & 0	0 & 0	0 & 0
Coho	2,393	8 & 8	0 & 0	0 & 0
Chinook	3,893	4 & 11	0 & 0	0 & 0
Sockeye	6,016	623 & 1,518	0 & 0	0 & 0
Totals	14,595**	635 & 1,537	0 & 0	0 & 0
Percents		4.4 & 10.5	0 & 0	0 & 0

* The minimum number assumes that only one fish per pool was virus positive when fish were pooled while the maximum number assumes that all fish in any given pool were virus positive.

** This number represents 53% of the total number of fish examined for various reasons to date and takes into account results that had not yet been entered into the computer data-base when the tables on bacterial prevalence were prepared.

The surveillance program has isolated on one occasion a non-pathogenic Birna virus (same group as IPN virus). However, it should be borne in mind that a Birna virus of this group has also been isolated from New Zealand waters.

The Canadian salmon disease surveillance program always uses at least two cell lines for each viral assay. The testing is carried out on salmon on their return to fresh water, when the stresses of the change and the population density tend to increase the likelihood of detection of diseases. The surveillance on returning salmon thus tends to describe a worst case. The risk of disease in ocean-caught fish is less.

The Birna virus isolated in British Columbia was from an Atlantic (cultured) salmon in a sea cage. Pacific salmon have not been found to be infected and studies elsewhere suggest that Pacific salmon are relatively resistant to infection with IPN virus.

---

[19] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994 in relation to GATT Article XXII:1.

IPN virus requires very high doses to establish infection by immersion or by mouth. Immersion of very juvenile Pacific salmon in 10^5 pfu/ml IPN virus for 4-5 hours may establish infection. Experiments with the British Columbia isolate of Birna virus were **not** able to establish infection in non-immunecompetent 40 day fish with 5 hours immersion in 10^4 pfu/ml.

During the July 1994 meeting, Dr Trevor Evelyn of the Pacific Biological Station was asked what tissues are carrier sites for IPN virus. He replied that the gills are a carrier site for some other viruses, but it is not known whether this is so for IPN virus. The kidney, spleen and pyloric caeca are the sites from which IPN virus is most readily cultured.

In fish dying of IPN there is a viraemia. In any viraemia, virus could be present in muscle. However, IPN deaths do not occur in mature, market size fish. IPN kills very small juveniles.

Pacific salmon are refractory to IPN. One does not find active (viraemic) IPN in adult, marketable salmonids.

So, Pacific salmon from the west coast of Canada do not pose a threat so far as IPN is concerned. This is because;

- IPN does not occur on the west coast.
- IPN viraemia does not occur in market size fish.
- Pacific salmon are refractory to infection with IPN.
- IPN virus is seldom found in tissues other than the gills and viscera, which are removed in the commodities under discussion.
- High doses of IPN virus are required to establish infection, even in non-immunecompetent young fish.

3.2 Infectious haematopoietic necrosis (IHN)

IHN has been found in Pacific salmon in Canada and the US, but the virus is not found in sea-run fish. It has been found in stressed fish in the fresh water spawning grounds (see Table 3.1 above).

There has been a case where IHN virus was isolated from 7 of 60 fish sampled returning from sea in September, late in the year. The salmon in this case were sockeyes.

The main route of IHN transmission is horizontal. IHN virus survives in salmonid eggs only when the embryo is actively growing and vertical transmission rarely, if ever, occurs.

The reservoir of IHN virus is still not defined. It is **possibly** present in a "masked" form in adults, being activated by stress.

IHN virus is normally isolated from the kidney and other visceral tissues, not the flesh. It is only in dying, viraemic fish that IHN virus will be detectable in flesh.

By analogy with other diseases (such as BKD, furunculosis) there are likely to be between 10,000 and 100,000 times fewer infectious particles in the flesh as compared to the viscera.

High doses of IHN virus are required to establish infection. Immersion for 1 hour in 10^4 pfu/ml may infect young fish. Injection of 10^2-10^3 pfu per fish may also establish infection.

(In New Zealand, people opposed to the importation of salmon flesh claim that flesh from fish dying from any one of a list of diseases could be imported. However, this claim ignores the fact that viraemic or septicaemic fish exhibit a number of signs which make them aesthetically unacceptable to the consumer - haemorrhages in the flesh, discolouration, softness - and for this reason they would be rejected or down-graded as suitable only for canning. The salmon processors have a compelling, commercial incentive in grading their product and sick fish are not processed as first grade product).

There are no controls in Canada or the US on the movement of eviscerated salmonids[20] and yet IHN is still confined to the west coast, despite decades of trading fish across the continent. As an example, some 40 million people live in the area around the Great Lakes and many tonnes of west coast salmon are consumed in the area each year. Nevertheless, IHN virus has never been found in the salmonid fish stocks of the Great Lakes. If IHN has not found its way into the Great Lakes, given the volume of west coast salmon consumed in the area, why should other importing countries be considered to be at risk?

IHN has only been recovered from sockeye salmon returning from sea once they have entered freshwater. On average, 40% of the fish caught each year at sea are sockeye and despite their never having been controls on the movement of these dead wild fish

---

[20] The Canadian Department of Fisheries and Oceans (DFO) only excluded the trade in eviscerated **cultured** (that is, farmed) fish from the Fish Health and Protection Regulations in 1992. However, when they changed the Regulations to exclude eviscerated fish, the decision was based on their assessment that there is minimal risk with transfer of eviscerated fish. DFO had permitted the transfer of eviscerated, **wild**-caught Pacific salmon to the east coast for decades without introducing marker diseases into the receiving areas.

Authorities in the USA reached the same decision, as reflected in the 1993 amendment to Title 50.

to other provinces or states IHN has not spread. Rainbow trout are highly susceptible to IHN. They are the major cultured fish east of the Rockies and yet, despite active surveillance, IHN virus has never been found east of the great divide.

(IHN virus **has** been introduced into hatcheries east of the Rockies in fertilised eggs, but was eradicated and intense surveillance has confirmed this).

IHN virus is a **rhabdovirus** and, as such, is relatively susceptible to damage by freezing and thawing. A single freeze-thaw cycle will reduce the titres of IHN virus in viscera by 10 to 100 fold. Being a rhabdovirus, IHN is also likely to be damaged by household detergents, such as are present in domestic waste water. IHN virus, even if present in domestic waste water, would be unlikely to survive passage through sewage.

IHN virus is not likely to be introduced in imports of headless, eviscerated wild, ocean-caught Pacific salmon because;

- IHN virus has rarely been isolated from ocean-caught Pacific salmon.
- IHN virus is found in flesh (as opposed to viscera) only in dying, viraemic fish.
- Despite a lack of internal controls on the movement of dead wild fish in Canada and the US, IHN has never established east of the Rockies.
- IHN is a rhabdovirus and as such is susceptible to damage by freezing and thawing and by detergents likely to be present in household waste water.
- High doses of IHN virus are required to establish infection.

3.3 Viral haemorrhagic septicaemia (VHS)

Two strains of VHS virus have been identified; the European strain which is a salmonid pathogen and the North American strain, which infects some Pacific marine fish but does not appear to cause disease.

European VHS does not occur in the Pacific and North American VHS is **not** pathogenic for salmonids. It has been isolated from healthy fish being tested routinely as part of a Canadian viral surveillance program, but not from Pacific salmon returning from sea and not in British Columbia (Table 3.1, Table 3.2).

Table 3.2: Assays of wild and cultured salmonids in British Columbia for VHS virus (1988 to end of 1993).[21]		
Year	No. Tested	No. with VHS virus
1988 & 1989	20,474	0
1990	8,647	0
1991	12,843	0
1992	6,180	0
1993	6,548	0

North American VHS virus is a marine virus. The Pacific herring may be its natural host. It is commonly isolated from **stressed** Pacific herring all along the Pacific coast of North America.

Titres of VHS virus are **very** low, even in viscera. In flesh, titres could be expected to be even lower.

VHS virus is a **rhabdovirus** and, as such, is relatively susceptible to freezing and thawing damage, like IHN virus. Similarly, like IHN virus, VHS virus is likely to be disrupted by the detergents found in household waste water.

VHS virus is not likely to be introduced in imports of headless, eviscerated wild, ocean-caught Pacific salmon because;

- The North American VHS virus is non-pathogenic for salmonids and is seldom found in Pacific salmon.
- Titres of North American VHS virus in the viscera of infected fish are very low and titres in muscle would be much lower.
- VHS virus is susceptible to damage by freezing and thawing and by detergents present in domestic waste water.

3.4 Viral erythrocytic necrosis (VEN)

VEN is also known as PEN, for piscine erythrocytic necrosis. VEN virus cannot be grown in tissue culture. It has been found in red blood cells. VEN virus can infect a

---

[21] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

large number of marine species. However, there is a suggestion that the strains isolated from the different species such as cod or Pacific herring may not be the same virus.

VEN has never been found in strictly fresh water fish. Pacific salmon probably become infected by eating herring.

VEN is probably ubiquitous. It is likely that it would be found in the waters off New Zealand and Australia if an adequate survey were conducted.

Even though anadromous species may become infected with VEN, and even though fresh water species are susceptible to experimental infection with VEN, one never sees VEN transmitted to fresh water fish naturally.

VEN virus is located within the cytoplasm of red blood cells, so it could be present in flesh. However, VEN is not a problem in wild Pacific salmon, nor is a problem in cultured salmon. Experimentally, juvenile salmon can be infected if injected with blood from herrings. If these experimentally infected fish are held together with others for long enough, VEN may be transmitted. No disease of salmonids is attributable to infection with VEN virus. Infection may predispose salmon to other diseases but because VEN is not a problem in either wild or cultured salmon in Canada and the US little research has been done on it and no good prevalence data exist.

The presence of VEN in the waters of the Pacific coast of North America should not be raised as a barrier to importing wild, ocean-caught Pacific salmon because;

- No disease of salmonids is attributable to infection with VEN virus.
- VEN virus is probably present in the marine environment of New Zealand.
- VEN virus infects red blood cells, not viscera or muscle.
- VEN is not transmitted readily between infected salmon and susceptible salmon.

3.5 Erythrocyte inclusion body syndrome (EIBS)

EIBS is not a problem in British Columbia. It has been found in cutthroat trout, but no prevalence data are available. EIBS is unimportant and the virus has not been cultured, so no quantitative studies have been carried out.

The virus is located within the cytoplasm of red blood cells. EIBS has not been found in Pacific salmon and no disease has been attributed to it.

The presence of EIBS in the waters of the Pacific coast of North America should not be raised as a barrier to importing wild, ocean-caught Pacific salmon because;

- EIBS has not been found in Pacific salmon.
- No disease of salmonids is attributable to infection with EIBS virus.
- EIBS virus is present in red blood cells rather than muscle tissue.

3.6 **Plasmacytoid leukaemia**

Plasmacytoid leukaemia (also known as lymphoblastic lymphoma, lymphoblastosis, marine anaemia) is due to infection with a cell-associated retrovirus. The infection is only seen as a **clinical** condition in chinook salmon in sea water, although infected fish may be found in fresh water.

Plasmacytoid leukaemia is **not** transmissible in sea water but can be transmitted in fresh water. It is probably transmitted **vertically** rather than horizontally.

Plasmacytoid leukaemia is not seen as a disease in wild salmon. However, infection with the virus has been detected occasionally in wild salmon. On one occasion a survey revealed a prevalence of around 1% in a sample of 500 adult wild-caught chinook salmon returning to fresh water.

The infection has only been detected in chinook salmon. However, it is possible to infect other salmon experimentally with plasmacytoid leukaemia virus; by **injection** with a large dose of a 10% tissue homogenate.

In fresh water, plasmacytoid leukaemia virus can transmit horizontally between chinook salmon cohabiting. Experimentally this has been demonstrated; 30 fish were infected by injection and held in a netpen with 30 susceptibles. As the inoculated fish died they were replaced with a further 20 inoculated fish. Transmission from the dying fish to those with which they were penned was demonstrated in so far as over an 8 month period **one** of the non-inoculated fish died. However, transmission did not occur to susceptibles held in adjacent netpens.

Plasmacytoid leukaemia is found only in chinook salmon. Chinooks make up less than 2% of the total catch.

The disease is a leukaemia; a proliferation of blood cells which accumulate in the kidney, eyes and other viscera. The virus is probably, therefore, present to some extent in all tissues. However, it is very difficult to detect the virus by electronmicroscopy. The cells must be disrupted and centrifuged to concentrate sufficient virions to see.

Plasmacytoid leukaemia is seen as a chronic low-grade problem in farmed chinook salmon. The virus probably predisposes infected fish to other infections.

The virus is inactivated at 37 °C.[22]

Plasmacytoid leukaemia does not occur naturally in coho salmon although these can be experimentally infected.

The presence of plasmacytoid leukaemia in the waters off the west coast of North America should not be raised as a barrier to importing wild, ocean-caught Pacific salmon because;

- Plasmacytoid leukaemia virus does not cause significant disease in salmon.
- Plasmacytoid leukaemia virus occurs only rarely in a single species (chinook) of wild, ocean-caught salmon.
- The one species of salmon naturally infected with plasmacytoid leukaemia virus (chinook) makes up a very small proportion (<2%) of the total catch.
- Plasmacytoid leukaemia virus is transmitted only with difficulty following prolonged cohabitation.

3.7 Pancreas disease

Pancreas disease has not been detected anywhere in Pacific salmon on the west coast of North America, not in Canada nor in the US. It was detected once, in 1986, in farmed Atlantic salmon in the state of Washington. Pancreas disease is not a concern on the Pacific coast of either British Columbia or the United States.

Pancreas disease is a condition of Atlantic salmon. The virus is not demonstrable in the tissues of infected fish, so is present in very low titres only. It is a disease of visceral tissue, not muscle.

The virus of pancreas disease is not culturable, but a viral aetiology for the disease has been demonstrated.

Pancreas disease should not be raised as a barrier to the importing of wild, ocean-caught Pacific salmon because;

- Pancreas disease is a disease of Atlantic, not Pacific, salmon.
- Pancreas disease has only once been demonstrated on the west coast, and that in farmed salmon.

---

[22] The significance of this is that the internal body temperature of mammalian or avian scavengers is greater than 37 °C.

- The target organ of pancreas disease is removed during evisceration.
- Virus is present only in low titres in fish affected by pancreas disease.

3.8 Furunculosis

If any disease is likely to be introduced in salmon flesh, it is furunculosis, a bacterial disease caused by *Aeromonas salmonicida*. This view is shared by most of the people with specialist knowledge consulted during the preparation of this analysis. For example, Fenwick[23] stated "of all the pathogens to be found in salmon, this is the only one causing significant muscle pathology, with abscesses in the muscle being one of the most common clinical and post-mortem findings, usually indicative of chronic infection. Furunculosis is usually introduced into farms by asymptomatic carriers or chronically infected fish. This organism, in my opinion, would be the most likely pathogen that could be introduced via infected fish fillets. Therefore an assessment of the likelihood of this organism being imported and infecting native fish is the most appropriate model to use to analyze the risks of importing any exotic fish pathogen. In other words, if the risk of importing *A. salmonicida* can be shown to be negligible, all other pathogens should have a similarly low risk of introduction."

A consultant at Stirling University's Institute of Aquaculture[24] stated that "...although the viral diseases infectious haematopoietic necrosis (IHN) and viral haemorrhagic septicaemia (VHS) are both present in the north west of North America in Pacific salmon, the main sources of these viruses are blood and sexual products, both unlikely to be involved in fillets from bled fish. I would agree with your basic premise to choose *Aeromonas salmonicida* and it may be worthwhile elucidating why you have chosen this..."

In a nutshell, fish affected by furunculosis typically have lesions in the skin and muscle tissue, and these lesions contain large numbers of the pathogen.

Infection with *A. salmonicida* is very uncommon in **wild**, ocean-caught Pacific salmon (Table 3.3). When 21,495 ripe salmon were tested **on their return to their fresh water spawning grounds**, by which time they had been stressed by the adaptation to fresh water, by the physiological change of sexual maturation, and crowding into infected waterways, 1,298 (6%) were found to be infected with *A. salmonicida*. However, even this low percentage does not represent the risk posed by ocean-caught fish.

---

[23] Dr SG Fenwick, Faculty of Veterinary Science, Massey University, personal communication with SC MacDiarmid, 12 August 1994.

[24] Hamish Rodger MRCVS, Veterinary Clinical Officer, Institute of Aquaculture, University of Stirling, Scotland, personal communication with SC MacDiarmid, 9 August 1994.

Table 3.3: Prevalence of[25] *Aeromonas salmonicida* (A.s.)* in wild, ripe or spent salmon in British Columbia from 1972 to 1993.[26]

Species	No. of fish	No. of fish cultured	No. of A.s. positive fish	No. of fish with flesh A.s. lesions
Pink	2,510	2,490	107	0
Chum	2,123	2,111	61	0
Coho	9,750	8,825	737	10
Chinook	5,908	5,381	364	2
Sockeye	5,371	2,688	29	2
Totals	25,662	21,495	1,298	14
Percents		83.8	6.0	0.05

* All isolates were typical *A. salmonicida* subsp. *salmonicida* where tested.

In a recent survey, Canadian Department of Fisheries and Oceans researchers sampled 300 wild sockeye and 300 wild chum salmon **before** they entered fresh water. *A. salmonicida* was not found in any of these fish. From this result one can say that the 99% confidence limits for the prevalence of *A. salmonicida* in ocean-caught sockeye or chum salmon are 0.00 to 1.75%. However, considering the two species together, the 99% confidence limits for ocean-caught salmon are 0.00 to 0.88%.

That is, there is a low probability of wild, ocean-caught Pacific salmon being infected with *A. salmonicida*.

---

[25] IMPORTANT NOTE; The data in this table on the prevalence of *Aeromonas salmonicida* in mature Pacific salmon were gathered from fish that had already returned from the sea to fresh water to spawn. The data thus also reflect infections that were acquired by the fish following their return to fresh water. In addition, the samples were not random samples; rather, they were, in many cases, obtained during investigations of mature fish that had developed problems (e.g., were showing pre-spawning losses, were developing external fungal lesions, etc.). The prevalences noted thus probably exaggerate the true prevalence for fish still at sea.

[26] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo BC, Canada on July 25-26, 1994 in relation to GATT Article XXII: 1.

These recent findings are consistent with Japanese reports.[27] Japanese studies have shown 33% of **spawning** wild salmon to be infected, but these fish have undergone the stresses of returning to crowded fresh water and sexual maturation and have picked up the infection on their return to fresh water and this was the conclusion of the Japanese researchers. They stated explicitly that they considered the salmon in their study were **not** infected with *A. salmonicida* until they entered the rivers to spawn.

The culture techniques used at the Department of Fisheries and Oceans Pacific Biological Station at Nanaimo are very sensitive and the researchers are experienced. It is clear that if *A. salmonicida* does occur in wild Pacific salmon at sea, it is at a very low prevalence. While the presence of carrier risk cannot be ruled out, the bacterial load of these fish must be extremely low. Experiments have shown that bacterial loading of the kidney is several hundred fold greater than in flesh (Table 3.4), so if carriage is undetectable by culturing kidney, it is improbable that there is any significant bacterial carriage in the flesh.

---

[27] Nomura T, Yoshimizu M, Kimura T. Prevalence of *Aeromonas salmonicida* in the chum salmon (Oncorhynchus keta), pink salmon (*O. gorbuscha*), and masu salmon (*O. masou*). Gyobyo Kenku 26, 139-147, 1991. [In Japanese, summary in English].

Nomura T, Yoshimizu M, Kimura T. An epidemiological study of furunculosis in salmon propagation. Pp 187-193. In Proceedings of the Oji International Symposium on Salmonid Diseases. T Kimura (ed.). Hokkaido University Press, Sapporo, Japan. 1992.

Nomura T, Yoshimizu M, Kimura, T. An epidemiological study of furunculosis in salmon propagation in Japanese rivers. Fisheries Research 17, 137-146, 1993.

Table 3.4: Viable counts on *Aeromonas salmonicida* (A.s.) in the flesh and kidney tissues of individual chinook salmon dead of experimentally induced furunculosis.[28]

Fish#	No. viable A.s. cells per g of:	
	Kidney	Flesh
1	4.0×10^8	2.0×10^4
2	9.7×10^7	8.3×10^4
3	7.6×10^8	2.6×10^5
4	2.1×10^7	1.7×10^3
5	3.0×10^7	8.0×10^2
Average	2.6×10^8	7.3×10^4

"Conclusion: The kidney tissues of fish **dead of furunculosis** contain approximately 10^3 more *A. salmonicida* cells than the flesh.

Note: Viable *A. salmonicida* counts in kidney tissues of healthy (carrier) chum and pink salmon averaged $10^{3.7}$ per g (Nomura et al, Gyobyo Kenku 26: 139-147, 1991). If the tissue distribution for **diseased** fish in the above table also holds true for **carrier** fish, this means that the carrier fish would contain fewer than 10 *A. salmonicida* cells per g of flesh. This level of *A. salmonicida* cells is not likely to provide an infectious dose when the findings of Rose et al (J. Fish Dis. 12: 573-578, 1989) are considered. After freezing, the flesh of carrier fish would contain essentially zero viable *A. salmonicida* cells because freezing decreases viable *A. salmonicida* cell numbers by two logs."

Fish disease experts with the Northern Ireland Department of Agriculture made the point that *A. salmonicida* can normally only be isolated from flesh in unhealthy fish suffering clinical furunculosis. In the carrier states, small numbers of the organism can be located in the internal organs but are unlikely to be present in the flesh.[29]

---

[28] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

[29] Menzies, F, McLoughlin, Marian, Department of Agriculture for Northern Ireland, personal communication with SC MacDiarmid, 26 August 1994.

Pacific salmon caught at sea, therefore, pose no significant risk so far as *A. salmonicida* is concerned. The prevalence of infection has been demonstrated to be extremely low. Where carrier fish have been detected, bacterial load is extremely low.

Studies at the Department of Fisheries and Oceans Pacific Biological Station at Nanaimo have demonstrated the **susceptibility of *A. salmonicida* to freezing.** They have demonstrated that a single freeze/thaw cycle will result in a two log decrease (100-fold decrease) in the number of viable bacteria in organs of infected fish (Table 3.5).

Table 3.5: Survival of *A. salmonicida* at various temperatures and following freezing/thawing (one cycle).[30]

Effect of heat

Trial 1:

% loss in viability in tryptic soy broth at 37 °C:
(No. viable cells per ml at time 0 = 2.8×10^8)

70	in	60 minutes
80	in	120 minutes
99	in	240 minutes

Trial 2:

% loss in viability in peptone-saline (0.1%-0.85%) at 40 °C:
(No. viable cells per ml at time 0 = 2.2×10^8)

98	in	30 minutes

Effect of freezing (-20 °C for 5-7 days)

Trial 1 :

Used chinook salmon dead of an infected challenge with *A. salmonicida*.
Viable *A. salmonicida* per g flesh:

---

[30] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

Fish No.	Before freezing	After freezing	% loss
Trial A			
1	1.1×10^4	6.7×10^1	99.4
2	3.0×10^4	2.0×10^2	99.3
3	5.2×10^3	0	100.0
4	5.8×10^3	0	100.0
5	1.8×10^4	0	100.0
Trial B			
1	1.2×10^2	NT	-
2	7.0×10^2	1.3×10^2	81.0
3	1.4×10^6	5.4×10^4	96.2
4	3.5×10^5	1.7×10^3	99.5
5	1.7×10^4	NT	-
6	8.0×10^4	NT	-
Trial C (with *A. salmonicida* in a 20% v/v flesh homogenate)			
	7.5×10^6	2.3×10^5	97.0
Trial d (with *A. salmonicida* in peptone-saline (0.1% - 0.85%))			
	8.3×10^8	2.6×10^4	99.9

"**Note:** In trial a, the temperature during freeze storage may have risen above -20 °C for short periods as the freezer door was briefly opened three times during the storage period. Note also, "before freezing" counts are given with some samples that were not counted "after freezing" to provide additional data on the *A. salmonicida* levels that occur in the flesh of Pacific salmon dead of furunculosis."

The **dose of *A. salmonicida* required to infect** salmon is relatively high. At Nanaimo, immersion of Pacific salmon in a concentration of 10^3 cells/ml *A. salmonicida* for 2-3 days failed to establish infection in stressed fish. Working with Atlantic salmon, Rose and colleagues[31] were unable to cause either clinical or subclinical infections by immersion in $3x10^3$ cfu/ml/day for 3 days. Neither were they able to infect salmon by immersion in 10^2 cfu/ml for **1 week.**

It is possible to infect Pacific salmon by mouth with *A. salmonicida*; work at Nanaimo has shown that a dose of 10^2 cells/kg by stomach tube will establish infection, but not readily. Rose and colleagues showed that infection of Atlantic salmon by intragastric intubation required $>10^5$ cfu/fish.

Lower doses by injection will establish infection more easily, but bear little relation to the natural situation.

Survival of *A. salmonicida* in the environment is very limited in the face of competition from other micro-organisms. Studies demonstrating prolonged survival in different types of water (fresh, brackish, sea water) have been in sterile media. When other micro-organisms are present, *A. salmonicida* survives only briefly.

Claims regarding the prolonged 'survival' of *A. salmonicida* in sediments[32] demonstrate a failure to appreciate the epidemiology of the disease or of the testing methods used. Indirect fluorescent antibody testing (IFAT) has detected **cells** of *A. salmonicida* in sediments. However, more advanced techniques have been unable to demonstrate the presence of **viable** *A. salmonicida* in such sediments. Viable organisms are not demonstrable in the environment. Although *A. salmonicida* cells may be detectable in fresh and sea water for many days using specialised techniques, they are non-culturable, have no detectable ability to respire or metabolise, are probably not viable

---

[31] Rose, AS, Ellis, AE, Munro, ALS. The infectivity by different routes of exposure and shedding rates of *Aeromonas salmonicida* subsp. *salmonicida* in Atlantic salmon, *Salmo salar* L., held in sea water. Journal of Fish Disease 12, 573-578, 1989.

[32] Anderson, C D. The risk of introducing exotic disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

and are probably unable to infect fish.[33] It is probable, in short, that *A. salmonicida* subsp. *salmonicida* does not enter a dormant state.

In an article arguing against the importation of salmon flesh[34] it was claimed that *A. salmonicida* not only may survive in mud and sediments, but may actually multiply. In support of this claim the writer cited a study carried out by Dubois-Darnaudpeys. However, an examination of that worker's report[35] reveals that Dubois-Darnaudpeys was studying the survival and multiplication of *A. salmonicida* in an **abiotic** environment; that is, an environment devoid of any competing bacteria. The relevance, therefore, of these findings to the natural environment must be questioned.

Similarly, some of the claims made about the concentration of *A. salmonicida* likely to be found in infected tissues[36] are quite unrealistic. The highest concentration of *A. salmonicida* isolated from a lesion in **Pacific** salmon at Nanaimo is 10^6 organisms/g, not the 10^{10} organisms/g claimed by Anderson (a claim based on work by Rose[37] in **Atlantic** salmon). Infected Pacific salmon have much lower numbers of *A. salmonicida* in their tissues (Tables 3.4 and 3.5).

---

[33] Rose, AS, Ellis, AE, Munro, ALS. Evidence against dormancy in bacterial fish pathogen *Aeromonas salmonicida* subsp. *salmonicida*. FEMS Microbiology Letters 68, 105-108, 1990.

Effendi, I, Austin, B. Survival of the fish pathogen *Aeromonas salmonicida* in seawater. FEMS Microbiology Letters 84, 103-106, 1991.

Morgan, JAW, Cranwell, PA, Pickup, RW. Survival of *Aeromonas salmonicida* in lake water. Applied and Environmental Microbiology 57, 1777-1782, 1991.

Rose, AS, Ellis, AE, Munro, ALS. The survival of *Aeromonas salmonicida* subsp. *salmonicida* in sea water. Journal of Fish Diseases 13, 205-214, 1990.

[34] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

[35] Dubois-Darnaudpeys, Annie. Epidemiologie de la furonculose des salmonides. I. Etude experimentale des conditions de survive et de multiplication de *Aeromonas salmonicida* dans un environnement abiotique. Bull. Fr. Piscic. 264, 121-127, 1977.

[36] Anderson, C D. The risk of introducing exotic disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

[37] Rose AS, Ellis AE, Munro ALS. The infectivity by different routes of exposure and shedding rates of *Aeromonas salmonicida* in Atlantic salmon, *Salmo salar* L., held in sea water. Journal of Fish Diseases 12, 573-578, 1989.

At the July 1994 meeting at Nanaimo, Evelyn discussed the **host range** of *A. salmonicida*. Where data are adequate to properly interpret reports in the literature, it is clear that non-salmonids are nearly always infected with "atypical" *A. salmonicida* (Austin's statements[38] about the host range misrepresent the situation and are a rather non-critical summary of the published literature). Non-salmonids become infected with "typical" *A. salmonicida* under unnaturally stressful conditions or when in close association with infected salmonids. That is, "typical" *A. salmonicida* is not naturally a cause of **disease** in non-salmonids and although 2-5% of *A. salmonicida* infections in non-salmonids may be due to "typical" stains, these infections do not usually cause clinical manifestations.

Studies conducted at Nanaimo[39] have demonstrated that filter feeders, such as mussels, take up the pathogen *Renibacterium salmoninarum* as it is shed into the aquatic environment and destroy it. Evelyn, one of the researchers involved in that study, believes that *A. salmonicida* is similarly destroyed in filter feeders. This is further evidence against the likelihood of its being introduced via sewage outfalls.

Wild, ocean-caught Pacific salmon from the west coast of North America are unlikely in introduce *A. salmonicida* when imported as headless, eviscerated fish because;

- Infection with "typical" *A. salmonicida* is very uncommon in ocean-caught Pacific salmon.
- Infection with "atypical" *A. salmonicida* has not been recorded in ocean-caught Pacific salmon in North American waters.
- In fish infected with *A. salmonicida* the bacterial load in muscle is 1,000 to 100,000 times lower than in visceral tissues which are removed during processing.
- Fish clinically diseased with furunculosis will not be in suitable condition to be graded for sale fresh or frozen.
- The bacterial load of *A. salmonicida* in the tissues of carrier fish is orders of magnitude less than in the tissues of clinically diseased fish.

---

[38] Austin B, Austin DA. Bacterial Fish Pathogens. Disease in Farmed and Wild Fish. Second edition. 384 pages. Ellis Horwood, New York. 1993.

[39] Paclibare JO, Evelyn TPT, Albright LJ and ProsperiPorta L. Clearing of the kidney disease bacterium Renibacterium salmoninarum from seawater by the blue mussel Mytilus edulis, and the status of the mussel as a reservoir of the bacterium. Diseases of Aquatic Organisms, 18, 129-133, 1994.

- Salmonids are not readily infected with *A. salmonicida*, requiring prolonged immersion in high concentrations for infection to become established.

- Non-salmonids are even less likely to become infected with *A. salmonicida*.

- Survival of *A. salmonicida* in the environment is very limited, with the organism being unable to survive competition from environmental micro-organisms.

- However, because *A. salmonicida* is the one pathogen of salmon likely to be present in a high concentration in the muscles of diseased fish, it is the subject of a separate quantitative risk assessment (see Part II below).

3.9 **Bacterial kidney disease (BKD)**

Department of Fisheries and Oceans staff have examined by laboratory techniques nearly 22,000 returning Pacific salmon for evidence of infection with *Renibacterium salmoninarum*, the bacterium which causes BKD. These fish have been examined by Gram stain and culture of kidneys, IFAT and, more recently, ELISA.

This surveillance has found that 4.6% of salmon show evidence of *R. salmoninarum* infection **once they have returned to fresh water** (Table 3.6). Many of these fish are without lesions. Most infections are seen in coho salmon which have been around for a while in fresh water. However, some evidence of *R. salmoninarum* infection is seen in salmon very soon after they return to fresh water; that is, before they can have acquired infection in fresh water. Some fish are, therefore, infected before they enter fresh water.

Table 3.6: Prevalence of *Renibacterium salmoninarum*[40] (R.s) in wild, ripe or spent salmon in BC from 1972 to September 1993.[41]

Species	Total No. of fish	No. of fish with microscopy done	No. of R.s. positive fish	No. of fish with flesh lesions
Pink	2,510	2,509	4	0
Chum	2,123	2,089	2	0
Coho	10,072	9,187	836	6
Chinook	5,908	5,330	98	1
Sockeye	5,371	2,884	71	0
Totals	25,984	21,999	1,011	7
Percents		84.7	4.6	0.03

R. salmoninarum infection is a salmonid disease. Natural infections of non-salmonids are not found. However, experimental inoculation or prolonged cohabitation with infected salmonids can result in the infection of some non-salmonid fishes.

The tissue distribution of *R. salmoninarum* has been studied in salmon dying of BKD. Studies at Nanaimo have shown that the number of bacteria in flesh is a least 1,000 times lower than in kidneys (Table 3.7).

---

[40] IMPORTANT NOTE; The data in this table on the prevalence of *Renibacterium salmoninarum* in mature Pacific salmon were gathered from fish that had already returned from the sea to fresh water to spawn. The data thus also reflect infections that were acquired by the fish following their return to fresh water. In addition, the samples were not random samples; rather, they were, in many cases, obtained during investigations of mature fish that had developed problems (e.g., were showing pre-spawning losses, were developing external fungal lesions, etc.). The prevalences noted thus probably exaggerate the true prevalence for fish still at sea.

[41] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994 in relation to GATT Article XXII:1.

Table 3.7: Viable counts on *Renibacterium salmoninarum* (R.s.) in the flesh and kidney tissues of individual chinook salmon dead of experimentally induced bacterial kidney disease (BKD).[42]

Fish #	No. viable R.s. cells per g of:	
	Kidney	Flesh
1	6.8×10^8	2.6×10^5
2	8.5×10^8	5.6×10^6
3	2.7×10^9	7.4×10^5
4	1.5×10^{10}	2.4×10^7
5	6.2×10^9	6.6×10^6
6	2.0×10^9	7.1×10^6
Average	4.6×10^9	7.4×10^6

"Conclusion: The flesh of fish **dead of BKD** contains approximately 10^3 times fewer *R. salmoninarum* cells than the kidney tissue."

Dr Trevor Evelyn commented "We have no hard data on the numbers of *R. salmoninarum* cells likely to be present in the kidney or flesh of healthy (*R. salmoninarum* carrier) fish. However, based on our inability to detect the bacterium (using fluorescent antibody techniques = FAT and culture) in the kidneys of any of 300 sockeye and 300 chum salmon recently sampled at sea in British Columbia, we would conclude that the number of *R. salmoninarum* present in the kidneys must have been extremely low (culture would have been capable of detecting viable cells had they been present in the kidney at levels of 10^3 per g). By ELISA, 3.3% of the sockeye and 4.7% of the chum salmon were *R. salmoninarum* carriers. Assuming a kidney-to-flesh distribution of *R. salmoninarum* cells similar to that found for diseased fish in the above table, the foregoing results suggest that the flesh of the carrier fish was essentially *R. salmoninarum*-free. It should be noted, however, that the kidneys of mature Pacific salmon caught at sea have on, occasion, contained *R. salmoninarum* cells detectable by FAT. This means that low numbers of *R. salmoninarum* cells could conceivably sometimes be present in the flesh of ocean caught Pacific salmon. However, this has never been demonstrated, and their numbers would be significantly reduced by freezing (see data in [Table 3.8 of this analysis] and also Olsen et al, Diseases of Aquatic Organisms 14, 207-212, 1992)."

---

[42] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held In Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

Fish health experts with the Northern Ireland Department of Agriculture support the view that *R. salmoninarum* can be found in muscle lesions but, if only grossly normal, eviscerated fish were accepted, the risks would be small.[43] It is worth noting that fish sick or dying of BKD are not likely to be traded as first grade fish; the flesh is unacceptable.

The immunological tests used to test fish for the presence of *R. salmoninarum*, such as ELISA and IFAT, are not very specific (that is, they commonly produce false positive results). This has been demonstrated when ELISA and IFAT results are checked against the newer PCR (polymerase chain reaction) tests.

In the sections on *A. salmonicida* reference was made to the recent survey in which 300 sockeye and 300 chum salmon were taken at sea and tested for the presence of diseases. Of the 600 salmon tested by ELISA for evidence of BKD, 3-5% were positive (Table 3.8). However, these results could not be confirmed by the more specific IFAT, nor could *R. salmoninarum* be cultured from their tissues.

Surveys in Alaska have found that around 9% of salmon may be infected with *R. salmoninarum* **once they have returned to fresh water** (a stressful situation which results in the recrudescence of latent infections and the spread of infections). As many as 100% of trout are found to be infected in similar surveys.

R. salmoninarum does not survive long in fresh water or sea water. In sterile water its survival is relatively short; in non-sterile water it is rapidly overgrown by other micro-organisms.

Transmission of *R. salmoninarum* is vertically through the egg or horizontally when fish cohabit.

In the environment *R. salmoninarum* **is taken up by filter feeders such as mussels** which readily digest it, thus removing infectivity from the aquatic environment.[44]

Experimentally, infection may be achieved by immersion of susceptible salmon in a concentration of 10^4 cells/ml. It may be possible to transmit infection at lower doses, but these have not been examined.

It is the vertical transmission which is responsible to *R. salmoninarum's* success as a pathogen.

---

[43] Menzies, F, McLoughlin, Marian, Northern Ireland Department of Agriculture, personal communication with SC MacDiarmid, 26 August 1994.

[44] Paclibare JO, Evelyn TPT, Albright LJ and ProsperiPorta L. Clearing of the kidney disease bacterium Renibacterium salmoninarum from seawater by the blue mussel Mytilus edulis, and the status of the mussel as a reservoir of the bacterium. Diseases of Aquatic Organisms 18, 129-133, 1994.

BKD is not a problem in Atlantic salmon. It has caused problems, however, when culturing of Pacific salmon was attempted. BKD was the number one disease problem in farmed Pacific salmon in British Columbia. In Atlantic salmon, *R. salmoninarum* is present but very seldom causes disease. It is not a problem in trout.

R. salmoninarum resists freezing better than *A. salmonicida*. Studies at Nanaimo have demonstrated that a single freeze/thaw cycle inactivates around 80% of renibacteria in tissues of infected salmon (Table 3.8). Smoking salmon at 25 °C results in a rapid decrease in the bacterium. The organism is rapidly destroyed at 37 °C (Table 3.8), as is *A. salmonicida* (Table 3.5).

Table 3.8: Recent data on *Renibacterium salmoninarum* from the Pacific Biological Station laboratory.[45]

1) Survival of *R. salmoninarum* in the flesh of chinook salmon following freezing (at-20 °C) and thawing (one cycle). Tests were conducted on fish that had died of BKD following challenge by prolonged association with *R. salmoninarum*-infected fish.

Average number viable *R. salmoninarum* cells/g flesh for 19 unfrozen fish = 8.0×10^6

Average number of viable *R. salmoninarum* cells/g flesh for 34 frozen fish = 1.8×10^6

% loss in viability on freezing = 77.5.

2) Survival of *R. salmoninarum* at various temperatures while suspended in KD broth (a medium designed for growing the bacterium).

Temperature (°C)	% loss at indicated time (hr)
4.0	0 at 71
15.0	0 at 71 (actually, cells grew!)
25.0	97 at 71
35.0	100 at 4.5

3) Prevalence of *R. salmoninarum* in unfrozen kidney tissue of BC salmon caught at sea shortly before their entry into fresh water to spawn.

Tested: 300 sockeye salmon (Alberni Inlet)

% *R. salmoninarum* positive by ELISA	=	4.7
% *R. salmoninarum* positive by culture	=	0
% *R. salmoninarum* positive by DFAT	=	1.0

Tested: 300 chum salmon (Satellite Channel)

% *R. salmoninarum* positive by ELISA	=	3.3
% *R. salmoninarum* positive by culture	=	0
% *R. salmoninarum* positive by DFAT	=	0

---

[45] Data provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

Note: None of the sockeye and chum salmon proved positive for the furunculosis agent *A. salmonicida* when unfrozen kidney samples were tested by culture.

In studies at the Pacific Biological Station, Nanaimo, researchers have **never been able to culture *R. salmoninarum* from the flesh of salmon** unless they are actually dying of BKD (and hence unacceptable for sale). In fish dying of BKD, bacterial counts in the order of 10^9/g in kidney and 10^6/g in muscle can be expected. In carrier fish, it is difficult to culture the organism from any tissue, including kidney, the numbers are so low (the threshold sensitivity of the culture technique is around 1,000 bacteria/g in the hands of the Nanaimo researchers).

There is an extremely low probability of finding *R. salmoninarum* in the muscle of fish not actually dying of BKD septicemia, in which case the flesh is grossly affected and hence is aesthetically unacceptable and not harvested.

The presence of *R. salmoninarum* in North American waters should not be used as a barrier to importing headless, eviscerated wild, ocean-caught Pacific salmon because;

- Carriers of *R. salmoninarum* are present at low prevalence only in ocean-caught Pacific salmon.
- The bacterium is not detectable in flesh of infected salmon unless they are actually dying from BKD, in which case the flesh is aesthetically unacceptable and so is rejected or downgraded by the processors.
- The most important route of transmission of *R. salmoninarum* is vertically; horizontal transmission appears to depend on cohabiting and rather high concentrations of the bacterium.
- *R. salmoninarum* does not survive at all well in the environment, being readily out-competed by other micro-organisms and removed and destroyed by filter-feeders such as mussels.
- *R. salmoninarum* is a relatively fragile bacterium, its numbers being reduced or eliminated by freezing, smoking at 25 °C or temperatures of 37 °C or higher.

