食物環境衞生署
Food and Environmental Hygiene Department

Risk Assessment Studies
Report No. 10B

Chemical Hazard Evaluation

DIETARY EXPOSURE TO HEAVY METALS OF SECONDARY SCHOOL STUDENTS

September 2002
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Chapter 1

Introduction

Background

1.1 Many different varieties of metals and metal compounds exist naturally around the world. Human exposure to metals occurred since pre-historic time in areas where the heavy metal content of water and food are naturally high. Other than exist as metal ores in the crust of the Earth, metals can also be transported to different parts of the world by various natural cycles. For example, metals, which exist in soil or in ores, can be dissolved by rain. The dissolved metals can then enter river and ground water systems and later the oceans and deposit as sediments. Metals may also be carried up into the atmosphere along with water vapour and subsequently deposit elsewhere.[1] The advancement of technology and industrialization had also contributed to the wider distribution of metals and the increased variety of metal compounds[1].

1.2 Three heavy metals, namely arsenic, cadmium and mercury, are chosen for dietary exposure assessment in this report in view of their toxicity and carcinogenicity. Cadmium and inorganic arsenic are known carcinogens and their intake are mainly concerned with food. Mercury is toxic, in particular its organic form, methyl mercury, to which pregnant women, young children and fetuses are particularly vulnerable to its adverse effects on the nervous system.

Objectives

1.3 The objectives of this study are -

- to assess the levels of heavy metals exposure of secondary school students through food consumption;
- to identify the dietary sources of the heavy metals; and
- assess the public health impact of the exposures.

Scope and Methods

1.4 This study utilizes two sets of data in estimating the dietary exposure of secondary school students to heavy metals. The first set of data on concentration of heavy metals in food was obtained from the food surveillance programme of the Department. The second set of data on food consumption was derived from the Food Consumption Survey conducted by this Department in late 2000. Secondary school students were chosen as the population of this study because they have relatively high-energy intake than adult and may be considered as a particularly at risk group. Moreover, they may be subject to chronic exposure to heavy metals. In addition, they are relatively more cooperative and comprehensive data can be obtained more easily. These make the collection of food consumption data manageable and feasible.

References

[1] Moffat CF, Whittle KJ editors. Environmental Contaminants in Food. Sheffield: Academic Press: 1999.

Chapter 2

Arsenic

2.1 Arsenic (As) is widely distributed in nature. It occurs as inorganic and organic compounds as well as trivalent As (III) and pentavalent As (V) states. Arsenic in nature is often associated with igneous and sedimentary rocks in form of inorganic arsenic compounds. Arsenic occurs naturally in soil and minerals and may enter the air, water, and land from wind-blown dust and may also get into water from runoff and leaching. Volcanic eruptions are another source of arsenic.

2.2 Arsenic compounds are used industrially in manufacturing of transistors, lasers, semiconductors, glass, pigments and others. To a lesser extent, they are used in agricultural chemicals such as insecticides, herbicides, fungicides and pesticides. Majority (97%) of the arsenic produced by human activities worldwide are in form of inorganic arsenic, arsenic trioxide, while the rest is used as additives in the metallurgy for

producing special lead and copper alloys[1].

2.3 Arsenic has been considered as an essential trace element for the normal growth and development in experimental animals[2]. However, arsenic is more often regarded as a contaminant rather than as an essential mineral[3].

2.4 In the general population, the primary route of exposure to arsenic compounds is through ingestion. Food is considered the main contributor to total arsenic intake while in places where drinking water contains relatively high levels of arsenic, drinking water can be a significant source of arsenic intake[4]. Other routes of exposure such as through inhalation of air and via dermal absorption only play a minor or negligible role.

Dietary Exposure

2.5 Most arsenic compounds can dissolve in water and this results in the presence of this heavy metal in aquatic food, especially in shellfish. The highest concentrations of arsenic in food are found in aquatic foods[5]. Arsenic concentrations in fish usually range from 1 to 10 mg/kg, but this

value may be as high as 100 mg/kg in bottom feeders and shellfish[6]. Arsenic-containing pesticides, herbicides and other agricultural products can lead to the accumulation of arsenic compounds in soils and plants, resulting in trace amount of arsenic found in foodstuff. In general, inorganic arsenic is the more toxic form. Arsenic in fish is usually present in its less toxic organic form, of which arsenobetaine is the most predominant form. Nonetheless, organic arsenic could be metabolised to inorganic arsenic by aquatic animals and may present potential toxicity problems[7].

Metabolism

2.6 The absorption of arsenic in the body is dependent upon the type of compound present, its solubility and its physical form. In general, inorganic forms are more readily absorbed than organic forms and pentavalent arsenic As (V) is more readily absorbed than trivalent As (III). Organic arsenic compounds are excreted more rapidly than inorganic arsenic compounds and As (V) compounds are excreted more rapidly than As (III) compounds. Excretion is primarily in urine and to a lesser extent in faeces.

Toxicological Effects of Arsenic Compounds

Acute Toxicity

2.7 Symptoms of acute toxicity include severe inflammation of gastrointestinal tract, leading to severe vomiting and diarrhoea, often with blood-tinged stools. This can be accompanied by secondary electrolyte disturbances with clinical features of muscular cramps, facial oedema and cardiac dysfunction. Sensory loss is one of the neurological presentations of arsenic intoxication. It has been reported that the fatal dose of ingested arsenic trioxide ranges from 70 to 180 mg[8].

Subacute Toxicity

2.8 Subacute toxicity occurs in paralytic form, which mainly affects the respiratory, gastrointestinal, cardiovascular, nervous and haematopoietic systems. Clinical presentations include facial oedema, anorexia, and upper respiratory symptoms followed by skin lesions and neurological signs.

Chronic Toxicity

2.9 Chronic exposure to inorganic arsenic compounds is associated with skin lesions, hyperkeratosis and chronic pathological liver changes. A high prevalence of a peripheral vascular disease called “blackfoot disease” was found in a population living in Taiwan, where the speculated causative factor was related to the arsenic exposure via drinking well water.

Carcinogenicity

2.10 Carcinogenicity in the skin, lung, bladder, kidney, liver, and lymphatic and haematopoietic systems of humans is strongly supported by epidemiological studies.[9,10,11,12] These increased cancer risks are especially prevalent among smelter workers and in those engaged in the production and use of arsenical pesticides rather than through the dietary route of exposure.

2.11 In 1980[13], the International Agency for Research on Cancer (IARC) of WHO concluded that there was sufficient evidence that inorganic arsenic compounds were skin and lung carcinogens in humans, but that the data for other sites were inadequate for evaluation.

2.12 In contrast, animal carcinogenicity tests with inorganic and

organic arsenicals have been negative[5, 14,15]. The majority of animal studies did not demonstrate the carcinogenicity of arsenic compounds even when the chemicals were administered near the tolerated dosages. A few observations of increased incidence of leukaemia and lung cancers suggested that inorganic arsenicals might be considered as cancer promoter instead of initiators.

Reference

[1] Eisler R editor. Handbook of Chemical Risk Assessment Health Hazards to Humans Plants and Animals Vol 3 Metalloids, radiation, cumulative index to chemicals and species. US: CRC Press;2000

[2] US Environmental Protection Agency Risk assessment forum special report on ingested inorganic arsenic. Skin cancer, nutritional essentiality. Washington DC, USA; 1988(EPA-625/3-87/013)

[3] National Academy of Sciences (NAS) Arsenic Natl Acad.Sci, Washington , DC

[4] Agency for Toxic Substances and Disease Registry (ASTDR). Toxicological Profile for Arsenic. Chapter 5- Potential for human exposure. US Department of Health and Human Services. Washington DC,US; September 2000. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp2.html

[5] World Health Organization (WHO). Joint FAO/WHO Expert Committee on Food Additives (JECFA) WHO Food Additives Series -570 Arsenic, No.18 Available from: http://www.inchem.org/documents/jecfa/jecmono/v18je17.htm

[6] World Health Organization (WHO). Joint FAO/WHO Expert Committee on Food Additives (JECFA) WHO Food Additives Series -658 Arsenic, No. 24. Available from: http://www.inchem.org/documents/jecfa/jecomon/vo24je08.htm

[7] Agency for Toxic substances and Disease Registry (ATSDR). Toxicological Profile for Arsenic, prepared by life systems, Inc for ATSDR US Public Health Service in collaboration with US Environmental Protection Agency. ATSDR/TP-88/02. Washington DC, USA; 1989b.

[8] Vallee BL, Ulmer DD, Wacher WE. Arsenic toxicology and biochemistry. Arch Ind Health 21:132-151

[9] Hsueh YM, Cheng GS Wu MM et al. Multiple risk factors associated with arsenic-induced skin cancer. Effects of chronic liver disease and malnutritional status BrJ Cancer. 1995. 77(1):109-14

[10] Hanpert TA, Wiersma JH, Goldring JM. Health effects of ingesting arsenic-contaminated groundwater. Wis Med J. 1996;95(2):100-104

[11] Alain G, Tousignant J, Rozen farb E. Chronic arsenic toxicity. Int J Dermatol. 1993; 32(12): 899-901

[12] Bickley LK, Papa CM. Chronic arsenicism with vitiligo, hyperthyroidism and cancer. NJMed 1989; 86(5):377-380

[13] International Agency on Research of Cancer (IARC). Arsenic and arsenic compounds (IARC) Summary of data reported and evaluation. 1980;Vol 23 Available from: http://www.inchem.org/documents/iarc/iarc 369.htm

[14] Kroes R, Vanlogten . Study on the carcinogencity of lead arsenate and sodium arsenate and on possible synergistic effects diethylnitrosamine. Food Cosmet Toxicol 1974;12(5-6):671-679

[15] Byron WR, Bierbower GW, Brouwer JB etal. Pathologic changes in rats and dogs from two-year feeding of sodium arsenite or sodium arsensate. Toxicol Appl Pharmacol. 1967; 10:132-147

Chapter 3

Cadmium

3.1 Cadmium (Cd) is a metallic element that occurs naturally in the Earth's crust. Cadmium is usually not present in the environment as a pure metal, but as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulphur (cadmium sulphate, cadmium sulphide). These different forms of cadmium compounds are solids that dissolve in water to varying degrees and are present in zinc, lead, and copper ores.

3.2 Cadmium is a by-product of zinc and lead mining and smelting, which are important sources of environmental pollution. Since the early twentieth century, cadmium has been used in a variety of applications in electroplating, pigment production, and the manufacture of plastic stabilizers and nickel-cadmium batteries.

3.3 The largest airborne sources of cadmium in the environment are from the burning of fossil fuels such as coal, oil and the incineration of municipal waste. Cadmium has also been added to the environment through industrial processes such as production of cadmium metal. Levels of cadmium are generally higher in the vicinity of metallurgical plants. For the general population, cadmium intakes from air are unlikely to exceed 0.8 μg/day[1]. Besides, cigarette smoking is another important source of cadmium particularly for exposures inside houses.

3.4 Cadmium particles in air can travel long distances before coming down to the ground as dust, or along with rain or snow. Cadmium does not break down in the environment, but it can change into different forms. Although very mobile in water, some forms of cadmium will bind to soil. Fish, plants, and animals can take up cadmium from water and the environment.

3.5 Fertilizers, often contain some cadmium, may enter the soil when they are applied to crops. Certain staple foods, such as rice and wheat, may accumulate cadmium naturally by absorption from the soil.

Cadmium can also enter the soil from spills or leaks at hazardous wastes sites if large amounts of dissolved cadmium are present at the sites.

Dietary Exposure

3.6 Food is recognized as the main source of cadmium intake for non-occupationally exposed people. Crops grown in polluted soil or irrigated with polluted water may contain increased concentrations of cadmium, as may meat from animals grazing on contaminated pastures.

