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LegCo Panel on Food Safety and Environmental Hygiene

Study on Dietary Exposure of Secondary School Students to Dioxins and Heavy Metals

PURPOSE

This paper presents the findings of the study conducted by the Food and Environmental Hygiene Department (FEHD) on dietary exposure of secondary school students to dioxins and heavy metals.

BACKGROUND

2. Dioxins and heavy metals are environmental contaminants which can cause adverse effects on human health. Dietary intake is one of the major routes of human exposure to these contaminants. In the Policy Address 2000, FEHD pledged to conduct a study on dietary exposure of local secondary school students to dioxins and heavy metals in 2001-02 in order to identify the major dietary sources of these contaminants and to evaluate the risk posed to the students. The study has been completed. Copies of the full reports of the study have been deposited with the Secretariat for perusal by Members.

DIOXINS

3. Dioxins are a group of polychlorinated aromatic compounds arising either naturally or as by-products of industrial activities e.g. metal smelting, bleaching of paper pulp, etc. They are toxic and stable. Once produced, they tend to persist in the environment and concentrate in the food chain. Dioxins are most commonly found in food items that are rich in animal fat such as meat, fish and dairy products. While acute oral toxicity is rare, studies have shown that chronic exposure to dioxins

may be cancer causing to humans.

HEAVY METALS

4. Heavy metals are environmental contaminants that are present naturally in the Earth's crust. They may also be discharged to the environment through industrial uses. The possible health effects of each kind of heavy metals vary depending on the unique features of individual metal and the route of exposure. Acute toxicity resulting from ingesting food contaminated with heavy metals is uncommon, but chronic exposure to these metals may result in undesirable toxic effects. In our study, three types of heavy metals, namely arsenic, cadmium and mercury, are covered. They are chosen for study among the various types of heavy metals mainly because of their relatively pronounced toxicities. Inorganic arsenic, the toxic form of arsenic, is a human carcinogen whereas cadmium can affect renal function. Mercury is a toxic chemical, particularly in its organic form, which affects particularly the nervous system. The highest concentration of arsenic in food are usually found in aquatic foods, especially shellfish, whereas cadmium is present in low concentrations in most food but aquatic food such as shellfish can contain relatively high concentrations. Mercury is commonly found in higher concentration in predatory fish such as shark, tuna and swordfish.

SCOPE AND METHOD OF THE STUDY

5. The level of dietary intake of the selected contaminants (i.e. dioxins, arsenic, cadmium and mercury) by secondary school students, which are the target population of our study, is evaluated by assessing the average level of these contaminants contained in the target food groups and the amounts of those foods consumed by the students. We conducted a food consumption survey in late 2000-2001 to obtain the food consumption data on a list of commonly consumed food items by secondary school students. Based on the data collected during the survey, the dietary patterns of 903 secondary school students from 27 secondary schools are analyzed in our study. As regards the estimation of the contaminant levels in the food items, we have made use of the data collected under our food surveillance programme between 1999 and 2001. The dietary exposure data thus computed are then compared with international reference on safety intake levels, and the adverse effects

likely to occur in the target population are estimated.

INTERNATIONAL REFERENCE ON SAFETY INTAKE LEVELS

Dioxins

6. The World Health Organisation (WHO) recommended a tolerable daily intake (TDI) of 1-4 TEQ[1] pg/kg of body weight (bw) for dioxins in 1998. TDI is the amount of a toxic substance, expressed on a body weight basis, which an individual may ingest daily over a lifetime without appreciable risk to health. It stresses on lifetime exposure. Occasional short-term excursions above the TDI would have no health consequences provided that the averaged intake over long period is not exceeded.

Heavy Metals

7. The Joint Food and Agriculture Organization (FAO) / WHO Expert Committee on Food Additives (JECFA) recommended the safe exposure levels in terms of Provisional Tolerable Weekly Intakes (PTWIs) for inorganic arsenic, cadmium and mercury at 15, 7 and 5μg/kg bw/week respectively. PTWI is an estimate of the amount of a chemical that can be ingested per week over a lifetime without appreciable risk. Transient excursions above the PTWI would have no health consequences provided that the averaged intake over long period is not exceeded as the emphasis of PTWI is on lifetime exposure.

ASSESSMENT OF DIETARY EXPOSURE

Dioxins

8. The study reveals that the dietary exposure of an average secondary school student to dioxins is 0.85 pg TEQ/kg bw/day. This level of intake is well below the TDI as recommended by WHO in 1998, thus implying that the average secondary school student in Hong Kong would not experience major toxicological effects of dioxins. To

[1] The toxicity of dioxins, expressed in toxic equivalence (TEQ), is measured relative to the toxicity level of the most toxic dioxin, known as TCDD. This tolerable intake level includes both dioxins and dioxin-like compounds. The latter is not included in this study.

estimate the dioxin exposure of high consumers, those students with above 95th percentile exposure level (with respect to dioxin) were studied. The dioxin exposure of these high consumers is estimated to be 2.07 pg TEQ/kg bw/day, a level falling within the range of TDI recommended by WHO. Meat, poultry and their products are identified as the major dietary sources of dioxins. Dioxin concentration in milk is not high but the relatively large consumption amount renders it an important dietary source of dioxins. Conversely, although the dioxin concentration in eggs is high, its low consumption level makes it a less significant contributor to total dioxin exposure.

Heavy Metals

9. The estimated dietary exposure of an average secondary school student to inorganic arsenic, cadmium and mercury are 2.52, 2.49 and 2.98 μg/kg bw/week respectively. Major toxicological effects arising from dietary exposure of the secondary school students to these heavy metals are not anticipated as the estimated dietary exposure levels are below the PTWI established by JECFA. For the high consumers, their dietary exposures to inorganic arsenic, cadmium and mercury are estimated to be 6.77, 5.71 and 6.41μg/kg bw/week respectively, which are below the PTWIs of these heavy metals established by JECFA except for mercury. As mentioned in paragraph 7 above, an intake above the PTWI does not automatically mean that health is at risk because the PTWI represents a tolerable weekly intake for lifetime exposure and that occasional short term excursions above the PTWI would have no major health consequences provided that the averaged intake over long period is not exceeded. For cadmium and inorganic arsenic, food items falling under the food group “seafood other than fish”, in particular shellfish, are the main dietary sources. Predatory fish is found to have the highest content of mercury.

LIMITATIONS OF THE STUDY

10. Similar to dietary exposure studies conducted by other countries which all have their inherent limitations such as time and resources constraints, our study, as the first of its kind ever conducted in Hong Kong, also has a number of limitations. As we have made use of existing data collected from the food consumption survey and the

food surveillance programme to compute the dietary exposure, the following limitations have to be noted:

(a) The use of food surveillance data in estimating the contaminant concentration levels of food items might produce biased results on the high side. It is because our food surveillance programme adopts a risk-based approach. As a result, food samples chosen for testing would tend to contain higher level of contaminants.

(b) The dietary consumption pattern of secondary school students is obtained through a food consumption survey using self-administered food frequency questionnaires. Although the questionnaire is very comprehensive, some food items may be missed out. This may lead to underestimation of the dietary exposure.

(c) The limits of detection (LOD)[2] adopted in the testing of food samples under the food surveillance programme are usually set at relatively higher levels. For food samples of which the level of contaminants is below LOD, we do not have any actual level but have to assign 1/2 LOD as the level of contaminants. This may result in overestimation of the level of contaminants. The overestimation is most obvious for those food groups with the majority of food samples below the LOD.[3]

11. While it would be ideal to collect very comprehensive data to enhance the accuracy of dietary exposure estimates, we are mindful of the long time needed (easily taking several years) and costs involved. A simplified, yet scientifically adequate approach using existing data like

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[2] The limit of detection is the smallest measure that can be detected with reasonable certainty for a given analytical procedure.

[3] For example, cereal and cereal products appear to be a major dietary source of mercury in the study. However, the majority (95%) of the samples are below LOD. Given that the pattern of the levels of contaminants in food tends to follow a positively skewed distribution in which the majority of the food have lower levels of contaminants and a smaller proportion of food have high levels, the use of 1/2 LOD to these samples in this circumstance have probably overestimated the true level of mercury.

our study, is able to provide information on our dietary exposures within a short period of time, providing early alert if actions need to be taken promptly. Despite the limitations of the study, these have not affected the overall findings and conclusions which compare favourably with those of other countries. It is prudent to conclude that an average secondary school student in Hong Kong would not experience major toxicological effects of dioxins or heavy metals studied.

THE NEXT STEP

12. To monitor the trend of the exposures and produce more accurate estimates, we will explore the possibility of conducting a population-based food consumption survey so that population-wide dietary exposure studies can be conducted in the future. In addition, we plan to use our newly established Food Research Laboratory in support of any further dietary exposure studies. In particular, we will employ analytical methods to detect contaminants in food samples at much lower concentrations. This will enable us to obtain a more accurate assessment on dietary exposure to food contaminants which will in turn contribute to the overall planning of our food control strategy.

CONCLUSION

13. The findings of the study, which is the first of its kind ever conducted in Hong Kong, have provided us with useful initial information on the major dietary sources of dioxins and heavy metals, and their risks posed to the population arising from dietary exposure. It can be used as a baseline for further dietary exposure studies to be carried out in future. While its results indicate that an average secondary school student in Hong Kong would not experience major toxicological effects of dioxins and heavy metals covered by the study, high consumers may be exposed to higher risks of undesirable health effects. As these are undesirable contaminants, consumers are recommended to maintain a balanced diet in order to reduce the risk of excessive exposure to these contaminants that may be concentrated within a small range of food items. Examples of risk reduction measures for lowering the intake of dioxins include consuming low-fat products, trimming fat from meat and meat products, reducing the amount of animal fat used in food preparation and

using cooking methods that can reduce fat since dioxins are mainly present in the fatty parts of foods. Similarly, as predatory fish such as sharks, tuna and swordfish tend to contain higher concentration of mercury whereas shellfish tend to contain higher concentrations of arsenic and cadmium, consumers are advised not to consume excessive amount of these products. Vulnerable groups such as children and pregnant women should be particularly careful in the selection of foods.

PUBLICITY

14. We will publicize the findings of the study through various channels to advise the public of the risk factors concerned and the ways to reduce the possible risk. The reports of the study will be uploaded onto the website of FEHD (http://www.fehd.gov.hk) and will be available at the Communication Resource Unit of FEHD, major public libraries as well as the Health Education Exhibition and Resources Centre of FEHD.

FOOD AND ENVIRONMENTAL HYGIENE DEPARTMENT
October 2002

食物環境衞生署

Food and Environmental Hygiene Department

DIETARY EXPOSURE TO DIOXINS OF SECONDARY SCHOOL STUDENTS

September 2002

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List of Abbreviations

AOAC	Association of Official Agricultural Chemists
bw	Body weight
U.S. EPA	United States Environmental Protection Agency
GC-HRMS	Gas chromatography-high resolution mass spectrometry
HKSAR	Hong Kong Special Administrative Region
IARC	International Agency for Research on Cancer
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LD_{50}	Lethal dose 50
LOD	Limit of detection
LOAEL	Lowest-observed-adverse-effect level
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo-para-dioxin
PCDF	Polychlorinated dibenzofuran
PTMI	Provisional tolerable monthly intake
TCDD	2,3,7,8-tetrachlordibenzo-para-dioxin
TDI	Tolerable daily intake
TEF	Toxic equivalency factors
TEQ	Toxic equivalent
TMI	Tolerable monthly intake
WHO	World Health Organisation

Executive Summary

Dietary Exposure to Dioxins of Secondary School Students

Purpose

The risks posed by dioxins to the local population was assessed through a dietary exposure study of secondary school students. This paper describes the methods and findings of the study. The implications of these findings and possible risk management options are also discussed.

Dioxins and Dietary Exposure

2. Dioxins, a group of chemicals consisting of polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), have raised public health concerns because of their possible health implication and potential cancer-causing effects. One of the

dioxin congeners, 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD), has been identified as a human carcinogen (International Agency for Research on Cancer classified TCDD as Group 1 carcinogen). Ongoing studies are undertaken to study whether other dioxin congeners are also cancer causing.

3. There is no industrial use of dioxins. These chemical contaminants were generated because of incomplete combustion in incinerators, automobile emissions, bleaching of paper pulp, manufacture or use of defoliants and pesticides, as well as other natural phenomena such as volcano eruptions and forest fires. Dioxins could enter the food chain through deposition in soils and plants and accumulated in animals. Though other routes of exposure exist, dietary exposure has been regarded as the major route of dioxin exposure in the general population.

4. In the past years, there have been global efforts in controlling dioxin accumulation in humans. Generally two approaches are undertaken. One is to reduce the source of dioxin emissions and the other is to establish a tolerable daily intake for dioxins. To date, the

most commonly described tolerable intake level was recommended by World Health Organisation (WHO) in 1998. The WHO recommended that the dietary exposure to dioxins and related compounds should not be higher than 1 - 4 pg toxic equivalent (TEQ) per kg body weight (bw) per day.

Scope and Method

5. This dietary exposure assessment used two sets of data, data on food consumption pattern and dioxin concentrations in relevant food groups.

6. In Hong Kong, data on food consumption pattern are limited. In late 2000, a Food Consumption Survey using food frequency questionnaire and food measurement aids were conducted among secondary school students. The survey collected food consumption pattern for high risk food items.

7. Under the food surveillance programme of the Food and

Environmental Hygiene Department, a wide variety of food samples are taken regularly at every stage of the supply chain: from import and manufacture to the wholesale and retail stages for microbiological, chemical and radiological testing, including testing for dioxins, which commenced in 1999. The laboratory testing of dioxins consisted of two stages, fat extraction process followed by gas chromatography – high resolution mass spectrometry. To study dioxin concentrations in food products, we used the data on dioxins extracted from the food surveillance programme. The results of 88 products belonging to the target food groups collected from January 2000 to April 2001 were used to estimate the dioxin concentrations.

8. In this study, we adopted the "selective studies of individual foods" approach for the estimation of dietary exposure to dioxins. This is a more flexible approach and is one of the approaches recommended by the WHO. The dietary exposure to dioxins could be estimated using the available data from the food surveillance programme and the food consumption data on relevant food groups collected from the Food Consumption Survey.

Dioxin Exposure of Secondary School Students

9. Since dioxins are commonly found in food items of high fat content, consumption data on food items under five food groups of interest were extracted from the Food Consumption Survey for the dietary exposure study. They were meat and meat products, poultry and poultry products, seafood, milk and milk products, as well as eggs and egg products.

10. The laboratory reports provided the concentration of all the 17 dioxin congeners in a food sample. Total dioxin concentration in the food sample, the toxic equivalent (TEQ) concentration, was then computed using the relevant Toxic Equivalency Factors (TEFs) with reference to the WHO–TEF scheme developed in 1997. TEF refers to the equivalent toxic effect of the concerned dioxin congener comparing with its most toxic counterpart – TCDD. The total dioxin concentration for each food sample was computed. Taking into account the skewed distribution of the dioxin concentrations, dioxin concentration for each food group was represented by its median.

11. Using the above two sets of data, dietary exposure to dioxins was determined. **For an average secondary school student in Hong Kong, the dioxin exposure was 0.8 pg WHO-TEQ (PCDD/F) per kg bw per day.** This level was within the range of tolerable daily intake recommended by WHO in 1998.

12. To estimate the dioxin exposure of high consumers, those above 95th percentile exposure level were studied. **The dioxin exposure of these high consumers could be up to 2.7 pg WHO-TEQ (PCDD/F) per kg bw per day.** This level was about 2.5 times that of average eaters.

13. The pattern of dietary exposure showed that seafood, meat as well as poultry and their products were the major dietary sources of dioxins. Dioxin concentration in milk was not high but the consumed amount made it an important source. On the other hand, dioxin concentrations in eggs were high but the consumption level was relatively low.

Implications and Limitations of the Findings

14. Comparing the results of this study with similar studies conducted overseas were difficult principally because different methodologies were adopted. From our study, it could be concluded that an average secondary school student would not experience major toxicological effects of dioxins.

15. Limitations of the study were identified. Firstly, only selected groups of food were chosen for the study and hence might not represent the full range of dioxin exposure. Secondly, dioxin concentrations expressed on fat basis were converted to product basis before the determination of dioxin exposure, based on the assumption that dioxins would be present in the fat portion only. Thirdly, number of samples for establishing the dioxin contamination data was small. Finally, the study was limited to dioxins and toxic effects of dioxin-like PCBs were not accounted for.

Control of Dioxin Exposure

16. Dietary intake is the major route of dioxin exposure. Since dioxins were products of environmental pollution, the ultimate goals of decreasing dioxin exposure are to reduce the dioxin emissions as well as interrupting their pathways into food and these rely on global effort in the international community.

17. International agreed regulatory standard and tolerable intake level for dioxins are still evolving. The Department will continue to monitor international development on regulations of dioxins and foods available in Hong Kong, especially on food items that may contain high levels of dioxins.

18. To minimize dietary exposure to dioxins, the public is advised to consume low-fat products, trim fat from meat and meat products and to use simple cooking methods. Moreover, a balanced diet is recommended to maintain health and to avoid excessive exposure to contaminants from a small range of food items.

Chapter 1

Introduction

1.1 "Dioxins" are a group of polychlorinated hydrocarbons that are a subset of polyhalogenated hydrocarbons. Effect of polyhalogenated hydrocarbons was first noted at the end of the nineteenth century. Some workers suffered from dermatitis due to the formation of polyhalogenated hydrocarbon contaminants during production of caustic potash by electrolysis of potassium chloride. During World War I, numerous incidents of chloracne that were associated with occupational exposure to polyhalogenated hydrocarbons, were reported because these chemicals were used in gas masks[1].

