Hazards Affecting Australian Seafood

Part 1:

Priority Listing of Issues and Risk Ranking of Hazards Affecting Australian Seafood

John Sumner

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SafeFish

AUSTRALIAN SEAFOOD COOPERATIVE RESEARCH CENTRE

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Background

SafeFish is a partnership of seafood experts that has been formed to assist the industry to resolve technical trade impediments, especially in relation to issues associated with food safety and hygiene. Partnership members of SafeFish include the Australian Quarantine Inspection Service (AQIS), Food Standards Australia New Zealand (FSANZ), the Australian Seafood Cooperative Research Centre, Codex Australia, the Seafood Access Forum and Seafood Services Australia (SSA).

The purpose of SafeFish is to:

- Provide rapid technical response to maintain free and fair access to key markets
- Ensure the safety and hygiene of seafood.

A number of potential food safety and market access issues have been raised by the SafeFish partnership members and by the Seafood Access Forum. SafeFish seeks to provide technical support to clarify these issues and to rank them on the basis of various factors such as severity and likelihood as applied to:

- Seafood captured or grown in Australian waters
- Imported seafood

Terms of Reference

1. Collate master list of current issues and associated data
2. Develop criteria for excluding issues that are not technical, or able to be progressed by technical work
3. Separate technical trade issues from food safety issues into two lists to be ranked independently
4. Develop hazard sheets for the food safety issues and summary sheets giving brief background on the technical trade issues
5. For the food safety issues undertake a qualitative risk ranking and, where data permit, a semi-quantitative ranking using Risk Ranger
6. Create criteria and score or rank technical trade issues on basis of consequence and likelihood
7. Provide a concise report describing tasks undertaken and recommendations on which issues should be addressed in the next one to five years and present findings to a stakeholder meeting (with SafeFish partnership members) and give partnership members input into the final rankings
8. Finalise report

Structure of the Report

This report is presented in two parts. Part 1 is a response to each TOR and Part 2 contains background information supporting responses to each TOR as appendices.

Part 2, Section A contains two appendices: Appendix 1: Food poisonings in Australia due to seafood and Appendix 2: Recalls of seafood in Australia. Part 2, Section B contains detailed listings of food poisonings associated with consumption of seafood in Tables B1-B4.

Hazard sheets are presented in full in Section C following the Background Tables. Key information from each hazard sheet is used in summary form where appropriate in TORs 4-6 (Part 1).

TOR 1: Master List of Potential Issues

Issues identified by the Seafood Access Forum and the SafeFish partners are:

1. Parasites in seafood
2. Norovirus trade issues (Hong Kong and Singapore)
3. Arsenic in seafood
4. EU MRL Cadmium in prawns (and crustacean)
5. Review of testing requirements for imported seafood
6. Ciguatoxins
7. Seafood allergens
8. Potential regulation of cyclic imines (e.g. pinnatoxins) in shellfish
9. Faecal pollution indicators
10. Shellfish market access into the US
11. Biosecurity
12. Export regulations: Unloading in foreign ports
13. Environmental trade barriers: Ecolabelling
14. Export trade data
15. Product testing issues
16. Mercury advisory
17. EU IUU legislation
18. XyRex Prawnfresh usage
19. Vibrios in Australian bivalve molluscs

A number of sources have been interrogated to inform on food safety hazards in seafood in the Australian context:

- National Risk Validation Project (NRVP)
- OzFoodNet food poisoning data
- FSANZ recalls
- AQIS sampling program for imported seafoods

The collated data for these identified hazards are presented in Part 2, Section A, Tables A1-4, the source data for which is contained in Part 2, Section B, Background Tables B1-B4.

TOR 2: Develop criteria for excluding issues that are not technical or able to be progressed by technical work

In ascribing an issue to a 'technical' or 'not technical and unable to be progressed by technical work' category, the following criteria have been used.

Technical

The issue is a hazard which is considered to present a food safety risk and can be progressed by technical work which, in the present document, may comprise a status report, hazard sheet, or a qualitative risk assessment e.g. arsenic in seafood.

Non-technical

The issue is not a food safety hazard *per se*, but rather a regulatory position which requires resolution between controlling authorities and/or industry bodies e.g. improving the timeliness of updating trade data.

TOR 3: Separate technical trade issues from food safety issues into two lists to be ranked independently

Based on the criteria set out in TOR 2 the segregation of hazards and issues is presented in Tables 1 and 2.

Table 1: Potential hazards in seafood consumed in Australia and in Australian export seafood

Biological Hazards
Parasites in seafood

Chemical Hazards
Arsenic in seafood
EU MRL Cadmium in prawns (and crustaceans)
Ciguatoxins
Seafood allergens
Mercury advisory

Table 2: Issues of a regulatory nature

Issue	Suggested Progression
Review of testing requirements for imported seafood	See issues sheet provided
Shellfish market access into the US	See issues sheet provided
Norovirus trade issues (Hong Kong and Singapore)	See issues sheet provided
Potential regulation of cyclic imines in shellfish	See issues sheet provided
Faecal pollution indicators	See issues sheet provided
Product testing issues	See issues sheet provided
Vibrios in Australian bivalves	See issues sheet provided
Biosecurity	Consult industry & AQIS
Export regulations: Unloading in foreign ports	Consult industry & AQIS
Environmental trade barriers: Ecolabelling	Consult industry & C/wealth
Export trade data	Consult industry & AQIS
EU IUU legislation	Consult AQIS & EU
XyRex Prawnfresh usage	Consult AQIS & Japanese author

TOR 4 and TOR 5:
Develop hazard sheets for the food safety issues and summary sheets giving brief background on the technical trade issues
Undertake a qualitative risk ranking for the food safety issues

Summaries for each of the six regulatory issues and six hazards identified under TOR 2 are presented in these TORs. With each hazard sheet is presented a qualitative risk assessment. Complete hazard sheets for the additional hazards that were identified through other data sources are presented in Part 2.

Qualitative Risk Assessment Tool

A qualitative framework for the rating of risk has been used based on premises published by the International Commission on Microbiological Specifications of Foods (ICMSF, 2002) and by Food Science Australia (2000). The ICMSF formulated descriptors for severity of illnesses caused by various pathogens, which was used in conjunction with a matrix of factors assembled by FSA (2000) to describe risk profiles of plant products.

Taken together, the present qualitative matrix is based on criteria for:

Severity

The severity of the identified hazards was classified according to the International Commission of the Microbiological Specifications of Food (ICMSF 2002) with level of severity defined as follows:

IA. Severe hazard for general population; life threatening or substantial chronic sequelae or long duration.

IB. Severe hazard for restricted populations; life threatening or substantial chronic sequelae or long duration.

II. High hazard; incapacitating but not life threatening, sequelae rare, moderate duration.

III. Moderate; not usually life threatening, no sequelae, normally short duration, symptoms are self limiting, can be severe discomfort.

Occurrence of illness

This is classified as low, medium or high based on the hazard's involvement as recorded in public health statistics.

Growth

An indication is given of whether growth of the pathogen in the product is required to cause disease. In general, microbiological hazards need to grow in the product or be present at high numbers before there is a significant risk of disease.

In the case of chemicals such as heavy metals where chronic exposure is needed over a protracted period, for the purposes of the qualitative risk assessment, the situation is considered analogous to that of growth.

Production, processing or handling of food

The production, processing or handling of the food may increase, decrease or not affect the concentration of the hazard.

Consumer terminal step

This element considers whether a consumer terminal step, such as cooking, is applied to the product. Cooking by the consumer will, for most biological hazards, reduce the subsequent risk of disease.

Epidemiology

Consideration is given as to whether the hazard-commodity combination has been recorded as a cause of food poisoning.

References

Food Science Australia. 2000. Final Report – Scoping study on the risk of plant products. SafeFood NSW, Homebush, NSW, Australia.

ICMSF. 2002. *Microorganisms in Foods: 7 Microbiological testing in food safety management.* New York, NY, United States of America: Kluwer Academic/Plenum Publishers.

Hazard Sheet: Cadmium

Hazard

In 1972, the Joint FAO/WHO Expert Committee on Food Additives and Contaminants (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) for cadmium of 400-500 µg per person, approximately 7-8 µg/kg per body weight per week (bw/w) and 60-70 µg per day for a 60 kg person.

In 1993, JECFA advised a PTWI of 7 µg/kg bw/w, a level confirmed in 1995 by the European Commission and reconfirmed by JECFA in 2003.

Currently the European Food Safety Authority (EFSA) has a TWI of 2.5 µg/kg bw, this after considering a JECFA proposal for a provisional tolerable monthly intake (PTMI) of 25 µg/kg bw (not radically different from their previous PTWI of 7µg/kg bw).

Cadmium can enter cells and bind with ligands; it is not easily cleared by the body and has a long residence time (half-life 10-30 years) in organs such as liver, kidney and the intestine. Cadmium has been associated with disorders of the kidneys, bones and nervous system, and is also identified as a carcinogen.

The EU set maximum levels (MLs) for foodstuffs, including seafood, based on the results of a dietary exposure assessment and the opinions of JECFA, as noted above (Table 1). Full details of the scientific underpinning is contained in a risk assessment carried out by EFSA (EFSA. 2009).

Table 1: Maximum levels for cadmium in seafood (EFSA, 2009)

Product	**ML (mg/kg)**
Muscle meat of fish, excluding species listed below	0.05
Bonito (*Sarda sarda*) Common two-banded sea bream (*Diplodus vulgaris*) Eel (*Anguilla anguilla*) Grey mullet (*Mugil labrosus labrosus*) Horse mackerel or scad (*Trachurus spp*) Louvar or luvar (*Luvarus imperialis*) Mackerel (*Scomber spp*) Sardine (*Sardina pilchardus*) Sardinops (*Sardinops spp*) Tuna (*Thunnus spp*, *Euthynnus spp*, *Katsuwonus pelamis*) Wedge sole (*Dicologoglossa cuneata*)	0.10
Muscle meat of bullet tuna (*Auxis spp*)	0.20
Muscle meat of anchovy (*Engraulis spp*) Swordfish (*Xiphias gladius*)	0.30
Crustaceans, excluding brown meat of crab and excluding head and thorax meat of lobster and similar large crustaceans (*Nephropidae* and *Palinuridae*)	0.50
Bivalve molluscs	1.0
Cephalopods (without viscera)	1.0

Issue

The issue in the Australian context is that, within a lot of production of prawns, a proportion may contain cadmium in excess of the European Union ML (0.5 mg/kg).

Over the period 2001-2011 the EU, via its Rapid Alert system, has rejected 133 consignments of crustaceans, of which 45 were prawns and the remainder crab, on the basis of the ML.

Among the rejections were 39 consignments of Australian frozen prawns with cadmium levels up to 2 mg/kg, reflecting the bioaccumulation of the metal by prawns in certain Australian waters.

Current Status

In 2007, the Australian government made a submission to the European Commission to review the ML for crustaceans citing, as evidence for its deletion that:

- The Codex Alimentarius Commission had set no ML for cadmium in crustaceans, on the basis that crustaceans represent a minor exposure to cadmium.
- While median levels for prawns in Australia's export trade were 0.11 mg/kg, the distribution is highly skewed and a proportion can not meet the ML criterion at the 95th percentile (e.g. 5%).
- Australian prawns do not contribute significantly to the cadmium ingested by European consumers.

The status of the submission is not known.

It is noted that all 39 rejections were during the period 2004-2007 and the fact that there have been no further rejections of prawns from any European country may indicate that:

1 The EU has ceased testing prawns

2 Only prawns from low-risk waters in Australia and other countries are being exported

3 Exports from Australia to EU have been curtailed.

Data do not exist to comment on possibilities 1 and 2 (above). However, ABARE data provide clear evidence that exports to the EU have diminished over the period 2005-06 until 2008-09 (Table 2).

Table 2: Exports of prawns from Australia to the EU (2005-2009)

	2005-06	2006-07	2007-08	2008-09
Export volume (t) to EU	2,666	1,316	548	340
Total exports (t)	8,744	6,376	4,916	4,797
Proportion (%) exported to EU	30.5	20.6	11.2	7.1
Value ($,000)	39,205	17,825	7,537	3,600
Total exports ($,000)	133,923	93,563	68,624	82,180
Proportion (%) exported to EU	34.4	19.1	11.0	4.4

ABARE data, as presented in Table 2, indicate the scale of the reduction in prawn exports to the EU. Whereas in 2005-06 2,666 t of prawn exports entered the EU, by 2008-09 the volume was 340 t, the more than 2,000 t diminution contributing greatly to an overall reduction in total exports over the same period of >4,000 t.

It is noted that, while there was some appreciation of the Australian dollar against some currencies e.g. USD and Yen, no such appreciation occurred against the Euro during the period cited in Table 1.

Qualitative Risk Assessment

Product/hazard	**Cadmium in Prawns**
Severity	IA
Occurrence of illness	Low
Chronic exposure required to cause illness?	Yes
Impact of processing, handling	None
Consumer terminal step?	None
Epidemiological link?	None
Assessed risk	Low

Going Forward

There seems *prima facie* evidence that the EU maximum Limit for cadmium in crustacea has had, and continues to have, a significant effect on the prawn industry in general, and the export prawn sector in particular.

It should be determined whether exports of other crustaceans to Europe (lobsters, crabs) are similarly affected.

Engaging the EU in risk-based dialogue on ingestion of cadmium from Australian exports should be referred to AQIS as a high priority.

References

EFSA. 2009. Scientific opinion: Cadmium in food. Scientific opinion of the panel on contaminants in the food chain. EFSA Journal 908:1-39.

Hazard Sheet: Arsenic

Hazard

A provisional maximum tolerable daily intake of 2 μg/kg body weight (b.w.) for inorganic arsenic was set by the JECFA in 1983 and confirmed as a provisional tolerable weekly intake (PTWI) of 15 μg/kg b.w. in 1989. The JECFA noted that organic forms of arsenic present in seafoods needed different consideration from the inorganic arsenic, based on the low toxicity and rapid metabolism of organoarsenicals.

Both Codex (CAC, 1995) and EFSA (Anon. 2009) note that arsenic in seafoods is mainly the non-toxic forms Arsenobetaine and Arsenosugars, both of which are metabolic products of marine algae and are accumulated by herbivorous species, especially filter feeders.

Exposure to arsenic in its inorganic forms (trivalent arsenite and pentavalent arsenate) has a number of effects, the most important being bladder cancer.

Neither Codex, EFSA nor the New Zealand FSA has a standard for arsenic in seafood, though the latter has for imported Hijiki seaweed.

Issue

The Food Standards Code contains a Maximum Limit for arsenic in seafood based on a determination that seafood, particularly molluscan shellfish, contributes significantly to dietary exposure to arsenic.

Commodity	ML (mg/kg)
Crustacea	2
Fish	2
Molluscs	1

Several conflicting statements contained in ANZFA (1999b) are of relevance, particularly in those sections highlighted in bold type:

1 'The main seafoods contributing to inorganic arsenic dietary exposure (>5%) from food alone were prawns (52%) and marine fish (14%). Although other seafood such as crabs, mussels and oysters are significant sources of inorganic arsenic per kilogram of food, **the relatively small consumption levels of these foods means they do not make a significant contribution to mean inorganic arsenic dietary exposure for the whole population (ANZFA 1999b).**'

2 'Dietary exposure estimates for high consumers of single food commodity groups indicate that high fish consumers could receive up to 4 per cent of the PTDI for inorganic arsenic, and that high consumers of molluscs and crustacea could receive up to 6 per cent and 18 per cent of the PTDI for inorganic arsenic respectively, **assuming that the inorganic content of seafood is 6 per cent of the total arsenic content and assuming that these consumers eat molluscs and crustacea every day over a lifetime (ANZFA 1999b).**'

Current Status

The assertion is that the ML is too low for molluscan shellfish.

Qualitative Risk Assessment

Product/hazard	Arsenic in Molluscs
Severity	IA
Occurrence of illness	Low
Chronic exposure required to cause illness?	Yes
Impact of processing, handling	None
Consumer terminal step?	None
Epidemiological link?	None
Assessed risk	Low

Going Forward

It appears that FSANZ encountered difficulty in estimating inorganic arsenic because: *Inorganic arsenic analyses are more expensive than total arsenic analyses. To make the best use of the available funds for analytical testing, total arsenic, rather than inorganic arsenic, is determined in most cases* (20th ATDS).

An assumption on the proportion of total arsenic which was inorganic was made. However, as FSANZ state: *There is no accepted ratio that can be used for all foods to convert the total arsenic content to inorganic arsenic. For this reason and to enable comparison of the results with the tolerable limit for inorganic arsenic, it was assumed that all arsenic detected in each food was in the form of the more toxic inorganic arsenic. This is a significant overestimate because not all arsenic is present as inorganic arsenic. This is demonstrated by the presence of total arsenic at levels above the LOR in all of the seafood samples while inorganic arsenic was not present above the LOR in any of the seafood samples* (20th ATDS).

It is recommended that a re-appraisal of the ML for arsenic in seafood be made.

The Australian Seafood CRC is in the process of recruiting a new postdoctoral scientist in the area of seafood toxicology. This specialist resource may be able to be utilised to progress work in this area, via an expert solicitation.

The study should follow the process undertaken by Borak and Hosgood (2007) in assessing the implications of consumption of arsenic in seafoods. These authors state that:

- Inorganic arsenic, the cause of illness in humans subject to chronic exposure, generally comprises 0.1% and almost always <3% of total arsenic in seafood; organic arsenic compounds comprise the vast bulk of total arsenic in seafood.
- Organic arsenic compounds found in seafood are mainly arsenobetaine and arsenosugars and are non-toxic.
- A margin of exposure of 1,000-10,000-times exists between carcinogenic doses used in rodent studies and those expected after consumption of large quantities of seafood.

The authors conclude: *The general absence of arsenic toxicity reported in humans and other mammals after consumption of large amounts of seafood (Andrewes et al. 2004) lends weight-of-evidence support to its lack of acute toxicity.*

References

Andrewes, P., Demarini, D. Funasaka, K. Wallace, K. Lai, V. Sun, H. Cullen, W. and Kitchin, K. 2004. Do arsenosugars pose a risk to human health? Environmental Science and Technology 38:4140-4148.

Scientific opinion on Arsenic in food. 2010. EFSA panel on contaminants in the food chain. EFSA Journal 7(10):1351.

