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Chemical Hazard Evaluation

DIETARY EXPOSURE TO DIOXINS OF SECONDARY SCHOOL STUDENTS

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Food and Environmental Hygiene Department
HKSAR

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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

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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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*

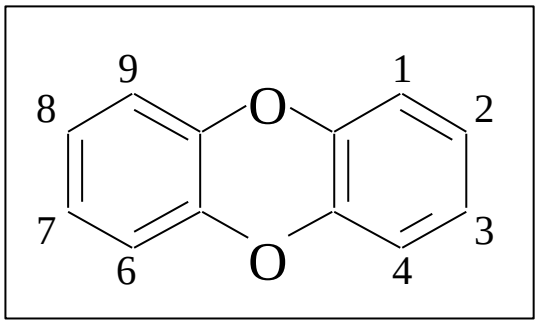
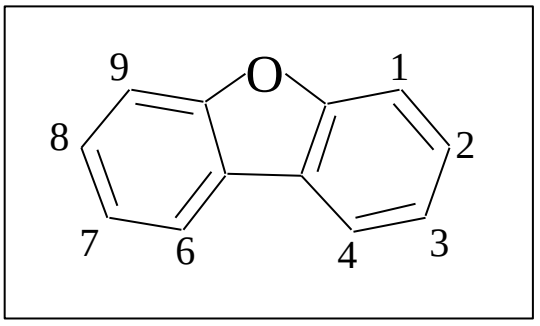


Figure 2.2: Basic Structure of PCDFs*



*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°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].

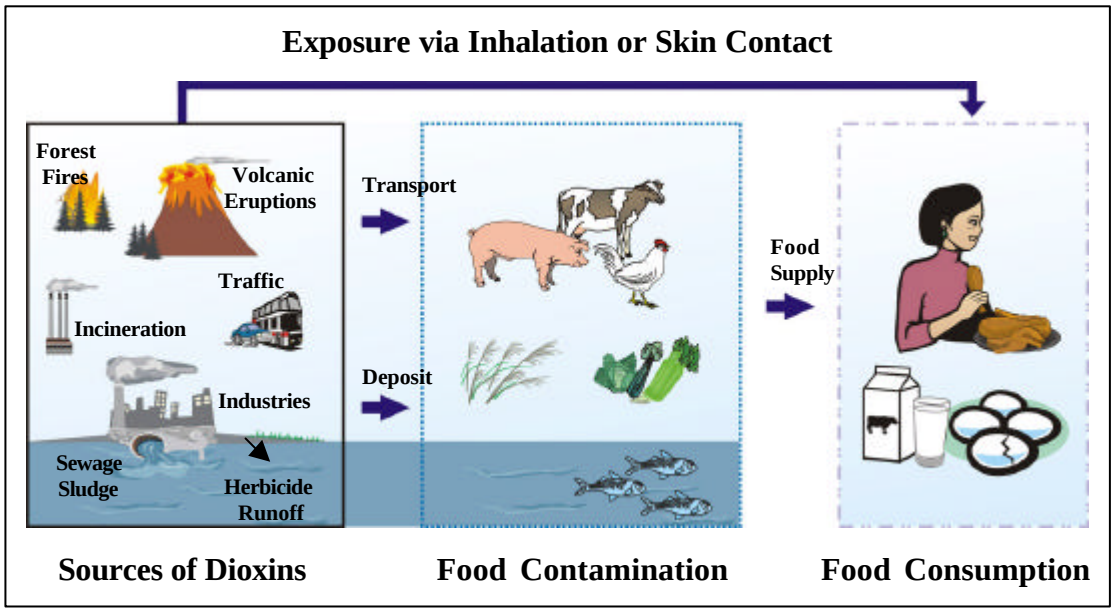
2.9 Sediments of surface waters are thought to be the ultimate sink of dioxins. Dioxins enter fish and other aquatic organisms through ingestion of sediments. The persistence of dioxins results in bioaccumulation in aquatic organisms[3].

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 2.1: WHO-TEF (1997) 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] –

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

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 2.2: 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.

References:

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

[4] International Programme on Chemical Safety. IPCS Environmental Health Criteria 210: Principles for the Assessment of Risks to Human Health from Exposure to Chemicals. International Programme on Chemical Safety; 1999.

[5] World Health Organisation. Report of a FAO/WHO Consultation: Food Consumption and Exposure assessment of Chemicals. World Health Organisation; 1997.

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[12] World Health Organisation. Report of a Joint USFDA/WHO International Workshop on Total Diet Studies in Cooperation with the Pan American Health Organisation: GEMS/Food Total Diet Studies. World Health Organisation; August 1999.