1.2 From the 1930s to the 1970s, polyhalogenated hydrocarbons were commonly manufactured and used as coolants and lubricants in electrical equipment in many industrialised countries. Consequently, concerns regarding their persistence, toxicity and inevitable

contamination of the environment and the food chain had been raised[1,2] and led to various scientific investigations followed by environmental monitoring and legislation among the industrialised countries. Total diet studies and other food surveillance programmes were initiated in the United States, Japan and some European countries in 1960s[1]. Studies on their biological effects, toxicity and carcinogenicity, as well as tolerable intakes have also been undertaken since then.

1.3 Some major dioxin-related incidents happened in 1960s to 1970s. During the Vietnam War from 1962 to 1971, researchers found that long-term exposure to Agent Orange, a defoliant containing dioxins used by the U.S. Air Force, would result in a higher chance of getting diabetes or other adverse health effects[1,3]. In 1976, a cloud of toxic chemicals, including dioxins was released into the air from a chemical factory in Seveso, Italy. Eventually an area of 15 square kilometers was contaminated and a total of 37,000 people were affected[1,4]. The affected population was found to have a higher risk of getting cancers[5].

1.4 Among the polyhalogenated hydrocarbons, dioxins and dioxin-like substances are the most studied chemical contaminants. Dioxins arise from either natural processes or industrial activities. Once produced, they tend to persist in the environment and concentrate in the food chain, especially in food of animal origin with high fat content. About 30 of these dioxin-related compounds are of toxicological concern, with 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD) being the most toxic and shown to be carcinogenic to human beings[4,6,7].

Local Situation and Development

1.5 The "Belgium Crisis" had aroused widespread concerns about dioxins in many places including Hong Kong Special Administrative Region (HKSAR) in 1999. In late May, poultry, eggs, pork, beef, milk and their products from Belgium was found to contain unusually high level of dioxins due to an earlier dioxin contamination of animal feed. Further investigations revealed that the affected animal feed had also been distributed to France, Germany and the Netherlands.

1.6 As a precautionary measure, the former Hygiene Division of the Department of Health of the HKSAR requested importers and retailers to temporarily withdraw the affected items from shelves. These affected items included poultry, eggs, pork, milk and milk products including milk formulae from the above four European countries. Dioxin concentrations in food samples are being monitored since 1999.

1.7 Because of the nature and public health implication of dioxins, the Food and Environmental Hygiene Department conducted a study of dietary exposure to dioxins in 2001.

Purpose

1.8 This study aims to assess dietary exposure to dioxins of secondary school students for the purpose of risk assessment and management.

Objectives

1.9 The objectives of this study are -

- to estimate the total dietary exposure to dioxins among secondary school students;
- to identify their major dietary sources of dioxins;and
- to assess the possible health impact of dioxin exposure.

Scope

1.10 This study focuses on the dietary exposure to dioxins in the HKSAR. Food items that are commonly consumed by local people and with potentially high dioxin concentration are selected as the studied items. As to the target food items, 17 dioxin congeners that are of toxicological concern as recommended by international authorities[5,7] are being analysed in our routine food surveillance programme. Making use of the data from the food surveillance programme of the Department and

the food consumption survey of secondary school students, dietary exposure to dioxins among secondary school students was estimated and then compared with tolerable intake values recommended by international authorities.

1.11 Secondary school students were chosen as the population of this study because they have relatively high-energy intake[8] and may be considered as a particularly at risk group. Moreover, they may be subject to chronic exposure to dioxins, probably up to many decades. 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.

Organization of this Report

1.12 In this report, we would first present a comprehensive account on dioxins (in Chapter 2) including their position in the food chain, their toxicological effects, as well as the international approaches in assessing

the cumulative toxic properties and the recommended tolerable intake of dioxins. We would then discuss the methodology and describe how the data were compiled for this assessment study in Chapters 3 and 4. The dioxin exposure estimation is presented in Chapter 5. In Chapter 6, we discuss the important findings and identify the limitations of the study. Finally, we summarise our findings in Chapter 7.

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[7] Codex Committee on Food Additives and Contaminants. Discussion Paper on Dioxins (CX/FAC 00/26). Codex Alimentarius Commission;December 1999.

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Chapter 2

Dioxins

2.1 "Dioxins" are a group of polychlorinated, planar aromatic compounds with similar structures, chemical and physical properties. According to the structure, dioxins can be grouped into polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs)[1] (Figures 2.1 and 2.2). There are 75 PCDD and 135 PCDF congeners.

Figure 2.1: Basic Structure of PCDDs*

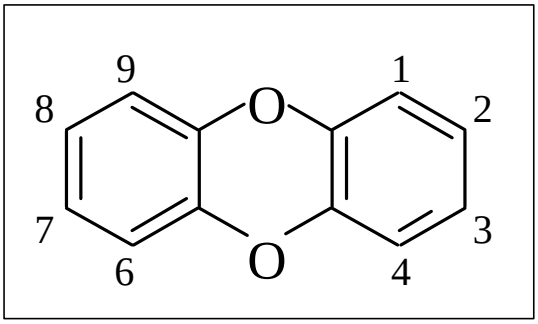
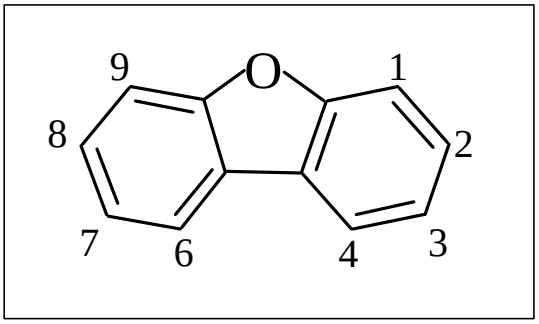


Figure 2.2: Basic Structure of PCDFs*



*These polychlorinated, planar aromatic compounds may have up to eight chlorine atoms attached to any carbon atoms at position 1, 2, 3, 4, 6, 7, 8, and 9.

2.2 Dioxins are colourless, odourless organic compounds[2]. They are sparingly water-soluble but highly lipophilic. In addition, they are

persistent in the environment and biological samples[3]. Incineration at temperature over 850^{o}C is the best available method to destroy dioxins though other methods under study are being developed[4].

Sources and Exposure

2.3 Dioxins have no commercial applications. They are formed mainly as by-products of industrial processes. Examples are combustion processes such as commercial or municipal waste incineration, manufacturing processes including bleaching of paper pulp using chlorine, and manufacture or use of defoliants, pesticides, steel, paint and some other chemicals. Other sources of dioxins in the environment include evaporation from chlorophenol wood preservatives as well as emission by smelting industries and traffic. Dioxins can also be formed naturally during volcanic eruptions and forest fires[1,2,5].

2.4 Most of the dioxins enter the environment by emission to air, then deposit on water, soil or plants near or far away from the source[5]. Some soils, sediments and animals may have higher level of dioxins

while water and air have a lower level[4]. Besides aerial transportation, soil and water may also be polluted by contaminated sewage sludge or composts, herbicide runoff and erosion from nearby contaminated areas[2,3].

2.5 Dioxins deposited on plants or soil may be degraded by photolysis in the presence of ultraviolet light. Hence dioxins that are on the soil surface have shorter half-lives (one to three years) than those deeper in the soil (10 to 12 years)[3]. However, relatively little is known about the fate of dioxins released into the environment, i.e. transport, distribution and transformation[1]. Since dioxins are extremely resistant to chemical and biological degradation, dioxins persist in the environment and accumulate in the food chain[1,2].

2.6 Dioxins are ubiquitous in the environment throughout the industrialised world. Human beings are exposed to dioxins through occupational exposure, accidental exposure or environmental exposure. Occupational activities with the production of unintentional amount of

dioxins, such as incineration and manufacture of pesticides, may result in a significant human exposure. Otherwise, occupational and accidental exposures have a relatively small contribution to the overall human exposure. For environmental exposure, small amounts of dioxin intakes may result from breathing in air containing trace amounts of dioxins on particles and in vapour form, inadvertent ingestion of soil containing dioxins and dermal absorption[6]. Nevertheless, international organisations estimated that over 90% of human exposure to dioxins is through dietary intake[1,2,6,7]. Bioavailability of dioxins from food containing fat is about 75% or higher[1].

2.7 Children show a relatively higher dietary exposure to dioxins, due to their relatively low body weight. Dioxins have also been shown to affect child growth and development[1,6,8,9], thus rendering children more vulnerable to the effects of dioxins. It has been shown that individuals who consume high-fat diets, or live near sites with relatively higher level of dioxins such as incineration plants, pulp plants and paper plants, are at greater risk from dioxins[4].

Dioxins and the Food Chain

2.8 Contamination of leafy vegetables and pastures is mainly due to atmospheric deposition of dioxins on the leaves. In addition, application of pesticides and spreading of contaminated sewage sludge may also pollute plants. Dioxins are then accumulated in livestock that ingests the contaminated plants and soil[3].

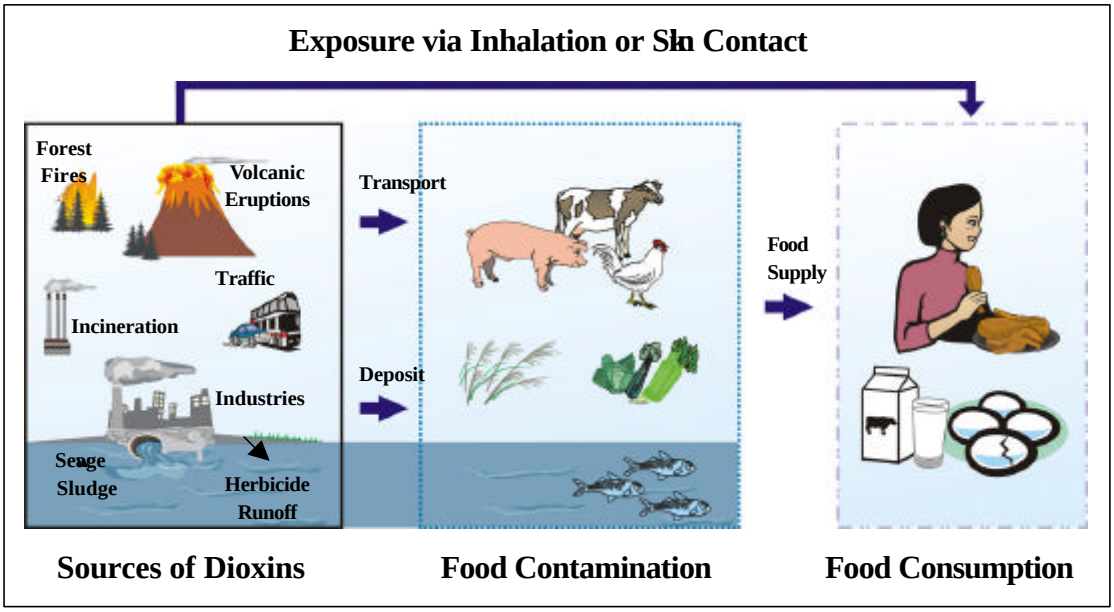
2.9 Sediments of surface waters are thought to be the ultimate sink of dioxins. Dioxins enter fish and other aquatic organisms through ingestion of sediments. The persistence of dioxins results in bioaccumulation in aquatic organisms[3].

2.10 Since dioxins are persistent and concentrate in the food chain, animals have higher dioxin concentration than plants, water, soil or sediments. In animals, dioxins tend to accumulate in fat. Hence dairy products, eggs, meat, poultry, fish and their products contribute most to

the dietary exposure to dioxins among the general population in industrialised countries. Moreover, animals with a longer life span may have a higher dioxin concentration in its fat tissue[2,3,4].

2.11 The flow of dioxins from their sources to human beings is described in Figure 2.3 –

Figure 2.3 Exposure to Dioxins



Toxicity

2.12 In animal studies, the oral LD_{50} (lethal dose for 50% animals

under study) in guinea pigs was 0.6 μg/kg body weight (bw) while that in hamsters was greater than 5000 μg/kg bw[9]. For human, a minimum toxic dose of 0.1 μg/kg has been reported[10]. However, the latest evaluation by Joint Food and Agriculture Organisation / World Health Organisation Expert Committee on Food Additives (JECFA) in 2001 concluded that an acute reference dose would not be appropriate for dioxins because of their long half-lives[9].

Human Effects

2.13 Chloracne and related dermal lesions such as skin rashes, skin discoloration and excessive body hair are frequently noted signs when people are exposed to large amounts of dioxins[4,6,7,11]. Short-term exposure may also result in altered liver functions[4]. Short-term exposure to TCDD, the most toxic congener, may lead to headache, fatigue, irritation of the gastrointestinal and respiratory tracts, dehydration and skin irritation[12].

2.14 Chronic exposure to dioxins may lead to diabetes and ischaemic heart disease, disruption of the thyroid and immunological functions as well as abnormal development of the nervous system and male reproductive system in foetus[4,12]. Animal studies have shown reduced sperm count in the offspring of rats that have been exposed to dioxins[13].

2.15 There is sufficient evidence that TCDD is carcinogenic to humans and experimental animals, and hence the International Agency for Research on Cancer (IARC) classified TCDD as Group 1 carcinogen in 1997. In addition, IARC has also evaluated dibenzo-para-dioxin and five other PCDDs (1,2,3,7,8-pentaCDD, 1,2,3,6,7,8-hexaCDD, 1,2,3,7,8,9-hexaCDD, 1,2,3,4,6,7,8-heptaCDD and 2,7-diCDD) as well as ten PCDFs. They considered that there is inadequate evidence to suggest that these chemicals are carcinogenic to humans, though there may be limited evidence of carcinogenicity in experimental animals[14].

2.16 TCDD is the one with the strongest epidemiological evidence

suggesting increased risks for all cancers combined[1,9,14]. Animal studies also showed that TCDD induced multiple-site tumours in multiple animal species in both sexes. Nevertheless, TCDD was shown to be negative in several short-term assays for genotoxicity. In a long-term study of carcinogenicity of TCDD in rats, the lowest observed effect level for hepatic adenomas in female rats was 10 ng/kg bw per day. Using the body burden approach (see para. 2.27 to 2.29), this is equivalent to a daily intake of 150 pg/kg bw in human beings[9].

Toxic Equivalency Factors (TEF) Scheme

2.17 To estimate the aggregate risks associated with exposure to dioxins is not easy since complex mixtures of different dioxin congeners are usually present in trace amounts in environmental and biological samples. Moreover, different congeners have different toxicity levels. As a result, schemes on the toxic equivalency of different dioxin congeners have been developed to facilitate toxicity assessment and regulatory control of dioxins. Recognising the need for an

internationally harmonized approach, the World Health Organisation (WHO) derived internationally agreed TEFs for PCDDs, PCDFs, and dioxin-like polychlorinated biphenyls (PCBs) in 1997[3, 15 , 16] . This WHO-TEF scheme has been commonly adopted by most international organisations and food control authorities.

2.18 The TEF concept relates the toxicity of an individual dioxin congener to the toxicity of TCDD, the most potent and most studied dioxin congener, and therefore transforms analytical results to toxicological information. Well supported by many studies, the TEF concept assumes additivity of toxic effects among dioxin congeners in a mixture[1,15,16].

2.19 In the WHO-TEF scheme, total dioxin concentration in an environmental or food sample is referred to as the toxic equivalent (TEQ) concentrations. It is obtained by summing the contribution from each congener, which was calculated by multiplying the concentration of each congener with the corresponding TEF using the following equation[6,15,16] –

$$TEQ = \sum (PCDD_i \times TEF_i) + \sum (PCDF_i \times TEF_i) + \sum (PCB_i \times TEF_i)$$

2.20 Total dioxin concentration in a sample or TEQs is often expressed as picogram (pg) WHO-TEQ per gram fat or nanogram (ng) WHO-TEQ per kilogram product. Under this WHO-TEF scheme, TEF is assigned to 17 congeners of dioxins and 12 dioxin-like PCBs, with reference to TCDD being the most toxic congener with a designated TEF of 1.0. TEFs for dioxins and dioxin-like PCBs are listed as follows[14,15] –

Table 21 : WHO -TEF (2005) Scheme for Dioxins and Dioxin-like PCBs

Group	Congener	TEF value
PCDDs	2,3,7,8-TetraCDD	1
	1,2,3,7,8-PentaCDD	1
	1,2,3,4,7,8-HexaCDD	0.1
	1,2,3,6,7,8-HexaCDD	0.1
	1,2,3,7,8,9-HexaCDD	0.1
	1,2,3,4,6,7,8-HeptaCDD	0.01
	OctaCDD	0.0001
PCDFs	2,3,7,8-TetraCDF	0.1
	1,2,3,7,8-PentaCDF	0.05
	2,3,4,7,8-PentaCDF	0.5
	1,2,3,4,7,8-HexaCDF	0.1
	1,2,3,6,7,8-HexaCDF	0.1
	1,2,3,7,8,9-HexaCDF	0.1
	2,3,4,6,7,8-HexaCDF	0.1
	1,2,3,4,6,7,8-HeptaCDF	0.01
	1,2,3,4,7,8,9-HeptaCDF	0.01
	OctaCDF	0.0001

Group	Congener	TEF value
Non-ortho PCBs	PCB 77	0.0001
	PCB 81	0.0001
	PCB 126	0.1
	PCB 169	0.01
Mono-ortho PCBs	PCB 105	0.0001
	PCB 114	0.0005
	PCB 118	0.0001
	PCB 123	0.0001
	PCB 156	0.0005
	PCB 157	0.0005
	PCB 167	0.00001
	PCB 189	0.0001

Polychlorinated Biphenyls (PCBs)

2.21 Twelve dioxin-like PCBs that are of similar toxicological properties as dioxins are included in the WHO-TEF scheme. PCBs are chlorinated aromatic hydrocarbons and consist of 209 congeners. Though most PCB congeners are non-planar, some may adopt a planar "dioxin-like" chemical structure and have toxicological properties that are similar to dioxins. These are often termed as "dioxin-like PCBs". Like dioxins, PCBs are also lipophilic and persistent. They tend to accumulate in the food chain[1].