ANZFA. 1999. P158 the review of metal contaminants in food.

Borak, J. and Hosgood, H. 2007. Seafood arsenic: Implications for human risk assessment. Regulatory Toxicology and Pharmacology 47:204-212.

Codex Alimentarius Commission. 1995. Codex general standard for contaminants and toxins in food and feed. Codex Standard 193-1995.

FSANZ. 2002. The 20th Australian total diet survey. Canberra, ACT 2610.

Hazard Sheet: Mercury

Hazard

Based on an acute mercury food poisoning that occurred in Japan during the 1950s, it is known that high levels of dietary mercury in seafood cause measurable deficits in the mental and physical development of young children exposed during gestation. Low levels of mercury are naturally present in the environment and in all foods. Inorganic mercury is poorly absorbed *via* the diet but, in aquatic environments, bacteria can convert inorganic mercury to Methyl mercury which is readily absorbed by the human body. Methyl mercury is accumulated in aquatic food chains, so all fish contain small amounts of Methyl mercury in their muscle tissue. Predatory fish or mammals such as whales at the top of the food web have the largest amounts.

Mercury levels in most commercially-harvested oceanic fish in Australia are <0.5 mg/kg Methyl mercury, but some large predators such as sharks, marlin and swordfish may have higher levels. Numerous studies have shown that nearly all the human exposure to Methyl mercury occurs *via* seafood (predominantly finfish) consumption. Therefore individuals who regularly consume large amounts of fish (particularly those fish with high mercury levels) could be exposed to dangerous levels of mercury (FDA, 1994; National Academy of Sciences, 2000).

Issue

FSANZ have issued an advisory which allows adults, including pregnant women, to eat 2-3 serves (300-450 g) per week of low-mercury species (almost all fish except shark, swordfish, billfish and marlin).

By contrast, the NZ Food Safety Authority (http://www.foodsmart.govt.nz/whats-in-our-food/chemicals-nutrients-additives-toxins/specific-foods/mercury-in-fish/) recommends that pregnant women eat seafood:

- Without restriction – a range of named finfish, shellfish and squid
- 3-4 serves/week – wide range of finfish and lobster
- 1 serve every 1-2 weeks – shark, marlin, swordfish, bluefin tuna

The amended advice by FSANZ and NZFSA accords with recent findings that consuming <340 g seafood/week may have a detrimental effect on foetal development (Hibbeln *et al.* 2007).

By following the revised FSANZ advice of '2-3 serves' consumers may consider it prudent to defer to two, rather than three serves, an intake which has been shown to be detrimental (Hibbeln *et al.* 2007).

Qualitative Risk Assessment

Product/hazard	Mercury in Seafood
Severity	IA
Occurrence of illness	Low
Growth in product required to cause illness?	Yes
Impact of processing, handling	None
Consumer terminal step?	None
Epidemiological link?	None*
Assessed risk	Low

* The Minamata poisoning notwithstanding

Current Status

There appears to be a general move towards allowing increased intake of seafoods, based on risk:benefit considerations e.g. US FDA, Hibbeln *et al*. (2007).

Going Forward

Given the NZ FSA stance on consumption of seafood it is requested that FSANZ review their current advice, with a view to aligning with the NZ approach.

It is noted that Thompson and Lee (2009) state: *NZFSA has recently agreed with FSANZ that both agencies should investigate removing fish monitored for mercury from New Zealand and Australia's respective 'risk lists' as exposure from mercury in fish would be better managed by an education programme such as NZFSA's advisory information for pregnant women.*

The process may be facilitated by data from the CRC's compositional seafood profiles to assess the mercury content of Australian species of fish and categorise them in categories similar to the NZFSA.

References

FDA. 1994. Mercury In Fish: Cause For Concern? FDA Consumer Magazine 28(7).

Hibbeln, J. 1998. 'Fish consumption and major depression'. Lancet 352: 1213.

National Academy of Sciences. 2000. Toxicological effects of mercury. The National Academy of Sciences, USA.

Thompson, B. and Lee, L. 2009. Mercury content in imported fin fish. Institute of Environmental Science and Research Ltd. Christchurch, NZ.

Hazard sheet: Allergens

Issue

Allergens represent a hazard to susceptible consumers and in seafood comprise two categories: naturally-occurring allergens and allergens added during processing.

The allergic reaction can vary in intensity from vomiting/nausea to anaphylactic shock and subsequent collapse.

Naturally-Occurring Allergens

Consumers generally are allergic to one type of seafood e.g. crustaceans or bivalve molluscs or finfish, and are able to eat other types with safety.

For vulnerable consumers allergic reactions may occur without seafood being ingested, e.g. via aerosols from seafood being cooked.

Consumers avoid contact with seafood causing the allergy by identifying it during purchase or when ordering food in a restaurant.

Because the quantity of allergen required to cause a reaction may be very small there is the possibility of inadvertent transfer during handling and marketing of seafoods. For example, there is opportunity for cross contact to occur when categories are mixed in a container being delivered to a wholesaler.

It is unlikely that fish markets, wholesalers and processors identify allergens as hazards reasonably likely to occur in the food safety plan.

Going Forward

A review commissioned by Sydney Fish Market found an absence of evidence that cross-contact between seafood categories is a hazard reasonably likely to occur during handling at the market (Murphy, 2011).

The same report recommended that further work be done on seafood handling, distribution and processing using the VITAL (Voluntary Incidental Trace Allergen Labelling) grid to assess the impact of allergen cross contact (http://www.allergenbureau.net/vital/).

Allergens Added During Processing

Allergens may be added during value-adding operations such as crumbing or battering of seafood. If the allergen is not declared the product must be recalled, and typical examples are presented in Table 1.

Table 1: Recalls of seafood due to presence of allergens (FSANZ 1999-2011)

Year	Product	Format	Hazard	Country of origin*
2005	Whiting	Frozen, crumbed	Allergens (casein)	Vietnam
2010	Fish	Frozen, battered	Allergen (dairy)	
2011	Fish	Frozen, crumbed	Allergen (peanuts)	
2011	Calamari	Frozen	Allergen (peanuts)	
2011	Dory	Frozen, crumbed	Allergen (peanuts)	

* Where known

Control of allergens at the plant level is by identifying allergens present in premixes used for coatings and ensuring that the label reflects the presence of an allergen.

Inadvertent addition of an allergen is prevented by good manufacturing practices such as cleaning equipment and food contact surfaces between batches.

The above information should be included in the company's food safety plan.

References

Murphy, R. 2011. Sydney Fish Market – allergen risk review. Rural Development Services, Hobart, Tasmania.

Hazard Sheet: Ciguatera

Hazard

Ciguatera poisoning is caused by eating subtropical and tropical reef fish which have accumulated naturally-occurring toxins produced by marine algae. The toxins are known to originate from dinoflagellates (predominantly *Gambierdiscus toxicus*), common to ciguatera-endemic regions in tropical waters. These toxins produce a range of gastrointestinal, neurological and cardiovascular symptoms which can persist for many weeks and may be re-triggered by dietary changes or exposure to low levels of toxin, months or years after initial exposure. Ciguatera poisoning is usually self-limiting with a low incidence of death.

Issue

According to data assembled by the National Risk Validation Project and OzFoodNet, over the period 1988-2010 there were 101 outbreaks of ciguatera food poisoning in Australia involving more than 597 consumers (Part 2, Table A1), with mackerels and coral trout the most-frequently implicated species (Part 2, Table A4). By far the majority of food poisoning incidents occurred in Queensland (Part 2, Table B4).

The issue is that ciguatera food poisoning is Australia's most frequent cause of illness due to consumption of seafood, with no reduction over the period 2001-2010 (OzFoodNet data) compared with 1988-2001 (NRVP data).

Qualitative Risk Assessment

Product/hazard	**Ciguatera in Seafood**
Severity	III
Occurrence of illness	High
Growth in product required to cause illness?	No
Impact of processing, handling	None
Consumer terminal step?	None
Epidemiological link?	Yes
Assessed risk	Medium

Going Forward

In general, ciguatera poisoning is sporadic, typically involves fewer than five people and may be misdiagnosed by medical staff.

It results from consumption of fish caught recreationally and commercially and the quantum from each sector is not known.

Current risk reduction strategies include limits on the size and/or type of certain fish species and restrictions on fishing in known toxic areas. Red bass, chinaman fish and paddletail have been regarded as unsuitable for sale in Queensland for 20 years due to their likely toxicity (Lehane, 1999). Other than in Queensland and Northern Territory recreational fishermen are unlikely to catch affected fish.

In subtropical and tropical regions professional fishermen are aware of areas to avoid which helps reduce the hazard to consumers. Size restrictions are potentially effective since larger fish may be more frequently toxic than small fish but the practice of filleting into portions for on-board packing and freezing makes this impossible to monitor as a CCP.

While some fish markets claim to not sell potentially poisonous fish such as reef fish, anecdotal evidence suggests that due to inconsistent naming, potentially ciguatoxic species may be sold. It is recommended that SafeFish and Sydney Fish Market undertake a benchmarking study on levels of ciguatoxin on species linked with ciguatera.

The feasibility should be considered whereby a wholesaler/distributor/marketer requires the supplying vessel to verify that the catch has been taken from 'safe' waters. This could be done by supplying a GPS log for each trip, analogous to supplying a temperature:time log for fish vulnerable to histamine formation.

This information could become a CCP in the HACCP plan of fishers and wholesalers/distributors/marketers. Widespread adoption of the Fish Names Standard would also assist.

References

Lehane, L. 1999. Ciguatera Fish Poisoning. A Review in a Risk-Assessment Framework. National office of Animal and Plant Health, Agriculture, Fisheries and Forestry Australia.

National Risk Validation Project. 2002. Food Science Australia and Minter Ellison.

OzFoodNet Annual reports 2000-2010.

Hazard Sheet: Parasites in Seafood

Hazard

The consumption of raw seafood as sashimi (pieces of raw fish) and sushi (pieces of raw fish or cooked prawns with rice and other ingredients) are popular foods in Australia. The ingestion of raw fish carries with it a number of risks of foodborne infection, including the risk of parasitic worms that can cause gastrointestinal disease in humans.

Among parasites associated with seafood are helminths (parasitic worms) and include nematodes (roundworms), cestodes (tapeworms) and trematodes (flat worms, or flukes). Of most concern are:

- Nematodes: *Anisakis simplex*
- Cestodes: *Diphyllobothrium*
- Trematodes: *Clonorchis sinensis*

Helminth parasites are sensitive to freezing and to relatively mild heating (i.e. normal cooking temperatures). Consequently, those parasites associated with seafood are generally passed to consumers via raw, minimally processed or inadequately cooked chilled products, the latter particularly associated with socio-cultural and behavioural factors (Adams *et al*. 1997).

A wide range of seafood products have been implicated in human infection (FDA, 1999):

- Ceviche (fish and spices marinated in lime juice; Latin America)
- Lomi lomi (salmon marinated in lemon juice, onions and tomato; Hawaii)
- Poisson cru (fish marinated in citrus juice, onions, tomatoes and coconut milk)
- Salmon roe
- Ako poki (Japanese and Hawaiian cephalopod dish)
- Sashimi (pieces of raw fish; Japan)
- Sushi (pieces of raw fish with rice and other ingredients; Japan)
- Green herring (lightly brined herring; Netherlands)
- Scandinavian gravlax
- Drunken crabs (crabs marinated in wine and peppers; China)
- Cold-smoked fish and undercooked grilled fish

Comprehensive lists of parasites that have been reported in Australian fish are presented in Beumer *et al.* (1982), Lester and Sewell (1989), Doupe *et al*. (2003) and Shamsi *et al*. (2010).

Data on prevalence of helminths in Australian species are presented in Part 2 of this report.

Current Status

There is one published record of anisakid infection in Australia (Shamsa and Butcher, 2011) in which a 41-year-old Australian woman of Tongan descent consumed raw mackerel.

In 2010, EFSA published a scientific opinion on risk assessment of parasites in fishery products and concluded that, while wild-caught fish can never be guaranteed not to have helminth parasites, the likelihood in farmed fish is much lower.

Of relevance in a listing of farmed fish, a number of species were identified which are both consumed in Australia, and exported in a chilled format, making them potential sources of infection. These species include Atlantic salmon, Pacific salmon, rainbow trout and tuna, to which should be added the farmed species Australian Kingfish (*Seriola lalandi*) and Mulloway (*Argyrosomas hololepidotus*).

In a survey of five South Australian species, Shamsi *et al*. (2010) did not detect anisakid larvae in ten samples of wild caught *S. lalandi*.

Qualitative Risk Assessment

Product/hazard	**Parasites in Seafood Consumed Raw**
Severity	II
Occurrence of illness	One case reported in Australia
Growth in product required to cause illness?	No
Impact of processing, handling	Freezing inactivates parasite
Consumer terminal step?	No
Epidemiological link?	Not in Australia
Assessed risk	Low

Going Forward

There has been activity recently where importers of Australian farmed species are being directed to a European Union directive (E.C. 853/2004) which, in Annex III, Section III, Chapter III, Paragraph D states:

D. REQUIREMENTS CONCERNING PARASITES

1. The following fishery products must be frozen at a temperature of not more than -20 °C in all parts of the product for not less than 24 hours; this treatment must be applied to the raw product or the finished product:

(a) fishery products to be consumed raw or almost raw;

(b) fishery products from the following species, if they are to undergo a cold smoking process in which the internal temperature of the fishery product is not more than 60 °C:

(i) herring;

(ii) mackerel;

(iii) sprat;

(iv) (wild) Atlantic and Pacific salmon;

and

(c) marinated and/or salted fishery products, if the processing is insufficient to destroy nematode larvae.

2. Food business operators need not carry out the treatment required under paragraph 1 if:

(a) epidemiological data are available indicating that the fishing grounds of origin do not present a health hazard with regard to the presence of parasites; and

(b) the competent authority so authorises.

3. A document from the manufacturer, stating the type of process they have undergone, must accompany fishery products referred to in paragraph 1 when placed on the market, except when supplied to the final consumer.

The reporting of the first case of anisakidosis in Australia, together with the position adopted by European countries prompts some investigation of parasites in seafoods.

This is an issue which requires a watching brief to monitor whether rigorous application of the EU directive is having a deleterious impact on Australian trade.

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Issues Sheet: Product Testing

Two issues are identified by SafeFish partners:

Issue 1: Biotoxin Testing of Bivalves

No biotoxin testing facility in Australia is accredited to diagnose marine biotoxins currently important in domestic and international trade. Around 30% of all diagnostic tests of Australian seafood (wildcatch and aquaculture) are undertaken in New Zealand by the Cawthron Institute.

Current Status

A recent report by Ridge Partners to the Seafood CRC recommends that a National Marine Biotoxin Centre be set up in Sydney; such a venture will allow all biotoxin testing to be done in Australia.

Issue 2: US Equivalence of Bivalve Testing

South Australian shellfish harvesters wish to export live product (oysters and mussels) to the USA, but this is impeded by the lack of a Memorandum of Understanding (MoU) between governments.

Current Status

In 2009, an audit of intending exporters was undertaken by SARDI in conjunction with Dorothy-Jean & Associates Ltd to advise on changes required to be able to meet USA requirements.

The report found that:

1 The SASQAP (SA Quality Assurance Program) is of a suitable status to advance an application to the USFDA for market access for whole, live shellfish.

2 The South Australian shellfish growing area would meet the general requirements for the USA National Shellfish Sanitation Program (NSSP) with some minor adaptations.

3 There are some hurdles that will need to be addressed to reach a successful MOU.

The recommendations were that:

1) AQIS must be advised of the industry request for market access of live bivalve molluscan shellfish to the USA. AQIS will be the primary Competent Authority and will play a central role in the Memorandum of Understanding arrangements with the USDFDA.

2) As there are multiple agencies involved in the administration of the South Australian shellfish quality assurance programme there will need to be good co-operation between the different agencies. There will also need to be evidence of written memoranda of

agreement with the agencies which share the programme responsibilities.

3) AQIS will likely need to provide to the USFDA a 'side by side' review of the Australian and USA shellfish food safety legislation.

4) A Central File will need to be established by AQIS that holds all the required programme records.

5) There needs to be training and appointment of a Shellfish Standardisation Officer for Australia who will then inspect any processing plants to be used for export of shellfish to the USA.

6) All premises used for processing shellfish must be listed on the Interstate Certified Shellfish Shippers List and this list must be maintained by AQIS.

7) Any laboratory used to support the shellfish programme will need to comply with the requirements of the NSSP manual and be supervised by a designated Laboratory Evaluation Officer. (Note the scope of this audit did not include Laboratory compliance).

8) South Australia currently uses a microbiological method for faecal coliforms in seawater that has not yet been approved by the NSSP. Equivalency will need to be negotiated with the USFDA.

9) Management plans for conditionally approved areas must be very specific in defining who, what and when environmental parameters are monitored to officially open and close a conditionally managed harvest area.

10) PIRSA shall record any polyculture activities at a growing area (more than one species on the farm). This information may be required for individual species management criteria.

11) The NSSP programme has specific patrol requirements. A Risk Management Plan for surveillance must be drafted for the growing areas exporting to the USA to justify the current surveillance programme (See Chapter VIII, Section B of the NSSP MO).

12) All harvesting vessels must have adequate toilet and hand sanitizing facilities on board.

13) On licensing harvest operators some public health information must be provided to them on the hazards with harvesting shellfish from closed areas and on the significance of discharging human sewage overboard.

14) Tagging of product for product from harvest area to dealer must be in compliance with Chapter VIII .02 F of the NSSP. Note that bulk tagging is acceptable provided that there is also a transaction record provided to the dealer.

15) There are specific HACCP and sanitation records required by the NSSP. These include mandatory CCPs, adequate training and weekly sign off on records. The NSSP is prescriptive in its sanitation requirements, for example wash hand basins must be supplied with hot water greater than 43 degrees Celsius. The Shellfish Standardization Officer will need to review the premises, process operation and records to assess full compliance with these requirements.

16) There will be specific requirements for labelling, temperature control storage and shipping conditions to the USA. However, it was not possible at this time to review likely compliance with these requirements. AQIS will need to verify these before certifying product for export.

