Risk Assessment of Cadmium in Australian Wild-Caught Prawn Muscle Tissue

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SUMMARY

The distribution of cadmium among prawns is highly skewed, with only small proportions from any location having a Cd concentration exceeding 0.5 mg/kg. King, Tiger and Banana prawns have mean Cd concentrations << 0.5 mg/kg, while Endeavour prawns have a mean Cd concentration of 0.4 mg/kg. The coral prawn *Metapenaeopsis crassissima* is a minor by-catch species, and the females of this species appear to have uniquely high concentrations of Cd in their muscle tissue.

There were two species found to have relatively high cadmium levels among Australian wild-caught prawn species, the coral prawn *Metapenaeopsis crassissima* and the blue endeavour prawn *Metapenaeus endeavouri*. Removal of these species from the risk assessment model had the same impact on reducing cadmium exposure from prawns as the removal of samples >0.5 mg/kg. Removal of these species would be a more reliable strategy for lowering cadmium exposure from prawns than application of a 0.5 mg/kg ML. The degree of benefit realised by a strategy of high-cadmium species removal has not been determined with sufficient certainty by this assessment, and would need to be considered in the context of costs to the prawn industry.

This quantitative risk assessment demonstrated that the 0.5 mg/kg maximum level (ML) stipulated by the EC may be an ineffective risk minimisation strategy for prawns and could result in the inappropriate rejection of prawn consignments. For typical (median-level) consumers of prawns in Europe, cadmium intake from prawn muscle tissue is minor. Prawns may be a substantial source of cadmium intake for a small proportion of the population (1-2%) who are high prawn consumers, however the dietary consumption patterns in this group are not adequately characterised. The risk assessment provides robust scientific evidence that the application of the European regulatory limit does not reduce dietary intake of cadmium significantly, and could be used to support future negotiations on market access standards for cadmium.

The ability to conduct food safety risk assessments for prawns is limited somewhat by information gaps on specific consumption patterns; the most recent dietary survey conducted for the Australian Bureau of Statistics cannot discriminate below the category of consumption of crustaceans. Community-based surveys of prawn consumption patterns, including information on consumption of “brown meat” (i.e. hepatopancreas tissues) would be a valuable addition to the knowledge base.

INTRODUCTION

Cadmium is a toxic metal that can contaminate air, water and soil. Cadmium can move between these three compartments, and as a consequence of its presence in soils and water, it is found in a wide range of food items of terrestrial, aquatic and marine origin. Cadmium can accumulate differentially in certain tissues, with kidney, liver, muscle and bone being important contributors to the overall body burden. Cadmium is excreted slowly from the body, the biological half-life being measured in decades. Therefore the risks to humans not subject to occupational contact are principally those of chronic, low-dose exposures, with tobacco smoke and diet being the main uptake conduits.

This work presents quantitative exposure and risk assessment considerations for cadmium in Australian wild-caught prawns. Using national and international population-based diet surveys, estimates of prawn muscle consumption patterns are presented. Those estimates, together with historical and contemporary data on cadmium concentrations in Australian prawns, are then used to direct Monte Carlo simulations to produce a risk assessment of the contribution of prawn muscle consumption to dietary cadmium exposure.

Also presented here are reviews of the biomedical literature relating to uptake mechanisms into marine fauna (including prawns) and the human health implications of exposure to cadmium, with a focus on dietary uptake and associated public health recommendations by international food safety regulatory agencies.

HAZARD IDENTIFICATION

CADMIUM CHEMISTRY

Cadmium, zinc and mercury are transition metals, occupying Group 12 of the periodic table. Zinc is an important component of many enzymatic reactions; cadmium and mercury are toxic and as metal (II) ions can substitute for Zn in enzyme structures [1]. Cd is stable in the Cd(II) (aq) ion configuration. The ionic radius of Cd, at 109pm, is sufficiently close to that of calcium (114pm), so Cd can accumulate in bone [1].

CADMIUM IN EDIBLE PRAWNS

Research work to detect and quantify cadmium in prawn tissues commenced in the early 1970s. Many studies have subsequently been published, describing cadmium concentrations ranging between "nil detects" and 31 mg/kg. A systematic review of cadmium in edible prawns, comprising 148 separate publications, is presented in Table 1 of the accompanying report: *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium*.

DISTRIBUTION OF CADMIUM IN PRAWN TISSUES

Observational and experimental studies have demonstrated that the decapod hepatopancreas is the organ in which the highest concentrations of cadmium, other trace metals and contaminants are generally found. Freshwater, estuarine and marine crayfish and crabs concentrate cadmium in the hepatopancreas [2-4]; indeed, alerts and advisories to avoid eating crab or lobster tomalley are made by some food safety experts and public health agencies due to excess levels of cadmium and other natural and anthropogenic contaminants in the hepatopancreas, e.g.

http://www.health.ny.gov/press/releases/2008/2008-08-01_do_not_eat_the_tomalley.htm

A broadly similar pattern for tissue distribution of cadmium is seen in prawns, whereby the hepatopancreas, which is located in the prawn head, accumulates higher Cd concentrations than other tissues (muscle or carapace). Pourang and Dennis examined Cd, Zn and Cu in hepatopancreas, exoskeleton and abdominal muscle of specimens of two penaeid prawn species captured from the Persian Gulf. Cadmium levels followed a gradient from highest in hepatopancreas to lowest in muscle [5]. Pierron *et al* investigated the influence of salinity and hypoxia on Cd bioaccumulation in *Palaemon longirostris*, an estuarine caridean prawn. Again, the hepatopancreas was the principal site of Cd concentration, with levels in the hepatopancreas almost 50-fold higher than in muscle. Decreased salinity increases the bioavailability of cadmium, and hypoxia results in increased absorption of Cd across the gills due to hyperventilation [6]. Animals living in low salinity environments, including freshwater systems, generally have higher cadmium concentrations in their tissues [7]. Canli *et al* found Cd concentrations in hepatopancreas were 7 to 19-fold higher than in muscle of a penaeid species from the Mediterranean; high Cd levels were also found in the gills [8]. Other workers investigating the tissue distribution of experimentally dosed cadmium in prawns have noted that the highest levels are found in the hepatopancreas [9-11]. Work conducted for the accompanying *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium* accords with international findings: analysis of hepatopancreas tissues has shown that cadmium concentrations are consistently higher than in muscle tissues. A detailed summary of the international peer-reviewed literature on cadmium levels measured in prawn hepatopancreas is presented in Table 1 of the accompanying *2012-13 survey* report.

CADMIUM EXPOSURE AND UPTAKE IN HUMANS: DIETARY SOURCES, METALLOTHIONEIN BINDING

Metallothioneins (MTs) are metal-binding low molecular weight proteins found in many animal tissues; in humans and experimental animals they are found in highest concentration in liver, kidney, pancreas and testis [12]. MTs appear to have important functions in homeostasis, transport and storage of essential trace metals like Zn and Cu; for non-essential metals, and in particular for Cd, for which MTs have high affinity, these proteins perform sequestration and detoxification roles [12, 13]. The question of the relative bioavailability of bound cadmium from dietary sources compared to that of Cd^{2+} in drinking water has been investigated experimentally in a few studies. This topic was reviewed by Asagba [14]; there would appear to be broadly similar absorption efficiencies for organic and inorganic forms, although there are conflicting reports in the literature. Metallothioneins are found in animal, fungal and plant domains, although debate surrounds the role of MTs in cadmium detoxification in plants, a role that is also performed by enzymatically-synthesised phytochelatins [15-17]. So while protein-bound cadmium from a variety of plant and animal foods appears to be effectively bioavailable, other dietary and metabolic factors can have considerable impact on the absorption efficiency of Cd. Experimental studies on rodents and human epidemiological investigations have demonstrated that fibre-rich diets inhibit intestinal absorption of Cd, whereas relative iron deficiency conditions (as seen, for example, in premenopausal women) will cause enhanced assimilation of dietary cadmium [14, 18]. Diets low in protein result in increased uptake of cadmium, and long-term nutritional status with respect to iron, zinc and calcium has an important influence on dietary cadmium assimilation. Chronic dietary deficiencies in either Fe, Zn or Ca can result in increased Cd uptake [14].

There is an apparent dearth of information regarding the bioavailability of cadmium in specific food matrices; this information gap may have particular implications for seafood consumption and the seafood industry. Vahter *et al* conducted a small volunteer study in adult women to investigate the relative bioavailability of cadmium from (unspecified) shellfish. Two groups of non-smokers were studied: those eating a mixed diet low in shellfish, and a group reporting regular shellfish consumption. The high shellfish diet contained twice the level of cadmium, yet there were no differences in blood or urine cadmium between the two groups, which was suggestive either of lower Cd absorption from shellfish, or different toxicokinetic profiles [19]. This and other studies have tended to fuel some level of disquiet in the seafood industry, as outlined by Kruzynski, who has questioned some risk assessment processes for Cd exposure and regulatory oversight of Cd in bivalves [20].

CADMIUM AND HUMAN HEALTH: RENAL TOXIN, CANCERS, BONE DEMINERALISATION

While some workers have considered the possibility that cadmium may be an essential ultra-trace element, with studies from the 1970s providing some support for this supposition [21, 22], current expert opinion places cadmium as a contaminant of a wide range of ecosystem compartments, including food webs and human food supplies [23-25].

Acute cadmium intoxication is rare; occupational exposures have occurred from inhalation of cadmium fumes. Food poisoning cases have been attributed to contamination of food and drinking water by cadmium in cooking utensils and containers, and cadmium in metal solder [25]. Chronic, sub-clinical exposure to cadmium is the main concern for the general population. Cadmium and cadmium compounds are listed as

Group I carcinogens, i.e. they are carcinogenic to humans [24]. Occupational and population epidemiology studies lend support for an increase in lung cancers related to cadmium exposure; prostate and renal cancers have also shown positive associations with cadmium exposure. Dietary or respiratory cadmium exposure is also suspected to contribute to increased risk of cancers of the breast, bladder and endometrium. Cadmium is thought to act as a metalloestrogen, thus implicating it in the aetiology of oestrogen-sensitive cancers such as those of the breast, uterus and prostate [26, 27]; however, the mechanisms of cadmium-induced carcinogenicity are probably multifactorial [28, 29]. Some retrospective and prospective epidemiological investigations suggest that dietary cadmium exposure is associated with an increased risk of breast cancer and endometrial cancer [30-34], although the epidemiology is not unequivocal [35]. Experimental exposures in laboratory animals support the conclusions that cadmium is carcinogenic [24, 28].

The kidney is the main target organ for chronic cadmium exposure. Clear dose-response relationships have been reported for airborne cadmium and proteinuria (a non-specific marker of renal damage) in occupational cohorts. Population-based epidemiological investigations from Japan and Belgium support the conclusion that cadmium is a renal toxicant [25, 36]. Glucosuria, impaired concentration capacity and excess excretion of calcium and phosphorus are also seen in cadmium-induced nephropathy [36].

Bone disease is a recognised feature associated with chronic cadmium poisoning, manifesting as osteomalacia, osteoporosis and spontaneous fractures. Cadmium-related skeletal pathology is considered to be secondary to renal dysfunction – increased urinary excretion of calcium and phosphorus [36]. Recent prospective epidemiological investigations indicate positive associations between dietary cadmium intake and decreased bone mineral density in post-menopausal women, with concomitant risks of osteoporosis and fractures [37].

HAZARD CHARACTERISATION

TOLERABLE DIETARY INTAKES OF CADMIUM

The 2011 Joint FAO/WHO Expert Committee on Food Additives (JECFA) safety evaluation of cadmium in food [38] established a provisional tolerable monthly intake (PMTI) of 25 µg/kg bw, and the European Food Safety Authority (EFSA) in the 2009 Scientific Opinion of the Panel on Contaminants in the Food Chain established a tolerable weekly intake (TWI) of 2.5 µg/kg bw for cadmium in food [39]. The JECFA level, expressed as a weekly intake is 5.8 µg/kg bw. Thus the scientific risk assessments of JECFA and EFSA arrived at different safe intake levels, with the EFSA establishing a lower tolerable level than JECFA.