3.7 According to Global Environment Monitoring System - Food Contamination Monitoring and Assessment Programme (GEMS/Food)[2] of WHO, the lowest cadmium levels are found in milk, eggs, fruit and meat muscles; medium levels are found in cereal and potatoes; highest concentrations are present in mollusks, crustacean and in kidneys, in which contamination is found to increase with the age of the animal. The Joint FAO/WHO Expert Committee on Food Additives (JECFA)[3] noted that high cadmium level found in animal kidneys might be due to metal accumulation in these organs. In the same evaluation, it was also pointed

out that fish contain normally only small amount of cadmium, whereas shellfish such as crustaceans and mollusks, may absorb larger amounts of cadmium from their environment.

3.8 Cigarette smokers are exposed to extra amount of cadmium. Smokers may double their daily intake of cadmium compared with non-smokers. Each cigarette may contain 1 to 2 μg of cadmium, and about 40 - 60% of the cadmium in the inhaled smoke can pass through the lungs into the body[1].

Metabolism

3.9 In healthy subjects, 4 - 8 % of the cadmium ingested are absorbed; in calcium and iron deficient people such as pregnant woman, the figure can reach 15 - 20 %[4].

3.10 Most of the cadmium that enters the body is concentrated in the kidneys and liver with biological half-life of 10-35 years[1]. Cadmium is bound to a low molecular weight protein, matallothionein which mitigates

the toxicity of the unbound ion. This cadmium-matallothionein complex is filtered at the glomerulus of the kidney but is reabsorbed by the proximal renal tubules[3].

3.11 Only a small portion of the cadmium that enters the body is excreted slowly in urine and feces. However, the lack of an effective elimination pathway leads to the accumulation of cadmium in the body and long biological half-life. Although the body can change most cadmium to a form that is less harmful, too much cadmium can overload this ability of the kidneys and liver, and thus damage health.

Toxicological Effects of Cadmium

Acute Toxicity

3.12 Human exposure to cadmium through inhalation, which is usually occupational in nature, may result in effects on the lung, including chemical pneumonitis and sometimes pulmonary oedema. In severe cases, there may be respiratory insufficiency, shock and death. A single acute

exposure to high levels of cadmium can result in long-lasting impairment of lung function.

Chronic Toxicity

3.13 With chronic oral exposure, the kidney, particularly the cortex, appears to be the most sensitive organ. Cadmium affects the resorption function of the proximal tubules, the first adverse effect being an increase in the urinary excretion of low-molecular-weight proteins, known as tubular proteinuria.

3.14 Other possible effects include aminoaciduria, glucosuria, and phosphaturia. Disturbances in renal handling of phosphorus and calcium may cause resorption of minerals from bone, which can result in the development of kidney stones and osteomalacia (fragile bones).

Carcinogenicity

3.15 According to JECFA's evaluation[3], some epidemiological

studies indicated an increased risk of cancer of the prostate in workers exposed to cadmium and an increased incidence of lung cancer was also indicated. IARC (1993)[5] classified cadmium and its compounds as Group 1 carcinogen, which means that the agents are carcinogenic to human.

References

[1] World Health Organization (WHO). Cadmium – Guidelines for Drinking Water Quality. 2nd ed. Vol.2 Health criteria and other supporting information. Geneva, WHO, 1996. Available from: http://www.who.int/water_sanitation_health/GDWQ/cadmiumfull.htm

[2] Golin F. Moffat and Kevin J. Whittle editors. Environmental Contaminants in Food. Sheffield: Sheffield Academic Press.

[3] Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Food Additives Series, No.4, 1972.

[4] John deVries Food Safety and Toxicity. CRC Press 1997. Netherlands

[5] International Agency on Research of Cancer (IARC). IARC Cancer Databases. Available from: http://www.iarc.fr

Chapter 4

Mercury

4.1 Mercury (Hg) exists naturally in abundance in the environment. It enters the environment by both natural and human means[1,2]. Volcanic and geothermal activities are the major known sources of natural mercury emission that enter the biosphere, where they may exist in form of vapour, solution and particles.

4.2 Mercury exists in three forms, namely metallic, inorganic and organic mercury. The forms can be altered under certain conditions[1]. Mercury metals can be oxidized to inorganic bivalent mercury with the presence of organic matters in water, or it can either be converted back to metallic mercury in a reducing environment in certain industrial effluent, or alkylated by a number of bacteria to dimethylmercury.

4.3 Industrial activities including mining and refining of cinnabar

and gold, manufacture of chlorakali chemicals (chlorine and sodium hydroxide), manufacture and use of mercury-containing lighting and temperature-monitoring devices, combustion of fossil fuels and electroplating are among the common industrial activities that add to the emission of mercury[2].

4.4 Other sources, including the use of mercuric compounds in fungicides and seed treatments, disposal of mercury-containing batteries and incinerator ashes in landfills, spreading of municipal sludge onto farmland etc. contribute to the increased level of mercury in soil[3].

4.5 A complex system of mercury cycling operates on a global scale. Metallic mercury on the surface, including those in soils and water can evaporate and enter the atmosphere. Mercury that entered the atmosphere is carried along with atmospheric activities and later deposited onto land with rain. It is then absorbed by soil or sediments. Mercury that was deposited with sediments could re-emerge during dredging or being carried by ocean currents.

4.6 Mercury and its compounds have no known physiological functions in animals. Their presence in human is undesirable and may be hazardous to health[2].

Dietary Exposure

4.7 Organic mercury compounds are more of a concern than inorganic mercury. Methylmercury is the most common form of organic mercury and is regarded as highly toxic. Contamination of food from both natural and human sources, dental amalgam and occupational exposure in agriculture and manufacturing sectors are possible routes of exposure to the chemicals. Traditional Chinese Medicines (TCM) and cosmetics would also be the possible sources of exposure.

4.8 Dietary intake is by far the most dominant source of exposure to mercury. Fish and other seafood products are the main source of methylmercury, of which large predatory species such as tuna and swordfish tend to accumulate relatively higher levels. Methylmercury bio-accumulates as it moves up the food chain, increasing in concentration

at the same time.

Metabolism

4.9 Elemental mercury can only be marginally absorbed (<0.01%) through the gastro-intestinal tract when ingested[4]. For inorganic mercury, about 7 - 15% oral intake of mercuric chloride is absorbed through the gastrointestinal tract, where the percentage of absorption is proportional to the solubility of the mercuric salt. Inorganic mercury (II) compounds are most likely to be accumulated in kidneys. They also do not cross the placenta and blood-brain barrier easily because of their ionic charge.

4.10 Clinical studies showed that inorganic mercury tends to accumulate in kidneys[5]. Effects of exposure are on the nervous system and the kidneys when the dose is low. Common responses to human exposure include immunological glomerular disease and proteinuria. Proteinuria is reversible when the intake of mercury and inorganic mercury ceased, whereas people exposed to mercuric chloride may have irreversible damages to the lining of the renal tubes. Inorganic mercury is mainly

eliminated in faeces and urine. About 75 - 92% of the metal is excreted 4 - 5 days after ingestion.

Toxicological Effect

Acute Toxicity

4.11 Acute toxicity is often a result of occupational exposure, and that from dietary exposure is rare. Acute effects include increased occurrence of lymphocytic aneuploidy, discolouration of the front surface of lens of the eyes, insomnia, tremors and hyperexcitablity. Effects of inorganic mercury are cumulative.

Chronic Toxicity

4.12 There is a long latent period before early symptoms of methylmercury poisoning including paraesthesia, malaise and blurred vision emerge. Constriction of visual field, deafness, dysarthria and ataxia may develop at a later stage. The patient may partly recover from

the symptoms in a less severe case or may fall into a coma and die as a result in a severe case. Damages to the central nervous system are highly localized and affects mostly sensation, vision and hearing. For ingestion of inorganic mercury (II) compounds, the kidneys are the critical organs[6].

4.13 The metallic, inorganic and organic forms of mercury are neurotoxicants. Foetuses exposed to organic mercury have been found to be born mentally retarded and with symptoms similar to those of cerebral palsy. Pregnant mothers who received low level of methylmercury by normal adult standard may give birth to children with serious cerebral palsy.

Carcinogencity

4.14 Mercury compounds are not classified as human carcinogens by IARC[7].

References

[1] Eisler, R. Handbook of Chemical Risk Assessment – Health Hazards to Humans, Plants and Animals. Vol. 1 – Metals. 2000. Lewis Publishers, Boca Raton, Florida, USA.

[2] Committee on the Toxicological Effects of Methylmercury, National Research Council. Toxicological Effects of Methylmercury. 2000. National Academy Press, Washington, DC, USA.

[3] Office of Water, US Environmental Protection Agency. Mercury Update: Impact on Fish Advisories – US EPA Fact Sheet. June 2001. US EPA, Washington, DC, USA.

[4] National Research Council. Toxicological Effects of Methylmercury. 2000. National Academy Press. Washington DC, USA.

[5] International Programme on Chemical Safety (IPCS). Inorganic Mercury. Environmental Health Criteria 118. World Health Organization. Geneva, Switzerland 1991.

[6] World Health Organization (WHO). Inorganic Mercury. Environmental Health Criteria 118. WHO. Geneva, Switzerland. 1991.

[7] International Agency on Research of Cancer (IARC). IARC Cancer Databases. Lyon, France. Avaliable from <http://www.iarc.fr>

Chapter 5

Data Compilation

5.1 This study used two sets of data available to the Department for estimating the levels of heavy metals that the population may be exposed from dietary sources. The first set of data was obtained from the food surveillance programme of this Department from 1999 to 2001. Results of food samples tested for heavy metals were extracted from the database to establish the levels of heavy metals in the local food supply. The second set of data was derived from the Food Consumption Survey, which was conducted by the Department in 2000 on secondary school students. The two sets of data are described in greater detail below.

Heavy Metal Contamination in Local Foods

Sample Collection

5.2 For surveillance and enforcement purposes, a food surveillance programme is in place. Food samples were collected at every stage of the supply process from the local market for chemical, microbiological and radiological testing. Data obtained from 1999 to 2001 which were relevant to the present study were extracted from the food surveillance database. These food samples can be categorized under six groups, namely (i) cereals and cereal products, (ii) vegetables, (iii) meat, poultry and their products, (iv) fish, (v) seafood other than fish, and (vi) milk and dairy products.

Analysis of Heavy Metals

5.3 The Government Laboratory (GL) undertook the metal analysis. For the analysis of arsenic and cadmium, an accurately weighed portion of the homogenized sample was digested using concentrated nitric acid and sulphuric acid in Kjeldahl digestion apparatus. The concentrations of the two heavy metals were determined by analysing the final digest with hydride-generation atomic absorption spectroscopy and graphite furnace atomic absorption spectroscopy respectively.

5.4 As for mercury, an accurately weighted portion of the homogenised sample was digested with a mixture of nitric acid, hydrochloric acid and sulphuric acid. The concentration of mercury in the sample was determined by analysing the final digest with cold vapour atomic absorption spectroscopy.

5.5 The analytical procedures for inorganic arsenic and methylmercury are technically difficult and expensive to conduct. Thus, total arsenic and mercury were analyzed while inorganic arsenic assay was performed on selected samples with high total arsenic level only. The limits of detection (LOD) for the metals in the food samples were as follows:

Table 5.1: The limits of detection (LOD) of Arsenic, Cadmium and Mercury

Heavy metal	Limit of detection (LOD)
Arsenic	0.076 mg/kg (ppm)
Cadmium	0.02 mg/kg (ppm)
Mercury	0.03 mg/kg (ppm)

Data Analysis

5.6 In the analytical reports issued by GL, the total arsenic content determined by the analysis were expressed as arsenic trioxide (As_2O_3) for enforcement purpose, since the maximum permitted level for arsenic was stipulated as arsenic trioxide in the law. In this study, the arsenic trioxide content was converted back to total arsenic level. There is no international consensus on how the level of inorganic arsenic can be estimated in various kinds of food based on the total arsenic level. The United States Food and Drugs Administration (US FDA)[1] estimated that in shellfish (bivalve mollusks and crustaceans), the inorganic arsenic was about 10% of total arsenic. The Ministry of Agriculture, Fisheries and Food[2] (MAFF) in the United Kingdom reported that inorganic arsenic accounted for approximately 1-3% of total arsenic in fish while the Australia New Zealand Food Authority[3] (ANZFA) reported that 2-6% of total arsenic was inorganic arsenic in seafood.