17) This audit focused on the USA food safety requirements for live bivalve molluscan shellfish. However, it should be noted that there may also be USA biosecurity requirements for the entry of live animals. This will need to be referred to AQIS for consideration.

Going Forward

This centres on the feasibility of AQIS incorporating the necessary work into their program to complete a MoU with USA. This will provide the potential for all Australian shellfish harvesters and processors to export to the market. Consideration could be given to SafeFish technical support to assist AQIS to reignite the MoU.

Issues Sheet: Potential Regulation of Cyclic Imines in Shellfish

Issue

Cyclic imines (CIs) comprise a family of marine biotoxins made up of spirolides, gymnodimines, pinnatoxins and pteriatoxins. The toxins cause neurotoxic symptoms in mice.

The issue in Australia concerns pinnatoxins and devolves around the fact that:

- A dinoflagellate capable of synthesising pinnatoxins has been confirmed in Australian and New Zealand oyster-growing waters
- Pinnatoxins were detected in oysters in South Australia in 2007, leading to a protracted closure of the fishery
- Acute toxicity of pinnatoxins has been demonstrated in mouse tests
- The possibility that the European Commission may impose a regulatory limit for CIs.

Current Status

In 2010, the European Food Safety Authority (EFSA) produced a scientific opinion on cyclic imines which declared, in summary, that:

- There have been no reports linking pinnatoxins to poisoning events in humans. Note, however that pinnatoxins were linked with consumption of razorshells in China at a workshop in South Australia (Rhodes *et al*. 2010).
- No country has a regulatory limit for CIs in shellfish.
- The acute toxicity of CIs has been established.
- No long-term studies exist to allow the establishment of a tolerable daily intake (TDI).
- No conclusions could be drawn on risk to consumers following consumption of pinnatoxins.
- Further method development is required for detection of CIs.
- More information is needed on occurrence of CIs in shellfish.

Going Forward

It is noted that the program for the 8th International Conference on Molluscan Shellfish Safety in June 2011 has a paper by Hess: First report of pinnatoxin in mussels and a novel dinoflagellate, *Vulcanodinium rugosum*, from France.

This report may provide EFSA with impetus to revisit the regulation of pinnatoxins and it is recommended that a watching brief be maintained.

References

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pteriatoxins). EFSA Journal 2010; 8(6):1628. [39 pp.]. doi:10.2903/j.efsa.2010.1628. available online: www.efsa.europa.eu

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Issues Sheet: Norovirus Trade Issues (Hong Kong and Singapore)

Issue

For some years, Hong Kong has been concerned about illness caused by consumption of raw bivalve molluscs. In 2000-2002 Cheng *et al*. surveyed oysters imported from 11 countries and found norovirus in 10.7% of the 507 samples investigated.

In September and October 2006, Hong Kong authorities identified oysters imported from Chile as the probable cause of an outbreak of norovirus.

In Hong Kong, the Centre for Food Safety:

- Requires that suppliers provide a health certificate issued by the controlling authority of the country of origin certifying the food is fit for human consumption
- Began testing imported shellfish for norovirus nucleic acid.

Health certification provided by AQIS for exporting bivalve molluscs to Hong Kong should certify that:

- The bivalve molluscs were collected or harvested from sanitary waters, which have not been polluted; or the molluscs have been cleansed by relaying in clean water for (state number) days or the molluscs have been cleansed in the approved shellfish purification plant at (address of plant).
- The molluscs were packed under hygienic conditions.
- The molluscs do not contain any substance or substances, including biotoxins, contaminants like pesticides, trace metals, etc. in such amount as to be poisonous, harmful or injurious to health.
- The molluscs are fit for human consumption and can be sold as food for human consumption in (state country of origin).

In Singapore the Agri-Food and Veterinary Authority (AVA) issued a circular to importers *New requirement of pre-export norovirus testing for frozen oysters exported to Singapore* to the effect that, from July 1, 2007, *the competent relevant government authority of the exporting countries will certify in the health certificate that the consignment of frozen oysters has been tested and found free of norovirus.*

Current Status

It is stated that consignments to Singapore and Hong Kong are held because of the presence of norovirus nucleic acid in consignments of oysters.

There is uncertainty whether the analysis is capable of determining whether the product under investigation constitutes a public health risk. The methodology does not allow the quantification of norovirus particles capable of infecting a consumer, merely identifies the presence of nucleic acid.

There are a number of current positions being developed on viral contamination of bivalves which are likely to:

- Extend the context from a Hong Kong/Singapore position to a global one

- Intensify current difficulties with methodologies surrounding opening and closing of harvest areas because of viral build-up in bivalves

These developments include, in chronological order during 2010:

1 Codex have published draft guidelines at Step 3 in which *inter alia*, the testing of bivalves for norovirus and HAV should be undertaken as a prerequisite for opening the harvesting area.

2 The Biohaz panel of the EFSA has decided to initiate a self-tasking issue with the purpose to provide up-to-date information on the present knowledge on the occurrence and control of food- and water-borne viruses. EFSA requests the BIOHAZ Panel by 31/12/2011:
   - To carry out a review of the available information in the scientific literature with regards to the biology, epidemiology also including ecological aspects, diagnosis and public health importance of food- and waterborne viruses. Where possible the review will cover primary production, food harvesting, food processing, and storage/retail until consumption. Data needs to support a risk assessment will also be identified.
   - To identify possible control options and their anticipated impact to reduce the number of food- and water-borne viral human infections.
   - To discuss the scientific reasons for and against the establishment of food safety criteria for viruses for certain food categories (e.g. fresh produce, bivalve molluscs etc).

3 The Food Safety Authority of Ireland has requested EFSA to provide an opinion on:
   - Use of real-time PCR as a means of detecting and quantifying norovirus in oysters
   - A safe limit for norovirus genogroups GI and GII in oysters as determined by real-time PCR (e.g. copy number per gram)
   - Treatment regimes that can be relied upon to reduce norovirus counts in oysters

Going Forward

Over recent years, the development of reverse transcriptase polymerase chain reaction methods (RT-PCR) and nucleotide sequencing methods allows detection and identification of viruses in faecal specimens and in shellfish.

Noroviruses can be quantified by determining the amount of norovirus RNA extracted from the shellfish sample and expressing the result as RT-PCR copies per gram. In NZ, for example, the technique has been used to monitor norovirus in shellfish harvested near an ocean sewage outfall; prevalence was 89.2% and concentration ranged to >10,000 norovirus RT-PCR units/g of shellfish (Greening *et al*. 2009).

A review of management options related to the detection of foodborne viruses in shellfish, currently being undertaken by McLeod and Kiermeier, raises the possibility of an acceptable level of norovirus particles in shellfish.

When completed, this review may be useful in determining whether methodology currently used by Hong Kong and Singapore is rejecting consignments of imported frozen oysters with concentrations which will not cause infection.

Based on the actions being undertaken by EFSA and by Codex it seems likely that testing of shellfish for viruses (NoV and HAV) may become a legislated reality in the short to medium term.

These developments prompt the strong recommendation that significant effort be expended on the development of methods that better reflect the infectivity of the virus. To this end, it is noted that there is a proposal for a collaborative project involving Australian and New Zealand scientists to improve the management of the risk of human enteric viruses in shellfish at harvest.

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Issues Sheet: Monitoring Indicators of Faecal Pollution in Bivalve Molluscs

Issue

At the Annual General Meeting of the Australian Shellfish Quality Assurance Advisory Committee (ASQAAC) a paper by Ogburn and White (2009) was discussed. The paper stated that in the NSW oyster industry:

- Indicators of faecal pollution are monitored both in growing waters and in muscle meat.
- An analysis of data from sites in three estuaries (Manning River, Port Stephens and Hawkesbury River) over the period 2000-2005 indicated that testing waters for faecal indicators correlated well with rainfall, salinity and temperature while that for testing muscle did not.
- It might be possible to maintain public health standards while minimising unnecessary disruptions and costs in the trade of fresh oysters.

Current Status

Two independent reviews were commissioned of the Ogburn-White paper by the NSW Food Authority.

A review by Dorothy-Jean & Associates Ltd, while in general agreement with the main findings of the paper, states that:

- Microbiological testing is an adjunct to the primacy of the sanitary survey.
- Neither testing water for faecal coliforms nor muscle for *E. coli* is a reliable indicator of the major hazard of public health significance – viruses.
- A result for faecal coliforms/100 mL in growing waters is not directly comparable with one for *E. coli*/100 g in muscle.
- It may be desirable to monitor both waters and muscle to allow export to the USA and European markets, respectively.

A review by Brenda Hay of AquaBio Consultants Ltd while canvassing points already covered in the review, above, raises a number of aspects about the design of the study, the interpretation of its results and the conclusions reached by the authors.

Going Forward

Given the breadth and depth of generally unfavourable comments raised in the Hay review it is important that Ogburn and White receive the opportunity to a rebuttal.

The process of review and rebuttal might be expedited by allowing authors and reviewers to present their views to the ASQAAC.

Taken together, the exercise would inform controlling authorities on whether they should emulate the conservative approach taken by the NSW Food Authority, a regulatory approach perhaps understandable given the public health problems associated with oysters in the State (Table 1).

Table 1: Selected outbreaks of viral illness linked with oyster consumption

Year	Origin	Agent	Cases	Reference
1978	Georges River	Norovirus	>2000	Kraa 1990a,b; Kraa 1995
1989	Georges River	Norovirus	370-412	Kraa 1990a,b; Kraa 1995
1990	Georges River	Norovirus	461-752*	Kraa 1990a,b; Kraa 1995
1996	Terranora	Norovirus	97	Stafford et al. 1997; Dalton 1997
1997	Wallis Lake	HAV	467	Conaty et al. 2000

* Includes 18 cases from Darwin (Ruben *et al.* 1992)

References

Conaty, S. Bird, P. Bell, G. 2000. Hepatitis A in New South Wales, Australia from consumption of oysters: the first reported outbreak. Epidemiology and Infection 124:121-130.

Dalton, C. 1997. An outbreak of Norwalk virus gastroenteritis following consumption of oysters. *Communicable Disease Intelligence* 21:321-322.

Kraa, E. 1990a. Oyster related food poisoning. NSW Public Health Bulletin 1:11.

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Ogburn, D. and White, I. 2009. Evaluation of fecal pollution indicators in an oyster quality assurance program: application of epidemiological methods. Journal of Shellfish Research 28:263-271.

Ruben, A. Ralston, A. Merianos, A *et al*. 1992. Investigations of an outbreak of food poisoning in Darwin. Communicable Disease Intelligence 16:278-279.

Stafford, R., Strain, D., Heymer, M., Smith, C., Trent, M. and Beard, J. 1997. An outbreak of Norwalk virus gastroenteritis following consumption of oysters. Communicable Disease Intelligence 21:317-320.

Issues Sheet: Vibrios in Australian bivalves

Issue

In 2001, a joint FAO-WHO panel assessed risk under the general heading *Vibrios in Seafoods*. The panel was convened in the wake of large outbreaks following consumption of oysters in the USA in the late 1990s. The FAO-WHO panel adopted a wide-ranging approach which aligned with the brief *Vibrios in seafoods* and published as FAO reports:

- Risk assessment of *V. vulnificus* in raw oysters (2005)
- Risk assessment of choleragenic *V. cholerae* 01 and 0139 in warm water shrimp in international trade (2005)
- Risk assessment of *V. parahaemolyticus* in raw oysters (in press)

Members of the FAO-WHO panel were also part of a USA panel working on *Quantitative risk assessment on the public health impact of pathogenic Vibrio parahaemolyticus in raw oysters*, published in 2005 (Anon. 2005). As a result, the USA model was used by the FAO-WHO panel as a default model. However, when data were obtained from various countries, particularly Australia and New Zealand, it was found that the USA greatly overestimated estimated cases oyster-borne illness caused by *V. parahaemolyticus*, overestimates which were corrected in the final draft.

Current Status

Recently, Codex has considered using the FAO-WHO models to provide estimates on whether implementing microbiological criteria for vibrios in bivalves might reduce illness. To this end the 42nd session of Codex Committee on Food Hygiene noted that:

Following the request of the 41st session of the Committee to address a number of issues relating to predictive risk models and testing methodology for Vibrio parahaemolyticus and Vibrio vulnificus in seafoods, it was noted that JEMRA had implemented an Expert Meeting to address these issues in September 2010. Direct replies to the requests of the Committee were provided in CX/FH 10/42/3 and the Representative highlighted the need for further guidance from the Committee on the next steps to be taken.

With regard to the future work on Vibrio spp. in seafood, the Delegation of Japan highlighted the importance of continuing with this work but considered that the next step should focus on methodology and data collection. This approach was supported by several other Delegations. In noting these recommendations, the Representative of FAO stated that there was potential to use existing frameworks such as those provided by the Global Foodborne Infections Network (GFN) 4 to facilitate this work. However, this would be a resource intensive activity, which would require support and resources from member countries as well as FAO and WHO, particularly those countries with a high level of expertise in this area. In addition, it was noted that some of the proposed aspects such as method validation were outside the remit of FAO and WHO and so could not be addressed as proposed. In light of these discussions, the Committee recommended that FAO and WHO continue with this work in the following manner:

***Step 1:** Provide recommendations on a range of test methods for quantifying V. parahaemolyticus (total and pathogenic (e.g. tdh+, trh+)) and V. vulnificus in seawater and bivalves and facilitate performance evaluation of the proposed methodologies;*

Step 2: *Develop data collection strategies (that would facilitate the collection of data) by countries to support the modification/development of models with a broader scope than those which currently exist;*

Step 3: *Encourage the collection of data in different regions, in different bivalve species and for geographically diverse strains of pathogenic V. parahaemolyticus and V. vulnificus according to the data collection strategy and using recommended test methods; and*

Step 4: *To modify/develop risk assessment models that could be used to address a range of risk management questions in a number of different regions and products, when adequate data becomes available.*

At issue is that use of the FAO-WHO models may not describe correctly the microbiological status of Australian oysters, particularly via the marketing chain.

Going Forward

It is important to be part of the Codex proposals captured under Steps 1-4 (above). In particular, there is a data gap on prevalence of *V. parahemolyticus*, in general, and of toxigenic strains in particular.

It is proposed that SafeFish both remain in contact with CCFH and undertake benchmarking studies on Australian bivalves, focusing on *V. parahemolyticus* and *V. vulnificus*.

References

Anonymous. 2005. Quantitative Risk Assessment on the Public Health Impact of Pathogenic *Vibrio parahaemolyticus* In Raw Oysters. Center for Food safety and Applied Nutrition Food and Drug Administration; US Department of Health and Human Services, Washington, USA.

Drafting group. Risk assessment of *Vibrio vulnificus* in raw oysters. FAO/WHO Microbiological risk assessment series 8 (2005).

Drafting group. Risk assessment of choleragenic *Vibrio cholerae* O1 and O139 in warm-water shrimp in international trade. FAO/WHO Microbiological risk assessment series 9 (2005).

Drafting group. Risk assessment of *V. parahaemolyticus* in raw oysters (in press).

TOR 6: Create criteria and score or rank technical trade issues on basis of consequence and likelihood

Ordering the twelve technical trade issues from 1-12 based on 'criteria and score or rank' proved difficult.

Criteria considered, and rejected, included:

- Financial impact

  Against this criterion was that a small sector would always be at the end of a long queue e.g. establishing an MoU for a handful of SA bivalve companies.

- Ability of the responsible agency to undertake work in a timely fashion

  However, because several issues are within the purview of FSANZ and AQIS, agencies which have established work programs and might not have the resources to respond in a timely manner.

- Immediacy – 'picking winners'

  While at first sight attractive, for every winner there are losers and without objective criteria, this was not considered suitable for prioritising purposes.

Ultimately it was decided to not fulfil the black letter requirements of the TOR, but rather to set out a framework by which each issue could be progressed. In adopting this approach it was necessary to ignore ratings previously assigned in favour of a *de novo* approach.

Perceived advantages of this approach include:

- Every issue has an equal chance of being progressed – no sector need be left behind
- Key agencies such as FSANZ and AQIS, while having active engagement, would be spared the onerous tasks of organising and progressing resolution of each issue
- Where appropriate, industry e.g. SSA and ASQAAC would lead resolution of a specific issue
- Research agencies would lead projects in which expert solicitation or laboratory research were required

Accordingly, issues are arranged under three broad headings:

- Area 1: Heavy metals in Australian Seafoods
- Area 2: Watching Brief
- Area 3: Market Access

Area 1: Heavy Metals in Australian Seafoods

The three issues within this area can be assessed by one technical panel, which will prepare technical material suitable for the activating agency to consider.