Both the EFSA and JECFA safety assessments utilised the concentration of a low molecular weight protein, beta-2-microglobulin (β2MG) in the urine to establish tolerable intakes of Cd in the diet. An increase in β2MG concentration in urine is an early biomarker of potential kidney impairment. The EFSA and JECFA used the same large database of urinary Cd (as µg/g creatinine) and β2MG concentrations (as µg/g creatinine), but applied different models to determine the urinary Cd concentration at which an increase in β2MG occurs. The concentration at which the increase occurs was examined for individuals of 50 years or older in both models. This age group was selected as cadmium has an average half-life in the kidney of approximately 15 years, and reaches a steady-state concentration in the kidney by age 45-60 years [38].

The model that EFSA used to examine the relationship between urinary Cd levels and β2MG concentrations associated 4 µg Cd/g creatinine with an increase in β2MG concentration. A chemical-adjustment factor of 3.9 was applied to account for individual variation, arriving at a level of 1µg Cd/g creatinine. A model created by Amzal et al (2009) [40] was used to examine the relationship between dietary Cd exposure and a urinary Cd concentration, and determined that 95% of the population would have a urinary Cd concentration below 1 µg Cd/g creatinine if dietary cadmium exposure was 2.5 µg/kg bw/week, and established this as the tolerable weekly intake (TWI).

The model and analysis by JECFA concluded that an increase in β2MG concentration occurs when the concentration of Cd in urine exceeds 5.24 µg Cd/g creatinine. JECFA also utilised the model of Amzal et al [40] to examine the relationship between dietary Cd exposure and urinary Cd concentration and concluded that a dietary Cd intake of 25 µg/kg bw/month, which is 5.8 µg/kg bw/week, to be a safe level [38]. Following the JECFA assessment the EFSA Panel on Contaminants in the Food Chain evaluated the JECFA and EFSA approaches to establishing tolerable intakes and concluded that the previous opinion of the EFSA was appropriate and the TWI of 2.5 µg/kg bw/week was maintained [39].

EPIDEMIOLOGICAL ASSOCIATIONS BETWEEN CADMIUM AND HARMFUL EFFECTS

A number of other epidemiological studies associating dietary Cd intakes and the wide range of harmful effects with which it is associated were considered by JECFA in their safety evaluation of cadmium in food, but these were not utilised to establish dose-response relationships, as either the study results were preliminary, studies produced conflicting or inconclusive results, or the harmful effects occurred at exposure levels higher than those associated with elevation of β2MG concentrations in urine. More recent epidemiological studies, subsequent to the EFSA and JECFA evaluations, have indicated associations between harmful effects and cadmium occurring at exposure levels lower than those associated with early indications of effects on the kidneys.

A recent study examined the dietary cadmium intakes and the occurrence of osteoporosis and fractures in women aged 56-69 yrs who have never smoked [37]. Effects in women were studied as low body iron stores are more common in women and those with low body iron stores have a higher uptake of Cd. Only 0.7% of the women in the study exceeded the EFSA safe dietary Cd intake of 2.5 µg/kg bw/week. Increased risks of osteoporosis and fracture were associated with those whose intake of Cd was >13 µg/day which for a 60 kg woman is >1.5 µg/kg bw/wk. The authors noted that the plant foods that are major contributors to Cd intake, also contain magnesium, iron and fibre which appeared to counteract the negative effects of cadmium on bone [37].

A recent epidemiological study of cadmium levels in urine of children found an increased risk of learning disability and special education utilisation in children from the highest quartile of urinary Cd concentrations compared with those in the lowest quartile [41]. Only four of the 2,189 children in the study had concentrations of Cd/g creatinine above the safe levels determined to avoid potential kidney impairment identified by the EFSA and JECFA in their risk assessments. When these four children were excluded from the study, the increased risk of learning disability and special education utilisation in the highest quartile of urinary Cd concentrations remained. This study examined urinary Cd concentrations in relation to various demographic factors and other variables, but did not quantify the contribution of the various sources of Cd intake (e.g. smoking in their residence, diet, mouthing/accidentally swallowing objects) by the children [41].

Future epidemiological studies should contribute to a better characterisation of dose-response relationships between cadmium and the harmful effects with which it is associated; and the mitigating effects of Fe, Zn, Ca and fibre in the human diet.

EXPOSURE ASSESSEMENT

Exposure assessment is the estimation of the intake of a harmful substance. Human exposure to cadmium (Cd) occurs through smoking, food, water and air, with the largest exposures occurring through smoking and food.

Many, if not most foods have some cadmium in them. The concentrations of Cd vary according to food type, and vary within a food type depending on where it was grown or caught. Total exposure in a person's diet is the sum of intake from each food type (=amount of food type consumed x the concentration of Cd in the food type). A food type with a low concentration of Cd can make the largest contribution to Cd intake, if it is a staple food consumed regularly and in large quantities. Conversely a food type which has a high concentration of cadmium, may only contribute a small amount to cadmium intake if it is consumed infrequently, or in small quantities. This is illustrated by the estimated exposure to consumers from food groups in Europe, where the highest exposures are from the cereals/grains food group and the vegetables/nuts/pulses food group which have moderate concentrations of Cd (mean concentrations 0.016 mg/kg and 0.019 mg/kg respectively), but which are staple foods consumed in large quantities (median consumption 257 and 194 g/day, consumers only). The third highest source of Cd in European diets is offal, which has a high mean concentration of Cd (0.126 mg/kg), but is consumed in smaller amounts (median consumption 24 g/day, consumers only) (Table 1).

Table 1. Estimated consumer exposure to Cadmium (Cd) by food group in European adult diets (see notes 1, 2)

Food Group	Mean Cd (mg/kg = µg/g)	Median food group consumption (consumers only) (g/day)	Cd exposure from food group (consumers only) (µg/day)
Cereals/grains	0.016	257	4.2
Vegetables, nuts, pulses	0.019	194	3.7
Offal	0.126	24	3.0
Starchy vegetables/roots	0.021	129	2.7
Alcoholic beverages	0.0004	413	1.7
Fish and seafood	0.027	62	1.7
Sugars	0.026	43	1.1
Milk and dairy products	0.004	287	1.1
Coffee, tea, cocoa	0.002	601	1.1
Meat	0.008	132	1.0
Juices, soft drinks, bottled water	0.001	439	0.4
Miscellaneous foods	0.024	14	0.3
Fats (vegetable and animal)	0.006	38	0.2
Tap water	0	349	0.1
Eggs	0.003	25	0.1

1. From EFSA Scientific Opinion, Cadmium in food (2009) [39]
2. Consumers in this sense are those who consume the food type e.g. vegetarians are non-consumers of meat, and have nil exposure to Cd from this food type

In addition to individual variations in diet, on a broader scale dietary patterns vary from country to country, and can vary from region to region within countries. In a multicultural country like Australia, dietary patterns may vary between ethnic and religious groups. In most diets cereals and vegetables are the main sources of Cd exposure from food, but this does not always apply. In Liaoning, a coastal, highly industrialised province of China, it is estimated that seafood is the source of 88% of Cd exposure from food (Table 2). This may reflect both the quantity and types of seafood in the diet and the extent of Cd pollution in harvest areas (Fig 1).

Table 2. Estimated adult exposure to Cadmium from food in some provinces of China (2007) (see note 1)

Province	Mean Cd exposure (µg/day)	Main sources of Cd exposure in foods (estimated % of total dietary exposure)
Heilongjiang	14.3	Meat (93%)
Liaoning	10.9	Seafood (88%)
Hebei	5.5	Legumes (36%) + Seafood (34%)
Shanxi	2.5	Vegetables (62%)
Henan	5.7	Cereals (50%) + Vegetables (45%)
Ningxia	1.1	Cereals (39%) + Potatoes (23%)
Shanghai	9.9	Vegetables (69%)
Fujian	30.9	Cereals (40%) + Vegetables (37%)
Jiangxi	47.0	Cereals (92%)
Hubei	20.4	Meat (53%) + Vegetables (41%)
Sichuan	76.7	Cereals (85%)
Guangxi	21.6	Cereals (37%) + Vegetables (25%)
National average	20.8	Cereals (32%) + Vegetables (25%)

1. From JECFA (2011) [38]

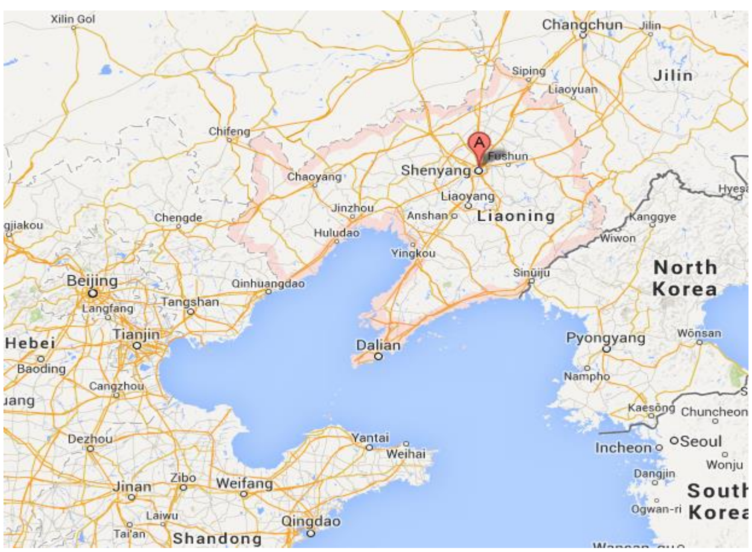


Figure 1. Liaoning Province of China (Google Maps)

Within the food group "fish and seafoods", Cd contamination levels are not uniform in sub-groups as accumulation varies according to the feeding habits and physiology of species (Table 3). Oysters and other bivalve molluscs accumulate the highest concentrations of Cd, and the muscle meat of finfish accumulates the lowest concentrations.

Table 3. Concentration of cadmium in fish and seafoods sampled in Europe (mg/kg) (see notes 1-4)

Seafood Category	n	% Samples <LOD	P5	Median	Mean	P95	Max
Fish and fish products - muscle meat	10172	47	0.00	0.01	0.02	0.1	0.66
Crustaceans - muscle meat	1896	44	0.00	0.01	0.09	0.5	2.30
Cephalopods	1017	13	0.01	0.12	0.28	1.2	2.30
Bivalve molluscs other than oysters	1388	2.3	0.03	0.16	0.38	1.4	4.53
Oysters	164	0.7	0.09	0.24	0.29	0.84	0.97

1. Data collated by the European Food Safety Authority (EFSA, 2009) [39]
2. n = number of samples, LOD = level of detection
3. P5 - 5% of samples had a Cd conc. ≤ P5 concentration.
4. P95 - 5% of samples had a Cd conc. ≥ P95 concentration.

A quantitative estimate of cadmium (Cd) intake from eating prawns requires knowing or estimating the quantity of prawns consumed and the concentration of cadmium in prawns. Mean values are frequently reported to indicate the level of exposure to the population of a country, which are calculated as follows:

$$\text{Mean exposure to cadmium from prawns per person per day } (\mu g/\text{day}) = \text{mean amount of prawns consumed per person per day } (g/\text{day}) \text{ x mean concentration of cadmium in prawns } (\mu g/g) \quad \textbf{Eq. 1}$$

Exposure is often quantified in relation to body weight (kg) rather than "per person", which is calculated as follows:

$$\text{Mean exposure to cadmium } (\mu g/\text{kg bw/ day}) = \frac{\text{Mean exposure to cadmium per person } (\mu g/\text{day})}{\text{Mean body weight per person (kg)}} \quad \textbf{Eq. 2}$$

Mean consumption averaged across a total population however has limited value in characterising the consumption of prawns and other crustaceans, as this food type is not eaten by all, and of those who do eat prawns they might not do so on a regular basis.