2.22 Starting from late 1920s, PCBs had been commonly used in a

number of industrial and commercial open and closed systems such as pigments, dyes, repellents and plasticizers, as well as transformers, capacitors, electric insulators and hydraulic fluids. Because of the environmental and health implications of PCBs, Organisation of Economic Cooperation and Development banned the use of PCBs in open systems in early 1970s and in new equipment in early 1980s. However, large amounts of PCBs are still present in electrical equipment, plastic products and the environment. Nevertheless, decrease in level of PCBs in the environment has been reported in many countries since implementation of the ban of PCBs[1,17].

2.23 People who were exposed to large amounts of PCBs may result in ocular effect and dermal effect such as acne and rashes. Some people may have liver damage eventually[17]. IARC has determined that PCBs are probably carcinogenic to humans and classified PCBs as group 2A carcinogen. This means that there is limited evidence of carcinogenicity in humans, though there is sufficient evidence of carcinogenicity in experimental animals[18].

2.24 It is recognised that some dioxin-like and non-dioxin-like PCBs may not bind to aryl hydrocarbon (Ah) receptor, which is an intracellular receptor protein for dioxins, to mediate actions such as liver enlargement and tumour promotion. The WHO meeting in 1997 concluded that TEF values would not be established for these compounds based on insufficient environmental and toxicological data[9,15].

Tolerable Intake

2.25 Tolerable intake describes permissible human exposure to chemical contaminants with cumulative properties over a certain period of time without causing any adverse effects. Most of these contaminants are unavoidably associated with the consumption of otherwise wholesome and nutritious foods[19]. Dioxins are one of these contaminants with such properties.

2.26 Tolerable intake could be expressed in daily, weekly or

monthly basis, e.g. Tolerable Daily Intake (TDI) and so on. The derivation of tolerable intake is based on the toxicological, epidemiological and pharmacokinetic data derived from animal studies. The most sensitive indicator of toxicity, such as the lowest-observed-adverse-effect level (LOAEL) in the most susceptible species of experimental animals, would be used to derive the TDI[16,20].

2.27 To account for differences in the sensitivities between animals and humans, as well as the susceptibilities within the human population, a safety factor, say 10, is applied to extrapolate the human TDI from the animal LOAEL[16,20].

2.28 For dioxins, the WHO established a TDI of 10 pg/kg bw for TCDD[16] based on animal studies on steady state liver TCDD concentration in December 1990[6,16].

2.29 Since dioxins have relatively long half-lives in biological systems, the “body burden” approach was found to provide a better

estimate in assessing the continuous exposure to dioxins than daily intake[2,6]. In humans, the half-lives of PCDDs and PCDFs range from several months to over 20 years. Because of their persistent and accumulative nature, toxicity of dioxins is related to the amount accumulated in the body during lifetime, i.e. the body burden. Moreover, the half-lives of dioxins in the body are related to amount of body fat, not the daily dose[9].

2.30 Since the concentration of dioxins at the target tissue is seldom known, the WHO estimated the body burdens of TCDD in human by transforming the animal body burdens using simple pharmacokinetic calculation[6,9,16] –

$$\text{Body Burden (ng/kg bw)} = \frac{f}{} \times \text{Intake(ng/kg bw/day)} \times \text{Half-life(day)} / \ln(2)$$

2.31 In the above formula, “f” denotes the fraction of absorbed dose (assumed to be 50% for absorption from food for humans) and the half-life for TCDD was estimated to be 7.5 years (2740 days)[6,9,16].

Consequently, the WHO in 1998 decided to adopt body burdens as the measures of dose rather than daily doses in the interpretation of toxicological data. In light of new scientific evidences, the TDI was modified to 1 - 4 TEQ pg/kg bw for dioxins and dioxin-like compounds[16].

2.32 In 2001, JECFA concluded that tolerable intakes for PCDDs, PCDFs and dioxin-like PCBs should be expressed as a monthly value because of their long half-lives and therefore over-month studies would be more appropriate to assess their long- or short-term risks. Eventually, JECFA established a Provisional Tolerable Monthly Intake (PTMI) of 70 pg/kg bw per month for PCDDs, PCDFs and dioxin-like PCBs based on two studies on PTMI[9].

2.33 Tolerable intakes that have been established by international authorities are summarized as follows –

Table 22 : Tolerable Intakes for Dioxins and Dioxin-like Compounds

Organisation	Tolerable Intake	Coverage
WHO, 1990	10 pg/kg bw per day	TCDD
WHO, 1998	1 - 4 pg/kg bw per day	Dioxins and dioxin-like compounds
JECFA, 2001	70 pg/kg bw per month	Dioxins and dioxin-like compounds

2.34 Among the above recommended tolerable intakes established in or after 1998, there is not much difference on average even though they are expressed on different time bases. In this study, tolerable intakes established by the WHO in 1998 will be used to compare with our estimated dietary exposure to dioxins.

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Chapter 3

Dietary Exposure Assessment

3.1 Environmental substances in food that may cause adverse health effects are food hazards. The chance of occurrence of an adverse effect and the magnitude of that effect on the population is defined as risk[1]. The environmental substance may not pose risk to a population when the people are not exposed to the substance. Hence exposure assessment is essential in assessing whether the population is at risk by evaluating the degree of contact with the substance[2]. Exposure assessment refers to both the qualitative or quantitative evaluation of magnitude, frequency, duration and the route of contact of an environmental substance[2,3].

3.2 Generally speaking, exposure is defined as the contact with a chemical, physical or biological agent[4]. Human beings are exposed to environmental substances by inhalation, ingestion and/or dermal

absorption from air, water, food and soil.

3.3 This study focuses on the dietary exposure to dioxins.

Dietary Exposure Assessment

3.4 Information on the consumption of relevant foods and the concentrations of the environmental substance or chemical in those foods are necessary for assessing dietary exposure of a chemical contaminant. The dietary exposure of a contaminant in a food is obtained by multiplying the contaminant concentration in the food by the amount of that food consumed. The total dietary exposure of the contaminant is estimated by summation of the exposures to all foods containing the contaminant. This can be expressed in the general formula below[5] –

$$\text{Dietary Exposure} = \frac{\text{Food Chemical Concentration} \times \text{Food Consumption}}{\text{Body Weight}}$$

3.5 To estimate the dietary exposure to a chemical contaminant in a population, the average body weight of the population will be used. Dietary exposure often expresses as mg/kg bw over one-day, one-week or one-month period.

3.6 To assess whether the consumer or population is at risk, dietary exposure to a chemical will be compared with relevant reference levels[5,6,7] such as TDI or tolerable monthly intake (TMI) recommended by international organisations such as WHO and JECFA.

Establishing Food Consumption Estimate

3.7 Food consumption estimate is used to describe dietary patterns of individuals or populations. With food consumption data, dietary exposure to food contaminants can be assessed. In general, food consumption pattern can be established by data collected in three different ways, (a) national/regional data of food availability, (b) data collected at household level or (c) data on individual food consumption.

3.8 National/regional food supply data can be calculated from the food balance sheets. A food balance sheet presents an overview of food supply of a region over a certain period of time. Data regarding production, stocks, trade (imports and exports), domestic utilization and population are needed for the compilation of food balance sheets. Food supply data from food balance sheets is expressed in kilograms per person per year[8].

3.9 Household food survey can be conducted to collect data for establishing food consumption pattern. There are two types of such surveys, household budget survey and household food consumption survey. The household budget survey records food purchases in terms of amounts and/or expenditure. The household food consumption survey records the movement of foods in and out the household. By measuring food purchases and food stock changes, food supply information at the household level can be obtained. Per person food consumption data can be derived from dividing the total amount of

household food supply by the number of persons in the household[9].

3.10 Food consumption survey targeting at individual food intake is considered most useful to establishing the pattern of actual food consumption. There are three commonly used methods for collecting information on individual food data, namely (a) 24-hour dietary recall, (b) food frequency questionnaire and (c) food diary/records.

3.11 Twenty-four hour recalls collect food consumption data by recalling intakes during a 24-hour period, usually over the past 24 hours. By asking probing questions, a skilled interviewer will assist respondents to describe the food preparation methods, types, amounts, time and location of food consumed. This method can provide comprehensive description on food consumption pattern. However, probing skills and variability among interviewers may affect the data collection[8,9,10,11].

3.12 Food frequency questionnaire is considered to be the most simple and straightforward method to collect food consumption data and

is most frequently used. Food frequency questionnaire is a structured questionnaire with pre-selected food items. Respondents are asked to recall how frequent they consume the selected foods. If pre-determined portion sizes of foods are given, the respondents can estimate the amount of foods consumed as well. However, information regarding pre-selected foods can only be obtained; therefore, it is very important that the selected food items are related to the purpose of the study[8,9,10,11].

3.13 Food record/diary is a self-administered data collection instrument. Respondents are requested to record the types and amounts of food consumed. The quantity of food intake can be measured by weighing or estimating, preferably with food measurement aids. This method usually collects food intake data for 1-7 days, depending on the purpose of the survey and resources available[8,9,10,11].

3.14 For the purpose of assessing dietary exposure, individual food consumption data are most useful to reflect the actual consumption pattern. Data collected at national and household levels can only reflect

food supply information.

Estimating Dietary Exposure to a Contaminant

3.15 There are generally three approaches of estimating dietary exposure to a chemical contaminant as recommended by WHO[6]: (a) total diet studies; (b) duplicate portion studies; and (c) selective studies of individual foods.

Total Diet Studies

3.16 Total diet studies are also known as market basket survey. Among the three approaches for assessing dietary exposure, total diet studies are considered to be the most accurate approach in estimating the actual exposure to contaminants. The effect of food preparation would have been incorporated in the testing procedure. Many countries have either been conducting or become interested in adopting this approach for dietary exposure assessment[12].

3.17 One key element of the total diet studies is to establish the food consumption pattern which is used for the design of the "standard" diet for the population concerned. Food samples representing this "standard" diet is obtained for study. The samples are then prepared for table-ready consumption and analysed individually or in combination with others of similar food groups. Chemical levels measured in the food samples are used in calculating the average daily exposure for each composite and for the whole diet[6].

3.18 Total diet studies are particularly useful in determining whether the chemicals of concern are widely distributed amongst all major foods, or are confined to a few classes of foods[6].

3.19 There are additional advantages of total diet studies. They can be used for monitoring the effectiveness of the existing regulatory systems. Moreover, they convey easily understandable information on the dietary exposure to all stakeholders. In addition, they can help

identify the major dietary sources of chemical contaminants in terms of either individual foods or food group composites depending on the approach of obtaining the data[6,7]. Some countries also utilize total diet studies to monitor the intake of specific nutrients in the population.

3.20 However, the estimated level of dietary exposure may be lower than the actual scenario when the food sample is analysed based on food group composites because of the "dilution effect". The dietary contribution of a contaminant in a food that is consumed in small amount may be diluted by other food items of the same food group to a level below the detection limit. In addition, it requires considerable resources and sophisticated set-up for the laboratory testings[6].

3.21 Countries that have conducted total diet studies include Australia, Canada, China, Czech Republic, Finland, France, Japan, New Zealand, Spain, the United Kingdom and the United States. In case of dietary exposure assessment for dioxins, countries including Canada, Japan, Spain, and the United Kingdom have adopted this approach[12].

Duplicate Portion Studies

3.22 Duplicate portion studies are variants of total diet studies. They involve collection of “representative diets” of individuals taken over a period of time by obtaining a duplicated set of samples of the meals consumed. In this way, the average food consumption pattern on a population basis will not be necessary. The duplicated meals in their ready-for-table consumption state are taken for laboratory analysis for contaminants[6].

3.23 Advantages of this approach are that the food actually consumed is being analysed and food consumption data are not necessary[6].

3.24 The disadvantages are that the data obtained may only cover a restricted population due to limitation in resources and the data may not

be representative of long-term average food consumption[6].

3.25 The United Kingdom adopted this approach for dietary exposure assessment for pre-school children in 1984[7].

Selective Studies of Individual Foods

3.26 This is a more flexible approach in studying dietary exposure. Contaminant concentrations in representative samples of various food items, especially staple foods, are measured. The samples could either be raw, or as consumed. Together with food consumption data, the average daily exposure to the contaminant could be estimated[6].

3.27 This approach is particularly suitable for contaminants which are predominantly contributed by one or two commodities and/or when food contamination monitoring programme has established average contaminant concentrations in the commodities[6].

3.28 The major advantages of this approach are that (1) it provides flexibility of estimating the dietary exposure to contaminants in a whole population or individual groups;(2) it can estimate the dietary exposure more accurately when a food composite approach may dilute the contaminant concentration to below the quantitation limit of the method; (3) food monitoring data on individual foods may be used directly;and (4) data available from other sources may be used to supplement incomplete data on food consumption or residue limits[6].

3.29 The disadvantage is that this approach has greater initial demands on testing resources when compared with the total diet studies. The effect of cooking on the contaminant concentration will also be ignored if the data are based on raw samples[6].

3.30 The United Kingdom had estimated average daily exposure to lead in selected food groups using food consumption data in 1970s[6].

Approach Undertaken by this Dioxin Study

3.31 In this study of dietary exposure to dioxins, the “selective studies of individual foods” approach is adopted because of their flexibility in assessing dietary exposure in population sub-groups, while food consumption data for the design of total diet study are not yet available. This approach is feasible also because dioxins are predominantly present in a few food groups, which have been tested by this Department under the food surveillance programme.

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Chapter 4

Data Compilation for the Study

4.1 This study consisted of two main steps. Firstly, data on food consumption pattern and dioxin concentration in selected food items were extracted from the Food Consumption Survey conducted in 2000 and the regular food surveillance programme of this Department respectively. Secondly, these data were compiled according to the WHO proposed methodologies with which the estimates for dioxin exposure were computed.

4.2 In this Chapter, we would focus on data compilation pertaining to the food consumption pattern and the dioxin concentration data in foods. Some crucial methodological issues would also be discussed. These data were used to estimate the dioxin exposure according to the formula as presented in Chapter 3.

Food Consumption Pattern of Secondary School Students

4.3 Food consumption pattern is vital for estimating the contaminant exposure level from food of the local population which in turn provides a clearer picture of the risks involved. Food consumption pattern of the secondary school students was obtained from a food consumption survey that was conducted by this Department in 2000.

4.4 The survey covered Form 1 to Form 5 secondary day school students with the exclusion of students from International/English Foundation schools. A total of 472 secondary schools with more than 380,000 students were covered by the sampling plan. A stratified three-stage sampling design was used. Consequently, 967 students of 27 schools participated in the survey, with a response rate of 77% at the school level and 96% at the student level. The mean weight of students participated in the survey was 52.0 kg[1].

4.5 Consumption data on individual food items were obtained using a self-administered food frequency questionnaire. Photographs of food items were provided to facilitate the participants in reporting the usual amount of food intake[1].

4.6 Food items covered by this survey fell into 13 categories. We have chosen food items for the exposure assessment in which dioxins are more likely to be found, principally foods of animal origins with high fat content. Five groups have been identified. They are: (1) meat and meat products, (2) poultry and poultry products, (3) milk and milk products, (4) eggs and egg products and (5) seafood. Offal was included in the group "meat and meat product" and sashimi in the group "seafood".

4.7 The food group consumption pattern was obtained using data from subjects who had responded to all food frequency questions for that particular food group. This provides a better average estimate for each food group. The food group consumption pattern of the average eaters

is given in Table 4.1.

Table 4.1 The List of Food Groups Selected and Modified for Dietary Exposure Assessment of Dioxins and their Consumption Patterns

Food Groups	Mean Consumption (g/day)
Meat and Meat Products	111.3
Poultry and Poultry Products	88.2
Milk and Milk Products	158.2
Seafood	133.7
Eggs and Egg Products	15.5
Total	**506**

Food Dioxin Concentration

4.8 Data on dioxin concentration in foods available on the local market was extracted from the food surveillance programme of this Department where food samples were collected from the local market and sent for microbiological and chemical analysis including the testing for dioxins.

4.9 At the Government Laboratory, samples underwent a fat extraction process followed by gas chromatography-high resolution mass spectrometry (GC-HRMS) for analysis of dioxin concentrations. For fat

extraction, organic solvents were used and the appropriate Association of Official Agricultural Chemists' (AOAC) methods were employed. Due to the difference in the fat contents of the samples, there was slight adjustment in the sample preparation procedure.

4.10 The samples were first homogenized and freeze-dried before being spiked with isotopically labelled 2,3,7,8-PCDDs/PCDFs. After the enrichment and purification process, the extract was analysed by GC-HRMS as described in the method 1617 of the United States Environmental Protection Agency (U.S. EPA) for dioxin analysis. Separated PCDDs or PCDFs were detected using mass spectrometer set at 10,000 mass resolution to detect the exact masses of the analytes in multiple ion detection mode. Formal quality assurance programme which involved regular analysis of blanks, duplicates, spiked samples and certified reference materials was used to monitor the ongoing performance.

4.11 Since the action level for our current dioxin surveillance

programme is 1 pg WHO-TEQ (PCDD/F) /g sample, the testing method adopted was aimed at detecting TEQ concentration at such level.

4.12 We have examined the available dioxin data from January 2000 through April 2001. Results of 105 food items were reported from the Government Laboratory during this period. After matching the data with our selected food groups, 88 fell into our selected food groups (see Table 4.2) while the remaining belonged to other food groups.