Mercury

Requirement	Review advice re consumption to align with NZ advice
How achieved	Advice from expert panel
Technical input	Data from CRC survey will allow segmenting of species Exposure assessment required
Lead organisation	SafeFish
Activating agency	FSANZ

Arsenic

Requirement	Review ML for molluscs
How achieved	Advice from expert panel
Technical input	Need work on inorganic:organic arsenic in bivalves. Exposure assessment required
Lead organisation	SafeFish
Activating agency	FSANZ

Cadmium

Requirement	Provide evidence for AQIS to consider
How achieved	Advice from expert panel
Technical input	Data from CRC survey
Lead organisation	SafeFish
Activating agency	AQIS

Area 2: Watching Brief

Allergens

Requirement	Impact of allergens in seafoods
How achieved	Attention to literature and electronic food safety sites such as FS Net and UTas Food Safety Centre (Seafood News for Today). SSA subscribes to VITAL (Voluntary Incidental Trace Allergen Labelling) grid.
Technical input	Brief report six-monthly or as necessary
Lead organisation	Seafood Services Australia
Activating agency	Seafood Services Australia

Potential regulation of cyclic imines (e.g. pinnatoxins) in shellfish

Requirement	Remain apprised of positions taken by EFSA
How achieved	Attention to EFSA website
Technical input	Update report as necessary
Lead organisation	SafeFish
Activating agency	SafeFish

Faecal pollution indicators

Requirement	Resolution of issues raised in publication by Ogburn and White
How achieved	Expert solicitation and workshop
Technical input	Material prepared by lead speakers
Lead organisation	ASQAAC
Activating agency	State regulators via ASQAP

Parasites

Requirement	Knowledge of distribution of parasites in Australian seafoods
How achieved	Keep up to date with Australian and international literature
Technical input	Safefish literature review and annual report
Lead organisation	SafeFish
Activating agency	SafeFish

Area 3: Market access

Shellfish to USA

Requirement	Address all issues identified in gap audit to enable an MoU with US FDA for export of live bivalves to be regenerated
How achieved	Industry assist AQIS to prepare all background materials required for MoU SafeFish recommends SAF prioritise as number one issue
Technical input	Materials to service all recommendations identified in gap audit
Lead organisation	Seafood Access Forum
Activating agency	AQIS

Ciguatoxin

Requirement	Reduction in number of ciguatera poisonings
How achieved	Validate a risk management system
Technical input	SSA investigate epidemiology (OzFoodNet data) SafeFish and Sydney Fish Market undertake survey of market fish susceptible to accretion of ciguatoxin
Lead organisation	SafeFish
Activating agency	SafeFish

Virus in bivalves

Requirement	Reducing burden of illness from viruses in bivalves
How achieved	Advice for risk managers
Technical input	Specific guidance on estimating the impact of pollution sources
Lead organisation	SafeFish
Activating agency	State Shellfish Quality Assurance programs (SQAPs)

Product testing

Requirement	Resolution on need for an Australian laboratory for testing bivalves
How achieved	ASQAAC to schedule process for resolution
Technical input	Report by Ridge Partners
Lead organisation	ASQAAC
Activating agency	ASQAAC

Review of import testing

Requirement	Review of import testing
How achieved	Expert panel to consider risk basis for import testing
Technical input	Confirm scientific underpinning for 'Risk' category
Lead organisation	FSANZ
Activating agency	FSANZ and AQIS

Vibrios in bivalve molluscs

Requirement	Assessment of vibrios in bivalve molluscs in Australia
How achieved	Laboratory program on *V. parahaemolyticus* and *V. vulnificus* in bivalves
Technical input	National temporal survey of vibrios in bivalves to validate the FAO-WHO models intended to be used to inform risk management options for different countries.
Lead organisation	SafeFish
Activating agency	State Shellfish Quality Assurance programs (SQAPs)

TOR 7: Present findings to a stakeholder meeting (with SafeFish partnership members) and give partnership members input into the final rankings

Conduct of the meeting

A draft report covering TORs 1-6 was distributed to stakeholders prior to a meeting held on June 3, 2011 in Canberra; attendees are listed in Table 1.

Table 1: Attendees at SafeFish food safety meeting (Canberra, June 3, 2011)

Cath McLeod	SARDI Food Safety
Duncan Lash	MAF New Zealand
Anthony Zammit	NSW Food Authority
Mark Boulter	Sydney Fish Market
Ann Backhouse	Codex Australia
Lynda Feazey	AQIS
John Dawson	Oyster grower, NSW
Natalie Dowsett	SARDI Food Safety
Dean Merrilees	AQIS
Colin Freeman	Pristine Oysters, Coffin Bay and others
Mark Schipp	AQIS
Jayne Gallagher	Seafood CRC
Mark Tamplin	University Tasmania
Tom Ross	University Tasmania
Ted Loveday	Seafood Services Australia
Ewan Colquhoun	Ridge Partners
Ben Daughtry	FSANZ
Beatrice Dias-Wanigasekera	FSANZ
Alison Turnbull	DHHS Tasmania
Paw Dalgaard	Technical University of Denmark
Steve Hathaway	MAF New Zealand
Dean Lisson	Abalone Council Australia Ltd
John Sumner	M&S Food Consultants Pty Ltd
Andrew Pointon	SARDI

The meeting considered the draft report and incorporated amendments into the final report.

Recommendations for future work

A number of issues were proposed for the future SafeFish program.

Australia's delegate to the 31st session of the Codex Committee on Fish and Fishery Products (Tromso, 2011) proposed that SafeFish schedule work on:

1. Screening of methods for biotoxins
2. Draft standard for fresh/live and frozen abalone
3. Public health significance of histamine in fish and fishery products
4. Food additives

In addition, a watching brief was recommended on the:

1. FAO report on *Salmonella* in bivalves
2. Review of the draft standard for smoked fish and draft code of practice for fish and fishery products
3. Review of the draft standard for quick frozen scallop adductor meat, including an expanded scope to add scallop with roe-on.

Details are provided in the delegate's report to SafeFish.

The Seafood CRC recommended that a risk assessment be undertaken on the effect of sulphite in canned abalone and prawns, with a focus on the Chinese market.

Representatives of the NZ Food Safety Authority (NZFSA) commented that parallel research was being carried out in Australia and NZ, and suggested that there were synergies to be obtained from cooperation between the NZFSA and the SafeFish scientific work program.

There was general agreement that synergies should be pursued.

The proposed work (above) is additional to the list of issues for SafeFish as lead agency re. follow-up work and/or keeping a watching brief:

- Heavy metals
- Cyclic imines/parasites
- Parasites
- Ciguatoxin
- Viruses in bivalves
- Vibrios in bivalves

Given the number and scope of projects directed to SafeFish, consideration will need to be given to resource allocation to permit this list of projects to progress.

Hazards Affecting Australian Seafood

Part 2:
Supporting Information

John Sumner

May 2011

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Section A: Appendices

Appendix 1: Food Poisonings in Australia due to Seafood (1988-2010)

There are two databases which provide information on illness associated with consumption of seafood. Food Science Australia and Minter Ellison Consulting published the National Risk Validation Project (NRVP) in 2002, which assembles an exhaustive database of food poisonings in Australia over the period1988-2001. In 2001, OzFoodNet began collating data on food poisoning outbreaks on a state-by-state basis.

In Table A1 are presented NRVP and OzFoodNet data on 209 outbreaks of food poisoning following consumption of oysters over the 23-year period 1988-2010. During the period, more than 3200 individuals became sufficiently ill to enter the medical system and become registered cases, of whom seven died.

Table A1: Hazards associated with seafoods implicated in food poisonings 1988-2010

	NRVP data (1988-2001)		OzFoodNet data (2001-2010)	
Hazard	**Outbreaks**	**Cases (deaths)**	**Outbreaks**	**Cases**
Ciguatera	36	>314	65	>283
Histamine	11	54	27	94
Shellfish poison	3	117	0	-
Waxy esters	1	>14	7	>84
Viruses	16	1864 (1)	7	135
Salmonella	0	-	5	60
S. aureus	0	-	1	3
Vibrio spp	8	8 (6)	1	3
L. monocytogenes	1	3	0	0
Unknown	0	-	20	151
Total	**76**	**2374**	**133**	**>812**

There were striking differences in major causes of illness during both periods:

- Viral illness fell significantly and while oysters from NSW waters were responsible for illness during 1988-2001, imported shellfish (predominantly frozen oysters) caused most of the illnesses in 2001-2010.
- There were no illnesses due to shellfish poisons during the second period.
- More outbreaks and illnesses occurred due to consumption of fish containing waxy esters in 2001-2010.
- The reduction in oyster-associated illness reflects the implementation of shellfish quality assurance programs by all states.
- Ciguatera and histamine poisoning remained important causes of illness during both periods, with Queensland being responsible for most cases of the former (Table A2).
- By far the largest number of outbreaks occurred from consumption of fish containing ciguatoxin; species are listed in Table A3.

- Histamine poisonings were second most numerous and though some obviously stemmed from imported products, a number were probably caused by temperature abuse of product caught domestically.
- The third most numerous category is 'Unknown' which accounted for 20 outbreaks, of which nine were associated with bivalve shellfish (suspect norovirus), one with tuna (suspect histamine) and one with Spanish mackerel (suspect ciguatera).
- Although imported bivalves appear to be responsible for some norovirus outbreaks it is possible some are of domestic origin.
- Waxy esters from Escolar and Rudderfish have also caused outbreaks.
- While there have been no illnesses associated with *L. monocytogenes*, salmon smoked domestically has been recalled.

Table A2: State of origin of seafoods associated with outbreaks 2001-10 (after OzFoodNet)

State	Number of outbreaks
Queensland	64
New South Wales	36
Victoria	16
Northern Territory	5
Western Australia	4
ACT	5
Tasmania	2
South Australia	1
Total	**133**

Table A3: Species associated with ciguatera outbreaks (after OzFoodNet)

Species	Number of outbreaks
Mackerel	22
Coral trout	10
Kingfish	7
Barracuda	3
Other	23
Total	**65**

A full listing of hazards and outbreak settings is presented in Tables B1-B4.

Appendix 2: Recall of Seafood in Australia (1999-2011)

In Table A4 are presented recalls of seafood over the period 1998-2011. Of the 28 recalls, the country of origin was recorded for eleven, eight of which were Asian. It is probable, based on the product and/or format, that most of the remainder were imported e.g. *P. monodon* and *P. vannamei* are likely imports as are *Tilapia* and anchovy.

Table A4: Recalls of seafood listed by FSANZ (1999-2011)

Year	Product	Format	Hazard	Country of origin*
1998	Trevally		May be puffer fish	
1999	Mussels	Smoked	*L. monocytogenes*	
1999	*Penaeus monodon*	Cooked peeled	*V. cholerae*	Thailand
2000	Queenfish	Fillets	Ciguatera	
2001	Mackerel	Dried	Histamine	Philippines
2002	Crab	Frozen, salted	Chemical contamination	China
2003	Mackerel	Salted	Histamine	Thailand
2004	Salmon	Smoked	*L. monocytogenes*	Denmark
2004	Mackerel	Fillets in oil	Histamine	
2004	*Penaeus monodon*	Cooked	*S.* Infants	
2005	Whiting	Frozen, crumbed	Allergens (casein)	Vietnam
2005	Mackerel	Fillets	Ciguatera	
2005	Anchovy	Canned	Histamine	Greece
2005	*Penaeus vannamei*	Cooked	Microbial contamination	
2006	Clams	Canned	Under processing	China
2007	Herring	Marinated	Glass	Sweden
2007	*Penaeus monodon*	Cooked	Metal	
2007	Prawns	Raw, peeled	Labelling (states cooked)	
2008	Tuna	Frozen steaks	Histamine	Indonesia
2009	Anchovy	Frozen	Histamine	
2010	Fish	Frozen, battered	Allergen (dairy)	
2010	Tuna salad	Chilled	*L. monocytogenes*	
2010	Tilapia	Frozen, fillets	Chemical (antibiotics)	Vietnam
2011	Fish	Frozen, crumbed	Allergen (peanuts)	
2011	Trout	Smoked	*L. monocytogenes*	
2011	Calamari	Frozen	Allergen (peanuts)	
2011	Dory	Frozen, crumbed	Allergen (peanuts)	
2011	Salmon	Smoked	*L. monocytogenes*	

* Where known

Section B: Background Tables

Background Data: Outbreaks of Food Poisoning Attributed to Seafood

Table B1: Outbreaks of food poisoning due to consumption of oysters (after NRVP, 2001)

Food Business	Pathogen	Cases (deaths)	Year	Reference
Caterer	Norovirus	>32	1990	NSW Health
Caterer	Norovirus	>48	1990	NSW Health
Caterer	Norovirus?	>19	1990	NSW Health
Caterer	Norovirus	30	1990	NSW Health
Caterer	Norovirus	>11	1990	NSW Health
Caterer	Norovirus	>39	1990	NSW Health
Caterer	Norovirus	18	1992	Ruben *et al.* 1992
Caterer	Norovirus, *V. parahaemolyticus*	>148	1990	NSW Health
Caterer	Norovirus	>25	1990	NSW Health
Caterer	Norovirus	>17	1990	NSW Health
Caterer	Norovirus	>74	1990	NSW Health
Community	*V. vulnificus*	1	1989	NSW Health
Community	*V. vulnificus*	(1)	1991	NSW Health
Manufacturer	*L. monocytogenes*	3	1991	Mitchell, 1991; Misrachi *et al*.1991.
Community	*V. parahaemolyticus*	1(1)	1992	Kraa, 1995
Community	*V. vulnificus*	(1)	1990	McAnulty, 1990
Community	Virus	>461	1990	Lees, 2000; Kraa, 1995
Community	Norovirus	>370	1989	Kraa, 1995
Community	Hepatitis A	467 (1)	1997	Anonymous, 1997; SafeFood NSW, 2001; Conaty *et al.* 2000
Community	Norwalk-like viruses	97	1996	NSW Health
Eating est.	Norovirus	>18	1990	NSW Health
Eating est.	*V. vulnificus*	(1)	1988	NSW Health
Eating est.	*V. vulnificus*	(1)	1990	NSW Health
Manufacturer	*V. parahaemolyticus*	(1)	1992	NSW Health
Retail	*V. vulnificus*	1	1989	NSW Health

Table B2: Outbreaks of food poisoning due to consumption of seafood contaminated with toxins (after NRVP, 2001)

Food Business	Product	Toxin	Cases (deaths)	Year	Reference
Community	Spanish mackerel	Ciguatoxin	63	1987	Capra, 1997
Caterer	Coral trout	Ciguatoxin	8	1991	Lehane, 1999
Private residence	Coral reef fish	Ciguatoxin	1	1991	
Retail	Coral trout	Ciguatoxin	2	1991	Merianos *et al.* 1991
Retail	Spanish mackerel	Ciguatoxin	43	1994	Kraa *et al.*, 1994; Kraa, & Campbell, 1994
Private residence	Coral trout	Ciguatoxin	4	1995	Fenner *et al.* 1997
Private residence	Coral trout	Ciguatoxin	4	1995	Anonymous, 1995
Retail	Spanish mackerel	Ciguatoxin	15	1995	Harvey, 1995
Retail	Rock cod	Ciguatoxin	2	1996	DOH
Community	Coral trout	Ciguatoxin	6	1997	Karalis *et al.* 2000
Community	Coral trout	Ciguatoxin	10	1997	Karalis *et al.* 2000
Community	Coral cod	Ciguatoxin	20	1997	Lucas, *et al.* 1997
Eating estab.	Maori Wrasse	Ciguatoxin	18	1997	Ng & Gregory, 2000; Andrews *et al.* 1998
Retail	Fish	Ciguatoxin	8	1997	DOH
Retail	Coral trout	Ciguatoxin	6	1997	Karalis *et al.* 2000
Private residence	Spotted cod	Ciguatoxin	12	1998	Karalis *et al.* 2000
Private residence	Barracuda	Ciguatoxin	7	1998	Williams & Dentith, 1998
Retail	Cod	Ciguatoxin	3	1998	DOH
Retail	Spotted cod	Ciguatoxin	10	1998	Karalis *et al.* 2000
Retail		Ciguatoxin	3	1998	Kirk *et al.* 1999
Retail		Ciguatoxin	5	1998	Kirk *et al.* 1999
Private residence	Mackerel	Ciguatoxin	2	1999	Queensland Health
Private residence	Queenfish	Ciguatoxin	7	1999	Queensland Health
Private residence	Spanish mackerel	Ciguatoxin	4	1999	Anonymous, 1999
Eating estab.	Cod	Ciguatoxin	3	2000	Queensland Health
Eating estab.	Sweetlip	Ciguatoxin	2	2000	Queensland Health
Private residence	Black trevally	Ciguatoxin	?	2000	OzFoodNet
Private residence	Coronation trout	Ciguatoxin	9	2000	Queensland Health

Food Business	Product	Toxin	Cases (deaths)	Year	Reference
Private residence	Spotted mackerel	Ciguatoxin	?	2000	OzFoodNet
Private residence	Queenfish	Ciguatoxin	?	2000	OzFoodNet
Private residence	Black kingfish	Ciguatoxin	?	2000	OzFoodNet
Private residence	Coral trout or cod	Ciguatoxin	?	2000	OzFoodNet
Retail	Coral trout	Ciguatoxin	4	2000	Queensland Health
Caterer	Spanish mackerel	Ciguatoxin	14	2001	Queensland Health
Retail	Coral trout	Ciguatoxin	17	2001	Anonymous, 2001
Retail	Spotted mackerel	Ciguatoxin	2	2001	Queensland Health
Community	Pipis	DSP	59	1997	DOH
	Pipis	DSP	56	1997	Quaine *et al.* 1997
	Shellfish	PSP	2	2000	Anonymous, 2000
Eating estab.	Flake	Marine toxin	2	2000	Queensland Health
Eating estab.	Pilchards	Scombrotoxin	>6	1995	Ross & Sanderson, 2000
Resort	Tuna	Scombrotoxin	4	1995	DOH
Retail	Marlin	Scombrotoxin	2	1997	Ross & Sanderson, 2000
Wholesaler	Thai fish cakes	Scombrotoxin	9	1998	Kirk *et al.* 1999
Wholesaler	Risotto	Scombrotoxin	3	1998	Kirk *et al.* 1999
Wholesaler	Tuna	Scombrotoxin	6	1998	Kirk *et al.* 1999
Eating estab.	Grenadier	Scombrotoxin	5	1999	Klessa & Csizmadia, 2000
Eating estab.	Pasta with tuna	Scombrotoxin	?	1999	OzFoodNet
Supermarket	Tuna	Scombrotoxin	4	1999	DOH
Eating estab.	Mahi Mahi	Scombrotoxin	4	2001	Queensland Health
Mobile canteen	Rudderfish or Caster oil fish	Wax esters	9	2001	DOH
Eating estab.	Rudderfish (Butterfish)	Wax esters	>14	1999	Kirk *et al.* 2000

Table B3: Food poisoning outbreaks due to the consumption of prawns in Australia 1990-2010 (after OzFoodNet)

Source	Hazard	Country	Cases (deaths)	Year	Reference
Retail	*V. parahaemolyticus*	Indonesia.	27	1990	NSW Health
Caterer	*V. parahaemolyticus*	Indonesia	100 (1)	1990	NSW Health; Kraa, 1995
Importer	*V. parahaemolyticus*	Indonesia	>50	1992	Kraa, 1995
Eating est.	*S.* Typhi	Thailand	4	1995-1996	NSW Health
Eating est.	Hepatitis A	Burma	23	1997	NSW Health
Eating est.	Hepatitis A	Burma	17	1997	Anonymous, 1997
Eating est.	*V. cholerae* non 01, non 139*	Not known	10	1999	OzFoodNet
Eating est.	Hepatitis A	Not known	2	2003	OzFoodNet
Eating est.	Unknown	Not known	2	2009	OzFoodNet

* Red claw crayfish

Table B4: Food poisoning outbreaks due to the consumption of seafood in Australia 1990-2010 (after OzFoodNet)