As described in the Hazard Identification and Hazard Characterisation sections of this report, impacts on kidney function and other disease from cadmium exposure in food are typically a result of low level chronic exposure and accumulation of cadmium in the body over time, not acute illness from single or short term exposures. Therefore, rather than a safe daily intake of cadmium in food, estimated safe or tolerable intake levels to avoid disease are specified over a week or month. Cadmium exposure in this report is specified as weekly exposure (µg/kg bw/week).

PRAWN CONSUMPTION

BACKGROUND

A driver for this risk assessment was the rejection of some Australian prawn consignments to Europe because test samples exceeded the maximum limit (ML) of 0.5 mg/kg. Spain is Australia's largest prawn export market in Europe, however only a minor proportion of prawns exported by Australia go to Spain (Table 4). The largest export markets are countries in the Asian region, with Japan the largest export market for Australian prawns (Table 4).

Table 4. Australian prawn exports by major export destination countries (t) (see note)

	Whole		Headless		Other		Total	
	2009–10	2010–11	2009–10	2010–11	2009–10	2010–11	2009–10	2010–11
Indonesia	252	0	0	0	0	0	252	0
Thailand	0	0	27	18	80	5	107	22
Taiwan	0	24	0	0	0	0	-	24
New Zealand	113	131	35	17	0	0	148	147
Spain	142	214	0	0	0	0	142	214
Korea, Rep. of	0	210	0	41	0	0	0	251
Other	214	170	36	76	46	211	296	458
Malaysia	0	0	48	218	121	272	169	490
Vietnam	161	436	0	0	67	232	228	669
Hong Kong	518	799	308	64	40	21	866	883
China	543	888	18	37	50	410	611	1335
Japan	1821	1896	19	30	0	0	1839	1925
Total	3764	4768	491	499	404	1151	4659	6419

Source: Australian Fisheries Statistics (ABARES)

In total, approximately 20% of Australian wild-caught prawns are exported, while the majority remaining are consumed by Australians (Table 8). Spain, Japan and Australia were originally selected as the countries for inclusion in this risk assessment. However as explained in further detail below, suitable food consumption data for Japan were not available, and there were significant limitations in the data available for Australia, including that the last comprehensive Australian food consumption survey was conducted in 1995.

Information on food consumption is sourced from national diet surveys. Survey data for national diet surveys in European countries is collated by the European Food Safety Authority (EFSA) and is readily accessible from the EFSA Comprehensive European Food Consumption Database (http://www.efsa.europa.eu/en/datexfoodcdb/datexfooddb.htm). In Australia the National Nutrition Survey (NNS) conducted in 1995 is the primary source of food consumption data. In Japan a National Nutrition Health and Nutrition Survey is conducted on a regular basis, with the last survey in 2007, but the results of this survey could not be accessed for this assessment.

The World Health Organisation has developed the Global Environmental Monitoring System (GEMS) "cluster diets" to represent the variety of diets in the world (http://www.who.int/foodsafety/chem/gems/en/index1.html). In the most recent update countries are clustered into 17 groups according to their food consumption patterns, and consumption amounts are reported as mean consumption for total populations.

Due to the wide range of foods consumed, dietary surveys do not report consumption for all food types, and foods are grouped into categories. Data are available for the category 'Crustacea' from dietary surveys for Spain, France, UK and GEMS groups, and for the category 'Crustacea and Molluscs' from the Australian NNS 1995 survey (Table 5).

For the purpose of this assessment "prawns" and "shrimp" are considered synonymous. In addition to prawns, seafoods in the division "crustacean" include all types of crabs and lobsters/crayfish. A wide variety of seafoods are in the category molluscs including scallops, oysters, clams, abalone, octopus, squid, mussels and cockles.

The mean consumption of Crustacea in the GEMS cluster diets range from 0.1 – 5.3 g/day. The highest mean consumption (5.3 g/day) among the GEMS cluster diets is the Group 9 diet which includes China, Indonesia, Thailand and 11 other countries in the South-East Asian region.

Table 5: Mean (median) consumption of crustaceans

Country	Survey	Food category	Age group	All subjects (g/day)	Percentage consumers (%)	Consumers only (g/day)
Spain	AESAN_FIAB 1999-2001	Crustaceans	Adult	5.2	33.1	15.8
France	INCA2 2005-07	Crustaceans	Adult	1.6	27.2	5.6
United Kingdom (UK)	NDNS 2000-01	Crustaceans	Adult	2.6	23.7	11.2
Australia	NNS 1995	Crustaceans and Molluscs	Adult	2.7	2.7	(69.5)
GEMS Group 07 (Australia, UK, France and 7 other countries)	WHO GEMS	Crustaceans	All	1.2	na	na
GEMS Group 08 (Spain and 3 other countries)	WHO GEMS	Crustaceans	All	0.09	na	na
GEMS Group 09 (China and 13 other countries)	WHO GEMS	Crustaceans	All	5.3	na	na
GEMS Group 10 (Japan and 13 other countries)	WHO GEMS	Crustaceans	All	1.3	na	na

Sources: Data from Spain, France and UK surveys sourced from the EFSA Comprehensive European Food Consumption Database; Data for Australia from the 1995 National Nutrition Survey; GEMS data from http://www.who.int/foodsafety/chem/gems/en/index1.html ; na=not available

In the 1995 National Nutrition Survey (NNS) for Australia the mean consumption of 'Crustacea and molluscs' for all those aged 19+ years was 2.7 g/day; however the proportion of consumers in this population was only 2.7% (Table 5). This small percentage of consumers indicates that the large majority of Australians do not eat crustacea/molluscs on a regular basis. The median consumption of 'Crustacea and molluscs' by Australian consumers was 69.5 g/day.

A 2004-05 survey of seafood consumption frequency in the city of Melbourne provides more detail on the pattern of prawn consumption (Table 6) [42]. This survey found only a minority of Melburnians eat prawns at least once a week in home (4%) or out of home (9%). The survey results do not indicate the proportion who eat prawns both at home and out of home, but there may be some who do both, suggesting that the proportion of Melburnians who eat prawns at least once a week is within the range 9 – 13%. That survey estimated that approximately one quarter of the population never eats prawns, 41% eat prawns at home only 1-6 times a year, and 34% eat prawns out of home 1-6 times a year. While the size, target populations and methodologies of the 1995 NNS and the 2004-05 Melbourne surveys were quite different and conducted a decade apart, the results are consistent in indicating that among Australians, prawns are a regular contribution to the total diet for a minority only. It is possible that the size of this minority increased between 1995 and 2005 as there was an increase in mean apparent consumption per person in Australia from 4.1 g/day to 5.4 g/day between 1995 and 2005, and a further increase to 6.4 g/day in 2009 (see Equations 3-5 and Table 8 for calculation method).

Table 6: Frequency of eating prawns in Melbourne in 2005

	In Home % Respondents	Out of Home % Respondents
More than once a week	1	3
Once a week	3	6
At least once a week	4	9
Once a fortnight	7	7
Once a month	15	18
At least once a month	26	35
Six times a year	10	8
Four times a year	8	9
Three times a year	5	6
Twice a year	9	7
Once a year	9	4
At least once a year	68	69
Less than once a year	6	4
Never	26	22

Source: Ruello et al (2005) [42]

Among the 19+ age group consumers of 'Crustacea and Molluscs' in the 1995 Australian NNS, the median intake was 69.5 g/day. Prawn consumption for adult Australians was estimated by adjusting the 'Crustacea and Molluscs' consumption by a factor calculated as the ratio of the Apparent Prawn Consumption (APC) to the Apparent 'Crustacea and Mollusc' Consumption (AC&MC) (Eq. 3-5). The APC and AP&MC (Table 7) were calculated with Equations 2-4, using Australian production, import and export data sourced from the Food and Agriculture Organisation (FAO) FishStatJ database for the year 2009 (http://www.fao.org/fishery/statistics/software/fishstatj/en).

$$\text{Apparent Prawn Consumption (t)} = \text{Prawn production (t)} + \text{Prawn import (t)} - \text{Prawn export (t)} \qquad \textbf{Eq. 3}$$

$$\text{Apparent 'Crustacea and Mollusc' Consumption (t)} = \text{'Crustacea and Mollusc' production (t)} + \text{'Crustacea and Mollusc' import (t)} - \text{'Crustacea and Mollusc' export (t)} \qquad \textbf{Eq. 4}$$

$$\text{Australian adjustment factor} = \frac{\text{Apparent prawn consumption (APC)}}{\text{Apparent 'Crustacea and Mollusc' consumption (AC\&}MC\text{)}} \qquad \textbf{Eq. 5}$$

Table 7. Apparent consumption of Crustaceans, Molluscs and Prawns in Australia in 2009

	Production (t)	Import (t)	Export (t)	Apparent Consumption (t)
Crustaceans	42 143	33 435	14 340	61 238
Molluscs	34 142	27 267	5 674	55 715
Crustaceans and Molluscs	76 264	60 702	20 014	116 952
Prawns	24 189	31 360	4 228	51 321

Source: FAO FishStatJ database, http://www.fao.org/fishery/statistics/software/fishstatj/en

The Australian adjustment factor to estimate prawn consumption from 'Crustacea and Molluscs' consumption was 0.44. Applying this factor to the median consumption of 'Crustacea and Molluscs', the estimated median consumption of prawns among Australian adult consumers was 30.6 g/day.

PRAWN CONSUMPTION IN JAPAN

Dietary intake data that characterised the % consumers and median intake by consumers could not be accessed for Japan. An indication of relative consumption levels among countries is obtained from the Apparent Consumption (AC) which is the gross sum of production and imports less exports. This differs from dietary consumption as it includes wastage (e.g. head, gut and exoskeleton removal, food thrown out at retail and wholesale outlets, restaurants or homes) and any use of the commodity for other purposes. Among the three countries of focus for this assessment (Spain, Japan and Australia), Spain had the highest mean apparent consumption per person in 2009, while the mean apparent consumption per person was similar in Australia and Japan (Table 8).

In Australia regular weekly or fortnightly consumption appears concentrated among a minority of 'high' consumers. However this may not be the pattern in Japan, where regular consumption of smaller quantities per meal might be consumed by a much broader section of the population. In the absence of data to better characterise consumption patterns in Japan, France and the UK were included in the assessment to represent countries with a lower dietary intake level across a broader section of the population; and Spain was included not only as the most important export destination in Europe, but the country with the highest apparent consumption (Tables 5, 8 and 9).

Table 8. Apparent Consumption (AC) of prawns by country in 2009

	Australia	Japan	Spain	France	UK
Production (t)	24 189	20 279	8 655	806	1 123
Import (t)	31 360	266 032	154 137	105 919	85 551
Export (t)	4 228	547	29 162	12 969	14 583
Apparent Consumption (t)	51 321	285 764	133 630	93 756	72 091
Population (millions)	22	128	47	65	62
Mean Apparent Consumption/person/yr (kg)	2.3	2.2	2.8	1.4	1.2
Mean Apparent Consumption/person/day (g)	6.4	6.1	7.8	4	3.2

Source: FAO FishStatJ database, http://www.fao.org/fishery/statistics/software/fishstatj/en

PRAWN CONSUMPTION IN SPAIN, FRANCE AND THE UNITED KINGDOM

The data that best characterised crustacean consumption patterns were available for European countries from the EFSA Comprehensive European Food Consumption Database, and included the mean, median and the 5th, 10th, 95th, 97.5th and 99th percentiles for consumption by consumers. These data allow a more meaningful estimation of cadmium intake by those who eat this food type; and within this group, by those who eat small, median and large quantities of crustaceans. France, the UK and Spain represent a range of crustacean consumption levels.

Adult consumption data were utilised. Cd intake occurs throughout life with the majority occurring in the adult years. There is a higher intake/kg bw in childhood, however as the established health impacts are associated with long term intake, the larger intakes of crustaceans in adult years is the most relevant. In addition the sample sizes of adult consumers were large enough in the available dietary surveys for Spain, France and the UK, to be statistically robust at the 99th percentile.

