What We Have Achieved: An Introduction to a Case Study on Dioxins

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Abstract

Our achievements in a research project involving a case study on dioxins are presented by introducing the results. These achievements include the identification of emission sources, description of the dynamics in environmental media based on the congener-specific analysis and development of the integrated methods to evaluate and manage various types of risks due to dioxins. The risks to fetuses due to dioxins and how to manage the risks arising from chemical use are discussed.

1. Overview of the case study on dioxins

Our project, entitled, " Establishment of a scientific framework for the management of toxicity of chemicals based on environmental risk-benefit analysis," aims at providing the Japanese society with a scientific basis for devising an environmental policy. The project consists of three major subjects: 1) to evaluate the human health risks from chemical use, 2) to evaluate the ecological risks from chemical use, various types of exploitation and fishing, and 3) to balance the risks against the benefits of chemical use. We are aiming at the development of a methodology for evaluating risks and balancing the risks against the benefits, by carrying out case studies of chemicals such as dioxins, endocrine-disrupting chemicals, DDT, benzene, mercury and indoor air pollutants.

Referring to the case study on dioxins, our achievements since 1996 are presented here. An overview of the case study on dioxins is given in Figure 1. The term dioxins in this study refers to a family of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) which sometimes are abbreviated as PCDD/Fs. This case study covers four major issues: 1) exposure analysis, 2) risk evaluation, 3) identification of emission sources, and 4) a proposal of how to manage risks due to dioxins.

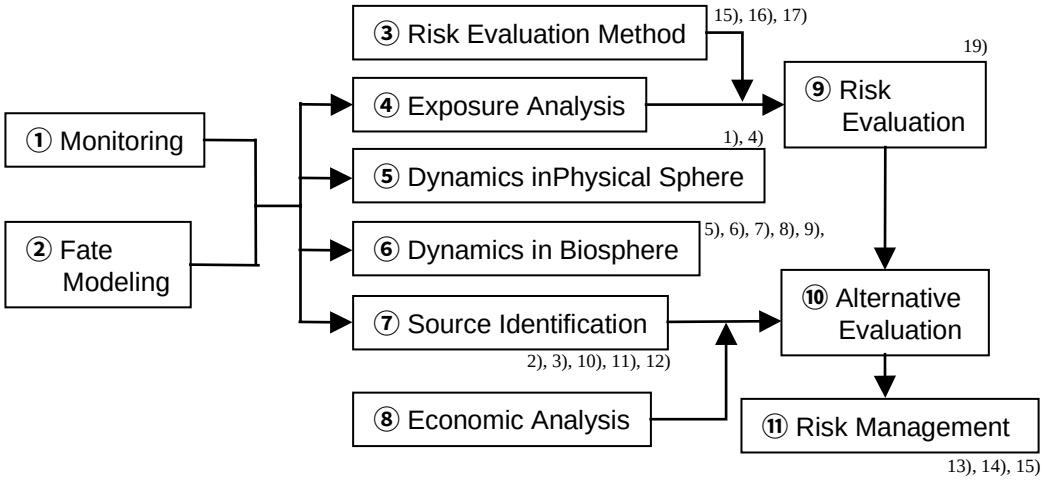

Figure 1 Overview of a Case Study on Dioxins.
-Congener Specific Analysis and Integrated Risk Evaluation-

2. Exposure and risk due to dioxins

First, the results of exposure analysis and the evaluation of the risk due to dioxins and the methodologies used for implementing them are summarized and presented.

2.1 Exposure analysis and a scenario

The magnitude of dioxin exposure via significant pathways was simulated for the general population (G), for local residents living near garbage incinerators (LR) and for heavy fish consumers (HFC). The monitored data published by governmental institutions and university researchers were used for G and for HFC, while models to estimate dioxin levels in various environmental media were formulated and used for LR. LR refers to humans who have lived, for thirty years, within a 1000m radius of a garbage incinerator emitting dioxins of a level that corresponds to the dioxin level in the soil within a 1000 m radius of the Shirotori Garbage Incinerator in Ibaraki Prefecture as reported by Miyata.[20] In addition, LRs are assumed to consume green vegetables affected by emissions from the incinerator. This estimate refers to the theoretical worst case, although there is no such thing as a worst case. In addition, an uncertainty analysis was conducted, the results of which are shown in Figure 2.[19]