Year	State	Setting	Hazard	Cases	Product
2005	NSW	Primary produce	Ciguatera	10	Barracuda
2003	QLD	Home	Ciguatera	5	Barracuda (*Sphyraena* spp.)
2005	NSW	Primary produce	Ciguatera	5	Black kingfish
2005	NSW	Primary produce	Ciguatera	2	Black trevally
2003	QLD	Home	Ciguatera	2	Cod fish heads
2003	QLD	Home	Ciguatera	7	Coral trout
2003	QLD	Home	Ciguatera	2	Coral trout
2005	Vic	Primary produce	Ciguatera	5	Fijian snapper
2003	QLD	Home	Ciguatera	3	Fish
2003	QLD	Home	Ciguatera	3	Fish head soup - Red Emperor
2003	QLD	Home	Ciguatera	4	Unknown
2003	QLD	Home	Ciguatera	3	Giant trevally
2002	Qld	Home	Ciguatera	5	Grunter bream
2009	QLD	Primary produce	Ciguatera	2	King snapper
2005	NSW	Primary produce	Ciguatera	4	Mackerel

Year	State	Setting	Hazard	Cases	Product
2003	QLD	Home	Ciguatera	3	Mackerel
2009	QLD	Primary produce	Ciguatera	2	Reef cod
2005	NSW	Primary produce	Ciguatera	17	Spanish mackerel
2005	NSW	Primary produce	Ciguatera	2	Spanish mackerel
2003	QLD	Restaurant	Ciguatera	15	Spanish mackerel
2002	Qld	Takeaway	Ciguatera	Unknown	Spanish mackerel
2002	Qld	Home	Ciguatera	2	Striped perch
2005	NSW	Primary produce	Ciguatera	2	Trevally
2005	NSW	Primary produce	Ciguatera	2	Yellowtail kingfish
2005	NSW	Primary produce	Ciguatera	8	Yellowtail kingfish
2008	QLD	Primary produce	Ciguatera	4	Yellow king - Samson fish
2006	QLD	Primary produce	Ciguatera	4	Black kingfish
2008	QLD	Primary produce	Ciguatera	6	Black kingfish
2006	QLD	Primary produce	Ciguatera	2	Cod
2008	QLD	Primary produce	Ciguatera	3	Cod
2006	Vic	Primary produce	Ciguatera	2	Coral perch or coral trout
2010	Qld	Primary produce	Ciguatera	2	Coral trout
2010	QLD	Primary produce	Ciguatera	6	Fish curry
2010	Qld	Primary produce	Ciguatera	4	Fish head soup
2010	QLD	Private residence	Ciguatera	4	Mackerel soup
2008	QLD	Primary produce	Ciguatera	6	Red throat emperor/ reef snapper
2006	NT	Primary produce	Ciguatera	14	Slate sweetlips
2006	QLD	Primary produce	Ciguatera	4	Spanish mackerel
2006	QLD	Primary produce	Ciguatera	2	Spanish mackerel
2009	QLD	Primary produce	Ciguatera	3	Spanish mackerel
2010	Qld	Primary produce	Ciguatera	2	Spanish mackerel
2006	QLD	Primary produce	Ciguatera	2	Trevally
2008	QLD	Primary produce	Ciguatera	2	Yellowtail kingfish
2001	QLD	Home	Ciguatera	3	Barracuda (*Sphyraena jello*)
2001	Vic	Home	Ciguatera	16	Coral trout

Year	State	Setting	Hazard	Cases	Product
2001	QLD	Home	Ciguatera	4	Coral trout
2001	QLD	Home	Ciguatera	14	Spanish mackerel
2001	QLD	Home	Ciguatera	9	Spanish mackerel
2002	NSW	Home	Ciguatera	Unknown	Spanish mackerel
2001	QLD	Home	Ciguatera	2	Spotted mackerel
2007	QLD	Primary produce	Ciguatera	3	Coral trout
2007	QLD	Primary produce	Ciguatera	5	Coral trout
2007	QLD	Primary produce	Ciguatera	2	Coral trout
2004	QLD	Restaurant	Ciguatera	4	Coral trout
2004	QLD	Private residence	Ciguatera	2	Fish species unknown
2004	QLD	Private residence	Ciguatera	2	Golden spotted trevally fish
2004	QLD	Private residence	Ciguatera	4	Grey mackerel
2004	QLD	Takeaway	Ciguatera	4	Grey mackerel
2007	QLD	Primary produce	Ciguatera	2	Mackerel
2007	QLD	Primary produce	Ciguatera	6	Mackerel
2007	QLD	Primary produce	Ciguatera	2	Mackerel
2007	NT	Primary produce	Ciguatera	2	Reef cod
2007	QLD	Primary produce	Ciguatera	2	Spanish mackerel
2004	QLD	Contaminated primary produce	Ciguatera	5	Spanish mackerel/golden trevally
2004	QLD	Private residence	Ciguatera	3	Trevally
2003	Vic	Restaurant	Escolar	3	Escolar
2003	QLD	Restaurant	Escolar	20	Escolar
2001	Hunter	Conference/ function	Escolar esters	20	Escolar
2009	QLD	Primary produce	Fish wax ester	27	Escolar/rudderfish
2004	Vic	Commercial caterer	Suspected	9	'Butterfish' (rudderfish)
2001	Vic	Restaurant	Wax ester	5	Butterfish
2009	ACT	Contaminated primary produce	Waxy esters		Escolar
2007	QLD	Private residence	Histamine	2	Imported Indonesian tuna
2007	Vic	Restaurant	Histamine	2	Mahi Mahi
2007	NT	Commercial manufactured food	Histamine	2	Tinned tuna

Year	State	Setting	Hazard	Cases	Product
2007	Vic	Restaurant	Histamine	2	Tuna
2007	NSW	Private residence	Histamine	3	Tuna kebab steaks
2007	QLD	Private residence	Histamine	4	Tuna kebabs
2007	NSW	Restaurant	Histamine	2	Tuna steak
2010	Vic	Private residence	Histamine	4	Tuna
2001	QLD	Restaurant	Histamine	4	Mahi Mahi
2003	QLD	Restaurant	Histamine	3	Dolphin Fish
2003	Vic	Restaurant	Histamine	22	Escolar
2005	Vic	Primary produce	Histamine	2	Fish
2003	NSW	Home	Histamine	2	Sardine
2009	NSW	Restaurant	Histamine	2	Tinned anchovies imported from Morocco
2009	QLD	Other	Histamine	6	Tuna
2005	Vic	Restaurant	Histamine	2	Tuna
2003	QLD	Home	Histamine	2	Tuna patties
2005	NSW	Private residence	Histamine	4	Tuna steak
2009	ACT	Private residence	Histamine	2	Tuna steak
2005	NSW	Restaurant	Histamine	2	Yellowfin tuna
2005	Tas	Restaurant	Histamine	2	Yellowfin tuna
2006	QLD	Private residence	Histamine	2	Blue fin tuna steaks
2006	Vic	Restaurant	Histamine	2	Kingfish
2008	NSW	Bakery	Histamine	1	Canned tuna
2006	NSW	Restaurant	Histamine	2	Tuna steaks
2006	NSW	Restaurant	Histamine	6	Yellowtail kingfish fillets
2010	NSW	Restaurant	Histamine	5	Mahi-mahi fish fillets
2003	WA	Restaurant	Norovirus	35	Japanese IQF oysters
2003	NT	Restaurant	Norovirus	48	Japanese IQF oysters
2004	NSW	Primary produce	Norovirus	24	Oysters
2004	QLD	Contaminated primary produce	Norovirus	4	Oysters (frozen)
2004	QLD	Contaminated primary produce	Norovirus	2	Oysters (frozen)
2004	WA	Commercial caterer	Norovirus	19	Prawns and cold meats

Year	State	Setting	Hazard	Cases	Product
2001	Vic	Community	*S.* Mississippi	6	Suspected oysters
2004	QLD	Restaurant	*Salmonella*	13	Sushi rolls
2006	NSW	Commercially manufactured	*Salmonella*	6	Tuna and salmon sushi rolls
2004	ACT	Restaurant	*Salmonella*	12	Ling
2007	WA	Takeaway	*Salmonella*	23	Sushi and Katsudon (made with eggs)
2003	ACT	Home	Unknown	3	Fish
2002	NSW	Takeaway	Unknown	2	Fish
2007	NSW	Takeaway	Unknown	2	Grilled tuna
2003	WA	Caterer	Unknown	17	Japanese IQF oysters
2008	NSW	Private residence	Unknown	3	Mussels - fresh
2008	NSW	Private residence	Unknown	2	Mussels - fresh
2007	NSW	Restaurant	Unknown	19	Oysters
2008	NSW	Restaurant	Unknown	4	Oysters
2008	NSW	Restaurant	Unknown	10	Oysters
2004	NT	Contaminated primary produce	Unknown	5	Oysters (frozen)
2007	Tas	Other	Unknown	19	Oysters, suspected
2009	QLD	Takeaway	Unknown	2	Prawn roll
2004	Vic	Contaminated primary produce	Unknown	7	Redfin
2007	NSW	Restaurant	Unknown	4	Seafood platter
2007	SA	Commercial caterer	Unknown	12	Sushi
2006	NSW	Private residence	Unknown	4	Suspect Nile perch
2006	NSW	Private residence	Unknown	6	Suspect oysters
2004	ACT	Restaurant	Unknown	16	Suspected calamari
2005	Vic	Restaurant	Unknown	11	Suspected Spanish mackerel
2009	NSW	Fair/festival/mobile service	Unknown	3	Unknown, possibly prawns or calamari
2006	QLD	Takeaway	*S. aureus*	3	Sushi roll
2006	NSW	Private residence	*Vibrio*	3	Whitebait

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Section C: Hazard Sheets

Norovirus and Hepatitis A

Hazard Identification

Shellfish have been associated with foodborne viral infection throughout the world. In 1991 in Shanghai, almost 300,000 people contracted hepatitis and nine died after consuming cockles contaminated with Hepatitis A virus (Tang *et al.*, 1991). In the USA in the period 1992-99, there have been 12 documented outbreaks of viral poisoning following consumption of shellfish (mainly oysters) involving 1473 persons (Smith de Waal, *et al.*, 2000).

In the period 1990-2000, Australian oysters were implicated more frequently in outbreaks than other shellfish (Table C1) whereas, in the period 2001-2010 viral illness fell significantly, with imported shellfish (predominantly frozen oysters) causing most of the illnesses.

Table C1: Outbreaks of food poisoning due to consumption of oysters (after NRVP, 2001)

Setting	Pathogen	Cases (deaths)	Year	Reference
Caterer	Norovirus	>32	1990	NSW Health
Caterer	Norovirus	>48	1990	NSW Health
Caterer	Norovirus?	>19	1990	NSW Health
Caterer	Norovirus	30	1990	NSW Health
Caterer	Norovirus	>11	1990	NSW Health
Caterer	Norovirus	>39	1990	NSW Health
Caterer	Norovirus	18	1992	Ruben *et al.* 1992
Caterer	Norovirus	>148	1990	NSW Health
Caterer	Norovirus	>25	1990	NSW Health
Caterer	Norovirus	>17	1990	NSW Health
Caterer	Norovirus	>74	1990	NSW Health
Community	Virus	>461	1990	Lees, 2000; Kraa, 1995
Community	Norovirus	>370	1989	Kraa, 1995
Community	Hepatitis A	467 (1)	1997	Anonymous, 1997; SafeFood NSW, 2001; Conaty *et al.* 2000
Community	Norovirus	97	1996	NSW Health
Eating est.	Norovirus	>18	1990	NSW Health

Viruses most commonly associated with these outbreaks are Norovirus (NoV) and Hepatitis A (HAV). The former typically cause mild, self-limiting diarrhoea while HAV infection can be severe.

Exposure Assessment

Cooking may inactivate virus particles in shellfish, but is often ineffective particularly because very mild cooking procedures are employed. The degree of virus inactivation varies with cooking method and type of virus and is unlikely to render safe a highly contaminated

product. However, at a low level of contamination in the product, cooking could reduce virus levels to ≤ 1 plaque forming units (pfu) per serving, so cooked product will be excluded from the exposure assessment. The Ministry of Agriculture, Fisheries and Food of the United Kingdom recommend that to inactivate viruses, the internal temperature of molluscs be maintained at 90°C for more than 1.5 minutes before consumption (Huss *et al*. 2000).

Perhaps >90% of oysters are eaten raw or only lightly cooked, leading to an estimate that approximately 30 million servings of uncooked, or effectively uncooked, oysters are consumed. Thus, the contribution of cooking to the reduction of risk from oysters in Australia is negligible.

There are no published data on the level or frequency of viral contamination in shellfish produced in Australian systems. American data indicate that up to 20% of shellfish from clean growing waters may be contaminated with viruses (Mosley, 1967; Goldfield, 1976; Gerba and Goyal, 1978; DeLeon and Gerba, 1990; Sobsey *et al.*, 1991). If this figure is applied to Australia then approximately six million servings of oysters could be contaminated with viruses while, with a contamination rate of 1%, some 300,000 servings could be contaminated.

All immunologically unprotected individuals (i.e. those without serological immunity) are susceptible to infection with human enteric viruses. There is no indication in the literature that immunocompromised individuals are at greater risk of infection from enteric viruses than are immunocompetent individuals.

Hazard Characterisation

Enteric viruses can be introduced into aquatic environments through contamination with sewage. They may persist longer than enteric bacteria in marine environments and can accumulate in bivalve molluscs. As a consequence, their presence in shellfish does not always correlate with bacterial indicators of faecal pollution in marine environments. Viruses may also take longer to depurate from contaminated shellfish than enteric bacteria (Jackson and Ogburn, 1996) and viruses are more resistant to inactivation during cooking than bacteria. Outbreaks of viral food poisoning associated with shellfish continue to occur in Australia and worldwide. Even where control systems are in place (e.g. USA) outbreaks continue to occur. In general, the incidence of seafood-borne viral food poisoning in Australia is low, suggesting that control strategies are effective. Australian outbreaks have been associated with failures or non-implementation of control strategies.

Viral hazards associated with consumption of seafood were the topic of a number of reviews (Mosley, 1967; Goldfield, 1976; Gerba and Goyal, 1978; Richards, 1985; CDC, 1990; DeLeon and Gerba, 1990; Sobsey *et al.*, 1991; Fleet *et al*., 2000; Lees, 2000), the latter two presenting up-to-date reviews of viral contamination of Australian oysters.

In summary, the main conclusions of these reviews are that:

- Viruses causing disease in fish are not pathogenic to humans.
- Food contaminated with human waste containing viruses that are infective *via* the faecal-oral route is a cause of foodborne disease.
- Seafood can be contaminated with enteric viruses through exposure to raw or treated sewage and during processing and preparation by contaminated water supplies and infected food handlers.

- Finfish and crustaceans are not usually associated with the spread of viral foodborne disease unless contaminated by food handlers.
- Consumption of both raw and cooked molluscan bivalves (shellfish) is a well documented cause of viral foodborne disease.
- Virus particles can remain detectable for several months under certain conditions in seawater and in food.
- Shellfish depuration techniques do not totally eliminate viral particles.
- Infectious doses are presumed to be low e.g. 10-100 virus particles.
- Human enteric viruses do not replicate in seafood products so that time and temperature of storage/handling are not risk factors.
- Viruses are resistant to moderate heat and pH conditions.

Norovirus

These are non-enveloped RNA viruses classified in the *Caliciviridae*. Caliciviruses display a Star of David surface structure when viewed by negative stain electron microscopy and at the genetic level have their capsid open reading frame fused to and contiguous with the non structural proteins. The group is described collectively as Small Round Structured Viruses (SRSV) and contains the Norovirus which is named after places where the outbreaks occurred e.g. Norwalk and Snow Mountain.

Illness Caused

Symptoms include nausea, vomiting, diarrhoea, fever and abdominal pain with an incubation period of 1-4 days, usually followed by recovery without complications. Human NLV cause epidemic gastroenteritis amongst all age groups (Caul, 1996a; Caul, 1996b; Clarke and Lambden, 1997) and may be the most significant cause of infectious intestinal disease. Attack rates for SRSV seafood-associated gastroenteritis in outbreaks are relatively high; Kirkland *et al.* (1996) reported 56% (27/48) in one outbreak and Linco and Grohmann (1980) reported 89% (25/28) in another.

Unique Host Susceptibility Factors

SRSVs are a major cause of foodborne gastroenteritis especially linked to the consumption of sewage-contaminated shellfish. It has been estimated that 20-25% of UK, 67% of Japanese and 39% of USA outbreaks, were food associated (Caul, 1996b). In those incidents in the UK and Japan, shellfish were identified as the major source.

Human caliciviruses are primarily a paediatric infection, and there is little evidence that they cause foodborne illness. Seroprevalance studies suggest that childhood infection gives rise to life-long immunity. Up to 90% of children and adults have specific antibodies to human caliciviruses which may explain why consumption of contaminated shellfish by adults rarely causes disease (Caul, 1996b).

Hepatitis A virus (HAV)

This virus is classified within the Hepatovirus genus of the *Picornaviridae* family. HAV has a single molecule of RNA surrounded by a small (27nm diameter), non-enveloped, protein capsid.

Hepatitis A is usually a mild illness characterised by sudden onset of fever, malaise, nausea, anorexia and abdominal discomfort, followed by jaundice, perhaps up to four weeks after exposure. The infectious dose is unknown but presumably is similar to other RNA enteroviruses (10-100 virus particles). HAV is excreted in faeces of infected people and can infect susceptible individuals when they consume contaminated water or foods. Water, shellfish, and salads are the most frequent sources. Contamination of foods by infected workers in food processing plants and restaurants is common. The virus has not been isolated from any food directly associated with an outbreak. Because of the long incubation period, the suspected food is often no longer available for analysis (Sobsey *et al.*, 1991; FDA, 1999). Shellfish have been associated worldwide with a large number of hepatitis outbreaks (Tang *et al.*, 1991; Xu *et al.*, 1992; Leoni *et al.*, 1998).