5.7 According to our local data, the proportion of inorganic arsenic

in total arsenic in seafood ranged from 0.2% to 6.0%, which was comparable to the ANZFA data. Data on the portion of arsenic being inorganic in other food groups are limited both locally and in overseas though ANZFA reported that this figure in rice was less than 10%[4]. In its dietary exposure assessment in 1999[4], ANZFA assumed 6% of arsenic being inorganic for all food. To err on the conservative side, we assumed that 10% of total arsenic was inorganic in all food groups in our calculation of dietary exposure to allow comparison to be made with the PTWI for inorganic arsenic recommended by JECFA.

Treatment of Non-Detected Results

5.8 When the analytical value was below the limit of detection (LOD), the true value could be anywhere between zero and the LOD. The treatment for these results is particularly important when a large percentage of the analytical results of a particular food group are below LOD.

5.9 A number of approaches have been used in dealing with non-detectable results. The most commonly employed technique involves

substitution of a single value as a proxy for each non-detectable data value, which includes zero, LOD and 1/2-LOD. Other more sophisticated methods that require more data manipulation have also been suggested, for example log-probit analysis or other robust methods. These methods require enough data points above the reporting limit to define the distribution function, and transforming and extrapolating the quantified data.

5.10 While it may not be appropriate to assume a zero concentration for all the samples with analytical values below LOD, assigning the non-detects the value of LOD would, however, grossly overestimate the dietary intake particularly when the LODs are relatively high. In this study, arsenic, cadmium and mercury are only detected in limited varieties of food, thus making the more sophisticated methods inapplicable. In order not to underestimate the risk, a value of 1/2-LOD was assigned to all results below LOD.

5.11 To estimate the dietary exposure, a measure of central tendency for each food group needs to be chosen. Since the distributions

of the data in this study are skewed to the right, the median value was chosen over the mean and mode as the median can better reflect the central tendency of the skewed distribution of results. The distribution curves can be read at the Annexes.

Food Consumption Data

5.12 The food consumption data in this report are based on results of the Food Consumption Survey of local secondary school students conducted in 2000. In the Survey, a stratified three-stage sampling plan was used, with a sampling frame of 472 secondary schools and more than 380,000 students, covering almost all the local secondary schools. A total of 967 students from 27 schools participated in the survey yielding a response rate of 77% at the school level and 96% at the student level. The mean weight of the participated students was 52.0 kg. Details of the survey were covered in a separate report[5].

Data analysis

5.13 The survey covered 93 food items categorized under 13 food groups. For the purpose of this study, the food items of the survey were re-categorized into six food groups as mentioned in para. 5.2. The mean consumption (in g/day) of the six selected food groups were used to estimate the dietary exposure to the heavy metals of an average student, while the 95th percentile of the exposure level was used to represent the exposure for high consumers.

Estimated Dietary Exposure for Particular Metal

5.14 The average dietary exposure of a particular metal for each food group can be estimated by multiplying the metal concentration of the food group by the mean dietary intake of that particular food group. The general formula adopted is:

$$\text{Dietary exposure} = \frac{\text{Metal contaminant concentration} \times \text{Average dietary intake}}{\text{Body weight}}$$

5.15 The daily dietary exposure was computed by summing up the intakes from the food groups studied. The daily dietary exposure is multiplied by seven to obtain a weekly exposure level. The estimated level is then compared with the PTWI to determine whether the tolerable weekly intake has been exceeded.

High Consumers among Secondary School Students

5.16 The concept of an average diet may not be useful to estimate particular at risk group like the high consumers. Therefore, the estimate of high exposure to heavy metal was also necessary as an indicator of the extreme cases of exposure. The 90th and above percentiles have been recommended for estimating the risk of high exposure to contaminants while the 95th percentile is frequently quoted by various organisations such as WHO[6,7] and U.S. EPA[8]. In this assessment, the 95th percentile exposure level was used to represent the dietary exposure to heavy metals for high consumers.

References

[1] United States Food and Drug Administration. Guidance Documents for Trace Elements in Seafood. Center for Food Safety and Applied Nutrition. Washington, DC, USA. 1993

[2] Ministry of Agriculture, Fisheries and Food (MAFF). 1994 Total Diet Study: Metals and Other Elements, Sheet No. 131. MAFF. London, UK. 1997.

[3] Australia New Zealand Food Authority (ANZFA). Proposal P157, Contaminants in Food – Metals Full Assessment. ANZFA. Canberra, ACT, Australia. 1999a,

[4] Australia New Zealand Food Authority (ANZFA). Development of joint Australia New Zealand Food Standards; As part of the process of the Review of the Food Standards Code. Contaminants in Foods – Metals. Full Assessment Report Proposal P157. Available from: http://www.anzfa.gov.au/document/p157_-_attach_5.doc

[5] Food and Environmental Hygiene Department. Food Consumption Survey 2000. Hong Kong, 2001.

[6] World Health Organisation. Guidelines for the Study of Dietary Intakes of Chemical Contaminants. World Health Organisation; 1985.

[7] World Health Organisation. Food Consumption and Exposure Assessment of Chemicals. World Health Organisation; 1997.

[8] U.S. Environmental Protection Agency. Guidelines for Exposure Assessment. Environmental Protection Agency; 1992. Available from: http://www.epa.gov/nceawww1/exposure.htm

Chapter 6

Heavy Metal Contamination in Food

6.1 Results of 1 324, 1 376 and 1 337 samples for arsenic, cadmium and mercury analyses respectively were extracted from the food surveillance database from 1999 to 2001. After matching the data with our selected food groups, a total of 2 486 results, comprising 817, 857 and 812 analyses for arsenic, cadmium and mercury respectively, fell into our selected six food groups as described in paragraph 5.2 and were used in the estimation of dietary exposure. The remaining food items belonged to other food groups such as beverages, herbs, spices, sauces and oils. They were generally not regarded as significant sources of dietary heavy metals and were generally consumed in small amounts. Table 6.1 provides the number of food samples in different food groups used in dietary exposure estimation. The median concentrations of total arsenic, cadmium and mercury in selected food groups is given in Table 6.2.

Table 6.1: Number of Samples for Dietary Exposure Assessment To Arsenic, Cadmium and Mercury

Food Groups	Number of Samples		
	Arsenic	Cadmium	Mercury
Cereal and cereal products	40	40	41
Vegetables	22	24	22
Meat, Poultry and their products	30	29	29
Fish	227	232	233
Seafood other than fish	475	509	464
Milk and dairy products	23	23	23
Total	**817**	**857**	**812**

Table 6.2: Median concentrations (μg/kg) of total arsenic, cadmium and mercury in selected food groups

Food group	Total Arsenic		Cadmium		Mercury	
	% of samples below LOD	Median concentration (μg/kg)	% of samples below LOD	Median concentration (μg/kg)	% of samples below LOD	Median concentration (μg/kg)
Cereals and cereal products	82.5	38	60.0	10	95.1	15
Vegetables	100.0	38	66.7	10	100.0	15
Meat, poultry and their products	100.0	38	96.6	10	93.1	15
Fish	23.8	606	78.4	10	35.2	50
Seafood other than fish	4.0	1894	19.3	120	79.1	15
Milk and dairy products	100.0	38	91.3	10	100.0	15
Total	**22.2**	-----	**43.1**	-----	**69.0**	-----

6.2 Of the six food groups, "seafood other than fish" was found to contain the highest amount of total arsenic and cadmium with median

concentrations of 1 849 µg/kg and 120 µg/kg respectively, whereas "fish" was found to contain the highest amount of mercury with median concentration of 50 µg/kg. The majority of the results of other food groups were below LOD.

6.3 Arsenic was detected in three food groups, namely, "seafood other than fish", "fish" and "cereal and cereal products", in descending order of frequency. Arsenic was not detected in the food groups "vegetables", "meat, poultry and their products" and "milk and dairy products".

6.4 Cadmium was detected in all food groups. It was most frequently detected in "seafood other than fish", followed by "cereal and cereal products", "vegetables", "fish", "milk and dairy products" and "meat, poultry and their products".

6.5 Mercury was detected in four of the six food groups. It was most frequently detected in "fish", followed by "seafood other than fish", "meat, poultry and their products" and "cereal and cereal products".

Mercury was not detected in “vegetables” and “milk and dairy products”.

6.6 Distributions curves of arsenic, cadmium and mercury concentrations in the six food groups are presented in Annex 1, 2 and 3 respectively.

Chapter 7

Dietary Exposure to Heavy Metals

7.1 The concentrations of the heavy metals in the six food groups together with food consumption data of secondary school students were used to estimate the dietary exposure of secondary school students. The estimation can then be compared against the Provisional Tolerable Weekly Intake (PTWI) as recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA).

7.2 The food consumption data for six food groups which were used to estimate the dietary exposure to heavy metals are given in Table 7.1.

Table 7.1: Food Consumption for Secondary School Students

Food Group	Mean Consumption (g/day)
Cereals and cereal products	499.4
Vegetables	314.5
Meat, poultry and their products	190.3
Fish	78.6
Seafood other than fish	50.5
Milk and dairy products	158.2
Total	**1 291.5**

Dietary Exposure for an Average Secondary School Students

7.3 The dietary exposure for an average secondary school student was 2.52, 2.49 and 2.98 µg/kg bw/week for inorganic arsenic (after conversion), cadmium and mercury respectively (Table 7.2). These levels fall well within the PTWIs of 15, 7 and 5 µg/kg bw/week for the three metals respectively established by JECFA (Table 7.3).

Table 7.2: Estimated Dietary Exposure to Inorganic Arsenic, Cadmium and Mercury for Average Eaters among School Students*

Food Group	Dietary Exposure in mg/kg bw/week (%)		
	Inorganic Arsenic	Cadmium	Mercury
Cereals and cereal products	0.25 (10%)	0.67 (27%)	1.01 (34%)
Vegetables	0.16 (6%)	0.42 (17%)	0.64 (21%)
Meat, poultry and their products	0.10 (4%)	0.26 (10%)	0.38 (13%)
Fish	0.64 (26%)	0.11 (4%)	0.53 (18%)
Seafood other than fish	1.29 (51%)	0.82 (33%)	0.10 (3%)
Milk and dairy products	0.08 (3%)	0.21 (8%)	0.32 (11%)
Total	**2.52** (100%)†	**2.49** (100%)†	**2.98** (100%)†

* The mean body weight of 52.0 kg is used

† Figures may not add up to total due to rounding

Dietary Exposure for High Consumers

7.4 Further analyses were undertaken to estimate the risk that high consumers might be exposed to. This study used the 95th percentile exposure level of the school students to estimate the dietary exposure to heavy metals for high consumers.

7.5 The exposure estimates for mercury exceeded the PTWIs established by JECFA while that for inorganic arsenic and cadmium still fell within the PTWI (Table 7.3).

Table 7.3: Comparison among JECFA Provisional Tolerable Weekly Intakes (PTWIs), Dietary Exposure for Average Eaters and High Consumers for Inorganic Arsenic, Cadmium and Mercury

Heavy Metal	JECFA PTWI (μg/kg bw/week)	Exposure for Average Eaters (μg/kg bw/week)	Exposure for High Consumers (μg/kg bw/week)
Inorganic Arsenic	15	2.52	6.77
Cadmium	7	2.49	5.71
Mercury	5	2.98	6.41

Chapter 8

Discussion

8.1 In this study, risks to health from metal contaminants in selected food were assessed by comparing estimates of dietary exposure with the Provisional Tolerable Weekly Intakes (PTWIs) recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA)[1]. PTWI is the recommended safe level of exposure which is the amount of contaminant that can be ingested over a lifetime without appreciable risk. Its value represents permissible human weekly exposure to a contaminant unavoidably with the consumption of food.