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	**506.8**

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 –

Product-based Dioxin Concentration = Fat-based Dioxin Concentration × % 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 were 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

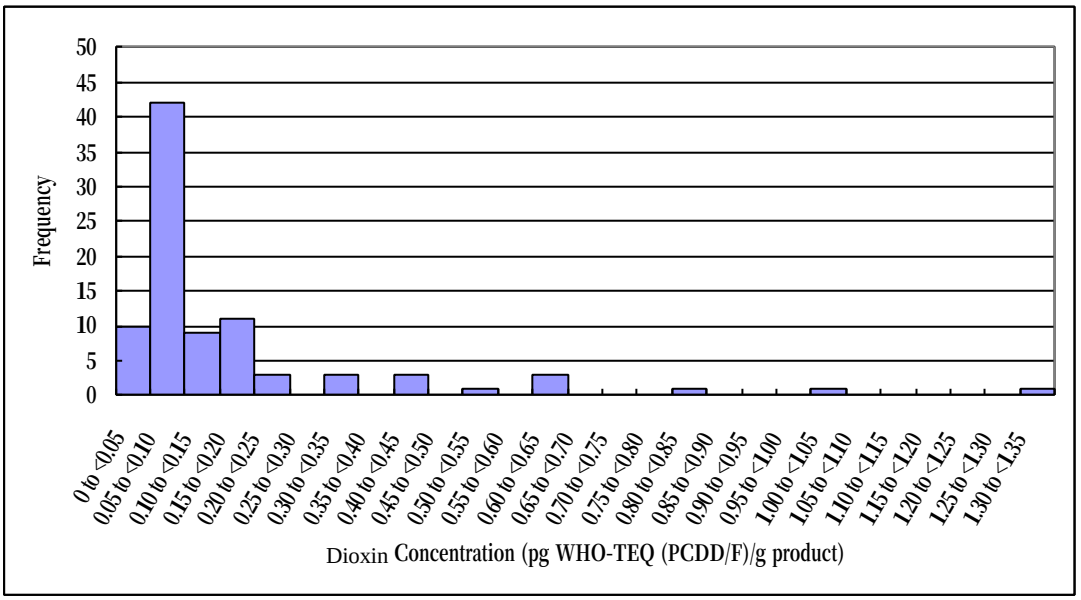
Group	Congener	Limit of Detection (Fat Basis) (pg/g)	Limit of Detection (Product Basis) (pg/g)
PCDDs	2,3,7,8-TetraCDD	0.2	0.04
	1,2,3,7,8-PentaCDD	0.2	0.04
	1,2,3,4,7,8-HexaCDD	0.5	0.1
	1,2,3,6,7,8-HexaCDD	0.5	0.1
	1,2,3,7,8,9-HexaCDD	0.5	0.1
	1,2,3,4,6,7,8,9-HeptaCDD	0.5	0.1
	OctaCDD	1	0.2
PCDFs	2,3,7,8-TetraCDF	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: Frequency 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: Frequency Distribution of Dioxin Concentrations in Meat and Meat Products

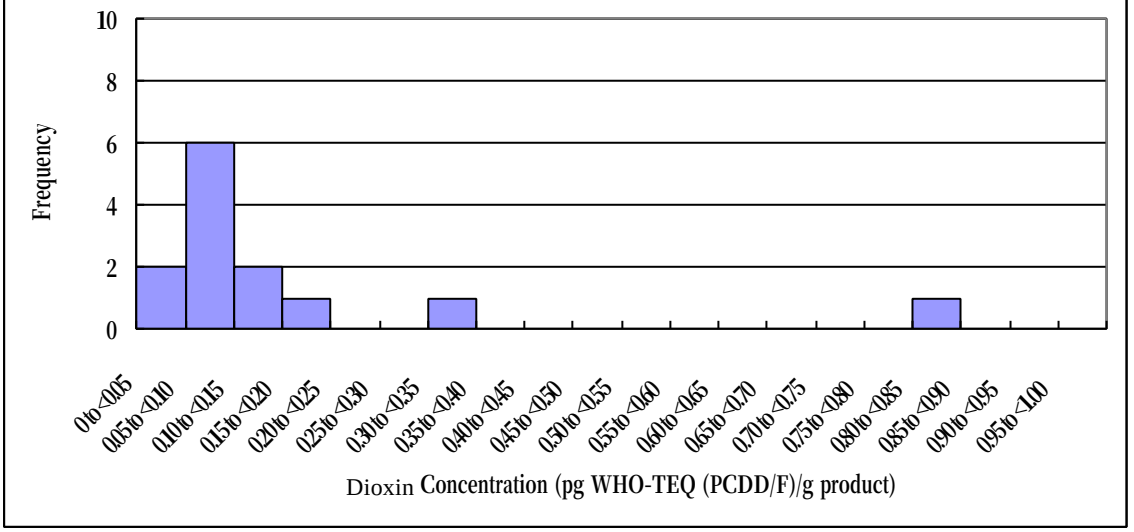


Figure 4.3: Frequency Distribution of Dioxin Concentrations in Poultry and Poultry Products