Illness Caused

The incubation period for Hepatitis A, which varies from 2-6 weeks (mean 4 weeks), is dependent upon the number of infectious particles consumed. Infection with very few particles results in longer incubation periods. The period of communicability extends from early in the incubation period to about a week after the development of jaundice. The greatest danger of spreading the disease to others occurs during the middle of the incubation period, well before the first presentation of symptoms. Many infections with HAV do not result in clinical disease, especially in children. When disease does occur, it is usually mild and recovery is complete in one to two weeks. Occasionally, the symptoms are severe and convalescence can take several months. Patients suffer from feeling chronically tired during convalescence, and their inability to work can cause financial loss. Less than 0.4% of the reported cases in the U.S. are fatal, usually occurring in the elderly (Sobsey *et al.*, 1991; FDA, 1999).

Unique Host Susceptibility Factors

All people who ingest the virus and are immunologically unprotected are susceptible to infection (Sobsey *et al.*, 1991; FDA, 1999). Hepatitis A infection in children is normally subclinical, while in adults overt hepatitis develops in the majority of individuals (Hollinger and Ticehurst, 1990). Infection results in long-term immunity as older individuals are more likely to have HAV antibodies than younger individuals. In one study of 245 blood donors, 57% of those aged 30-49 years were antibody positive, compared with only 30% of those aged 18-29 years. Immunity appears to be protective as repeat attacks are rare (Boyd and Marr, 1980). Other host factors affecting the severity of hepatitis infection are poorly characterised. Immune impairment is less significant than for other foodborne pathogens. Existing liver damage (e.g. cirrhosis) may be significant (Cliver, 1989).

Table C2: Variability in reported incidence of viruses in seafood

Food (% positive)	Viruses detected	Reference
Shellfish (21%)	HAV, EV*	Apaire Marchais *et al.* (1995)
Oysters (50%)	RV (17-246/100g) EV (124-200/100g)	Boher *et al.* (1995)
Oysters (58%)	EV	Chung *et al.* (1996)
Mussel & cockle (73%)	HAV	Crance *et al.* (1995)
Oysters (20%), mussels (0/15)	RV	Hafliger *et al.* (1997)
Cockles & mussels 67%	HAV	Le Guyader *et al.* (1993)
Cockles & mussels	EV (22%), HAV (14%) RV (20%)	Le Guyader *et al.* (1994)
Cockles	EV (89%), HAV (84%)	Le Guyader *et al.* (1995)

* (HAV = Hepatitis A; EV = enteroviruses; RV = rotavirus)

Norovirus and HAV have been linked to shellfish-associated gastroenteritis in Australia (Cross *et al.*, 1979; Kraa, 1990a,b; Bird and Kraa, 1995; Dalton, 1997; Grohmann, 1997; Murphy, *et al*., 1979 and Stafford *et al.*, 1997). The first outbreak occurred in 1977 when oysters harvested from Georges River in Sydney and frozen on the half-shell caused problems in the UK. Product conformed with the microbiological standard of <2.3 *E. coli*/g and no bacterial pathogens were isolated at levels which would cause gastroenteritis. The oysters had been harvested during rainfall events when the *E. coli* count had exceeded the standard, though freezing may have killed *E. coli* but not viruses. Oysters from Georges River were also implicated in an outbreak of gastroenteritis involving more than 2000 consumers in 1978 and though NoV was isolated from patients, it was not isolated from oysters.

These incidents resulted in changes to oyster regulation in NSW with the mandating of depuration for all oysters before sale (Ayres, 1991). However, in 1984, NoV was implicated in an outbreak of gastroenteritis in Tamworth, NSW (Kraa, 1990a) and also in 1996, when oysters harvested from estuarine waters in northern NSW and depurated were implicated in an outbreak of gastroenteritis involving 96 people. The virus was isolated from one sample of oysters but not from faecal samples.

The first case of HAV from shellfish in Australia was attributed to incompletely cooked mussels from contaminated waters in Victoria with seven out of the 10 consumers who ate the mussels developing symptoms of Hepatitis A (Locarnini and Gust, 1978).

The largest outbreak of Hepatitis A in Australia occurred during 1996-97 following consumption of oysters from the Wallis Lake region of Australia (CDI, 1997) when almost 500 people were affected and one died. Oysters from the area tested positive for HAV (by PCR) and also for enterovirus and adenovirus, but not NoV. The *E. coli* level was <2.3/g and the oysters had passed through a depuration process. However, a coronial enquiry established several point sources of faecal contamination into growing areas and the question of bacteriological criteria for oysters was again questioned (Wilcox, 1999).

Seafood-associated viral gastroenteritis has been reported following the consumption of seafood that passed microbiological criteria (Chalmers and McMillan, 1995). Shellfish associated with outbreaks in Australia, however, have generally failed to comply with microbiological standards indicating faecal contamination prior to harvest (Cross *et al.*, 1979;

Bird and Kraa, 1995). In an American risk assessment of viral contamination of shellfish an average level of viruses of 10 pfu/100g was determined from beds considered to be 'uncontaminated'. This corresponds to an exposure of 6-24 pfu in a serving (60-240 g) of shellfish (Rose and Sobsey, 1993).

Due to the high infectivity and low infectious dose of these viruses there is potential for secondary cases of infection spread *via* the faecal-oral route. In a clam-associated Hepatitis A outbreak in Italy there was a secondary attack rate of 7.9% (Leoni *et al.*, 1998).

The infectious dose of human enteric viruses is not known but is presumed to be of the order of 10-100 particles (FDA, 1999). For HAV, higher doses of virus have been reported to cause more severe disease.

Growth and Survival

While enteric viruses do not replicate in seafood products, they remain detectable for long periods of time. Several studies have demonstrated no, or very little, reduction in virus numbers during prolonged (up to 28 days) storage at refrigeration temperatures (5-8°C). In the 1978 incident of Norovirus in NSW oysters the agent survived several months freezing on the half shell. Thus if enteric viruses are present in shellfish at the time of harvest, it is likely that they will persist until purchased (Sobsey *et al.*, 1991). The effect of these conditions on their infectivity is unknown.

Heat Treatment

Shellfish are consumed in various forms ranging from raw to thoroughly cooked. When cooked, heat treatments are usually minimal because excessive heating results in unacceptable organoleptic changes. Enteric viruses in shellfish may survive heat processes including stewing, frying, baking, and steaming (Sobsey *et al.*, 1991) and HAV was recovered from experimentally contaminated mussels steamed for five minutes after the shells opened (Abad *et al.*, 1997). Epidemiological data also attests to the heat resistance of enteric viruses. In outbreaks associated with Norovirus-contaminated oysters, persons who reported eating only thoroughly cooked oysters (grilled, stewed, fried (McDonnell *et al.*, 1997) or steamed (Kirkland *et al.*, 1996) were as likely to become ill as those who ate raw oysters. Several outbreaks have been associated with cooked ready-to-eat molluscs (Little *et al.*, 1997). The outbreak cases cited above suggest that cooking is unlikely to render a highly contaminated product safe for consumption unless it is so intensive that the sensory aspects of the product are impaired.

In the United Kingdom, the Ministry of Agriculture recommended an internal meat temperature of 90°C for 1.5 minutes for commercial cooking, parameters which have been adopted by the European Community (Anon., 1993). It is likely that home and restaurant cooking is only partially effective as a control measure, offering more protection with smaller species e.g. mussels compared with larger species such as oysters.

Depuration Efficacy

The ability of depuration to eliminate microbial pathogens in shellfish was reviewed by Jackson and Ogburn (1996). In general the efficacy of depuration depends on the physiology of the oyster (or other mollusc), the pathogen load initially present, the management of the

depuration operation, and other factors. Several studies report failure to remove all viruses (Apaire Marchais *et al.*, 1995; Boher *et al.*, 1995; Abad *et al.*, 1997). In one study, for example, although depuration for 48 h gave a 95% reduction of bacteria it only gave a 7% reduction of Norovirus titres (Schwab *et al.*, 1998). Variables affecting the efficacy of depuration for oysters include the level of contamination, the health of the animals, the management of the depuration process etc (Jackson and Ogburn, 1996).

Among the conclusions of Jackson and Ogburn (1996) was that depuration is satisfactory for removal of moderate contamination with bacterial pathogens, but that continued food poisoning outbreaks in Australia, despite mandatory depuration, indicates that the process is not sufficient to ensure the safety of shellfish product. In particular, they point to strong evidence that viral agents often persist in shellfish both in growing areas and after depuration for periods long after bacterial indicators of faecal contamination can no longer be detected. In particular, experimental studies have shown the long persistence of Hepatitis A virus in oysters.

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Mercury in Finfish

Hazard identification

Based on an acute mercury food poisoning that occurred in Japan during the 1950s, it is known that high levels of dietary mercury in seafood cause measurable deficits in the mental and physical development of young children exposed during gestation. Low levels of mercury are naturally present in the environment and in all foods. Inorganic mercury is poorly absorbed *via* the diet but, in aquatic environments, bacteria can convert inorganic mercury to methylmercury which is readily absorbed by the human body. Methylmercury is accumulated in aquatic food chains, so all fish contain small amounts in their muscle tissue. Predatory fish or mammals such as whales at the top of the food web have the largest amounts.

Mercury levels in most commercially harvested oceanic fish in Australia are <0.5 mg/kg methylmercury, but some large predators such as sharks, marlin and swordfish may have higher levels. Numerous studies have shown that nearly all the human exposure to methylmercury occurs *via* seafood (predominantly finfish) consumption. Therefore individuals who regularly consume large amounts of fish (particularly those fish with high mercury levels) could be exposed to dangerous levels of mercury (FDA, 1994; National Academy of Sciences, 2000).

Exposure Assessment

In Australia there is significant production of species associated with elevated levels of mercury. Predatory fish are associated with elevated levels of mercury and are defined in the Food Standards Code as: *Gemfish, billfish (including marlin), southern bluefin tuna, barramundi, ling, orange roughy, rays and all species of shark.*

Over the past two decades there have been several surveys of Australian finfish (Table C3), all of which have found that most seafood contains low levels of mercury (Health Commission, 1978; Working Group on Mercury in Fish, 1979; WA Food Monitoring Program, 1993; Bureau of Resource Sciences, 1997a and 1997b; White, 1999).

However, these surveys have also established that sharks, particularly warm water sharks (*Carcharinus*) and large game fish, such as swordfish and marlin, have mercury levels much higher than the maximum recommended level of 1 mg/kg (Food Standards Code). Interestingly, although tuna is a large predatory fish it generally has mercury levels <0.5 mg/kg.

In a NSW survey, 3/26 shark samples and 3/8 swordfish samples exceeded the 1 mg/kg limit (White, 1999). The maximum level of mercury found in shark and swordfish in this survey was 2.3 mg/kg and 1.65 mg/kg, respectively. Several other recent surveys have found fish with mercury levels above 1 mg/kg. The 1989-1993 NSW health survey found that nearly 3% of 1,095 fish samples, all shark and swordfish, exceeded the standard.

In the USA, the Food and Drug Administration have also published mercury content ranges for orange roughy (0.42-0.71 mg/kg), swordfish (0.26-3.22 mg/kg) and marlin (0.10-0.92 mg/kg) (FDA, 1994).

Table C3: Mercury levels in predatory fish in Australia

	Mean mercury (mg/kg) Number of samples in parentheses			
	Health Com., 1978	**Working Group on Mercury in Fish, 1979**	**WA Food Monitoring Program, 1993**	**White, 1999**
Gemfish	-	0.68 (148)	-	-
Tuna, Skipjack	-	0.15 (20)	-	-
Tuna, Southern Bluefin	-	0.22 (219)	-	-
Tuna, Yellow Fin	-	0.38 (20)	-	-
Swordfish	1.98	-	-	0.98 (8)
Marlin, Black	-	7.27 (42)	-	0.57 (3)
Shark	-	-	-	-
Angel	-	0.36 (36)	-	-
Blacktip Whaler	-	1.48 (8)	0.41 (14)	-
Blue Pointer	-	1.93 (2)	0.83 (2)	-
Blue Whaler	-	0.41 (2)	-	-
Bronze Whaler	-	0.72 (159)	0.52 (33)	-
Carpet	-	1.02 (76)	0.69 (12)	-
Gummy	-	0.44 (507)	0.29 (4)	-
'Shark'	-	-	-	0.48 (26)

Hazard Characterisation

The tissue concentration of mercury in its organic form, methylmercury, is poorly regulated by vertebrate and invertebrate fish. Inorganic mercury can be methylated by biological (predominantly microbiological) processes in the aquatic environment. Methylmercury accumulates in the food chain, with the highest concentrations found in predatory fish. More than 95% of the total mercury content in edible fish tissue is in the form of methylmercury. Most regulatory bodies have adopted this level as the maximum permissible limit in large predatory fish destined for human consumption.

Farmed vertebrate fish are likely to be safer than their counter-parts caught in the wild. Methylmercury is predominantly taken up from food, and in marine aquaculture systems as well as other systems, fish are generally fed formulated diets. As feeds will, or should, have low mercury content, the harvested fish will likewise have low tissue concentrations of mercury. Moreover, mercury accumulates in fish during their lifetime and tissue concentrations are greater in older and larger fish. Since farmed fish are usually harvested young, they would be expected to have low tissue concentrations even if their feed contained mercury (FAO/NACA/WHO, 1999).

Illness caused

Methylmercury is rapidly absorbed from the gut and enters the brain of adults and foetuses, where it accumulates and is converted to inorganic mercury. Methylmercury is highly toxic and has adverse effects over the lifetime of an individual. The most severe effects were seen

following two methylmercury incidents in Iraq (contaminated grain) and Japan (contaminated seafood) and included mental retardation, cerebral palsy, deafness, blindness and dysarthia in individuals who were exposed *in utero* and sensory and motor impairment in exposed adults.

More recently, chronic, low-dose prenatal exposure to methylmercury from maternal consumption of fish has become associated with impaired performance in neurobiological tests which measure attention, language, memory and fine-motor function.

There is also evidence that exposure to methylmercury can affect the cardiovascular system (blood pressure regulation, variable heart rate and heart disease).

The developing foetus is particularly susceptible to the toxic effects of mercury. The first trimester of pregnancy appears to be the critical period of exposure. Since dietary practices immediately before pregnancy will have a direct bearing on foetal exposure during the first trimester, women of child-bearing age who might become pregnant are at risk from mercury exposure. Australian Bureau of Statistics data indicate that around 200,000 women are pregnant any given time in Australia. Of these, one third (in the first trimester) are at greater risk, or perhaps more correctly their foetuses are at greater risk. Thus, a group around 60,000 are at increased risk at any given time.

Unless otherwise acknowledged the following material on the hazard of methylmercury exposure and current studies is drawn from (Kjellstrom *et al.*, 1986, 1989; Davidson *et al*., 1998; Johnson, 1998; Levin, 1998; Mahaffey, 1998; Myers, 1998).

Nearly all the human exposure to methylmercury occurs via fish consumption. Richardson, (1995) states that there are two primary exceptions: accidental releases, usually in industrial processes and usually for short periods, and mercury used in tooth filling amalgams. Methylmercury obtained from the diet typically resides in the human body for several weeks. To date, mercury health risk estimates have primarily relied on data from a 1970 acute poisoning incident in Iraq that involved severe, rapid exposure from consumption of contaminated grain, and caused some deaths.

Those data are the basis for the current United States EPA Reference Dose (RfD) which is defined as an estimate of a daily exposure to the human population (including sensitive subpopulations) that is likely to be without a risk of adverse effects when experienced over a lifetime. The US EPA reference dose for methylmercury is 0.1 µg/kg body weight/day and includes a safety factor of ten. The health standard for mercury in Australia is the reference acceptable exposure value established by the Joint FAO/WHO Expert Committee on Food Additives (JEFCA). This committee established a provisional tolerable weekly intake (pTWI) for methylmercury of 5 µg/kg body weight/week (White, 1999).

The only documented account of mercury poisoning involving seafoods occurred in people living around Minamata Bay in Japan during the 1950s. In all, there were more than 700 cases of poisoning and 46 deaths. Finfish and shellfish harvested from the area had mercury levels up to 29 mg/kg and were eaten at least daily by most people to give an estimated average methylmercury intake of 0.3 mg/day (Coultate, 1992). For a Japanese woman weighing 50 kg this equates to 6 µg/kg body weight/day, or 42 µg/kg body weight/week, more than eight times the pTWI and 90 times the RfD.

Scientific studies are currently being conducted to clarify what levels of mercury in children can produce adverse outcomes. Three studies of children exposed to mercury *via* fish consumption were being undertaken: the Seychelle Islands in the Indian Ocean, the Faroe Islands in the North Atlantic Ocean and New Zealand populations, all having diets that are highly dependent on marine life. The Seychellois usually eat fish twice a day with an average

mercury content of fish consumed of 0.3 mg/kg which is similar to the majority of fish consumed in Australia.

The National Academy of Sciences expert panel found that children in the Seychelles study had no significant mercury effect (NAS, 2000). By contrast, the Faroe and NZ studies indicated that children who were exposed prenatally to the highest mercury levels had slight abnormalities in development at age seven. The NAS panel has recommended the retention of the EPA's RfD of 0.1 µg/kg body weight/day.

Advice to consumers

FSANZ have issued an advisory which allows adults, including pregnant women, to eat 2-3 serves (300-450 g) per week of low-mercury species (almost all fish except shark, swordfish, billfish and marlin).

By contrast the NZ Food Safety Authority (www.foodsmart.govt.nz/whats-in-our-food/chemicals-nutrients-additives-toxins/specific-foods/mercury-in-fish) recommends pregnant women to eat seafood:

- Without restriction – a range of named finfish, shellfish and squid
- 3-4 serves/week – wide range of finfish and lobster
- 1 serve every 1-2 weeks – shark, marlin, swordfish, bluefin tuna

The amended advice by FSANZ and NZFSA accords with recent findings that consuming <340 g seafood/week may have a detrimental effect on foetal development (Hibbeln *et al.* 2007).

By following the revised FSANZ advice of '2-3 serves' consumers may consider it prudent to defer to two, rather than three serves, an intake which has been shown to be detrimental (Hibbeln *et al.* 2007). While advice on avoiding particular species is appropriate, by specifying serves and quantities FSANZ may unwittingly be prompting consumers to limit seafood intake.

Given the NZ FSA stance on consumption of seafood it is recommended that FSANZ review their current advice, with a view to aligning with the NZ approach.