Dietary Exposure

Average Exposure of Secondary School Students

8.2 We estimated that the dietary exposure to inorganic arsenic,

cadmium and mercury was 2.52, 2.49 and 2.98 µg/kg bw/week respectively for an average secondary school student. These levels fall within the PTWI of 15, 7 and 5 µg/kg bw/wk respectively as established by JECFA. The dietary exposure estimates of our results suggest that secondary school students are unlikely to experience major undesirable health effects to these heavy metal contaminants.

High Consumers among Secondary School Students

8.3 The dietary exposure estimates of high consumers for mercury have exceeded the PTWI established by JECFA, while that for inorganic arsenic and cadmium were below the PTWI. However, an intake of these heavy metals above the PTWIs does not automatically mean that health is at risk. These PTWIs represent a tolerable weekly intake for lifetime exposure and that occasional short-term excursions above the PTWIs would have no major health consequences provided that the average intake over long periods is not exceeded.

Arsenic

8.4 According to our estimation of dietary exposure to inorganic arsenic, the group "seafood other than fish" (51%) made the greatest contribution, and was followed by the groups "fish" (26%) and "cereals and cereal products" (10%). Our findings were consistent with data reported in overseas dietary exposure studies including those conducted in Australia[2], USA[3] and UK[4] which reported seafood accounted for the majority of dietary exposure to arsenic.

8.5 Data from our food surveillance programme indicated that the concentration of arsenic found in seafood was much higher than those in other food groups. High arsenic concentrations were found especially in the group "seafood other than fish" with median concentration of 1.9 mg/kg. This was consistent with findings obtained in overseas studies[2,3,4].

8.6 The LOD of 0.076 mg/kg for arsenic employed in our current study was high compared with the LODs used in overseas dietary exposure studies; an LOD of 0.03 mg/kg was used in USA[3] and 0.01 mg/kg in

Australia[2]. This relatively high LOD probably led to overestimation of the heavy metal content especially for food groups, such as "cereals and cereal products" and "vegetables", in which most of the samples were below LOD, since a value of 1/2-LOD was given to samples with concentrations below LOD.

8.7 There are concerns that arsenic from drinking water can be an important source of exposure to inorganic arsenic[5]. A maximum level of 0.01mg/litre has been set in the WHO Drinking Water Guidelines[6].

8.8 We have examined the exposure to arsenic from drinking water. According to the food consumption survey, water consumption for an average secondary school was 1.01 litre/day. Data from the Water Supplies Department (WSD)[7] showed that the average concentration of arsenic in drinking water in Hong Kong was less than 0.001 mg/litre. Assuming that 100% of the arsenic present in water is inorganic, the exposure to inorganic arsenic from drinking water for an average secondary school student was less than 0.13 μg/kg bw/week, an additional contribution of less than 0.9% to the PTWI. Therefore, drinking water is

considered an insignificant source of inorganic arsenic exposure in Hong Kong.

Cadmium

8.9 Results from our dietary exposure estimates suggested that "seafood other than fish" (33%) was the major source of cadmium, followed by "cereals and cereal products" (27%) and "vegetables" (17%). "Seafood other than fish" alone could contribute 33% of the daily intake of cadmium. This food group was also found to have the highest concentration for cadmium, and this finding was echoed in other studies that shellfish was found to contain higher concentration than did most other food[8,9]. Therefore it is less desirable for seafood–lovers to consume large amount of shellfish on a regular basis.

8.10 Another important dietary sources of cadmium include "cereals and cereal products" and "vegetables". Foods of plant origin including vegetables and cereals may take up cadmium from contaminated soil, resulting from cadmium-containing fertilizers and wastes from industrial

discharge.[10] Therefore, when considering dietary exposure to cadmium, vegetables and cereals could be of significance. This had been pointed out in the ANZFA dietary exposure study[11] in which potato (39%) and white bread (11%) were identified as significant contributors to the dietary cadmium exposure.

8.11 For smokers, significant contribution of cadmium exposure can be attributed to cigarette smoking. Breathing of cigarette smoke can absorb a substantial amount of cadmium[12]. According to the Thematic Household Survey conducted in 2000, an average current daily smoker in Hong Kong smokes 15 cigarettes per day[13] making an additional exposure from cigarette smoking of 0.71 μg/kg bw/week (i.e. 28% of the average weekly exposure) and even higher for heavy smokers. This estimation is based on the assumption that each cigarette contains 2μg of cadmium of which 10% is inhaled.[14] It is important to note that the absorption of cadmium from inhalation (15-40%) is much greater than oral exposure (4-8%)[15]. As a result, the amount of cadmium being absorbed via smoking may double the daily absorbed burden of cadmium.

Mercury

8.12 In our findings, "cereal and cereal products" (34%) contributed the highest dietary mercury exposure, followed by "vegetables" (21%). Although mercury levels of food samples from these two groups were mostly below LOD, the relatively high consumption of them might have exaggerated their contribution to the overall dietary exposure to mercury.

8.13 The LOD for mercury used in our study was 0.03 mg/kg which is higher than those used in overseas dietary exposures studies such as in US (0.01mg/kg), Australia (0.01 mg/kg) and the UK (0.003mg/kg). As discussed above, this high LOD probably led to overestimation of the concentration of mercury in food groups such as cereal and cereal products and vegetables, in which most of the results were below LOD. The overestimated mercury level would further be magnified after multiplying the high amount of consumption. The contribution of cereal and cereal products as a source of mercury would be overestimated as rice, which represented the majority of consumption in this group, was tested for mercury in its raw state, but its weight would increase by about three times

when consumed after cooking.

8.14 The surveillance results employed in the current study indicated that fish contain the highest level of mercury among the six food groups. Of the food analysed, large predatory fish such as tuna, swordfish and shark tail skirt had the highest level of mercury, similar to overseas findings.[15,16]

8.15 The estimated average dietary exposure to mercury in our study was well below the PTWI, and the International Programme of Chemical Safety of the World Health Organization (WHO/IPES)[17] opined that the general population "does not face a significant health risk from methylmercury." However, those who consume large amount of fish, especially consumption of predatory fish that are of large size or old age, and fetuses may be of particular risk. The US FDA thus advised pregnant women or women that may become pregnant to limit consumption of predatory fish such as shark and swordfish to no more than once a month because of the potential high methylmercury levels. ANZFA also shares the same view as US FDA, and has advised pregnant women to limit consumption of predatory fish.

International Comparison

8.16 Estimates of dietary exposure to arsenic, cadmium and mercury contaminants are compared with findings in overseas studies and presented in table 8.1. However, direct comparison of the data has to be done with caution because of the differences in research methodology, food group categorization, methods of collection of consumption data, methods of analyzing the contaminant concentration and methods of treating results below detection limits.

Table 8.1 A Comparison of Average Weekly Exposure of Arsenic, Cadmium and Mercury

Country	Average Weekly Dietary Exposure (μg/person/week)		
	Total arsenic	Cadmium	Mercury
Australia[2]	478-546	47-84	26-126
USA[3]	407	80.5-99.4	8.75
UK[4]	840	98	21.7
Netherlands[18]	--	--	14
Spain[19]	2108	75	129
New Zealand[20]	609	196	51
Japan[21]	1980	246	72
China[22]	-	96.6	72.1
Hong Kong	1311*	129.5	154.7

*The mean weekly dietary exposure of total arsenic is presented for an easy comparison.

8.17 Table 8.1 shows that our findings on arsenic and cadmium exposure are comparable to the dietary exposures estimated from other dietary exposure studies conducted in other places.

8.18 The dietary exposure to mercury estimated in our study is relatively high when compared with the values obtained elsewhere. In our study, the important dietary sources of mercury were "cereals and cereal products" and "vegetables" in which the high LOD coupled with high consumption are identified to be the major attributes to the apparent high value as discussed previously.

Limitations

Food Consumption Pattern

8.19 The method adopted for the collection of food consumption data may also influence the accuracy of the estimates on dietary exposure. In this study, food consumption pattern of secondary school students was

collected using a food frequency questionnaire. Although the food frequency questionnaire was very comprehensive, it was not possible to cover every single food item, some of which might be relevant to heavy metal exposure.

Food Sampling

8.20 In the food surveillance programme, food samples were taken from the local market adopting a risk-based approach. Although the number of food groups selected was considered sufficient to produce reasonable dietary exposure estimates, it fell short of representing the full range of food products consumed and might thus underestimate the exposure to heavy metals. A total diet study conducted in the US suggested that the food groups selected in this study, in the US situation, would contribute to over 90% of the total dietary exposure to arsenic and mercury and about 70% of total dietary exposure to cadmium.

Concentration of heavy metal contaminants

8.21 To make the best use of the analytical resources, metal contaminants analyses for arsenic and mercury were determined in form of total arsenic and total mercury rather than inorganic arsenic and methylmercury which have greater public health implication. Conversion factor and conservative assumption such as assuming that 100% of non-seafood arsenic was inorganic arsenic have been introduced in estimating inorganic arsenic. This would attribute to an overestimation of the metal contaminant exposure.

8.22 Food surveillance results primarily are used for the purpose of enforcement and low LODs are considered as a luxury rather than a necessity. For some of the food groups, the heavy metals concentrations of most of the samples were below LOD. By assigning a value of 1/2 LOD in the calculation would most likely overestimated the exposure.

References

[1] World Health Organization (WHO). Summary of Evaluations Performed by the Joint FAO/WHO Expert Committee on Food Additives (FECFA), 1956-1997 (First through Forty-Ninth Meeting). Geneva, Switzerland; 1999.

[2] Australia New Zealand Food Authority (ANZFA). The 19th Australian total diet survey. Australia & New Zealand: ANZFA; 2001.

[3] Egan SK, Tao SSH, Pennington JAT, Bolger PM. US food and Drug Administration's Total Diet Study: intake of nutritional and toxic elements 1991-96. Food Additives and Contaminants. 2002; 19(2):103-125.

[4] Ministry of Agriculture, Fisheries and Food (UK). Total Diet Study—Aluminium, Arsenic, Cadmium, Chromium, Copper, Lead, Mercury, Nickel, Selenium, Tim and Zinc. Food Surveillance Information Sheet No.191, 1999. Available from: URL: http://www.foodstandards.gov.uk/maff/archive/food/infsheet/1999/no191/191tds.htm

[5] World Health Organisation (WHO). Water and Sanitation United Nations Synthesis Report on arsenic in Drinking Water. Available from: URL: http://www.who.int/water_sanitation_health /Arsenic/ArsenicUNReptoc.htm.

[6] World Health Organisation (WHO). Guidelines for dinking water quality 2nd edition Vol. 1. WHO; 1993.

[7] Water Supplies Department (HKSAR). Drinking Water Quality for the Period April 2000-March 2001. Available from: URL: http://www.info.gov.hk/wsd/wq/wq-a2-b.htm

[8] Moffat GF, Whittle KJ, editors. Environmental Contaminants in Food. Sheffield: Academic Press; 1999.

[9] Joint FAO/WHO Expert Committee on Food Additives (JECFA). Evaluation of Cadmium. WHO Food Additives Series. 1988; 24.

[10] Watson DH. Food Chemical Safety Volume1: Contaminants. Cambridge, England: Woodhead Publishing Limited; 2001.

[11] New South Wales (NSW) Health Department. Metal Contamination of Major NSW Fish Species available for human consumption. New South Wales, Australia: NSW Health Department; 2001.

[12] Agency for Toxic Substances and Disease Registry (ASTDR). ToxFAQs – Cadmium. US Department of Health and Human Services. Washington DC, USA; April 1993 . Available from: URL: http://www.atsdr.cdc.gov/tfacts5.html

[13] Census and Statistic Department (HKSAR). Thematic Household Survey. Report No. 5. Hong Kong; 2001.

[14] Ros JPM, Sloff W, editors. Integrated criteria document cadmium. Bilthoven Netherlands National Institute of Public Health and Environmental Protection (Report no. 758476004); 1987.