3.10 Enteric redmouth (ERM)

Enteric redmouth is caused by *Yersinia ruckeri* which is endemic in New Zealand but which does not cause problems in British Columbia.

Several species may act as carriers of *Y. ruckeri*, including some birds and mammals.

Y. ruckeri requires relatively high water temperatures to cause problems and the water temperatures in British Columbia are not high enough.

ERM or *Y. ruckeri* are not seen in Pacific salmon in British Columbia. Researchers at Nanaimo have examined 21,495 salmon returning to fresh water by bacterial culture of the kidney and found *Y. ruckeri* carriage in 0.31% only. Only one of these was carrying the bacterium in numbers high enough to attribute disease to.

Table 3.9: Prevalence[46] of *Yersinia ruckeri* (Y.r.) in wild, ripe or spent salmon in BC from 1972 to 1993.[47]

Species	No. of Fish	No. of fish cultured	No. positive for Y.r.
Pink	2,510	2,490	2
Chum	2,123	2,111	1
Coho	9,750	8,825	13
Chinook	5,908	5,381	33
Sockeye	5,371	2,688	17
Totals	25,662	21,495	66
Percents		83.8	0.31

"**NOTE**; Only one fish appeared to have died of *Y. ruckeri*. All other *Y. ruckeri* positive fish appeared to be carriers."

The Nanaimo researchers have not looked for *Y. ruckeri* in salmon in the ocean before they have begun their return to fresh water.

Y. ruckeri is found in fish in many fresh water locations in Canada.

---

[46] The data in the table on the prevalence of *Yersinia ruckeri* in mature Pacific salmon were gathered from fish that had already returned from the sea to fresh water to spawn. The data thus also reflect infections that were acquired by the fish following their return to fresh water. In addition, the samples were not random samples; rather, they were, in many cases, obtained during investigations of mature fish that had developed problems (e.g., were showing pre-spawning losses, were developing external fungal lesions, etc.). The prevalences noted thus probably exaggerate the true prevalence for fish still at sea.

[47] Data provided to Australian officials at the Canada-Australia Technical Meeting Held in Nanaimo BC, on July 25-26, 1994 in relation to GATT Article XXII:1.

The presence of *Y. ruckeri* in North America should not be raised as a barrier to importing wild, ocean-caught Pacific salmon because;

- *Y. ruckeri* is endemic in New Zealand.
- *Y. ruckeri* infection is extremely uncommon (less than 0.33%) in ocean-caught Pacific salmon.

3.11 Salmonid rickettsial septicaemia

Salmonid rickettsial septicaemia has only very occasionally been a cause of problems in chinook salmon held in netpens. In British Columbia, salmonid rickettsial septicaemia is definitely not the problem it is in Chile, leading to speculation that the organisms are different strains.

When salmonid rickettsial septicaemia has occurred in British Columbia it has been as a low-grade chronic condition. In Atlantic salmon (farmed) it has presented as a low-grade mortality of around 0.1%/day in conjunction with other diseases such as vibriosis and BKD.

Salmonid rickettsial septicaemia has not been seen in ocean-caught salmon.

Experimentally, it has been possible to infect several salmonid species by injection. Flounders can be infected by injection. Salmonid rickettsial septicaemia can be spread among farmed salmonids by cohabitation. It is probably not vertically transmitted.

Salmonid rickettsial septicaemia has, on a single occasion in Chile, occurred in fresh water (but some have questioned the accuracy of the diagnosis).

It is likely that an as-yet-unidentified marine reservoir exists for the agent. The causative agent of salmonid rickettsial septicaemia is an intracellular parasite.

Clinical disease is infected fish is obvious, with crater-like lesions in the liver.

There appears to be very significant difference between the variants of this infection as seen in Chile and North America. In Chile coho salmon have been affected.

John Nickum[48] confirmed that salmonid rickettsial septicaemia has not been detected in US waters.

---

[48] Dr John G Nickum, United States Fish and Wildlife Service at Canada-Australia Salmon Technical Meeting under auspices of GATT Article XXII. Nanaimo, British Columbia, July 25-26, 1994.

The presence in North American waters of salmonid rickettsial septicaemia should not be raised as a barrier to the importation of wild, ocean-caught Pacific salmon because;

- Salmonid rickettsial septicaemia, as seen in British Columbia, is a minor condition only affecting farmed salmon.
- Salmonid rickettsial septicaemia has not been detected in ocean-caught Pacific salmon in Canadian or US waters.
- Salmonid rickettsial septicaemia does not readily spread among fish.

3.12 **Rosette agent**

The so-called rosette agent has never been detected in British Columbia although there has been a single identification in Puget Sound.

The causative agent has not been characterised. However, the signs of infection with rosette agent are very obvious and the disease would have been noticed if present in the thousands of fish screened in the Department of Fisheries and Oceans salmon surveillance program.

Rosette agent should not be raised as a barrier to the importation of wild, ocean-caught Pacific salmon from Canada and the US because;

- Rosette agent has never been detected in British Columbia, despite surveillance.
- Rosette agent has been detected on a single occasion only in Puget Sound.

3.13 ***Loma salmonae***

Loma salmonae is a microsporidium. It is a gill parasite found in Pacific salmon on the west coast of the US and Canada and in Japan. The parasite *L. salmonae* is found inside enlarged cells called xenomas. It has never been seen in ocean-caught fish, but causes occasional problems in fresh water species.

L. salmonae has a direct life cycle. Spores are released from the xenomas to infect cohabiting fish. The parasite has been recorded as spreading in sea water in netpens which have received infected smolt from fresh water hatcheries.

L. salmonae has never been found in flesh of Pacific salmon (although it has been recorded in the flesh of some species of trout). In processing, any cysts released from the gills would be washed off.

The presence of *Loma salmonae* in salmonids in North America is no reason not to permit the importation of wild, ocean-caught Pacific salmon because;

- *Loma salmonae* is not an infestation of ocean-caught Pacific salmon.
- *Loma salmonae* is a gill parasite and the gills are removed during the process of evisceration; the parasite has never been found in flesh of Pacific salmon.

3.14 *Enterocytozoon salmonis*

Enterocytozoon salmonis is a microsporidian parasite. It affects some Pacific salmon as well as rainbow trout.

E. salmonis is an intracellular parasite of red blood cells and is found in the waters of British Columbia and the US Pacific coast. *E. salmonis* infection has been found in salmon in fresh water and sea water. It has sometimes been seen in association with plasmacytoid leukaemia.

The life cycle of *E. salmonis* is as yet undefined, but is assumed to be direct, like all microsporidians. Little is known about its stability. The route of infection is unknown, but is horizontal.

The presence of *Enterocytozoon salmonis* in North American waters is no reason not to permit the importation of wild, ocean-caught Pacific salmon because;

- *Enterocytozoon salmonis* is not the cause of significant disease in salmonids.
- *Enterocytozoon salmonis* is an intracellular parasite of red blood cells and is unlikely to be present to a significant extent in muscle (other than in residual blood).

3.15 **Edwardsiellosis**

Although salmonids **may** be infected with *Edwardsiella tarda* or *E. ictaluri*, edwardsiellosis is primarily a warm water disease of catfish, eels etc. Edwardsiellosis is not found as a disease in British Columbia. *Edwardsiella* species have been isolated

in British Columbia, but not from salmonids.[49] In the US, edwardsiellosis is not a disease of salmon or of cold water fish.[50]

Edwardsiella species can be carried in the intestines of birds and some mammals, including humans. That is, *Edwardsiella* species could be brought into New Zealand by travellers.

Imports of wild, ocean-caught Pacific salmon do not pose a risk so far as edwardsiellosis is concerned because;

- Edwardsiellosis is a disease of warm water fishes and Pacific salmon are caught in cold waters.
- Although salmon can be infected with *Edwardsiella* species, such infections are rare and have not been recorded in British Columbia.

3.16 **Proliferative kidney disease (PKD)**

PKD is a European problem, not a problem of the Pacific coast of North America.

PKD is caused by the kidney being infected by the developmental stage of a myxosporean parasite.

PKD has been found in North America, where some outbreaks have been recorded in juvenile fish in fresh water hatcheries.

PKD does not affect flesh; the parasite is not found in muscle tissue.

PKD is a warm water disease, requiring temperatures greater than 15 °C for disease to occur.

The life cycle of the myxosporean parasite is indirect, requiring an alternate host, a fresh water oligochaete which has not yet been identified. Myxosporean alternate hosts are specific, so the introduction of PKD would require the introduction not only of viable myxosporeans but also the specific oligochaete host. An unlikely coincidence.

---

[49] Dr Trevor Evelyn (Canadian Department of Fisheries and Oceans) at Canada-Australia Salmon Technical Meeting under auspices of GATT Article XXII. Nanaimo, British Columbia, July 25-26, 1994.

[50] Dr John G Nickum (United States Fish and Wildlife Service) at Canada-Australia Salmon Technical Meeting under auspices of GATT Article XXII. Nanaimo, British Columbia, July 25-26, 1994.

PKD is not seen in adult (market size) fish, only in juveniles in fresh water hatcheries. PKD is not an issue in salmon farming in British Columbia.

The presence of PKD in North America should not be a cause for concern so far as imports of wild, ocean-caught Pacific salmon are concerned because;

- Proliferative kidney disease is uncommon in North American salmonids.
- Proliferative kidney disease is not a disease of adult, market size fish.
- Proliferative kidney disease is caused by a parasite confined to kidney tissue, which is removed during processing.
- The myxosporean parasite which causes PKD requires a specific oligochaete alternate host for its indirect life cycle.

3.17 *Kudoa thyrsites*

Kudoa thyrsites is a marine parasite, widespread in the Pacific ocean. It has been found off southern Australia, South Africa, Japan, Canada, the US etc. It is probable that appropriate surveying would detect *K. thyrsites* in marine fish off New Zealand's coasts.

K. thyrsites is a marine myxosporean, life cycle not defined. It lodges in the muscles of fish causing rapid autolysis of flesh once the fish has been caught.

If New Zealand waters were free of *K. thyrsites* the parasite would more likely be introduced in ships' ballast water or in imports of marine fish.

Kudoa thyrsites is no reason not to import wild, ocean-caught Pacific salmon because;

- *Kudoa thyrsites* is present in marine fish throughout the southern ocean.
- *Kudoa thyrsites* could equally be introduced in any marine fish, or, more likely, in ships' ballast water.

3.18 *Ceratomyxa shasta*

Ceratomyxa shasta is found only on the west coast of North America. It is a fresh water myxosporean requiring a specific oligochaete intermediate host.

C. shasta is present in wild stocks of salmon in British Columbia. Juveniles may be infected when they leave fresh water, but all infected juveniles die when they go to sea; that is, mortality is 100% and one never sees adult, market size salmon infected with *C. shasta* when they are at sea.

Adult salmon can become infected with *C. shasta* when they return to an infected river to spawn.

C. shasta enters the fish through the alimentary tract and then spreads throughout all the viscera.

Infection with *C. shasta* is not a problem in hatcheries in British Columbia.

Large volumes of Pacific salmon have been exported to many countries without *C. shasta* being introduced (see Section 2.2).

Ceratomyxa shasta is no reason not to import wild, ocean-caught Pacific salmon because;

- *Ceratomyxa shasta* has never been spread beyond the west coast of North America, despite huge exports of Pacific salmon to many countries.
- *Ceratomyxa shasta* is a fresh water parasite requiring a specific intermediate host; that is, the parasite and the intermediate host would need to be introduced for disease to establish.
- *Ceratomyxa shasta* is never seen in adult, market-size fish caught at sea.

3.19 *Herpesvirus salmonis*

Herpesvirus salmonis does not occur in British Columbia. It has been identified once in the US, in steelhead trout. It is not a problem. It was associated with post-spawning mortalities on the occasion it was detected in the US.

3.20 Vibriosis

Vibriosis caused by *Vibrio ordalii* and *V. anguillarum* occurs in New Zealand, where it is said to have little economic impact on salmon farming.[51] In British Columbia vibriosis is seldom a problem in salmon hatcheries and is not seen in adult salmon returning from sea.

The bacterial culture techniques used to screen 21,495 returning salmon for *Yersinia ruckeri* would have detected *Vibrio* species if present. However, none were isolated.

---

[51] Anderson, C D. The risk of introducing exotic disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

3.21 Hitra disease

Hitra disease, also known as cold-water vibriosis, is a condition of Atlantic salmon caused by *Vibrio salmonicida*. It has recently been identified as a cause of significant mortalities among Atlantic salmon on Canada's east coast.

Hitra disease does not occur on the west coast of North America and the extensive surveillance of Pacific salmon for bacterial diseases (referred to in previous sections) has failed to detect *V. salmonicida*.

The presence of Hitra disease in Atlantic salmon on the east coast of Canada is no reason to block the importation of Pacific salmon from the west coast.

3.22 *Henneguya salmonicola*

Henneguya salmonicola is a myxosporean protozoan parasite which may be found in the muscle and skin of wild salmon and sea trout. It may be responsible for a condition referred to as milky flesh disease. Transmission occurs to juveniles in fresh water.

Evidence suggests that *Henneguya*, like other myxosporeans, requires a specific oligochaete worm to complete its life cycle. The requirement for this specific intermediate host is the probable reason for the parasite's very limited distribution; it may occur in one river while the neighbouring river remains free of *Henneguya*. This also explains why much of the world's waterways remain free of the parasite.

Henneguya salmonicola has not spread, despite years of exports of eviscerated wild, ocean-caught, Pacific salmon worldwide. Japan and several European countries (see Table 2.1) are major importers of Pacific salmon but have remained free of the parasite.

3.23 Parvicapsular disease

Protozoa of *Parvicapsula* species have been found infecting the kidney of netpen-reared salmon but the role of the parasite in disease is unclear as concurrent infection with *Renibacterium* and *Vibrio* often occur in outbreaks of disease. *Parvicapsula* has been isolated from a number of species of salmon and trout.[52]

---

[52] Kent, M L, Diseases of Seawater Netpen-reared salmonid Fishes in the Pacific Northwest. 76 pages. Department of Fisheries and Oceans, Nanaimo, British Columbia. 1992.

A *Parvicapsula* species has been recorded in Pacific cod caught near netpens containing infected coho salmon and it has been suggested that Pacific cod could be a reservoir for the parasite. *Parvicapsula* species has also been found in wild salmon off the coast of British Columbia.

The life cycle of this parasite is complicated and the mode of transmission is unclear. *Parvicapsula* species is believed not to be able to produce its spore stage in salmonids and, because the spores are the infectious stage of the parasite's life cycle, flesh containing the parasite would not be able to act as a vehicle to introduce the disease.

The presence of this parasite in North American waters is not a valid reason to bar the importation of headless, eviscerated salmon. It is present uncommonly in a tissue which is removed during processing (kidney) and is believed to be unable to reach an infectious stage in salmonids.

PART II: QUANTITATIVE RISK ASSESSMENT

4. Furunculosis

It is clear from the information presented above that, because of the nature of the disease, furunculosis is the one fish disease most likely to be introduced in the flesh of imported salmon because of the intramuscular location of the causative agent.

Furunculosis is a septicaemic disease caused by the Gram-negative bacterium *Aeromonas salmonicida*. The disease has long been recognised as a serious problem in wild and farmed salmonid fish in fresh water. Fish may carry *A. salmonicida* without clinical signs.

Fish may become infected through close contact with diseased fish, by ingesting infected material or by entry of infection through abrasions or wounds caused by external parasites.

A. salmonicida has been found in a large number of fish species, including marine species. Three subspecies of *A. salmonicida* are currently recognised and all are capable of infecting salmonids. However, in pen-reared salmon in the Pacific northwest of Canada, only one subspecies has been encountered as a problem: *A. salmonicida* subsp. *salmonicida*.

The different strains of *A. salmonicida* have different affinities for salmonids. Those strains which commonly affect salmonids under natural conditions are referred to as "typical" strains and, while they **may** infect non-salmonid species, such infections are not the rule. Similarly, the so-called "atypical" strains of *A. salmonicida* which, under natural conditions affect non-salmonid fishes, do not readily infect salmonids (see below Section 5.3.1).

It is generally accepted that infected fish are the main reservoir of infection. Fish clinically affected by, and dying from, furuculosis produce and release large numbers of *A. salmonicida* (10^5-10^8 cfu/fish/hour)[53]into their environment. It is this **sustained** release of large numbers of bacteria which results in the spread of infection, despite the poor survival of the pathogen in an environment where other bacteria are present.

In very acute outbreaks of furunculosis the only signs noticed may be a sudden increase in the number of fish deaths. In less acute cases fish may first exhibit anorexia, darkening of the skin and lethargy. Later, haemorrhages and reddening of the skin and fins may be seen. More chronically-affected fish may exhibit furuncles which are boil-like swellings which may rupture leaving large ulcers. However,

---

[53] Rose, AS, Ellis, AE, Munro, ALS. The infectivity by different routes of exposure and shedding rates of *Aeromonas salmonicida* subsp. *salmonicida* in Atlantic salmon, *Salmo salar* L., held in sea water. Journal of Fish Disease, 12, 573-578, 1989.

furuncles are uncommon and more commonly the lesions in chronically affected fish are areas of haemorrhagic, liquefied muscle with little host response.

4.1 Why furunculosis?

There are a number of diseases which affect salmon on the west coast of Canada, and some of these have not yet been recorded in New Zealand waters. Some of these are potentially more damaging than furunculosis, should they be introduced. Nevertheless, *A. salmonicida* was chosen as the organism on which this risk assessment is based because it is clearly the pathogen most likely to be introduced in the commodity under discussion, namely headless, eviscerated fish.

None of the other pathogens affect the muscle tissues of salmon to the same extent as *A. salmonicida* and none is present in such large numbers in muscle tissue. Selection of *A. salmonicida* as the exotic pathogen most likely to be introduced through fillets constitutes a "worst case" risk estimate. The reasons for this have been clearly outlined in the preceding sections of this analysis.

4.2 "Atypical" *Aeromonas salmonicida*

Currently three subspecies are recognized:

A. salmonicida subspecies *salmonicida*
A. salmonicida subspecies *achromogenes*
A. salmonicida subspecies *masoucida*

Furunculosis of salmonids is caused by *A. salmonicida* subspecies *salmonicida*, the so-called "typical" strain of the bacterium. The other, "atypical", strains of *A. salmonicida* cause outbreaks of disease in species other than salmonids. The "typical" strain of *A. salmonicida* is in reality atypical, in so far as it is less common in nature than the so-called "atypical" strains.[54] The "atypical" strains have very wide distribution in marine fish and fresh water fish the world over. While the "typical" strain of *A. salmonicida* is the one which causes furunculosis in salmon and may cause localised "ulcer disease" in non-salmonid fishes, it is much less common in nature than the so-called "atypical" strains.

On the west coast of Canada so-called "atypical" strains of *A. salmonicida* are common in herring while on the east coast of North America these strains are common in cod. However, in both these species the occurrence of lesions is uncommon. **"Atypical" strains of *A. salmonicida* have not been isolated from Pacific salmon** in British

[54] Dr Trevor P Evelyn, Pacific Biological station, Nanaimo, British Columbia, personal communication with SC MacDiarmid during a visit to Nanaimo, November 1993.

Columbia, despite the extensive surveillance program described in preceding sections (see especially Table 3.3).

4.3 The method

One of the principles established under the Uruguay Round of the General Agreement on Tariffs and Trade (GATT) is that import measures applied in the name of protecting animal health should be based on sound science and risk assessment principles and should not form disguised barriers to trade.[55] Risk analysis is a decision-making tool which employs science but is not itself a pure science. It is a blend of art and science which is used as a way of examining the components of risk in a structured way.[56] Risk analysis must deal with situations as they arise and must tolerate the mathematical limitations of the disease prevalence estimates or other data.

By breaking down the overall risk into its various components (the "input variables") a risk assessment is designed to focus debate on the specific risk steps which must be met before a disease introduction occurs.

Until relatively recently risk analyses have assumed and combined a series of average, conservative and worst-case values to derive a point estimate of risk that is seen to be conservative. However, such an approach has major limitations, making it very difficult to put estimates into perspective and often, through their bias, focusing on scenarios which will rarely, if ever, happen.[57] Rather than focusing solely on 'worst-case' scenarios risk analyses based on a Monte Carlo simulation using variables which are defined by a range of values give decision makers and stakeholders a much better picture of the risk and the uncertainty surrounding it. Such an approach is also more fair, in that it neither overstates nor understates the risk in favour of those promoting a certain proposal or those opposed to it.

To assess the risk of introducing *Aeromonas salmonicida* (the agent of furunculosis) into New Zealand's salmon fisheries through imports of headless, eviscerated wild, ocean-caught Pacific salmon, the essential steps in the pathway of possible disease

---

[55] Kellar, JA. The application of risk analysis to international trade in animals and animal products. Revue Scientific et Technique de l'Office International des Epizooties 12, 1023-1044, 1993.

[56] MacDiarmid, SC. Risk analysis and the importation of animals and animal products. Revue Scientifique et Technique de l'Office International des Epizooties 12, 1093-1107, 1993.

[57] Thompson, KM, Burmaster, DE, Crouch, EAC. Monte Carlo techniques for quantitative uncertainty analysis in public health risk assessments.

Risk Analysis 12, 53-63, 1992.

introduction must be identified, broken down into their components, and estimates of risks applied to each component. For each input variable examined minimum, most likely and maximum estimates (that is, triangular distributions) were obtained by examination of published material and consultation with various people having specialist knowledge. However, it should be noted that the use of such triangular distributions may still lead to the risk being overstated.[58]

The risk estimates are derived from a simulation model which makes calculations iteratively using the PC software program @RISK. At each iteration @RISK selects at random a value out of each triangular distribution. These are then multiplied together as outlined in the model below and the result of each iteration is stored. Final risk estimates are then displayed as probability distributions.

The quantitative risk assessment was carried out on **four** commodities processed from wild, ocean-caught Pacific salmon. These are;

- Chilled, headless, eviscerated fish.
- Frozen, headless, eviscerated fish.
- Chilled skinless fillets.
- Frozen skinless fillets.

4.4 The major areas of perceived risk

Concerns that imports of wild, ocean-caught Pacific salmon from Canada could introduce *A. salmonicida*, the agent of furunculosis, tend to be based on five premises.

These are;

- **Premise 1:** The belief that a large percentage of Pacific salmon carry *A. salmonicida* with them at sea, that is are carriers of infection, and that these fish carry large numbers of the pathogen;
- **Premise 2:** The belief that Canadian fish processing plants employ procedures that are so unsanitary that;
    - all headless, eviscerated Pacific salmon or fillets thereof emerging from them will be contaminated with *A. salmonicida* and that;

---

[58] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with SC MacDiarmid, 7 January 1993.

- the numbers of *A. salmonicida* cells associated with the headless, eviscerated salmon or their fillets will be sufficient to establish infections in fish that contact the cells;

- **Premise 3:** The contention that contact with as few as ten *A. salmonicida* cells (presumably by ingestion of infected/contaminated Pacific salmon product or by exposure to the water-borne pathogen released from the product) will inevitably establish infections in aquatic animals contacted;

- **Premise 4:** The contention that significant portions of the product will end up being discarded (because of unsightly blemishes) and/or being fed to New Zealand fish and that *A. salmonicida* cells carried by the product will end up infecting New Zealand's fish and

- **Premise 5:** The assertion that the pathogen is so catholic in the range of potential hosts that, once introduced, it has a good chance of becoming established in a New Zealand waterway, whether or not salmonids are present to serve as hosts.

Each of these premises has either been dealt with above in Section 3.8 or will be addressed below in the body of the quantitative risk assessment.

5. Chilled, eviscerated, headless fish

5.1 The probability of importing contaminated, potentially infective fish

The first probability to be assessed is the probability that contaminated, potentially infective fish flesh will arrive in the country. This probability depends on the proportion of the year's catch which is infected and the number of fish represented in each tonne of imported commodity. It is also affected by the extent to which the risk is reduced by the various processing steps (inspection and grading, eviscerating and/or filleting, washing, freezing).

The range of input variables the simulation model uses to assess the probability of importing contaminated, potentially infective fish when the commodity is chilled, headless, eviscerated fish is as follows;

Table 5.1: Input variables used to calculate probability of importing contaminated fish flesh. Chilled, headless, eviscerated salmon.

	Minimum	Most Likely	Maximum
Proportion of fish diseased/year? (P)	0.0*	0.02	0.06
Evisceration reduces risk by;	0.8*	0.95	0.99
to; (R2)	1 - 0.8	1 - 0.95	1 - 0.99
Inspection & grading reduces risk by;	0.1*	0.5	0.7
to; (R3)	1 - 0.1	1 - 0.5	1 - 0.7
Washing reduces risk by;	0	0.05	0.3
to; (R4)	1 - 0	1 - 0.05	1 - 0.3
Mean weight of ocean caught salmon? (K)	1.5	2.5	5.0
Proportion remaining after evisceration (F)	0.65	0.71	0.73
No. fish represented/tonne (N) = 1000/FxK			
No. *diseased* fish/tonne = PxN			
DISEASED FISH *IMPORTED*/TONNE (D) = PxNxR2xR3xR4			

* Note: In the simulation model prevalence (P), effectiveness of inspection and grading (R3) and effectiveness of evisceration (R2) are inter-related. As prevalence (P) increases, the effectiveness of inspection and grading (R3) **tends** to increase while the effectiveness of evisceration (R2) **tends** to decrease. See below (Section 5.1.1.4) for discussion.

5.1.1 The evidence

5.1.1.1 Proportion of diseased fish per year (P)

The proportion of wild, ocean-caught Pacific salmon which are infected with *A. salmonicida* is a reflection of the prevalence of the disease in the wild salmon population. One consultant[59] has suggested that, at times, the prevalence of furunculosis in some batches of wild, ocean-caught Pacific salmon could be as high as 30-80%. However, these figures are unrealistically high. In 1990, no furunculosis was diagnosed in chinook salmon.[60] A document from the Canadian Department of Fisheries and Oceans reported furunculosis lesions in 0.047% of 19,000 spawning fish examined. [In the earlier version of this assessment this figure was mistakenly cited as 0.4%. The error was pointed out by Dr T Evelyn, Pacific Biological Station, Nanaimo]. However, the most telling evidence for the prevalence of *A. salmonicida* in wild, ocean-caught Pacific salmon is the data presented in Section 3.8 above.

There are no reports of *A. salmonicida* infections occurring in harvest-size Pacific salmon caught at sea. The evidence is that, as a worst case, based on the prevalence of infection in salmon **once they have entered fresh water on their return to their spawning grounds**, and thus after they have undergone a series of stresses which result in a recrudescence of any latent infections and a spread of infections amongst crowded, stressed fish, the prevalence of *A. salmonicida* does not exceed 6.0%. More importantly, a recent survey of wild, ocean-caught chum and sockeye salmon **before** they returned to fresh water failed to detect **any** carriers of *A. salmonicida*, establishing 99% confidence limits of between 0 and about 2.0%.

This information demonstrates the indefensibility of part of Premise 1, outlined above (Section 4.4), that a large percentage of Pacific salmon carry *A. salmonicida* with them at sea; that is, are carriers of infection.

---

[59] Anderson, C D, Veterinary Investigation Officer, MAF Quality Management, personal communication with Sue Cotton and Stuart MacDiarmid, 1992-93.

[60] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with Stuart MacDiarmid, 1993.

5.1.1.2 The probability of accepting a diseased fish for processing

Although salmon infected with *A. salmonicida* may develop large abscesses which burst through the skin, it is likely that affected fish will not display signs sufficient to cause their rejection prior to processing. This is because, as has been demonstrated in Section 3.8 above, clinical furunculosis is not seen in wild, ocean-caught Pacific salmon. If, however, a salmon clinically affected with furunculosis were caught, it could escape notice until it reached the processor.

5.1.1.3 The extent to which evisceration reduces the risk (R2)

The site of choice for isolating *A. salmonicida* from **carrier** salmon is the kidney.[61] Organisms may also be present in the spleen and, in diseased fish, in furuncles in muscle. In fish inhabiting bodies of fresh water with heavily-infected salmonid populations, *A. salmonicida* may also be readily cultured from mucus. This does not, of course, apply to wild, ocean-caught Pacific salmon in which furunculosis has not been seen and in which the prevalence of even subclinical infections is rare.

Evisceration significantly reduces the risk. It removes from the fish all visceral organs and tissues, gill, kidney tissue, heart and attached mesentery and vessels. This process substantially reduces the numbers of virtually all pathogens even though, in practice, small traces of dorsal kidney tissue, in the order of a gram or so, are often left in eviscerated fish.

A. salmonicida, if it occurs at all in wild, ocean-caught Pacific salmon, must do so only at low prevalence. In addition, one would expect the majority of any *A. salmonicida* cells in carrier fish to occur in visceral tissues, including the gills, rather than in the flesh. As discussed earlier (see Section 3.8), in fish actually dying from dead furunculosis, the visceral tissues (kidney) carry 10^3 to 10^4 times as many *A. salmonicida* cells/g as the flesh. It is almost certain that this disparate tissue distribution also holds true for the bacterium in clinically normal carrier fish. The vast majority of any *A. salmonicida* cells carried in grossly normal salmon would be removed from the product along with the visceral tissues during processing prior to export.

While evisceration could result in cross contamination, by bringing bacteria sequestered in viscera into contact with knives, work surfaces and flesh, its overall effect is overwhelmingly one of reduction in risk. Evelyn, who has worked extensively with *A. salmonicida* in salmonids considers the reduction to be near to 99%.[62] This figure is derived from data in Table 3.3, with the prevalence of carriers (6.0%) being

---

[61] Dr TP Evelyn, Pacific Biological Station, Nanaimo, British Columbia, personal communication to SC MacDiarmid during a visit to Nanaimo, November 1993.

[62] Dr TP Evelyn, Pacific Biological Station, Nanaimo, British Columbia, personal communication with SC MacDiarmid during a visit to Nanaimo, November 1993.

divided into the percentage of fish with lesions (0.08%) being an estimate of what the risk is reduced to.

5.1.1.4 The extent to which inspection and grading reduces the risk (R3)

While some have expressed the opinion that inspection and grading would reduce the risk by no more than 10%[63], others believe that inspection and grading markedly reduces the risk. In grading, commercial imperatives exclude diseased fish from the top grades. For example, at British Columbia Packers, Vancouver, fish are placed into three grades. The first and second grades are sold fresh or frozen, the third is for canning. **Any blemish leads to fish being downgraded.** On evisceration the inside of the fish is inspected for lesions, haemorrhages, discolouration etc. Even softness is considered a reason for downgrading. Septicaemic fish, or those affected with furuncles, would either be rejected or downgraded to canning grade (after extensive trimming). Adequate assurances about the health status of wild, ocean-caught salmon would be provided by permitting only first grade fish to be imported.

Depending on whether furunculosis occurs as the chronic or acute form, a wide spectrum of gross pathological signs may be seen. Changes range from a darkening of the skin through haemorrhages and reddening of the skin and fins to the development of furuncles (vesicles containing blood-tinged fluid) or large, bloody ulcers. Internally, affected fish exhibit diffuse reddening and petechiae are common on all serosal surfaces (such as are inspected after the fish has been eviscerated).[64]

Necrotic muscle lesions or furuncles are easily visible in most diseased individuals in chronic infections.[65] Inspection and grading of headless, eviscerated salmon will detect any cases of active furunculosis. Inspection and grading will reduce the risk of permitting the export of a case of furunculosis by at least 90%. However, it must be remembered that no case of clinical furunculosis has been found in wild, ocean-caught Pacific salmon. Subclinical carrier fish, undetectable by inspection, have been found in **salmon once they have returned to fresh water to spawn** and while inspection and grading will not reduce the risk of selecting a subclinically infected fish, subclinically infected fish will not be carrying a detectable *A. salmonicida* burden in the flesh; the pathogens will be removed during evisceration. If signs of furunculosis are not obvious

---

[63] Anderson, C D, Veterinary Investigation Officer, MAF Quality Management, personal communication with Sue Cotton and Stuart MacDiarmid, 1992-93.

[64] Kent, M L. Diseases of Seawater Netpen-Reared Salmonid Fishes in the Pacific Northwest. 76 pages. Canadian Special Publication of Fisheries and Aquatic Sciences 116, Department of Fisheries and Oceans, Nanaimo. 1992.

[65] Anderson, CD. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19 (1), 12-13, 1992.

in infected fish, the probability of their flesh carrying *A. salmonicida* is very low. If signs of septicaemia or furunculosis are obvious, then the fish will be rejected.

Because an increase in prevalence of infection leads to an increase in likelihood of clinical disease, the simulation model links prevalence with the probability of rejecting an infected fish at the time of harvest and at inspection and grading. That is, the higher the prevalence of *A. salmonicida* infection, the more likely there will be clinical signs. These, in turn, will mean that more infected fish will be rejected during inspection and grading. In the model, this linkage is achieved by making "inspection and grading reduces risk" dependent on "proportion of fish diseased"[66] by a coefficient of 0.4. That is, as "proportion of fish diseased" increases, "inspection and grading reduces risk" tends to increase also, and this dependency is given a correlation of 0.4. Similarly, because on increase in clinical furunculosis could reduce the effectiveness of evisceration as a risk reducing measure, "evisceration reduces risk" is made dependent on "proportion of fish diseased" by a coefficient of -0.4.

5.1.1.5 The extent to which washing reduces the risk (R4)

Washing will not reduce the risk from lesions in the flesh but will significantly reduce surface contamination. However, as pointed out earlier an argument raised against importation of wild, ocean-caught Pacific salmon is that stated in what I have called **Premise 2** (Section 4.4 above), namely that Canadian fish processing plants employ procedures that are so unsanitary that;

- all headless, eviscerated Pacific salmon or fillets thereof emerging from them will be contaminated with *A. salmonicida* and that;
- the numbers of *A. salmonicida* cells associated with the headless, eviscerated salmon or their fillets will be sufficient to establish infections in fish that contact the cells.

For this premise to have any validity, ocean-caught Pacific salmon would have to have a high prevalence of *A. salmonicida* carriers and the carriers would have to be carrying large numbers of the pathogen. Also, conditions in Canadian fish processing plants would have to be substandard, with the processing procedures, including the

---

[66] @RISK Risk Analysis and Simulation Add-In for Lotus 1-2-3. Version 2.01 Users Guide. Palisade Corporation, Newfield, New Jersey, 1992.

David Vose, Risk Analysis Services, Wincanton, Somerset, advised on linkage and generated the scatter plots on which the choice of the degree of correlation was made. The choice of the degree of correlation was, however, made by SC MacDiarmid.

frequency of washing, being totally inadequate. A digression into processing and inspection procedures is, therefore, appropriate at this point.[67]

During visits to Canada and in written communications, officials of Canada's Department of Fisheries and Oceans have described how the Canadian fish inspection system is well respected, having evolved over nearly 100 years into a sophisticated, multi-faceted program that focuses on the critical steps in harvesting, transporting and processing fish products to ensure the production of safe fish products of acceptable quality. The Canadian Department of Fisheries and Oceans was the first government food inspection agency in the world to introduce the requirements of Hazard Analysis Critical Control Point (HACCP) principles as a condition of processing fish. The system in place in Canada is referred as the Quality Management Program and, according to officials of the Department of Fisheries and Oceans, has received international recognition as an effective and efficient approach to ensuring the production of safe fish products of high quality. Other countries, including the United States, have used the Canadian Quality Management Program as a model in the development of their own HACCP-based programs.

Pacific salmon produced for export from British Columbia are subject to the requirements of the multi-faceted inspection program which includes the Quality Management Program.

All vessels licensed to harvest and transport salmon must meet regulated construction and operating standards to ensure that the fish are handled properly and not subject to temperature abuse or contamination. These requirements are enforced by inspectors of the Department of Fisheries and Oceans and the level of compliance of the vessels is high. Most fish holds of these vessels are constructed of either aluminium or fibreglass and are designed to facilitate the cleaning and sanitization of all fish contact surfaces. All fishing vessels are required to preserve the catch either in ice at no greater than 4 °C or in chilled seawater which must be capable of holding the fish at -1 °C.

Salmon processed in registered processing plants must meet the requirements of the Quality Management Program. All fish landings must be sampled and inspected prior to processing to ensure that the fish meet the minimum acceptable safety and quality standards. Any fish exhibiting lesions of any kind are rejected and the landing hand-culled prior to processing.

Prior to the processing of any whole or dressed fish, the fish must be adequately washed. In recent companies have been installing mechanical fish washers which are effective in removing slime and mucus from the surface belly cavity of dressed fish.

---

[67] Information on Department of Fisheries and Oceans inspection procedures is based on personal communication from Iola M Price, Director, Aquaculture and Habitat Science Branch, Biological Sciences Directorate, Department of Fisheries, Barry O'Neil, Chief Veterinary Officer, September 1994 and on information gathered by SC MacDiarmid during a visit to British Columbia in July 1994.

During the on-line processing of salmon, critical control points throughout the process are monitored to ensure that the fish will meet the final product standards for safety and quality. One such critical control point is the dressing of whole fish. Under the processing plant's Quality Management Program, fish are routinely sampled after the dressing process and inspected to ensure that all viscera have been removed and that the belly cavity has been thoroughly washed of all slime and blood. These inspections are recorded and the identification of any defects results in immediate corrective action being required.

Prior to the freezing of fish or the icing and boxing of fresh fish, the fish are individually inspected and graded. Again, any fish that exhibited lesions of any sort are culled from the lot. Fish with remnants of viscera or which have been poorly washed are culled from the line and reprocessed. These inspections are again recorded and when reject fish are identified, appropriate corrective action is initiated by the plant.

Under the Quality Management Program, the final product is subject to one more inspection. All lots of final product are randomly sampled and inspected to ensure that they meet the final product grade standards for safety and quality.

In addition to the controls implemented on product and process, the Quality Management Program also requires that the processing plant perform routine inspections of construction and sanitation conditions of the processing facility. These inspections are recorded and all deficiencies result in immediate corrective action. These inspections not only cover plant conditions but also monitor the hygienic practices of the employees to ensure that proper sanitation measures are followed. Examples of the requirements include that employees must wash and disinfect their hands and gloves after each absence from the processing area and all outer garments and protective clothing must be cleaned and disinfected after each break. The sanitation checks are performed by the plant on a daily basis.

The Inspection Branch of the Department of Fisheries and Oceans is responsible for verifying that the fish processing plants are effectively implementing their in-plant Quality Management Program, operating in compliance with the Fish Inspection Regulations and producing a commodity which is safe and of acceptable quality. Department of Fisheries and Oceans staff carry out regular inspections of the processing plants to ensure this. Such inspections are a verification that the processor's in-plant Quality Management Program meets the requirements of the Fish Inspection Regulations, that the records of inspection are up to date and accurate, that the monitoring and inspections procedures are being carried out correctly and at the specified frequency, and that the plant conditions and final product meet the requirements of the Regulations. The inspections are comprehensive and involve the physical inspection of the plant and sensory evaluation of the salmon. The frequency of inspections is based on the history of compliance of the plant and can vary from once every 2 weeks to once every 2 months.

Officials from Canada's Department of Fisheries and Oceans pointed out that in 1993, based on an evaluation of Canada's fish inspection system, the European Union

exempted Canada from the EU inspection regime and Canadian fish now receive facilitated access to the EU market. Canada was the first country to receive this exemption.

Concerns, then, that salmon with lesions of furunculosis could somehow be pass inspection and grading procedures and be classed as first grade for export to New Zealand appear to be without foundation. Similarly, claims that processing would result in mass contamination of commodity are also baseless. Even if it were true that Pacific salmon were likely to contaminate various surfaces with *A. salmonicida*, multiplication of the pathogen on such surfaces is less likely than its disappearance. All publications on the topic support the contention that the pathogen does very poorly when it has to compete with "environmental" bacteria (see Section 3.8).

Further, as already discussed, *A. salmonicida*, if it occurs at all in Pacific salmon at sea, must do so only at a very low prevalence (see Section 3.8). What is more, the majority of any *A. salmonicida* cells in carrier fish occur in visceral tissues, including the gills, rather than in the flesh. For example, unpublished data obtained at the Pacific Biological Station, Nanaimo, British Columbia,[68] for fish actually dying from furunculosis, indicate that the flesh carries 10^3 to 10^4 times fewer *A. salmonicida* cells/g than visceral tissues (e.g. kidney). This disparate tissue distribution almost certainly holds true for the bacterium in carrier fish. The vast majority of any such *A. salmonicida* cells in subclinically infected carrier salmon would be removed from the product during the process of evisceration.

The numbers of viable *A. salmonicida* cells reported as present in samples of kidney from carrier chum and pink salmon averaged $10^{3.7}$ (range from $10^{1.7}$ to $10^{5.4}$ cfu/g).[69] In flesh, one would reasonably expect the numbers of bacterial cells in such carrier fish to be considerably lower (based on the results mentioned above, about $10^{3.5}$ times lower, which is why fish pathologists studying the disease never consider flesh samples as suitable for determining the prevalence of infections with bacteria such as *A. salmonicida*).