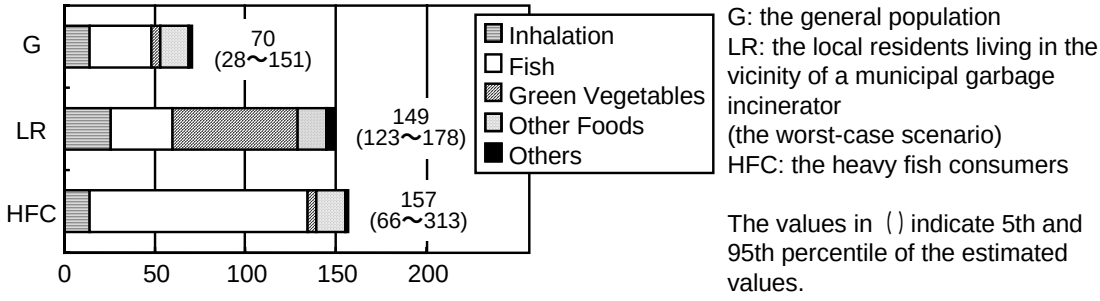

Figure 2 Lifetime Average Daily Dose of Dioxins [19]

2.2 Human health risk analysis using a dosimetry of daily intake

Based on the results of exposure analysis, human health risks were evaluated for four endpoints: cancer, reproductive dysfunction and endometriosis in adults and developmental neurologic abnormalities in terms of the development of cognitive ability in infants exposed to dioxins in utero and through breast-feeding. Our conclusions drawn from the risk analysis include: 1) the risk level of LRs is to some extent, but not significantly greater than, that of the general population. A percentage of HFCs is at more risk than LRs. 2) If it is assumed, as it is done by the Japanese government and the WHO for establishing the value of tolerable daily intake (TDI), that dioxins do not act as initiators but, rather, as promoters in the process of carcinogenic action, then the risk due to dioxins for the three population categories is negligible. However, if it is more appropriately assumed that dioxins act as neither initiators nor promoters but rather activators, as is claimed by the USEPA[21)], the excess cancer risk for a lifetime due to dioxins can be calculated to be, on average, 1.3×10^{-4}, 2.7×10^{-4}, 2.9×10^{-4} for the general population, for LRs and for HFCs, respectively. 3) The estimated margin of safety (MOE) values for cancer, reproductive dysfunction and endometriosis were sufficiently high to guarantee safety. 4) The estimated MOE values for neurobehavioral effects on infants and fetuses suggested that dioxins may to some extent pose risks to infants and fetuses, although the effects were not severe.[19)]

3. Risk evaluation for fetuses and infants

3.1 Risk to fetuses

Following the results in the previous section, we carried out more precise evaluation of risks to fetuses: 1) maternal body burden was used as a dosimetry of dioxins instead of daily intake, and 2) assumptions regarding toxicity were matched with those which the Japanese government used for establishing the TDI of 4pg/kg/day of dioxinlike compounds in terms of 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalents (TEQ) in 1999, where the dioxinlike compounds refer to dioxins and Co-PCBs. The TDI is based on the lowest-observed-adverse-effect level (LOAEL) from animal tests that identified the relationship between the maternal body burden of dioxins and the changes of immunological indicators and morphological abnormalities of genital organs in the fetus. Here, the no-observed-adverse-effect level (NOAEL) for the immunological effects for the fetus was assumed to be 27 TEQ ng/kg of body weight (bw kg), one third of the LOAEL value. In addition, it was assumed that the interhuman variability regarding the individual sensitivity to dioxins follows a log-normal distribution with the geometric standard deviation of 1.7, which corresponds to an assumption of a 3.16-fold uncertainty factor. We thought that a 3.16-fold uncertainty factor is reasonable, because the LOAEL is based on the effects for a sensitive subpopulation, in other words, the developing fetus and because the government used a tenfold uncertainty factor for combined uncertainties arising from individual variability and estimation of NOAEL from LOAEL.

The geometric mean and geometric standard deviation of dioxin maternal body burden were calculated, respectively, to be 3.66 WHO(1997)-TEQ-ng/bw kg and 1.47 for PCDD/Fs and 6.29 TEQ-ng/bw kg and 1.45 for the sum of PCDD/Fs and Co-PCBs based on the data of dioxin levels in the breast milk of the 415 mothers monitored.[22)]

Subsequently, the distribution of maternal body burden was combined with the distribution of individual sensitivity and a Monte Carlo simulation was performed. The results indicate that the risk was calculated to be 1×10^{-3} for PCDD/Fs and 2×10^{-2} for the sum of PCDD/Fs and Co-PCBs, where the risk is defined as the probability that the maternal body burden exceeds the NOAEL value. The details of the

process for evaluating the risk to fetuses are illustrated in Figure 3 and 4.