It is noted that Thompson and Lee (2009) state: *NZFSA has recently agreed with FSANZ that both agencies should investigate removing fish monitored for mercury from New Zealand and Australia's respective 'risk lists' as exposure from mercury in fish would be better managed by an education programme such as NZFSA's advisory information for pregnant women.*

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Parasites in Raw Fish

Hazard Identification

Sashimi (pieces of raw fish) and sushi (pieces of raw fish or cooked prawns with rice and other ingredients) are increasingly popular foods in Australia. The ingestion of raw fish carries with it a number of risks of foodborne infection, including the risk of parasitic worms that can cause disease in humans.

Among parasites associated with fish and seafoods, most of those known to cause disease in humans are helminths (parasitic worms) and include nematodes (roundworms), cestodes (tapeworms) and trematodes (flat worms, or flukes). Over 50 species of helminths from fishes, crabs, snails and other molluscs are known to cause human illness. Of most concern are:

- Nematodes *Anisakis simplex, Pseudoterranova decipiens*, *Eustrongylides* and *Gnathostoma*
- Cestodes *Diphyllobothrium*
- Trematodes *Clonorchis sinensis, Opisthorchis*, *Heterophyes*, *Metagonimus*, *Nanophyetes salminicola* and *Paragonimus*

The helminth parasites are sensitive to freezing and to relatively mild heating (i.e. normal cooking temperatures). Consequently, those parasites associated with seafood are generally passed to man by consumption of raw, minimally processed or inadequately cooked chilled products which are mostly associated with socio-cultural and behavioural factors, particularly the consumption of raw or undercooked seafood (Adams *et al*., 1997).

A wide range of seafood products have been implicated in human infection (FDA, 1999):

- Ceviche (fish and spices marinated in lime juice; Latin America)
- Lomi lomi (salmon marinated in lemon juice, onions and tomato; Hawaii)
- Poisson cru (fish marinated in citrus juice, onions, tomatoes and coconut milk)
- Salmon roe
- Ako poki (Japanese and Hawaiian cephalod dish)
- Sashimi (pieces of raw fish; Japan)
- Sushi (pieces of raw fish with rice and other ingredients; Japan)
- Green herring (lightly brined herring; Netherlands)
- Scandinavian gravlax
- Drunken crabs (crabs marinated in wine and peppers; China)
- Cold-smoked fish and undercooked grilled fish

Exposure Assessment

Volumes of Product

Parasites are killed by freezing in a time and temperature-dependent manner. It is, therefore, assumed that only products that are not, and have not been, frozen will pose a risk to consumers.

There is one published record of anisakid infection in Australian consumers (Shamsa and Butcher, 2011) in which a 41-year-old Australian woman of Tongan descent consumed raw mackerel.

Sushi/sashimi: Consumption Frequency and Demographics

Ruello (1999) researched consumption of sushi in Sydney during Spring 1998 and Summer 1999 finding that 19% of Sydney residents consumed seafoods and that the average serve size was 377 g. Of those consumers, only 1-2% reported consuming sushi. Thus, it is estimated that in Sydney (population 3,500,000) of the order of 10,000 sushi meals are consumed per week. Of the sushi consumed out-of-home 41% was at restaurants and 33% from a fast food/take away outlet. There was a strong indication that sushi was consumed by those with higher household incomes ($60-80,000 p.a.) and with a tendency towards younger consumers (<40 years old). The 'in-home' consumption survey indicated no sushi consumption. Intuitively, it seems most unlikely that only 10,000 raw fish portions are eaten each week in Sydney, with the figure expected to be an order of magnitude higher. Scaling from Sydney to a national consuming public with similar demographic to that of the Sydney survey leads to total servings between 1.5 and 15 million servings/ annum. One possible estimate of consumption pattern is that very few (1%) Australians consume raw fish on a monthly basis.

Comprehensive lists of parasites that have been reported in Australian fish are presented in Beumer *et al*. (1982), Lester and Sewell (1989), Doupe *et al*. (2003) and Shamsi *et al*. (2010).

Nematodes (roundworms) and Cestodes (tapeworms)

Infection with *Anisakis simplex* has been documented in many species of fish, rockfish, herring, cod, halibut, mackerel, wild-caught salmon, yellowfin and skipjack tuna and squid from many regions of the world. The rates of infection are often high with 10-90% of samples carrying the parasite (Bouree *et al*., 1997) and multiple larvae in each fish are also commonly recorded. Where species and geographical differences exist, it is often due to the feeding habits of the fish, their size and age (older and larger fish typically contain more parasites) and whether definitive hosts (e.g. marine mammals) and intermediate hosts are present to complete the life cycle of the parasite. Due to the need for a mammalian host to complete the parasite's life cycle, correlations between the presence and density of marine mammals and level of contamination of fish have been demonstrated. Pereira-Bueno (1992) discusses some aspects of the distribution and epidemiology of human anisakiasis.

In relation to risks due to Australian species, Humphrey (1995) reports that *A. simplex* has been isolated from flathead (*Platycephalus speculator*) and anisakids have also been recovered from other flatheads, mackerel (*Scomberomorus spp.)*, mackerel tuna (*Euthynnus alleteratus)*, striped trumpeter and farmed salmonids from Tasmania. (Ross, 2000) It is not known, however, whether the strains isolated are pathogenic to humans.

In Tasmania, the incidence of anisakids in wild caught stripey trumpeter and farmed Atlantic salmon (included in Humphrey, 1995) was 91% (39/43) of samples of striped trumpeter and 1/60 farmed Atlantic salmon. At that time striped trumpeter were being used and promoted for raw fish dishes, as were Atlantic salmon and Jack mackerel (Mure and Mure, 1993). Lester and Sewell (1989) reported the incidence of parasites in fish from Heron Island. Anisakis larvae were found in the organs or viscera of six species of fish including blue grenadier, gemfish, Jack mackerel and orange roughy. Beumer *et al*. (1982) provide a list of parasites of fishes in Australian waters.

In 2010, EFSA published a scientific opinion on risk assessment of parasites in fishery products and concluded that, while wild-caught fish can never be guaranteed not to have helminth parasites, the likelihood in farmed fish is much lower.

Of relevance in a listing of farmed fish, a number of species were identified which are both consumed in Australia and exported in a chilled format, making them potential sources of infection. These species include Atlantic salmon, Pacific salmon, rainbow trout and tuna, to which should be added the farmed species Australian Kingfish (*Seriola lalandi*) and Mulloway (*Argyrosomas hololepidotus*).

In a survey of five South Australian species, Shamsi *et al*. (2010) did not detect anisakid larvae in ten samples of wild caught *S. lalandi*.

Infections of *A. simplex* have been reported in New Zealand (Goldsmid and Speare, 1997) and specifically in New Zealand teleost fishes (Hurst, 1984).

Anisakiasis is a relatively common infection in Japan, largely because fish is often eaten raw, lightly cooked or pickled. Infection is also relatively common in northern Europe where cured fish, such as pickled herring, are part of the diet. Deardorff and Overstreet (1991) report that in Japan, the annual incidence of anisakiasis is >1000 cases/annum.

In contrast, the number in the US, where raw fish is not currently a major part of the cuisine, is negligible; anisakiasis is not mentioned in a review of the role of seafood in foodborne diseases in the USA (Lipp and Rose 1997). Although the frequency of infection in Europe is apparently lower than in Asian countries, the number of reported cases has been increasing in recent years, particularly in France (Audicana *et al*., 1995). A retrospective study in France during January 1985-September 1987 by 32 laboratories specialising in parasitology (Hubert *et al*., 1989) confirmed 21 cases of anisakiasis involving several fish species.

The Netherlands was one of the nations recognised to have endemic Aniskiasis. Nonetheless, in the period 1955 (first identified) and 1968 (before control measures were introduced), only 161 cases were reported ~10-15 cases per annum. After the introduction of control measures (freezing and gutting on board) the incidence has decreased to virtually nil (Hayunga, 1997).

FDA (1999) reports that ~10 cases per year are reported in the USA, but consider that many cases go unreported.

Table C4: Guidelines for tolerable levels of parasites in fish (FDA, 1999)

Product	Guideline
Tullibies, ciscoes, inconnus, chubs and whitefish	50 cysts per 45.45 kg (100 lb)
Blue fin and other freshwater herring averaging 1 lb or less	60 cysts per 100 fish, if 20% of the fish examined are infested
Blue fin and other freshwater herring averaging >1 lb	60 cysts per 45.45 kg (100 lb), if 20% of the fish examined are infested
Rose fish (red fish and ocean perch)	3% of fillets examined contain one or more copepods accompanied by pus pockets

At this time, there are no published records of anisakid infections in Australian consumers, nor do there appear to be any unreported cases (Ross, 2000). Despite the widespread

incidence of anisakid larvae in wild fish populations and market fish, there are several reports (Deardorff and Kent, 1989; Angot and Brasseur, 1993; Ross, 2000) indicating that farmed Atlantic and other salmon do not develop infections. Similarly, for cod, Hemmingsen *et al*. (1993) showed that the accumulation of parasites ceased when wild caught Atlantic cod (*Gadus morhua)* were caged and fed artificial diets. This was believed to result from interruption of the parasite life cycle by the feeding of fish with artificial feed thereby preventing infection of the fish by intermediate hosts. Infections of mariculture species by nematodes has not been confirmed (Durborow, 1999).

Diphyllobothrium spp. have been reported to be present in Australian fish (Humphrey, 1995) but there is little detail of the parasite species or species of fish affected. Goldsmid and Speare (1997) indicate that *D. latum* is endemic in North and South America and in parts of Europe, particularly Finland.

Trematodes (flatworms)

Fish-borne trematode disease is highly endemic in southeast China but also in other parts of Asia. *Clonorchis sinensis* affects an estimated 7,000,000 people worldwide. It is the most common parasite in Hong Kong where 30-60% of the population are believed to be infected. Opisthorchiasis (*O. viverrini*) is a major cause of death in northeast Thailand and it is estimated that 7,000,000 are infected in that country. The infection is also very common in Laos (Durborow, 1999).

Imported cases may occur in other parts of the world. Shipments of imported dried or pickled fish are considered a likely source. Additionally, chilled freshwater fish from endemic areas are flown daily into the United States (Benenson, 1995).

Fish hosts of the liver fluke include silver carp (*Hypothalamicthys molitrix*) and the grass carp (*Ctenophargyndon idellus*) both of which are aquaculture species (Durborow, 1999).

Hazard Characterisation

Unless otherwise acknowledged, the following is drawn from FDA (1999), FAO (1999), Hayunga, (1997) and Deardorff and Overstreet (1991). Adams *et al*. (1997) also reviewed helminthic infections acquired from fish and shellfish including risk mitigation and preventive measures. For ease of reading, the hazard characterisation is presented separately for each of the three groups of parasites considered.

Of the parasites, the Anisakidae are the most important and will be given the most attention. The means of controlling Anisakids are equally relevant to other seafood-borne parasites.

Nematodes (rounds worms)

Among the nematodes those of most concern are *Anisakis*, *Capillaria*, *Gnasthostoma* and *Pseudoterranova*, the infective cysts of which are found predominantly in the liver, belly cavity and flesh of marine fish. Of these organisms, *A. simplex* causes the greatest numbers of illnesses which tend to be more severe than those caused by other helminths. Nonetheless, occurrence is considered rare as the infective stage of the parasite is killed by cooking (e.g. 60°C for one minute) or freezing (e.g. -20°C for 24 h).

Anisakis simplex is commonly called the 'herring worm'. Its final hosts are dolphins, porpoises and sperm whales. The larval (worm-like) stage in fish and squid is usually 18-36 mm long, 0.24-0.69 mm wide and pinkish to whitish in colour i.e. it is visible, when exposed, to the naked eye. Anisakiasis, the human illness caused by *A. simplex*, is associated with eating raw fish such as sushi, sashimi, lomi lomi, ceviche, Dutch green herring, marinated fish and cold-smoked fish or undercooked fish (Ward *et al*., 1997).

Pseudoterranova decipiens, commonly called codworm or sealworm, is another important parasitic nematode. The usual final hosts of *Pseudoterranova* are grey seals, harbour seals, sea lions and walruses. The larval stage in fish are 5-58 mm long, 0.3-1.2 mm wide and yellowish, brownish or reddish in colour. These nematodes are related to *A. simplex* and the disease associated with infections is also termed anisakiasis. They are also transmitted to humans through raw or undercooked fish (Ward *et al.*, 1997).

Several forms of illness are recognised. A non-invasive form (more usually associated with *P. decipiens*) involves a tingling sensation in the throat when worms are released from ingested seafood and migrate up the oesophagus, typically to be spat out. The invasive form usually associated with *A. simplex* occurs when the organism penetrates the mucosa or sub-mucosa of the stomach or small intestine resulting in gastric pain, vomiting, and diarrhoea about 12 hours after ingestion. The chronic form can mimic gastric ulcer, appendicitis, enteritis, or gastric carcinoma. Endoscopic identification and removal of the worm is the most reliable treatment. Juvenile larvae have also been mistaken for, and linked with, cancerous cells in the host.

Most infections are reported from Japan and the Netherlands but numbers in other countries are increasing. This has generally been attributed to changing dietary habits, whether in established or migrant populations; although Oshima (1987) maintains that the apparent increase (since 1972) is due to better diagnosis of infections.

A third form of illness - an allergic response to *A. simplex* antigens - has been recognised more recently (Audicana *et al*., 1997). While freezing and cooking may kill *A. simplex* it may not protect consumers against reactions to ingested *A. simplex* antigens. Reactions to the allergens, and an association of chronic *A. simplex* infections with some gastric cancers, are increasingly being recognised as discrete, long term, disease syndromes (Daschner *et al*., 1998).

Gnathostoma infections are associated with consumption of inadequately cooked or marinated freshwater fish. As such they are unlikely to be spread by sushi and sashimi. Nonetheless, *G. spinigerum* is endemic in Australia and is the only species recorded to have caused illness in Australia. It is also found in Asia. Larvae can also penetrate the skin during handling. Nausea, abdominal pain, and vomiting usually develop 24-48 hours after eating. Infection can also involve the eye or cause subcutaneous swelling (Goldsmid and Speare, 1997). The larvae migrate and may invade the central nervous system (CNS) resulting in meningitis or neuropathy. As with anisakiasis chemical treatment is ineffective and surgical removal of the larvae is required.

Capillaria phillipensis is observed mainly in Thailand and the Philippines and causes enteropathy and malabsorption (Goldsmid and Speare, 1997). It utilises many species of freshwater fishes as intermediate host, but its life cycle is incompletely understood.

Disease is usually caused by the direct effects of the presence of the parasite in the host tissue due to mechanical or biochemical damage to cells, tissues or organs of the host or competition for space and nutrients. In the case of helminthic infections, clinical disease in

the host is usually dependent on the worm load, which is in turn dependent on the infecting dose. An exception to this rule is *Capillaria phillipensis* a fish-borne parasite (Goldsmid and Speare, 1997).

In Japan, achlorhydria or hypochlorhydria was found in at least 50% of cases and may be a predisposing factor. In the allergic form of sickness, prior exposure is required to cause sensitisation, though some of the allergens cross react with other organisms (Benenson, 1995). Interestingly, there is some evidence that the Japanese custom of drinking sake wine with raw fish may reduce the risk of establishment of an infection (Durborow, 1999).

As discussed above, factors affecting the dose include the degree of processing (duration and severity of heating, freezing, brining) and the age and size of the fish. The handling of the fish immediately post-harvest has also been implicated as a risk factor. Migration of the parasites from the viscera to the muscle tissue after catch has been demonstrated and most parasites in fish are found initially in the intestinal or belly flap regions. Rapid freezing, or evisceration, can reduce the likelihood or migration to the edible portions of the fish.

Person to person transmission does not occur. Apparently there is universal susceptibility (Benenson, 1995). Anisakid larvae are generally presumed to be killed by freezing and cooking (Angot and Brasseur, 1993). Accordingly only those products that have never been frozen and which are eaten raw or lightly cooked or lightly preserved pose a hazard. A survey by Adams *et al*. (1994) of sushi and sashimi in restaurants and retail shops in Seattle, USA, supported this assumption. All juveniles found in sushi were dead, most likely the result of using previously frozen fish. The larvae are generally killed after freezing and holding at -20°C for 24 hours though Bier (1976) found that the larvae of some types may survive for as long as 52 h at -20°C. Marques *et al*. (1995) studied ten fishes from the Brazilian Coast and isolated approximately 48 anisakid larvae of the genera *Contracaecum*, *Phocanema* and *Anisakis*. Twenty five fishes were held chilled or frozen, and larval survival determined; 98% of the *Contracaecum* larvae survived storage at 0° for five days and 96.4% survived storage at -18°C for 24 h. FDA regulations for the elimination of nematodes in fish by freezing are either seven days at -20°C or 15 h at -35°C. It should be noted that microbial inactivation is a stochastic process. That is, a constant proportion of organisms will be inactivated during any given interval under lethal conditions. Thus, the time required to inactivate all the pathogens or parasites will depend on the number of them initially present.

Additional details of the survival of nematodes under conditions relevant to seafood handling and processing are provided in ICMSF (1996).

Anisakis larvae survival was studied in brines by Karl *et al*. (1994). Traditional German and Danish procedures for pickling herring require at least five and six weeks respectively to eliminate viable larvae. A reduction in the salt phase from 9% to 4.3% w/w, without change in the acetic acid concentration, resulted in the nematode survival time increasing from 35 days to more than 119 days. Hayunga (1997) states that cold smoking and most methods of brining fish are not reliable preventative methods.

Gnathostoma larvae can be killed by cooking or immersing in strong vinegar for >5 h. Immersion in lime juice or chilling at 4°C for >1 month were not completely lethal. The salt tolerance of *Gyrodactylus salaris* is poor, and it begins to die at salt concentrations >7.5 g/kg, with normal seawater causing rapid inactivation (Soleng and Bakke, 1997). Similar results were found for *Gyrodactylus derjavini* by Buchman (1997).