---

[68] Dr TP Evelyn, Pacific Biological Station, Nanaimo, British Columbia, personal communication to SC MacDiarmid, 1994.

[69] Nomura T, Yoshimizu M, Kimura T. Prevalence of Aeromonas salmonicida in the chum salmon (Oncorhynchus keta), pink salmon (*O. gorbuscha*), and masu salmon (*O. masou*). Gyobyo Kenku 26, 139-147, 1991. [In Japanese, with English summary.]

The figure of 10^{10} cfu/g cited by Anderson[70] is based on counts made by Rose and colleagues[71] on flesh lesion material from highly *A. salmonicida*-susceptible Atlantic salmon which had died of experimentally induced furunculosis; the source fish were therefore not carrier fish! By way of contrast, data obtained by workers at the Pacific Biological Station, Nanaimo[72] from Pacific salmon (chinook salmon) dead of experimentally induced furunculosis, demonstrate that the numbers of *A. salmonicida* cells in the flesh were considerably lower than the 10^{10} cfu/g reported for Atlantic salmon by Rose and coworkers. The numbers of *A. salmonicida* cells in the tissue of Pacific salmon dying from furunculosis ranged from 10^2 to 10^6 cfu/g and averaged only 10^5 cfu/g (see Tables 3.4 and 3.5). These figures for *A. salmonicida* in the flesh of Pacific salmon dying from furunculosis mean that Anderson's calculations are based on an unrealistically high number of *A. salmonicida* cells. They also mean that even if product from a Pacific salmon with active furunculosis were to be shipped by some error to New Zealand, the risk posed by such a product would still be considerably less than that feared Anderson. Such an error would, of course, be highly unlikely because, at sea, an actively diseased salmon would be quickly eliminated by predation and thus would not be available to the commercial fishery (see prevalence figures in Section 3.8). It is difficult to visualize a situation in which the product could provide the continuity of exposure required to establish infections with the doses of the pathogen as low as could reasonably be expected to be present, even should contamination of commodity occur during processing.

To sum up, the contention that the number of *A. salmonicida* cells likely to be present in carrier Pacific salmon are so high as to result in widespread contamination of processing plants and salmon passing through them is unsustainable.

In fact, the washing process to which salmon are subjected at a number of stages during processing[73] will reduce any contamination of flesh which may occur during the removal of viscera from subclinically infected carrier fish. A maximum reduction of 30% is used here.

---

[70] Anderson, CD. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19 (1), 12-13, 1992.

[71] Rose AS, Ellis AE, Munro ALS. The infectivity by different routes of exposure and shedding rates of *Aeromonas salmonicida* subsp. *salmonicida* in Atlantic salmon, *Salmo salar* L., held in sea water. Journal of Fish Diseases 12, 573-578, 1989.

[72] Dr TP Evelyn, Pacific Biological Station, Nanaimo, British Columbia, personal communication with SC MacDiarmid during visit to Nanaimo, November 1993.

[73] Visit by SC MacDiarmid to British Columbia Packers, Vancouver, July 1994.

5.1.1.6 Mean weight of ocean caught salmon (K)

A number of New Zealand sources were consulted[74] and a range of weights was selected for use in the first edition of this risk assessment.[75] However, at a meeting at the Pacific Biological Station, Nanaimo, in November 1993 Dr L Margolis pointed out that the mean weights for New Zealand ocean-caught salmon used in the first assessment were too high for Canadian Pacific salmon. Ocean caught salmon on the British Columbia coast are about half what was used in the first edition. Average weights of Pacific salmon are shown in Table 5.2. The data in Table 5.2 also provide background recommended by Boustead[76] as being useful to readers of an analysis such as this.

---

[74] Anderson, C D, Veterinary Investigation Officer, MAF Quality Management, personal communication with Sue Cotton and Stuart MacDiarmid, 1992-93.

Unwin, M, National Institute of Water and Atmospheric Research Ltd, personal communication with Sue Cotton, 1993.

McNeil, A, General Manager, Angus McNeil & Company, Nelson, personal communication with Sue Cotton, 1993.

[75] MacDiarmid, S C, Cotton, S. The Risk of Introducing Aeromonas salmonicida into New Zealand Salmon Fisheries Through the Vehicle of Frozen Fillets of Ocean-Caught Canadian Salmon. 46 pages. NASS Publication 93-1. Ministry of Agriculture and Fisheries, Wellington, 1993.

[76] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with SC MacDiarmid, 7 January 1993.

Table 5.2: Data on the commercial salmon catch in British Columbia, Canada, for 1990.[77]

Species	*Number*	*% of catch*	*Kg (round wt.)*	*Average wt.*
All	39,128,000	-	96,350,000	2.46
Sockeye	14,188,000	36.3	37,133,000	2.62
Pink	17,224,000	44.0	26,240,000	1.52
Chum	3,176,000	8.1	17,180,000	5.41
Coho	3,872,000	9.9	10,569,000	2.73
Chinook	668,000	1.7	5,228,000	7.83

5.1.1.7 Percent remaining after evisceration and removal of the head (F)

The percentage of a Pacific salmon remaining after the head and internal organs have been removed is based on data in Table 5.3.

[77] Data taken from INPFC Statistical Yearbook 1990, Vancouver, Canada, and provided to Australian officials at the Canada-Australia Salmon Technical Meeting held in Nanaimo, BC, Canada on July 25-26, 1994, in relation to GATT Article XXII:1.

Table 5.3: Percentage of body weight of body parts of Pacific salmon.[78]

Organ or body part	Percent of body weight
Blood	4.1
Liver	1.16
Kidney	0.8
Gills	3.9
Skin	4.66
Bone	3.84
Gut (abdominal contents except liver and kidney)	8.52
Muscles (flesh)	46.55*
Head	10.3
Tail, fins, everything not mentioned above	16.17
	100.00

* This value appears to relate to muscle recoverable as flesh. The actual percentage of muscle in a Pacific salmon (*Oncorhynchus* species) is between 57 and 66% of body weight.[79]

From Table 5.3 it can be seen that the weight of a headless, eviscerated Pacific salmon is approximately 71.22.% of its live weight.

---

[78] Price, Iola M, Director, Aquaculture and Habitat Science Branch, Biological Sciences Directorate, Department of Fisheries and Oceans, Ottawa, citing a personal communication from Dr Francis Law, Simon Fraser University, Vancouver. Personal communication with SC MacDiarmid, 2 September 1994. Modified slightly on the basis of information provided by PS Davie and H Thorarensen, Massey University, personal communication with SC MacDiarmid, 27 September 1994.

[79] Davie, PS, Thorarensen, H, Department of Physiology and Anatomy, Massey University, Palmerston North, personal communication with SC MacDiarmid, 27 September 1994.

5.1.2 Simulation results

5.1.2.1 The number of fish represented/tonne of headless, eviscerated salmon

The first graph (Figure 5.1) shows the most likely number of fish per tonne and the probable distribution of values around this expected number. The estimates are based on 5,000 iterations of the @RISK program.

It can be seen that, under the assumptions used in this model, the number of fish per tonne is;

Expected/mean result	=	508.9
Maximum result	=	962.7
Minimum result	=	282.8

Probability;	That the number of fish is less than;
90%	687.3
95%	753.8
99%	849.3

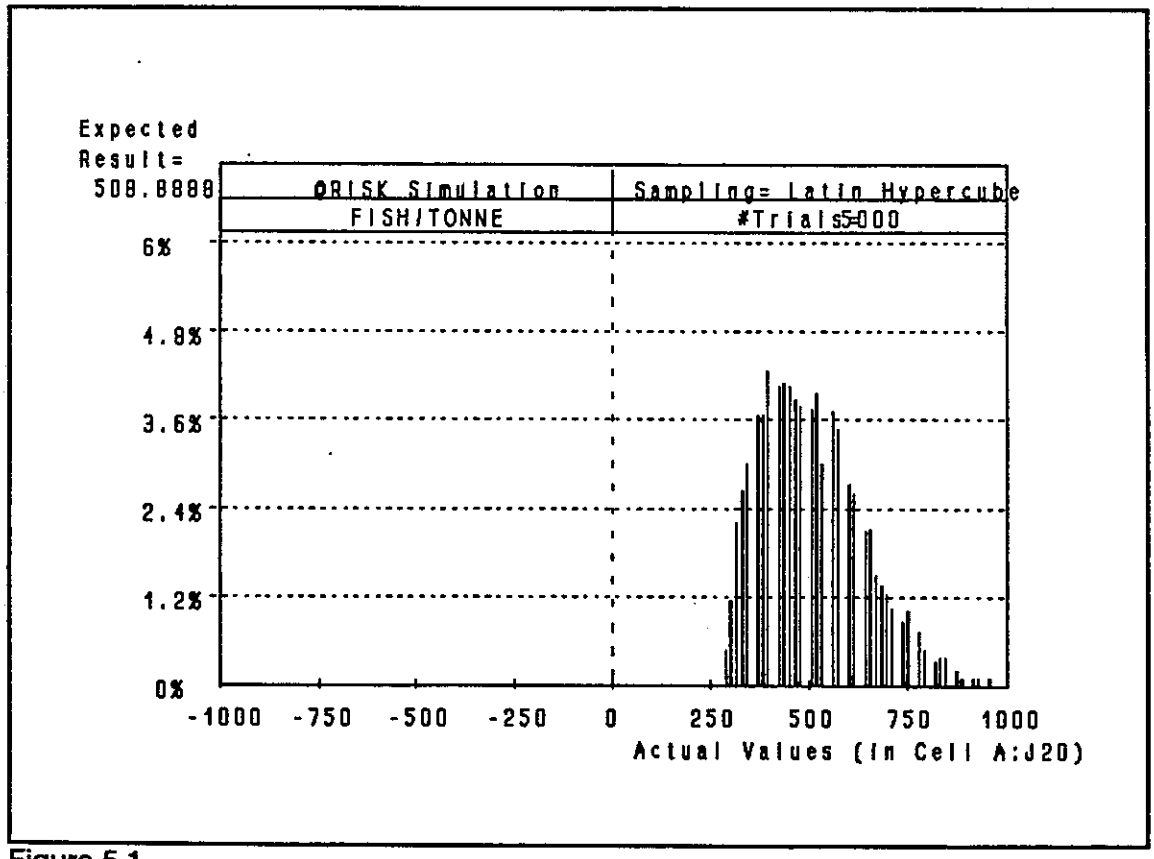


Figure 5.1

That is, there is a 95% probability that a tonne of headless, eviscerated Pacific salmon represents fewer than 754 fish. On average though, one can expect a tonne to represent about 509 fish.

5.1.2.2 The number of *diseased* fish represented/tonne

Figure 5.2 shows the probable number of *diseased* fish represented in each tonne of headless, eviscerated Pacific salmon. With the assumptions used in this model, the number of diseased fish included in a tonne is;

Expected/mean result	=	13.5
Maximum result	=	52.0
Minimum result	=	0.004

Probability;	That the number of fish is less than;
90%	23.6
95%	27.3
99%	34.2

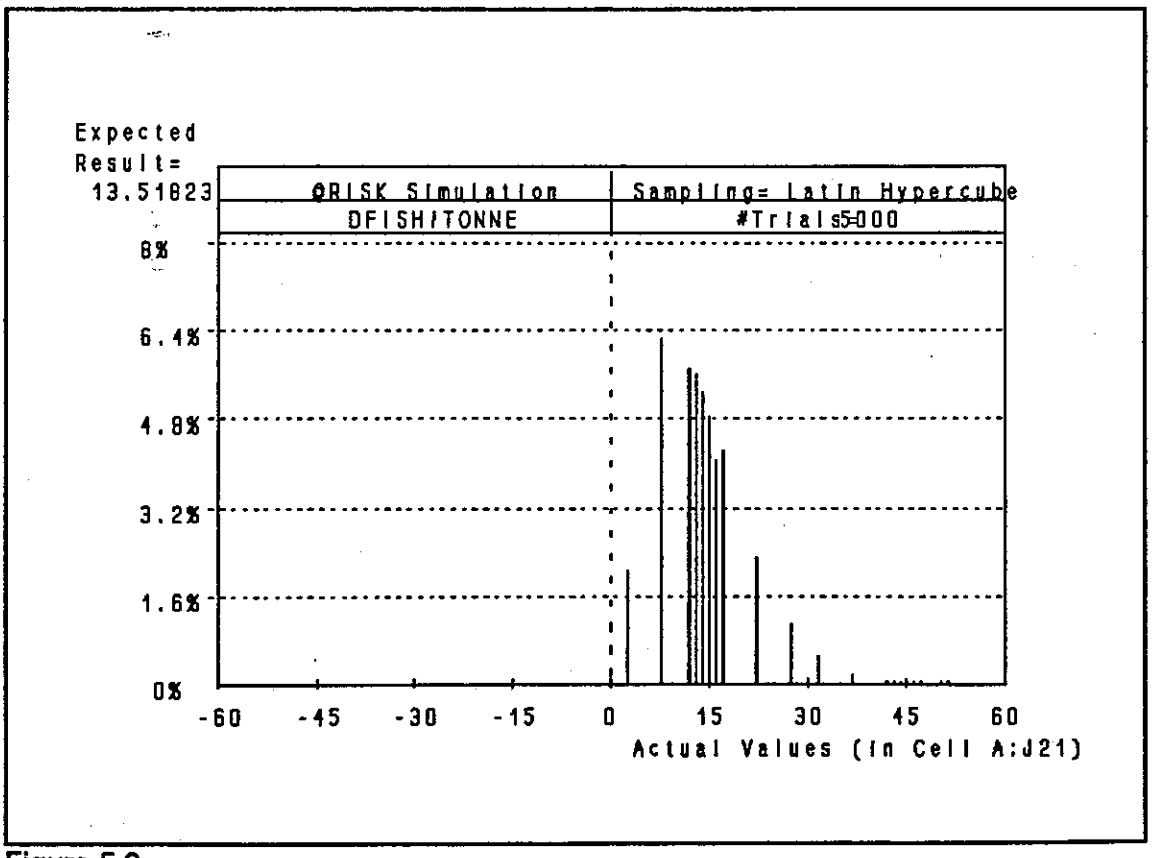


Figure 5.2

That is, there is a 95% probability that a tonne of headless, eviscerated Pacific salmon will include fewer than 28 infected fish. On average though, one can expect that a tonne will contain about 14 infected fish.

5.1.2.3 The probability of *importing* contaminated fish, without freezing

Figure 5.3 shows the probable number of potentially infective, contaminated fish represented in each tonne imported. The next graph (Figure 5.4) shows the *cumulative* probability of a given number of contaminated fish contributing to each tonne imported. It can be seen that the number of contaminated fish contributing to each tonne imported is;

Expected/mean result	=	0.6
Maximum result	=	4.5
Minimum result	=	8×10^{-5}

Probability;	That the number of fish is less than;
90%	1.3
95%	1.6
99%	2.4

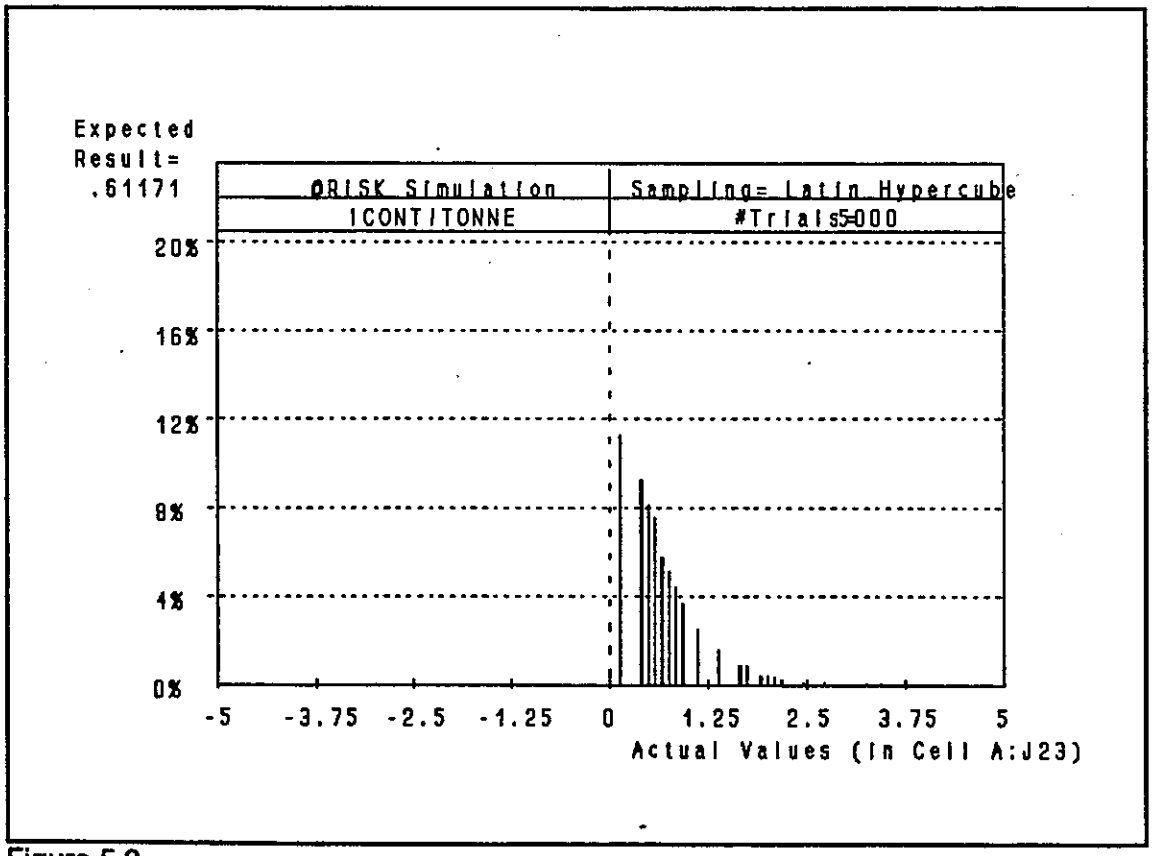


Figure 5.3

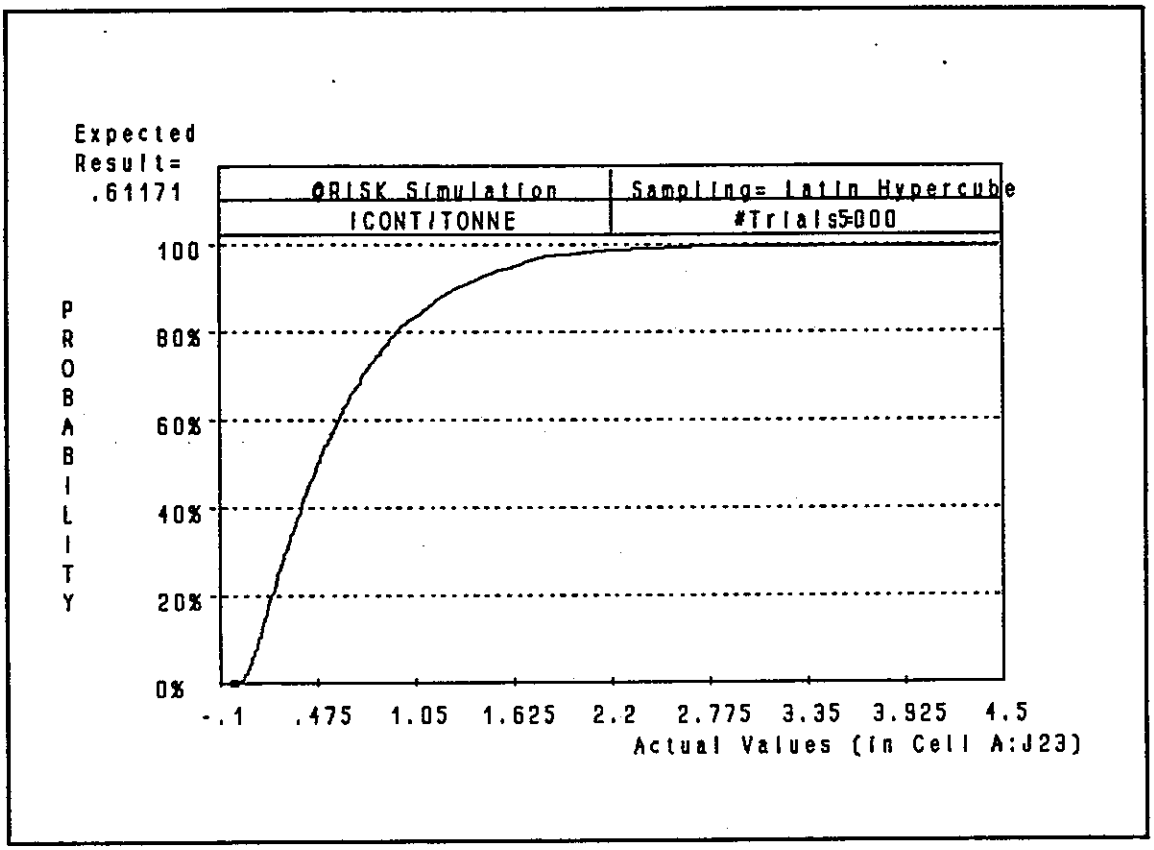


Figure 5.4

That is, there is a 95% probability that the number of contaminated, potentially infective fish contributing to each tonne imported is fewer than 2 per tonne. This figure takes into account the extent to which the risk has been reduced by evisceration, inspection, grading and washing of headless, eviscerated Pacific salmon. With the assumptions used in the model, it can be seen that each tonne imported into New Zealand is likely to contain flesh contributed by <1 fish which is, theoretically, capable of introducing disease. However, there are many steps between importation of contaminated flesh and the introduction of disease, as the following sections illustrate.

5.2 Distribution of imported fish within the country

Quite clearly the distribution of imported ocean-caught Pacific salmon within New Zealand will be strongly correlated with the distribution of the human population. For the purposes of this model, New Zealand has been divided into three regions (see Figure 5.5). The highest human population is in what I have called Region 1 ('North' in Figure 5.5), roughly the north of the North Island as far as a line joining Kawhia Harbour with the Bay of Plenty. The rest of the North Island comprises Region 2 ('Central' in Figure 5.5) and contains most of the North Island's salmonid fisheries. Region 3 ('South' in Figure 5.5) is the South Island.

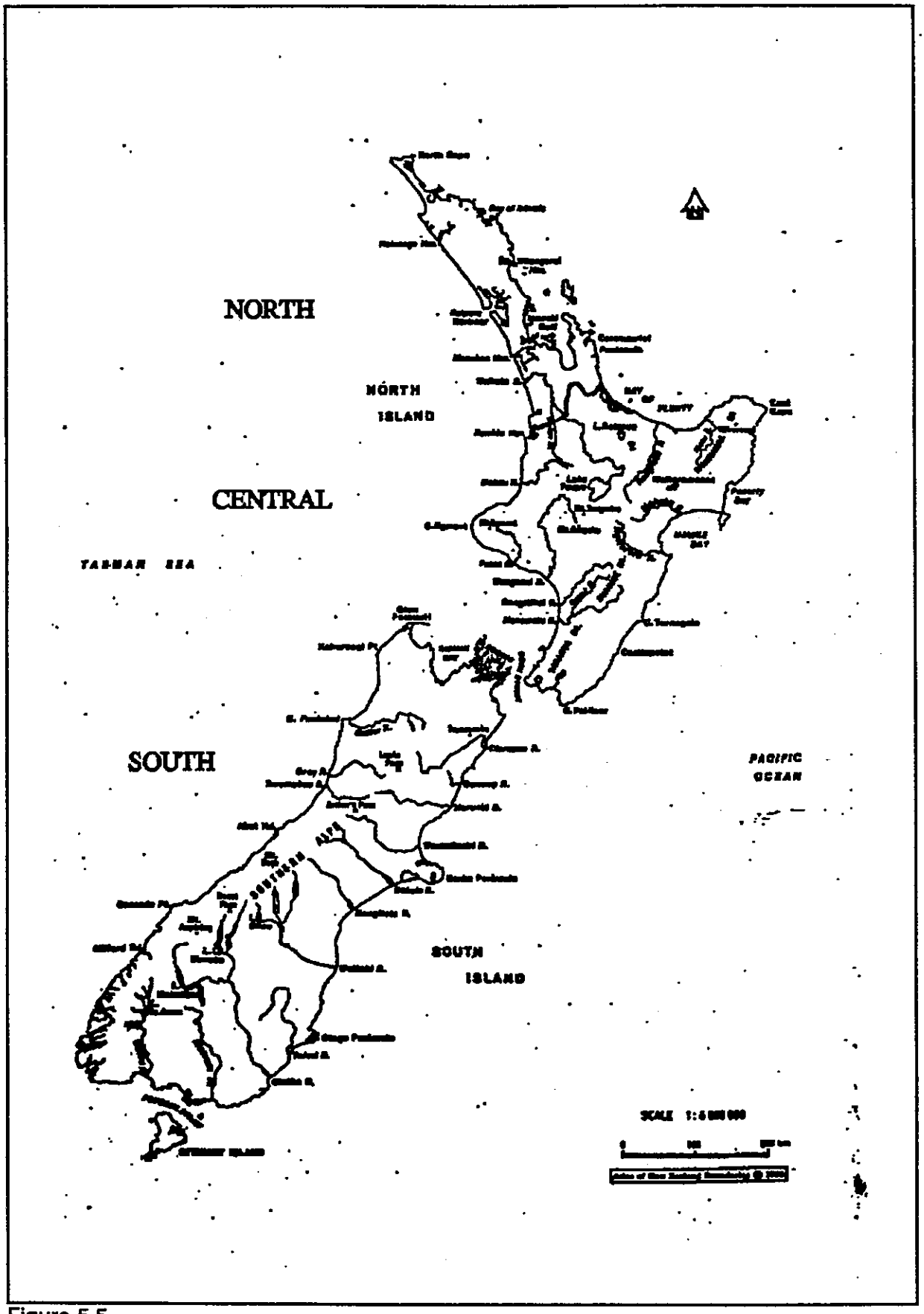

NORTH
NORTH
ISLAND
CENTRAL
TASMAN SEA
SOUTH
PACIFIC
OCEAN
SOUTH
ISLAND

Figure 5.5

The distribution of the human population between the three regions is shown in Table 5.3.

Table 5.3: Distribution of human population within New Zealand

Human pop. Region 1 (Hp)r1	1,367,300	39.8%
Region 2 (Hp)r2	1,184,600	34.5%
Region 3 (Hp)r3	880,700	25.7%
Total population	**3,432,600**	**100.0%**

The volume of fish imported into each region is assumed to be proportional to the human population plus or minus 10%. Obviously, when the model randomly selects a value of up to plus 10% for one region it must make a compensatory reduction in another region.

Table 5.4: Input variables for proportion of salmon imported into each region of New Zealand.

	Minimum	Most Likely	Maximum
Proportion imported into Region 1 (I)r1	29.8%	39.8%	49.8%
into Region 2 (I)r2	24.5%	34.5%	44.5%
into Region 3 (I)r3	15.7%	25.7%	35.7%

5.2.1 The evidence

Figures for the human population of New Zealand are readily available and are not contentious.[80] The proportion of imported product consumed in each region is clearly related to the human population. It is probable, however, that proportionally more of the expensive imported commodity will be consumed in the main urban centres such as Auckland. While this will reduce any risks, the extent to which a disproportionate volume of the product is consumed in the major centres cannot be estimated.

[80] Department of Statistics. *New Zealand Official Yearbook.* 95th edition. Wellington. 1992.

5.3. The distribution of salmonid fish stocks within the country

The salmonid fisheries (wild trout stocks, wild salmon and farmed salmon stocks) are not distributed evenly throughout the country. In fact, there are virtually no salmonid fisheries in Region 1, the region with the greatest human population and hence the region where the greatest amount of imported salmon is likely to be consumed.

The distribution of salmonid stocks within New Zealand is shown in Table 5.5.

Table 5.5: Input variables used for the distribution of salmonid stocks within New Zealand.

	Minimum	Most Likely	Maximum
Proportion of salmonid fishery in Region 1 (Sp)r1	0.25%	0.25%	0.25%
in Region 2 (Sp)r2	18.0%	23.0%	28.0%
in Region 3 (Sp)r3	72.0%	77.0%	82.0%

5.3.1 The evidence

The *wild salmon* population of New Zealand is relatively small and is virtually all located in the South Island. The total population of catchable *trout* (> 40 cm) is approximately 2.5 million. This figure does not include the large number of early year classes of trout. The population estimate is calculated as follows;[81] There are 186,000 km of river in New Zealand and an estimated 170,000 km sustain trout. On average there are 15 fish per km. The distribution of wild stocks of trout is estimated to be;

Region 1; maximum 0.5%
Region 2; minimum 35%, most likely 45%, maximum 55%
Region 3; minimum 45%, most likely 55%, maximum 65%

The wild salmon population is not significant compared with the trout population. The figure for wild stocks of salmonids will, therefore, be approximately the same as those for wild stocks of trout.

[81] Jellyman, D, National Institute of Water and Atmospheric Research Ltd, personal communication with Sue Cotton, 1993.

The total *farmed salmon* population of New Zealand is approximately 10 million individual fish [82], but these are concentrated in a few localities. There are three large salmon farming operations in New Zealand (in Nelson, Blenheim and Stewart Island). The three major companies have approximately 2 million fish each[83] and several smaller companies probably own a similar total number.

All salmon farms are in the South Island, concentrated around Nelson, Blenheim and Stewart Island, with some smaller ones in Canterbury.[84]

With the inclusion of farmed stocks, the salmon and trout populations of New Zealand are approximately equal. However, all the salmon are located in the South Island. The distribution of the combined salmonid population approximates;

Region 1; maximum 0.25%
Region 2; minimum 18%, most likely 23%, maximum 28%
Region 2; minimum 72%, most likely 77%, maximum 82%

In the simulation, whatever values are selected in any one iteration must add up to 100%.

In this risk analysis the possibility of fish other than salmonids becoming infected with *A. salmonicida* was considered. There is no denying that *A. salmonicida* (the typical form, which causes furunculosis in salmonid fishes [Section 4.2]) is capable of infecting a wide range of other fish species. However, the most reasonable interpretation of the relevant literature is that the typical form of the bacterium persists poorly in non-salmonids and that its host range in non-salmonids is far narrower than is actually reported. The supposedly wide host range of typical *A. salmonicida* is the result of a general failure of authors to provide enough information on their isolates to permit one to determine which strain of the bacterium (typical or atypical) was present. Under these circumstances typical *A. salmonicida* was usually assumed to be the infecting agent. In fact, however, in cases where adequate testing was done, the atypical form of the organism accounted for most of the infections reported in non- salmonids. Reports of infections in non-salmonids with typical *A. salmonicida* have usually represented recent infections caused by association with salmon suffering furunculosis outbreaks or have involved non-salmonids held under

---

[82] This figure does include a large number of early year classes. If these are excluded the population is around 4.5 million, a similar order of magnitude to the wild trout population.

[83] Wilson, G, Southern Oceans Seafoods Ltd, Nelson, personal communication with Sue Cotton, 1993.

[84] Russell, D, New Zealand Fishing Industry Board, personal communication with Sue Cotton, 1993.

stressful conditions of confinement. So, as reported earlier in Section 3.8, non-salmonids become infected with typical *A. salmonicida* under unnaturally stressful conditions or when in close association with infected salmonids. Typical *A. salmonicida* is not naturally a cause of disease in non-salmonids.[85]

Further, as demonstrated in Section 3.8, atypical strains of *A. salmonicida* have never been isolated from wild, ocean-caught Pacific salmon examined in the Canadian salmon disease surveillance program.

With respect to other aquatic organisms becoming infected and acting as a reservoir for *A. salmonicida*, Austin and Austin[86] cite a study whereby a total of 2,954 vertebrate and invertebrate specimens collected from ponds during an epizootic of furunculosis were examined for the presence of *A. salmonicida* with negative results. This is clear evidence against the likelihood of organisms other than fish acting as reservoirs.

Nevertheless, following sections which examine the likelihood of infectious doses of *A. salmonicida* entering waterways following importation of Pacific salmon products, are also relevant in addressing the possible risks to non-salmonid fish species.

5.4 The probability that scraps will introduce infection

In examining the potential pathways by which infected salmon flesh could enter New Zealand waterways, one must decide on the extent to which one *lumps* or *splits* the possibilities. It has been claimed that scraps of flesh could find their way into waterways via kitchen waste water, picnickers throwing away uncooked scraps, fishermen using scraps as bait, scavenging animals and birds carrying scraps from dumps into waterways etc.

Of the possibilities listed, kitchen waste water is clearly a significant one and is dealt with separately. All others are lumped together in this section and the estimates of each variable and the calculation to assess the likelihood that scraps *per se* will introduce infection are stated in the following sections.

---

[85] Dr Trevor Evelyn (Canadian Department of Fisheries and Oceans), Dr John G Nickum (United States Fish and Wildlife Service) and Dr Eva-Maria Bernoth (CSIRO Fish Diseases Laboratory, Australia) at Canada-Australia Salmon Technical Meeting under auspices of GATT Article XXII. Nanaimo, British Columbia, July 25-26, 1994.

[86] Austin, B, Austin, DA. Bacterial Fish Pathogens. Disease in Farmed and Wild Fish. Second edition. Ellis Horwood, New York. 384 pages. 1993.

5.5 Region 1, scraps introduce infection

For each tonne imported, the number of contaminated, potentially infective fish entering Region 1 is the product of the number of contaminated fish imported into the country D and the proportion imported into the region (I)r1. That is; (D)r1 = Dx(I)r1

Table 5.6: Input variables used to calculate probability of scraps introducing infection into Region 1.[87]

	Minimum	Most Likely	Maximum
Proportion disposed of as uncooked scraps (P1)r1	0.1	0.17	0.34
Proportion not incinerated (P2)r1	0.95	0.98	1.0
Proportion not buried (P3)r1	10^{-4}	0.001	0.005
Contaminate fresh water? (R6)r1	0.001	0.01	0.05
Proportion of fishery in region 1 (Sp)r1			
Infect suscept. host when present? (R7)r1	10^{-5}	10^{-4}	0.01
Infect suscept. host in Region 1 (SpxR7)r1			
Infection introduced into fresh water (S1)r1 = (DxP1xP2xP3xR6xR7xSp)r1			
Contaminate estuarine water? (R8)r1	0.001	0.01	0.05
Infect suscept. host when present? (R9)r1	10^{-5}	10^{-4}	0.01
Infection introduced into estuarine water (S2)r1 = (DxP1xP2xP3xR8xR9xSp)r1			
Contaminate sea water? (R10)r1	0.001	0.01	0.05
Infect suscept. host when present? (R11)r1	10^{-5}	10^{-4}	0.005
Infection introduced into sea water (S2)r1 = (DxP1xP2xP3xR10xR11xSp)r1			
SCRAPS INTRODUCE INFECTION INTO REGION 1/TONNE (I1)r1 = (S1 + S2 + S3)r1			

[87] Each input variable is represented by an upper case letter and a number. For example "P1" is short-hand for "Proportion 1". Each input variable may also be qualified by a lower case letter "r" and a number. This is short-hand for "Region 1", "Region 2" or Region 3". (P1)r1, therefore, stands for "Proportion 1 for Region 1".

5.5.1 The evidence

5.5.1.1 Proportion disposed of as scraps (P1)

Salmon is a relatively expensive commodity, with imported salmon likely to be substantially more expensive than the local product.[88] For this reason, consumers are unlikely to be profligate in the amount they discard as uncooked scraps. While it is possible that on many occasions skin, fins, tail and/or bones may be discarded prior to cooking, it is likely that in about 50% of cases salmon will be cooked with the skin on and most of the bones still in.[89] The maximum amount discarded as scraps will, therefore, be unlikely to exceed 34.6% of what is imported (from Table 5.3, this figure is estimated as follows ((4.66 [skin]+3.84 [bone]+16.17 [tail and fins])/71.22 [headless, eviscerated fish]). Although approximately half of this, that is 17%, is likely to be discarded uncooked, some product could be discarded as spoiled[90] so the full value of 34% is used as the maximum value in the simulation model. There is no reason why this figure should vary between regions.

5.5.1.2 Proportion not incinerated (P2)

Some domestic rubbish is destroyed in home incinerators. While up to 16% of households may incinerate at least some of their rubbish[91], the amount of **kitchen** refuse destroyed by burning is likely to be much less. A MAF survey into the way households dispose of **fruit** wastes indicated that only about 2% of households use incineration as their primary means of disposing of such waste.[92] (A further 3% use incineration as their secondary means of disposal.) Most rubbish containing scraps

---

[88] Cavanagh, R, Director Seafood, Woolworths (NZ) Ltd, personal communication with SC MacDiarmid, 1994.

Hall, R, Managing Director, James Crisp Ltd, personal communication with SC MacDiarmid, 1994.

Warren, T, Southfresh Ltd, personal communication with SC MacDiarmid, 1994.

[89] Kyaw Tay, Head Chef, Terrace Regency Hotel, Wellington, personal communication with Martin Van Ginkel, September 1994.

[90] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with Stuart MacDiarmid, 1993.

[91] Dolan, L. *Where to Now? Christchurch Solid and Hazardous Waste Management Public Discussion Document.* Christchurch City Council. 30 pages, 1992?

[92] Viggers, Elizabeth. The Potential risk of Introducing Exotic Pests to New Zealand through Domestic Disposal of Waste Plant Material. Project BH 004. MAF Quality Management. 12 pages. June 1993.

will be disposed of by other methods. There is no reason why this figure should vary between regions.

5.5.1.3 Proportion not buried (P3)

Salmon scraps and wrappers from domestic or commercial kitchens will be disposed of with other kitchen waste. This kitchen waste will, in turn, be diluted with other non-kitchen solid waste. Residential waste will be further diluted by industrial and office waste. So, from the time that salmon scraps are generated they are going to be diluted with other solid waste.

Many households (34%) use burial or composting as their primary means of disposal of fruit wastes and it is probable that this applies to other kitchen wastes, such as that containing salmon scraps. However, most households dispose of their kitchen waste into the rubbish bin and such disposal is the primary means of disposal for 40% of households and the secondary means for a further 20%.[93]

About 16-18% of urban solid waste is collected residential garbage[94] of which approximately 26% to 37% is 'putrescibles' or kitchen waste. A further 30% of urban solid waste is residential rubbish, largely garden rubbish ('yard waste'), delivered to landfills by private vehicles. So, for every 1,000 kg of urban solid waste, around 300 kg (1,000 x 0.3) will be residential 'yard waste'. About 180 kg (1,000 x 0.18) will be collected residential rubbish, of which 67 kg (180 x 0.37) will be kitchen waste and 113 kg (180 - 67) will be non-kitchen waste. This means that each 1 kg of kitchen waste will be diluted by 6.16 kg ([300 + 113]/67) of non-kitchen residential waste.

For every 1,000 kg of urban waste, 67 kg will be kitchen waste and the balance will be domestic non-kitchen waste, office waste, industrial waste etc. This means that the dilution of kitchen waste is [(1,000-67)/67=] 13.92 per kg. That is, every 1 kg of kitchen waste is diluted, on average, into 13.92 kg of other rubbish.

---

[93] Viggers, Elizabeth. The Potential risk of Introducing Exotic Pests to New Zealand through Domestic Disposal of Waste Plant Material. Project BH 004. MAF Quality Management. 12 pages. June 1993.

[94] Dolan, L. *Where to Now? Christchurch Solid and Hazardous Waste Management Public Discussion Document.* Christchurch City Council. 30 pages, 1992?

Thorstensen, L, Planning Engineer, Wellington City Council, personal communication with Stuart MacDiarmid, 1993.

Meilink, A, Solid Waste Management Section, Wellington City Council, personal communication with SC MacDiarmid, 9 March 1994.

Between 61% and 74% of New Zealanders eat fish at least once a week, but salmon is eaten as a main meal by only around 4-5% of fish-eaters[95] and much of this is in the form of canned salmon. The average serving of fish per person is between 100 g[96] and 220-250 g[97], with servings of (fresh or frozen) salmon tending to be a little larger, in the order of 250-300 g.[98]

In an attempt to estimate the likely volume of salmon to be imported into New Zealand, should restrictions be eased, advice was sought from companies which had expressed an interest in importing from Canada.[99] Would-be importers expect Canadian salmon to be more expensive than locally-produced farmed salmon. Imported salmon is seen as filling a "...small niche market" and would-be importers expect the volume imported to be substantially less than the volume presently produced locally. Estimates of actual tonnages likely to be imported ranges from 5 to 10 tonnes, with a maximum of 20 tonnes, up to 100 tonnes per year.

---

[95] Horwath, C, Parnell, W, Birkbeck, J, Wilson, N, Russell, D, Herbison, P. *Life in New Zealand. Commission Report. Volume VI. Nutrition.* Hillary Commission for Recreation and Sport, Wellington, 1991.

McNair, A G B. *Survey of Fish Eating Habits. A Report for the New Zealand Fishing Industry Board.* A G B McNair Ltd, Wellington, 1990.

McNair, A G B. *Survey of Fish Consumption Patterns. Prepared for the New Zealand Fishing Industry Board.* A G B McNair Ltd, Wellington, 1991.