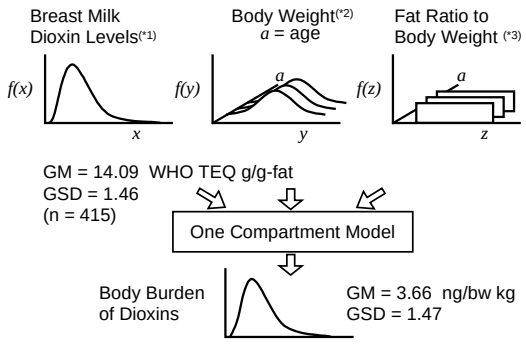

Figure 3 Estimation of Dioxin Body Burden

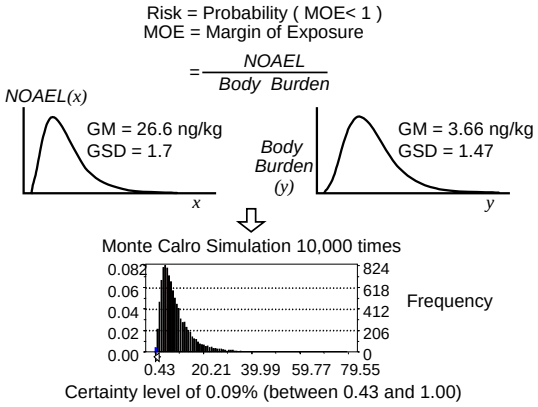

Figure 4 Risk Calculation Considering Uncertainties

3.2 Risks to infants due to breast-feeding

Many papers contain warnings of the possible adverse immunological effects due to dioxins in breast milk. However, the dose-response relationship has not been well established in any of the reports, and generally the postnatal effects are less than the prenatal ones except in terms of cancer risk.[23] Therefore, it may be valid to assume that the magnitude of the immunological risks to infants due to breast-feeding is not greater than that for the fetus. In addition, it was assumed that the excess cancer risk due to dioxins in breast milk is similar to the cancer risks estimated in Canada of 1.5×10^{-4} due to PCDD/Fs, because the level of PCDD/Fs in Japanese breast milk is similar to that in Canadian breast milk. In addition, it is noted that the excess cancer risk associated with exposure to 15 organochlorines, such as α-chlordane, γ-chlordane, chlordane metabolite-oxychlordane, p,p'-DDE, o,p'-DDT, p,p'-DDT, dieldrin, heptachlor epoxide, α-hexachlorobenzene, β-hexachlorocyclohexane, γ-hexachlorocyclohexane, hexachlorocyclohexane, Mirex, PCBs and PCDD/Fs in Canadian breast milk was calculated to be 2×10^{-4}.[23]

4. Identification of dioxin emission sources

4.1 Principal component analysis

In order to reduce emission, it is of utmost importance to make an inventory of emission sources. Recognizing that this task is not only important but also difficult in the case of dioxins, which are unintentionally produced and related to a broad spectrum of activities, we have been studying this subject for a decade. Principal component analysis was used to identify the possible major origins of dioxins. A family of dioxins has numerous congeners. In the case of PCDDs and PCDFs with four or more chlorines, the number of congeners is 136. Among them, 17 are generally determined, because only these 17 congeners are thought to be toxic whereas the others nontoxic. However, we have determined 87 of the 136 congeners, because we believed that we could obtain information about the origins of dioxins, based on the fact that the congener profile of dioxins differs depending on the origin.

Using principal component analysis, we extracted three major components, in some cases, four components including an unknown component. The first one was attributed to aerial deposition, the second to impurities of CNP, chloronitrophen, a herbicide for paddy fields, and the third to impurities of PCP, pentachlorophenol, also a herbicide for paddy fields. This revealed that impurities of outdated

herbicides still persist in sediments in Tokyo Bay, in Lake Shinji and Lake Kasumigaura, and their contribution is still significant.[2),3),10),11)] However, it did not directly indicate that both the herbicides contain the toxic congeners, because the analysis was performed with the aid of the congener profile of dioxins containing nontoxic congeners.