[15] United States Food and Drugs Administration (US FDA). FDA Announces Advisory on Methyl Mercury in Fish. FDA Talk Paper. March 2001. Available from: URL: http://www.cfsan.fda.gov/~lrd/tphgfish.html

[16] Canadian Food Inspection Agency (CFIA). Mercury and Fish Consumption. Consumer Fact Sheet. 1999. Available from: URL: http://www.inspection.gc.ca/english/corpaffr/foodfacts/mercurye.shtml

[17] International Programme of Chemical Safety (IPCS) of the World Health

Organization. [CD-ROM] IPCS Inchem. Geneva, Switzerland; July 2001.

[18] Dabeka RW and Mckenzie AD. Survey of lead, cadmium, fluoride, nickel, fluoride, nickel and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988. Journal of AOAC International . 1995. Vol. 78, 897-909.

[19] Jalón M, Urieta I, Macho ML, Azpiri M. vigilancia de la contaminación quimica de los alimentosen laComunidad Autónoma del Pais Vasco, 1990-1995 (Food chemical surveillance in the Basque Country, 1990-1995). Bilbao, Spain: Servicio Central de Publicaciones del Gobierno Vasco. 1997.

[20] Ministry of Health. 1997/98 New Zealand Total Diet survey-- Part 2 Elements: Selected contaminants and nutrients. Feburary 2000.

[21] Tsuda T, Inoue T, Kojima M, Aoki S. Market basket and duplicate portion estimation of dietary intakes of cadmium, mercury, arsenic, copper, manganese, and zinc by Japanese adult. Journal of AOAC International. 1995; 78(6):1363-68.

[22] Hong, W. Journal of Hygiene Research. Vol. 22 Supplement. Institute of Nutrition and Food Hygiene, Chinese Academy of Preventive Medicine; May, 1993. Beijing, China.

Chapter 9

Conclusions and Recommendations

9.1 The dietary exposures to the three heavy metals for an average secondary school student were 2.52, 2.49 and 2.98 μg/kg bw/week for inorganic arsenic, cadmium and mercury respectively. They are all within the PTWIs as recommended by the JECFA for the respective heavy metals. It can be concluded that an average secondary school student would be unlikely to experience major toxicological effects of the three heavy metals. The dietary exposures for high consumers were 6.77, 5.71 and 6.41 μg/kg bw/week for inorganic arsenic, cadmium and mercury respectively. The exposure for mercury exceeded the PTWI. For inorganic arsenic and cadmium, the exposure fell within the PTWIs.

Major Dietary Source of Arsenic, Cadmium and Mercury

9.2 "Cereal and cereal products" had been identified as a major

source of dietary exposure to mercury (1.01 μg/kg bw/week). This figure, however, was likely to be overestimated as the majority of samples in this food group were below LODs for the heavy metals, relatively high LODs used in laboratory testing and the relatively large amount of products being consumed.

9.3 The group "seafood other than fish", particularly shellfish, was identified as the main dietary source of cadmium and inorganic arsenic. Since this group had the highest median concentration of cadmium and arsenic. While "fish" particularly predatory fish, had the highest concentration in mercury, they can be significant sources of heavy metals especially when they are consumed in large amount.

Recommendations

9.4 Food is recognized as one of the major sources of heavy metals exposure. Since heavy metals are products of environmental pollution resulting from various industrial activities, the ultimate goals of reducing heavy metals exposure are to control heavy metals emissions as well as

interrupting their pathways into food.

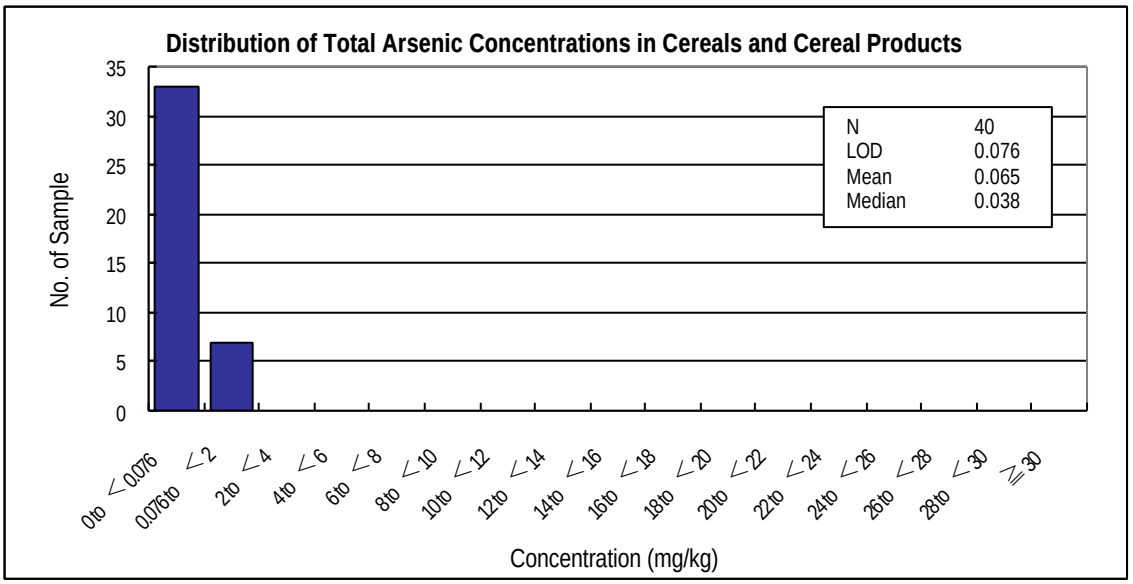
9.5 The limits of detection (LOD) of heavy metals testing were relatively high as they were designed for food surveillance and enforcement purposes. This would introduce uncertainties in exposure studies significantly especially when a large proportion of results were below LOD. We recommend that use of analytical methods with LOD set at level as low as practicable for dietary exposure studies. With the establishment of the Food Research Laboratory (FRL) by this Department, lowering reporting limits of contaminants could be achieved.

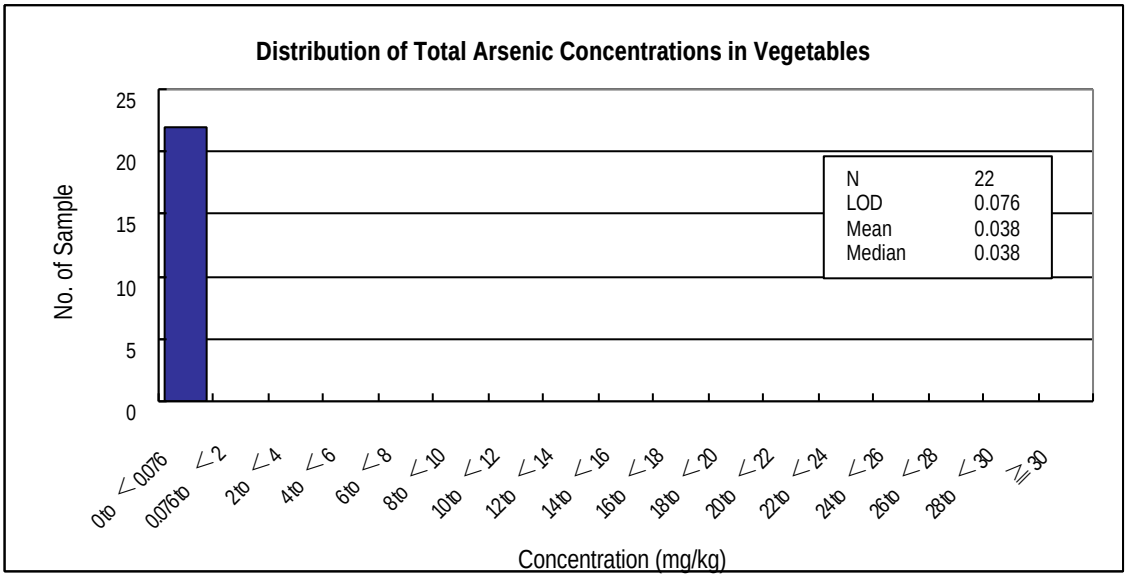
9.6 In this study, consumption data was obtained from the Food Consumption Survey conducted by this Department on secondary school students. To monitor the trend of the exposure and produce more accurate estimates, we will explore the possibility of conducing a population-based food consumption survey so that population-wide dietary exposure studies can be conducted in the future.

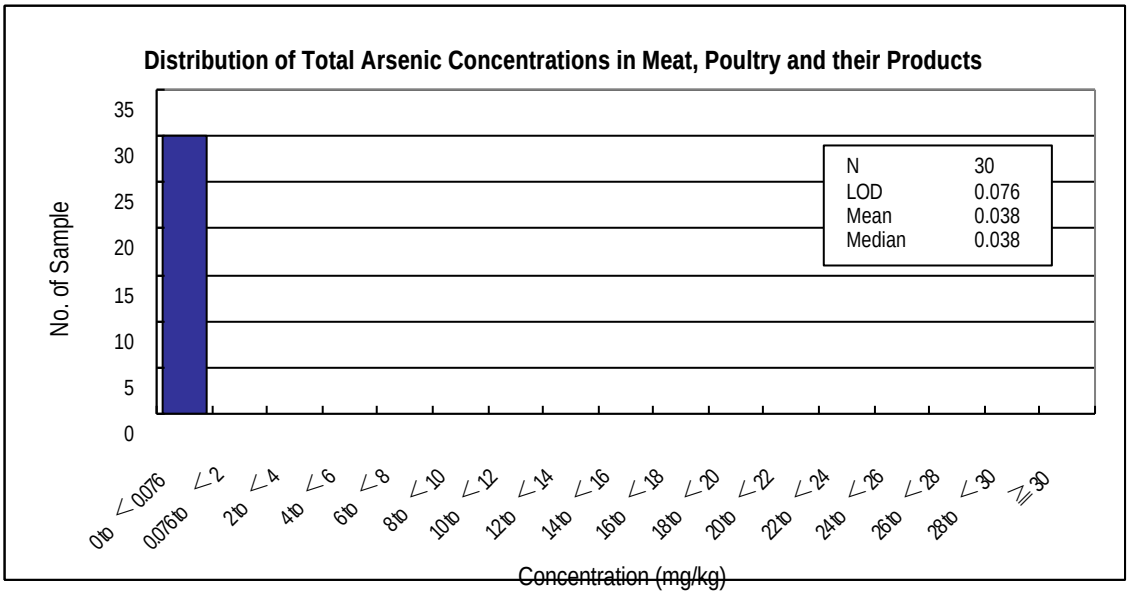
9.7 A balanced diet is essential to avoid excessive exposure to

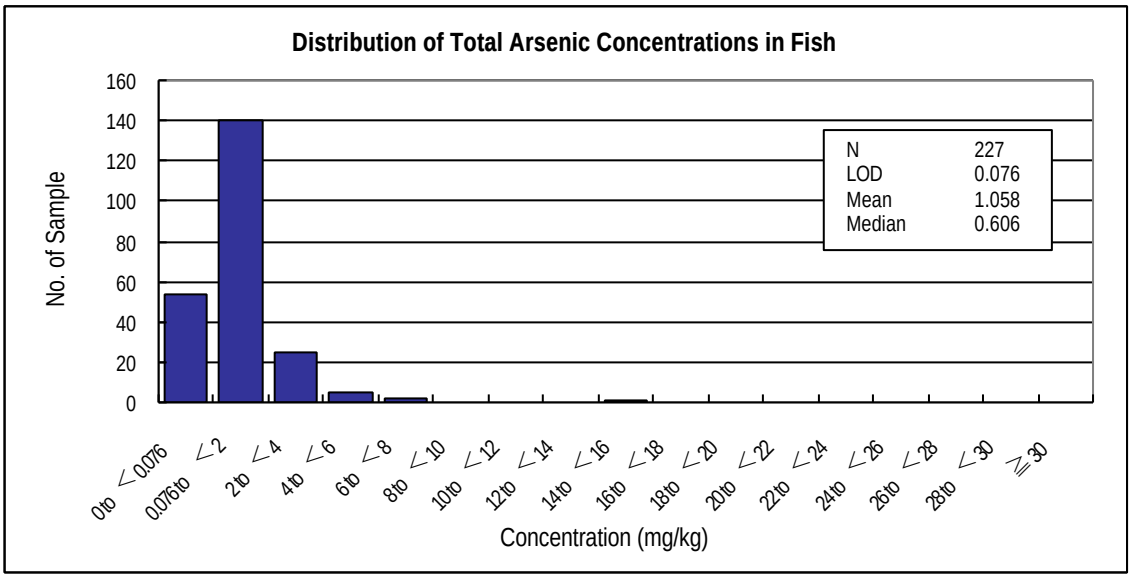
contaminants from a small range of food items. Vulnerable groups such as children and pregnant women should be careful in the selection of food, in particular, they are advised not to consume excessive amount of predatory fish such as shark, tuna and swordfish, which may contain higher concentrations of mercury. While fish are excellent sources of high-quality protein and low in saturated fat, and moderate consumption is recommended. Food safety authority in countries like the UK, USA, Australia and Canada also shares the same view. Consumers are also advised not to overindulge in shellfish as they tend to contain higher level of arsenic and cadmium, as well as other food hazards.