Phipps, Jean. Press release. New Zealand Fishing Industry Board. 12 October 1990.

[96] Phipps, Jean. Press release. New Zealand Fishing Industry Board. 12 October 1990.

[97] Kyaw Tay, Head Chef, Terrace Regency Hotel, Wellington, personal communication with Martin Van Ginkel, 28 september 1994.

[98] Kyaw Tay, Head Chef, Terrace Regency Hotel, Wellington, personal communication with Martin Van Ginkel, 28 september 1994.

[99] Cavanagh, R, Director Seafood, Woolworths (NZ) Ltd, personal communication with SC MacDiarmid, 1994.

Hall, R, Managing Director, James Crisp Ltd, personal communication with SC MacDiarmid, 1994.

Warren, T, Southfresh Ltd, personal communication with SC MacDiarmid, 1994.

In 1992 2,600 tonnes of New Zealand-produced salmon was exported[100] while between 10% and 15% of locally-produced salmon was consumed within New Zealand.[101] From these figures one can estimate that between 289 and 459 tonnes of locally-produced salmon is consumed annually within New Zealand. As the amount of imported salmon is likely to be substantially less than the amount of locally-produced salmon consumed, these figures allow an upper estimate to be placed on the amount of imported product entering the country. For the purposes of this analysis, an upper value of 200 tonnes annually has been chosen as providing a conservative safety margin.

Approximately 10.5 kg of garbage per week is collected from each urban household.[102] The average number of persons per household is 2.9.[103] If the volume of salmon imported per year were 200 tonnes (an improbably high estimate) this would be sufficient to provide 300 g servings of salmon to each person in 4,600 households, with each household recieving, on average, 870 g per week of imported product. If each household disposed of 17% of this **uncooked** as kitchen scraps (see Section 5.5.1.1 above), this would be a 'concentration' of 150 g uncooked salmon scraps per 10,500 g (10.5 kg) waste from each of the consuming households. There are about 1.185×10^6 households in New Zealand[104] so the waste from each household consuming salmon would be diluted into the waste from over 1.1804×10^6 other households. Therefore, each 150 g of uncooked salmon scraps from consuming households would be diluted by approximately 12,512 tonnes of collected residential waste. That is, each 1 g of uncooked salmon scraps would be diluted into approximately 18.13 **tonnes** of

---

[100] New Zealand Official Yearbook. 97th edition. Statistics New Zealand, 587 pages, 1994.

[101] Smith, F. National Institute of Water and Atmospheric Research Ltd, personal communication with Martin Van Ginkel, 23 September 1994.

O'Sullivan, K. Chairman, New Zealand Salmon Farmers Association, personal communication with Martin Van Ginkel, 26 September 1994.

[102] Dolan, L. *Where to Now? Christchurch Solid and Hazardous Waste Management Public Discussion Document.* Christchurch City Council. 30 pages, 1992?

Thorstensen, L, Planning Engineer, Wellington City Council, personal communication with Stuart MacDiarmid, 1993.

Meilink, A, Solid Waste Management Section, Wellington City Council, personal communication with SC MacDiarmid, 9 March 1994.

[103] New Zealand Official Yearbook. 97th edition. Statistics New Zealand, Auckland, 587 pages, 1994.

[104] New Zealand Official Yearbook. 97th edition. Statistics New Zealand, Auckland, 587 pages, 1994.

other collected residential waste. On top of this, each 1 kg of collected residential waste is diluted with 13.92 kg of waste from other sources (see above).

Refuse in New Zealand is disposed of into landfills. The establishment and operation of landfills are activities subject to the provisions of the Resource Management Act 1991. The provisions of the Act apply not only to all aspects of the establishment, filling and post-closure of new proposals, but also to the performance of existing landfills.[105]

The establishment of a landfill requires the granting of consents by a Regional Council and/or Territorial Local Authority. Matters requiring consent include a land use consent, a discharge permit, a water permit for the collection and control of stormwater and a permit for the discharge of treated leachate.

Landfill site conditions must be managed in such a way as to control litter, dust, odour, birds, pests etc. The way refuse is placed, spread, compacted and covered are all subject to control. Daily covering of refuse should be provided at all landfills. Leachate is also controlled.

So, modern waste management practices require that waste is buried within hours of being dumped, dumps are fenced, measures are taken to control litter and leachate.[106] The proportion of solid waste not buried is unlikely to be very great. Certainly, not as high as 1%. For a city like Wellington or Christchurch 1% would be around 400-700 tonnes of residential solid waste remaining unburied per year. This is clearly an absurdly high figure.

The quantity of rubbish littering New Zealand has been decreasing steadily in recent years, with surveys demonstrating a 39% decrease in litter since 1986.[107]

---

[105] Centre for Advanced Engineering. Our Waste: Our Responsibility. Towards Sustainable Waste Management in New Zealand. Project Report. University of Canterbury. 467 pages. 1992.

[106] Thorstensen, L, Planning Engineer, Wellington City Council, personal communication with Stuart MacDiarmid, 1993.

Meilink, A, Solid Waste Management Section, Wellington City Council, personal communication with SC MacDiarmid, 9 March 1994.

[107] Drum, Tracy, National Manager, Keep New Zealand Beautiful Society Inc., personal communication with Martin Van Ginkel, 1994.

While the quality of waste management will vary around the country, with smaller centres probably having less secure systems, the larger urban centres, generating the most waste, have the best quality systems.[108]

The estimates for salmon scraps not buried (P3) used in this simulation are conservatively high and almost certainly overstate the risk.

5.5.1.4 Contaminate fresh water? (R6)

A relatively high proportion of kitchen waste is disposed of through kitchen sink waste units directly into waste water and will be dealt with in a later section. This section examines only that kitchen waste dealt with by burial.

If uncooked salmon scraps do escape burial, what is the likelihood of their contaminating fresh water? Most residential kitchen waste is disposed of by burial or composting in the home garden or by collection in rubbish bags That is, much of the kitchen waste is inside bags. While leachate from urban landfills is controlled and is likely to be directed into sewage systems[109], the inability of *A. salmonicida* to survive in the environment in competition with other bacteria (see Section 3.8) means that any which finds its way into landfills will be destroyed long before it could percolate out in leachate. Dumped rubbish is buried within hours of disposition.

From the time that salmon scraps are generated in the kitchen they will start to putrefy. Putrefaction will progress during the time scraps are stored in household garbage, collected, transported to a landfill and buried. The process of putrefaction is likely to destroy *A. salmonicida* and other pathogens as it is recognised that *A. salmonicida* does not compete well with environmental bacteria and, in fact, is rapidly destroyed in media where environmental bacteria are present.

It has been suggested[110] scavenging birds such as seagulls, which are commonly attracted to rubbish tips, could transfer *A. salmonicida* in or on their bodies to contaminate waterways. However, taking, for example, a worst-case (and essentially impossible) scenario whereby a 2 g trimming was discarded from a fillet derived from a Pacific salmon that was actually dying of furunculosis, on average, such a trimming

---

[108] Thorstensen, L, Planning Engineer, Wellington City Council, personal communication with Stuart MacDiarmid, 1993.

[109] Thorstensen, L, Planning Engineer, Wellington City Council, personal communication with Stuart MacDiarmid, 1993.

Meilink, A, Solid Waste Management Section, Wellington City Council, personal communication with SC MacDiarmid, 9 March 1994.

[110] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

would contain a total of around $2x10^5$ viable *A. salmonicida* cells before freezing and $2x10^3$ viable cells after freezing and thawing. Even if this trimming was scavenged before putrefaction (and hence destruction of *A. salmonicida* by competing environmental bacteria could take effect), this dosage would likely be insufficient to establish infections in susceptible fish; it failed to cause infections in highly susceptible Atlantic salmon by ingestion, and exposure of Atlantic salmon to water containing 10^3 *A. salmonicida* cells/ml for 3 days also failed to cause infection (see Section 3.8).

Further, if a trimming were eaten by a warm-blooded animal such as a rat or a gull, the pathogen would be rapidly killed by the high body temperatures of these animals. *A. salmonicida* cells, unlike those of certain of the other fish pathogens, are rapidly killed at temperatures of 37 °C and over. Studies at the Pacific Biological Station, Nanaimo[111] have shown that cells of the pathogen suspended in peptone-saline suffered over a 98% loss in viability within 30 minutes when exposed to a temperature of 40 °C. At 37 °C in tryptic-soy broth, a medium capable of supporting growth of *A. salmonicida*, there was a 70% loss of viability in 60 minutes and a 99% loss in 240 minutes. Ingestion of uncovered garbage (trimmings) by gulls and rats would therefore be a "certain end" to any *A. salmonicida* cells that escaped from the kitchen to the environment.

The calculations made above were based on bacterial numbers in salmon actually dying from furunculosis rather than carrier fish. With carrier fish, the numbers of the pathogen present in flesh would, on average, be considerably less (on average $10^{3.5}$ times less) if evenly distributed; even if distributed unevenly, as in lesions (furuncles, which, by definition, would not be found in clinically normal carrier fish), the numbers in the lesions would be unlikely to exceed the numbers found in the flesh of the Pacific salmon dead of furunculosis. These cell numbers are on average 10^5 times less (10^7 times less after freezing) than the unrealistic number (10^{10}) cited by Anderson.[112]

As discussed above, *A. salmonicida* does not survive long at elevated temperatures. The internal body temperature of a variety of gull species ranges from 40.7 to 42.6 °C That of the black backed gull (*Larus dominicanus*) is 40.9 °C. This means that 98% of any *A. salmonicida* present on the hypothetical piece of discarded salmon tissue would be destroyed in 30 minutes at avian internal body temperatures. (A typical avian gut residence time is 3 hours.)

---

[111] Evelyn TP, personal communication with SC MacDiarmid during a visit to Nanaimo, July 1994, and Price, Iola M, Director, Aquaculture and Resource Development, Biological Sciences Directorate, Department of Fisheries and Oceans, Ottawa, personal communication with Barry O'Neil, Chief Veterinary Officer, September 1994.

[112] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

Further, the avian digestive system has a pH lower than the optimum for survival of *A. salmonicida*, ranging from 2.6-5.0 in the gizzard of domestic fowl and becoming more basic (5.5-7.5) through progression to the small intestine, so there is no basis for suggesting that *A. salmonicida* would survive passage through the acid stomach of fish, birds, and mammals. Indeed, poor survival of the bacterium in the gastrointestinal tract of fish may explain why such high numbers of the pathogen are required to establish infections in fish by the oral route (see Section 3.8). In birds and mammals, as discussed earlier, survival in the gastrointestinal tract is not likely because of the high body temperatures in these animals.

The article referred to[113] suggests that *A. salmonicida* could be transported from rubbish dumps to waterways **on** the bodies of scavengers such as gulls. Should gulls become sufficiently contaminated externally with a particular bacterium they could carry it on their bill, plumage or feet.[114] However, given the information already presented on the numbers of *A. salmonicida* present in the flesh of carrier fish, the poor survival of the organism in competition with environmental bacteria, and the high doses required to establish infection in susceptible fish of any species, introduction of infection on the exterior of gulls is improbable in the extreme.

It is difficult to quantify the extent to which dumped scraps might contaminate waterways because the probability of the pathogen surviving through the necessary chain of steps is so small. Careful consideration of the information presented, however, leads inevitably to the conclusion that the likelihood of such contamination must be very small indeed. However, the possibility of picnickers dropping or throwing salmon scraps into waterways has been raised as a concern by some.[115] Clearly it is difficult to put probability estimates on the likelihood of this event happening. It is, however, probable that most people would eat salmon cooked and that most scraps disposed of in salmon sandwiches and the like will, thus, not be raw.

Furthermore, whatever the annual consumption of imported Pacific salmon, most of it will **not** be consumed by picnickers at the seaside or on river banks. Most will be prepared in kitchens. The proportion consumed in situations where raw scraps could be thrown directly into water is unlikely to be great. Nevertheless, this possibility,

---

[113] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

[114] Bartle, JA, Curator of Birds, Museum of New Zealand, personal communication with SC MacDiarmid, 10 August 1994.

[115] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with SC MacDiarmid, 23 December 1993.

remote as it is, requires a high "Maximum" value to be entered into the simulation model.[116]

Given the low prevalence of *A. salmonicida* infection in ocean-caught Pacific salmon, the low bacterial counts which would be expected in export grade product, and the relatively high dose required to infect susceptible fish (of any species) by mouth, the spectre of picnickers feeding trout with uncooked salmon sandwiches cannot, realistically, be seen as a compelling enough reason to deny access for this commodity.

5.5.1.5 Infect a susceptible host when present? (R7)

Assuming that some infective salmon scraps do escape burial and do contaminate fresh water, what is the probability that they would come into infective contact with a susceptible host, if such a host were present in the waterway?

This is extremely difficult to quantify. It is likely that scraps would be attractive to fish, but many non-susceptible host species would be competing with susceptible species for the available scraps (see Sections 3.8 and 5.3.1). It is also likely that a proportion of scraps would not come into contact with any fishes, sinking instead to the bottom of waterways and putrefying.

In Section 3.8 it was shown that the dose of *A. salmonicida* required to infect fish is relatively high. For example, immersion in a concentration of 10^3 cells/ml *A. salmonicida* for 2-3 days failed to establish infection in stressed Pacific salmon. Atlantic salmon did not become infected following immersion in $3x10^3$ cfu/ml/day for 3 days nor following immersion in 10^2 cfu/ml for 1 week.

Data were also presented in Section 3.8 showing that doses in the order of 10^2 cells/kg to $>10^5$ cells/fish are required to infect salmon by mouth. Non-salmonids are even less susceptible to infection with typical *A. salmonicida*, the strain found in Pacific salmon.

Tables 3.4 and 3.5 presented information on the *A. salmonicida* numbers found in tissues of fish dying of furunculosis. As has been pointed out, the Pacific salmon intended for export to New Zealand will not be clinically affected with furunculosis, although a low percentage could have been carrying low numbers of the bacterium in those tissues which were removed during the process of evisceration.

It is highly unlikely that sufficient *A. salmonicida* cells could be present in imported wild, ocean-caught Pacific salmon for scraps to be capable of infecting New Zealand

---

[116] Nelson Boustead has described the situation whereby a fish processing plant discharges fish waste directly into an estuary (personal communication with SC MacDiarmid, 23 December 1993). For this reason, commercial importations of wild, ocean-caught, headless, eviscerated Pacific salmon intended for further processing within New Zealand could only be permitted to approved premises where the waste disposal systems meet appropriate standards.

fish by the oral route. It is almost inconceivable that such product could carry a bacterial loading sufficient to produce the sustained concentration of *A. salmonicida* necessary to introduce infection by immersion. It is difficult to visualize a situation in which the product could provide the continuity of exposure required to establish infections with such low doses of the pathogen.

The values used in this simulation model are conservatively, probably unrealistically, high.

5.5.1.6 Contaminate estuarine water? (R8)

Similar arguments to those used in R6 apply, although it is considered that there is a lower risk of contaminating estuarine water.

5.5.1.7 Infect a susceptible host when present? (R9)

Arguments similar to those used in R7 apply.

5.5.1.8 Contaminate sea water? (R10)

As in R8.

5.5.1.9 Infect a susceptible host when present? (R11)

Arguments similar to those used in R7 apply, although, because of the much more rapid dilution in such a large body of water, and the poor survival of *A. salmonicida* in sea water, lower risk estimates are used. These are, nevertheless, probably too high.

5.5.2 Simulation results

The graph below (Figure 5.6) shows the probable number of contaminated, potentially infective fish imported into Region 1.

Expected/mean result	=	0.2
Maximum result	=	1.9
Minimum result	=	2×10^{-5}

Probability;	That the number of fish is less than;
90%	0.5
95%	0.6
99%	1.0

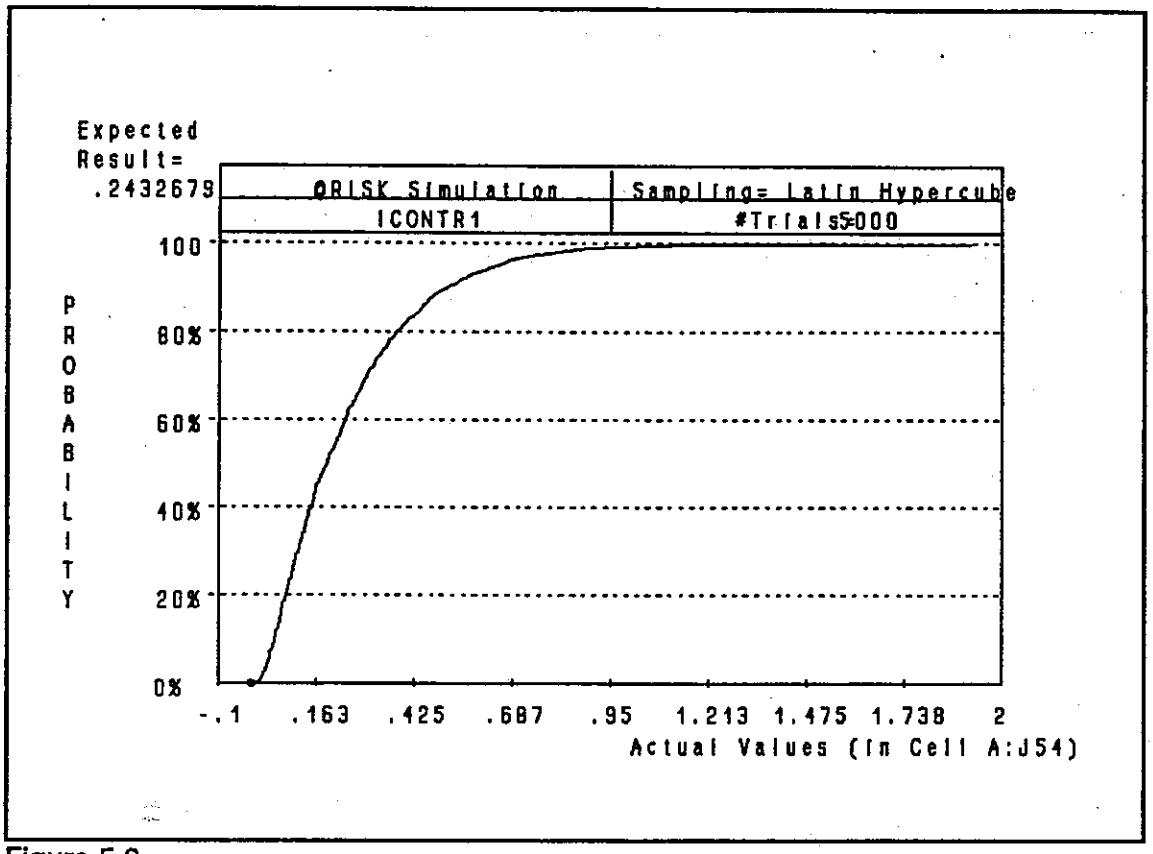


Figure 5.6

This simulation shows that there is a 95% probability that the number of contaminated fish imported into Region 1 is fewer than 0.6 for every tonne imported into New Zealand.

Figure 5.7 shows the probability of scraps serving as the vehicle by which *A. salmonicida* gets introduced into a susceptible salmonid in waterways in Region 1.

Expected/mean result	=	1.7×10^{-10}
Maximum result	=	3.5×10^{-9}
Minimum result	=	0[117]

Probability;	That risk is less than;
90%	4.1×10^{-10}
95%	6.0×10^{-10}
99%	1.2×10^{10}

[117] Values smaller than 10^{-10} are reported as 0.

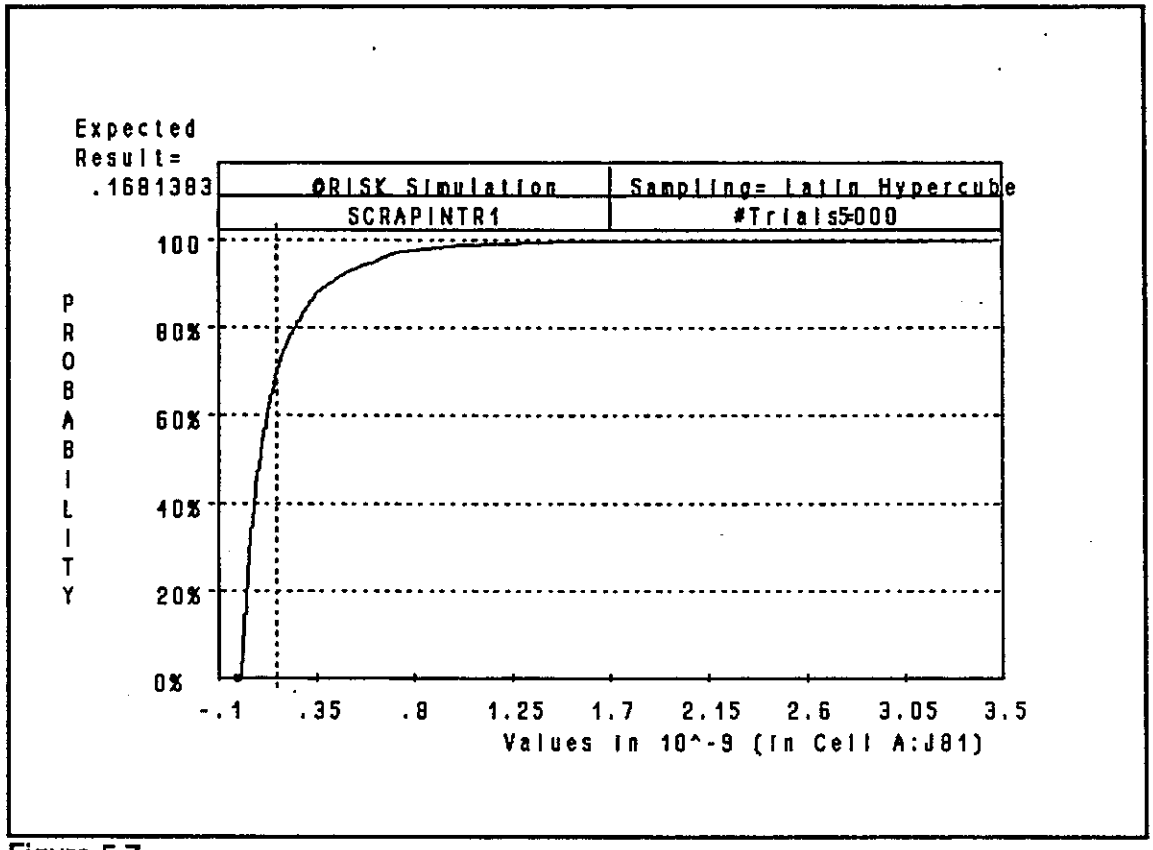


Figure 5.7

This simulation shows that there is a 95% probability that the risk of introducing infection into Region 1 through the medium of scraps is less than 6 disease inroductions per 10,000 million tonnes of headless, eviscerated ocean-caught Pacific salmon imported into New Zealand. That is, the disease risk is, essentially, zero.

5.6 Region 2, scraps introduce infection

For each tonne imported, the number of contaminated, potentially infective fish entering Region 2 is calculated;

$$(D)r2 = Dx(I)r2$$

Table 5.7: Input variables used to calculate probability of scraps introducing infection into Region 2.

	Minimum	Most Likely	Maximum
Proportion disposed of as uncooked scraps (P1)r2	0.1	0.17	0.34
Proportion not incinerated (P2)r2	0.95	0.98	1.0
Proportion not buried (P3)r2	10^{-4}	0.001	0.005
Contaminate fresh water? (R6)r2	0.001	0.01	0.05
Proportion of fishery in region 2 (Sp)r2			
Infect suscept. host when present? (R7)r2	10^{-5}	10^{-4}	0.01
Infect suscept. host in Region 2 (SpxR7)r2			
Infection introduced into fresh water (S1)r2 = (DxP1xP2xP3xR6xR7xSp)r2			
Contaminate estuarine water? (R8)r2	0.001	0.01	0.05
Infect suscept. host when present? (R9)r2	10^{-5}	10^{-4}	0.01
Infection introduced into estuarine water (S2)r2 = (DxP1xP2xP3xR8xR9xSp)r2			
Contaminate sea water? (R10)r2	0.001	0.01	0.05
Infect suscept. host when present? (R11)r2	10^{-5}	10^{-4}	0.005
Infection introduced into sea water (S2)r2 = (DxP1xP2xP3xR10xR11xSp)r2			
SCRAPS INTRODUCE INFECTION INTO REGION 2/TONNE (I1)r2 = (S1 + S2 + S3)r2			

5.6.1 The evidence

The evidence used in 5.5.1 applies equally to this section.

5.6.2 Simulation results

Figure 5.8 shows the probable number of contaminated fish imported into Region 2.

Expected/mean result	=	0.2
Maximum result	=	1.4
Minimum result	=	3×10^{-5}

Probability;	That the number of fish is less than;
90%	0.5
95%	0.6
99%	0.8

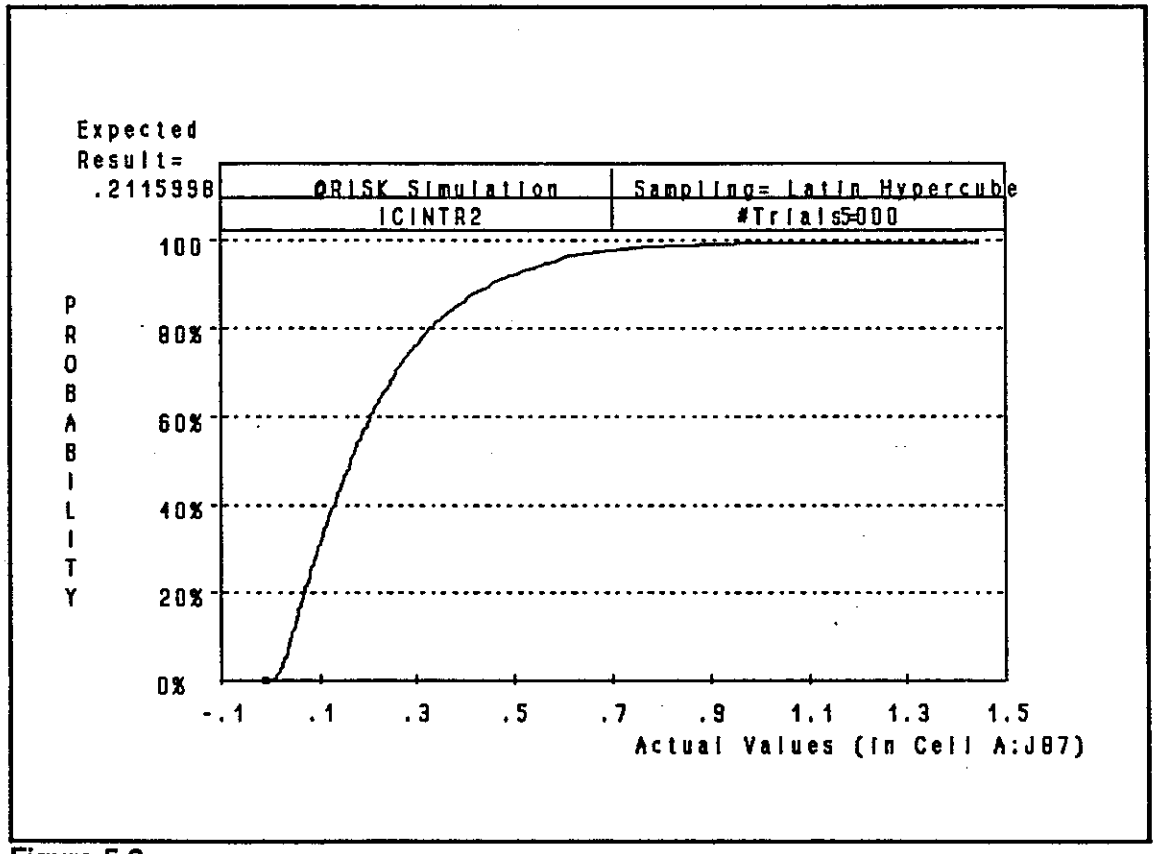


Figure 5.8

This simulation shows that there is a 95% probability that the number of contaminated fish imported into Region 2 is fewer than 0.6 for every tonne imported into New Zealand.

Figure 5.9 below shows the probability of scraps serving as the vehicle by which *A. salmonicida* gets introduced into a susceptible salmonid in waterways in Region 2.

Expected/mean result	=	3.4×10^{-9}
Maximum result	=	1.3×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	8.3×10^{-9}
95%	1.2×10^{-8}
99%	2.3×10^{-7}

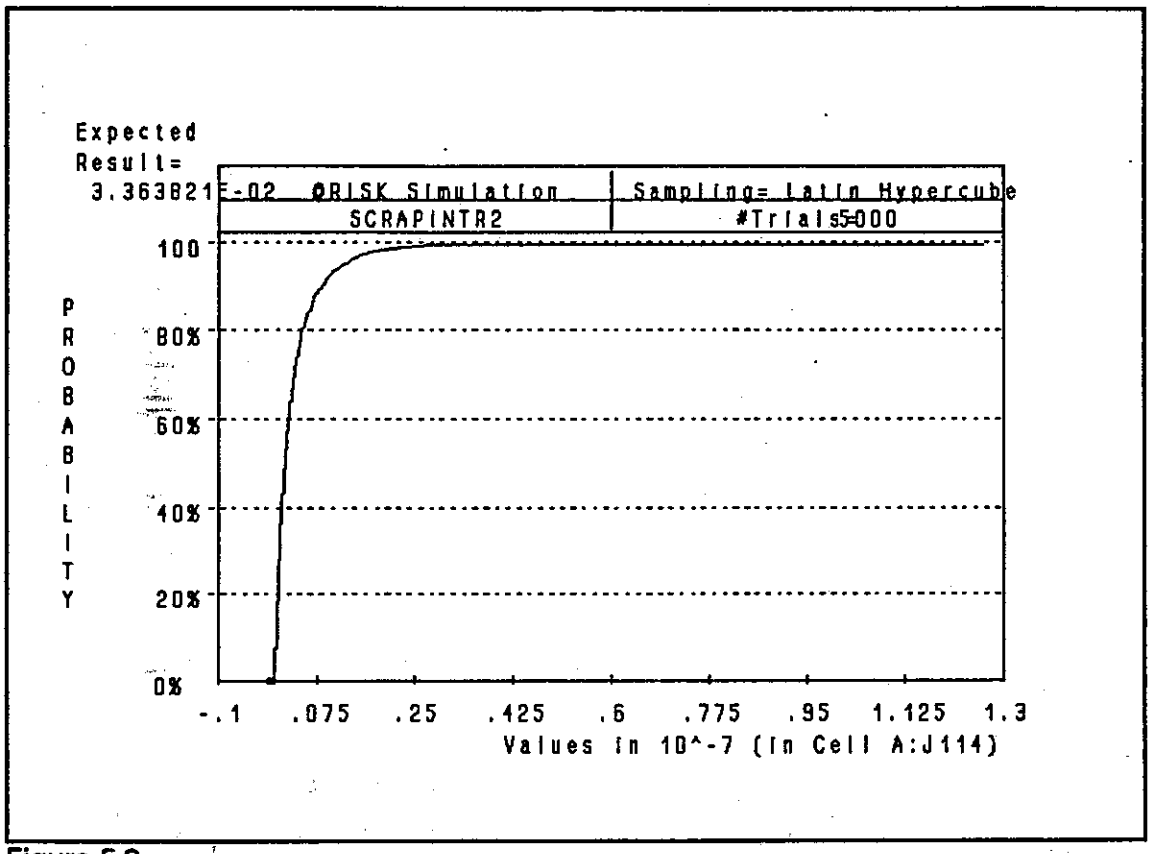


Figure 5.9

This simulation shows that there is a 95% probability that the risk of introducing infection into Region 2 through the medium of scraps is less than 1.2 disease introductions per 100 million tonnes of headless, eviscerated ocean-caught Pacific salmon imported into New Zealand. That is, the risk is, essentially, zero.

5.7 Region 3, scraps introduce infection

For each tonne imported, the number of contaminated, potentially infective fish entering Region 3 is calculated;

$(D)r3 = Dx(I)r3$

Table 5.8: Input variables used to calculate probability of scraps introducing infection into Region 3.

	Minimum	Most Likely	Maximum
Proportion disposed of as uncooked scraps (P1)r3	0.1	0.17	0.34
Proportion not incinerated (P2)r3	0.95	0.98	1.0
Proportion not buried (P3)r3	0.0001	0.001	0.005
Contaminate fresh water? (R6)r3	0.001	0.01	0.05
Proportion of fishery in region 3 (Sp)r3			
Infect suscept. host when present? (R7)r3	10^{-4}	10^{-3}	0.01
Infect suscept. host in Region 3 (SpxR7)r3			
Infection introduced into fresh water (S1)r3 = (DxP1xP2xP3xR6xR7xSp)r3			
Contaminate estuarine water? (R8)r3	0.001	0.01	0.05
Infect suscept. host when present? (R9)r3	10^{-4}	10^{-3}	0.01
Infection introduced into estuarine water (S2)r3 = (DxP1xP2xP3xR8xR9xSp)r3			
Contaminate sea water? (R10)r3	0.001	0.01	0.05
Infect suscept. host when present? (R11)r3	10^{-4}	10^{-3}	0.005
Infection introduced into sea water (S2)r3 = (DxP1xP2xP3xR10xR11xSp)r3			
SCRAPS INTRODUCE INFECTION INTO REGION 3/TONNE (I1)r3 = (S1 + S2 + S3)r3			

5.7.1 The evidence

The same evidence used in 5.5.1 applies here. There is no reason to assume that these values differ significantly between regions.

5.7.2 Simulation results

The next graph (Figure 5.10) shows the probable number of contaminated fish imported into Region 3.

Expected/mean result	=	0.2
Maximum result	=	1.1
Minimum result	=	3×10^{-5}

Probability;	That the number of fish is less than;
90%	0.3
95%	0.4
99%	0.6

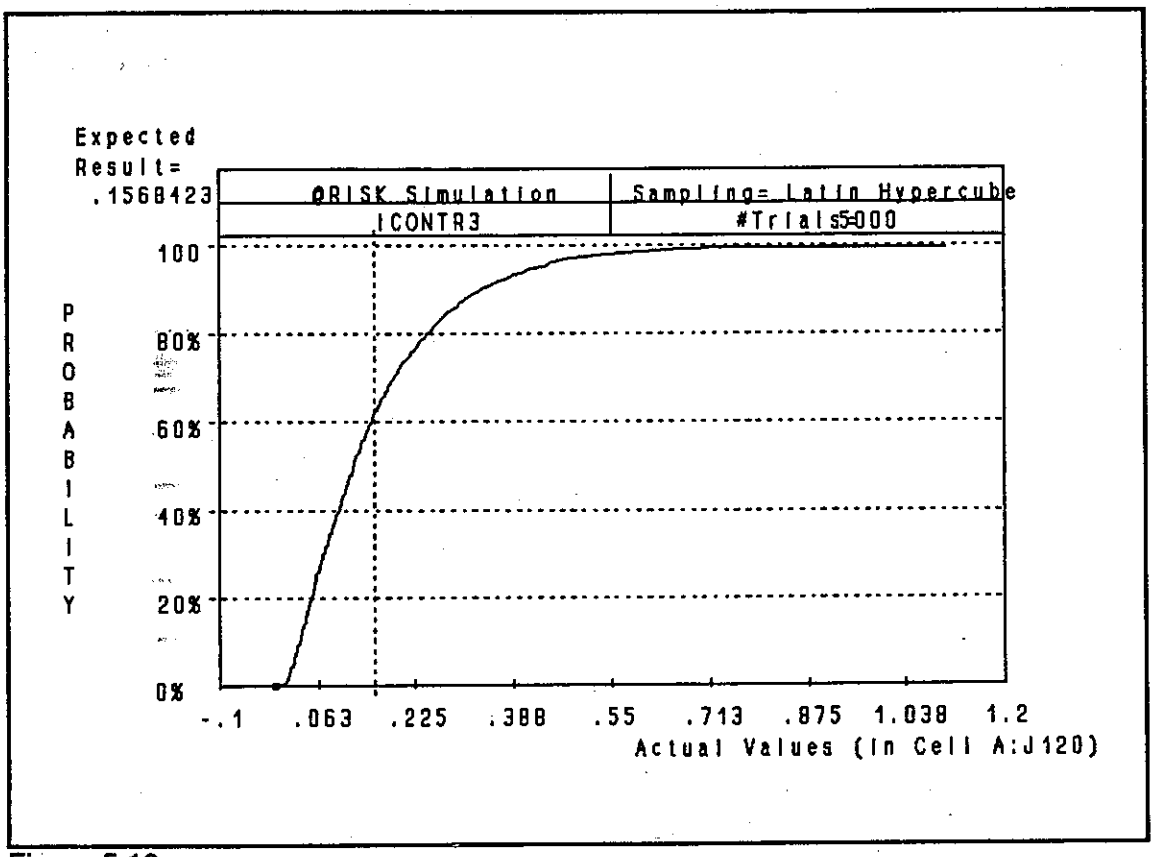


Figure 5.10

This simulation shows that there is a 95% probability that the number of contaminated fish imported into Region 3 is fewer than 0.4 for every tonne imported into New Zealand.

Figure 5.11 shows the probability of scraps serving as the vehicle by which *A. salmonicida* gets introduced into a susceptible salmonid in waterways in Region 3.

Expected/mean result	=	3.3×10^{-9}
Maximum result	=	1.1×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	8.3×10^{-9}
95%	1.3×10^{-8}
99%	2.9×10^{-8}

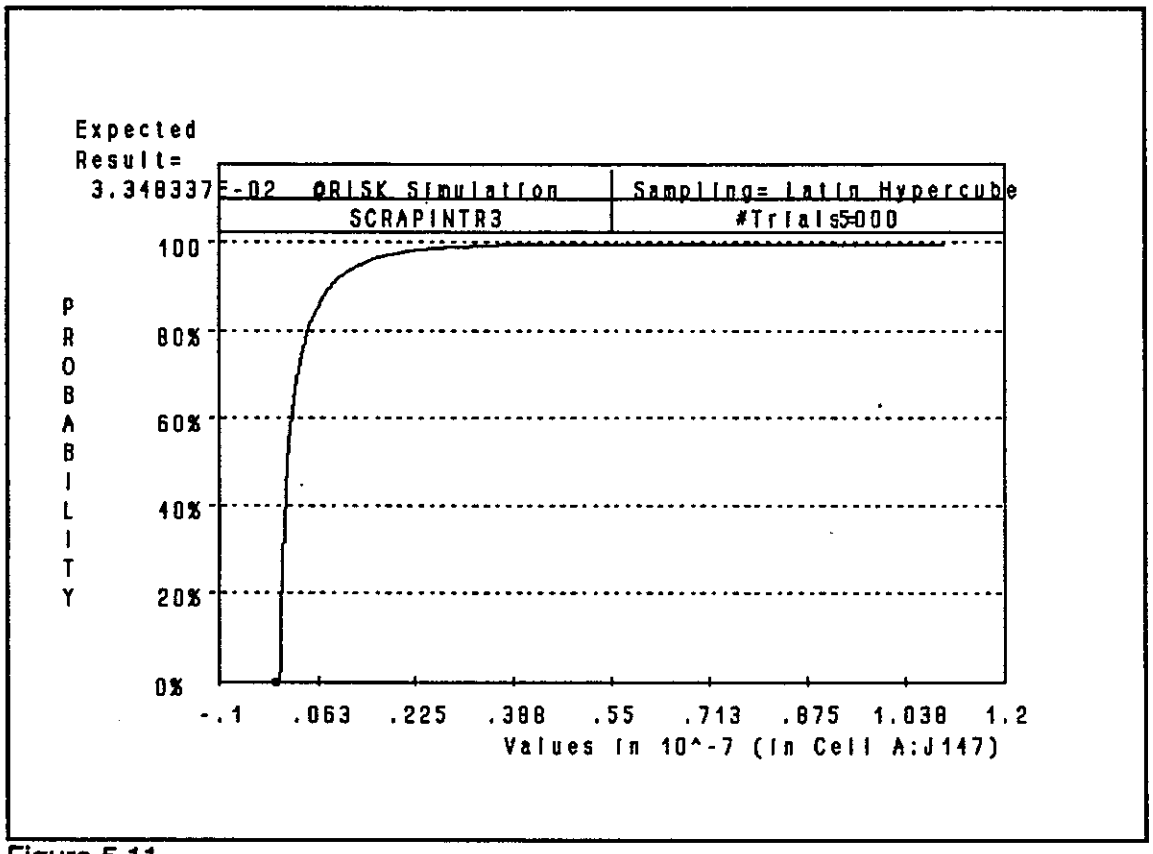


Figure 5.11

This simulation shows that there is a 95% probability that the risk of introducing infection into Region 3 through the medium of scraps is less than 1.3 disease introductions per 100 million tonnes of headless, eviscerated ocean-caught Pacific salmon imported into New Zealand. That is, the risk is essentially zero.

5.8 Cumulative risk of scraps introducing infection

The cumulative risk of scraps acting as the vehicle by which *A. salmonicida* could be introduced is calculated by adding the risks for each region. That is, the risk is;

$$(I1)r1 + (I1)r2 + (I1)r3$$

This cumulative risk is shown graphically in Figure 5.12.