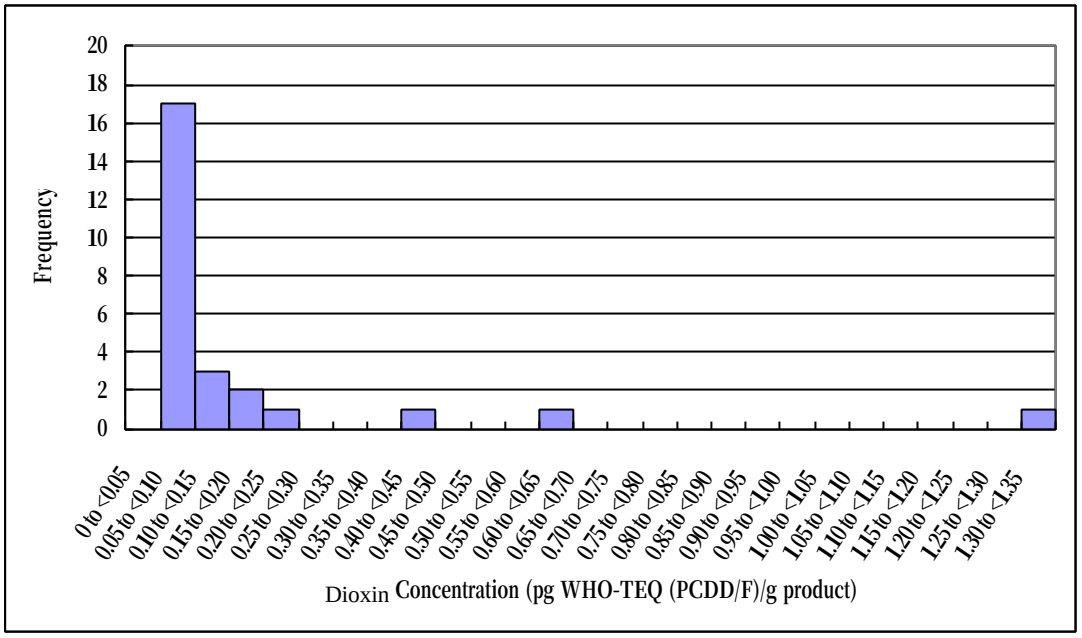


Figure 4.4: Frequency Distribution of Dioxin Concentrations in Milk and Milk Products