Trematodes (flat worms, flukes)

Fish-borne flatworm (trematode) infections are a public health problem in about 20 nations, particularly in south-east Asia, where freshwater fish are intermediate hosts. In terms of human infection the most important species are from the genera *Clonorchis* and *Opistorchis* (liver flukes), *Paragonimus* (lung flukes) and to a lesser extent *Heterophyes* and *Echinochasmus* (intestinal flukes). Freshwater fish are the intermediate hosts in the life cycle of *Clonorchis* and *Opistorchis* and freshwater crustaceans in the case of *Paragonimus.*

Reservoir hosts of *Clonorchis sinensis* are wild and domestic mammals. Metacercariae (the infective stage) have also been found in crayfishes. Metacercariae encyst in fish gills, fins, muscles or under the skin. Adult worms (1.2-2.4 cm long and 0.3-0.5 cm wide) reside in the bile duct. Pancreatitis may also occur, and an association with choleangiocarcinoma has also been reported.

Infection by *Paragonimus westermani* (human lung fluke) can occur through eating raw or improperly cooked freshwater crabs or crayfish. It is common in Asia and to a lesser extent in Africa and India, and is reported throughout SE Asia/Micronesia, and South Pacific islands. Important hosts include freshwater and brackish-water crabs of the genera *Eriocheir*, *Potamon* and *Sundathelphusa* and the crayfish *Procambrus*.

When eaten by the definitive host the metacercariae of *C. sinensis* encyst in the duodenum, migrate into the bile duct and grow to adulthood. Symptoms may be slight or absent in light infections, the symptoms resulting from local irritation of the bile ducts by the flukes. Loss of appetite, diarrhoea and abdominal pressure are early symptoms of infection, which may take up to 30 days to become apparent. Rarely jaundice may result in enlargement and tenderness of the liver, and progressive ascites and oedema followed by cirrhosis. The organisms may live in human host for 25-30 years. Diarrhoea, epigastric pain, and anorexia are common manifestations of acute disease. Adult worms can produce localised tissue damage that may interfere with bile function, leading to secondary bacterial infection. It is usually a mild disease, and often asymptomatic, but is a significant risk factor for the development of cholangiocarcinoma.

Susceptibility appears to be universal. Direct person to person transmission does not occur (Benenson, 1995). Severity of symptoms is related to the intensity and duration of infection - infections with as many as 500-1000 worms have been reported.

Freezing at -20°C or below for seven days, or -35°C for about 20 h, will kill infective stages of these parasites. Fan (1998) reported that metacercariae of *C. sinensis* remained viable and infective after 10-18 days storage at -12°C, as did those stored for 3-7 days at -20°C. Those kept in a heavily salted fish at 26°C for 5-7 days also remained viable and infective. While pickling does not kill the parasite, boiling for a few minutes does. Infections can also be acquired from crab/crayfish juices.

Cestodes (tape worms)

Cestodes are tapeworms with segmented bodies and a structure that allows them to attach to the intestinal wall of their hosts. The species of most concern is *Diphyllobothrium latum*, the broad tapeworm. Its distribution is worldwide and *D. latum* parasitises a variety of fish-eating mammals of the northern latitudes. A similar species is found in the southern latitudes and is associated with seal hosts. Cases have been reported from around the world including Australia. It is the largest human tapeworm, growing up to 9 m, but infections may be mild or asymptomatic.

Fish are intermediate hosts and infective larvae may be found in trout, whitefish, pike and salmon. Cestode larvae found in fish range from a few millimeters to several centimeters in length and are white or grey in colour. *Diphyllobothrium* tapeworms primarily infect freshwater fish, but salmon and related fish can also carry the parasite. *Diphyllobothrium* tapeworms are usually found unencysted and coiled in musculature or encysted in viscera.

Infection is related to dietary and culinary practices. As with the nematodes, human infections are linked with consumption of raw or minimally processed fish. Freezing and cooking temperatures lethal for anisakids will kill the infective stage of *D. latum*.

Common symptoms are nausea, abdominal pain, diarrhoea and weakness, but it may also cause pernicious anaemia and vitamin B12 deficiency if the worm attaches to the jejunum. *D. denderiticum* and *Ligula intestinalis* (tapeworms of fish eating birds) and *D. pacificum* (tapeworm of seals) have also been found in humans. The infection is usually mild, or even asymptomatic and often of long duration. Massive infections may be associated with diarrhoea and obstruction of the intestinal tract, because the mature worm may be up to 10 m long in the human host.

People are universally susceptible and there appears to be no induction of immunity (Benenson, 1995). FDA (1999) suggests that people of Scandinavian heritage may be genetically more susceptible. In such cases a severe anaemia may develop because of the tapeworm's great requirement for and absorption of Vitamin B12. Victims may harbour more than one worm. As indicated above, the presence of more than one worm can amplify the symptoms of infection.

Acanthocephala (spiny headed worms).

These burrowing worms are widespread in nature and infect, as secondary hosts, amphipod crustaceans, freshwater and marine fish (paratenic hosts) and other, non-aquatic species. They are intestinal parasites and may cause an inflammatory response at the site of proboscis attachment, although usually there are no clinical signs. Wild aquatic birds (such as ducks, swans and geese), dogs and their relatives, pigs and Central and South American monkeys are the definitive hosts. These worms are considered to pose little risk to humans because they are relatively scarce in fish eaten by man, and because the worms are usually localised in the viscera of fish and thus less likely to be eaten.

Similarly, most reviews completely discount the potential for protozoan infections from consumption of fish. The recent review of Durborow (1999) mentions the potential for aquaculture species to lead to infection with *Cryptosporidium* sp., *Enterocytozoan* sp. or *Giardia lamblia*, but indicates that there is currently no evidence of the role of fish in the transfer of these diseases.

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Ciguatoxins

Each year, a number of Australians develop an illness known as ciguatera poisoning after eating certain species of fish. The illness, characterised by gastrointestinal, neurological and cardiovascular symptoms, may be acute and long-term. This assessment acknowledges the reviews by Lehane (1999) and Lehane and Lewis (2000).

Hazard Identification

Ciguatera poisoning is caused by eating subtropical and tropical reef fish which have accumulated naturally-occurring toxins produced by marine algae. The toxins are known to originate from several dinoflagellate algae species (predominantly *Gambierdiscus toxicus*) that are common to ciguatera-endemic regions in tropical waters. These toxins produce a range of gastrointestinal, neurological and cardiovascular symptoms which can persist for many weeks and may be re-triggered by dietary changes or exposure to low levels of toxin, months or years after initial exposure. Ciguatera poisoning is usually self-limiting with a low incidence of death.

Exposure Assessment

Because of the distribution of *Gambierdiscus* most ciguatoxic fish are caught in Queensland and Northern Territory waters, with some coming from northern Western Australia (Port Hedland to Bunbury). A list of fish involved in outbreaks in Queensland during the period 1965-1984 (Table C5) compiled by Gillespie *et al*. (1986) indicates the species involved and relative frequency. By far the most numerous cause of ciguatera is mackerels in the genus *Scomberomorus*, particularly the Spanish mackerel (*S. commerson*). In Tables A1, A3 and B2 are listed details of ciguatera outbreaks in the period 1998-2010 using data from the National Risk Validation Project (1998-2001) and OzFoodNet annual data (2000-2010)

Table C5: Cases of ciguatoxin illness in Queensland 1965-1984 (after Gillespie *et al*., 1986)

Scientific Name (common name)	Number of cases	Number of outbreaks
Scomberomorus commerson (Spanish mackerel)	226	30
Scomberomorus spp (mackerels, species unknown)	161	62
Sphyraena jello (barracuda)	29	13
Plectropomus spp (coral trout)	27	18
Epinephelus fuscoguttatas (flowery cod & other epinephalids)	27	14
Lutjanus sebae (red emperor) and *Lutjanus bohar* (red bass)	16	9
Scomberoides commersonnianus (giant dart)	8	3
Lethrinus nebulosa (yellow sweetlip)	4	1
Seriola lalande (yellowtail kingfish and other seriolids)	6	1
Caranx sp (trevally, species unknown)	4	2
Cephalopholis miniatus (coral cod)	3	2
Chelinus trilobatus (maori wrasse)	3	3
Choerodon venustus (venus tusk fish)	2	1
Trachinotus sp (dart)	1	1
Paracesio pedlryi (southern fuselier)	1	1
Lates calcarifer (barramundi)	1	1
Other and unknown	14	16

Despite Spanish mackerel being most frequently implicated, Gillespie *et al*. (1986) stated that red bass (*Lutjanus bohar*), chinamen fish (*Symphorus nematophorus*) and paddletail (*Lutjanus gibbus*) were recognised as high risk species in Queensland and were not accepted for sale (at that time) by the Queensland Fish Board. The same authors added that, despite its well-known toxicity, red bass was commonly eaten by Queensland fishers with apparently few cases of ciguatoxin poisoning. In addition to the list compiled by Gillespie *et al.* (1986), Lehane (1999) adds parrot fish (*Scarus* spp), grunter bream (*Pomadsys sp*) and moray eel (*Lycodontis* or *Gymnothorax javanicus*) to the list of potentially ciguatoxic species.

It is important to review the scientific and common names listed by Gillespie *et al*. (1986) and Lehane (1999) for their current correctness according to the Australian Seafood Handbook (Yearsley *et al.*, 1999). Names not listed in the Handbook are *Lutjamus gibbus*, paddlefish, *Epinephalus fuscoguttatus*, *Paracesio pedlryi* and southern fusilier. Changed names include *Lethrinus nebulosa* (now *Diagramma* or *Plectorhynchus* sp) and altered specific epithets include *Cephalopholis miniatus* (*C. cyanostigma*) and *Cheilinus trilobatus* (*C. undulatus*). Such information is not of mere semantic moment. Ross (2000) reports anecdotal evidence of a ciguatoxicosis in NSW attributed to ‘queenfish’ which, while not considered a potentially ciguatoxic species by some, was identified by the Handbook as *Scomberoides commersonnianus*, a species regularly implicated in ciguatera poisonings.

Prevalence of intoxication

Estimates of the number of ciguatera cases worldwide are, variously, 20,000 (Durburow, 1999), 50,000 (Benenson, 1995) and 10-50,00 (Lehane, 1999).

In Australia, ciguatera fish poisoning usually occurs as sporadic isolated cases (Fenner *et al.*, 1997; Lucas *et al.*, 1997), although at least two larger outbreaks (>30 cases) have been reported (Hallegraeff, 1998). One hundred and sixty-six confirmed outbreaks affecting 479 people were reported in Australia between 1976 and 1984 (Glaziou and Legrand, 1994). In

Queensland, several thousand cases were notified to authorities over a 10-year period (Ting *et al.*, 1998) with an estimated 1.8-2.5% of the population in that state affected (Glaziou and Legrand, 1994; Lehane, 1999). The average incidence in Queensland (1985-1990) was 1.6 cases/100,000, although in coastal Queensland the annual prevalence was estimated at 33/100,000 (Capra and Cameron, 1988).

Ross (2000) cites a personal communication from Voetsch who collated 41 cases of ciguatera poisoning in NSW for the period 1996-1998, noting also that the list was not comprehensive. Due to under-reporting of this often mild illness, these data represent the minimum prevalence in NSW. There have also been several large outbreaks in Sydney at restaurants. In 1987, 63 people became ill after eating Spanish mackerel (*Scomberomorus commerson*) which had been caught in Hervey Bay, Queensland. Another mass poisoning occurred in 1994 in which 43 people were affected after eating Spanish mackerel from Queensland (Kraa, 1994: Capra, 1997). In Darwin, three people were affected after consuming locally-purchased coral trout (Merianos *et al*., 1991) and there was an outbreak in Brisbane in 1995 involving 15 people who ate Spanish mackerel caught east of Fraser Island (Harvey, 1995). In Victoria in 1997 there were 30 cases of ciguatera after a 16.2kg live Maori Wrasse was consumed at a banquet at an Asian restaurant (Ng and Gregory, 2000).

Seriola dumerili, known in Australia as amberjack and a common component of the catch in WA and Queensland, was associated with 21% (17/81) of ciguatera fish poisoning outbreaks in Hawaii between 1975 and 1981. All commercially harvested *S. dumerili* caught in Hawaii between 1979 and 1981 were tested with an immunoassay for ciguatoxin (detection limit 1 ng/mL). Fifteen percent (824/5,529) gave borderline or positive results, as did 17% (269/1,585) of fish weighing 9 kg or more (Katz *et al.*, 1993). Serological test kits for the detection of ciguatoxin are now available commercially (e.g. Cigua-Check Fish Poison Test Kit Oceanit Test Systems, Inc., http://www.cigua.com), but little reliable data on the incidence of ciguatoxin in Australian fish have been published (Ting *et al.*, 1998). Mackerel and barracuda from mid to northeastern Australian waters have been reported to be frequently ciguatoxic (Price and Tom, 1999).

Hazard Characterisation

When herbivorous fish are eaten by carnivorous fish, dinoflagellate toxin is converted to the more potent ciguatoxin (Durborow, 1999). These toxins accumulate through the food chain, from small fish grazing on algae on coral reefs into the organs of larger top-order predators. Toxin is concentrated in the head, liver and viscera of fish (Ting *et al.*, 1998), but levels are lower in the muscle, the part more usually eaten. The occurrence of toxic fish is sporadic and not all fish of a given species or from a given locality will be toxic (Benenson, 1995). If fish cease ingesting the dinoflagellate the toxin will slowly be purged from the fish. There is one report of ciguatera poisoning associated with consumption of jellyfish (Zlotnick *et al.*, 1995) and breast milk (Lehane, 1999).

Ciguatoxicosis in humans usually involves a combination of gastrointestinal, neurological, and cardiovascular disorders. Initial signs of poisoning occur within six hours of consumption and include perioral numbness and tingling (paresthesia) which may spread to the extremities, nausea, vomiting and diarrhoea. Neurological signs include intensified paresthesia, arthralgia, myalgia, headache, temperature sensory reversal and acute sensitivity to temperature extremes, vertigo and muscular weakness to the point of prostration. Cardiovascular signs include arrhythmia, bradycardia or tachycardia, and reduced blood pressure.

Ciguatera poisoning is usually self-limiting and signs of poisoning often subside within several days of onset. However, in severe cases the neurological symptoms persist from weeks to months and in rare cases, for several years. Sometimes, recovered patients experience recurrence of neurological symptoms months to years after recovery. There is a low incidence of death resulting from respiratory and cardiovascular failure. Clinical testing procedures are not presently available for the diagnosis of ciguatera in humans, which is based entirely on symptoms and recent dietary history. The disease has only recently become known to the general medical community and may be under-reported because of the generally non-fatal nature and short duration of the disease.

All humans are believed to be susceptible to ciguatera toxins. Populations in tropical/subtropical regions are most likely to be affected because of the relatively higher frequency of exposure to toxic fishes. Repeated ciguatoxin exposures are associated with more severe illness (Glaziou and Martin, 1993; Katz *et al.*, 1993).

Infectious Dose/Dose Response

The ciguatoxins are lipid-soluble toxins. These are relatively inert molecules, and remain toxic after cooking and exposure to mild acidic and basic conditions. Ciguatoxin (CTX)-1 is the major toxin (on the basis of both quantity and total toxicity) present in fish, except for certain herbivorous species which accumulate mostly gambiertoxins and less polar ciguatoxins. CTX-1 typically contributes ~90% of total lethality. On the basis of available outbreak data, Lehane (1999) estimated the minimum toxic dose to be ~50 ng in an adult of 50 kg weight (i.e. ~1 ng/kg body weight). In one well-documented incident, six U.S. soldiers became ill after eating fish containing approximately 20 ng ciguatoxin/g flesh. They all presented with nausea, vomiting, watery diarrhoea and abdominal cramps 5-8 h after consumption and some also had numbness in the extremities or perioral region, bradycardia and scalp paresthesia (Poli *et al.*, 1997).

Several studies have demonstrated that increased toxin dose is equated with increased severity of cardiovascular effects in animals and humans (Katz *et al.*, 1993). However, Arcilaherrera *et al.* (1998) found no association between the amount of toxic fish ingested and the latency period or the severity and duration of the symptoms in a study of 10 incidents. It is well recognised that ciguatoxin displays a cumulative exposure phenomenon, with repeated exposure resulting in more severe and prolonged symptoms.

Critical Control Points and/or Management Options

There are few control strategies available to eliminate the risk of exposure to ciguatera affected fish. Current risk reduction strategies include limits on the size and/or type of certain fish species and restrictions on fishing in known toxic areas. Red bass, chinaman fish and paddletail have been regarded as unsuitable for sale in Queensland for 20 years due to their likely toxicity (Lehane, 1999).

Ciguatera poisoning is regularly reported in Queensland which may reflect the impact of recreational fishing. In the US it has been reported that approximately 80% of cases are due to recreational fishers who are unfamiliar with the types of fish that are commonly ciguatoxic (Kuenster, 1991. Other than in Queensland and the Northern Territory, recreational fishermen are unlikely to catch affected fish.

In subtropical and tropical regions professional fishermen are aware of areas to avoid which helps reduce the hazard to consumers. Size restrictions are potentially effective since larger fish may be more frequently toxic than small fish but the practice of filleting into portions for on-board packing and freezing makes this impossible to monitor as a CCP.

While some fish markets claim to not sell potentially poisonous fish such as reef fish, anecdotal evidence suggests that due to inconsistent naming of fish, potentially ciguatoxic species may be sold.

In the USA, current passive control measures include posting warning signs and the issue of pamphlets advising about the hazards of particular species. An active control system involving development of a reliable and inexpensive test for ciguateric fish, regulating fishing of suspected species, testing these fish either on board fishing vessels or at dock site, and educating consumers, commercial and sports fishers and health professionals has been recommended (Ahmed, 1991; Miller, 1991). Recent developments in diagnostic kits for ciguatera toxin have seen the commercial release of several immunological assays. Increased testing for ciguatera toxin using such kits would help define the magnitude of the problem. Their use may be economically viable with high value fish (e.g. coral trout) as a routine screening test.

Introduction of a system that would allow trace-back of fish to the area where they were caught (e.g. tagging) would help define problem areas. However, such a system is dependent on the reporting/diagnosis of ciguatera poisoning incidents. Currently ciguatera poisoning incidents are under reported because cases are rarely fatal, typically involve few (< 5) people, are sporadic and are easily misdiagnosed by medical staff (Todd, 1995; Lehane, 1999). Adoption of a trace back system would need to be backed up with an educational campaign for clinicians and public health workers to increase their awareness of ciguatera poisoning.

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