Expected/mean result	=	6.9×10^{-9}
Maximum result	=	1.7×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.6×10^{-8}
95%	2.2×10^{-8}
99%	4.1×10^{-8}

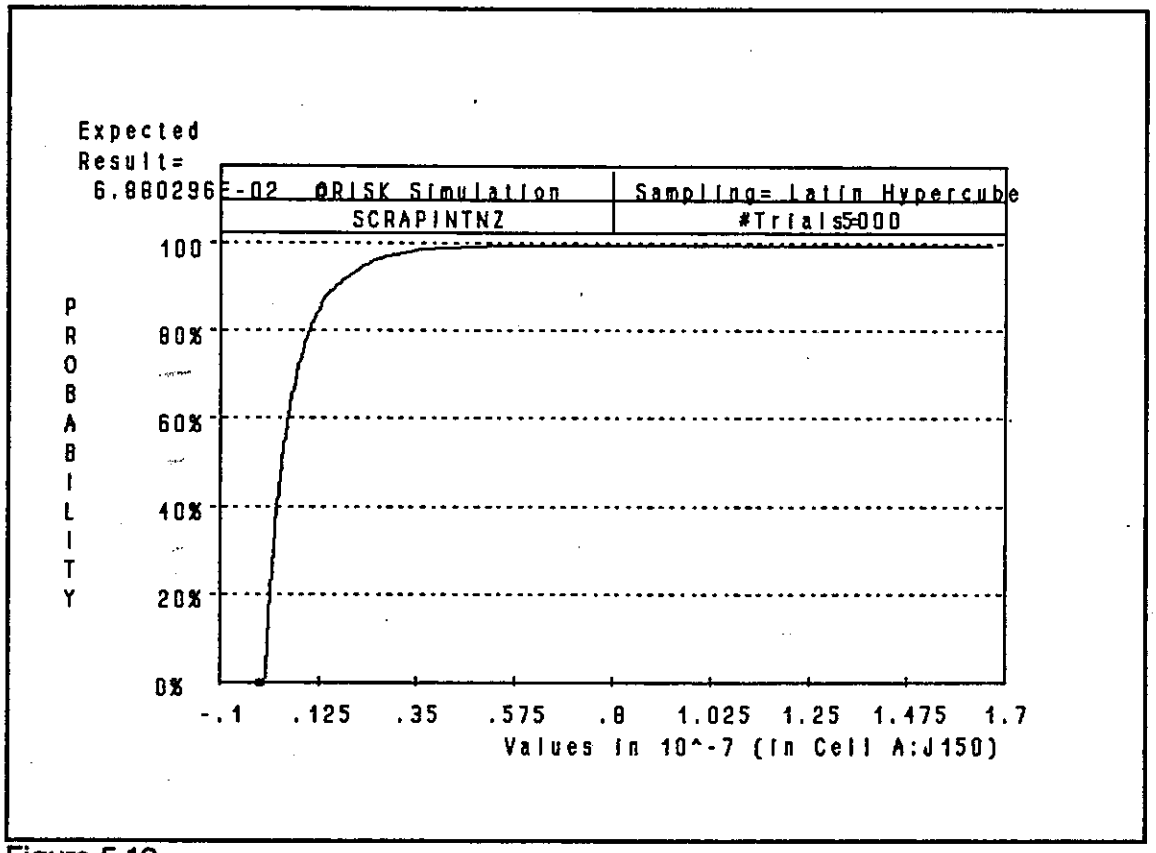


Figure 5.12

This simulation shows that, under the assumptions outlined above, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand through the medium of scraps is less than 2.2 disease introductions per 100 million tonnes of **chilled**, headless, eviscerated ocean-caught Pacific salmon imported. That is, the disease risk from scraps is essentially zero.

5.9 The probability that wrapping will introduce infection

It has been claimed that the material used to wrap salmon or salmon flesh contaminated with *A. salmonicida* has a high probability of itself becoming contaminated and acting as a vehicle for the introduction of infection. Wrapping material not promptly burned or buried may be blown, washed or carried by

scavengers into watercourses. The assumptions made and the method of assessing the risk that wrapping will introduce infection are shown below.

5.10 Region 1, wrapping introduces infection

It will be recalled (from Section 5.5 above) that for each tonne imported, the number of contaminated, potentially infective fish entering Region 1 is calculated;

$$(D)r1 = Dx(I)r1$$

The estimated values for (D)r1 have been given in Section 5.5.2. The other assumptions are:

Table 5.8: Input variables used to calculate probability of wrapping introducing infection into Region 1.

	Minimum	Most Likely	Maximum
If contaminated fish, probability that wrapping contaminated? (R12)r1	0.2	0.3	0.5
Wrapping contaminated in Region 1 (DxR12)R1			
Proportion not incinerated (P4)r1	0.95	0.98	1.0
Proportion not buried (P5)r1	10^{-4}	0.001	0.005
Contaminate fresh water? (R13)r1	10^{-4}	0.01	0.1
Proportion of fishery in Region 1 (Sp)r1			
Infect suscept. host when present? (R14)r1	10^{-5}	10^{-4}	0.001
Infection suscept. host in Region 1 (SpxR14)r1			
Infection introduced into fresh water (W1)r1 = (DxR12xP4xP5xR13xR14xSp)r1			
Contaminate estuarine water? (R15)r1	10^{-4}	0.01	0.1
Infect suscept. host when present? (R16)r1	10^{-5}	10^{-4}	0.001
Infection introd. into estuarine water (W2)r1 = (DxR12xP4xP5xR15xR16xSp)r1			
Contaminate sea water? (R17)r1	10^{-4}	0.01	0.1
Infect suscept. host when present? (R18)r1	10^{-5}	10^{-4}	0.001
Infection introduced into sea water (W3)r1 = (DxR12xP4xP5xR17xR18xSp)r1			
WRAPPING INTRODUCE INFECTION REGION 1/TONNE (I2)r1 = (W1 + W2 + W3)r1			

5.10.1 The evidence

5.10.1.1 The probability that wrapping is contaminated (R12)

An article arguing that importation of salmon was likely to introduce *A. salmonicida*[118] postulated that the wrappers from around the product could serve to introduce furunculosis because they would be "...contaminated by their close contact with infected tissue [and] could also be blown or washed into waterways." However, this assertion was based on an unrealistic estimate of the bacterial loading likely to be present in imported wild, ocean-caught Pacific salmon. Earlier sections (see especially 3.8) have shown that clinical furunculosis has never been recorded in ocean-caught Pacific salmon off the west coast of North America and subclinical carriers, although they do occur, are uncommon. Further, it has been shown that *A. salmonicida*, if it occurs at all in the flesh of eviscerated carrier salmon, is present in very small numbers. While it cannot be excluded that wrappers could be contaminated with *A. salmonicida*, such contamination is unlikely and could not involve a significant number of bacterial cells.

5.10.1.2 Proportion of wrapping not incinerated or buried, (P4) and (P5)

The derivation of these estimates has been described is Sections 5.5.1.2 and 5.5.1.3. It could be argued that a higher proportion of wrappers than scraps would be incinerated, but this does not seem worth changing the estimates for.

5.10.1.3 Contaminate fresh water? (R13)

Much of what has been said in section 5.5.1.4 for R6 applies here. However, it could be argued that wrappings could be blown by wind and thus more easily escape from a dump site and find their way into waterways. For this reason a greater maximum value is used. However, the bacterial loading on wrappings will certainly be less than that in the actual salmon scraps and so more liable to destruction by desiccation, sunlight etc. For this reason a lower minimum value is used in the simulation.

5.10.1.4 Infect a susceptible host when present? (R14)

The risks of contaminated wrappings infecting susceptible hosts, when present in waterways, are likely to be lower than those for scraps (R7) outlined in section 5.5.1.5 because wrappers are less likely to be ingested by susceptible hosts and the bacterial loading is likely to be lower than in scraps and hence more rapidly diluted below an infective threshold. Considering the low numbers (if any) of *A. salmonicida* cells likely to be associated with the product, it is inconceivable that a fish eating even a whole

---

[118] Anderson, C. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19(1), 12-13, 1992.

wrapper would obtain a dose of *A. salmonicida* cells sufficient to establish an infection (see Section 3.8 for doses required to establish infection by the oral route). Then too, even if a contaminated wrapper were to be discarded directly into water, there would be a rapid reduction of bacterial numbers on it due dilution and to the lethal competition from environmental bacteria present in water.

5.10.1.5 Contaminate estuarine water? (R15)

As in 5.5.1.6 and R13 above.

5.10.1.6 Infect susceptible host when present? (R16)

As in 5.5.1.7 and R14 above.

5.10.1.7 Contaminate sea water? (R17)

As in 5.5.1.8 and R13 above.

5.10.1.8 Infect susceptible host when present? (R18)

While the arguments used in 5.5.1.9 apply, the dilution of surviving bacteria on the wrappings is likely to be so much more rapid in the large body of water that the risk values cannot be seen as greater than those used above in R14 and R16.

5.10.2 Simulation results

Figure 5.13 shows the probability of wrappings serving to introduce *A. salmonicida* into salmonid fish stocks in Region 1.

Expected/mean result	=	6.6×10^{-11}
Maximum result	=	1.4×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.6×10^{-10}
95%	2.2×10^{-10}
99%	0.5×10^{-10}

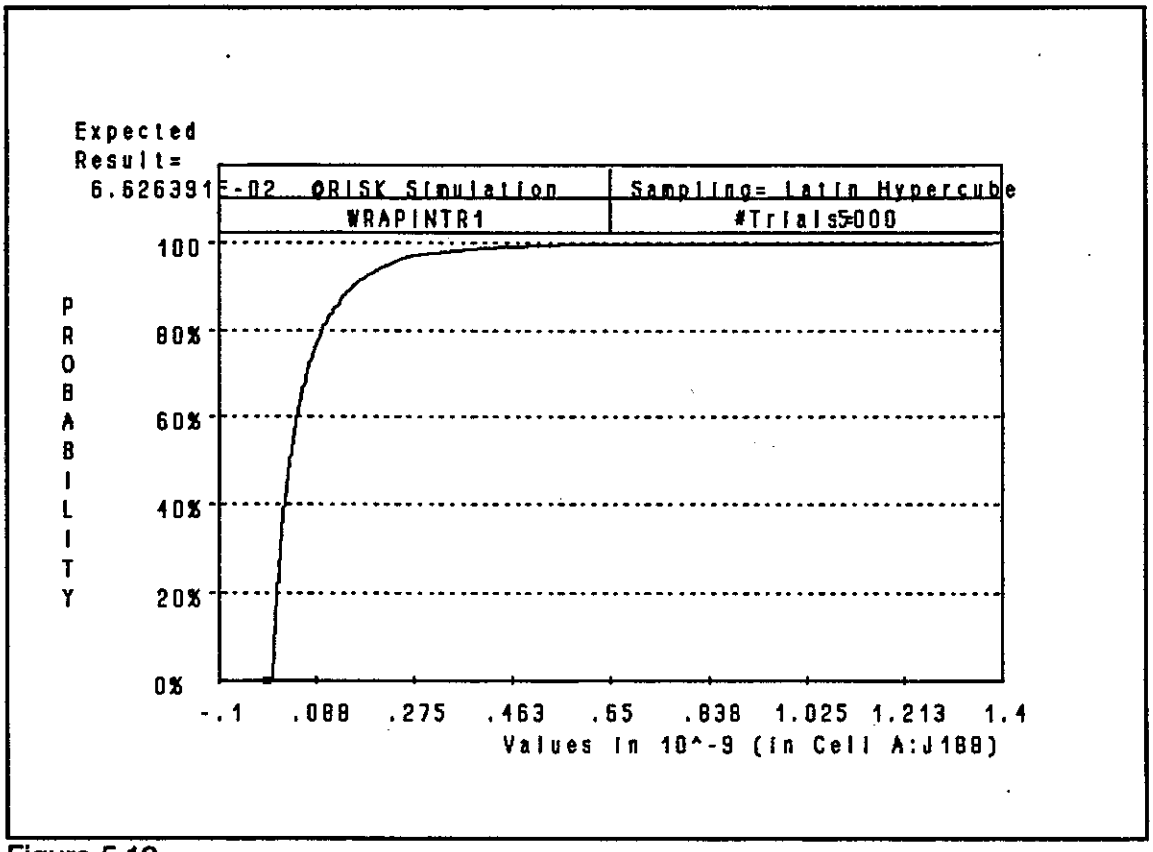


Figure 5.13

It can be seen that there is a 95% probability that the risk of wrappings introducing infection into Region 1 is less than 2.2 disease introductions per 100 million tonnes of **chilled**, headless, eviscerated Pacific salmon imported into New Zealand.

5.11 Region 2, wrapping introduces infection

The figures for the numbers of contaminated fish imported into Region 2, (D)r2, have been given in Section 5.6.2. The other assumptions used are;

Table 5.9: Input variables used to calculate probability of wrapping introducing infection into Region 2.

	Minimum	Most Likely	Maximum
If contaminated fish, probability that wrapping contaminated? (R12)r2	0.2	0.3	0.5
Wrapping contaminated in Region 2 (DxR12)r2			
Proportion not incinerated (P4)r2	0.95	0.98	1.0
Proportion not buried (P5)r2	0.0001	0.001	0.005
Contaminate fresh water? (R13)r2	10^{-4}	0.01	0.1
Proportion of fishery in Region 2 (Sp)r2			
Infect suscept. host when present? (R14)r2	10^{-5}	10^{-4}	0.001
Infection suscept. host in Region 2 (SpxR14)r2			
Infection introduced into fresh water (W1)r2 = (DxR12xP4xP5xR13xR14xSp)r2			
Contaminate estuarine water? (R15)r2	0.0001	0.01	0.1
Infect suscept. host when present? (R16)r2	10^{-5}	10^{-4}	0.001
Infection introd. into estuarine water (W2)r1 = (DxR12xP4xP5xR15xR16xSp)r1			
Contaminate sea water? (R17)r2	10^{-4}	0.01	0.1
Infect suscept. host when present? (R18)r2	10^{-5}	10^{-4}	0.001
Infection introduced into sea water (W3)r2 = (DxR12xP4xP5xR17xR18xSp)r2			
WRAPPING INTRODUCE INFECTION REGION 2/TONNE (I2)r2 = (W1 + W2 + W3)r2			

5.11.1 The evidence

The same arguments as used in 5.10.1 apply here.

5.11.2 Simulation results

Figure 5.14 shows the probability of wrappings serving to introduce *A. salmonicida* into salmonid fish stocks in Region 2.

Expected/mean result	=	2.8×10^{-9}
Maximum result	=	6.9×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	6.5×10^{-9}
95%	9.8×10^{-9}
99%	2.1×10^{-9}

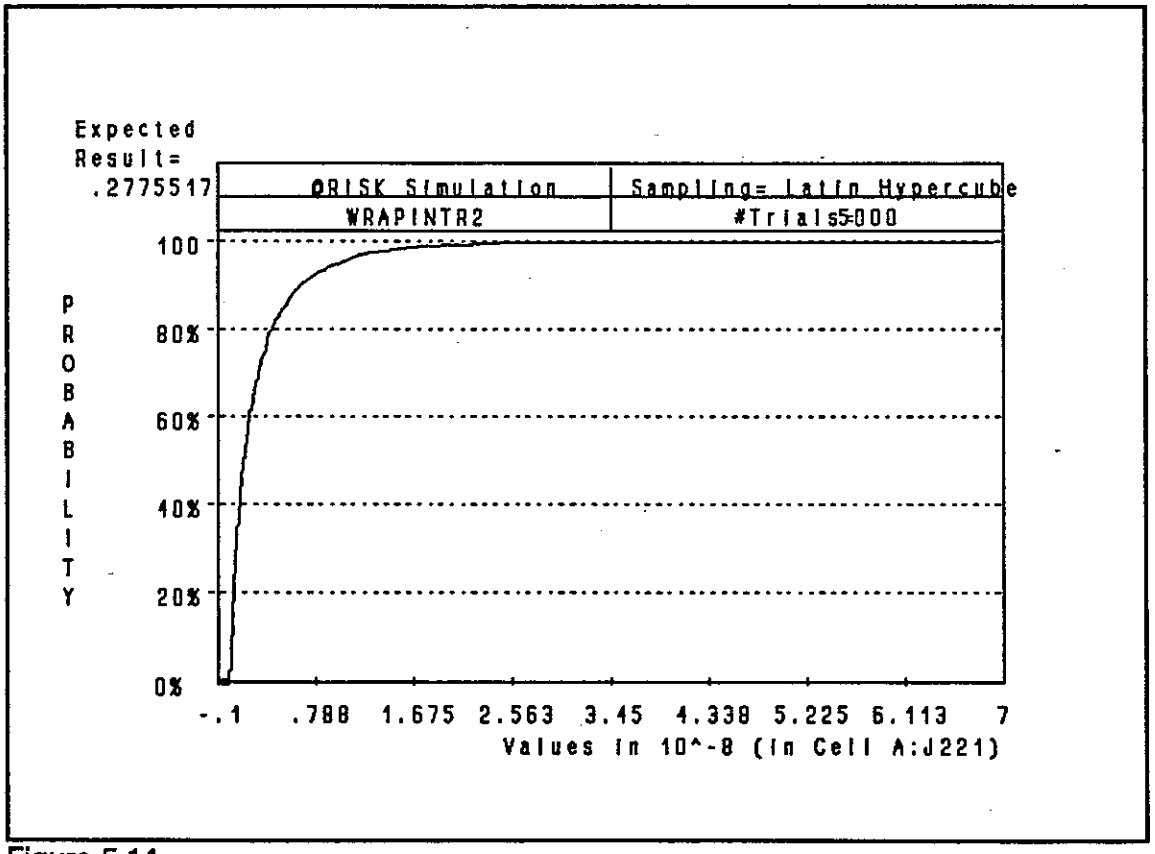


Figure 5.14

It can be seen that there is a 95% probability that the risk of wrappings introducing infection into Region 2 is less than 1 disease introduction per 100 million tonnes of **chilled**, headless, eviscerated Pacific salmon imported into New Zealand.

5.12 Region 3, wrapping introduces infection

The figures for the numbers of contaminated fish imported into Region 3, (D)r3, have been given in Section 5.7.2. The other assumptions used are;

Table 5.10: Input variables used to calculate probability of wrapping introducing infection to Region 3.

	Minimum	Most Likely	Maximum
If contaminated fish, probability that wrapping contaminated? (R12)r3	0.2	0.3	0.5
Wrapping contaminated in Region 3 (DxR12)R3			
Proportion not incinerated (P4)r3	0.95	0.98	1.0
Proportion not buried (P5)r3	0.0001	0.001	0.005
Contaminate fresh water? (R13)r3	10^{-4}	0.01	0.1
Proportion of fishery in Region 3 (Sp)r3			
Infect suscept. host when present? (R14)r3	10^{-5}	10^{-4}	0.001
Infection suscept. host in Region 3 (SpxR14)r3			
Infection introduced into fresh water (W1)r3 = (DxR12xP4xP5xR13xR14xSp)r3			
Contaminate estuarine water? (R15)r3	10^{-4}	0.01	0.1
Infect suscept. host when present? (R16)r3	10^{-5}	10^{-4}	0.001
Infection introd. into estuarine water (W2)r1 = (DxR12xP4xP5xR15xR16xSP)r1			
Contaminate sea water? (R17)r3	10^{-4}	0.01	0.1
Infect suscept. host when present? (R18)r3	10^{-5}	10^{-4}	0.001
Infection introduced into sea water (W3)r3 = (DxR12xP4xP5xR17xR18xSp)r3			
WRAPPING INTRODUCE INFECTION REGION 3/TONNE (I2)r3 = (W1 + W2 + W3)r3			

5.12.1 The evidence

The same arguments as used in 5.10.1 apply here.

5.12.2 Simulation results

Figure 5.15 is the graph showing the probability of wrappings serving to introduce *A. salmonicida* into salmonid fish stocks in Region 3.

Expected/mean result	=	3.2×10^{-9}
Maximum result	=	6.2×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	7.8×10^{-9}
95%	1.2×10^{-8}
99%	2.0×10^{-8}

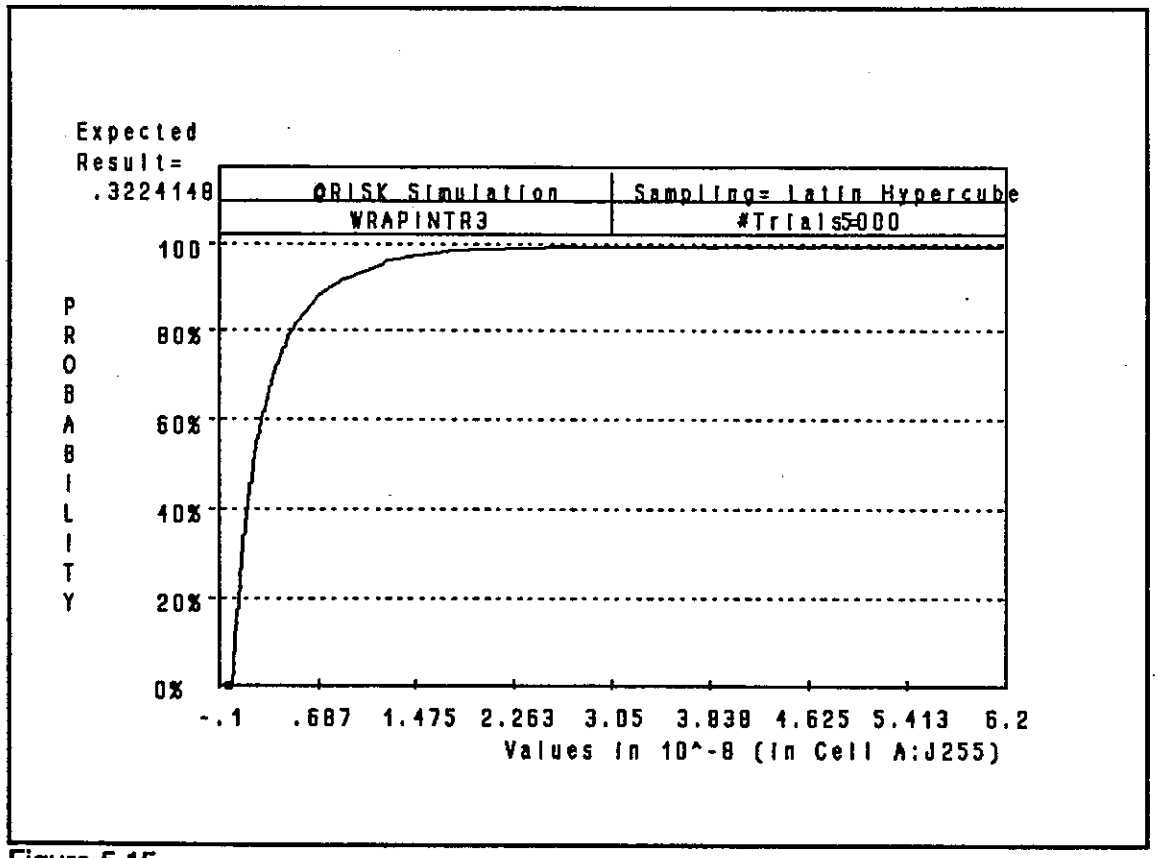


Figure 5.15

It can be seen that there is a 95% probability that the risk of wrappings introducing infection into Region 3 is less than 1.2 disease introductions per 100 million tonnes of **chilled**, headless, eviscerated Pacific salmon imported into New Zealand.

5.13 Cumulative risk of wrapping introducing infection

The cumulative national risk of *A. salmonicida* being introduced into New Zealand via the vehicle of contaminated wrappings is the sum of the regional risks. That is;

$$(I2)r1 + (I2)r2 + (I2)r3$$

This cumulative national risk is shown in Figure 5.16.

Expected/mean result	=	6.1×10^{-9}
Maximum result	=	8.3×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.4×10^{-8}
95%	1.9×10^{-8}
99%	3.4×10^{-8}

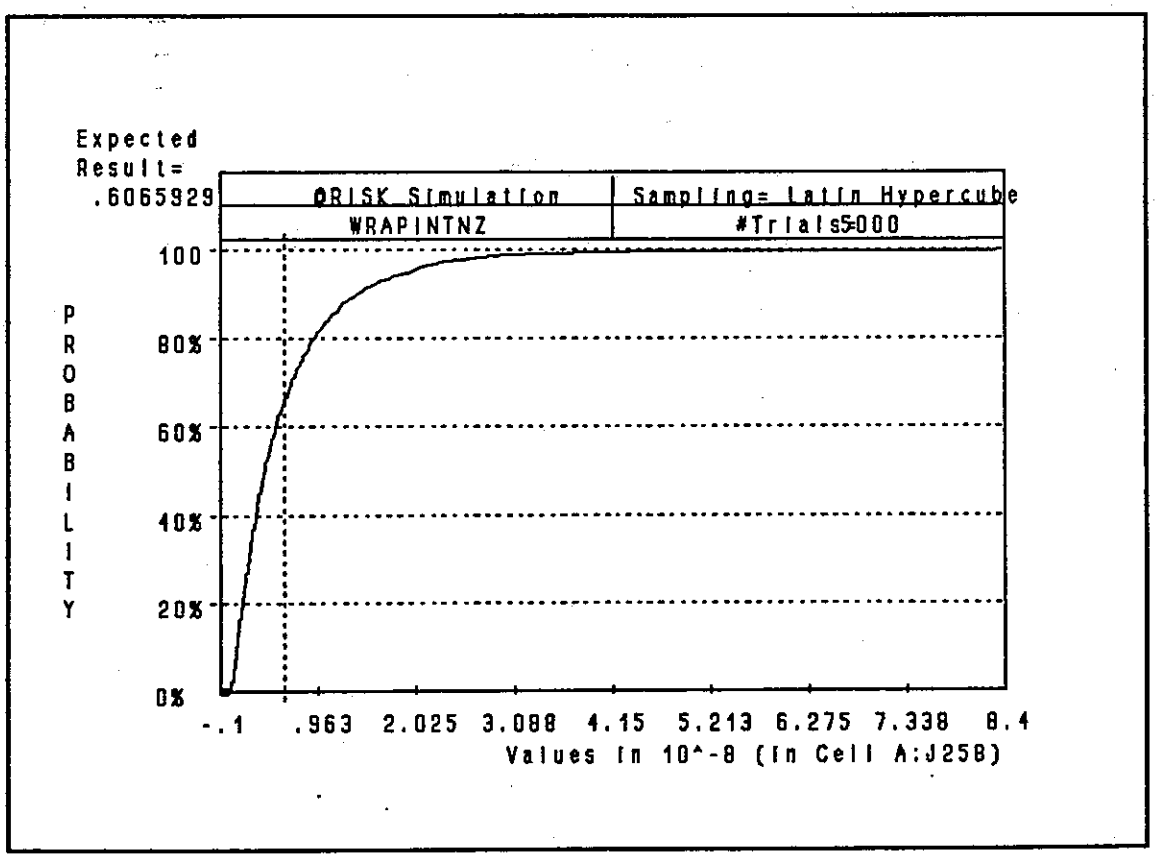


Figure 5.16

This simulation shows that, under the assumptions outlined in the previous sections, there is a 95% probability that the risk of introducing *A. salmonicida* through the medium of contaminated wrapping is less than 2 disease introductions per 100 million tonnes of **chilled**, headless, eviscerated ocean-caught Pacific salmon imported into New Zealand.

5.14 The probability that waste water will be the vehicle by which infection is introduced

Kitchen waste water is seen to be a route by which *A. salmonicida* could find its way into waterways and infect a susceptible host. The extent to which kitchen waste water (commercial or domestic) is able to contaminate waterways is strongly affected by the extent to which sewage is treated adequately. The extent to which sewage is not treated adequately varies between regions (Table 5.11).

Table 5.11: Proportion of sewage not treated adequately in New Zealand.

	Minimum	Most Likely	Maximum
Proportion of sewage NOT treated adequately in Region 1 (SW)r1	20.0%	75.0%	80.0%
in Region 2 (SW)r2	75.0%	89.0%	90.0%
in Region 3 (SW)r3	30.0%	89.0%	90.0%

Similarly, the extent to which sewage is discharged into fresh, estuarine or sea water affects the probability of contaminated outflow introducing infection into salmonid stocks. The extent to which sewage is discharged into each type of water varies between regions.

Table 5.13: Input variables used to calculate probability of waste water introducing infection into Region 1.

	Minimum	Most Likely	Maximum
If fish contaminated, probability that kitchen waste water contam? (R19)r1	0.1	0.2	0.5
Waste water contamination in Region 1 (DxR19)r1			
Sewage NOT treated adequately (SW)r1			
Proportion discharging into fresh water (DF)r1			
Contaminate fresh water? (SWxDF)r1			
Proportion of fishery in Region 1 (Sp)r1			
Infect suscept. host when present? (R22)r1	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infect suscept. host in Region 1 (SpxR22)r1			
Infection introduced into fresh water (WW1)r1 = (DxR19xSWxDFxR22xSp)r1			
Contaminate estuarine water? (SWxDE)r1			
Infect suscept. host when present? (R24)r1	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infection introduced into estuarine water (WW2)r1 = (DxR19xSWxDExR24xSp)r1			
Contaminate sea water? (SWxDS)r1			
Infect suscept. host when present? (R26)r1	1×10^{-7}	5×10^{-7}	1×10^{-6}
Infection introduced into sea water (WW3)r1 = (DxR19xSWxDSxR26xSp)r1			
WASTE WATER INTRODUCE INFECTION, REGION 1/TONNE (I3)r1 = (WW1 + WW2 + WW3)r1			

5.15.1 The evidence

5.15.1.1 If fish contaminated probability that kitchen waste water is contaminated? (R19)

It has been argued[123] that the risk of contaminated fish contaminating kitchen waste water is quite high. However, this argument is based on the very high bacterial contents of the furuncles found in the flesh of **Atlantic** salmon clinically affected with furunculosis. As has already been outlined (Section 3.8, Tables 3.4 and 3.5), the numbers of *A. salmonicida* found in the tissues of **Pacific** salmon actually dying of furunculosis are much lower than the 10^{10} cfu/ml cited by Anderson.

Further, as has already been pointed out (Section 3.8), clinical furunculosis has never been detected in wild, ocean-caught Pacific salmon and, although a small percentage (perhaps as many as 6%) of these fish might be subclinical carriers of *A. salmonicida*, the bacterium is sequestered in those viscera which are removed during processing. If any *A. salmonicida* are present in the flesh they can only be present in very low numbers. For example, the number of viable *A. salmonicida* reported in kidney tissue of carrier chum and pink salmon averages $10^{3.7}$ cfu/g (range $10^{1.7}$ to $10^{5.4}$ cfu/g).[124]

Studies outlined in Section 3.8 and shown in Table 3.4 supports the contention that if *A. salmonicida* were present in the flesh of carrier Pacific salmon it would be in numbers about $10^{3.5}$ times lower than in kidney tissue; that is, as few as 0.01 to 79.4 cfu/g, and certainly no more than a few tens to a few hundreds per gram.

Given the lability of *A. salmonicida* in the face of temperatures above 37 °C and in the presence of competing bacteria, the likelihood of waste water becoming contaminated cannot be very high.

However, some 18% of New Zealand households use a sink waste unit to dispose of **fruit** wastes[125]and it is probable that these households would also dispose of any

---

[123] Anderson, C D. The risk of introducing exotic disease with imported fish flesh. *Surveillance 19(1),* 12 - 13, 1992.

Anderson, C D, Veterinary Investigation Officer, MAF Quality Management, personal communication with Sue Cotton and Stuart MacDiarmid, 1992-94.

[124] Nomura T, Yoshimizu M, Kimura T. Prevalence of *Aeromonas salmonicida* in the chum salmon (Oncorhynchus keta), pink salmon (*O. gorbuscha*), and masu salmon (*O. masou*). Gyobyo Kenku 26, 139-147, 1991. [In Japanese, summary in English].

[125] Viggers, Elizabeth. The Potential risk of Introducing Exotic Pests to New Zealand through Domestic Disposal of Waste Plant Material. Project BH 004. MAF Quality Management. 12 pages. June 1993.

salmon scraps (cooked or uncooked) by this method. Even in households without sink waste units there is a likelihood that some salmon scraps would enter kitchen waste water, and some *A. salmonicida* cells which might be washed free of tissues, food preparation surfaces and kitchen utensils.

5.15.1.2 Infect susceptible host when present? (R22), (R24) and (R26)

Even were *A. salmonicida* to be liberated into a kitchen sink, very few of the bacteria would survive their passage from the kitchen through the sewage system and into a body of water containing fish. The extent to which bacteria would be inactivated by hot water in the immediate outflow of the sink cannot be guessed at, but the extent to which the infective material is likely to be diluted can be estimated.

As outlined in Section 5.5.1.3, if the volume of Pacific salmon imported per year were 200 tonnes (an improbably high estimate), this would be sufficient to provide around 4,600 households per week with around 0.87 kg of imported product each. This equates to 657 households per day, on average, consuming imported Pacific salmon. However, to err on the side of conservatism, let us assume that, say, 700 households use the 0.87 kg of imported salmon **on the same day** (that is, the weekly consumption all occurs in one day).

If each household disposed of 17% of this imported salmon **uncooked** as kitchen scraps (see Section 5.5.1.1 above), and if an improbably high 50% disposed of this via kitchen waste water (either through a sink waste unit or by some other means of maceration and flushing), this would lead to as much as [700x0.87x0.17x0.5=] 51.76 kg uncooked imported salmon being disposed of into waste water on one day.

As the proportion of wild, ocean-caught Pacific salmon carrying *A. salmonicida* is unlikely to exceed 6% (see Section 3.8 for discussion), the proportion of the uncooked **and contaminated** salmon scraps disposed of into waste water is unlikely to exceed this figure. However, to ensure that a conservative estimate of risk is obtained, let us assume that 10% of what is disposed of is contaminated. That is, on the day in question, the one day in the week when 700 households eat imported salmon, 5.17 kg of contaminated salmon is disposed of into waste water.

It has been explained already that clinical furunculosis has not been reported in wild, ocean-caught Pacific salmon. However, if one assumes that somehow tissue from Pacific salmon dying from furunculosis escapes Canadian inspection and grading procedures is imported (an improbable and unsustainable proposition) the flesh could be expected to contain, on average, 7.3×10^4 *A. salmonicida* per gram. If **all** the tissue flushed into the waste water carried this bacterial load (unlikely!), then on the day in question [$7.3 \times 10^4 \times 5.17 \times 10^3 =$] 3.77×10^8 *A. salmonicida* would be released into the household waste water.

A. salmonicida is relatively heat sensitive (see Section 3.8, for instance) being inactivated rather rapidly at temperatures of 37 °C and over. While temperatures much higher than this are commonly achieved in kitchen waste water for brief

periods, the effect of temperature inactivation of the pathogen can be ignored at this stage of the deliberations. Thus, ignoring any inactivation of *A. salmonicida* which may occur as a result of washing the sink and utensils in hot water, these $3.77x10^8$ bacteria will be diluted into the daily water usage from the affected households. One source consulted reports that each person in New Zealand generates, on average, 185 litres of sewage per day.[126] The average household comprises 2.9 people so a household could be expected to generate around 550 litres of sewage per day. This is similar to the reported domestic water usage of $5x10^5$ ml water per household per day[127]. Using these figures for the volume of sewage generated, the 700 households releasing $3.77x10^8$ *A. salmonicida* into their waste water on the day in question would dilute these to a concentration of [$3.77x10^8/700x5x10^5$=] 1.08 bacteria/ml, **even if all 700 households discharged their waste water into the same sewage system!** The proposition that the entire national weekly consumption of imported salmon occurs on the same day, in the same city, is stretching credibility and so it will be assumed that the households consuming the salmon are distributed around the country.

However, for the 700 households discharging contaminated waste water, around $1.1837x10^6$ will be discharging uncontaminated water. Further, domestic water consumption is matched nationally by a comparable industrial consumption, although this varies between localities. For instance, only 20% of the sewage discharged into the sea from the Hutt Valley is derived from industrial sources.[128] Nevertheless, the domestic waste water is likely to be further diluted by industrial waste water. Even if *A. salmonicida* were to escape inactivation by sewage treatment and competition with other bacteria, it will be further enormously diluted (that is, by several orders of magnitude) when discharged from the sewage system into a body of water (major river, estuary or sea).

The inability of *A. salmonicida* to survive or multiply in the environment has been discussed in Section 3.8. The literature on this topic is voluminous but can be faithfully rendered down to a few simple statements. First, in both fresh and sea water, the organism will survive for short periods if there are no competing bacteria and no nutrients present. Second, if nutrients but no competing bacteria are present, survival will be enhanced and there may even be growth, the amount of growth

---

[126] Centre for Advanced Engineering. Our Waste: Our Responsibility. Towards Sustainable Waste Management in New Zealand. Project Report. University of Canterbury. 467 pages. 1992.

[127] Department of Statistics. *New Zealand Official Yearbook.* 95th edition. Wellington,1992.

[128] Steven, Fitzmaurice and Partners. Disposal of Hutt Valley Wastewaters. Report submitted to the Hutt Valley Drainage Board. April Steven, Fitzmaurice and Partners, Christchurch, New Zealand, 1981. Cited by Anderlini, VC. The effect of sewage on trace metal concentrations and scope for growth in *Mytilus edulis aoteanus* and *Perna canaliculus* from Wellington Harbour, New Zealand. The Science of the Total Environment 124, 263-288, 1992.

depending on the amount of nutrient provided. Third, when both nutrients and competing bacteria are present, survival of the pathogen is drastically reduced compared to that occurring when competing bacteria are absent. Fourth, the presence of particulate material (eg. sand, mud, wood, etc) in the system helps to enhance survival but when such systems also contain competing bacteria, culturable *A. salmonicida* cells always show drastic declines.

A theory advanced by Austin and his group[129] is that *A. salmonicida* converts to a non-culturable form when held in aqueous systems. This is a view not shared by many other researchers who work with *A. salmonicida*.[130] However, even if Austin's results could be confirmed by others, it turns out that when the countable (by culture) bacteria decline to the stage where they are no longer detectable by culture on routine media (eg. tryptic soy agar), the sample becomes non-infectious for *A. salmonicida*-susceptible fish **(even when injected)**. Thus, even if Austin's peculiar forms of *A. salmonicida* do exist, they are incapable of establishing infections in susceptible fish.

Multiplication and prolonged persistence of the pathogen outside of the host in the natural environment has never been demonstrated and seems highly unlikely in view of the laboratory findings. In fact, sediment samples from under a salmon farm with active furunculosis failed to show evidence of the viable pathogen even though a new culture technique capable of growing the bacterium in the face of contaminating bacteria was used.[131] Indeed, in the real world, the rather brief persistence of the bacterium in the environment sometimes permits salmon farmers to avoid the bacterium in fish hatcheries. This avoidance is achieved if circumstances permit the hatchery operators to exclude fish (the source of the bacterium) from the water supply.

---

[129] See, for example, the discussion in Austin, B, Austin DA, Bacterial Fish Pathogens. Disease in Farmed and Wild Fish, Second edition. Ellis Horwood, New York, 384 pages, 1993.

[130] Evelyn, TP, Head, Fish Health and Parasitology Section, Biological Sciences Branch, Pacific Biological Station, Nanaimo, British Columbia, personal communication with SC MacDiarmid, 25 August 1994.

Rose, AS, Ellis, AE, Munro, ALS. Evidence against dormancy in bacterial fish pathogen *Aeromonas salmonicida* subsp. *salmonicida*. FEMS Microbiology Letters 68, 105-108, 1990.

Rose, AS, Ellis, AE, Munro, ALS. The survival of *Aeromonas salmonicida* subsp. *salmonicida* in sea water. Journal of Fish Diseases 13, 205-214, 1990.

[131] Evelyn, TP, Head, Fish Health and Parasitology Section, Biological Sciences Branch, Pacific Biological Station, Nanaimo, British Columbia, personal communication with SC MacDiarmid, 25 August 1994.

Salmonids are unlikely to be found in close proximity to sewage outfalls. They do not favour environments highly enriched in organic matter.[132] The low oxygen content and high ammonia concentration[133] will be outside the range tolerated by salmonids, thus making sewage outfalls unattractive to these fish. Sewage effluent is toxic to fish and exerts a high oxygen demand directly and indirectly through the demand of settled organic matter.

A study in British Columbia[134] showed that salmon are not found within about 1,000 m of a sewage outfall. Fewer fish of other species are also found in the proximity of the outfall. The oxygen levels measured in the vicinity of the sewage outfall were such that marine and anadromous species, including salmonids, would be expected to experience oxygen distress.

So, even should all 700 houses disposing of an improbably-high proportion of contaminated imported salmon scraps all be discharging their sewage, untreated, at the same outfall, the concentration of *A. salmonicida* would not be greater than 1.08 bacteria/ml **at the immediate point of outfall.**

The dose of *A. salmonicida* required to infect salmon is relatively high. In Section 3.8 information has been presented to demonstrate failure to infect salmon by immersion in concentrations of 10^3 cells/ml *A. salmonicida* for 2-3 days or 10^2 cells/ml for 1 week. Clearly it would not be possible to attain or sustain concentrations of this magnitude at sewage outfalls, even with discharges of contaminated salmon material far in excess of what can realistically be expected.