Table 9. Consumption of Crustaceans by Adult Consumers in Spain, France and the UK (g/day) (see note)

	Sample size (n)	P5	P10	P50	P95	P97.5	P99
Spain	325	3	5	12.3	46.3	60	68.8
France	620	0.7	1.1	4.3	15.3	18.5	22.4
UK	409	1.3	2	8	28.6	37	46.4

Source: EFSA Comprehensive European Food Consumption Database

Note. P5=5% of consumers, consume ≤ this quantity of prawns (g/day); P50=50% of consumers, consume ≤ this quantity of prawns (g/day); P95=5% of consumers, consume > this quantity (g/day); P99=1% consumers, consume > this quantity (g/day)

Prawn consumption in a particular country was estimated by adjusting "Crustacea" consumption by a factor calculated as the ratio of the apparent prawn consumption to the apparent crustacea consumption for that country. "Crustacea" includes all prawn, crab and lobster species. The apparent prawn consumption and apparent crustacean consumption were calculated from country production, import and export quantities sourced from the Food and Agriculture Organisation (FAO) FishStatJ database for the year 2009 (available at http://www.fao.org/fishery/statistics/software/fishstatj/en). The equations to calculate the APC:ACC ratio (Table 10) were Eq. 3, 6 and 7.

$$\text{Apparent crustacean production (t)} = \text{Crustacean production} + \text{Crustacean import} - \text{Crustacean export} \quad \textbf{Eq. 6}$$

$$\text{APC:ACC Adjustment factor} = \frac{\text{Apparent prawn consumption (APC)}}{\text{Apparent crustacea consumption (ACC)}} \quad \textbf{Eq. 7}$$

Table 10. Ratio Apparent Prawn Consumption (APC):Apparent Crustacean Consumption (ACC) for Spain, France and the UK in 2009

	Apparent Prawn Consumption (t)	Apparent Crustacean Consumption (t)	APC:ACC Adjustment Factor
Spain	133 630	176 143	0.76
France	93 756	143 628	0.65
UK	72 091	111 381	0.65

Prawn consumption (Table 11) was estimated by application of the APC:ACC adjustment factors (Table 10) to crustacean consumption amounts from the dietary survey data (Table 9).

Table 11. Estimated prawn consumption by adult consumers by country (g/day) (see note)

	P5	P10	P50	P95	P97.5	P99
Spain	2.3	3.8	9.4	35.2	45.6	52.3
France	0.5	0.7	2.8	9.9	12	14.6
UK	0.8	1.3	5.2	18.6	24.1	46.4

Note. P5=5% of consumers, consume ≤ this quantity (g/day); P50=50% of consumers, consume ≤ this quantity (g/day); P95=5% of consumers, consume > this quantity (g/day); P99=1% consumers, consume > this quantity (g/day)

Additionally available from the European national dietary surveys in the EFSA Comprehensive European Food Consumption Database is consumption as g/day per kilogram body weight. These values, adjusted by the APC:ACC ratio, were used as prawn consumption values for the exposure assessment. The minimum consumption values were estimated as 10% of the P5 value and the maximum consumption values were estimated as 110% of the P99 value (Table 12).

Table 12. Estimated Prawn Consumption by Adult Consumers by country (g/kg bw/day) (see note)

	Min	Max	P5	P10	P50	P95	P97.5	P99
Spain	0.003	0.085	0.033	0.0458	0.13	0.546	0.611	0.772
France	0.0007	0.274	0.0066	0.01	0.0411	0.152	0.186	0.249
UK	0.0009	0.467	0.0089	0.0174	0.071	0.26	0.359	0.425

Note. P5=5% of consumers, consume ≤ this quantity (g/kg bw/day); P50=50% of consumers, consume ≤ this quantity (g/kg bw/day); P95=5% of consumers, consume > this quantity (g/kg bw/day); P99=1% consumers, consume > this quantity (g/kg bw/day)

CADMIUM IN AUSTRALIAN WILD-CAUGHT PRAWNS

Two sets of data of Cd levels in Australian wild-caught prawns were available for this assessment. The first, a large dataset of over 2300 samples that were collected and analysed between 1991 and 2010, was a collation of data collected by various companies and the Australian National Residue Survey (NRS). The second data set were 140 prawn samples collected in a representative survey conducted for this project in 2012 and 2013. The representative sampling design for the sample collection in 2012-2013 dataset was based on production volumes from the four (4) major Australian prawn fisheries and random sampling based on production quantities in small scale spatial areas within these major fisheries. The sampling, handling and analytical techniques, and level of quality assurance are well defined for the 2012-2013 dataset, however this information was not available for the data in the historical dataset. The design of the 2012-2013 sampling program and collection, processing and testing methods are described in the accompanying report: *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium*.

At the time of preparing a first draft of this report the 2012-2013 dataset was not available, and a preliminary exposure assessment and risk characterisation were undertaken utilising the historical dataset. The characteristics of the historical dataset are presented here for comparison with the 2012-2013 dataset, however due to the representative nature of the sampling design of the 2012-2013 dataset and the quality control associated with the processing and analysis of samples, the exposure assessment and risk characterisation in this report were prepared utilising only the 2012-2013 dataset.

THE HISTORIC DATA SET

The historic data set illustrated the pattern of Cd levels in Australian wild-caught prawns. The historic dataset included samples of banana, tiger, king and endeavour prawns (Fig 3). Some samples of banana, tiger, king and endeavour prawns had Cd >0.5 mg/kg (the European maximum limit (ML)) for prawn muscle tissue. Samples with Cd concentrations >0.5 mg/kg occurred across northern Australia north of 30° latitude (Fig 2). The Cd concentrations in 90% of samples in the historic data set were <0.5 mg/kg. While there may be some variation in cadmium accumulation among species or genera of prawn, it appeared from this dataset that the most significant factor in cadmium accumulation in Australian wild-caught prawns is their geographic location.

The tissue type for which Cd levels were determined in the historic dataset were specified as "tail", "flesh", "head", "whole", or not specified at all. The highest mean and median Cd levels were in the prawns where the tissue tested was specified as "head" or "whole", as Cd accumulation is concentrated in the hepatopancreas in the head part of the prawn. The Cd concentrations in the samples where the tissue type was not specified were within the range of concentrations in those samples where the tissue type was "tail" or "flesh" (Table 13).

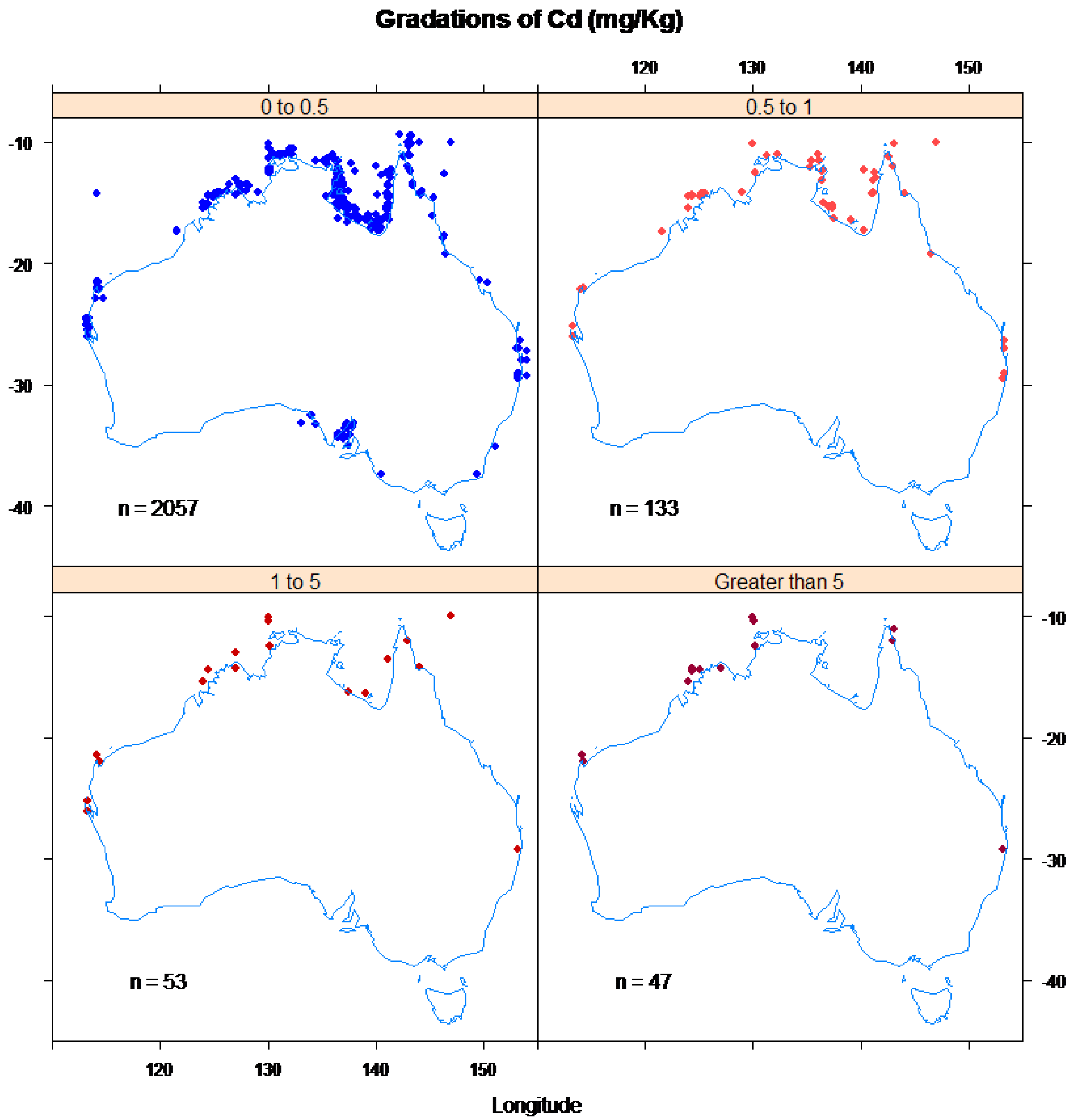


Figure 2. Sampling locations of prawn samples and cadmium concentrations in the historic dataset