Table 4.2 The List of Food Groups for Dioxin Assessment

Food Groups	Number of Food Items
Meat and Meat Products	13
Poultry and Poultry Products	26
Milk and Milk Products	10
Seafood	28
Eggs and Egg Products	11

(total 88 items)

4.13 Information on the concentration of each of 17 dioxin congeners and the fat content of the food sample were given in the test reports. Typically, for samples with high fat contents, the test results will be reported on fat basis. For other samples, the results will be reported on whole sample basis (i.e. product basis).

4.14 For each food sample, the TEQ concentration was obtained by summing the contribution from each congener, which was calculated by multiplying the concentration of each congener with the corresponding TEF (see para. 2.19).

4.15 When calculating TEQ concentration, conversion may be required for results derived from fat based measurement. The formula for converting fat-based results into product-based results is as follows –

$$\text{Product-based Dioxin Concentration} = \text{Fat-based Dioxin Concentration} \times \% \text{ Fat Content}$$

Treatment of Non-Detected Results

4.16 Since not all dioxin congeners are present in a concentration that can be detected, problems with the interpretation of analytical results may arise. This is particularly important when a significant portion of the test results has a chemical concentration below the limit of detection

(LOD). When the analytical value was below LOD, the true value would be anywhere between zero and the LOD.

4.17 A number of approaches have been used in dealing with non-detectable results. The most commonly encountered technique involves substitution of a single value as a proxy for each non-detectable data value, which include 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 quantified data above the LOD to define the distribution function of the set of data, and transforming and extrapolating the quantified data[2].

4.18 In this study, 74% of the test results of individual congeners were below LOD (Table 4.3). It may not be appropriate to assume a zero concentration for the samples with test results below LOD since dioxins are ubiquitous in the environment. On the other hand, assigning the non-detects to a value of LOD would, however, grossly overestimate

the dietary exposure particularly when the LODs are high. Also because quantified data for each dioxin congener in different food groups were limited, the more sophisticated methods for non-detects were inapplicable. Thus, a value of 1/2 LOD was assigned to all results below LOD, which would better reflect the true values of these samples.

Table 4.3: Percentages of Results that are Below LOD

Food Groups	No. of Samples	No. of Test Results	No. of Results Below LOD	Percentage of Results Below LOD
Meat and Meat Products	13	221	187	85%
Poultry and Poultry Products	26	442	328	74%
Milk and Milk Products	10	170	105	62%
Seafood	28	476	387	81%
Egg and Egg Products	11	187	104	56%
All Samples	**88**	**1496**	**1111**	**74 %**

4.19 The LODs for the 17 dioxin congeners being tested in our food surveillance programme are listed in Table 4.4.

Table 4.4: The Limits of Detection (LODs) for the 17 Dioxin Congeners

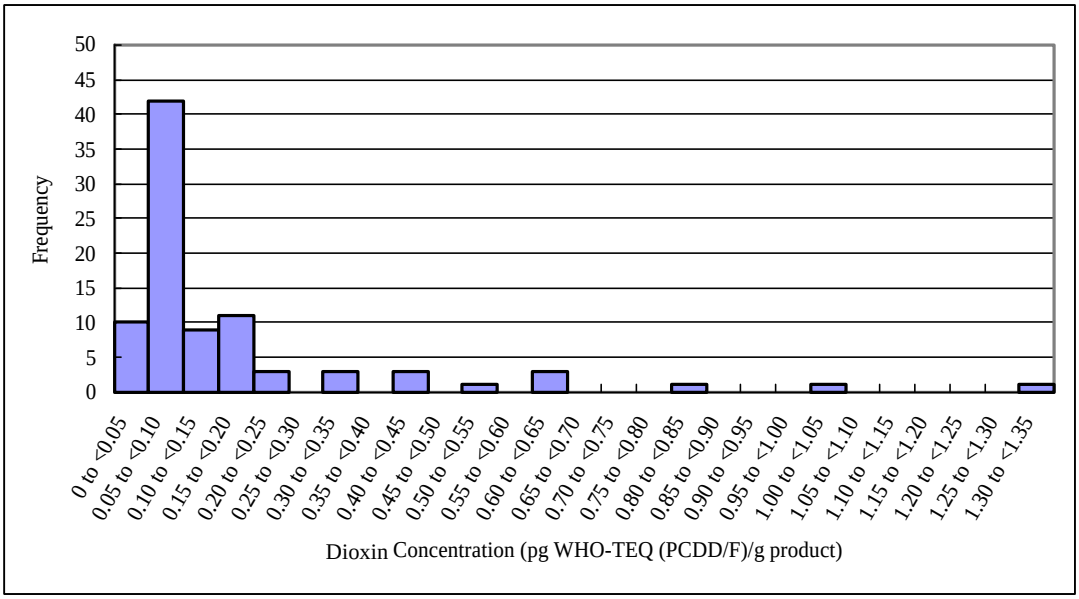
Group	Congener	Limit of Detection (Fat Basis) (pg/g)	Limit of Detection (Product Basis) (pg/g)
PCDDs	2,3,7,8-TetraCDD	0.2	0.04
	1,2,3,7,8-PentaCDD	0.2	0.04
	1,2,3,4,7,8-HexaCDD	0.5	0.1
	1,2,3,6,7,8-HexaCDD	0.5	0.1
	1,2,3,7,8,9-HexaCDD	0.5	0.1
	1,2,3,4,6,7,8,9-HeptaCDD	0.5	0.1
	OctaCDD	1	0.2
PCDFs	2,3,7,8-TetraCDF	0.2	0.04
	1,2,3,7,8-TetraCDF	0.2	0.04
	2,3,4,7,8-PentaCDF	0.2	0.04
	1,2,3,4,7,8-HexaCDF	0.5	0.1
	1,2,3,6,7,8-HexaCDF	0.5	0.1
	1,2,3,7,8,9-HexaCDF	0.5	0.1
	2,3,4,6,7,8-HexaCDF	0.5	0.1
	1,2,3,4,6,7,8-HeptaCDF	0.5	0.1
	1,2,3,4,7,8,9-HeptaCDF	0.5	0.1
	OctaCDF	1	0.2

Dioxin Concentration for a Food Group

4.20 Dioxin concentration for a food group can be represented by median or mean values. If the results have a normal distribution, the median and the mean values would approximate to each other. However, for contaminant data, the distribution is often skewed (Figure 4.1). In these situations, the use of median value would be less affected by outliers.

4.21 In interpreting contaminant data, WHO[3], Australia New Zealand Food Authority[4] and JECFA[5] shared similar views and chose to use the median value, whereas some countries in the European Union including the UK had used mean values in their assessments. A dioxin study[6] commissioned by the UK Government suggested that mean was preferred for individual consumer exposure assessment while median was preferred for whole population assessment.

Figure 4.1 Freqency Distribution of Dioxin Concentrations in All Food Samples



4.22 The frequency distribution for dioxin concentration (Figure 4.1) was skewed to the right. Dioxin concentrations in the 88 food

samples ranged from 0.01 to 1.32 pg WHO (PCDD/F)/g product basis. This illustrates that for dioxin concentrations the adoption of median value for assessment would be less likely to be affected by the skewed distribution.

4.23 The data was further broken down according to food groups and the frequency distribution with respect to each food group all showed a skewed distribution (Figures 4.2 – 4.6).

Figure 4.2: Freqency Distribution of Dioxin Concentrations in Meat and Meat Products

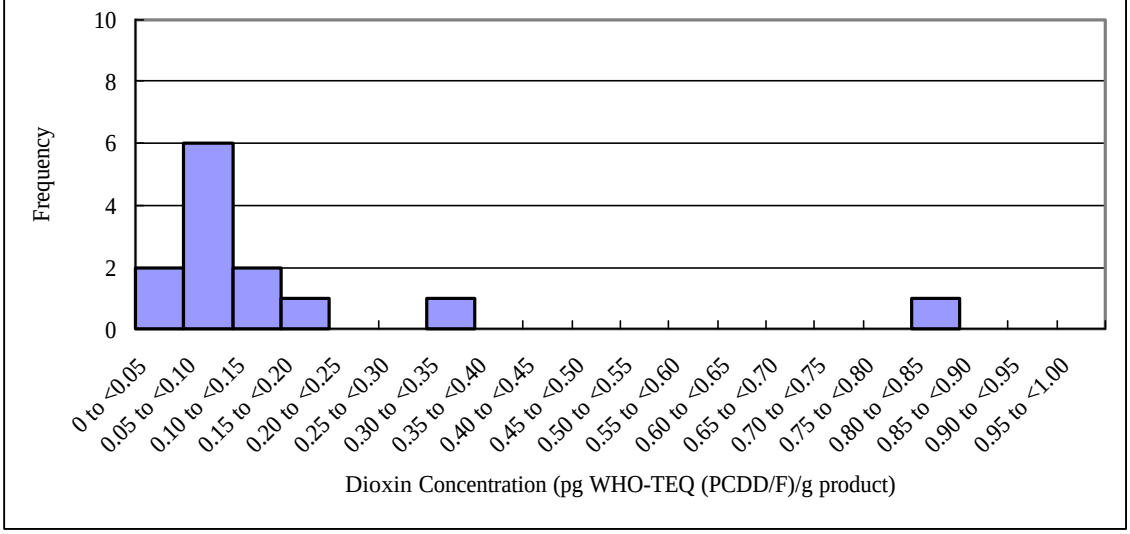


Figure 4 3:Freqency Distribution of Dioxin Concentrations in Poultry and Poultry Products

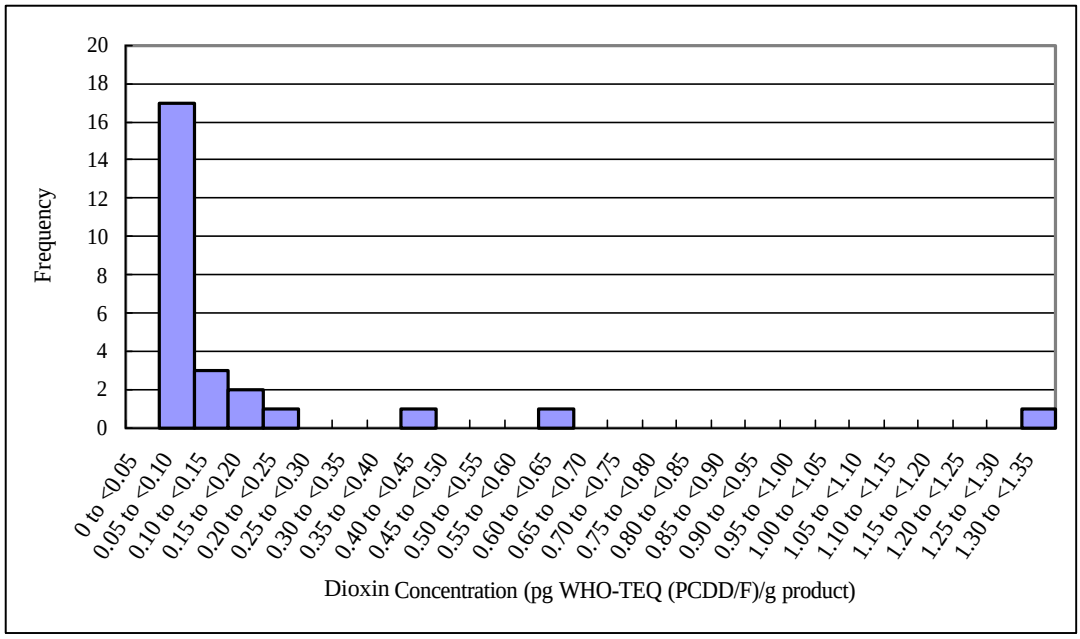


Figure 4 4:Freqency Distribution of Dioxin Concentrations in Milkand Milk Products

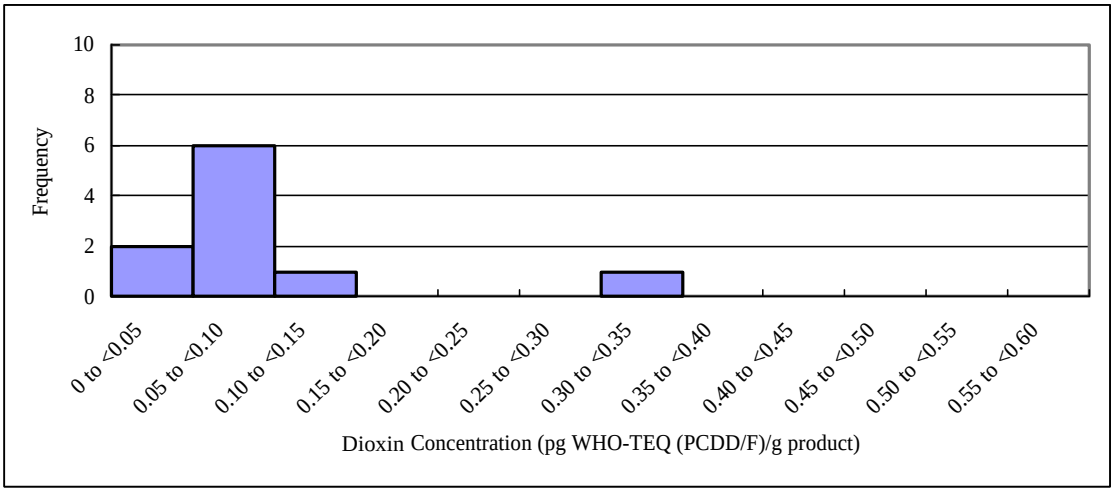


Figure 4 5:Freqency Distribution of Dioxin Concentrations in Seafood

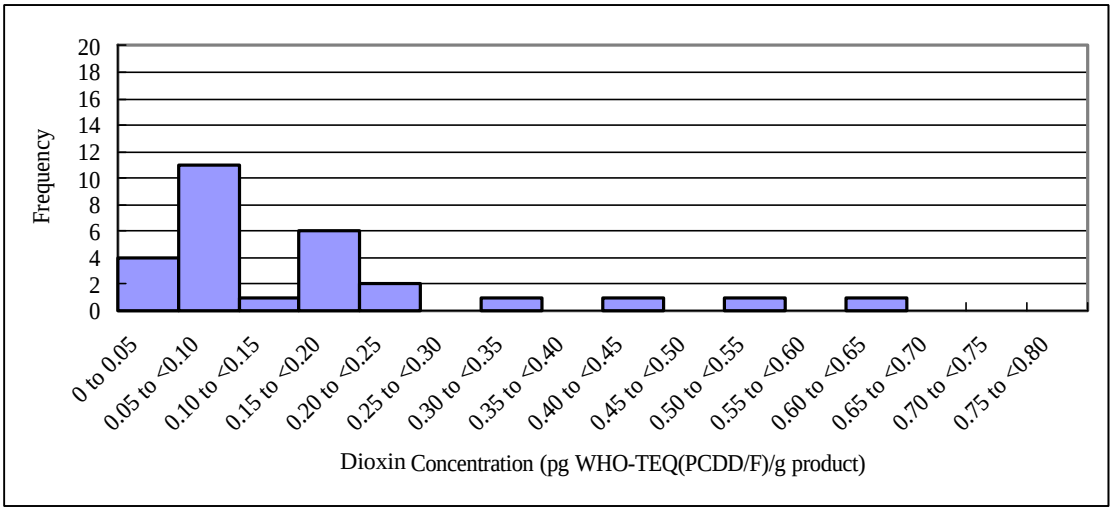
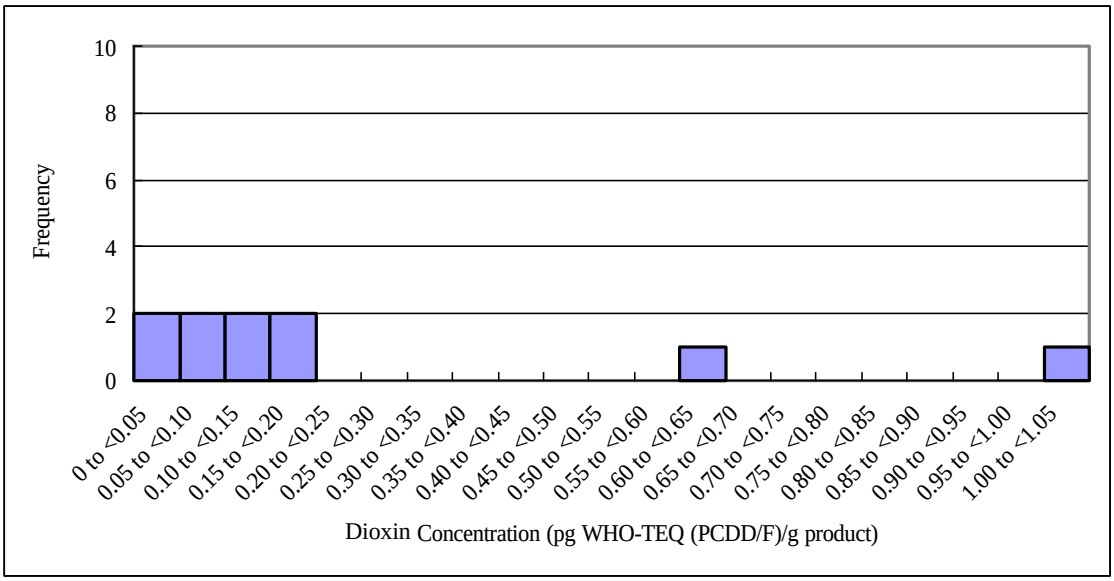


Figure 4 6:Freqency Distribution of Dioxin Concentrations in Eggs and Egg Products



4.24 Dioxin concentrations of the selected food groups are summarised below (see Table 4.5).

Table 4.5 Dioxin Concentration in Food Items Sampled in Hong Kong

Food Group	Median Concentration of Dioxins (pg WHO-TEQ/g product)
Meat and Meat Products	0.090
Poultry and Poultry Products	0.092
Milk and Milk Products	0.069
Seafood	0.099
Egg and Egg Products	0.117

References:

[1] Food and Environmental Hygiene Department of HKSAR. Food Consumption Survey 2000. Food and Environmental Hygiene Department;October 2001.