As discussed in Section 3.8, it is possible to infect salmon by mouth with *A. salmonicida* but not readily. Doses in the order of $>10^5$ cfu/fish would be required. However, as has been discussed in a number of previous sections, doses of this magnitude are just not likely to be attained in tissues from the grade of wild, ocean-caught Pacific salmon being imported. Studies outlined in Section 3.8 and shown in Table 3.4 supports the contention that if *A. salmonicida* were present in the flesh of carrier Pacific salmon it would be in numbers around a few tens to a few

---

[132] Klontz, G W. Environmental requirements and environmental diseases of salmonids. In *Fish Medicine*. Stoskopf, M K, (Editor). W B Saunders. Philadelphia. Pp333-342. 1993.

[133] Mara, D, Cairncross, A. *Guidelines for the Safe Use of Wastewater and Excreta in Agriculture and Aquaculture*. World Health Organisation, Geneva. 187 pages. 1989.

[134] Birtwell, IK, Greer, GL, Nassichuk, MD, Rogers, IH. Studies on the impact of municipal sewage discharged onto an intertidal area within the Fraser river estuary, British Columbia. Canadian Technical Report of Fisheries and Aquatic Sciences No. 1170. Department of Fisheries and Oceans, Vancouver. 55 pages. 1983.

hundreds per gram, and these would have to escape destruction through competition with competitive environmental bacteria or following ingestion by non-susceptible invertebrates.

At the point where the water in the vicinity of the sewage outfall has become tolerable to salmonids the dilution will have ensured that the maximum risk of infecting a susceptible host (of any species) is virtually zero.

5.15.2 Simulation results

Figure 5.17 shows the probability of kitchen waste water acting as the route by which *A. salmonicida* is introduced into waterways in Region 1.

Expected/mean result	=	1.6×10^{-10}
Maximum result	=	2.0×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	3.6×10^{-10}
95%	4.9×10^{-10}
99%	8.9×10^{-10}

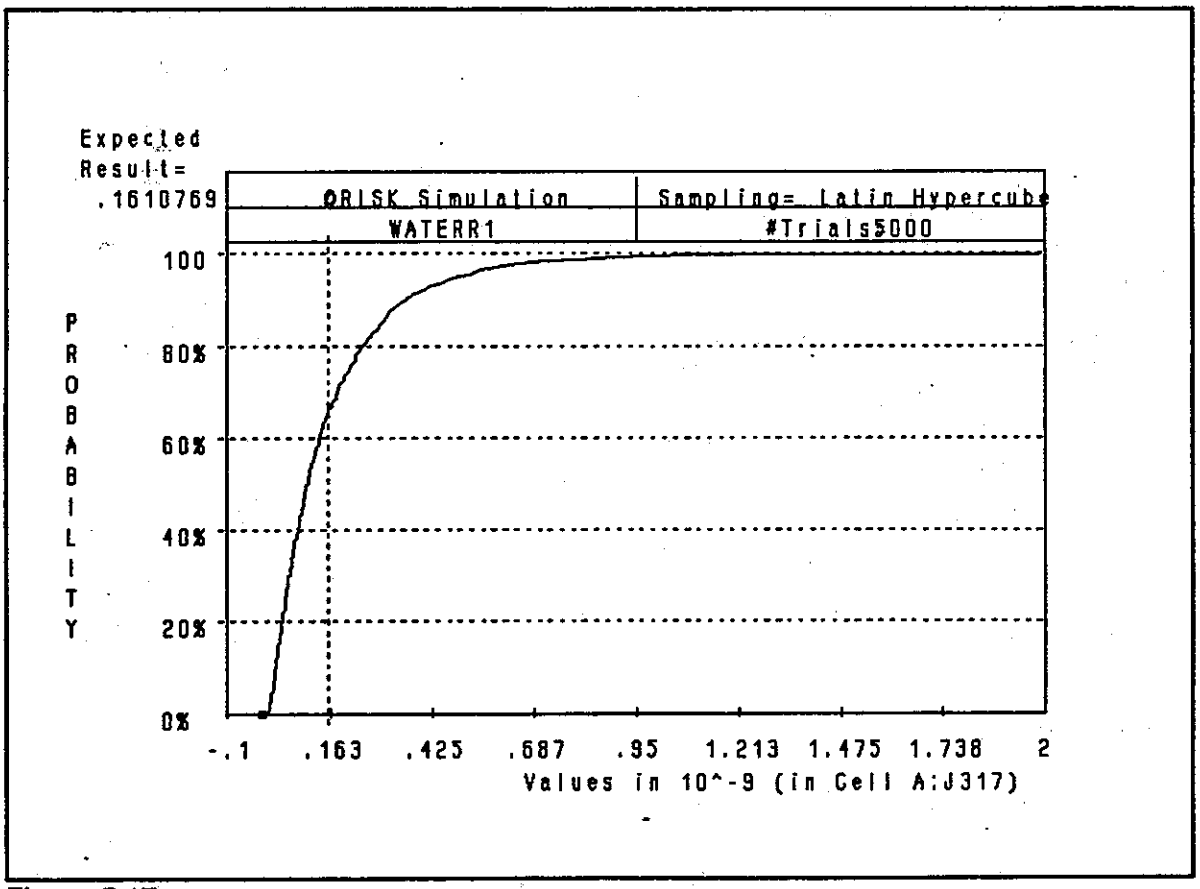


Figure 5.17

There is a 95% probability that the risk of waste water introducing infection into Region 1 is less than 5 disease introductions per 100 million tonnes imported into the country.

5.16 Region 2, waste water introduces infection

The number of contaminated fish imported into Region 2, (D)r2, has been given in Section 5.6.2. The other assumptions used in this simulation appear below.

Table 5.14: Input variables used to calculate probability of waste water introducing infection into Region 2.

	Minimum	Most Likely	Maximum
If fish contaminated, probability that kitchen waste water contam? (R19)r2	0.1	0.2	0.5
Waste water contamination in Region 2 (DxR19)r2			
Sewage NOT treated adequately (SW)r2			
Proportion discharging into fresh water (DF)r2			
Contaminate fresh water? (SWxDF)r2			
Proportion of fishery in Region 2 (Sp)r2			
Infect suscept. host when present? (R22)r2	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infect suscept. host in Region 2 (SpxR22)r2			
Infection introduced into fresh water (WW1)r2 = (DxR19xSWxDFxR22xSp)r2			
Contaminate estuarine water? (SWxDE)r2			
Infect suscept. host when present? (R24)r2	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infection introduced into estuarine water (WW2)r2 = (DxR19xSWxDExR24xSp)r2			
Contaminate sea water? (SWxDS)r2			
Infect suscept. host when present? (R26)r2	1×10^{-7}	5×10^{-7}	1×10^{-6}
Infection introduced into sea water (WW3)r2 = (DxR19xSWxDSxR26xSp)r2			
Waste water INTRODUCE INFECTION, REGION 2/TONNE (I3)r2 = (WW1 + WW2 + WW3)r2			

5.16.1 The evidence

Evidence outlined above in 5.15.1 applies equally here.

5.16.2 Simulation results

Figure 5.18 shows the probability of kitchen waste water acting as the route by which *A. salmonicida* is introduced into waterways in Region 2.

Expected/mean result	=	3.8×10^{-9}
Maximum result	=	5.3×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	8.5×10^{-9}
95%	1.2×10^{-8}
99%	1.9×10^{-8}

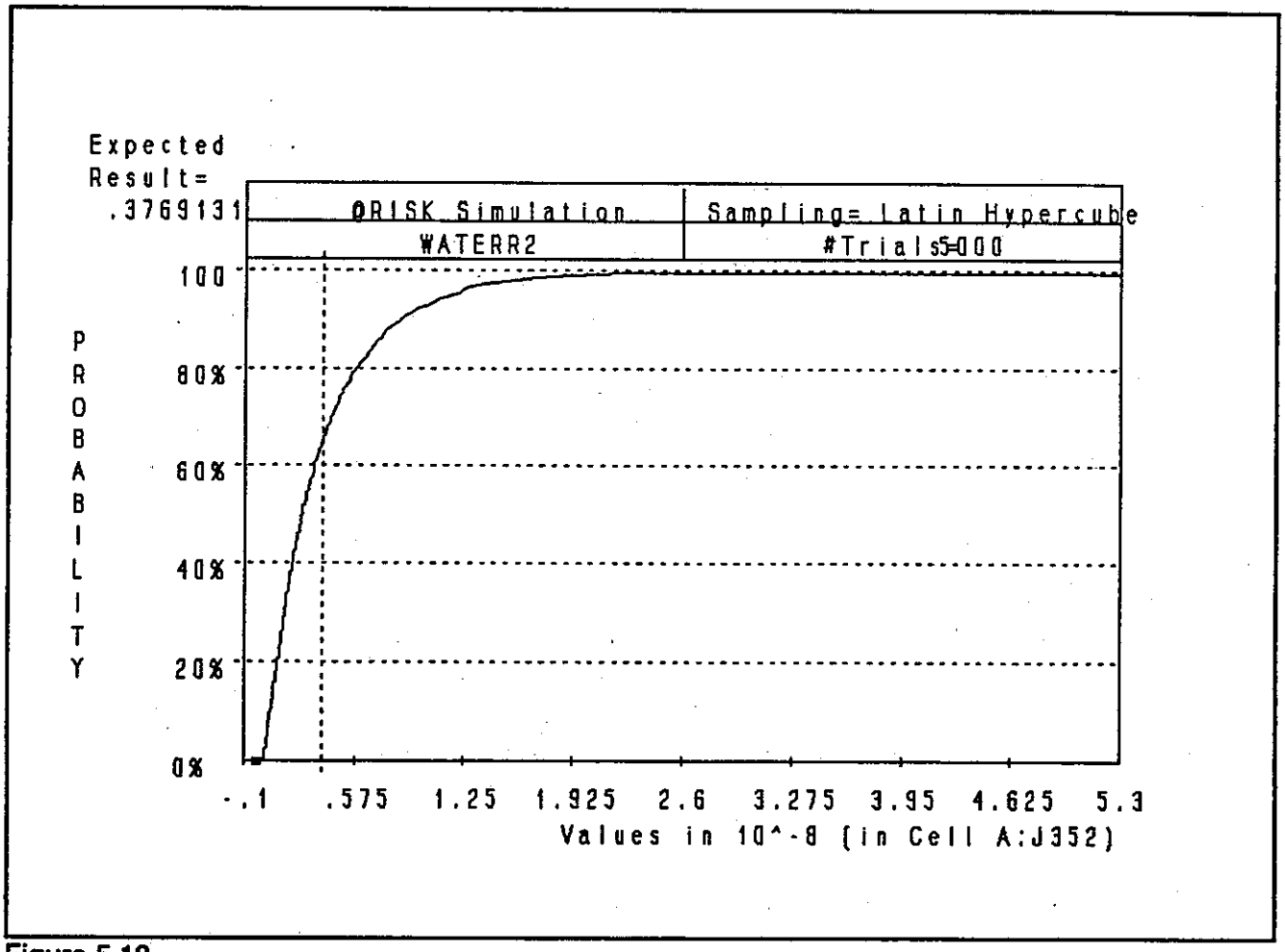


Figure 5.18

There is a 95% probability that the risk of waste water introducing infection into Region 2 is less than 1.2 disease introductions per 100 million tonne of ocean caught salmon imported into the country.

5.17 Region 3, waste water introduces infection

Section 5.7.2 outlines the probable number of contaminated fish imported into Region 3, (D)r3. Other assumptions which are used in this simulation are listed below.

Table 5.15: Input variables used to calculate probability of waste water introducing infection into Region 3.

	Minimum	Most Likely	Maximum
If fish contaminated probability that kitchen waste water contam? (R19)r3	0.1	0.2	0.5
Waste water contamination in Region 1 (DxR19)r3			
Sewage NOT treated adequately (SW)r3			
Proportion discharging into fresh water (DF)r3			
Contaminate fresh water? (SWxDF)r3			
Proportion of fishery in Region 3 (Sp)r3			
Infect suscept. host when present? (R22)r3	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infect suscept. host in Region 3 (SpxR22)r3			
Infection introduced into fresh water (WW1)r3 = (DxR19xSWxDFxR22xSp)r3			
Contaminate estuarine water? (SWxDE)r3			
Infect suscept. host when present? (R24)r3	1×10^{-7}	1×10^{-6}	5×10^{-6}
Infection introduced into estuarine water (WW2)r3 = (DxR19xSWxDExR24xSp)r3			
Contaminate sea water? (SWxDS)r3			
Infect suscept. host when present? (R26)r3	1×10^{-7}	5×10^{-7}	1×10^{-6}
Infection introduced into sea water (WW3)r3 = (DxR19xSWxDSxR26xSp)r3			
WASTE WATER INTRODUCE INFECTION, REGION 3/TONNE (I3)r3 = (WW1 + WW2 + WW3)r3			

5.17.1 The evidence

The evidence discussed in section 5.15.1 applies here also.

5.17.2 Simulation results

Figure 5.19 shows the probability of kitchen waste water acting as the route by which *A. salmonicida* is introduced into waterways in Region 3.

Expected/mean result	=	7.9×10^{-9}
Maximum result	=	1.0×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.7×10^{-8}
95%	2.3×10^{-8}
99%	4.1×10^{-8}

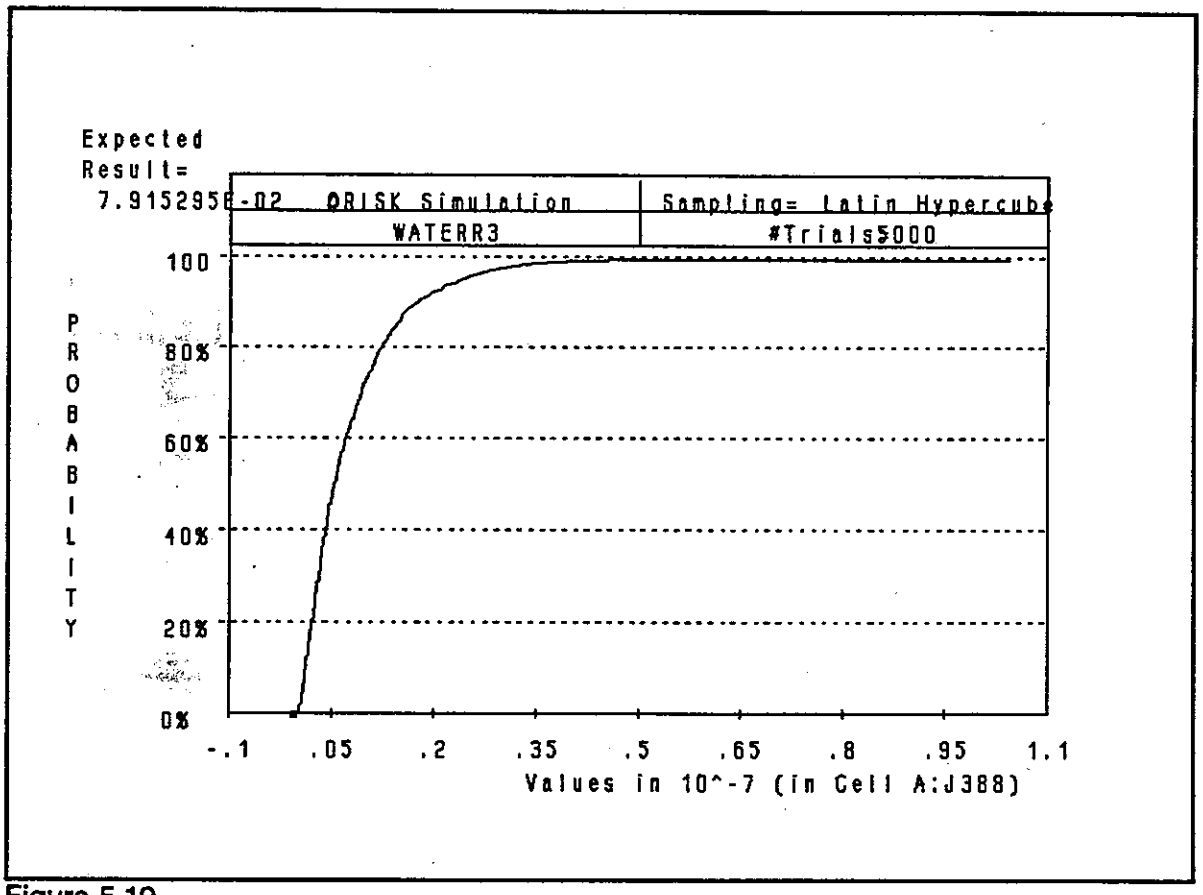


Figure 5.19

There is a 95% probability that the risk of waste water introducing infection into Region 3 is less than 2.3 disease introductions per 100 million tonnes imported into the country.

5.18 Cumulative risk of waste water introducing infection

The three regional risks of waste water introducing infection are summed to give the national risk. The combined risk from waste water is;

$$(I3)r1 + (I3)r2 + (I3)r3$$

This cumulative national risk is shown in Figure 5.20

Expected/mean result	=	1.2×10^{-8}
Maximum result	=	1.3×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	2.6×10^{-8}
95%	3.4×10^{-8}
99%	5.4×10^{-8}

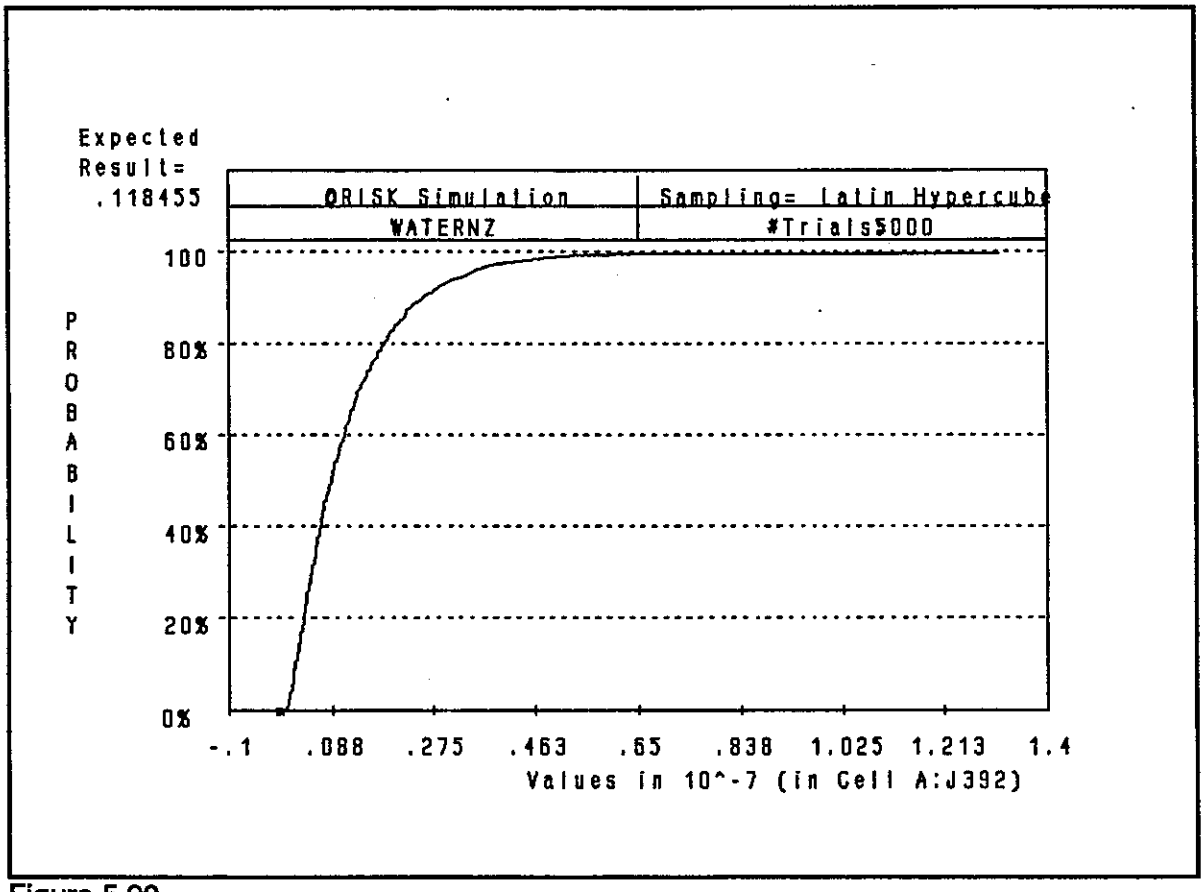


Figure 5.20

Under the assumptions used in the present simulation, there is a 95% probability that the risk of waste water serving as the vehicle by which *A. salmonicida* is introduced into New Zealand salmonid stocks is less than 3.4 disease introductions per 100 million tonnes of **chilled**, headless, eviscerated ocean-caught Pacific salmon imported.

5.19 The combined probability of infection being introduced through chilled headless, eviscerated Pacific salmon

The final estimate of risk/tonne is the sum of the individual risks of infection being introduced into each region via scraps, wrappings and waste water. The cumulative risk per tonne of imported chilled, eviscerated, headless, ocean-caught Pacific salmon is;

$$(I1 + I2 + I3)$$

The cumulative probability graph which combines the three regional risks of the three potential routes by which infection could enter local fish stocks (through scraps, through contaminated wrappings or through kitchen waste water) is shown in Figure 5.2.1.

Expected/mean result	=	2.5×10^{-8}
Maximum result	=	2.3×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	5.3×10^{-8}
95%	6.8×10^{-8}
99%	1.1×10^{-7}

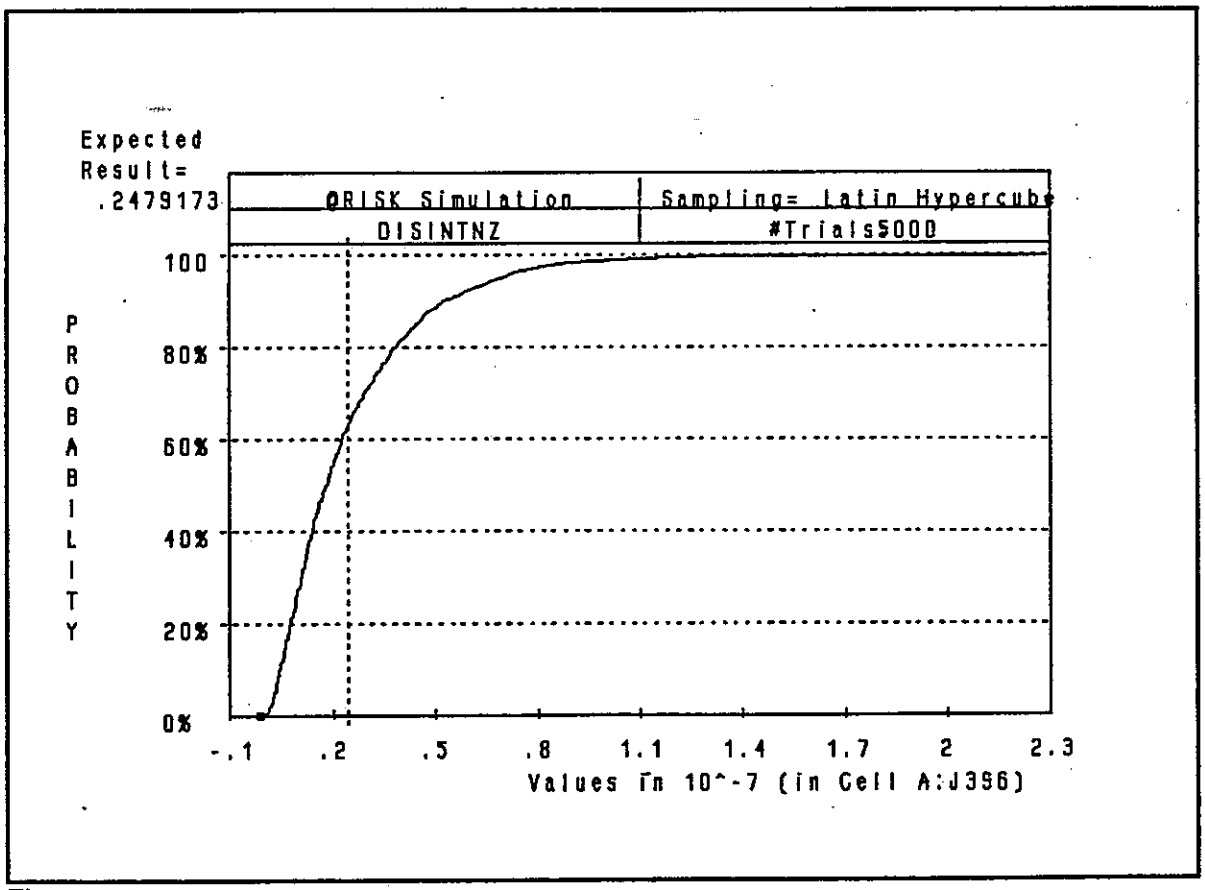


Figure 5.21

Thus, when the individual risks for each route of introduction in each region are combined, the results of the simulation show that, under the assumptions outlined, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand's salmonid fisheries is less than 1 disease introduction [0.68] per 10 million tonnes of chilled, headless, eviscerated, ocean-caught Pacific salmon imported.

To put this risk into perspective, one should note that the entire annual production of ocean-caught salmon in British Columbia is between 80,000 and 100,000 tonnes.[135] That is, the disease risk posed by importing chilled, headless, eviscerated, wild, ocean-caught Pacific salmon is negligible.

[135] Canada-Australia Salmon Technical Meeting Under Auspices of GATT Article XXII, Nanaimo, British Columbia, July 25-26 1994.

6. Frozen headless, eviscerated fish

In the previous section the risks posed by **chilled** headless, eviscerated wild, ocean-caught Pacific salmon have been assessed. This section assesses the effect of a further risk reducing measure (**freezing**) on the estimated risk of introducing *A. salmonicida*.

6.1 The probability of importing contaminated, potentially infective fish

When the commodity is frozen, the risk of importing contaminated, potentially infective fish flesh into the country is less than when chilled product is imported. The range of estimates the calculation uses to assess the probability of importing contaminated fish are as follows;

Table 6.1: Input variables used to calculate probability of importing contaminated fish flesh. Frozen, headless, eviscerated salmon.

	Minimum	Most Likely	Maximum
Proportion of fish diseased/year? (P)	0.0*	0.02	0.06
Evisceration reduces risk by;	0.8*	0.95	0.99
to; (R2)	1 - 0.8	1 - 0.95	1 - 0.99
Inspection and grading reduces risk by;	0.1*	0.5	0.7
to; (R3)	1 - 0.1	1 - 0.5	1 - 0.7
Washing reduces risk by;	0	0.05	0.3
to; (R4)	1 - 0	1 - 0.05	1 - 0.3
Freezing reduces risk by;	0.9	0.95	0.99
to; (R5)	1 - 0.9	1 - 0.95	1 - 0.99
Mean weight of ocean caught salmon? (K)	1.5	2.5	5.0
Proportion remaining after evisceration (F)	0.65	0.71	0.73
No. fish represented/tonne (N) = 1000/FxK			
No. *diseased* fish/tonne = PxN			
DISEASED FISH *IMPORTED*/TONNE (D) = PxNxR1xR2xR3xR4xR5			

* Note: As in Table 5.1 P, R3 and R2 are linked so that an increase in P tends to lead to an increase in R3 but a decrease in R2.

6.1.1 The evidence

All evidence supporting the choice of input variables used in this section is the same as for Section 5.1.1, with the exception of that relating to the effect of freezing on *A. salmonicida*.

6.1.1.1 The extent to which freezing reduces the risk (R5)

In Section 3.8 studies were described which demonstrated the susceptibility of *A. salmonicida* to freezing. These studies demonstrated that a single freeze/thaw cycle will result in a two log decrease (100-fold decrease) in the number of viable bacteria in organs of infected fish (Table 3.5). This means that with a frozen product, the number of viable *A. salmonicida* present in the flesh would be reduced by an additional two orders of magnitude, thus further reducing any hypothetical threat posed by the product. The bacterial numbers present in the frozen and thawed flesh of carrier Pacific salmon are thus likely to be zero or close to zero. Certainly, the numbers present would be many orders of magnitude less (perhaps 10^9 times less) than the number (10^{10} cfu/g) seen in the necrotic furuncular material from active cases of the disease in Atlantic salmon and cited[136] as being likely to be present in the flesh of imported salmon.

6.1.2 Simulation results

6.1.2.1 The number of fish represented/tonne of headless, eviscerated salmon

The number of fish represented per tonne of headless, eviscerated salmon is, of course, unchanged by freezing. The estimate given in Section 5.1.2.1 are applicable here.

6.1.2.2 The number of *diseased* fish represented/tonne

The number of *diseased* fish represented per tonne of headless, eviscerated salmon is not affected by freezing and so estimates given previously in Section 5.1.2.2 are again applicable here.

---

[136] Anderson, CD. The risk of introducing exotic fish disease with imported fish flesh. Surveillance 19 (1), 12-13, 1992.

6.1.2.3 The probability of *importing* contaminated fish, with freezing

Figure 6.1 shows the probable number of contaminated, potentially infective fish represented in each tonne imported. The next graph (Figure 6.2) shows the *cumulative* probability of a given number of contaminated fish contributing to each tonne imported. It can be seen that the number of contaminated fish contributing to each tonne imported is;

Expected/mean result	=	0.03
Maximum result	=	0.3
Minimum result	=	5.8×10^{-6}

Probability;	That the number of fish is less than;
90%	0.07
95%	0.10
99%	0.14

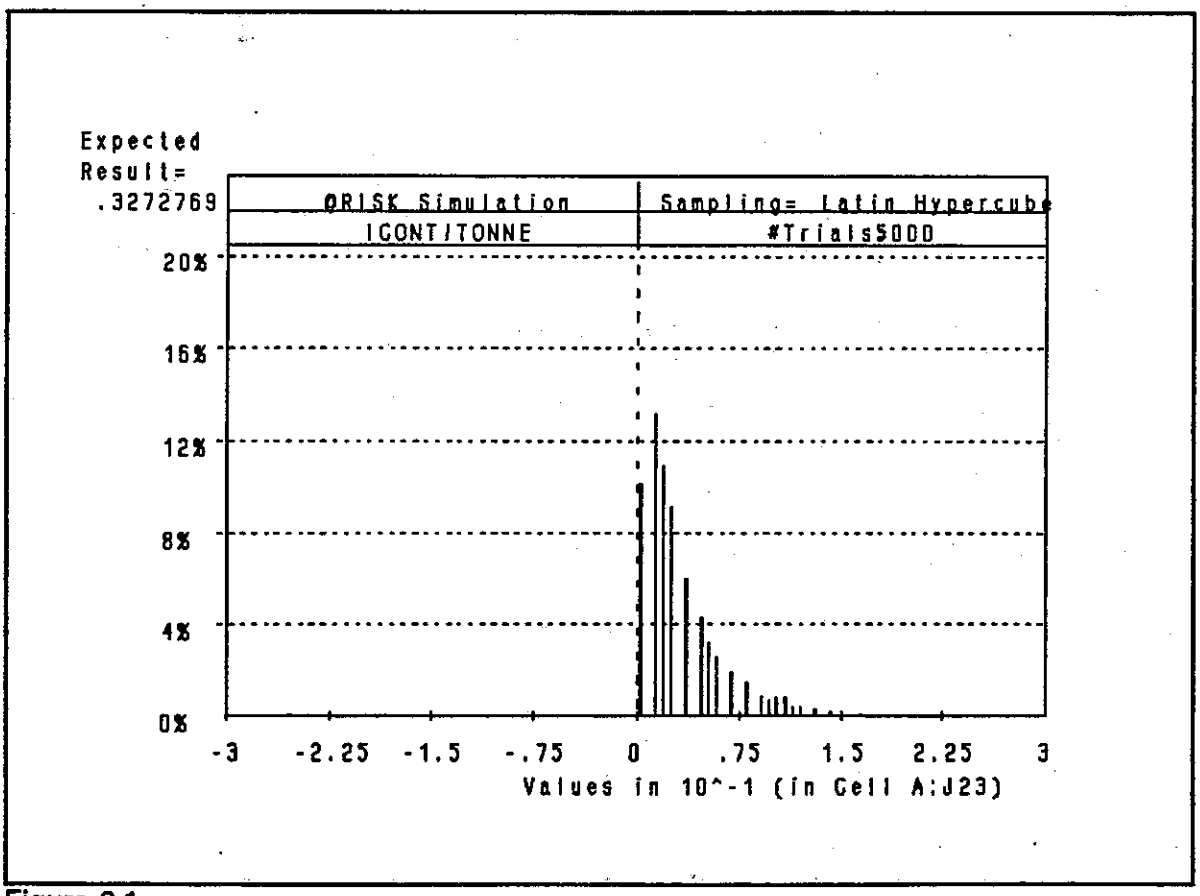


Figure 6.1

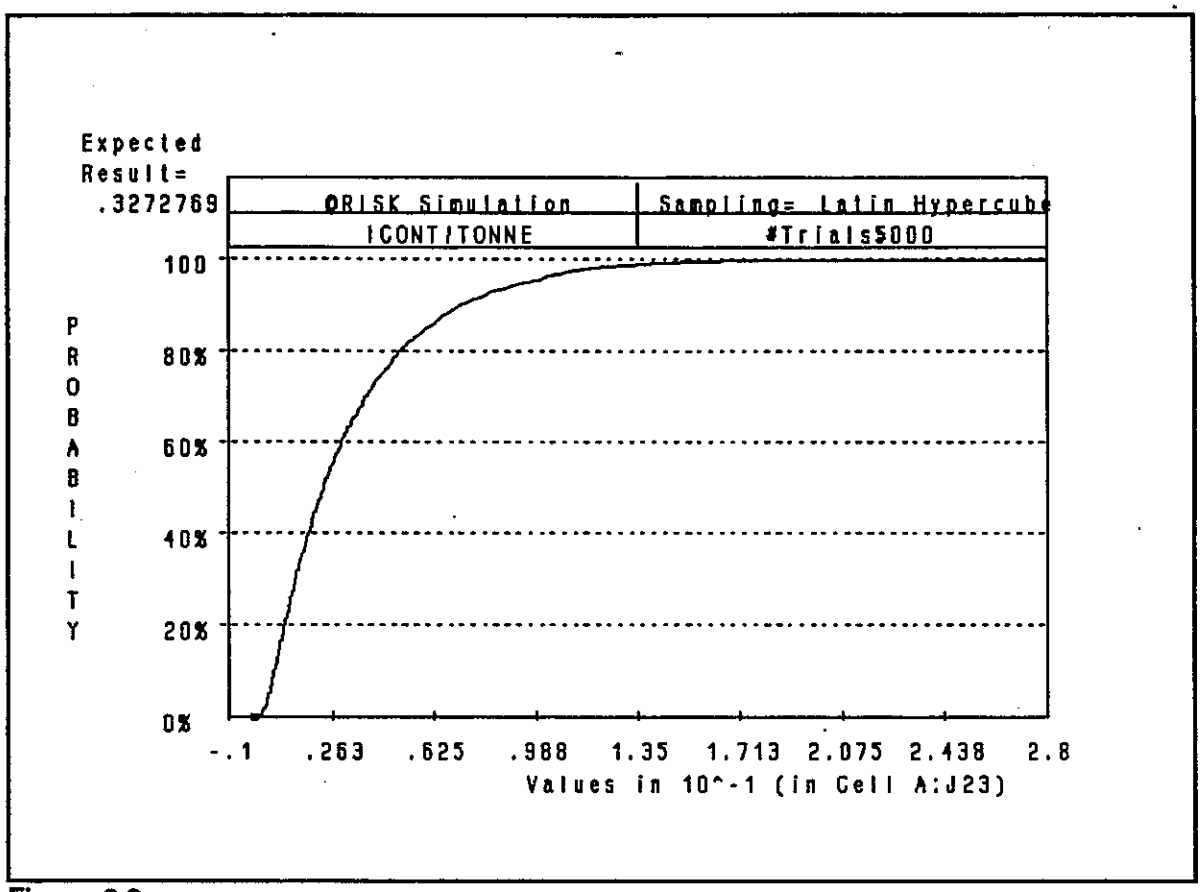


Figure 6.2

That is, there is a 95% probability that the number of contaminated, potentially infective fish contributing to each tonne imported is fewer than 1 per 10 tonnes. This figure takes into account the extent to which the risk has been reduced by evisceration, inspection and grading, washing **and freezing** of headless, eviscerated Pacific salmon. With the assumptions used in the model, it can be seen that each tonne imported into New Zealand is likely to contain flesh contributed by 0.03 fish which are, theoretically, capable of introducing disease. However, there are many steps between importation of contaminated flesh and the introduction of disease, as the following sections illustrate.

6.2 Distribution of imported fish within the country

The assumptions and input variables are the same as used already in Section 5.2 above.

6.3 The distribution of salmonid fish stocks within the country

The assumptions and input variables are the same as used already in Section 5.3 above.

6.4 The probability that scraps will introduce infection

The effect of freezing on *A. salmonicida* affects the input variables used for the probability that scraps will introduce infection. The input variables which are different from those already given in Tables 5.6, 5.7 and 5.8 are shown in Table 6.2 below.

Table 6.2: Input variables affected by freezing of commodity when considering scraps as the vehicle by which disease introduced.

	Minimum	Most Likely	Maximum
Fresh water, infect suscept. host when present? (R7) r1, r2 and r3	1×10^{-6}	5×10^{-5}	0.001
Estuarine water, infect suscept. host when present? (R9) r1, r2 and r3	1×10^{-6}	5×10^{-5}	0.001
Sea water, infect suscept. host when present? (R10) r1, r2 and r3	1×10^{-6}	5×10^{-5}	0.001

The values shown in the above Table 6.2 were substituted in the @RISK model for those used when chilled commodity was being considered.

6.4.1 The evidence

The evidence presented in Section 5.15.1 applies here but is influenced by the fact that a single freeze/thaw cycle reduces the numbers of *A. salmonicida* by one to two orders of magnitude (Table 3.5).

6.5 Simulation results; cumulative risk of scraps introducing infection

As the method has been outlined already (Section 5) only the cumulative risk of scraps introducing infection following the importation of **frozen**, headless, eviscerated ocean-caught Pacific salmon are presented here.

This cumulative risk is shown graphically in Figure 6.3.

Expected/mean result	=	0.4×10^{-10}
Maximum result	=	7.7×10^{-10}
Minimum result	=	0

Probability;	That risk is less than;
90%	0.9×10^{-10}
95%	1.4×10^{-10}
99%	2.6×10^{-10}

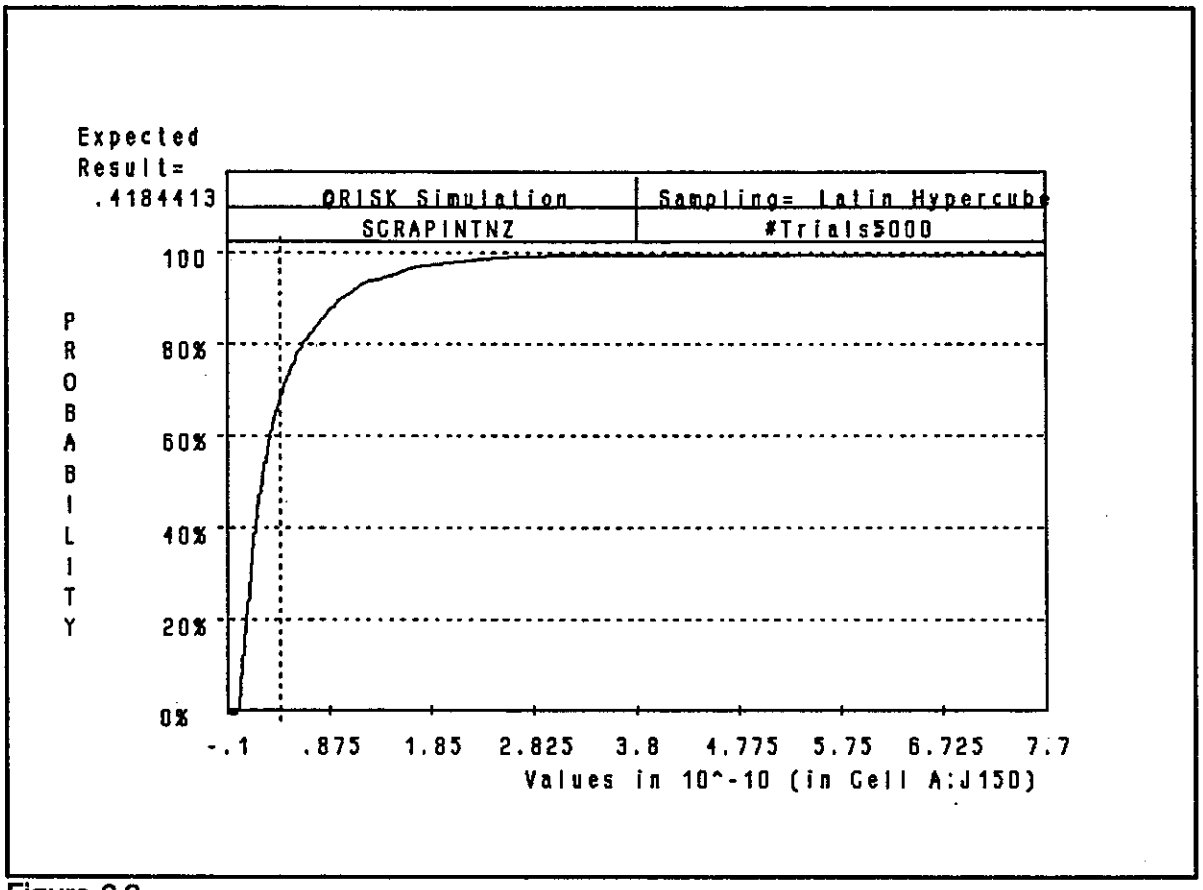


Figure 6.3

This simulation shows that, under the assumptions outlined above, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand through the medium of scraps is less than 1.4 disease introductions per 10^{10} tonnes of **frozen,** headless, eviscerated ocean-caught Pacific salmon imported.