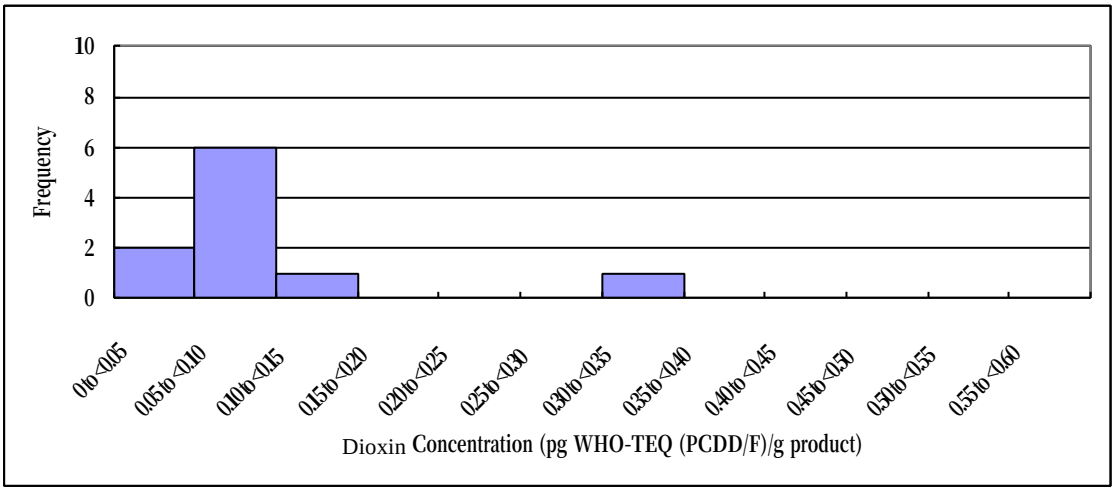


Figure 4.5: Frequency Distribution of Dioxin Concentrations in Seafood

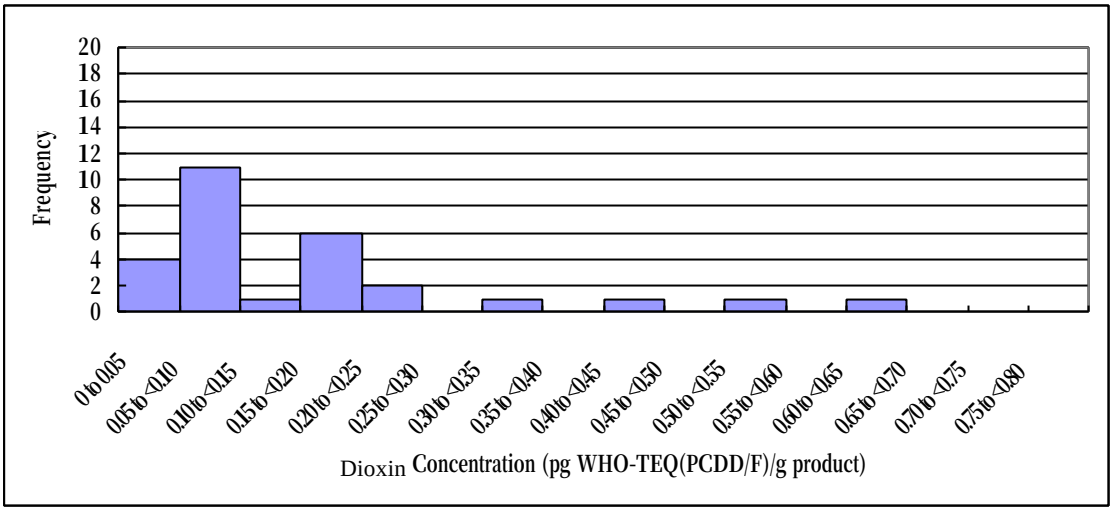
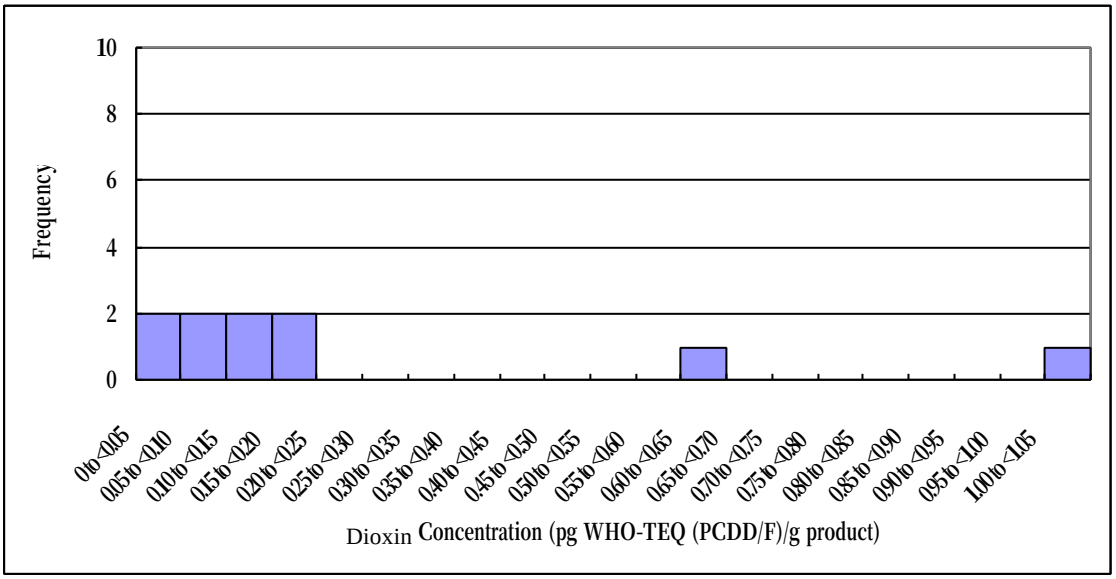


Figure 4.6: Frequency Distribution of Dioxin Concentrations in Eggs and Egg Products



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 (PCDD/F)/ 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
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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:

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

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›**	**-----**	**0.85›**

* 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 **0.85 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 **2.07 pg WHO-TEQ (PCDD/F)/ kg bw/ 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 6.1: 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	**0.85**

* 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].

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[12] U.S. Environmental Protection Agency. Questions and Answers about Dioxins; U.S. Environmental Protection Agency; July 2000.

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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