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

[3] World Health Organisation. Methodology for Exposure Assessment of Contaminants and Toxins in Food. World Health Organisation;June 2000.

[4] Australia New Zealand Food Authority. The 19th Australian Total Diet Survey – A Total Diet Survey of Pesticide Residues and Contaminants. Australia New Zealand Food Authority;2001.

[5] Joint FAO/WHO Expert Committee on Food Additives. Summary and Conclusions of the Fifty-seventh Meeting. JECFA; June 2001. Available from: http://www.who.int/pcs/jecfa/Summary57-corr.pdf

[6] AEA Technology Plc. Compilation of EU Dioxin Exposure and Health Data: Report for European Commission DG Environment, United Kingdom Department of the Environment Transport and the Regions. October 1999.

Chapter 5

Estimating Dietary Exposure to Dioxins

5.1 With the two sets of data, namely consumption pattern of five groups of at risk food items and their respective dioxin concentrations, we would in this chapter estimate the dietary exposure to dioxins of secondary school students. The general formula for this estimation is shown below –

General Formula for Dietary Exposure:

$$\text{Dietary Exposure} = \frac{\text{Food Chemical Concentration} \times \text{Food Consumption}}{\text{Body Weight}}$$

5.2 To estimate the average dietary exposure to dioxins, dioxin concentrations in the food groups concerned were expressed in TEQ. The average dioxin exposure by consumption of the concerned food

group, say food group A, would be estimated using the dioxin concentration and the consumption pattern of food group A according to the following formula. The average dietary exposure to dioxins is expressed in pg WHO-TEQ (PCDD/F)/ kg bw/ day.

Estimation of Dioxin Exposure by Average Eaters of Food Group A:

$$\text{Average Dioxin Exposure by Consumption of Food Group A (pg WHO-TEQ (PCDD/F)/ kg bw / day)} = \frac{\text{Dioxins in Food Group A (pg/ g)} \times \text{Mean Consumption of Food Group A (g/ day)}}{\text{Body Weight (kg)}}$$

Average Dioxin Exposure of Secondary School Students

5.3 For the estimation of average dietary exposure to dioxins, the mean consumption listed in Table 4.1 and the median concentration of dioxins listed in Table 4.5 in Chapter 4 were used. The total dietary exposure to dioxins for an average secondary school student was obtained by the summation of dioxin exposure across all the food groups. Using the average body weight of secondary school students of 52.0 kg, the results of the average dioxin exposure of secondary school students were

shown in the following table.

Table 5.1 : Dietary Exposure to Dioxins for Average Eaters

Food group	Mean consumption (g/ day)	Median concentration of dioxins (pg WHO -TEQ (PCDD/F) /g product)	Dietary exposure to dioxins* (pg WHO -TEQ (PCDD/F) / kg bw/day)
Meat and meat products	111.3	0.090	0.19
Poultry and poultry products	88.2	0.092	0.16
Milk and milk products	158.2	0.069	0.21
Seafood	133.7	0.099	0.25
Eggs and egg products	15.5	0.117	0.03
Total	**506.8**†	-	**0.83** †

* Average body weight of secondary school students in this study = 52.0 kg

† Figures may not add up to total due to rounding

5.4 From the above estimation, we found that the average dietary exposure to dioxins was **0.83 pg WHO -TEQ (PCDD/F) / kg bw/ day** for an average secondary school student in Hong Kong.

High Consumers among Secondary School Students

5.5 The concept of an average diet may not be useful to estimate particular at risk group like the high consumers, as the data for this group

may be even out in the averaging process. Therefore, the estimate of high exposure to dioxins 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[1,2] and U.S. EPA[3]. In this assessment, the 95th percentile exposure level was used to represent the dietary exposure to dioxins for high consumers.

5.6 The dietary exposure to dioxins of high consumers was **207 pg WHO-TEQ (PCDD/F) / kg b.w. day**. This level was about 2.5 times that of average eaters.

Effects of the Non-Detected Results

5.7 In this study, a value of 1/2 LOD was assigned to all test results below LOD. However, considering the ubiquitous nature of dioxins in the environment, the true value could lie anywhere between zero and the LOD.

5.8 To address this issue of uncertainty, dioxin concentration in each food sample was also estimated using an upper bound and lower bound estimates. The upper bound was calculated by setting results below LOD to the LOD while the lower bound was calculated by setting results below LOD to zero.

5.9 Using these upper and lower bound estimates, the dietary exposure to dioxins was calculated. The dioxin exposure of an average secondary school student would be anywhere between 0.31 (lower bound estimate) and 1.39 (upper bound estimate) pg WHO-TEQ (PCDD/F)/ kg bw/ day while that of high consumers could be anywhere between 0.78 (lower bound estimate) to 3.41 (upper bound estimate) pg WHO-TEQ (PCDD/F)/ kg bw/day.

References:

[1] World Health Organisation. Guidelines for the Study of Dietary Intakes of Chemical Contaminants. World Health Organisation; 1985.
[2] World Health Organisation. Food Consumption and Exposure Assessment of Chemicals. World Health Organisation;1997.
[3] U.S. Environmental Protection Agency. Guidelines for Exposure Assessment. Environmental Protection Agency;1992. Available from: http://www.epa.gov/nceawww1/exposure.htm

Chapter 6

Discussion

6.1 In Chapter 5, we have estimated the dietary exposure to dioxins of secondary school students. In this chapter, we discuss the health implications and other issues arising from the estimation and examine the limitations of this study.

Dietary Exposure to Dioxins

6.2 The dietary exposure to dioxins was estimated to be 0.85 pg WHO-TEQ (PCDD/F)/ kg bw/ day for an average secondary school student in HKSAR. This level was within the range of TDI recommended by the WHO in 1998 of 1 - 4 pg WHO-TEQ/ kg bw/ day. Hence it could be concluded that an average secondary school student would be unlikely to experience major toxicological effects of dioxins.

6.3 The WHO recommended that the dioxin intake should be reduced to a level below 1 pg TEQ/ kg bw/ day while the upper range of the TDI of 4 pg TEQ/ kg bw/ day represents a maximal tolerable intake. However, an intake above this upper range does not automatically mean that health is at risk. According to the WHO consultation, the TDI of 1 - 4 TEQ pg/ kg bw for dioxins and dioxin-like compounds, was derived by applying an uncertainty factor of 10 to the range of LOAELs of 14 - 37 pg TCDD/ kg bw/day. The consultation emphasised that the TDI represents a tolerable daily intake for life-time exposure and that occasional short-term excursions above the TDI would have no health consequences provided that the averaged intake over long periods is not exceeded[1].

6.4 Food products of plant origin such as cereals, fruits and vegetables as well as edible vegetable oil such as corn oil, olive oil and margarine were not included in this study since our food surveillance programme focused mainly on high risk food items. According to report

on dioxin exposure in some European countries, these foods may contribute to 6 - 45% of the total dietary exposure[2].

6.5 Dietary exposure to dioxins and related PCBs conducted by member states in the European Union[2] revealed that dioxin concentrations in meat and meat products were much higher than those in vegetables, cereals, fruits products as well as vegetable oil. The latter products had dioxin concentrations of around or below the limit of detection. Nevertheless, inclusion of these products into the dietary exposure study would present a more complete picture of dioxin exposure.

6.6 On the other hand, the TDI of 1 - 4 pg WHO-TEQ/ kg bw/ day recommended by the WHO has included the dietary exposure to dioxin-like PCBs besides dioxins. If dioxin-like PCBs were taken into account, the daily total TEQ exposure would be increased[3]. In the Netherlands, dietary exposure to dioxins and PCBs in 1991 was shown to be approximately equal. In Spain, the PCB exposure contributed

48-62% of total TEQ exposure in 1996 while the PCB exposure contributed to 49-57% of total TEQ in Sweden in 1990[4]. Hence doubling the estimate of exposure to dioxins will give a rough estimate to the total TEQ exposure.

6.7 Since food samples collected in this study were only sent for analysis of dioxins, total dietary exposure to dioxins and dioxin-like PCBs for secondary school students can be estimated by doubling the figure due to dioxins alone.

6.8 Based on the above, the estimate for total dietary exposure to dioxins and dioxin-like PCBs was 1.69 pg WHO-TEQ/ kg bw/ day for average eaters of the secondary school students in HKSAR. This level was within the range of tolerable daily intake recommended by the WHO in 1998 of 1 to 4 pg WHO-TEQ/ kg bw/ day.

6.9 However, for high consumers, the total dietary exposure to dioxins and dioxin-like PCBs was 4.14 pg WHO-TEQ/ kg bw/ day.

Hence there is a chance for the high consumers to have dioxin exposure above the recommended tolerable intake. However, TDI stresses on lifetime exposure and occasional short-term excursion above the TDI would have no health consequences provided that the average intake over long period is not exceeded.

6.10 In view of the significant contribution of dioxin-like PCBs in the total TEQ exposure, a more detailed study of PCB concentrations in food samples will give a better estimate of the total dietary exposure to dioxin and dioxin-like PCBs in HKSAR.

Major Dietary Sources of Dioxins

6.11 Based on the available data, we can identify the major dietary sources of dioxins for secondary school students in HKSAR. Table 6.1 shows the dietary exposure to dioxins from different food groups including meat and meat products, poultry and poultry products, milk and milk products, seafood, as well as eggs and egg products.

Table 6 The Concentration of Dioxins in Food and the Dietary Exposure to Dioxins for Average Eaters

Food Group	Dietary Exposure to Dioxins (pg WHO-TEQ (PCDD/F) / kg bw/day)
Meat and Meat Products	0.19 (23%)*
Poultry and Poultry Products	0.16 (18%)
Milk and Milk Products	0.21 (25%)
Seafood	0.25 (30%)
Eggs and Egg Products	0.03 (4%)
Total	**0.8**

* figures in brackets denote percentage contribution to total dietary exposure

6.12 The above table shows that seafood, meat and meat products as well as poultry and poultry products were significant dietary sources of dioxin exposure. Dioxin exposure from seafood was 0.25 pg WHO-TEQ (PCDD/F)/kg bw/day, which contributed to 30% of the dioxin exposure. Whereas exposure from meat and meat products as well as poultry and poultry products were 0.19 pg WHO-TEQ (PCDD/F)/ kg bw/ day (23% of the dioxin exposure) and 0.16 pg WHO-TEQ (PCDD/F)/ kg bw/ day (18% of the dioxin exposure) respectively.

6.13 Milk and milk products were also important dietary source to dioxin exposure for secondary school students. Dioxin exposure via the consumption of milk and milk products was 0.21 pg WHO-TEQ

(PCDD/F)/ kg bw/ day. It contributed to 25% of the dioxin exposure. Regarding the food consumption pattern, daily consumption of milk and milk products for average eaters was 158.2 g and was the highest among the selected food groups.

6.14 Among these five groups of food, eggs and egg products had a relative small contribution to the dioxin exposure, accounting for an exposure of 0.03 pg WHO-TEQ (PCDD/F)/ kg bw/ day or about 4% of the dietary exposure to dioxins. Even though dioxin concentrations in egg and egg products were high, their contribution to dioxin exposure was the least due to the relatively low daily consumption of 15.5 g for an average eater.

6.15 Similar pattern was observed in some European countries, in which fish and fish products contributed to 2-63% of dioxin exposure, meat and meat products contributed to 6-32% of dioxin exposure and milk and dairy products contributed to 16-39%[2].

International Comparison

6.16 Estimates of dietary exposure to dioxins produced by some industrialised countries are summarised in Table 6.2.

Table 6.2: Dietary Exposure to Dioxins in Other Countries

Countries	Year of Publishing the Study	Dioxin Exposure for Average Eater (pg WHO-TEQ (PCDD/F) / kg bw/day)
Canada[5]	1991	0.49 – 2.0
Denmark[4]	1995	2.44
Finland[4]	1991	1.36
Germany[4]	1995	0.99
Japan[5]	1998	0.63
Netherlands[5]	1997	1.1
New Zealand[6]	1998	0.18
Spain[4]	1996	3.0
Sweden[4]	1997	1.75 – 2.45
United Kingdom[6]	2000	0.8
United States[5]	1996	0.52 – 2.57
HKSAR	2002	0.85

6.17 It can be seen that the daily dietary exposure to dioxins of an average eater in industrialised countries ranged from 0.18 to 3 pg WHO-TEQ (PCDD/F) /kg bw /day. Estimate for dietary exposure to dioxins for secondary school students in HKSAR is comparable with those for adults in some other countries, except in Denmark and Spain, where higher exposure estimates were observed. However, direct

comparison of the results of this study with other studies conducted overseas has to be done with caution. This is because exposure data were obtained in different years; different methodologies including analytical methods were adopted, for example, different types of food samples were selected in different studies, some may be in cooked form while others may be in raw form;and different methods for treatment of non-detected results in estimating dioxin concentrations were employed[5].

Limitations of the Study

6.18 Limitations in terms of food sampling, food consumption pattern and dioxin concentration are discussed below.

Food Sampling

6.19 Most food products available in HKSAR are imported from the Mainland or overseas countries. In our food surveillance programme, because of resource constraints, only a limited number of food items were sampled from the local market for dioxin testing. The

number of samples tested for dioxins in the surveillance programme might not be representative of the average food being consumed by the general population and be subject to statistical variation. This may affect, to a certain extent, the reliability of dioxin exposure estimate.

6.20 All samples taken for the dioxin testing were in the raw (uncooked) state. However, dioxin residues in food vary with different cooking methods. The U.S. EPA[7] had conducted a study to investigate the effect of broiling on dioxin concentrations in meat and fish. The results showed that dioxin concentration in hamburger remained the same after broiling while the concentration increased by 84% in bacon and decreased by 34% in catfish. Therefore, testing of dioxin concentrations in raw food samples may not be able to reveal accurately the actual intake of dioxins.

Food Consumption Pattern

6.21 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 used was very comprehensive, it was not possible to cover some less important food items, some of which may be relevant to dioxin exposure. As a result, the dietary exposure estimate might have been underestimated. To expand the scope of dietary exposure, a comprehensive population based food consumption survey is recommended, which would facilitate the conduct of a wide range of dietary exposure assessment studies in the future.

Dioxin Concentration

6.22 Dioxin concentrations expressed on fat basis were converted to product basis before the determination of dioxin exposure, based on the assumption that dioxins would be present in the fat portion only.

Risk Management Measures

6.23 Dioxin exposure can be reduced through the reduction of dioxin emissions and the interruption of their pathways into the food chain.

6.24 Environmental control is the primary measure to minimise total exposure to dioxins. This involves global effort in the reduction of dioxin emission from industries and incinerators, the reduction of the manufacture and use of dioxins and PCBs, as well as the ban of use of PCBs, especially in open system[8]. In the United Kingdom, the manufacture and general use of PCBs was banned in 1986 and subsequently dietary exposure to both dioxins and PCBs was reduced by about 75%[9]. For incinerators, advances in technology have introduced the high temperature incineration, which could reduce the emission of dioxins from these incinerators[10].

6.25 International organisations are developing code of practices or

quality control programmes with an aim to reduce dioxin contamination in foods[8]. For example, Codex Alimentarius Commission is drafting a code of practice for source directed measures to reduce dioxin contamination of food[8,11].

6.26 Since HKSAR depends mainly on imported food, the impact of local environmental control on total exposure to dioxins, particularly dietary exposure, is unlikely to be great. The Food and Environmental Hygiene Department will continue to monitor foods available in HKSAR, especially on those high-risk food items such as milk, seafood, meat, poultry and their products.

6.27 At present, there is no international consensus on the regulation of dioxins in food. The Codex Alimentarius Commission is still discussing the surveillance standard for dioxins in food. In HKSAR, the action level for food surveillance on dioxins is 1 pg WHO-TEQ (PCDD/F) / g sample on product basis. Annex shows the action levels adopted by some developed countries. The Department will continue to

monitor the international development on regulation of dioxins.

6.28 To monitor the exposure of the population to dioxins, dietary exposure to dioxins to be conducted at periodic intervals is considered useful.

6.29 Though meat, poultry, seafood, milk and their products were the major dietary sources of dioxins, avoidance of these food items is not necessary as they are good sources of protein and other nutrients.

6.30 As an ultimate goal, WHO recommended that dioxin exposure should be reduced to a level below 1 pg TEQ/ kg bw /day. To minimise dietary exposure to dioxins, the public is advised to consume low-fat products, to trim fat from meat and meat products, to reduce the amount of animal fat used in food preparation and to use cooking methods that reduce fat (e.g. broiling). As a general advice, a balanced diet is recommended to maintain health and to avoid excessive exposure to contaminants from a small range of food items[9,12].

References:

[1] World Health Organisation. Executive Summary: Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI) – WHO Consultation. World Health Organisation;May 1998. Available from: http://www.who.int/pcs/pubs/dioxin-exec-sum-final.html (November 1999)

[2] European Commission. Reports on Tasks for Scientific Cooperation: Assessment of Dietary Intake of Dioxins and Related PCBs by the Population of EU Member States. European Commission; 7 June 2000

[3] Codex Committee on Food Additives and Contaminants. Discussion Paper on Dioxins (CX/FAC 00/26). Codex Alimentarius Commission;December 1999.

[4] AEA Technology, Plc. Compilation of EU Dioxin Exposure and Health Data: Task 4 – Human Exposure: Report Produced for European Commission DG Environment and United Kingdom Department of the Environment, Transport and the Regions (DETR) . AEA Technology, Plc.;October 1999.