6.6 The probability that wrapping will introduce infection

The probability that wrappings or packing material will introduce *A. salmonicida* is also influenced by freezing. The input variables considered to be affected by freezing of the commodity are shown below in Table 6.3. All other variables remain as outlined previously in Section 5.9.

Table 6.3: Input variables affected by freezing when considering wrappings as the vehicle by which disease introduced.

	Minimum	Most Likely	Maximum
If contaminated fish, probability that wrapping contaminated? (R12) r1, r2 and r3	0.1	0.2	0.3
Fresh water, infect suscept. host when present? (R14) r1, r2 and r3	1×10^{-6}	5×10^{-6}	1×10^{-4}
Estuarine water, infect suscept. host when present? (R16) r1, r2 and r3	1×10^{-6}	5×10^{-6}	1×10^{-4}
Sea water, infect suscept. host when present? (R18) r1, r2 and r3	1×10^{-6}	5×10^{-6}	1×10^{-4}

6.6.1 The evidence

Freezing and thawing significantly reduces the numbers of viable *A. salmonicida* present in a medium (Table 3.5). This reduction in bacterial numbers will reduce, but not eliminate, the risks of wrapping becoming contaminated. However, the reduction in numbers of the pathogen which could be present on wrappers will mean a concomitant reduction in likelihood of introducing infection to any susceptible hosts in any body of water into which wrappers may be washed or blown.

6.7 Simulation results; cumulative risk of wrapping introducing infection

Once the different input variables based on frozen commodity were placed into the simulation model, it was run as described in Section 5.

Only the cumulative risk is given here, although every step carried out in the simulation for chilled salmon was also carried out for the frozen commodity.

The cumulative national risk of *A. salmonicida* being introduced into New Zealand via the vehicle of contaminated wrappings from frozen, headless, eviscerated Pacific salmon is the sum of the regional risks. This cumulative national risk is shown in Figure 6.4.

Expected/mean result	=	0.2×10^{-10}
Maximum result	=	2.9×10^{-10}
Minimum result	=	0

Probability;	That risk is less than;
90%	0.4×10^{-10}
95%	0.6×10^{-10}
99%	0.1×10^{-10}

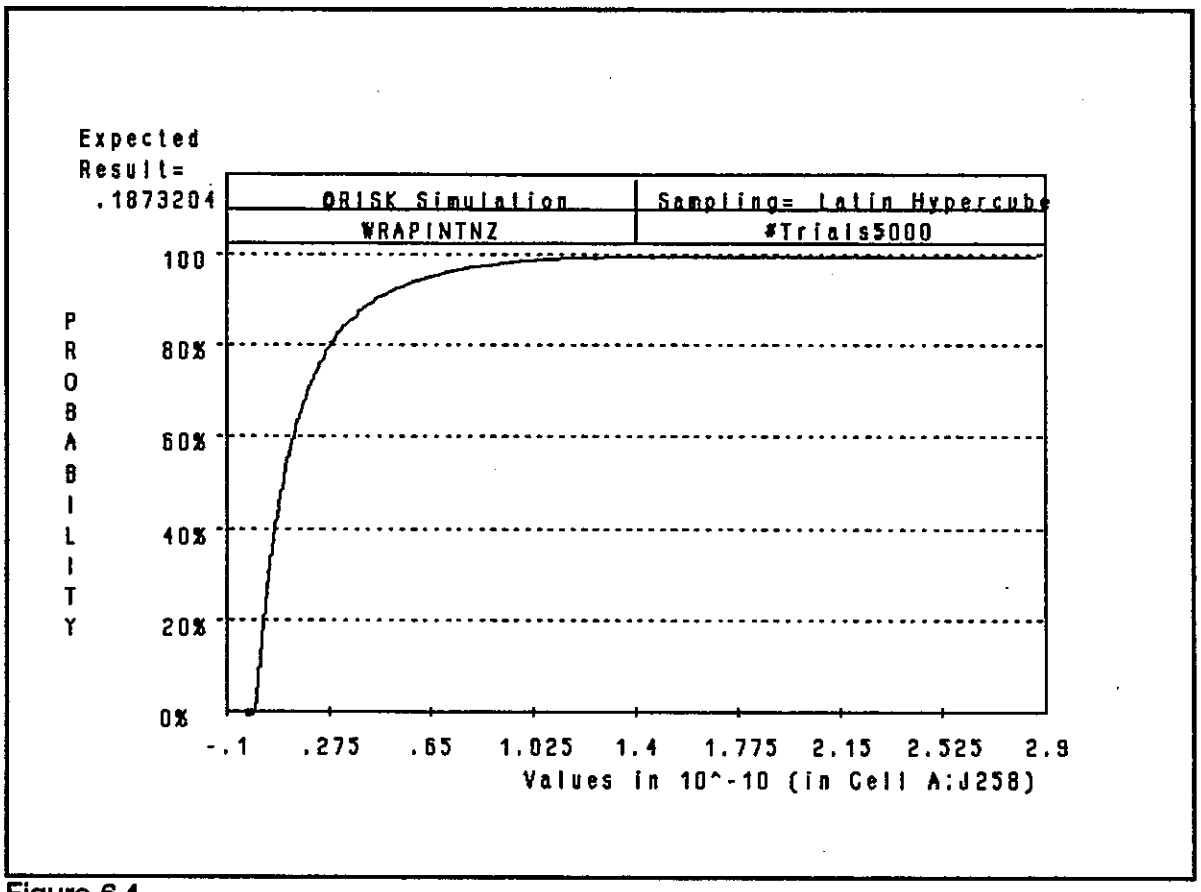


Figure 6.4

This simulation shows that, under the assumptions outlined in the previous sections, there is a 95% probability that the risk of introducing *A. salmonicida* through the medium of contaminated wrapping is negligible should **frozen**, headless, eviscerated ocean-caught Pacific salmon be imported into New Zealand.

6.8 The probability that waste water will be the vehicle by which infection is introduced

The effect of freezing the imported salmon will be to reduce any possible *A. salmonicida* contamination. This reduction in possible contamination will affect the likelihood of waste water serving as the route by which the pathogen could enter New Zealand fish stocks. The input variables affected by freezing of the salmon are shown below in Table 6.4. All other input variables used in the simulation are those described previously in Section 5.14.

Table 6.4: Input variables used to calculate probability of waste water introducing infection when commodity has been frozen.

	Minimum	Most Likely	Maximum
If fish contaminated, probability that kitchen waste water contam? (R19) r1, r2 and r3	0.1	0.2	0.3
Fresh water, infect suscept. host when present? (R22) r1, r2 and r3	1×10^{-7}	5×10^{-7}	1×10^{-6}
Estuarine water, infect suscept. host when present? (R24) r1, r2 and r3	1×10^{-7}	5×10^{-7}	1×10^{-6}
Sea water, infect suscept. host when present? (R26) r1, r2 and r3	5×10^{-8}	1×10^{-7}	5×10^{-7}

6.8.1 The evidence

6.8.1.1 If fish contaminated, probability that kitchen waste water is contaminated? (R19)

As already discussed, a single freeze/thaw cycle is likely to reduce the numbers of *A. salmonicida* which could be present in the flesh of imported wild, ocean-caught Pacific salmon by two orders of magnitude. This inevitably reduces the likelihood of flesh contaminating waste water. That is, the numbers of any *A. salmonicida* which could enter waste water must be at least 10 to 100 times less than for the chilled product. However, some *A. salmonicida* could still enter waste water, even if their number was less than when chilled product is considered.

6.8.1.2 Infect susceptible host when present? (R22), (R24) and (R26)

The smaller number of *A. salmonicida* which might be released into waste water following the preparation of frozen salmon will mean that the likelihood of an infectious dose of organisms being discharged into waterways, and thus infecting a susceptible host of any species, will be significantly less.

6.9 Simulation results; cumulative risk of waste water introducing infection

Again, although the entire simulation was run using input variables based on frozen salmon, only the cumulative risk of kitchen waste water acting as the route by which *A. salmonicida* might be introduced is given here.

This cumulative national risk is shown in Figure 6.5.

Expected/mean result	=	1.8×10^{-10}
Maximum result	=	3.3×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	4.2×10^{-10}
95%	5.6×10^{-10}
99%	9.2×10^{-10}

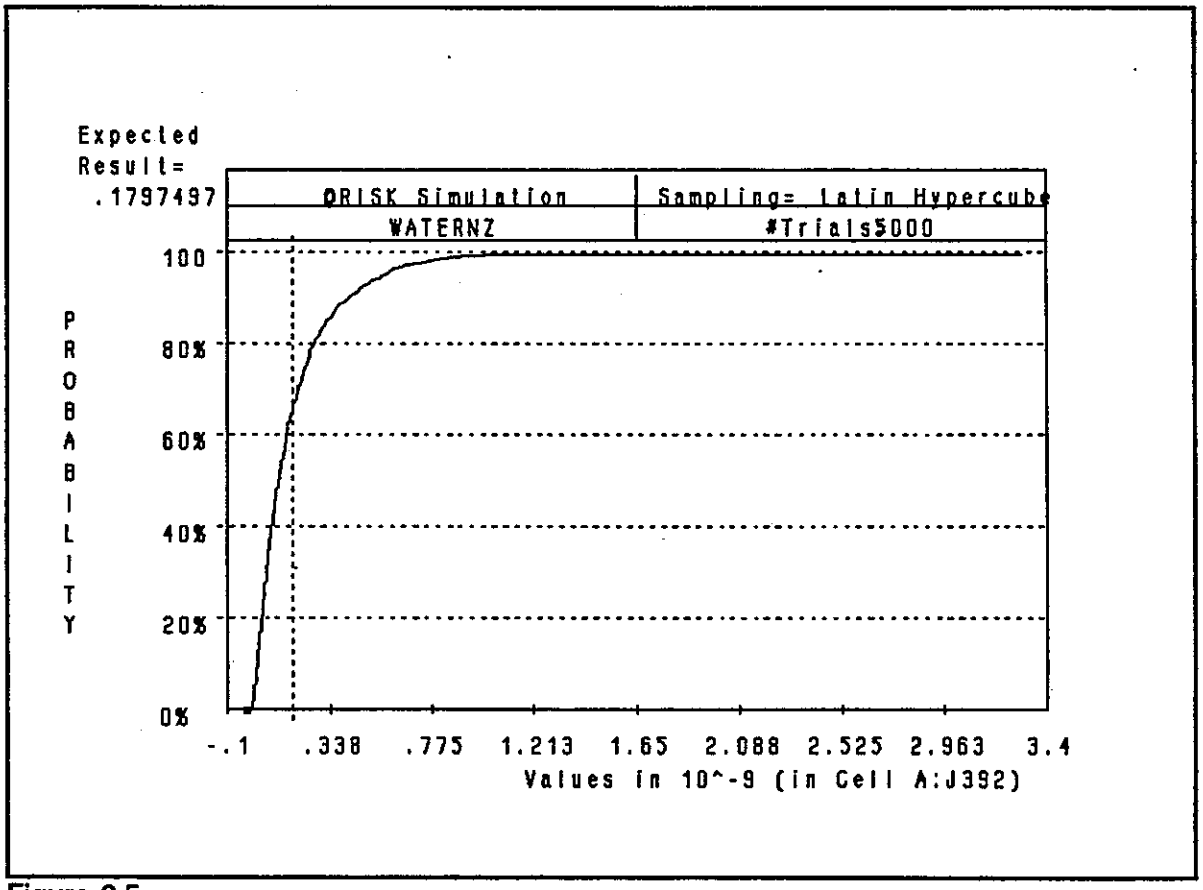


Figure 6.5

Under the assumptions used in the present simulation, there is a 95% probability that the risk of waste water serving as the vehicle by which *A. salmonicida* is introduced into New Zealand salmonid stocks is less than 5.6 per 10^{10} tonnes of frozen, headless, eviscerated ocean-caught Pacific salmon imported.

6.10 The combined probability of infection being introduced through frozen, headless, eviscerated Pacific salmon

The final estimate of risk/tonne is the sum of the individual risks of infection being introduced into each region via scraps, wrappings and waste water. The cumulative probability graph which combines the three regional risks of the three potential routes by which infection could enter local fish stocks (through scraps, through contaminated wrappings or through kitchen waste water) is shown in Figure 6.6.

Expected/mean result	=	2.4×10^{-10}
Maximum result	=	3.5×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	5.5×10^{-10}
95%	7.4×10^{-10}
99%	1.2×10^{-9}

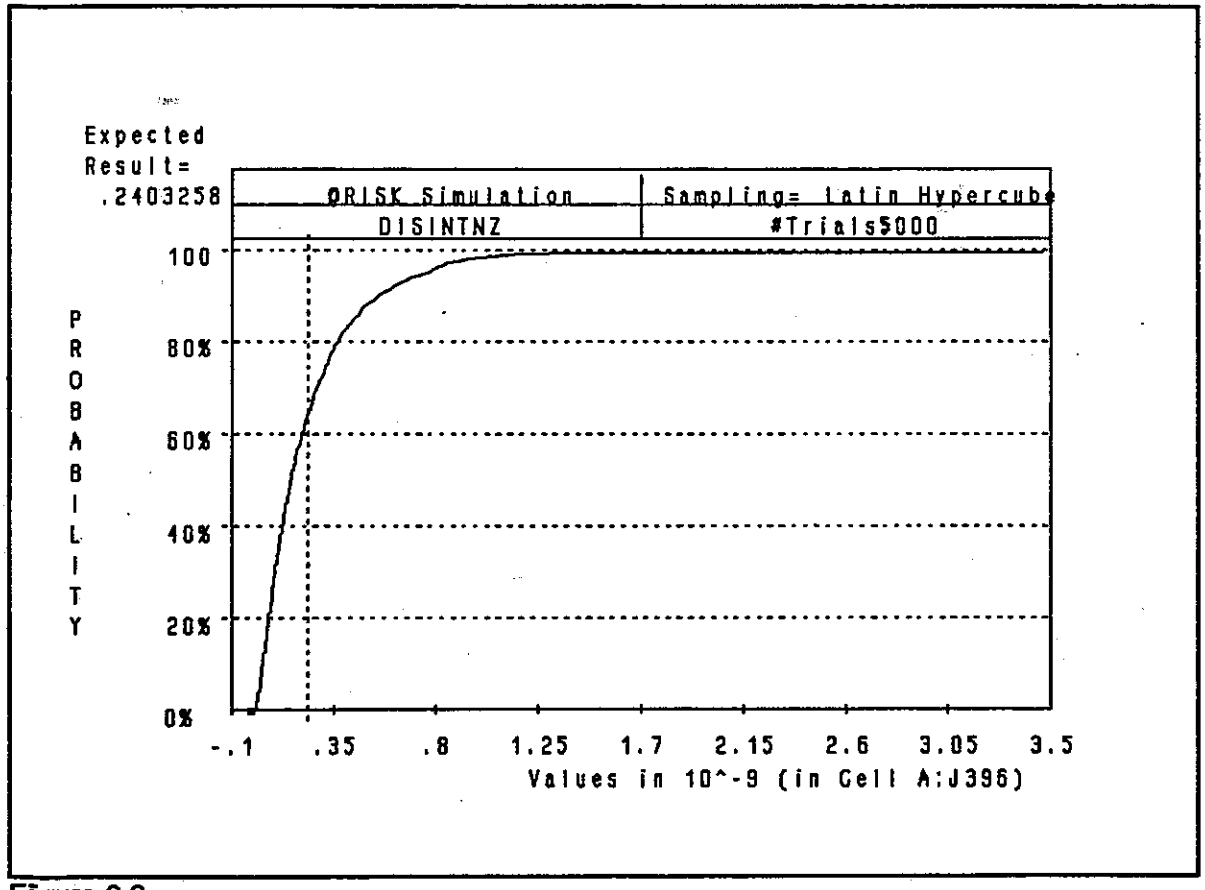


Figure 6.6

Thus, when the individual risks for each route of introduction in each region are combined, the results of the simulation show that, under the assumptions outlined, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand's salmonid fisheries is less than 1 disease introduction [0.74] per 10^{10} tonnes of frozen, headless, eviscerated, wild ocean-caught Pacific salmon imported. This risk is negligible.

7. Chilled skinless fillets

In Section 5 the disease risk posed by importation of chilled, headless, eviscerated, wild, ocean-caught Pacific salmon was quantified. In this section, the commodity assessed is chilled, skinless fillets. The process of filleting and skinning reduces any possible disease risk by reducing the amount of potentially contaminated waste tissue which is disposed of in New Zealand and by increasing the likelihood of diseased tissue being detected and rejected during the grading process at the packhouse.

7.1 The probability of importing contaminated fish

Many of the input variables used in assessing the likelihood of introducing *A. salmonicida* through the vehicle of chilled skinless fillet are the same as used in Section 5 where the risks from chilled, headless, eviscerated fish were assessed. Those which differ are given below a Table 7.1.

Table 7.1: Input variables used to calculate probability of importing contaminated fish flesh. Chilled, skinless fillets.

	Minimum	Most Likely	Maximum
Evisceration and filleting reduces risk by;	0.9*	0.95	0.99
to; (R2)	1 - 0.9	1 - 0.95	1 - 0.99
Inspection and grading reduces risk by;	0.2*	0.6	0.9
to; (R3)	1 - 0.2	1 - 0.6	1 - 0.9
Washing reduces risk by;	0	0.05	0.3
to; (R4)	1 - 0	1 - 0.05	1 - 0.3
Proportion recovered as fillets (F)	0.4	0.46	0.48

* Note: The effectiveness of these steps is linked to the prevalence of infection in the manner described in 5.1.1.4.

7.1.1 The evidence

7.1.1.1 The extent to which evisceration and filleting reduces the risk (R2)

The process of evisceration has a very significant risk-reducing effect. Not only does evisceration remove those tissues in which *A. salmonicida* would be sequestered in grossly normal carrier fish, it also exposes the serosal lining of the body cavity to

inspection, thus revealing lesions or other changes associated with active infection with this or other pathogens. Filleting further reduces any risk. Should fish actually affected with furunculosis (that is, diseased fish) be presented for processing, filleting would reveal the muscle lesions associated with the disease. There is no doubt about this; the lesions of furunculosis are obvious. Very early cases could escape detection, but most clinically affected salmon would be detected at the next step (inspection and grading). However, it must be borne in mind that clinical furunculosis has never been detected in wild, ocean-caught Pacific salmon (see Section 3.8).

The risk-reducing effect of evisceration and filleting is clearly greater than that associated with evisceration alone (Section 5.1.1.3). However, this increase in effectiveness is more likely to be manifest on the "Minimum" and "Most Likely" values, rather than the "Maximum".

7.1.1.2 The extent to which inspection and grading reduces the risk (R3)

Flesh in the form of skinless fillets presents more surfaces to visual inspection than when headless, eviscerated fish are considered. "Minimum", "Most Likely" and "Maximum" values will all be affected.

7.1.1.3 The extent to which washing reduces the risk (R4)

It may be that possible surface contamination could be more easily washed off skinless fillets. However, no attempt is made here to assess this and the same values used in Section 5.1.1.5 are used here.

7.1.1.4 Percent recovered as fillets (F)

In conducting the initial risk analysis on imports of Canadian salmon[137] several people were consulted on the percentage of a salmon recovered as fillets.[138] Estimates of

---

[137] MacDiarmid, SC, Cotton, S. The Risk of Introducing *Aeromonas salmonicida* into New Zealand Salmon Fisheries Through the Vehicle of Frozen Fillets of Ocean-Caught Canadian Salmon. 46 pages. NASS Publication 93-1. Ministry of Agriculture and Fisheries, Wellington, 1993.

[138] Anderson, C D, Veterinary Investigation Officer, MAF Quality Management. 1992-93. Personal communication with Sue Cotton and Stuart MacDiarmid.

Unwin, M, National Institute of Water and Atmospheric Research Ltd. 1993. Personal communication with Sue Cotton.

McNeil, A, General Manager, Angus McNeil & Company, Nelson. 1993. Personal communication with Sue Cotton.

around 60% of the live weight were common. However, some of those answering the question may have been giving their estimates for fillets with the skin on, as otherwise these estimates do not match what is known of the actual composition of Pacific salmon (see Section 5.1.1.7, Table 5.3). Based on the data in Table 5.3 an estimate of 46% recoverable as fillets is used as the most likely estimate.

7.1.2 Simulation results

7.1.2.1 The number of fish represented/tonne of fillets

Figure 7.1 shows the most likely number of fish per tonne of fillets and the probable distribution of values around this expected number. The estimates are based on 5,000 iterations of the @RISK program.

It can be seen that, under the assumptions used in this model, the number of fish represented per tonne is;

Expected/mean result	=	794.5
Maximum result	=	1,547.8
Minimum result	=	431.3

Probability;	That the number of fish is less than;
90%	1,076.5
95%	1,175.6
99%	1,353.1

Margolis, L, Pacific Biological Station, Nanaimo, British Columbia, personal communication to SC MacDiarmid during visit to Nanaimo, November 1993.

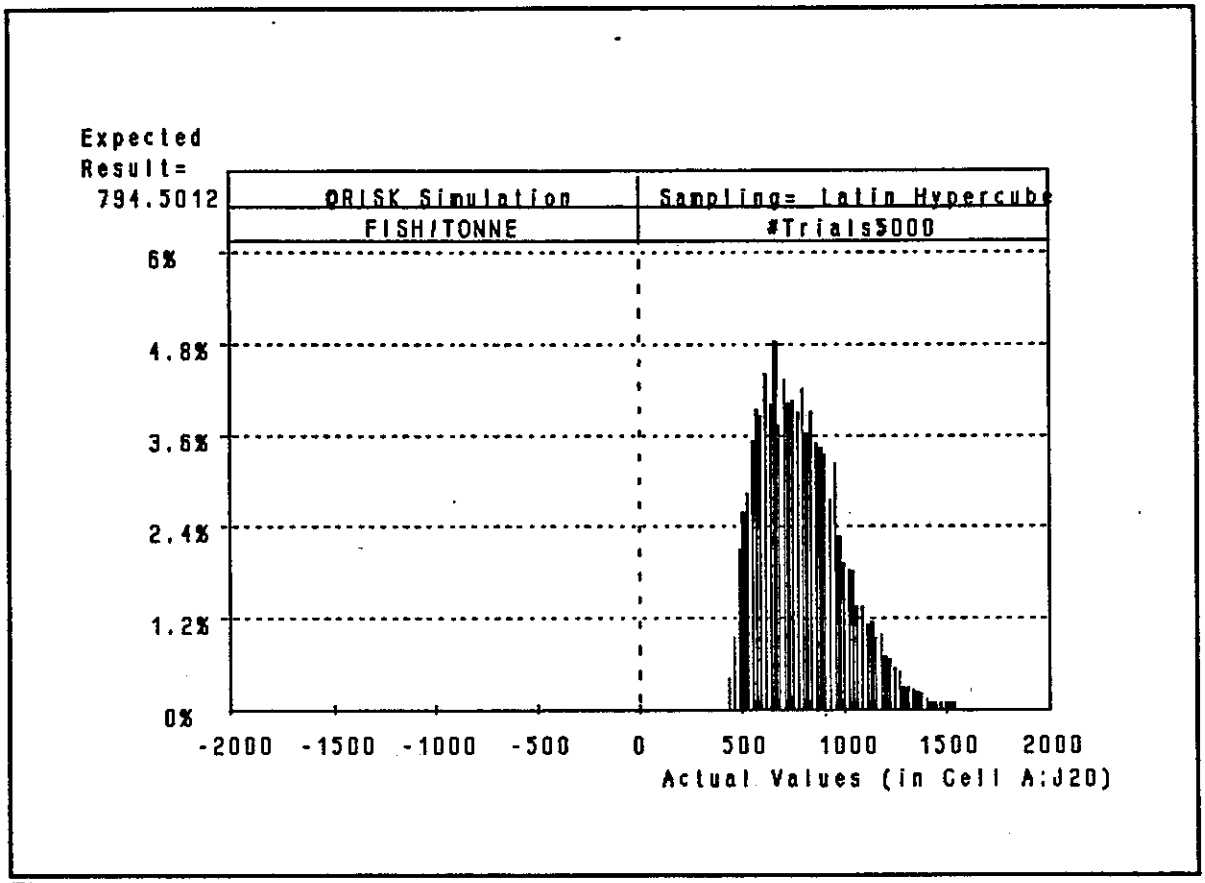


Figure 7.1

That is, there is a 95% probability that a tonne of fillets represents fewer than 1,176 fish. On average though, one can expect a tonne of fillets to represent about 795 fish.

7.1.2.2 The number of *diseased* fish represented/tonne

Figure 7.2 shows the probable number of *diseased* fish represented in each tonne of fillets. With the assumptions used in this model, the number of diseased fish included in a tonne of fillets is;

Expected/mean result	=	21.2
Maximum result	=	83.6
Minimum result	=	0.005

Probability;	That the number of fish is less than;
90%	36.8
95%	43.0
99%	54.7

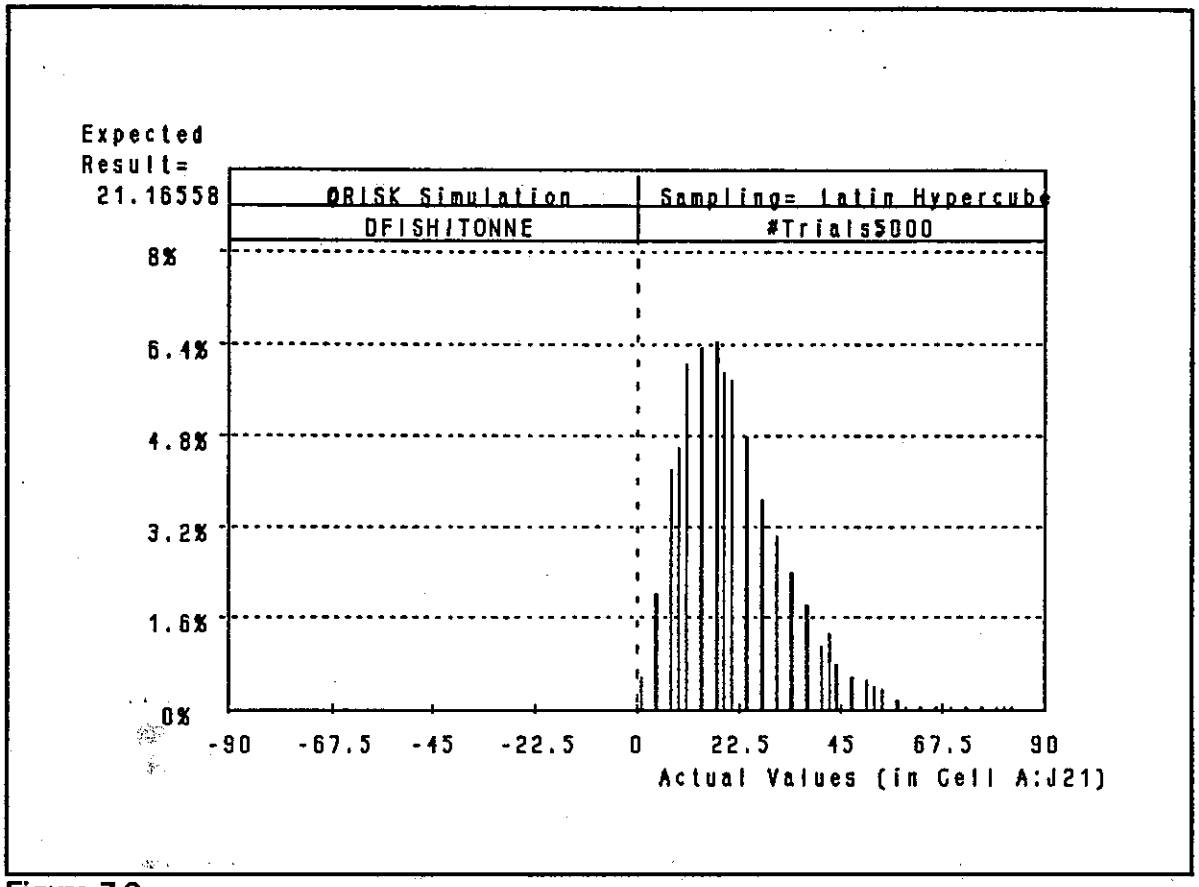


Figure 7.2

That is, there is a 95% probability that a tonne of fillets will include fillets from fewer than 43 infected fish. On average though, one can expect that a tonne of fillets will contain fillets from about 22 infected fish.

7.1.2.3 The probability of *importing* contaminated, potentially infective fillets

Figure 7.3 shows the probable number of potentially infective fish represented in each tonne of fillets imported. The next graph (Figure 7.4) shows the *cumulative* probability of importing fillets representing a given number of infected fish. It can be seen that the number of contaminated fish represented per tonne of fillets imported is;

Expected/mean result	=	0.4
Maximum result	=	3.5
Minimum result	=	9×10^{-5}

Probability;	That the number of fish is less than;
90%	0.9
95%	1.1
99%	1.6

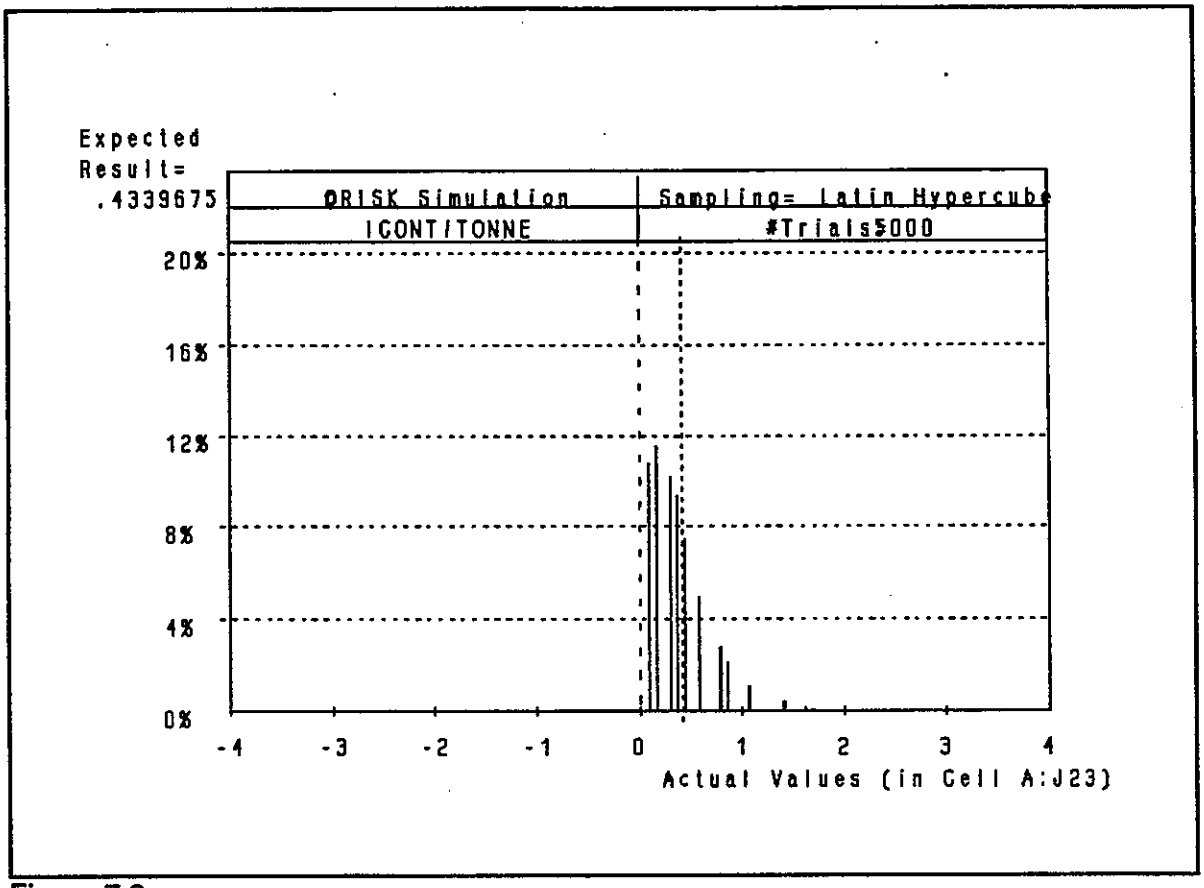


Figure 7.3

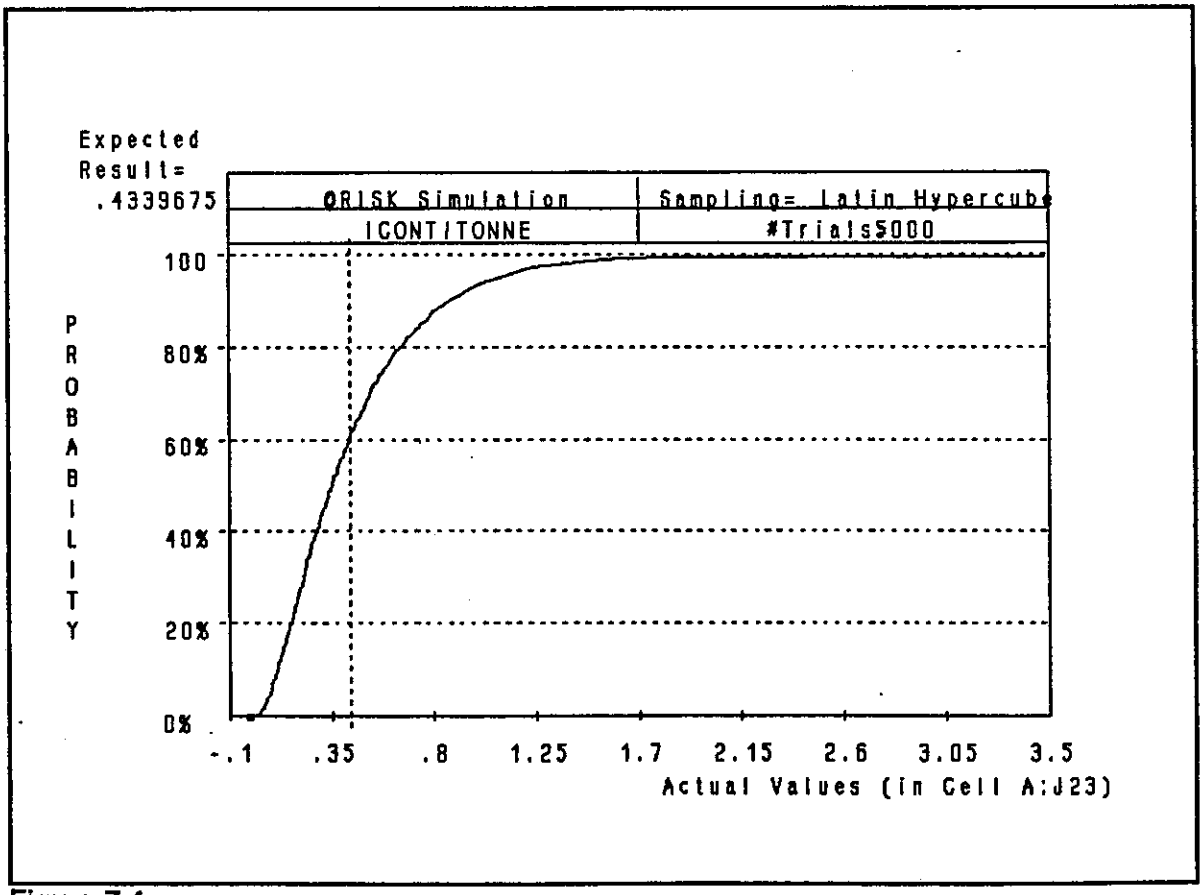


Figure 7.4

That is, there is a 95% probability that the number of fish contributing contaminated, potentially infective fillets is fewer than 1.1 per tonne. This figure takes into account the extent to which the risk has been reduced by evisceration and filleting, inspection and grading and washing. With the assumptions used in the model, it can be seen that each 10 tonnes of fillets imported into New Zealand is likely to contain fillets from around 4 fish which are, theoretically, capable of introducing disease. However, there are many steps between importation of contaminated flesh and the introduction of disease, as the following sections illustrate.

7.2 The probability that scraps will introduce infection

Filleting and skinning wild, ocean-caught Pacific salmon affects the input variables used to estimate the risk of scraps introducing *A. salmonicida*. Only those input variables which are different from those used for chilled, headless, eviscerated fish (Section 5.4) are shown here (Table 7.2 below).

Table 7.2: Input variables used to calculate probability of scraps introducing infection when chilled fillets imported.

	Minimum	Most Likely	Maximum
Proportion disposed of as scraps (P1) r1, r2 and r3	0.001	0.01	0.06

7.2.1 The evidence

7.2.1.1 Proportion disposed of as scraps (P1)

Skinless fillets are likely to be more expensive than cuts which have not had the skin and bones removed. Virtually none of the product is likely to be disposed of as scraps during preparation, certainly no more than 5%.[139] Some product could be discarded as spoiled[140], but again this is unlikely to be a large volume. There is no reason why this figure should vary between regions.

---

[139] Brugger, M, Executive Chef, Park Royal Hotel, Wellington, personal communication with Sue Cotton, 1993.

[140] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication with Stuart MacDiarmid, 1993.

7.2.2 Simulation results

Once those input variables which differ from those used in Section 5 were put into the simulation model, it was run for 5,000 iterations. The cumulative risk of scraps introducing *A. salmonicida*, when chilled, skinless fillets of Pacific salmon are the commodity imported, is shown graphically in Figure 7.5.

Expected/mean result	=	5.5×10^{-10}
Maximum result	=	1.5×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.3×10^{-9}
95%	1.8×10^{-9}
99%	0.4×10^{-9}

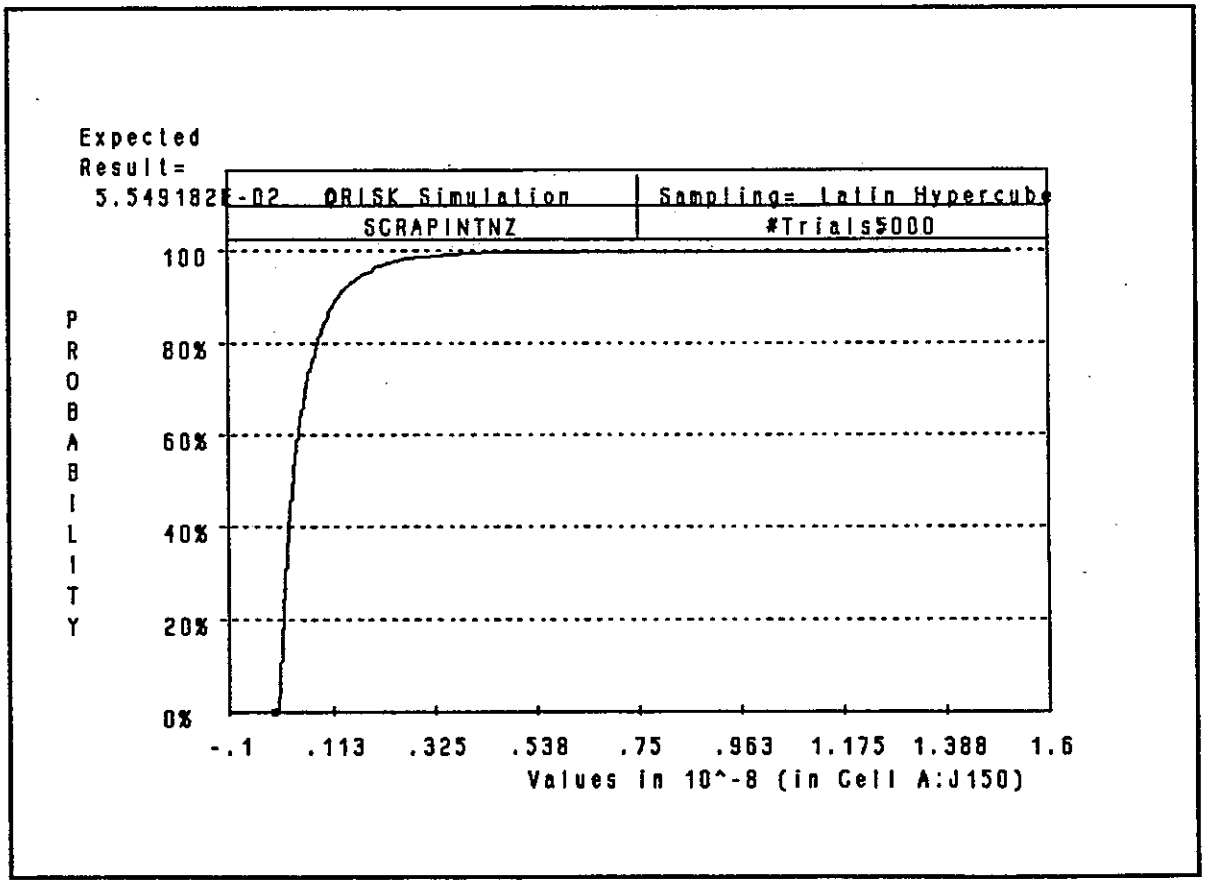


Figure 7.5

This simulation shows that, under the assumptions outlined above, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand through the medium of scraps is less than 1.8 disease introductions per 1,000 million tonnes of chilled ocean-caught salmon fillets imported.