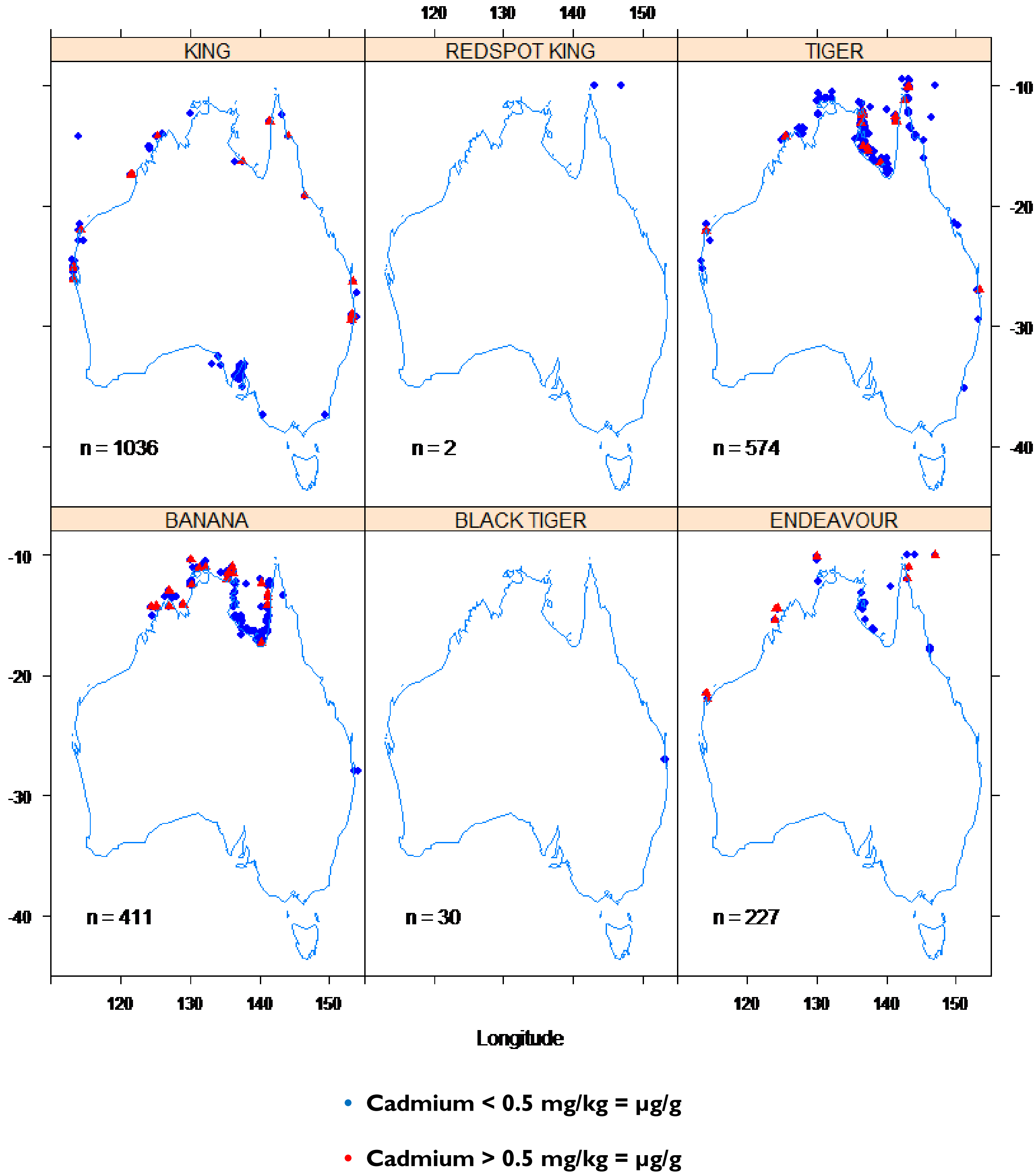


Figure 3. Sampling locations of species in the historic dataset

Table 13. Data sources, tissue types and Cd concentrations in Australian wild-caught prawns

Data Source	Number of samples (n)	Sample Type	Range Cd (µg/g)	Mean Cd (µg/g)	Median Cd (µg/g)
A	1494	Tail or Flesh	0.009 – 7.7	0.21	0.11
B	273	Not specified	0.005 – 0.77	0.15	0.11
C	111	Not specified	0.025 – 0.89	0.099	0.025
D	62	Not specified	0.01 – 0.07	0.028	0.027
E	15	Not specified	0.04 – 0.51	0.23	0.17
NRS (a)	65	Tail or Flesh	0.022 – 0.52	0.16	0.13
NRS (b)	283	Head or Whole	0.01 - 23	2.28	0.22
SARDI 2012-2013	140	Muscle tissue	0.01 - 4.6	0.16	0.084

Sources: Historical data from de-identified commercial companies (A-E) and the NRS = National Residue Survey. SARDI 2012-2013 - data from survey conducted for this project.

Where the whole prawn is consumed there will be a higher exposure to Cd due to the consumption of the hepatopancreas. This may be relevant to some individuals or ethnic groups, and anecdotally may be more relevant to the consumption of prawns in Asian countries, although data on consumption ratios of whole versus tail only in different populations were not available for this assessment. Some individuals who de-head and peel prawns in order to access muscle tissues will also aspirate or otherwise use the liquid fraction before discarding the head. Referred to colloquially as "brown meat", "tomalley" or "prawn mustard", the prawn hepatopancreas is regarded by some as a delicacy; many recipes, food blogs and food chat sites can be found extolling the culinary virtues of this tissue, see for example http://www.davesrecipes.org/recipe/26/Prawns-with-chilli-garlic-and-lemon/, http://www.bbc.co.uk/food/techniques/peeling_prawns, http://www.sydneyfishmarket.com.au/?TabId=171

This assessment is focused on the consumption of muscle tissue in the tail of the prawn only. Exposure to cadmium from whole prawns or prawn tomalley should be assessed separately. The leaching of cadmium from whole prawns or prawn heads in dishes where the medium in which prawns are cooked is eaten e.g. stocks, soups, braises, paellas, was also not included in this assessment. Leaching is possible but has not been quantified.

THE 2012-2013 DATASET

The details of the 2012-2013 dataset are given in the in the accompanying report: *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium*. The distribution of Cd concentrations in Australian wild-caught prawns in this survey is presented as a distribution in Fig. 4. The distribution illustrates that the Cd concentration in 4.3% of the samples exceeded the European maximum limit (ML) of 0.5 mg/kg for muscle tissue. This frequency of exceedance was similar to that in tail meat samples in the historic dataset (5.3% in prawns sampled 2001-2010).

The distribution is highly skewed with a large proportion of samples having a low Cd concentration, and a minor proportion of samples having high Cd levels above the European ML (Fig 4). This distribution pattern was also observed in the historic dataset. A characteristic of this type of skewness (right skew or positive skew) is that the median value (P50) is lower than the mean value (Table 13).

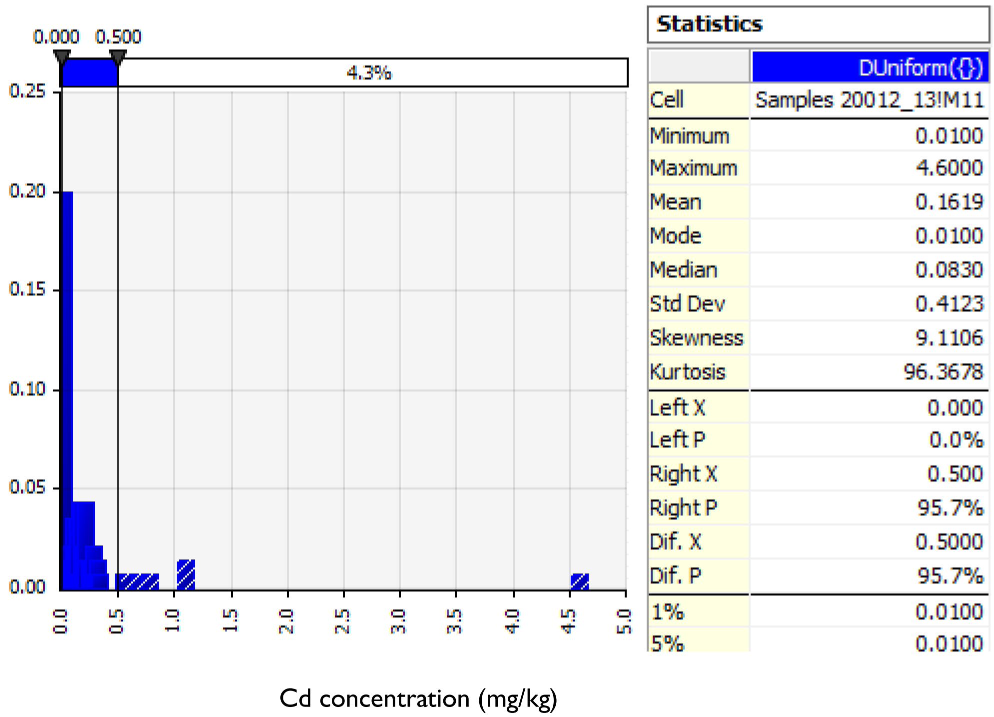


Figure 4. Distribution of Cd concentrations in prawn muscle tissue of samples from the 2012-13 survey.

MODELLING CADMIUM DISTRIBUTION IN PRAWNS CONSUMED IN EXPORT MARKETS

The minor by-catch species *Metapenaeopsis crassissima*, which has the common name coral prawn, was excluded from the model for export countries, as it was considered unlikely this species would be exported because they are a small prawn and non-target species. Subsequently, information became available that while *Metapenaeopsis crassissima* is not exported to Europe, some of these prawns are exported to China, Japan and Vietnam, but we have no information on the tonnage or proportion exported. Some of the prawns sent to Vietnam are returned to Australia after de-shelling as peeled prawns (Errol Sporer, WA Dep't of Fisheries *pers. comm*).

The South Australian catch of Western King prawns, *Penaeus latisulcatus*, is approximately 10% of total production from the four fisheries surveyed, but only 1% of these prawns are exported, representing 0.6% of exported prawns (Table 14). Therefore the distribution of samples was reduced to 121 samples from the WA + NPF + Qld combined fisheries, and one sample from SA. Rather than select a single sample result from the SA samples, a random selection from the SA samples was selected for each iteration of the Monte Carlo simulation. The distribution of Cd concentrations in exported prawns is illustrated in Fig 5. In this distribution 3.3% of samples exceeded the 0.5 mg/kg ML. The mean concentration of Cd from this distribution is 0.141 mg/kg and the median 0.096 mg/kg.

Table 14. Production, export and domestic use quantities for Australian wild-caught prawns

	Production		Export		Domestic		Survey 2012-13	
Fishery	T	%	T	%	T	%	n	%
C'wealth, WA & Qld	22017	90%	3869	99%	18148	88%	123	88%
SA	2383	10%	23	1%	2360	12%	17	12%
Total	24400	100%	3892	100%	20508	100%	140	100%

Source: Australian Fisheries Statistics (ABARE), Quantities are average annual quantities for the years 2008-09, 2009-10 and 2010-11, NSW and Victorian fisheries excluded

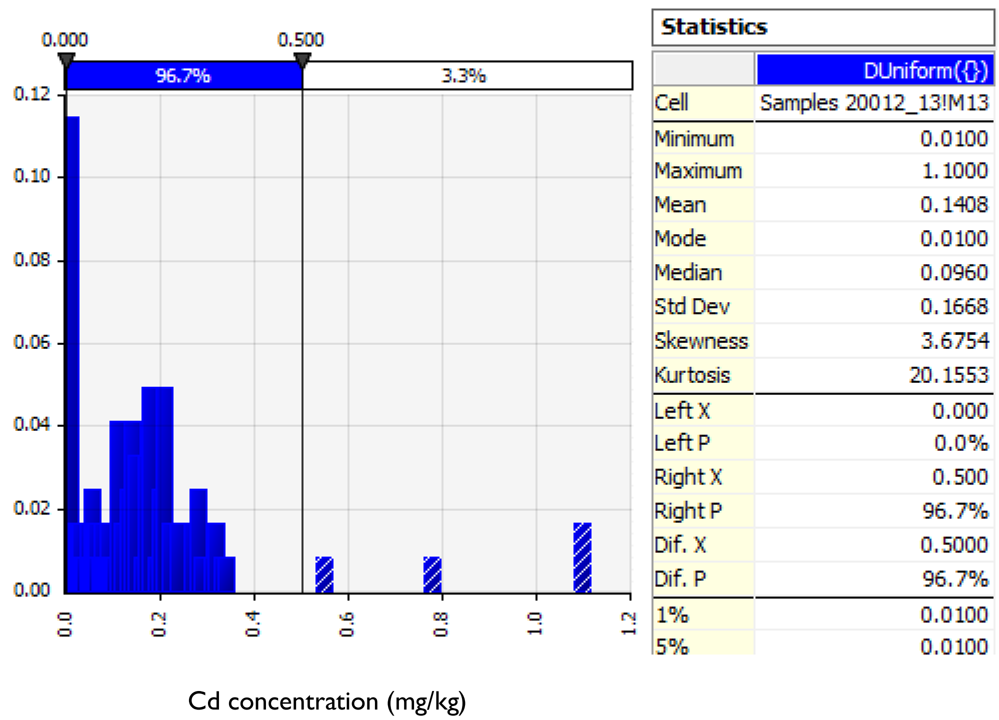


Figure 5. Distribution of Cd concentrations in prawn muscle tissue of exported prawns.

The following two scenarios were also simulated for exported prawns:

1. Removal of samples exceeding the 0.5 mg/kg ML.
2. Removal of samples of *Metapenaeus endeavouri.*

MODELLING CADMIUM DISTRIBUTION IN PRAWNS CONSUMED IN AUSTRALIA

The proportion of samples in the 2012-2013 survey reflected the proportions of wild-caught prawns sold in the domestic market from the SA fishery and the three northern fisheries (Table 14), so there was no adjustment of the 2012-2013 survey distribution of Cd concentrations (Fig 4).