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[9] UK Food Standards Agency. Food Standards Agency and Environment Agency Question and Answer Briefing on Dioxins and PCBs, and the Associated Statement by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). Food Standards Agency;16 November 2001. Availab le from: http://www.foodstandards.gov.uk/pressreleases/statements/pcbsdioxin.htm

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Chapter 7

Conclusion

7.1 For an average secondary school student in Hong Kong, dioxin exposure from food was 0.85 pg WHO-TEQ (PCDD/F)/kg bw/day. This was within the tolerable daily intake limit established by WHO in 1998. The dioxin exposure of high consumers, i.e. those above 95th percentile exposure level was 2.07 pg WHO-TEQ (PCDD/F)/kg bw/day. This level was about 2.5 times that of average eaters. From our study, it could be concluded that an average secondary school student would be unlikely to experience major toxicological effects of dioxins.

7.2 The pattern of dietary exposure showed that seafood, meat as well as poultry and their products were the major dietary sources of dioxins. Dioxin concentration in milk was not high but the consumed amount made it an important source. On the other hand, dioxin

concentrations in eggs were high but the consumption level was relatively low.

7.3 International efforts in the reduction of dioxin emission and their subsequent contamination of food are essential to minimise the dietary exposure to dioxins. Locally, the Food and Environmental Hygiene Department will continue to monitor dioxin concentration in foods available in Hong Kong, especially on those high-risk food items.

7.4 Though meat, poultry, seafood, milk and their products were the major dietary sources of dioxins, avoidance of these food items is not necessary as they are good sources of protein and other nutrients. Furthermore, milk and milk products are rich in calcium. To minimise dietary exposure to dioxins, the public is advised to consume low-fat products, to trim fat from meat and meat products, to reduce the amount of animal fat used in food preparation and to use cooking methods that reduce fat (e.g. broiling). As a general advice, a balanced diet is recommended to maintain health and to avoid excessive exposure to

contaminants from a small range of food items.

Annex

Action Levels for Monitoring Dioxins in Food

Action Level for Monitoring Dioxins in Food

Products	European Union[1] [(a)]	United States[2]	Canada[3]
Milk and milk products, including butter fat	3 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Hen eggs and egg products	3 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Meat and meat products derived from ruminants (bovine animals, sheep)	3 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Meat and meat products derived from poultry and farmed game	2 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Meat and meat products derived from pigs	1 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Liver and derived products	6 pg WHO-PCDD/F-TEQ/g fat[(b,c)]	--	--
Muscle meat of fish and fishery products and products thereof	4 pg WHO-PCDD/F-TEQ/g fresh weight[(b)]	50 ppt (TCDD)	20 ppt (TCDD)
Animal fat from ruminants	3 pg WHO-PCDD/F-TEQ/g fat [(b)]	--	--
Animal fat from poultry and farmed game	2 pg WHO-PCDD/F-TEQ/g fat[(b)]	--	--
Animal fat from pigs	1 pg WHO-PCDD/F-TEQ/g fat [(b)]	--	--
Animal fat from mixed animal fat	2 pg WHO-PCDD/F-TEQ/g fat[(b)]	--	--
Vegetable oil	0.75 pg WHO-PCDD/F-TEQ/g fat [(b)]	--	--
Fish oil intended for human consumption	2 pg WHO-PCDD/F-TEQ/g fat[(b)]	--	--
Drinking water	--	$3x10^{-8}$ mg/L (TCDD)	--

Note:

[(a)] upper bound concentrations; upper bound concentrations are calculated based on the assumption that all values of the different congeners less than the limit of determination are equal to the limit of determination.

[(b)] These maximum levels shall be reviewed for the first time by 31 December 2004 at the latest in the light of new data on the presence of dioxins and dioxin-lie PCBs, in particular with a view to the inclusion of dioxin-like PCBs in the levels to be set and will be further reviewed by 31 December 2006 at the latest with the aim of significantly reducing the maximum levels.

[(c)] The maximum levels are not applicable for food products containing less than 1% fat.

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Food and Environmental Hygiene Department

DIETARY EXPOSURE TO HEAVY METALS OF SECONDARY SCHOOL STUDENTS

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Table of Contents

Executive Summary

Dietary Exposure To Heavy Metals of Secondary School Students

Purposes

1. This study aims to determine the dietary exposure to heavy metals of secondary school students in Hong Kong so as to assess whether there are any risks to their health. The potential for any risks to health are assessed by comparing the dietary exposure to heavy metals with the appropriate safe exposure levels - Provisional Tolerable Weekly Intakes (PTWIs) recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA). PTWI is an estimate of the amount of a contaminant that can be ingested over a lifetime without appreciable risk.

Heavy Metals and Dietary Exposure

2. Three heavy metals, namely arsenic, cadmium and mercury, were chosen for this study, principally because of their toxicities. These heavy metals are environmental contaminants that are present naturally in the Earth's crust. They may also be discharged to the environment through industrial uses.

3. The possible health effects of heavy metals vary, depending on the unique features of the metals and the route of exposure. Human may be exposed to these metals through the food chain, after the food has been contaminated. Acute toxicity resulting from ingesting food contaminated with these heavy metals is uncommon, but chronic exposure to these metals may result in undesirable toxic effects. Of the three metals studied, mercury is a toxic chemical, particularly in its organic form, which is neurotoxic. Inorganic arsenic, a human carcinogen, is the more toxic form of arsenic. Cadmium is toxic to the kidney.

4. Dietary exposure to a chemical is determined by its concentrations in foods and the amounts of foods eaten. A food that contains high levels of a particular chemical can make a significant

contribution to dietary exposure even if it is eaten in small amounts. Conversely, a food that contains low concentrations but is eaten in large quantities can also make a large contribution to dietary exposure.

Scope and Method

5. In determining the dietary exposure of secondary school students to the heavy metals, two sets of data were used. The first set of data on concentrations of arsenic, cadmium and mercury in food was obtained from the food surveillance programme of the Department. Data on 2510 food samples collected between 1999 and 2001 were extracted from the food surveillance database. These food items were categorised under six target food groups, namely “cereals and cereal products”, “vegetables”, “meat, poultry and their products”, “fish”, “seafood other than fish” and “milk and dairy products”.

6. The second set of data on food consumption of the above food groups of secondary school students was derived from the food consumption survey conducted by this Department in late 2000.

Dietary Exposure to Heavy Metals of Secondary School Students

7. Using the above two sets of data, dietary exposure to heavy metals was estimated. **For an average secondary school student in Hong Kong, the dietary exposures to inorganic arsenic, cadmium and mercury were 23, 29 and 29 μg/kg bw/week respectively.** They fell within the PTWIs of 15, 7 and 5 μg/ kg bw/ week for inorganic arsenic, cadmium and mercury respectively.

8. To estimate the heavy metals exposure of high consumers, those above 95th percentile exposure level were studied. **The dietary exposures to inorganic arsenic, cadmium and mercury were 68 and 61 μg/kg bw/week respectively.** The 95th percentile of exposure was approximately two to three times the average exposure.

9. 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 life-time 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.

10. The results of the study showed that predatory fish, such as shark, tuna and swordfish, had the highest concentration of mercury while "seafood other than fish", particularly shellfish, was identified as the main dietary source of cadmium and inorganic arsenic.

Implications and Limitations of the Study

11. Comparing the results of this study with other similar studies conducted in China, Australia, USA and the UK, it was found that the dietary exposures to arsenic and cadmium were similar. However, dietary exposure to mercury was slightly higher compared to these countries. This could be explained by different consumption patterns, analytical methods, and numerical values assigned to non-detected results.

12. From our study, **it could be concluded that the estimated dietary exposures to inorganic arsenic, cadmium and mercury for an average secondary school student were below the PTWI established by JECFA and an average secondary school student would not experience major toxicological effects of these heavy metals.**

13. Several limitations of this study were identified. This study used food surveillance data which might produce biased results in dietary exposure assessment, as they were examined chiefly for enforcement purpose and might contain higher levels of contaminants. The limits of detection (LOD) were relatively high, which might result in overestimating the heavy metal content, particularly among those food groups with the majority of food samples having metal concentrations below LOD. 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.

Recommendations

14. Dietary intake is one of the major routes 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.

15. The LOD for heavy metals for food surveillance and enforcement purpose were relatively high and would introduce uncertainties in exposure studies especially when a significant proportion of food samples had below LOD concentrations. We recommend the 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 by this Department, lower reporting limits of contaminants in foods can be achieved which in turn provide more accurate dietary exposure assessment.

16. Consumption data used in this study were obtained from a

consumption survey conducted in secondary school children using food frequency questionnaires. 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.

17. 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. However, as fish are excellent sources of high-quality protein and low in saturated fat, 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 concentrations of arsenic and cadmium, as well as other food hazards.

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.

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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.

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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; medi um 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 Committed 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.

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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].

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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 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:

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

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 90^{th} and above percentiles have been recommended for estimating the risk of high exposure to contaminants while the 95^{th} percentile is frequently quoted by various organisations such as WHO [6,7] and U.S. EPA [8]. In this assessment, the 95^{th} percentile exposure level was used to represent the dietary exposure to heavy metals for high consumers.

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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/g) of total arsenic,cadmium and mercury in selected food groups

Food group	Total Arsenic		Cadmium		Mercury	
	% of samples below LOD	Median concentration (μg/g)	% of samples below LOD	Median concentration (μg/g)	% of samples below LOD	Median concentration (μg/g)
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	**222**	**-**	**341**	**-**	**100**	**-**

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	**1291**

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	**2.53** (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 (mg/kg bw /week)	Exposure for Average Eaters (mg/kg bw/week)	Exposure for High Consumers (mg/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 ANZEA 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.

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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.

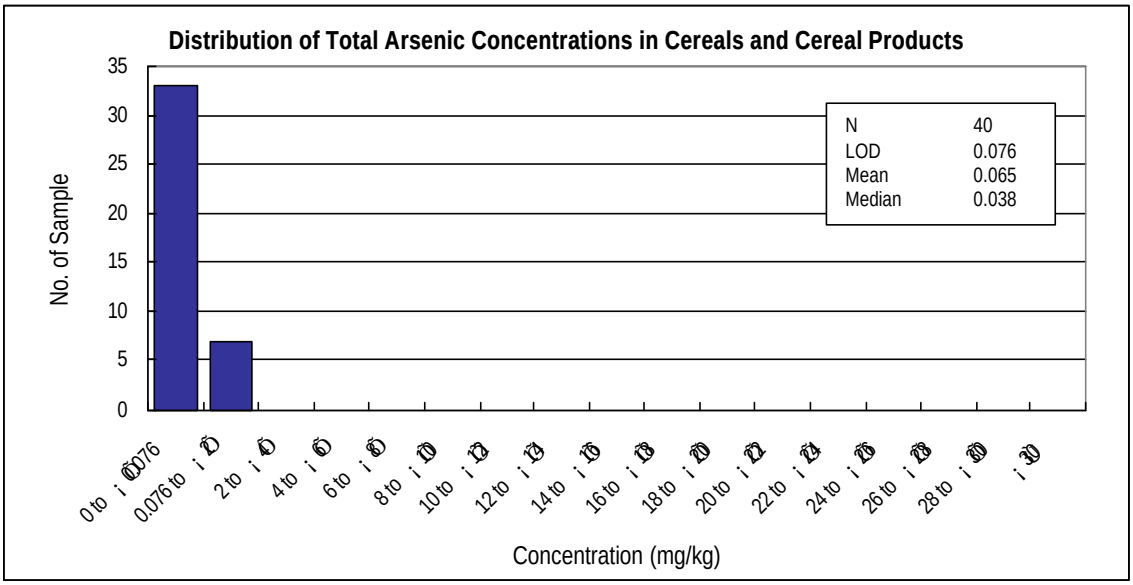
9.5 The limits of detection (LOD) of heavy metals testing were relatively high as they were designed for food surveillance and enforcement purposes. This would introduce uncertainties in exposure studies significantly especially when a large proportion of results were below LOD. We recommend that use 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.

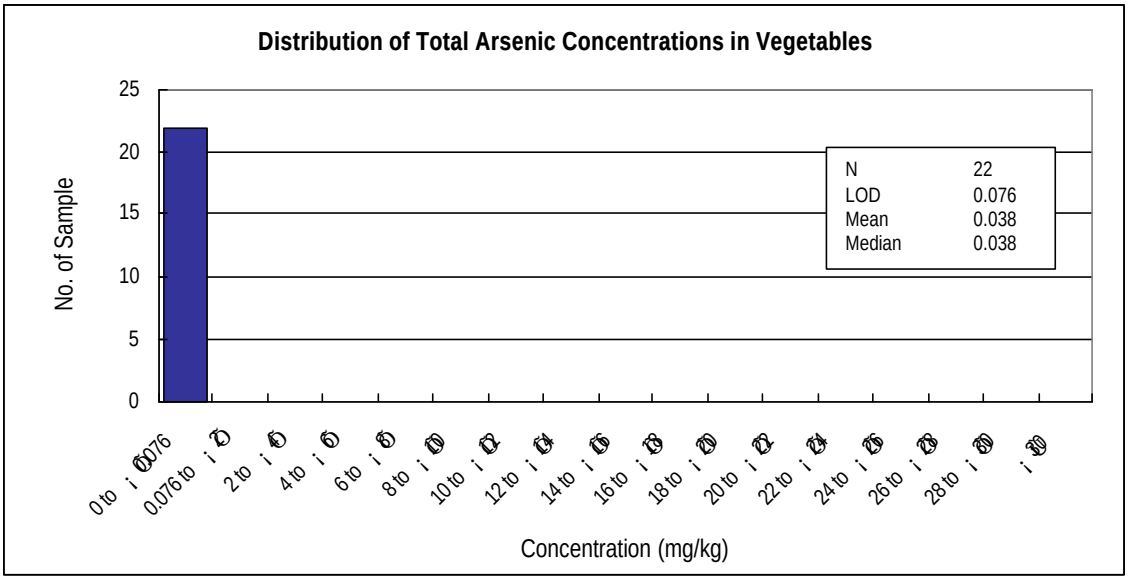
9.6 In this study, consumption data was obtained from the Food Consumption Survey conducted by this Department on secondary school students. To monitor the trend of the exposure and produce more accurate estimates, we will explore the possibility of conducing a population-based food consumption survey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

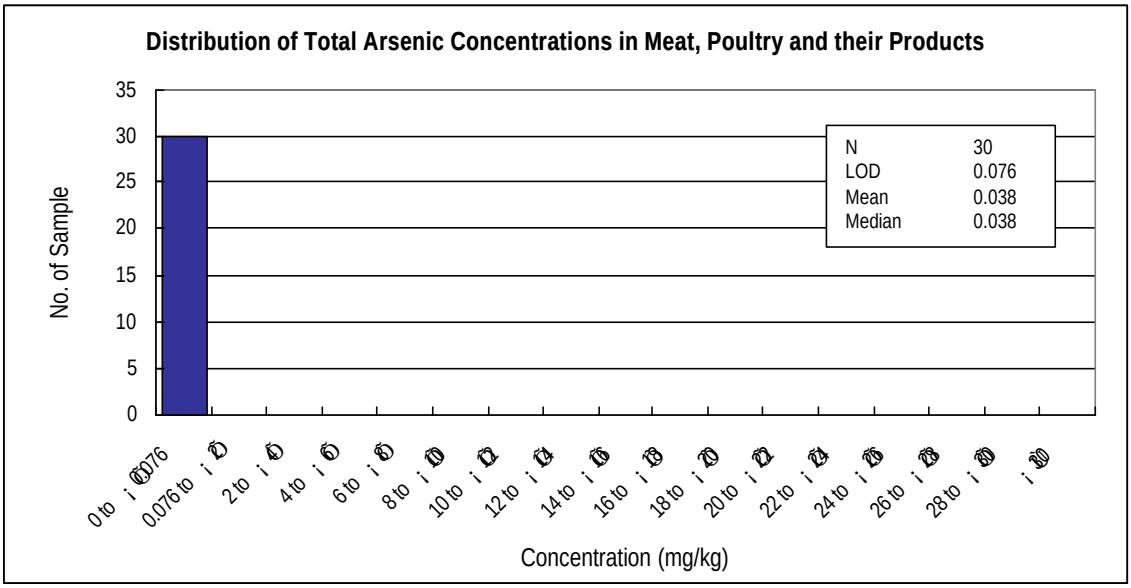
9.7 A balanced diet is essential to avoid excessive exposure to

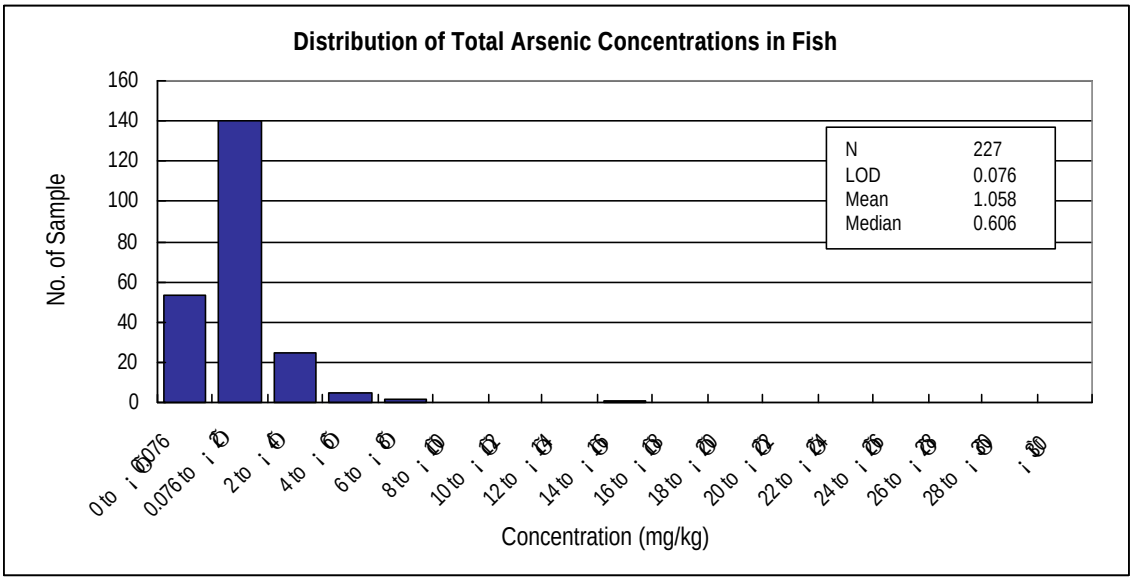
contaminants from a small range of food items. Vulnerable groups such as children and pregnant women should be careful in the selection of food, in particular, they are advised not to consume excessive amount of predatory fish such as shark, tuna and swordfish, which may contain higher concentrations of arsenic and 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.