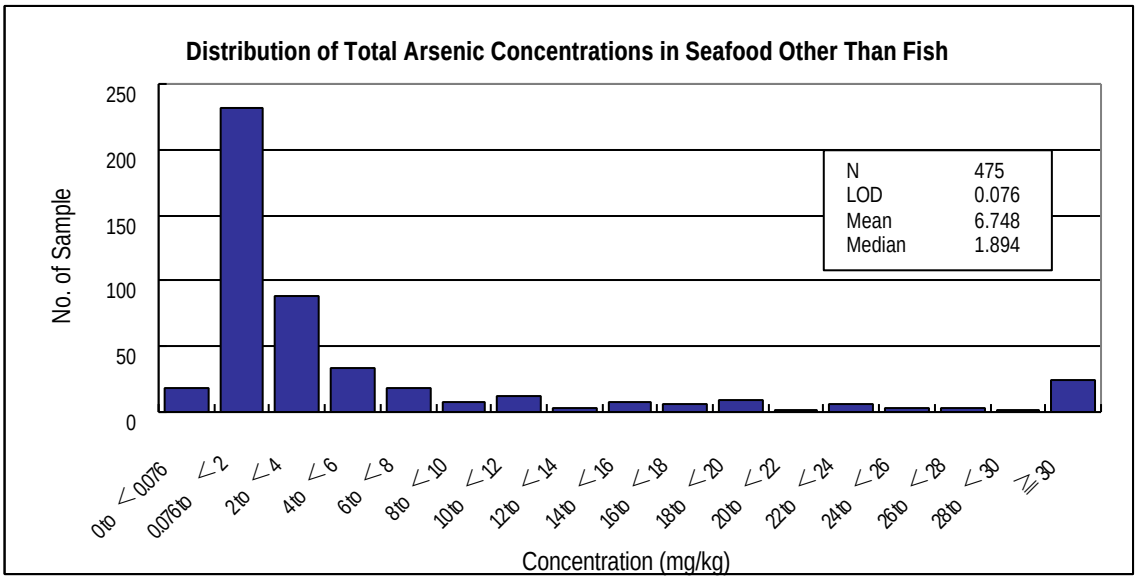
Annex 1: Distributions of Total Arsenic Concentration in Six Food Groups

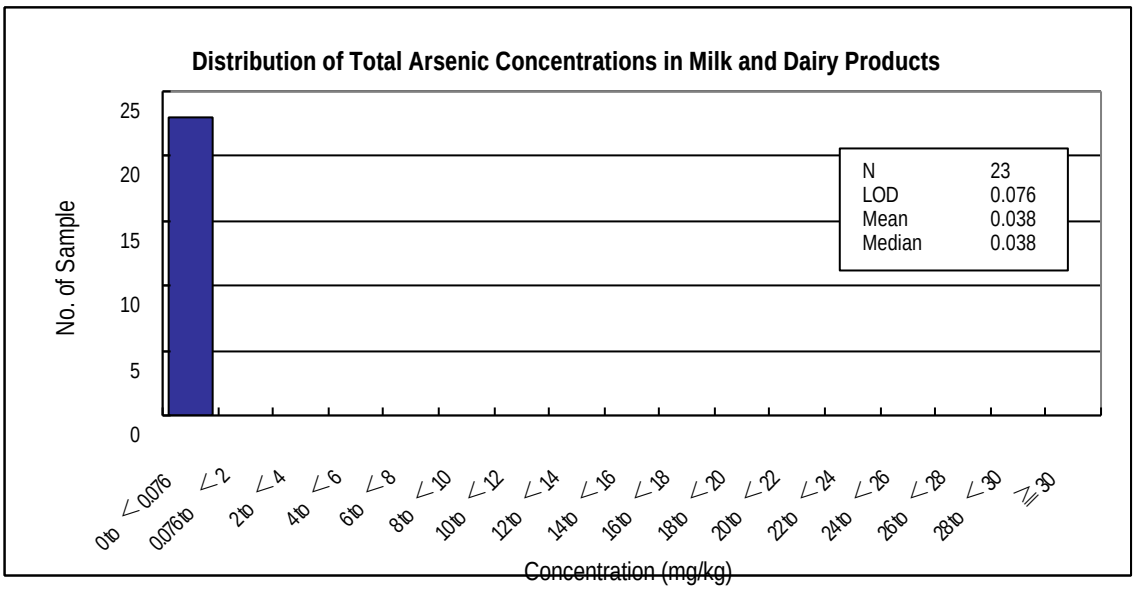




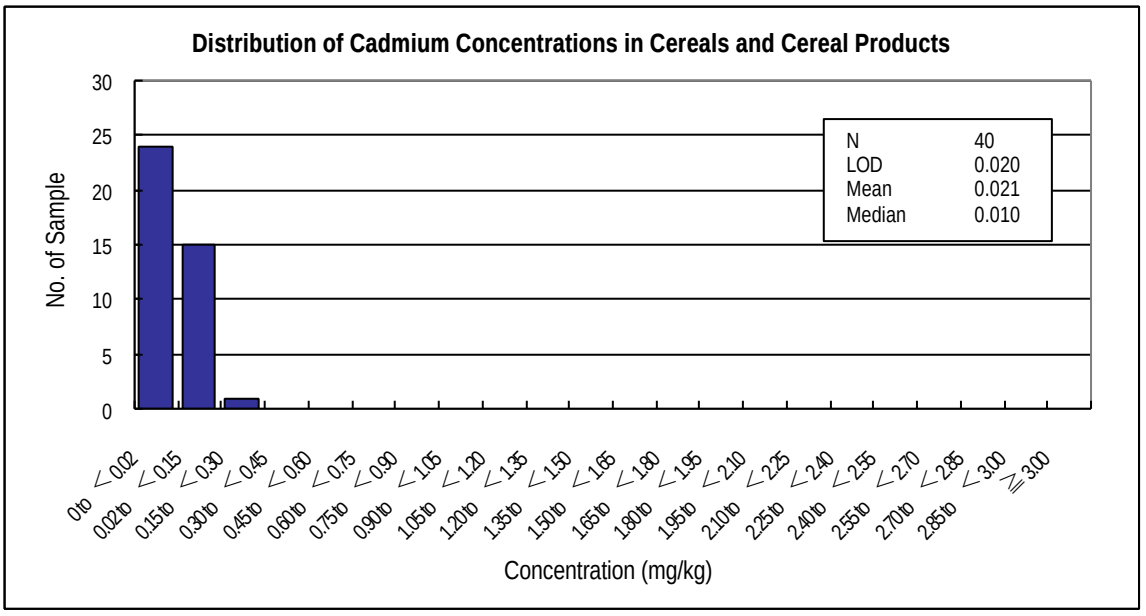


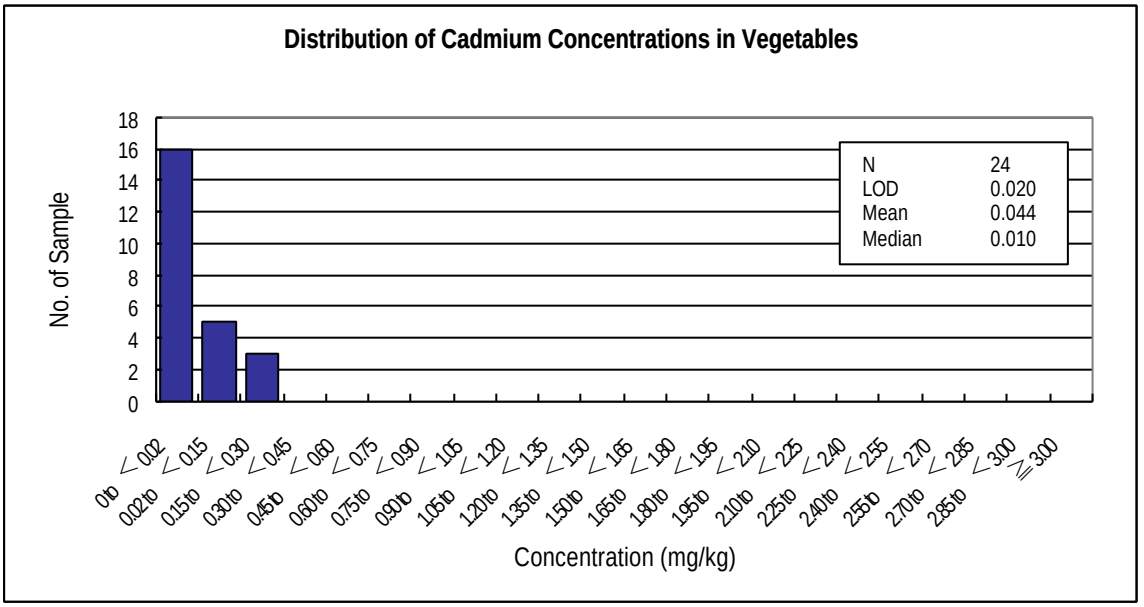

Distribution of Total Arsenic Concentrations in Fish
No. of Sample
Concentration (mg/kg)
N 227
LOD 0.076
Mean 1.058
Median 0.606

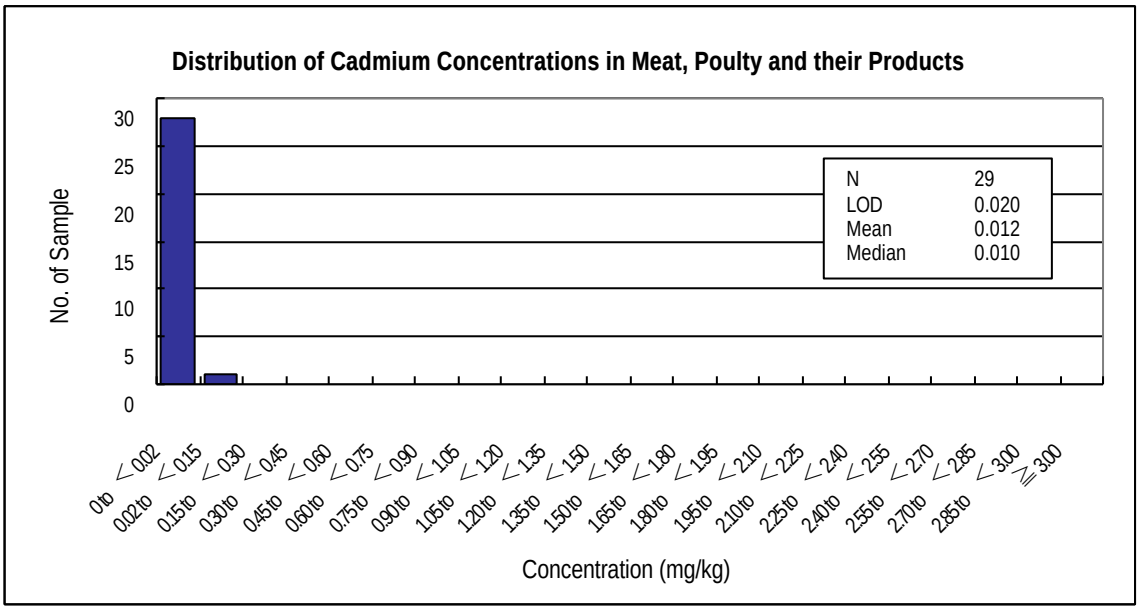

Distribution of Total Arsenic Concentrations in Seafood Other Than Fish
No. of Sample
Concentration (mg/kg)
N 475
LOD 0.076
Mean 6.748
Median 1.894