The following three scenarios were also simulated for domestically consumed Australian wild-caught prawns:

1. Removal of samples exceeding the 0.5 mg/kg ML.
2. Removal of samples of *Metapenaeopsis crassissima.*
3. Removal of samples of *Metapenaeopsis crassissima* and *Metapenaeus endeavouri.*

EXPOSURE SIMULATION

Exposure was estimated by Monte Carlo simulation using the risk modelling software "@Risk" version 6.1.2 (Palisade Corporation, New York, USA). Exposure per day was modelled with the country daily prawn consumption data and the relevant distribution of cadmium (Cd) concentrations in the muscle meat of Australian wild-caught prawns as inputs. Yearly exposures for a range of consumption levels were calculated by summing exposure over 365 days, and the average weekly cadmium intake by dividing the yearly intake by 52.

To estimate exposure for those who consume small quantities, those who consume median quantities, and those that consume large quantities of prawns, the 5th, 50th and 95th consumption percentiles (P5, P50, P95) from the Spain, France and UK dietary surveys were utilised as fixed input consumption quantities.

For Australian consumers, the median (50th percentile) prawn consumption amount was the only available datum. Also as consumption quantity was not available in relation to the body weights of the survey participants from the Australian NNS, a 70 kg body weight was used to estimate intake as µg/kg bw/wk. Consequently the only variable input for the Australian simulations was the distribution of cadmium in prawns.

One hundred thousand iterations of the models were run to simulate the distributions of exposure for adult populations.

PRAWNS CONSUMED IN EXPORT MARKETS

The distributions of the weekly intake of cadmium (µg/kg bw/wk) among consumers have a normal distribution shape, with very similar mean and median values (Fig 6-8). The distributions were narrow. For example 90% of median adult consumers in France had a Cd intake in the range 0.037 – 0.045 (mean 0.041) µg/kg bw/wk (Fig 6), and 90% of median adult consumers in Spain had a Cd intake in the range 0.12 – 0.14 (Mean 0.13) µg/kg bw/wk (Fig 8). The mean Cd intakes for low, median and high prawn consumers are detailed in Table 15.

Table 15. Mean weekly Cd intake from the consumption of prawns by adult consumers (µg/kg bw/wk)

	% Consumers	P5	P50	P95
France	27%	0.0066 (0.0004)	0.0407 (0.0025)	0.1498 (0.0093)
UK	24%	0.0088 (0.0005)	0.0702 (0.0044)	0.2567 (0.016)
Spain	33%	0.0327 (0.002)	0.1287 (0.0079)	0.5396 (0.0335)

Note: Standard deviation in brackets; P5, P50, P95 are low, median and high prawn consumers respectively.

The scenarios where all samples >0.5 mg/kg were removed and the scenario where *M. endeavouri* prawns were removed produced similar outcomes with the Cd intake from prawns reduced on average 18% and 21% respectively (Tables 16 and 17).

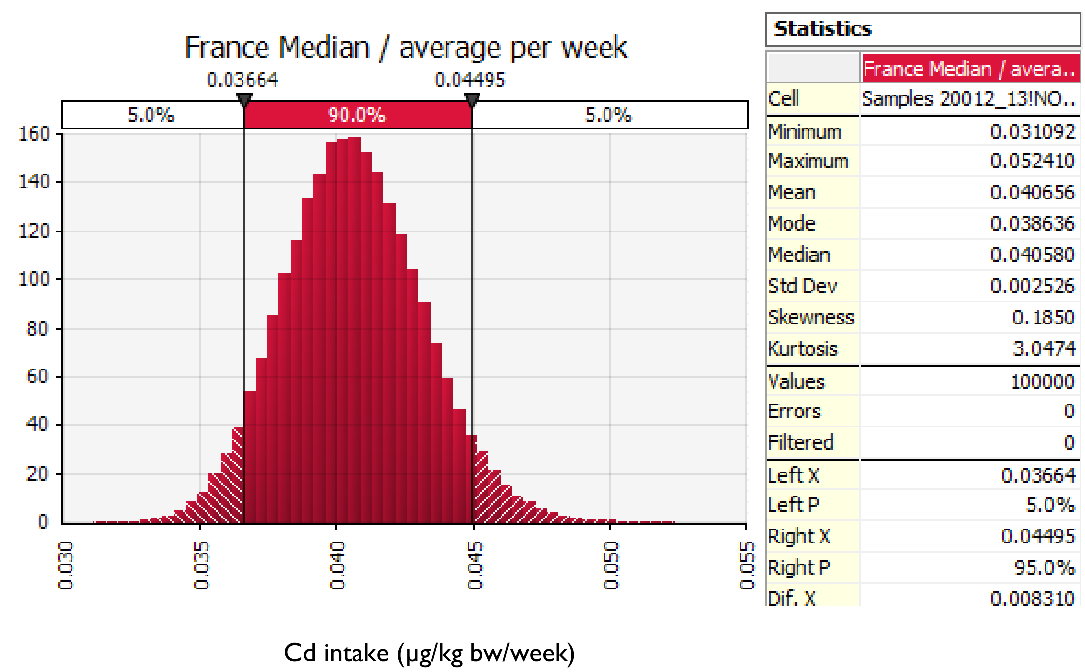
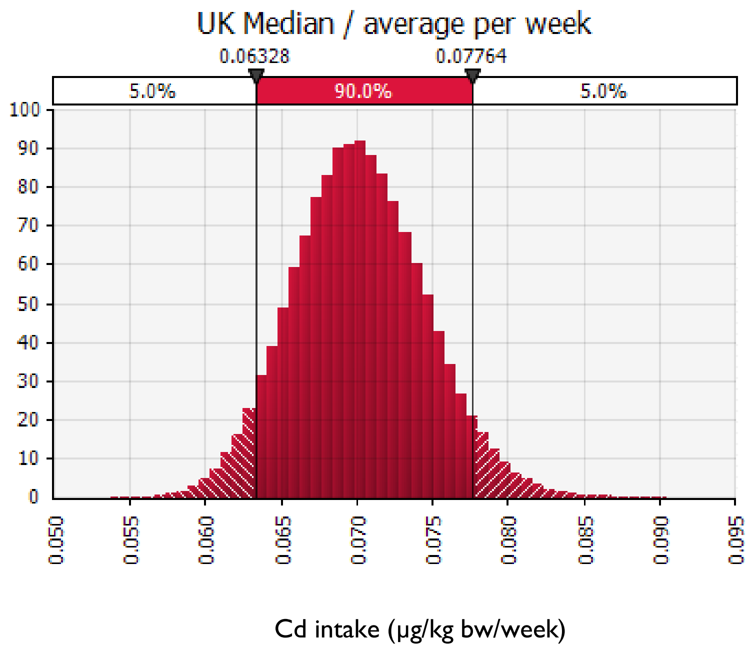
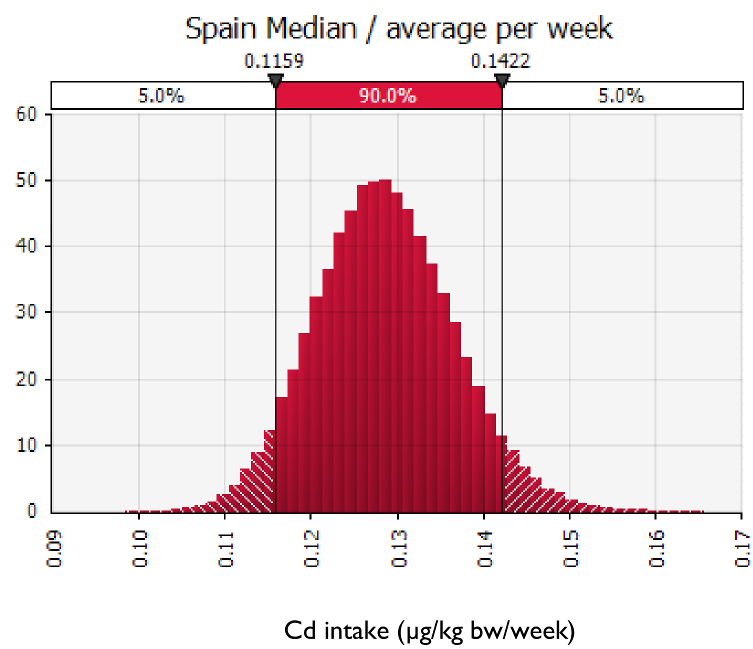


Figure 6. Weekly Cd intake distribution from the consumption of the median prawn amount by adult consumers in France.



Statistics	
	UK Median / average p..
Cell	Samples 20012_13!NO..
Minimum	0.053708
Maximum	0.090533
Mean	0.070229
Mode	0.065302
Median	0.070098
Std Dev	0.004363
Skewness	0.1850
Kurtosis	3.0474
Values	100000
Errors	0
Filtered	0
Left X	0.06328
Left P	5.0%
Right X	0.07764
Right P	95.0%
Dif. X	0.014355

Figure 7. Weekly Cd intake distribution from the consumption of the median prawn amount by adult consumers in the United Kingdom.



Statistics	
	Spain Median / averag..
Cell	Samples 20012_13!NO..
Minimum	0.098390
Maximum	0.165851
Mean	0.128656
Mode	0.127131
Median	0.128415
Std Dev	0.007993
Skewness	0.1850
Kurtosis	3.0474
Values	100000
Errors	0
Filtered	0
Left X	0.1159
Left P	5.0%
Right X	0.1422
Right P	95.0%
Dif. X	0.026297

Figure 8. Weekly Cd intake distribution from the consumption of the median prawn amount by adult consumers in Spain.

Table 16. Export scenario 1: Prawns with Cd > 0.5 mg/kg removed; Cd intake (see notes)

	P5	P50	P95
France	0.005	0.033	0.123
UK	0.007	0.058	0.211
Spain	0.027	0.106	0.443

Notes:
1. P5, P50, P95 are low, median and high prawn consumers respectively
2. Mean weekly Cd intake from the consumption of prawns by adult consumers (µg/kg bw/wk)

Table 17. Export scenario 2: *M. endeavouri* prawns removed; Cd intake (see notes)

	P5	P50	P95
France	0.005	0.032	0.116
UK	0.007	0.055	0.199
Spain	0.025	0.100	0.419

Notes:
1. P5, P50, P95 are low, median and high prawn consumers respectively
2. Mean weekly Cd intake from the consumption of prawns by adult consumers (µg/kg bw/wk)

PRAWNS CONSUMED IN AUSTRALIA

The Cd intake by median consumers of prawns in Australia was higher, with a mean intake of 0.5 µg/kg bw/week and 90% of consumers having an intake in the range 0.4 – 0.62 µg/kg bw/week (Fig 9). However the Australian NNS only identified 2.7% of the adult population as consumers of "Crustacea and Molluscs". It appears that the methodology of this survey only identified the highest consumers, who eat prawns on a weekly basis.

The scenario where all samples >0.5 mg/kg were removed reduced the mean Cd intake from prawns by 36%. The scenarios where *Metapenaeopsis crassissima* prawns were removed, and where both *Metapenaeopsis crassissima* and *Metapenaeus endeavouri* prawns were removed reduced the mean Cd intake by 22% and 40% respectively (Table 18).