7.3 The probability that wrapping will introduce infection

It is not considered likely that the risk of disease being introduced through wrappings will be significantly affected by filleting. For this reason there were no changes made in the input variables used to estimate this risk. The input variables used were the same as used in Section 5.9.

7.4 The probability that waste water will be the vehicle by which infection is introduced

Any risks associated with importation of skinless fillets will be less than those associated with the importation of headless, eviscerated salmon. One reason for this is that the amount of commodity which is trimmed and disposed of as scraps will be markedly less. In terms of waste water serving to introduce infection, the risk will be less when fillets are involved, because in the preparation of fillets in the kitchen there will be fewer and smaller scraps to be disposed of down the sink.

In estimating the risk of waste water serving to introduce *A. salmonicida* those input variables seen to be different from those used earlier (in Section 5.14) are;

Table 7.3: Input variables used to calculate probability of waste water introducing infection when chilled, skinless fillets are imported.

	Minimum	Most Likely	Maximum
If fillets contaminated probability that kitchen waste water contam? (R19) r1, r2 and r3	0.05	0.1	0.5

7.4.1 The evidence

7.4.1.1 If fillets contaminated probability that kitchen waste water is contaminated? (R19)

Skinless fillets require less handling and, obviously, less trimming than cuts which still contain bone or have the skin attached. Their preparation will involve less opportunity for contamination of waste water and definitely less opportunity for scraps to be flushed into waste water. Nevertheless, should they be contaminated with *A. salmonicida*, fillets are likely to contaminate food preparation surfaces and utensils which, in turn, may contaminate the water used to wash them.

7.5 Cumulative risk of waste water introducing infection

The three regional risks of waste water introducing infection following the importation of chilled, skinless fillets are summed to give the national risk. This cumulative national risk is shown in Figure 7.6.

Expected/mean result	=	6.8×10^{-9}
Maximum result	=	5.8×10^{-8}
Minimum result	=	0

Probability;	That risk is less than;
90%	1.5×10^{-8}
95%	1.9×10^{-8}
99%	3.2×10^{-8}

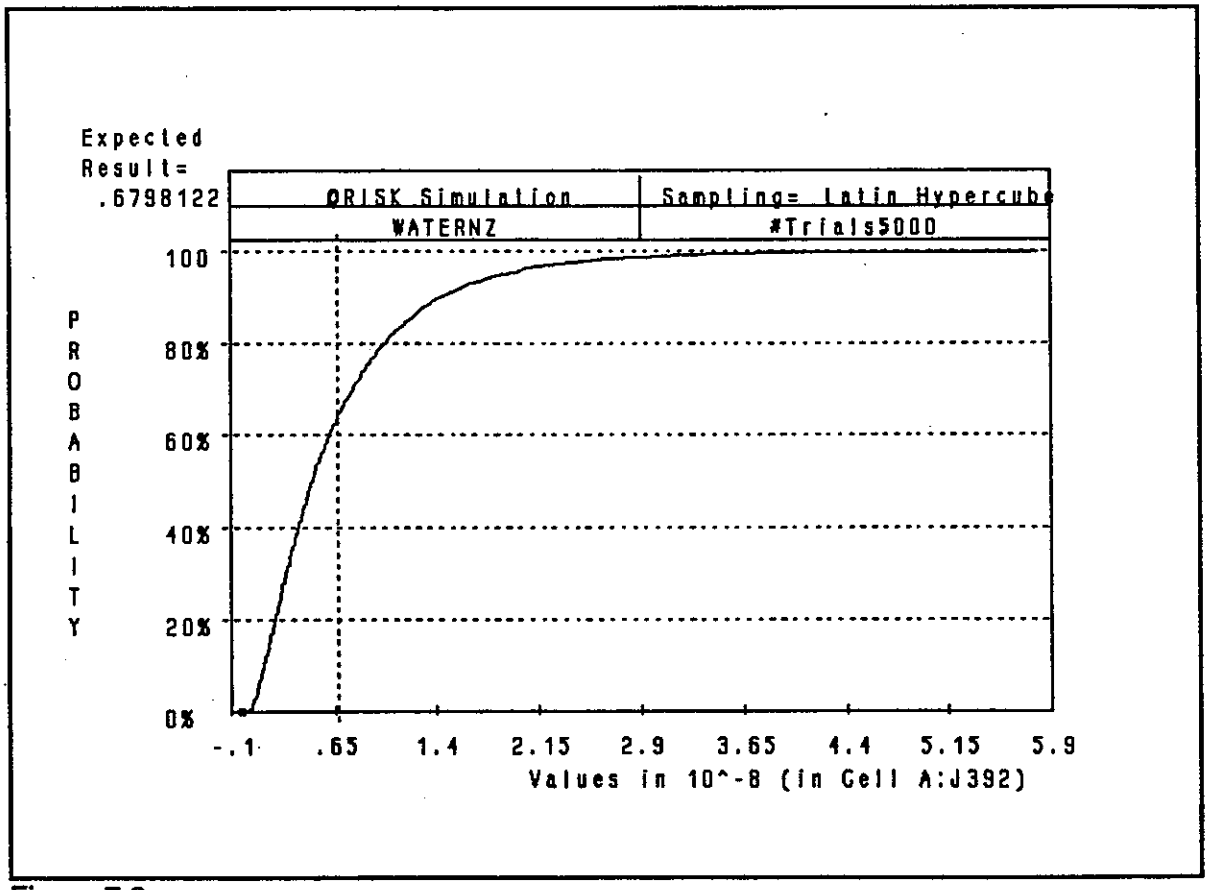


Figure 7.6

Under the assumptions used in the present simulation, there is a 95% probability that the risk of waste water serving as the vehicle by which *A. salmonicida* is introduced into New Zealand salmonid stocks is less than 2 disease introductions per 100 million tonnes of chilled, skinless ocean-caught Pacific salmon fillets imported.

7.6 The combined probability of infection being introduced in chilled, skinless fillets

The final estimate of risk/tonne is the sum of the individual risks of infection being introduced into each region via scraps, wrappings and waste water. The cumulative risk per tonne of imported ocean-caught salmon fillets is shown in Figure 7.7.

Expected/mean result	=	1.2×10^{-8}
Maximum result	=	1.2×10^{-7}
Minimum result	=	0

Probability;	That risk is less than;
90%	2.5×10^{-8}
95%	3.2×10^{-8}
99%	5.3×10^{-8}

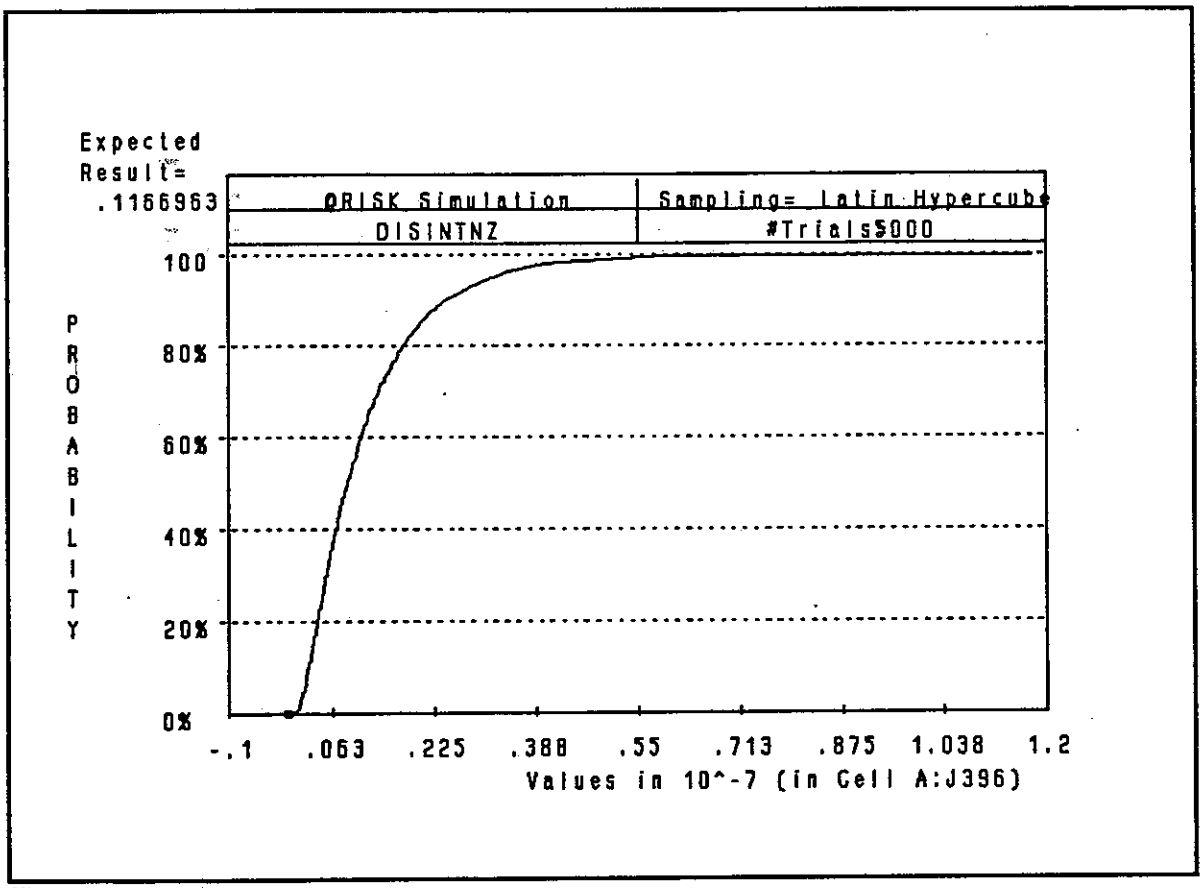


Figure 7.7

Thus, when the individual risks for each route of introduction in each region are combined, the results of the simulation show that, under the assumptions outlined, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand's fish stocks is less than 3.2 disease introductions per 100 million tonnes of chilled, skinless ocean-caught Pacific salmon fillets imported. This risk is negligible.

8. Frozen skinless fillets

The final commodity to be assessed is **frozen** skinless fillets of wild, ocean-caught Pacific salmon. This assessment takes into account the risk-reducing effects of filleting (reducing waste, improving inspection) and freezing (reduces numbers of *A. salmonicida*, if present).

All the input variables have already been described in the previous sections and the evidence supporting the assumption upon which they are based has been outlined.

The appropriate input variables were put into the @RISK simulation model which was then run 5,000 times to generate the results given below.

8.1 The probability of *importing* contaminated fish

Figure 8.1 shows the probable number of contaminated, potentially infective fish represented in each tonne of fillets imported. The next graph (Figure 8.2) shows the *cumulative* probability of importing fillets representing a given number of contaminated fish. It can be seen that the number of contaminated fish represented per tonne of fillets imported is;

Expected/mean result	=	0.02
Maximum result	=	0.24
Minimum result	=	4.0×10^{-6}

Probability;	That the number of fish is less than;
90%	0.05
95%	0.06
99%	0.98

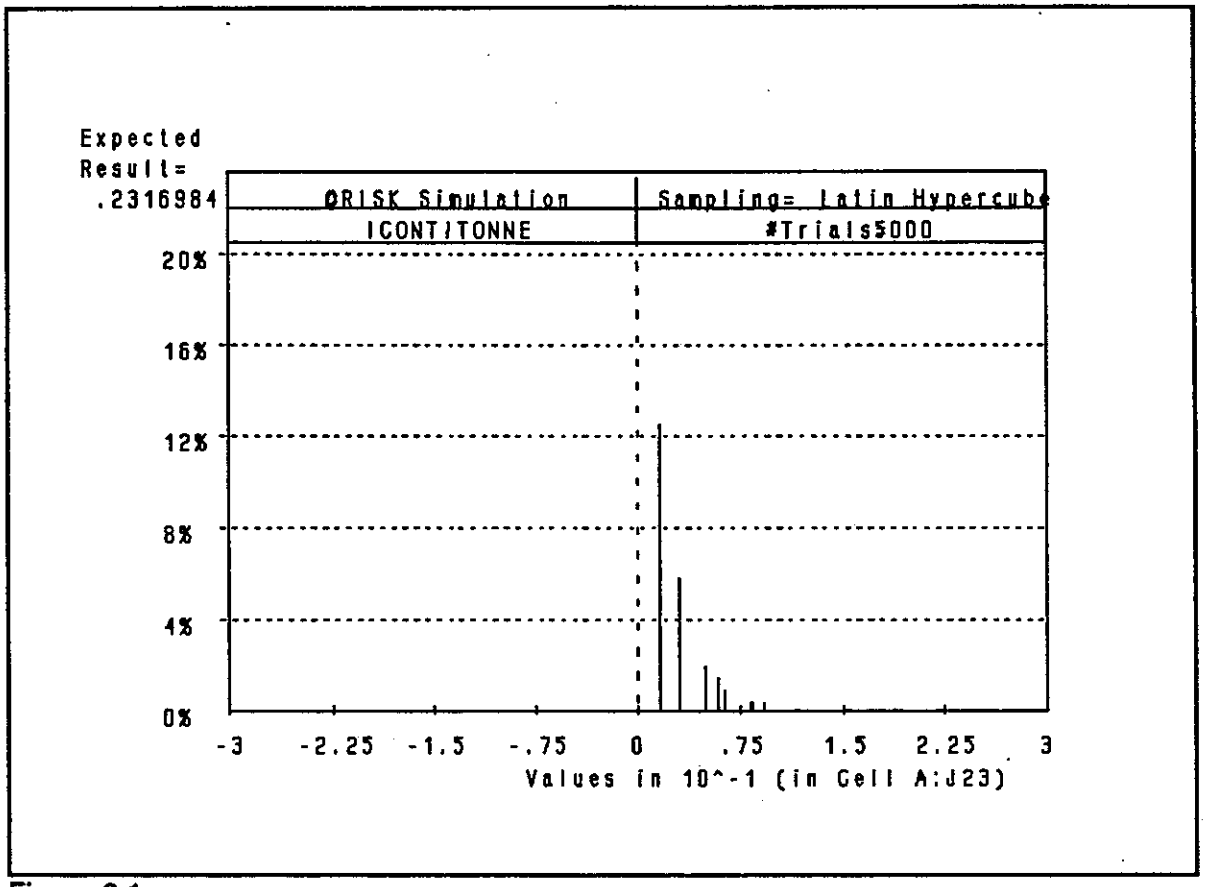


Figure 8.1

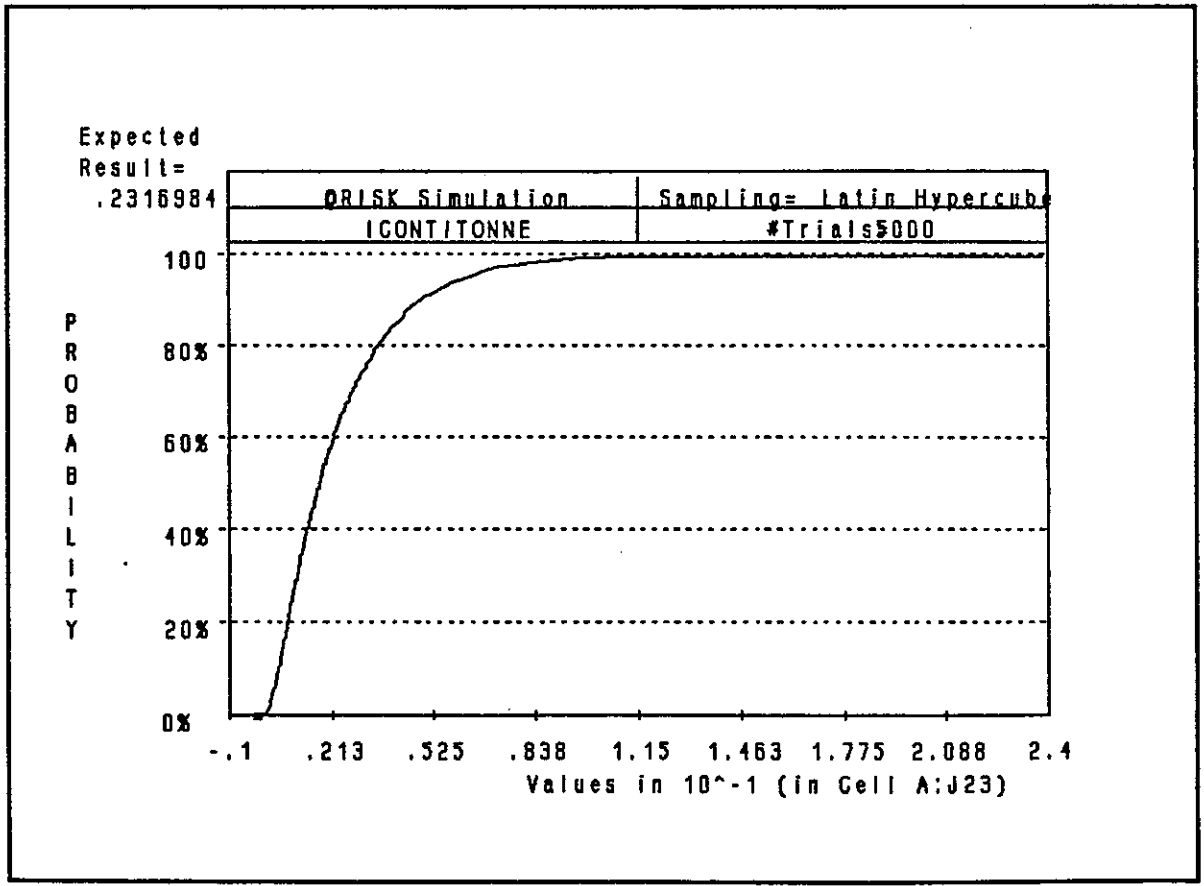


Figure 8.2

That is, there is a 95% probability that the number of fish contributing contaminated, potentially infective fillets is fewer than 6 per 100 tonnes. This figure takes into account the extent to which the risk has been reduced by evisceration and filleting, inspection and grading, washing and freezing. With the assumptions used in the model, it can be seen that each 100 tonnes of fillets imported into New Zealand is likely to contain 2 fillets which are, theoretically, capable of introducing disease.

8.2 Cumulative risk of scraps introducing infection

The cumulative risk of scraps acting as the vehicle by which *A. salmonicida* could be introduced is shown graphically in Figure 8.3.

Expected/mean result	=	3.4×10^{-12}
Maximum result	=	1.2×10^{-10}
Minimum result	=	0

Probability;	That risk is less than;
90%	8.1×10^{-12}
95%	1.2×10^{-11}
99%	2.2×10^{-11}

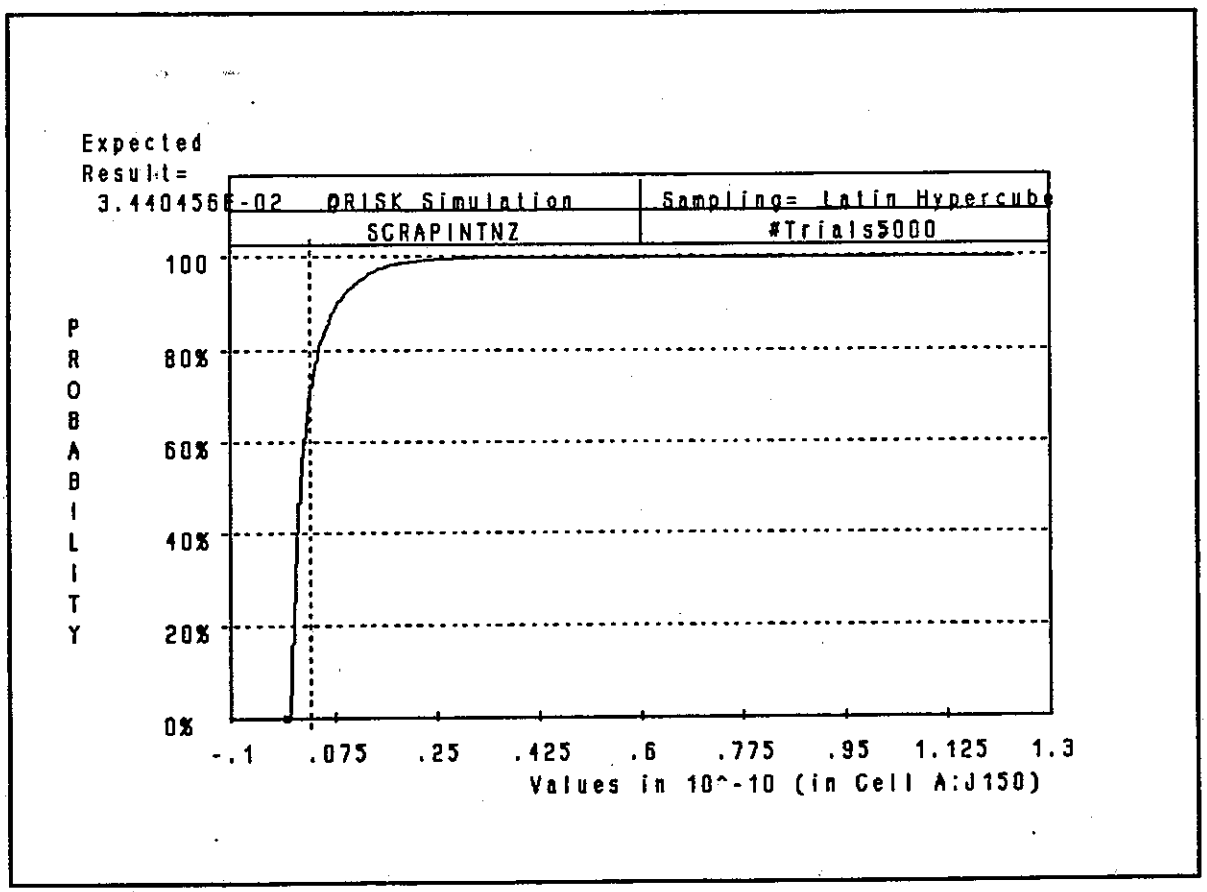


Figure 8.3

This simulation shows that, under the assumptions outlined above, there is negligible risk of introducing *A. salmonicida* into New Zealand through the medium of scraps when frozen ocean-caught salmon fillets are imported.

8.3 Cumulative risk of wrapping introducing infection

The cumulative national risk of *A. salmonicida* being introduced into New Zealand via the vehicle of contaminated wrappings is shown in Figure 8.4.

Expected/mean result	=	1.3×10^{-11}
Maximum result	=	4.7×10^{-10}
Minimum result	=	0

Probability;	That risk is less than;
90%	3.0×10^{-11}
95%	4.3×10^{-11}
99%	8.1×10^{-11}

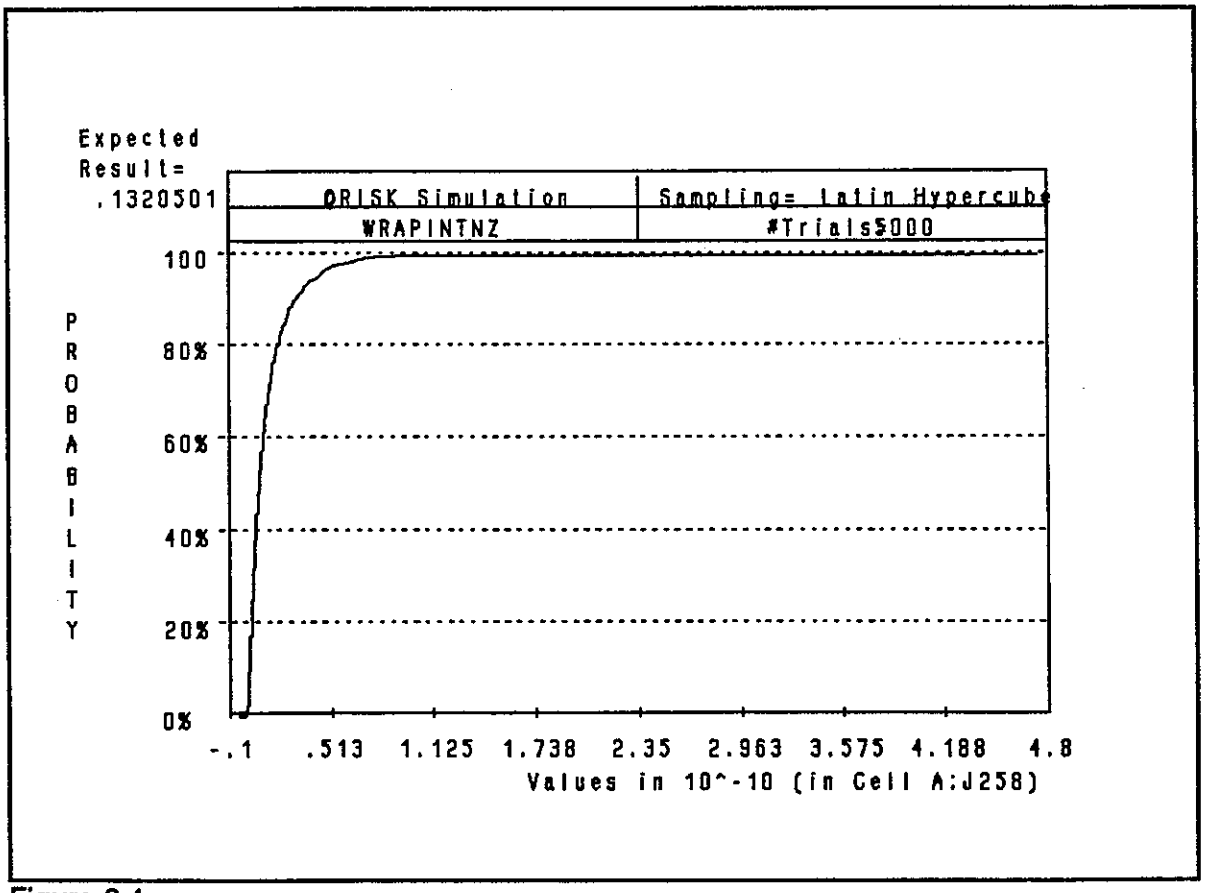


Figure 8.4

This simulation shows that, under the assumptions outlined in the previous sections, there is negligible risk of introducing *A. salmonicida* through the medium of contaminated wrapping when frozen ocean-caught Pacific salmon fillets are imported into New Zealand.

8.4 Cumulative risk of waste water introducing infection

The three regional risks of waste water introducing infection are summed to give the national risk which is shown in Figure 8.5.

Expected/mean result	=	0.9×10^{-10}
Maximum result	=	1.1×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	2.1×10^{-10}
95%	2.8×10^{-10}
99%	5.0×10^{-10}

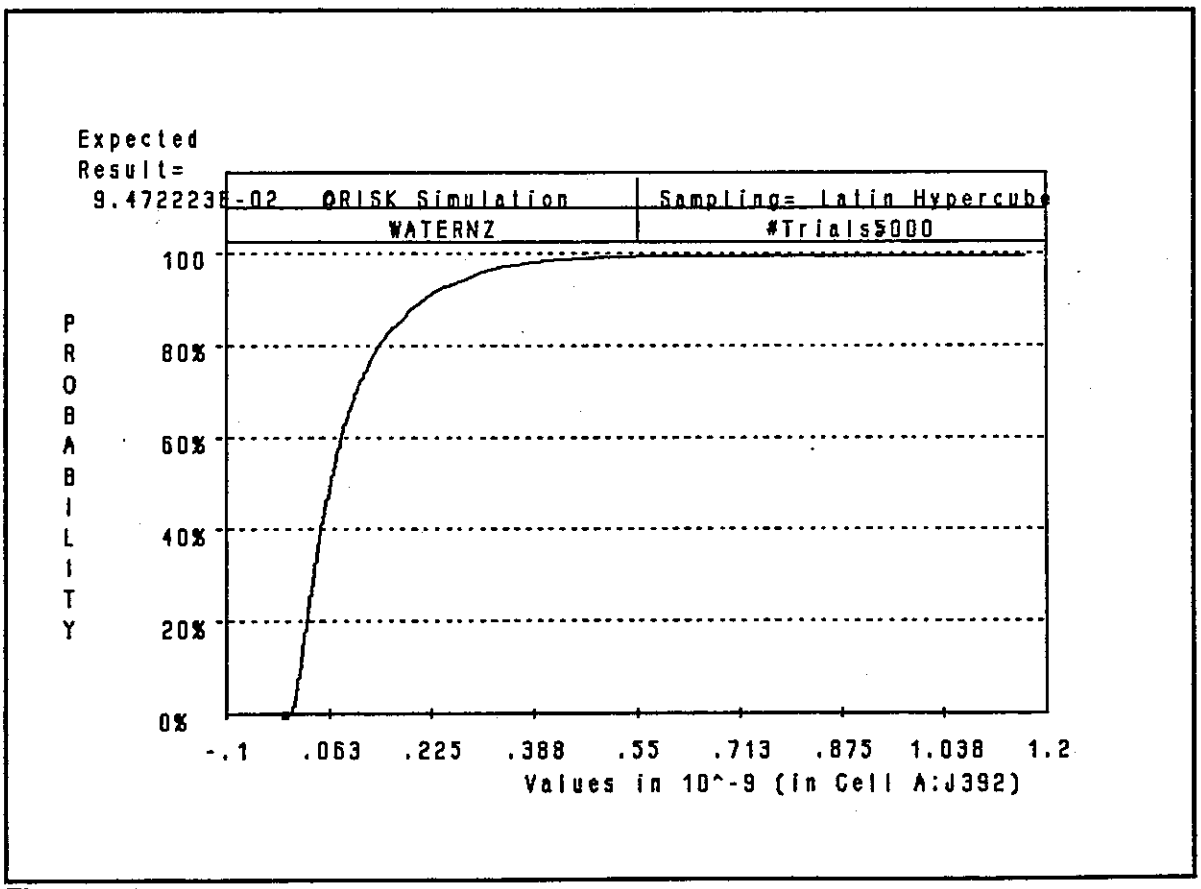


Figure 8.5

Under the assumptions used in the present simulation, there is a 95% probability that the risk of waste water serving as the vehicle by which *A. salmonicida* is introduced into New Zealand salmonid stocks is less than 3 disease introductions per 10^{10} tonnes of frozen, skinless ocean-caught Pacific salmon fillets imported.

8.5 The combined probability of infection being introduced/tonne of fillets

The final estimate of risk/tonne is the sum of the individual risks of infection being introduced into each region via scraps, wrappings and waste water. The cumulative probability graph which combines the three regional risks of the three potential routes by which infection could enter local fish stocks (through scraps, through contaminated wrappings or through kitchen waste water) is shown in Figure 8.6.

Expected/mean result	=	1.1×10^{-10}
Maximum result	=	1.3×10^{-9}
Minimum result	=	0

Probability;	That risk is less than;
90%	2.5×10^{-10}
95%	3.3×10^{-10}
99%	5.7×10^{-10}

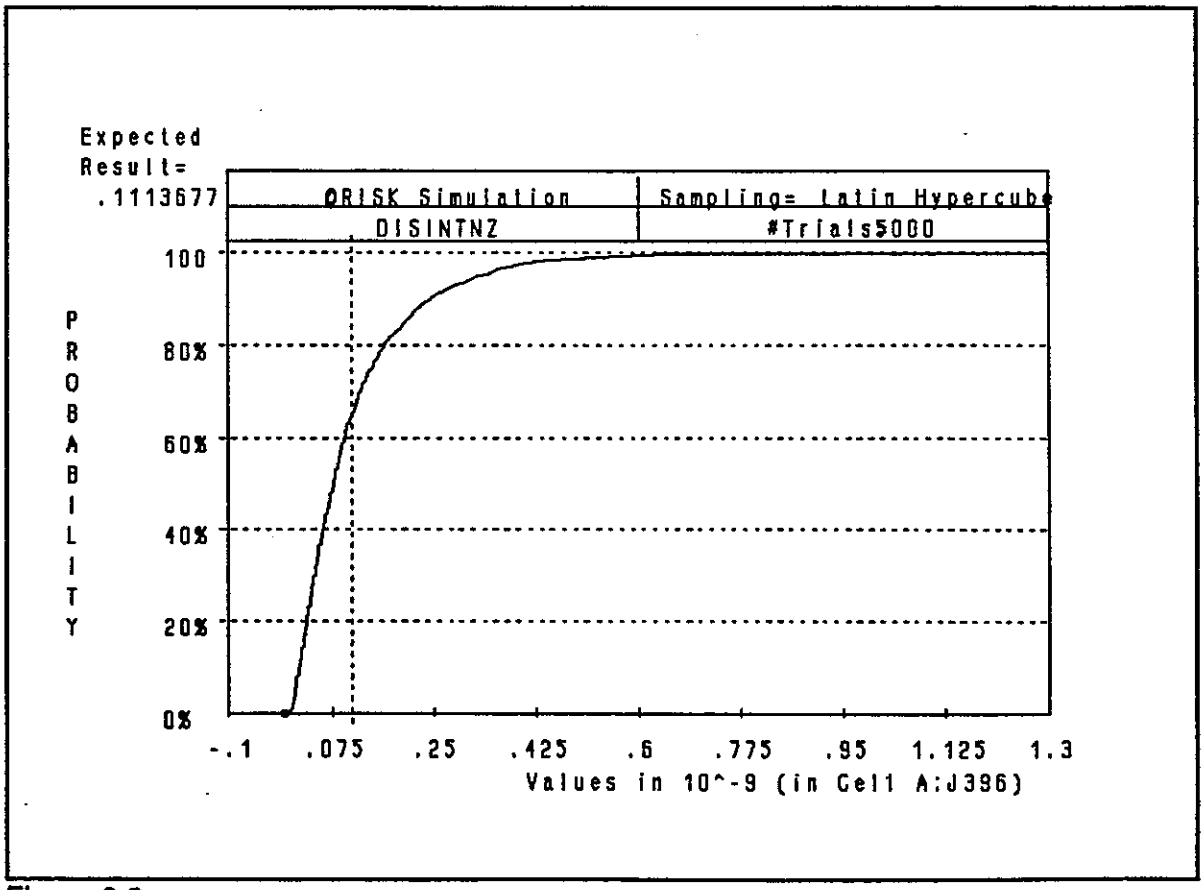


Figure 8.6

Thus, when the individual risks for each route of introduction in each region are combined, the results of the simulation show that, under the assumptions outlined, there is a 95% probability that the risk of introducing *A. salmonicida* into New Zealand's salmonid fisheries is less than 2.5 disease introductions per 10^{10} tonnes of frozen, skinless ocean-caught Pacific salmon fillets imported. This risk is essentially zero.

9. Conclusions

A qualitative risk analysis of the risks of introducing exotic fish diseases through importations of wild, ocean-caught Pacific salmon from Canada has demonstrated that none of 23 diseases of salmonids present in North America is likely to be introduced should imports be permitted.

Consideration of the various diseases leads to the conclusion that of all the exotic diseases present in North American salmonids, furunculosis, caused by the bacterium *Aeromonas salmonicida*, is the disease which would be most likely to be carried in the type of commodity under consideration. This conclusion, supported by a number of experts consulted during the risk analysis, is based on the fact that, of all the diseases considered, none result in greater numbers of pathogen being present in the flesh of infected fish. *A. salmonicida* is also capable of infecting, under appropriate conditions, a relatively wide range of fish species.

A quantitative risk assessment was conducted on four commodities;

- Chilled, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Frozen, headless, eviscerated, wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Consumer packs of skinless, boneless chilled fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.
- Consumer packs of skinless, boneless frozen fillets of wild, ocean-caught Pacific salmon harvested off the west coast of Canada.

The quantitative risk assessment took into account what is known of the prevalence of *A. salmonicida* in wild, ocean-caught Pacific salmon, the distribution and numbers of *A. salmonicida* found in infected Pacific salmon, the effect of processing on the numbers of *A. salmonicida* in the flesh of infected fish, the survival of *A. salmonicida* in the environment, the dose of *A. salmonicida* required to infect susceptible fish (of any species), and waste management practices in New Zealand. It also pointed out that the importation of commodity for further processing in New Zealand could constitute a significantly different risk from importation for consumption without further processing, and that such importations should be restricted to premises with approved waste disposal systems. The risk assessment also acknowledged that the risks posed by farmed salmon could be greater than those posed by wild-caught salmon but that substitution of product is improbable because of the higher cost of farmed salmon in Canada.

The quantitative risk assessment was based on a Monte Carlo-type simulation model in which each input variable was defined as a distribution of possible values from which the model randomly selected values at each of 5,000 iterations.

The quantitative risk assessment demonstrated that the risk of introducing *A. salmonicida* into New Zealand's farmed, recreational or native fish stocks is extremely remote. With the least-processed commodity (chilled, headless, eviscerated salmon) the model estimated that there is a 95% probability that there would be fewer than 1 disease introductions per 10 million tonnes imported. To put this into perspective, the analysis pointed out that the entire annual production of wild, ocean-caught Pacific salmon is no more than 100,000 tonnes.

Boustead has correctly pointed out[141] that the risks associated with other diseases would be cumulative to the risks posed by *A. salmonicida*, and to ignore this is to understate the real likelihood of disease introduction. Any risk posed by any of the other diseases must be added to that posed by furunculosis. However, as has been explained, there are compelling reasons for considering that no disease is more likely to be introduced than *A. salmonicida*. Given that for many of the diseases discussed in Section 3 there is virtually no risk of introduction in the commodities under discussion, and for the remainder the risks are likely to be orders of magnitude smaller than that for furunculosis, the cumulative risk of disease introduction is unlikely to be significantly greater than the range of risk estimates described here for *A. salmonicida*. That is, the overall risk of introducing diseases of salmon through the vehicle of headless, eviscerated, wild, ocean-caught Pacific salmon, appropriately certified by the Canadian Government authorities as to origin and grade, is negligible and poses no threat to either New Zealand's wild and farmed salmonid stocks or to non-salmonid fish stocks.

---

[141] Boustead, N, National Institute of Water and Atmospheric Research Ltd, personal communication to SC MacDiarmid, 23 December 1993.

10. Acknowledgements

Sue Cotton, Technical Advisory Officer, Animal Products Imports, MAF Regulatory Authority, assisted with the design of the model and derived many of the variables used in the risk assessment through personal communications.

Vaughan Seed, National Adviser, Import/Export, MAF Regulatory Authority, advised on many of the calculations, detected significant errors, and obtained more up to date information than had been used in early drafts.

Geoff Allen, formerly National Adviser, Animal Disease Surveillance, MAF Regulatory Authority, scrutinised many of the calculations and detected significant errors which had appeared in early drafts.

Martin Van Ginkel, Technical Advisory Officer, Agricultural Security and Animal Health, MAF Regulatory Authority, gathered information including that relating to salmon consumption and composting.

David Vose, Risk Analysis Services, Wincanton, Somerset, advised on the simulation model and the means of linking prevalence to efficacy of evisceration and inspection.

David Banks and Peter Beers, Australian Quarantine and Inspection Service, Canberra, generously shared their own model used for assessing the risks posed by imports of New Zealand salmon to Australian fisheries.

Eve Archer, Librarian, MAF, was tireless in obtaining information on water usage, volume of domestic garbage produced etc, etc.

Randy Morley, Chief, Animal Health Risk Assessment, Agriculture Canada, pointed out the need to tie the model to volume.

Peter Davie, Massey University, suggested modifying the model to take into account the different distribution of the human and salmonid populations. He made other valuable suggestions.

Trevor P T Evelyn, Head, Fish Health and Parasitology Section, Biological Sciences Branch, Pacific Biological Station, Nanaimo, British Columbia, spent many hours discussing diseases of salmon during Stuart MacDiarmid's two visits to Nanaimo and provided copies of many papers unavailable through the New Zealand library system as well as many important unpublished data on fish pathogens.

Iola M Price, Director, Aquaculture and Resource Development, Biological Sciences Directorate, Department of Fisheries and Oceans, Ottawa, provided copies of documents unavailable in New Zealand, answered numerous questions about the inspection and regulatory activities of her Department and arranged for SC MacDiarmid to be invited to attend as observer the Canada-Australia Salmon Technical Meeting under the Auspices of GATT Article XXII, Nanaimo, British Columbia, 25-26 July 1994.

T G Carey, Senior Program Advisor, Aquaculture and Fish Health, Aquaculture and Resource Development, Biological Sciences Directorate, Department of Fisheries and Oceans, described aspects of Canada's Fish Health Protection Regulations.