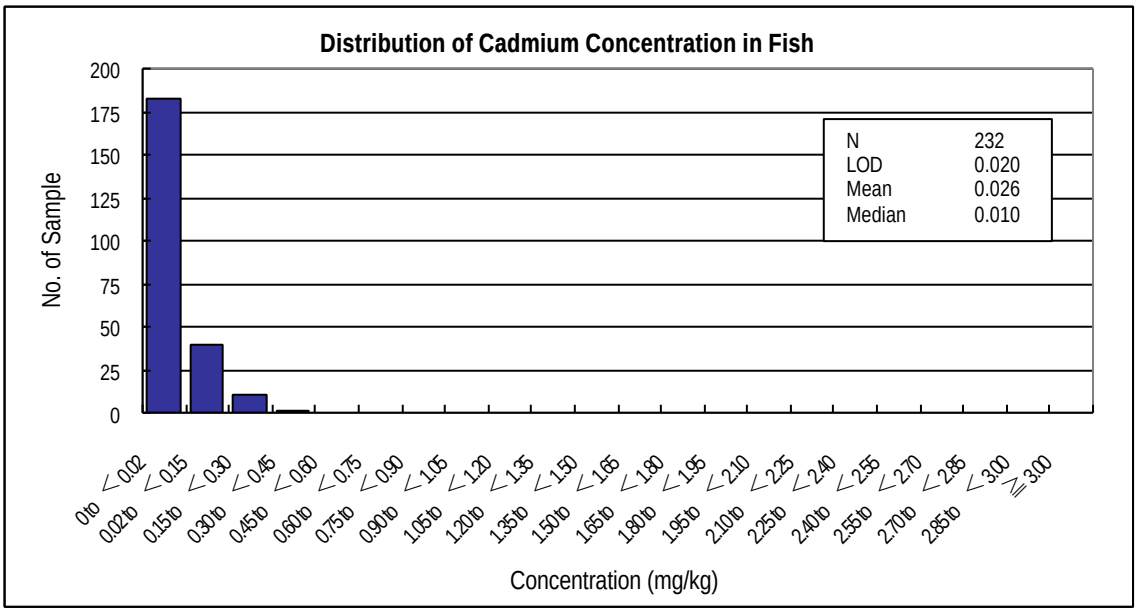

Distribution of Total Arsenic Concentrations in Milk and Dairy Products
No. of Sample
Concentration (mg/kg)
N 23
LOD 0.076
Mean 0.038
Median 0.038

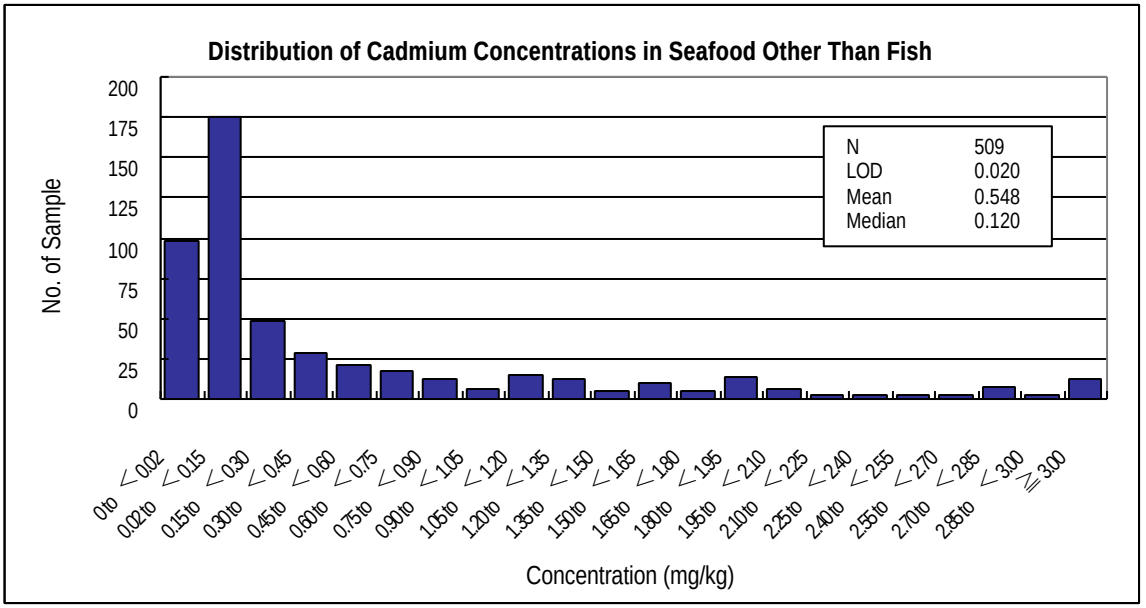
Annex 2: Distributions of Cadmium Concentration in Six Food Groups

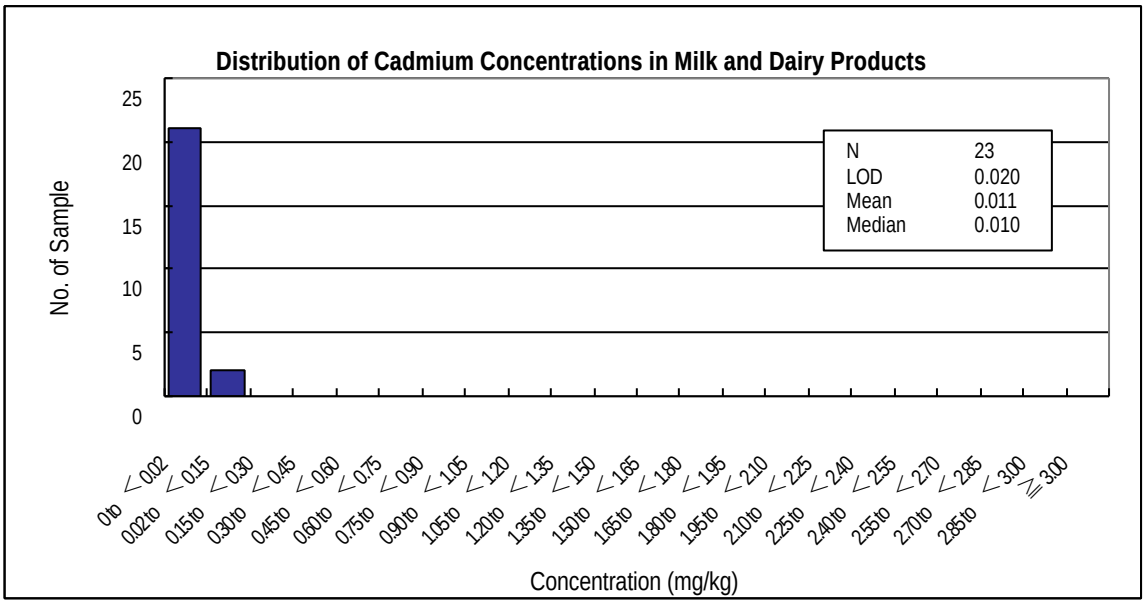




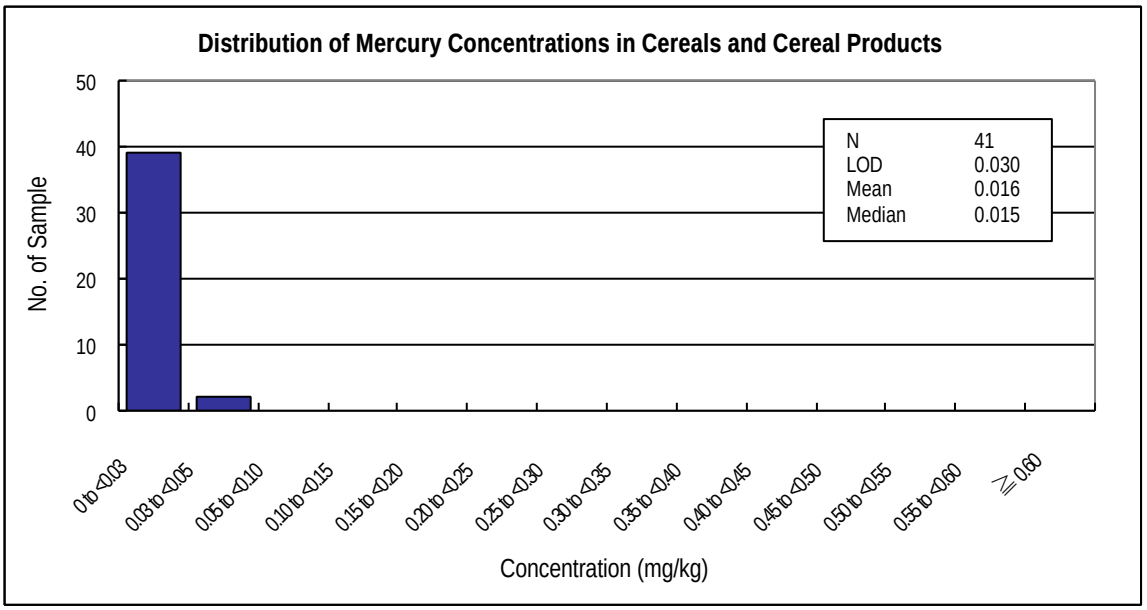


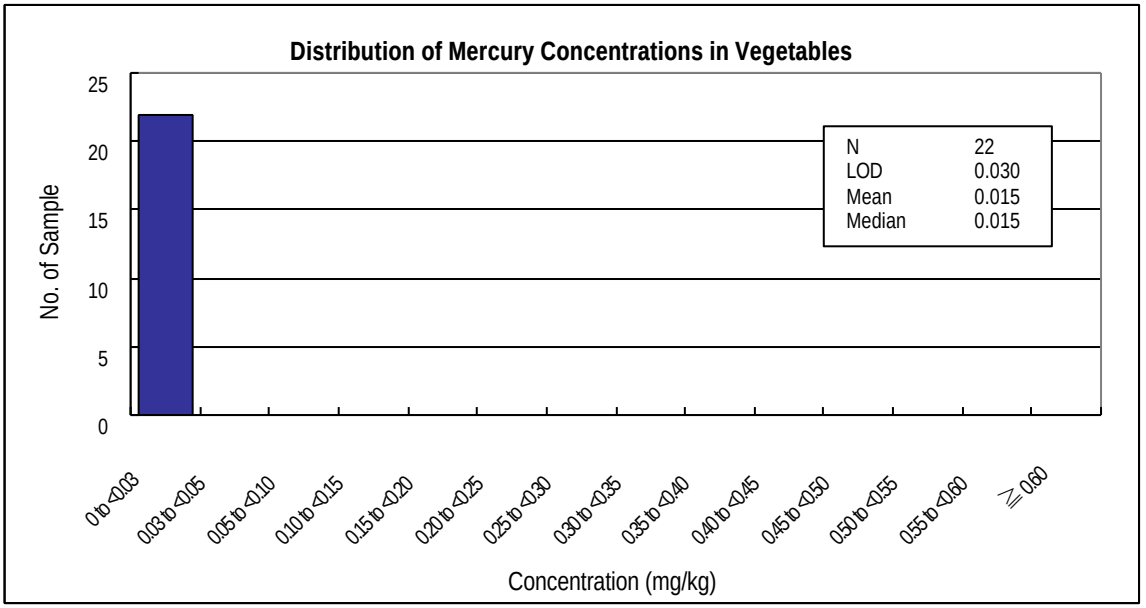

Distribution of Cadmium Concentration in Fish
No. of Sample
200
175
150
125
100
75
50
25
0
N 232
LOD 0.020
Mean 0.026
Median 0.010
0to
0.02to
0.15to
0.30to
0.45to
0.60to
0.75to
0.90to
1.05to
1.20to
1.35to
1.50to
1.65to
1.80to
1.95to
2.10to
2.25to
2.40to
2.55to
2.70to
2.85to
Concentration (mg/kg)