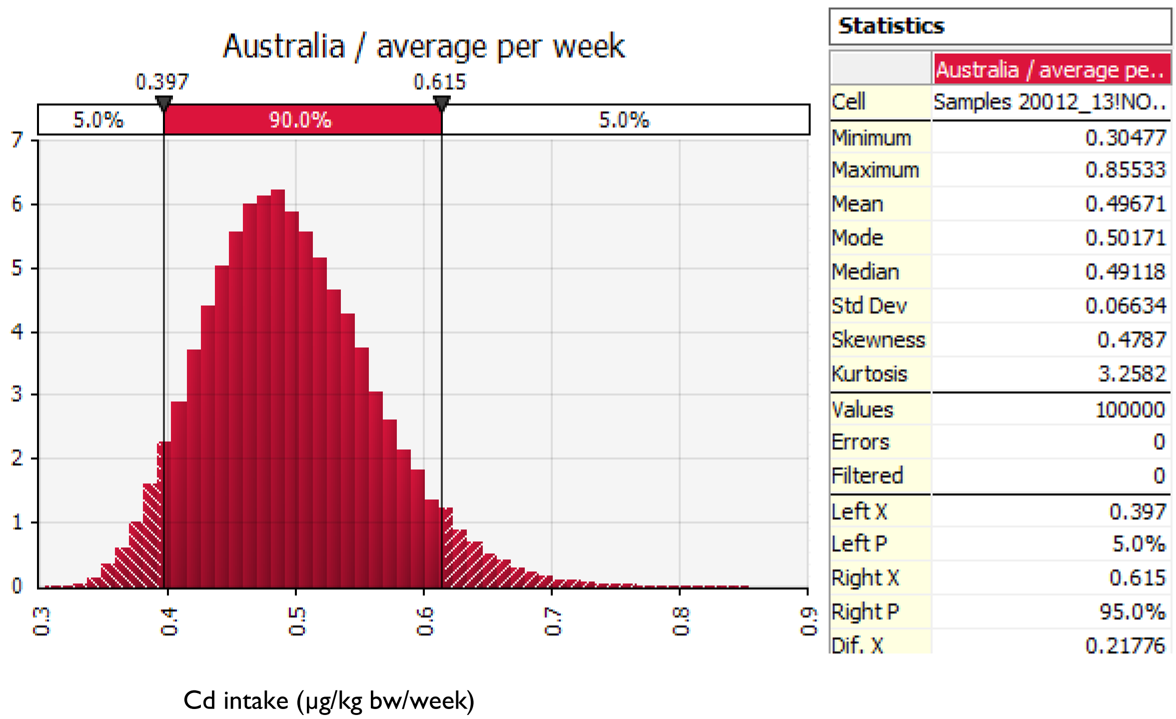


Figure 9. Weekly Cd intake distribution from the consumption of the median prawn amount by adult consumers in Australia.

Table 18. Mean weekly Cd intake (µg/kg bw/wk) from prawns by median consumers of prawns in Australia

Scenario	Mean Weekly Cd Intake From Prawns (µg/kg bw/wk)	% Reduction in Cd Mean Weekly Intake From Prawns
No removal of samples	0.497	-
Remove samples Cd >0.5 mg/kg	0.317	36%
Metapenaeopsis crassissima prawns removed	0.386	22%
Metapenaeopsis crassissima and *M. endeavouri* prawns removed	0.297	40%

RISK CHARACTERISATION

The concentration of Cd in the muscle tissue of Australian wild-caught prawns varies according to their species and geographic capture location.

CADMIUM LEVELS AND SPECIES

P. longistylus, P. monodon, P. indicus, M. ensis, and *Metapenaeus* spp. not identified to the species level are not included in this discussion of species, as they were represented by 3 or less samples in the SARDI dataset. The SARDI 2012-2013 survey found statistically significant differences between several pairs of species and the mean concentration of Cd in muscle tissue decreased in the order *Mp. crassissima* > *M. endeavouri* > *P. plebejus* = *P. esculentus* > *P. semisulcatus* > *P. merguiensis* = *P. latisulcatus* (Table 19). Despite the non-representative nature of the historical dataset, and the lack of scientific species identification, there was a substantial similarity in the mean and median concentrations for species between the two datasets (Table 19). The maximum Cd concentrations for King, Banana, Tiger and Endeavour prawns were higher in the historic dataset than the SARDI 2012-2103 dataset (Table 19). The number of samples for these prawn species in the historic dataset was much larger and there would be a greater probability that it would include more of the rarer prawns that have high Cd concentrations. The mean Cd concentrations in the muscle tissue of the Australian species were well below the 0.5 mg/kg ML, with the exception of *Metapenaeus endeavouri* (Blue Endeavour prawns) which was close to the ML with a mean concentration of 0.41 mg/kg. The coral prawn species *Metapenaeopsis crassissima*, while represented by only two samples, had a mean Cd concentration of 2.65 mg/kg.

Female *Metapenaeopsis crassissima* prawns from WA have exceptionally high concentrations of Cd in their muscle tissue compared with other prawn species analysed for cadmium [43] – see also Table 1 in the accompanying report *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium*. The Cd concentration in the midgut/hepatopancreas tissue of prawns is typically at least 10-fold higher than the concentration in the muscle tissue (Table 1 in accompanying report *2012-13 survey of Australian wild-caught prawns for analysis of cadmium and selenium*), and consistent with this the concentration of Cd in midgut tissue of male *Metapenaeopsis crassissima* is 21-fold higher than in their muscle tissue [44]. However the concentration of Cd in the muscle tissue of female *Metapenaeopsis crassissima* prawns sampled from Western Australia is similar to the high concentrations in their midgut tissue [44]. This phenomenon may be associated with a variant form of metallothionein in the WA female *Metapenaeopsis crassissima* prawns. Other *Metapenaeopsis* species are caught commercially, generally as non-target (i.e. by-catch) species in small quantities from the main Australian prawn fisheries, and are often collectively named “coral” prawns [45, 46]. An investigation of *Mp crassissima* from other locations and of other *Metapenaeopsis* species would indicate whether or not this phenomenon is only associated with a genetically isolated group of prawns in the WA fishery.

Species differences in Cd concentrations in prawns may be due to genetic differences and foraging habits, including the types of animal, plant and detrital material consumed, and variation in foraging area characteristics. There are some examples in the historic dataset of multiple samples collected from one location on a single date, which indicate substantial variation among the individuals in a species from a single location on a single date (Fig 10). This variation may be explained by variations in the Cd levels in

Table 19. Cadmium concentration (mg/kg = ppm) in Australian wild-caught prawn muscle tissue (see notes)

Species		Historic Dataset (1991 - 2010)					SARDI Survey Dataset (2012-2013)				
Common name	Scientific name	n	Mean	Median	Min	Max	n	Mean	Median	Min	Max
Western King	*Penaeus latisulcatus*						43	0.06	0.056	<LOQ	0.28
Eastern King	*P. plebejus*						23	0.12	0.087	0.046	0.24
Red-spot King	*P. longistylus*						1	0.14	0.15	0.03	0.31
King (north of 30 latitude)		601	0.22	0.15	0.01	3					
King (south of 30 latitude)		318	0.02	0.02	0.01	0.14					
King (all)	-	919	0.15	0.08	<0.02	3					
Red Spot		2	0.2	0.2	0.17	0.22					
Brown Tiger	*P. esculentus*						22	0.14	0.125	<LOQ	0.55
Grooved Tiger	*P. semisulcatus*						6	0.12	0.087	0.046	0.24
Black Tiger	*P. monodon*						3	<LOQ	<LOQ	<LOQ	<LOQ
Tiger (all)		524	0.18	0.11	<0.05	4.7					
Banana	*P. merguiensis*						22	0.06	0.03	<LOQ	0.27
Red-legged Banana	*P. indicus*						2	0.26	0.26	0.2	0.32
Banana		407	0.17	0.11	<0.05	2.2					
Blue Endeavour	*Metapenaeus endeavouri*						13	0.405	0.22	0.17	1.1
Red Endeavour	*M. ensis*						1	0.57	0.57	0.57	0.57
	Metapenaeus spp.						2	0.0205	0.0205	0.02	0.021
Endeavour		192	0.49	0.14	<0.05	7.7					
Coral	*Metapenaeopsis crassissima*						2	2.65	2.65	0.7	4.6

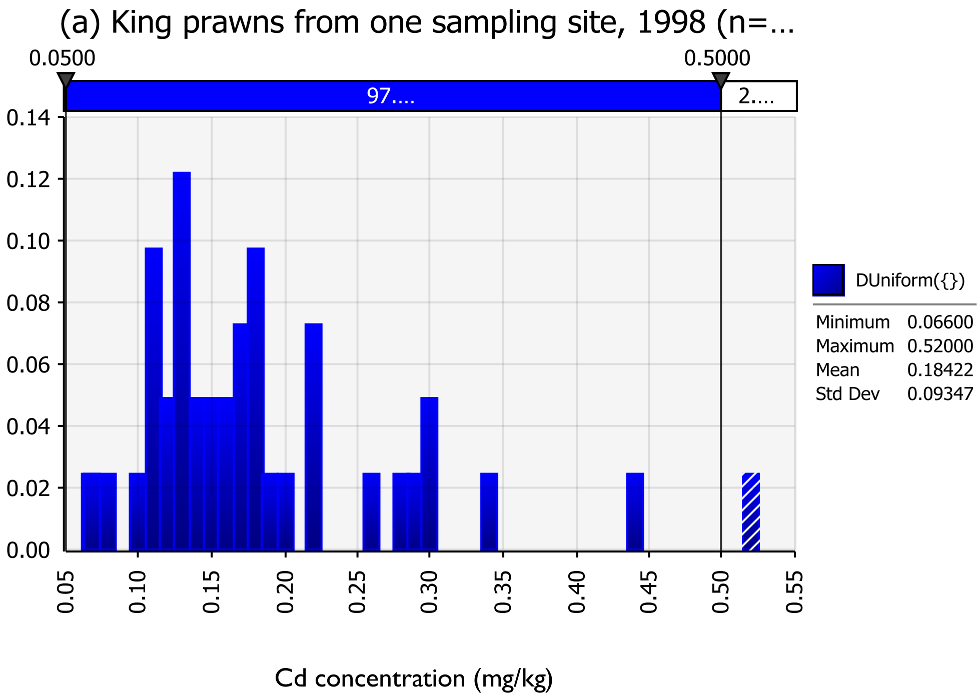
Notes: 1 Historic dataset is an amalgamation of data from various sources and is non-representative in terms of production and catch areas. Also sampling method and analytical techniques and quality assurance are unknown. Scientific species identification/names not available from the historic data set.

2. SARDI 2012-2013 Dataset is from a survey designed to be representative of production and catch areas, with defined sampling and analytical protocols and quality assurance.

3. LOQ = Level of quantitation samples reported at <LOQ allocated a Cd concentration of 0.5*LOQ (=0.01 ppm)

4. In historic dataset samples reported as <y allocated a Cd concentration of 0.5*y

dietary sources over the lifetime of the prawns at the time of capture, and overlays the between-species variation.



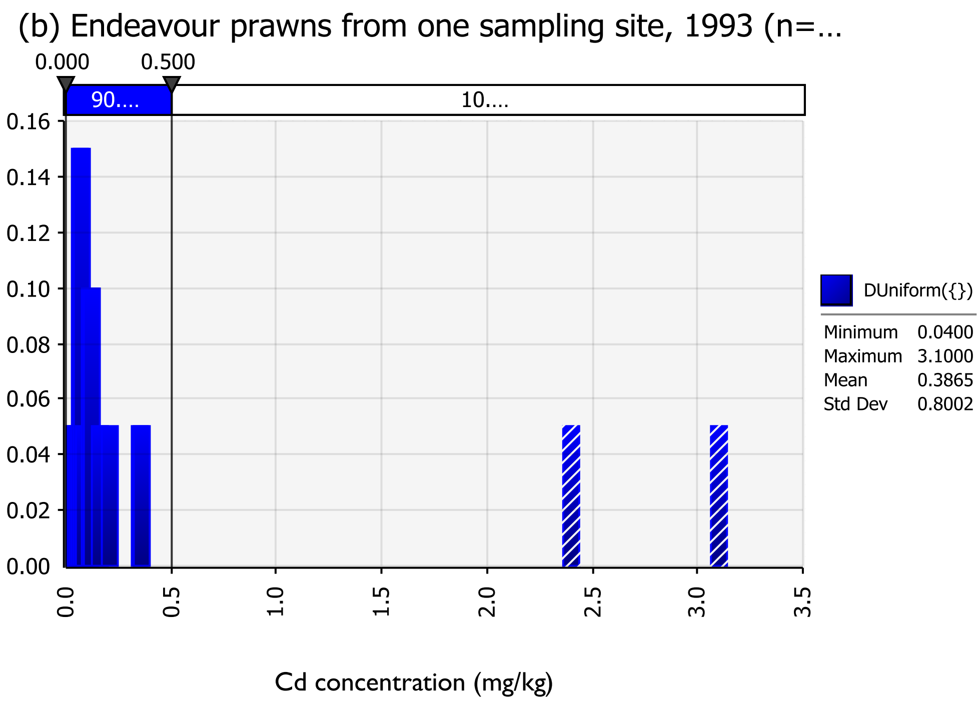


Figure 10. Cd concentrations in multiple samples from a single location on a single day (a) King prawns from a single location on a single date (b) Endeavour prawns sampled from a single location on a single date. Data from the historic dataset, company sources de-identified

CADMIUM AND FISHERY LOCATION

Mapping of the historic dataset suggested that prawns south of 30° latitude have low Cd concentrations (Fig 2). But as the data from samples collected south of 30° latitude were dominated by a single species (*P. latisulcatus* from South Australia), species as well as location may be factors in this pattern.