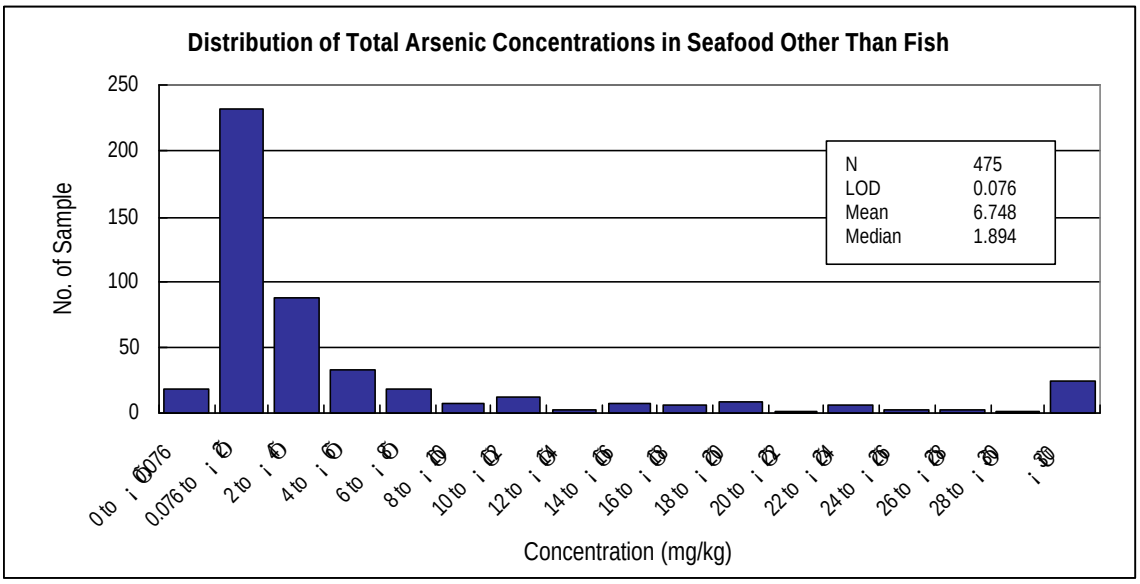
Annex 1: Distributions of Total Arsenic Concentration in Six Food Groups

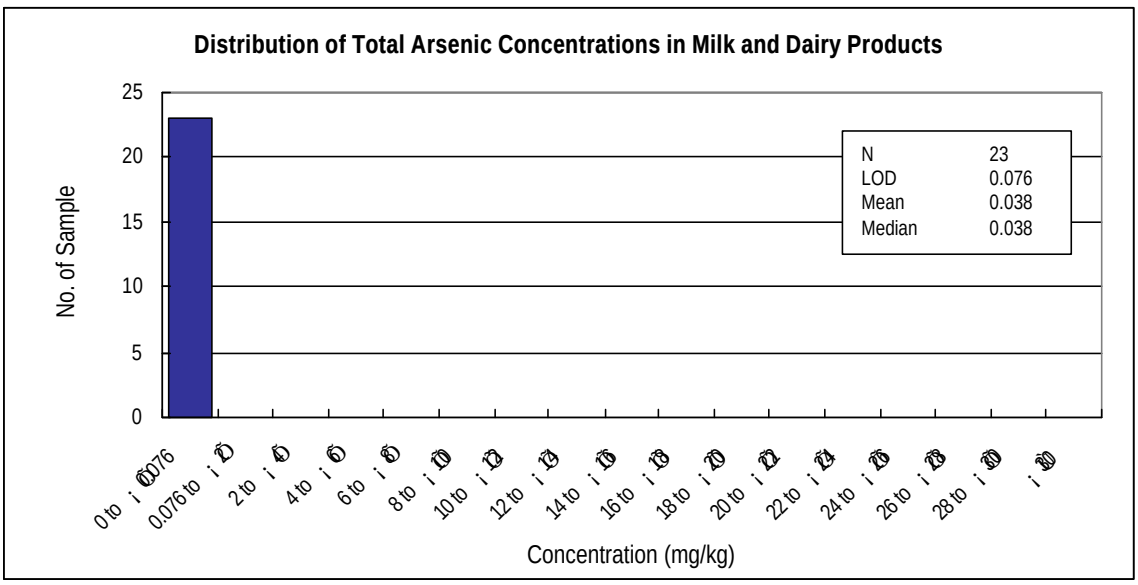




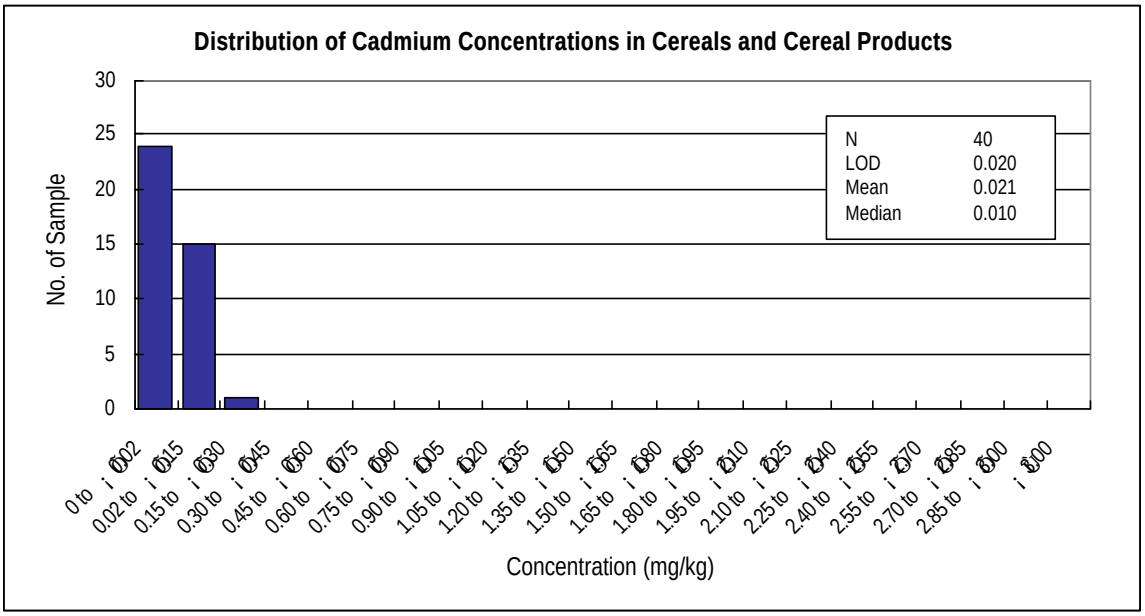


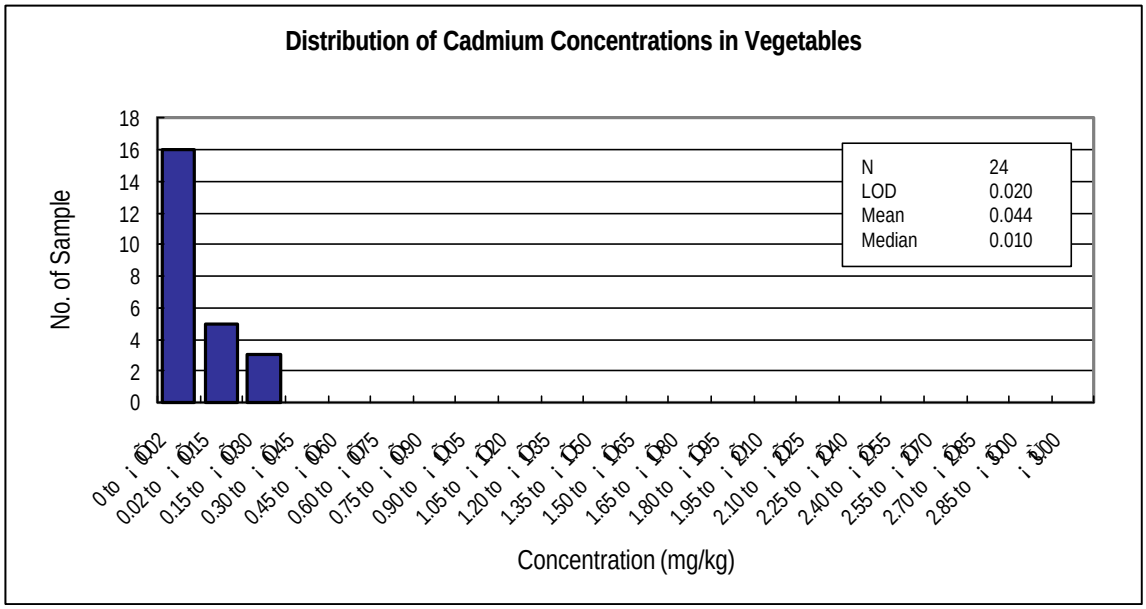

Distribution of Total Arsenic Concentrations in Fish
No. of Sample
Concentration (mg/kg)
N 227
LOD 0.076
Mean 1.058
Median 0.606

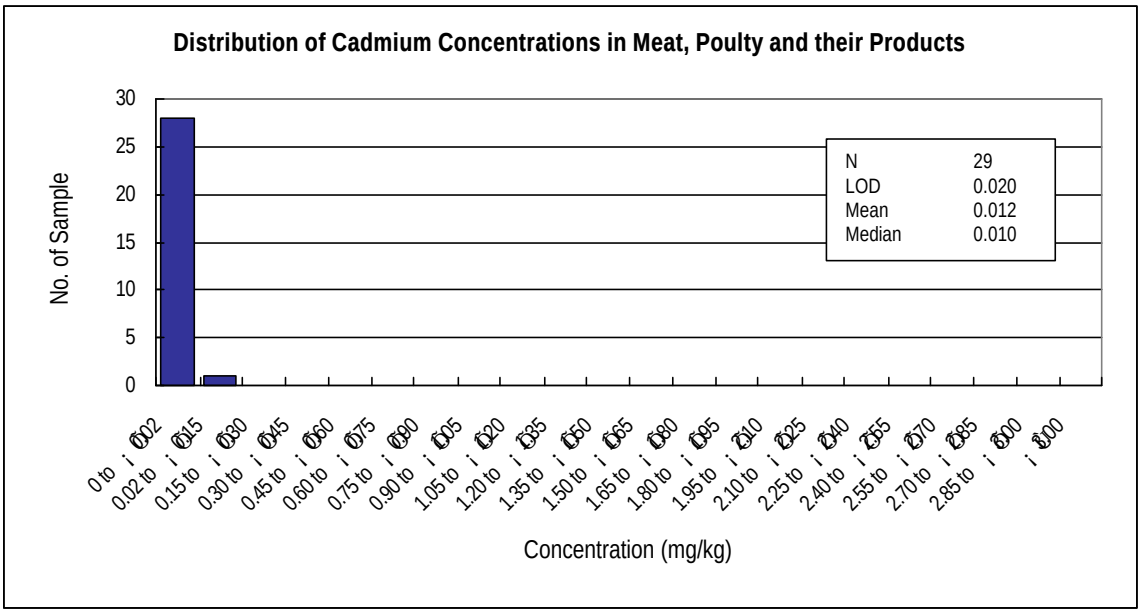

Distribution of Total Arsenic Concentrations in Seafood Other Than Fish
No. of Sample
Concentration (mg/kg)
N 475
LOD 0.076
Mean 6.748
Median 1.894

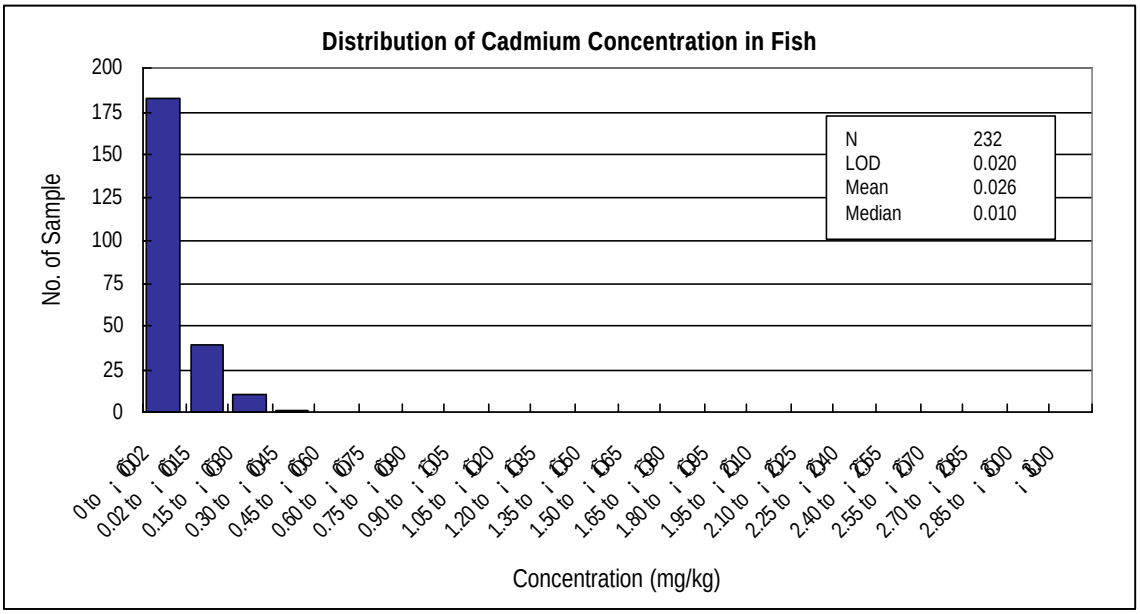

Distribution of Total Arsenic Concentrations in Milk and Dairy Products
No. of Sample
Concentration (mg/kg)
N 23
LOD 0.076
Mean 0.038
Median 0.038

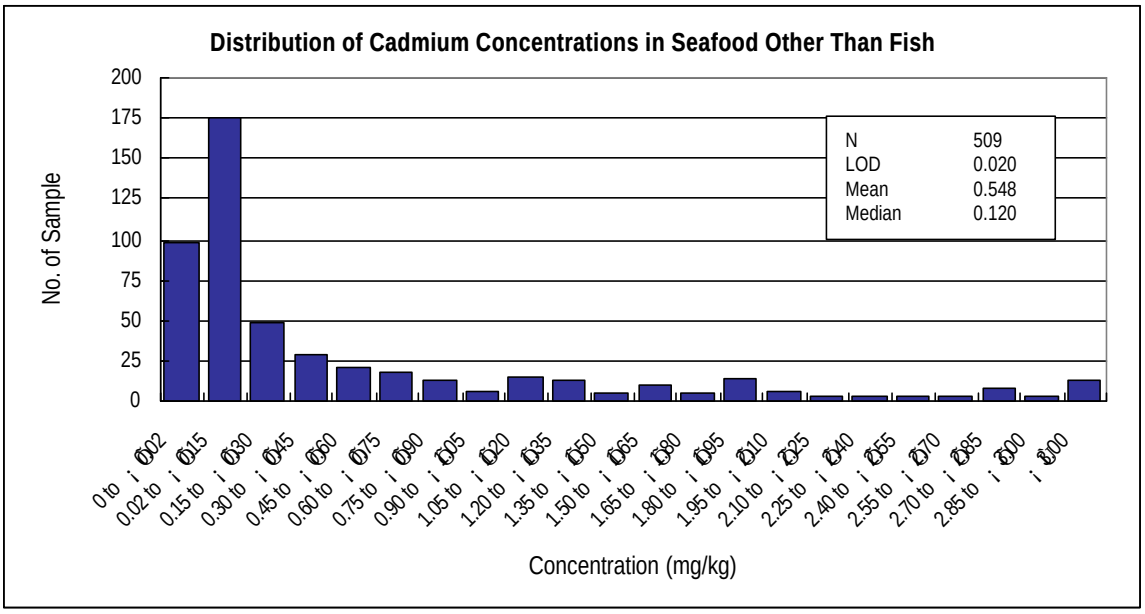
Annex 2: Distributions of Cadmium Concentration in Six Food Groups