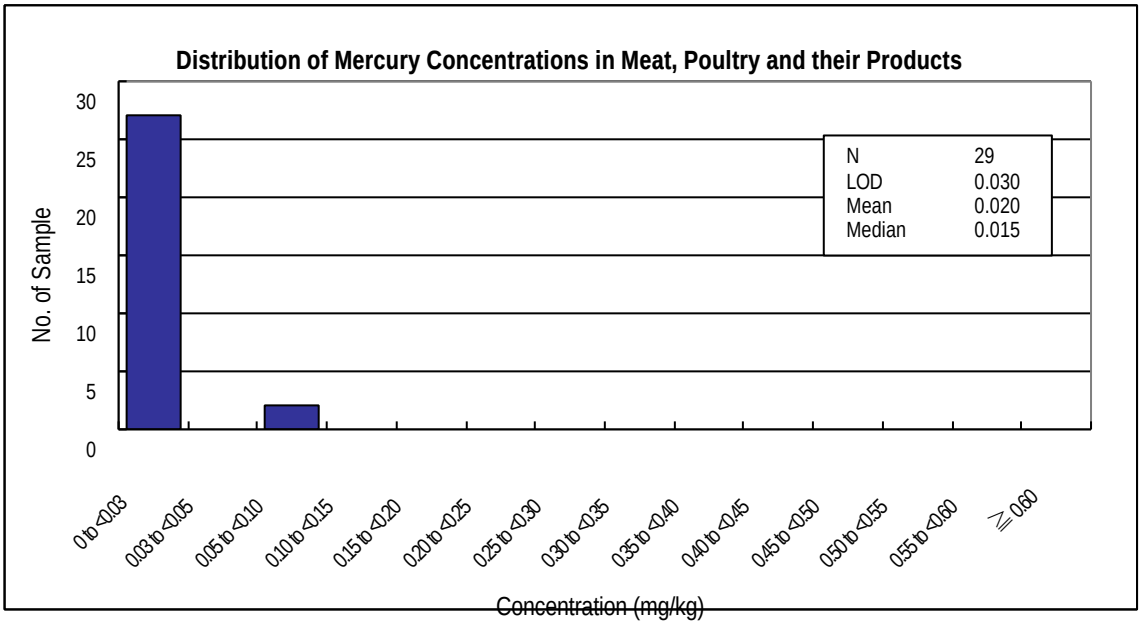

Distribution of Cadmium Concentrations in Seafood Other Than Fish
No. of Sample
200
175
150
125
100
75
50
25
0
N 509
LOD 0.020
Mean 0.548
Median 0.120
Concentration (mg/kg)

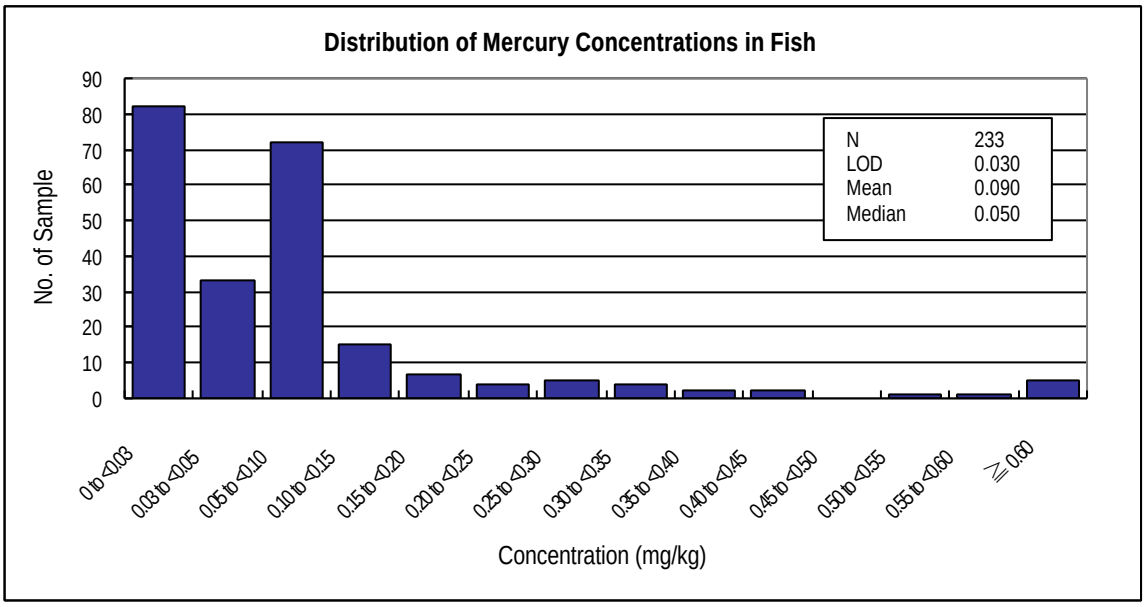

Distribution of Cadmium Concentrations in Milk and Dairy Products
No. of Sample
25
20
15
10
5
0
N 23
LOD 0.020
Mean 0.011
Median 0.010
Concentration (mg/kg)

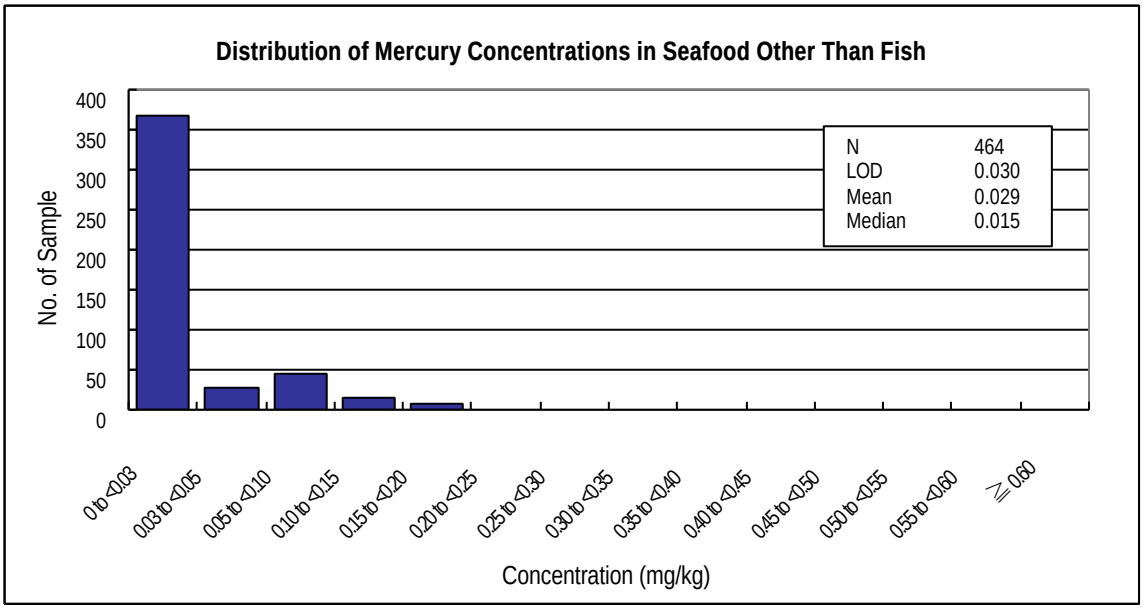
Annex 3: Distributions of Mercury Concentration in Six Food Groups

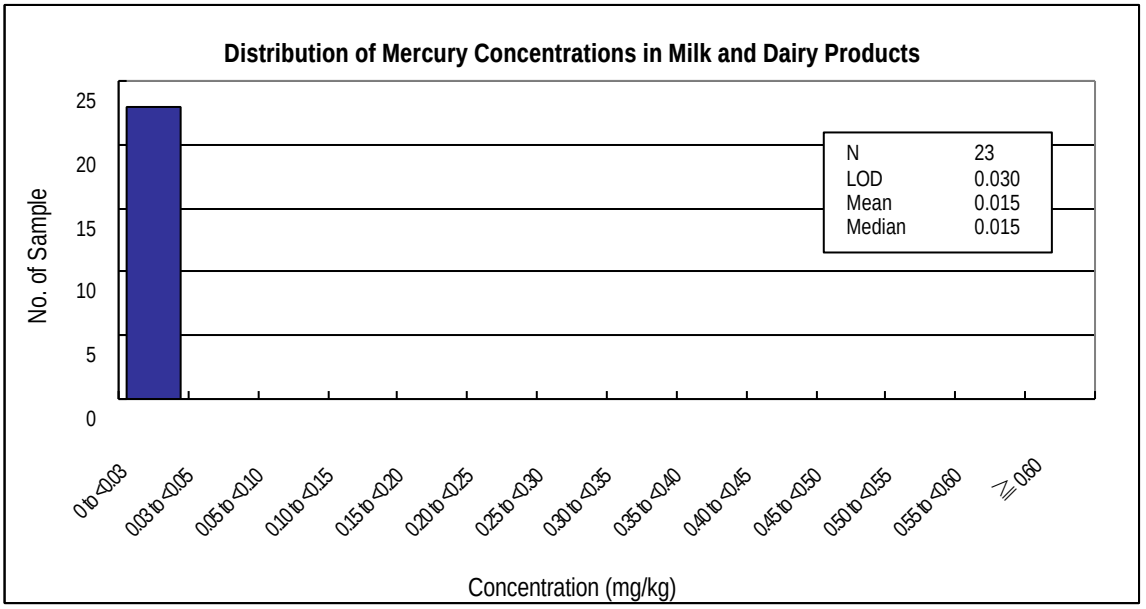







Distribution of Mercury Concentrations in Fish
No. of Sample
90
80
70
60
50
40
30
20
10
0
N 233
LOD 0.030
Mean 0.090
Median 0.050
0 to <0.03
0.03 to <0.05
0.05 to <0.10
0.10 to <0.15
0.15 to <0.20
0.20 to <0.25
0.25 to <0.30
0.30 to <0.35
0.35 to <0.40
0.40 to <0.45
0.45 to <0.50
0.50 to <0.55
0.55 to <0.60
≧ 0.60
Concentration (mg/kg)


Distribution of Mercury Concentrations in Seafood Other Than Fish
No. of Sample
400
350
300
250
200
150
100
50
0
N 464
LOD 0.030
Mean 0.029
Median 0.015
0 to <0.03
0.03 to <0.05
0.05 to <0.10
0.10 to <0.15
0.15 to <0.20
0.20 to <0.25
0.25 to <0.30
0.30 to <0.35
0.35 to <0.40
0.40 to <0.45
0.45 to <0.50
0.50 to <0.55
0.55 to <0.60
≧ 0.60
Concentration (mg/kg)


Distribution of Mercury Concentrations in Milk and Dairy Products
No. of Sample
25
20
15
10
5
0
N 23
LOD 0.030
Mean 0.015
Median 0.015
0 to <0.03
0.03 to <0.05
0.05 to <0.10
0.10 to <0.15
0.15 to <0.20
0.20 to <0.25
0.25 to <0.30
0.30 to <0.35
0.35 to <0.40
0.40 to <0.45
0.45 to <0.50
0.50 to <0.55
0.55 to <0.60
≧ 0.60
Concentration (mg/kg)