Analysis of the SARDI 2012-13 survey did show the following statistically significant differences: (i) Cd concentrations in *P. latisulcatus* were lower in South Australia than Western Australia, (ii) Cd concentrations in *P. merguiensis* were higher in the Northern Prawn Fishery than in the Eastern Queensland fishery, and (iii) Cd concentrations in *P. esculentus* were higher in the Northern Prawn Fishery than in the Eastern Queensland fishery.

In fisheries where Cd concentrations are higher, and where they cannot be explained by genetic differences between prawn populations of the same species, the differences may be explained by the variation in Cd concentrations in dietary sources. The causes of this variation in Australian wild-caught prawns have not been determined.

WEEKLY INTAKE OF CADMIUM FROM AUSTRALIAN WILD-CAUGHT PRAWNS

France, the United Kingdom and Spain represented a range of prawn consumption levels across Europe and also encompass prawn consumption levels in Asian countries where data on consumption amounts by prawn consumers were not available. For median consumers of prawns in these European countries, the intake of Cd from prawns represented only a minor proportion of the Tolerable Weekly Intakes established by JECFA (1-2%) and EFSA (2-5%), and the reduction in Cd intake by the removal of samples >0.5mg/kg or species with the highest Cd concentrations was minimal (Table 21).

For those who were high consumers of prawns (95^{th} percentile) in the European countries the intake of Cd from prawns was 3-9% of the JECFA TWI and 6-22% of the EFSA TWI (Table 22). As not all of the population are consumers, the proportion of the total population consuming these amounts of prawns or higher are 1.4%, 1.2% and 1.7% of the adult populations of France, the UK and Spain respectively. *Mp. crassissima* was not included in these analyses as this species is not exported to Europe. The removal of samples with Cd > 0.5 mg/kg or the removal of *Metapenaeus endeavouri* reduced the Cd intake levels for these high consumers by similar amounts; however the impact of these interventions was modest, with the Cd intake level reduced to 2-8% of the JECFA TWI or 5-18% of the EFSA TWI (Table 22).

The Australian NNS only identified 2.7% of the adult population as consumers and 1.4% consume the median amount of 30.6 g or more per day. The mean Cd intake for a 70 kg person consuming 30.6 g prawns per day is 8.6% of the JECFA TWI or 20% of the EFSA TWI. Removal of samples > 0.5 mg/kg or removal of the two species with the highest Cd concentrations (*Metapenaeopsis crassissima* and *Metapenaeus endeavouri*) reduced the Cd intake levels by similar amounts to 5.2-5.5% of the JECFA TWI or 12-15.6% of the EFSA TWI (Table 23).

EFFICACY OF A 0.5 MG/KG MAXIMUM LIMIT (ML) FOR RISK MINIMISATION

This assessment indicates that for median consumers of prawns in Europe, Cd exposure from prawns is small when considered in relation to either the JECFA TWI or the EFSA TWI. Consequently the application of the 0.5 mg/kg ML has minimal impact on Cd exposure for these consumers.

For high consumers of prawns in Spain – the European country with the highest prawn consumption – application of the 0.5 mg/kg ML did not have a large impact on reducing Cd exposure. Percent JECFA TWI exposure was reduced from 9% to 8%, and % EFSA TWI exposure was reduced from 22% to 18%.

Australian "median" consumers had similar exposure levels to "high" consumers in Spain. The two factors contributing to the higher exposure amounts in this group of Australians were the large consumption amounts and the inclusion of the coral prawn species *Metapenaeopsis crassissima* in the Australian diet.

In summary this assessment found the >0.5 mg/kg regulation had limited effect in reducing exposure to Cd from prawns. It is also noted that in removing all samples that had Cd > 0.5 mg/kg in the simulation model, the impact of the 0.5 mg/kg ML was overestimated, as not all consignments containing some prawns with Cd >0.5 mg/kg would be detected and removed.

Table 21. Cd intake by a median consumer as a percentage (%) JECFA and EFSA Tolerable Weekly Intakes (TWI)

				Samples > 0.05 Removed			*M. endeavouri* samples removed*		
	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI
France	0.041	1%	2%	0.033	1%	1%	0.032	1%	1%
UK	0.070	1%	3%	0.058	1%	2%	0.055	1%	2%
Spain	0.129	2%	5%	0.106	2%	4%	0.100	2%	4%

Table 22. Cd intake by a high consumer as a (%) JECFA and EFSA Tolerable Weekly Intakes (TWI)

				Samples > 0.05 Removed			*M. endeavouri* samples removed*		
	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI	Mean weekly Cd intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI
France	0.150	3%	6%	0.123	2%	5%	0.116	2%	5%
UK	0.257	4%	10%	0.211	4%	8%	0.199	3%	8%
Spain	0.540	9%	22%	0.443	8%	18%	0.419	7%	17%

Table 23. Cd intake by Australian median consumers as (%) JECFA and EFSA Tolerable Weekly Intakes (TWI)

Scenario	Mean Weekly Cd Intake (μg/kg bw/wk)	% JECFA TWI	% EFSA TWI
No removal of samples	0.53	9.1%	21%
Remove samples Cd > 0.5 mg/kg	0.37	6.4%	14.8%
Metapenaeopsis crassissima removed	0.43	7.4%	17%
Metapenaeopsis crassissima and *M. endeavouri* removed	0.35	6.0%	14%

This assessment found that the distribution of Cd in prawns is far from uniform. Even prawns sampled from one location on a single day have a skewed distribution with a small percentage of prawns >0.5 mg/kg and the large majority <0.5 mg/kg (Fig 10). The effect of this when samples are taken from a consignment for testing is that prawns >0.5 mg/kg will be missed by the sampling, and when a sample >0.5 mg/kg is detected it is unlikely to be representative of the mean concentration of Cd in prawns in the consignment, and therefore consignments may be rejected inappropriately.

COMPARISON OF CADMIUM DISTRIBUTION IN PRAWNS CAUGHT IN AUSTRALIAN WATERS AND THOSE FROM OTHER AREAS

The exposure assessment was undertaken as if all the prawns eaten were Australian wild-caught prawns. In reality in the countries to which Australian prawns are exported, Australian prawns are a minor fraction of the prawns consumed. Japan is the largest export market for Australian prawns. In 2009-10 Australia exported 1,839 tonnes (T) of prawns to Japan which represented only 0.6% of the 285,764 T of apparent prawn consumption in Japan in 2009 (Tables 4 and 8). In 2009-10 Australia exported 142 tonnes (T) of prawns to Spain which represented only 0.1% of the 133,630 T of apparent consumption of prawns in Spain in 2009 (Tables 4 and 8).

Any consideration of the efficacy of the 0.5 mg/kg maximum limit (ML) regulation in Europe and other export destinations would probably be considered in the context of cadmium levels in prawns from many sources. A comparison of the characteristics of the SARDI 2012-1013 survey, and a 2003-2008 European dataset utilised in the EFSA 2009 scientific opinion of cadmium in foods [39] indicates that they are broadly similar (Table 24). The mean of both datasets is <<0.5 mg/kg, and the medians are less than the means indicating a skewed distribution of Cd concentrations among samples in both datasets. The proportion of samples >0.5 mg/kg is also similar in both datasets. This suggests that the finding of this risk assessment that the 0.5 mg/kg ML is ineffective in minimising Cd exposure from Australian wild-caught prawns is also likely to apply to prawns in European diets sourced from other areas.

Table 24. Comparison of Cadmium in European Crustaceans and Australian prawns

	European Data 2003 – 2008* Crustaceans	Australian Prawns SARDI Survey 2012-2013
Number of samples (N)	1896	140
P5 (mg/kg)	0.0005	0.01
Median (mg/kg)	0.013	0.083
Mean (mg/kg)	0.093	0.162
P95 (mg/kg)	0.5	0.33
Max (mg/kg)	2.3	4.6
% Samples > 0.5 (mg/kg)	4.90%	4.30%

Source: European data from EFSA (2009) [39]

CONSIDERATION OF THE REMOVAL OF SPECIES WITH HIGH CADMIUM CONCENTRATIONS

There were two species found to have relatively high Cd levels among Australian wild-caught prawn species, the coral prawn *Metapenaeopsis crassissima* and the blue endeavor prawn *Metapenaeus endeavouri*. These findings were consistent with more extensive datasets from industry sources and the National Residue Survey, as well as literature reports [43, 44]. Removal of these species had the same impact on reducing Cd exposure from prawns as the removal of samples >0.5 mg/kg. Removal of these species would be a more reliable strategy to reducing Cd exposure from prawns than application of a 0.5 mg/kg ML.

Metapenaeopsis crassissima and *Metapenaeus endeavouri* contribute only a small proportion to the total Australian wild-catch production, and consequently the results of the simulation modelling indicate that the benefits from removal of these species from the human diet would be minimal for median consumers of prawns, and would result in only modest reductions of cadmium intake by high consumers of prawns. The extent of the benefit of a strategy of high-Cd species removal has not been determined with sufficient accuracy by this assessment, and would need to be considered in the context of costs to the prawn industry by risk managers.

This risk assessment cannot provide an accurate estimate of the extent to which Cd exposure from prawns by high consumers would be reduced by removal of *Metapenaeopsis crassissima* and *Metapenaeus endeavouri* due to (i) a lack of accurate information on prawn consumption patterns by those identified as high consumers of prawns in dietary surveys, and (ii) a lack of detailed quantitative information on the distribution of the higher Cd species in the Australian domestic market and in export markets.

Dietary consumption data is the most significant source of uncertainty in estimating the extent that Cd exposure among high consumers would be reduced by removal of the two species with high Cd concentrations. Dietary survey methodologies differ in how they obtain information e.g. day-to-day diaries, recall over differing periods, interviews and questionnaires. Due to practical limitations, the most detailed surveys are typically conducted over short periods e.g. 1-2 weeks, with seasonal variations accounted for by techniques such as conducting the survey on a rolling basis throughout a year. For a non-staple food such as prawns, it is difficult to obtain accurate information on the amounts consumed and regularity of consumption by those within the high consumer group. Consequently extrapolation from dietary survey data conducted over a short period to consumption over an annual period has limitations. This is particularly evident in the 1995 Australian NNS which only identified 2.7% of the Australian population as consumers of prawns. It is possible that consuming prawns each week may be a consumption pattern in a small proportion of Australian consumers, but accurately estimating this proportion and the amounts and types of prawns consumed in this manner would require more extensive and detailed prawn consumption data than is currently available.

Another source of uncertainty in estimating the extent that Cd exposure would be reduced by removal of the two species with high Cd concentrations, is a lack of information on the distribution of these species into both domestic and export markets. *Metapenaeopsis crassissima* and *Metapenaeus endeavouri* contribute the smallest production volumes to the total of Australia's wild-catch production species, and in markets where Australian prawns are a minor proportion of prawn consumption, their significance as a source of Cd may diminish quite significantly. Domestically, the simulation model is a simplified representation of reality in that it assumes that prawns are distributed to all consumers in proportion to production volumes. If there are locations where consumption of *Metapenaeopsis crassissima* and *Metapenaeus endeavouri* is substantially higher than the average, due to availability and price, exposure to Cd through prawns for some Australians will be higher than estimated in this assessment.

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