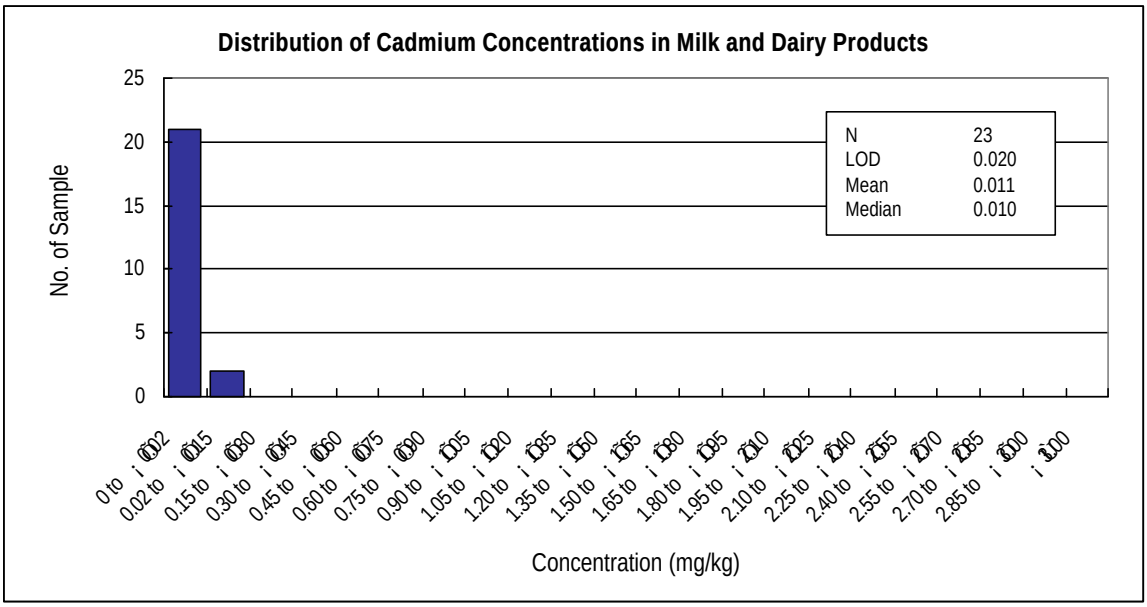




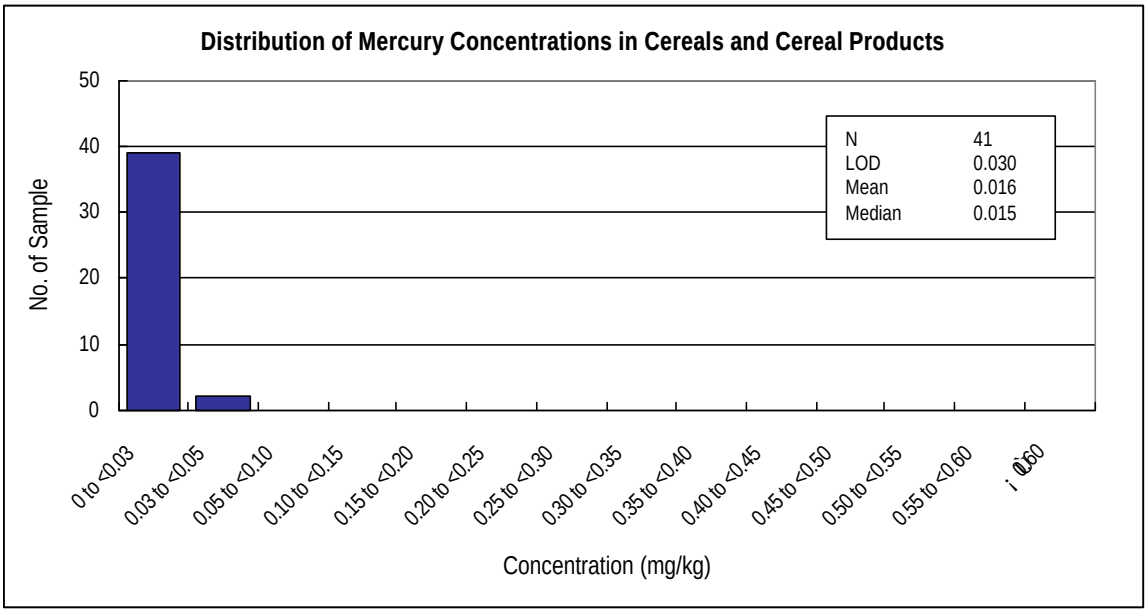


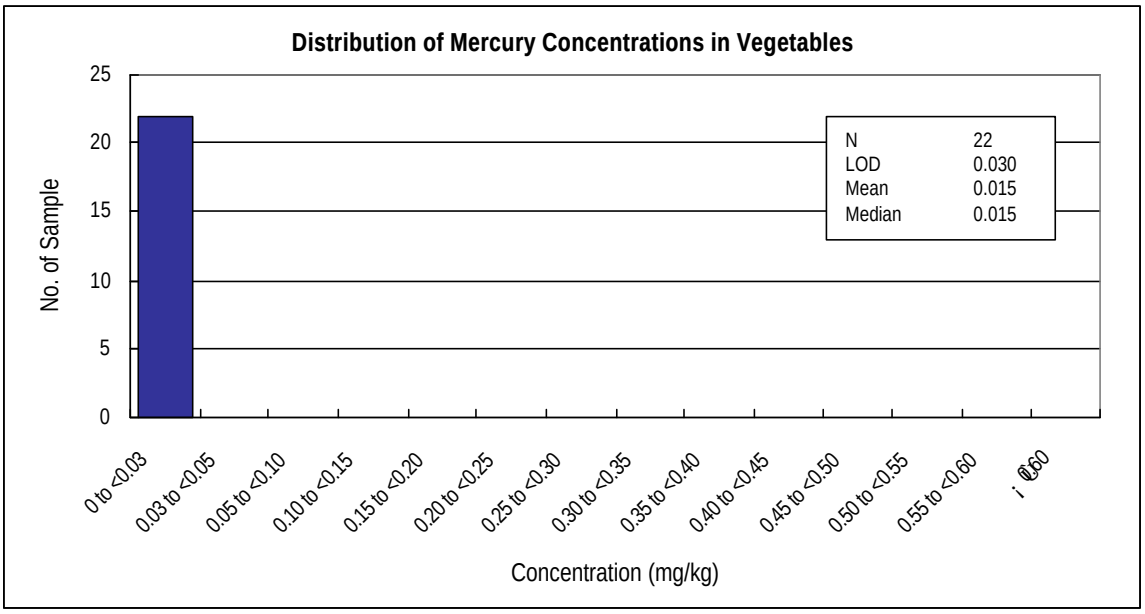

Distribution of Cadmium Concentration in Fish
No. of Sample
200
175
150
125
100
75
50
25
0
N 232
LOD 0.020
Mean 0.026
Median 0.010
0 to 0.02
0.02 to 0.15
0.15 to 0.30
0.30 to 0.45
0.45 to 0.60
0.60 to 0.75
0.75 to 0.90
0.90 to 1.05
1.05 to 1.20
1.20 to 1.35
1.35 to 1.50
1.50 to 1.65
1.65 to 1.80
1.80 to 1.95
1.95 to 2.10
2.10 to 2.25
2.25 to 2.40
2.40 to 2.55
2.55 to 2.70
2.70 to 2.85
2.85 to 3.00
3.00 to 3.15
Concentration (mg/kg)

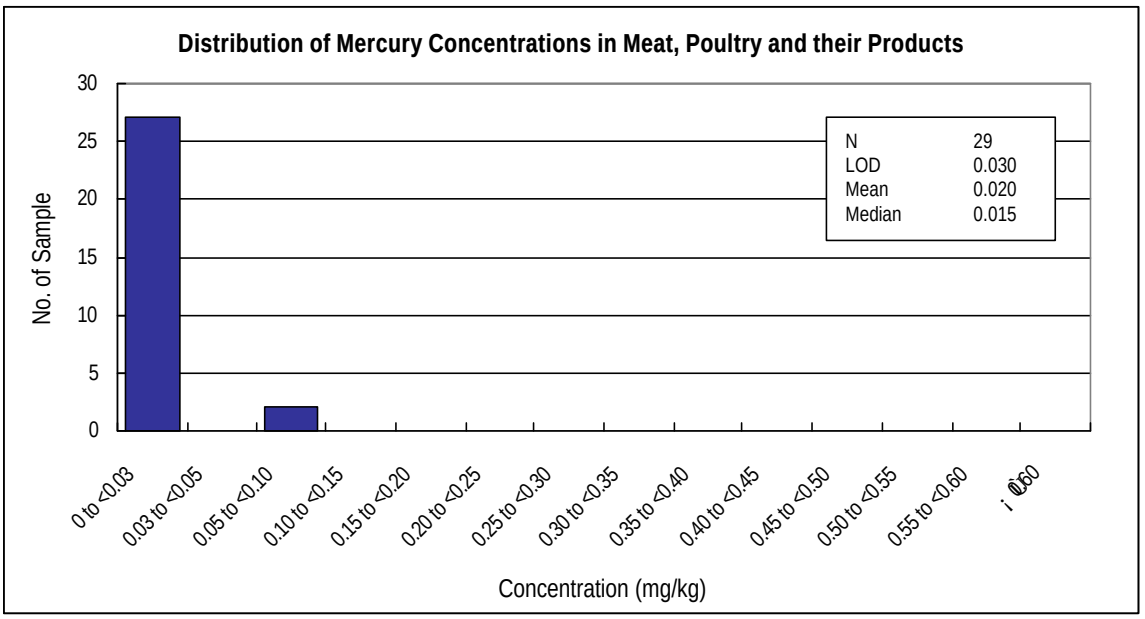

Distribution of Cadmium Concentrations in Seafood Other Than Fish
No. of Sample
200
175
150
125
100
75
50
25
0
N 509
LOD 0.020
Mean 0.548
Median 0.120
0 to 0.02
0.02 to 0.15
0.15 to 0.30
0.30 to 0.45
0.45 to 0.60
0.60 to 0.75
0.75 to 0.90
0.90 to 1.05
1.05 to 1.20
1.20 to 1.35
1.35 to 1.50
1.50 to 1.65
1.65 to 1.80
1.80 to 1.95
1.95 to 2.10
2.10 to 2.25
2.25 to 2.40
2.40 to 2.55
2.55 to 2.70
2.70 to 2.85
2.85 to 3.00
3.00 to 3.15
Concentration (mg/kg)

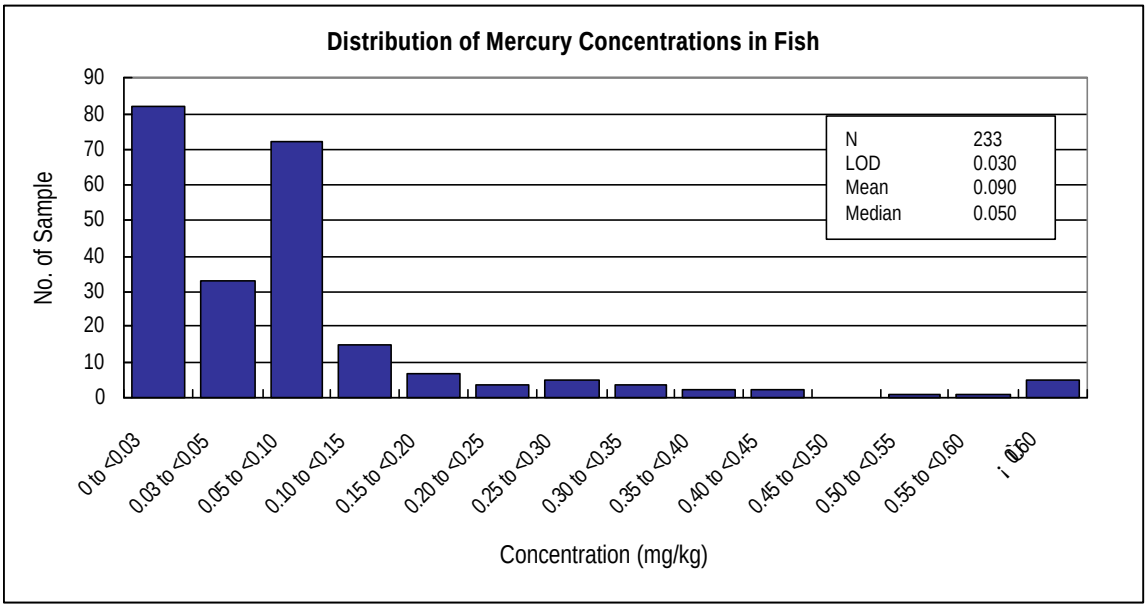

Distribution of Cadmium Concentrations in Milk and Dairy Products
No. of Sample
25
20
15
10
5
0
N 23
LOD 0.020
Mean 0.011
Median 0.010
0 to 0.02
0.02 to 0.15
0.15 to 0.30
0.30 to 0.45
0.45 to 0.60
0.60 to 0.75
0.75 to 0.90
0.90 to 1.05
1.05 to 1.20
1.20 to 1.35
1.35 to 1.50
1.50 to 1.65
1.65 to 1.80
1.80 to 1.95
1.95 to 2.10
2.10 to 2.25
2.25 to 2.40
2.40 to 2.55
2.55 to 2.70
2.70 to 2.85
2.85 to 3.00
3.00 to 3.15
Concentration (mg/kg)

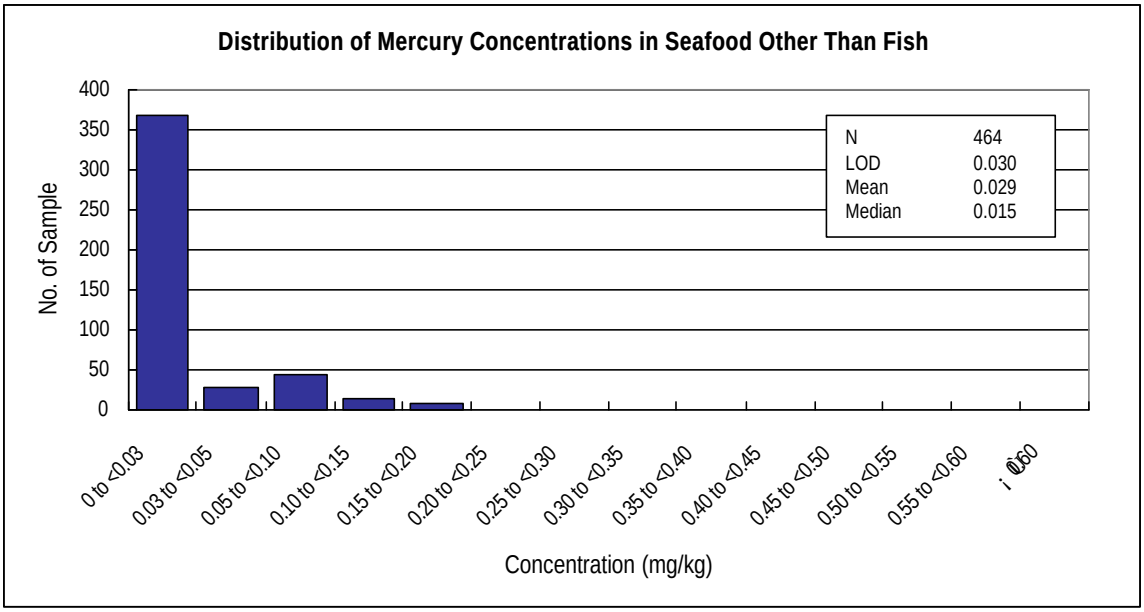
Annex 3: Distributions of Mercury Concentration in Six Food Groups

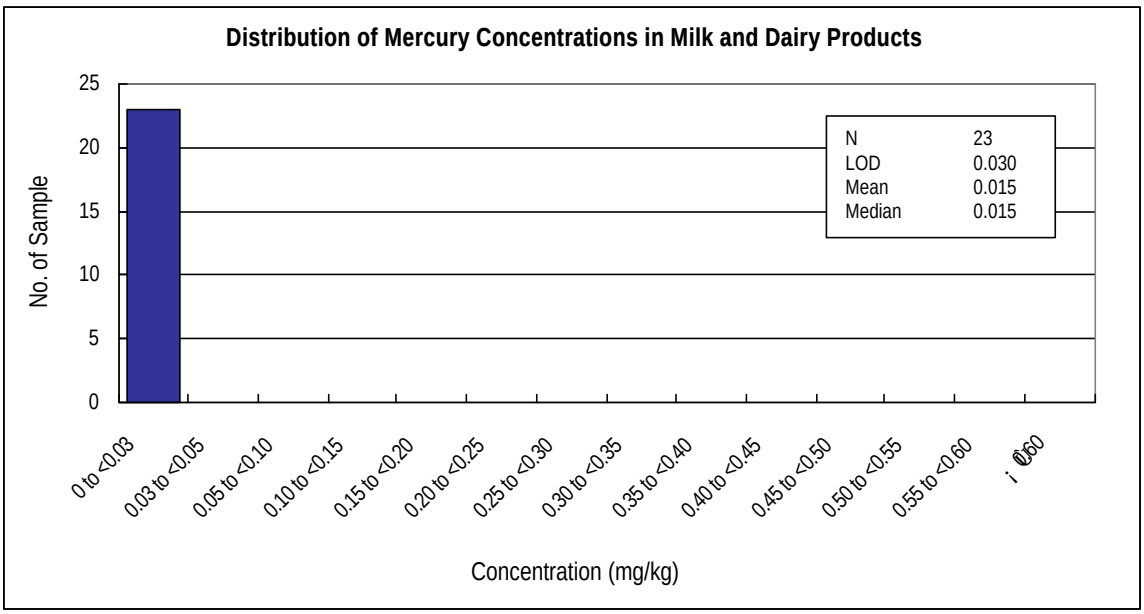







Distribution of Mercury Concentrations in Fish
No. of Sample
90
80
70
60
50
40
30
20
10
0
N 233
LOD 0.030
Mean 0.090
Median 0.050
0 to <0.03
0.03 to <0.05
0.05 to <0.10
0.10 to <0.15
0.15 to <0.20
0.20 to <0.25
0.25 to <0.30
0.30 to <0.35
0.35 to <0.40
0.40 to <0.45
0.45 to <0.50
0.50 to <0.55
0.55 to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