Although it was known that dioxin impurities are contained in CNP and PCP, no information has been published regarding the detection of toxic congeners in either of the herbicides manufactured in Japan. Recognizing that dioxin impurities in organochlorine agrochemicals, particularly in old ones, should be reexamined, thus we sought out old agrochemicals and determined their dioxin impurities. The results revealed that high concentrations of toxic dioxins were contained in both the herbicides.[12)] Based on the toxic dioxin concentration and the consumed amount of both the herbicides, the amounts of dioxins emitted in the paddy fields in terms of TEQ were estimated from 1955 for four decades. The estimates revealed the surprising fact that the amounts of dioxins of both the herbicides were more than fourfold the total amounts of discharged from municipal garbage and industrial waste incinerators for the four decades. These results indicate that the general belief that the major dioxins originate from municipal garbage incinerators is a complete misconception. Considering that the governmental policy regarding dioxin pollution prevention has been devised based on this misconception, the matter is significant.

4.2 Multiple regression analysis

Next, the relative contribution of the three main components in the environment was determined with the aid of multiple regression analysis and the information regarding the toxic congener profile of the three main components. The analysis of the sediment core in Lake Shinji indicated that aerial deposition, CNP and PCP accounted for 14 %, 0 %, and 86 % of the total dioxins in terms of WHO-TEQ, respectively, in the sediment in 1965, and 33 %, 8 % and 59 %, respectively, in the sediment in 1993. The contribution of aerial deposition is greater than expected based on the results in section 4.1, probably because there are other sources of aerial deposition than municipal garbage incineration and industrial waste incineration. However, it should also be noted that this multiple regression analysis result is not decisive, because the data of the toxic congener profile are not conclusive due to the small number of samples.

4.3 Fate of dioxins applied in paddy fields

The results in the previous section indicate that the total amount of dioxins applied in paddy fields is estimated to be 590 kg- I-TEQ, of which 400 kg comes from PCP and 190 kg from CNP, and is comparable to or more than the 170 to 550 kg-I-TEQ (as estimated by Westing) of dioxins in the Agent Orange utilized by the US Air Force in Vietnam. Assuming that 590 kg of dioxins was applied over three million ha, the rice acreage in the 1970s, the average dioxin level in the soil of paddy fields is calculated to be about 100 pg-TEQ/g of soil. According to the recently monitored data by the Environment Agency, the average dioxin level in the soil of paddy fields was 50.8 pg-TEQ /g of soil, the maximum is 130 and the minimum is 15 (n=20). Thus, it is assumed that approximately half of the applied dioxins still remain in the paddy fields. The research on dioxins in the sediment core in Lake Shinji indicates that dioxins in paddy fields still flow into lakes, seas and rivers.[4)] Considering that the Japanese ingest major amounts of dioxins via fish consumption, dioxin transfer from paddy fields to fish plays a crucial role in human health risk.

Yoshida estimated the origins of dioxins in breast milk and their amounts in the year 1999 based on an analysis using a mathematical model: 10.3 pg-I-TEQ / g of fat from herbicide impurities and 6.2 pg/g of fat from incineration.

5. Congener-specific toxicity analysis

The information on the congener profile is also important for estimating the magnitude of toxic effects of dioxins in the human body, because the toxicity and the bioavailability of congeners of dioxins in the human body depend on their chemical and physical properties. It is generally accepted that the relative toxicity of congeners is represented by the Toxic Equivalency Factor (TEF) determined by the WHO. As the toxicity of dioxins is initiated through the same mechanism of action, the toxicity of a mixture of these compounds is thought to be additive, and is usually expressed in TEQ. The TEQ currently used is suitable to estimate the toxicity of the mixture of dioxin congeners, when the toxicity is controlled by the daily intake of dioxins. However, when the toxicity is controlled by the dioxin body burden, toxic equivalence factors must be transformed so that they can also reflect bioavailability. The bioavailability depends on the product of the half life and the absorption ratio at the digestive duct.

In order to estimate the bioavailability of congeners, the half life was estimated based on the published data of the half life of dioxins in the human body and the absorption ratio was assumed to be equal to that of dioxin congeners in breast milk in nursing infants. The product of the half life and absorption ratio of congeners relative to 2,3,7,8-TCDD is an indicator representing the relative bioavailability and here, is called the Yoshida-Nakanishi Factor (YNF). The values of YNF are shown in Table 1.

Table 1 The values of YNF

Congener	Abb reviation	YNF	Reference value			
			Absorption ratio %	Half life		
				Liem et al.[24]	Flesch-Janys et al.[25]	Van den Berg et al.[26]
2,3,7,8-TCDD	4D	**0.97**	0.97	6.2	5.2	5.8/7.1
1,2,3,7,8-PeCDD	5D	**1.37**	0.99	8.6	15.7	--
1,2,3,4,7,8-HxCDD	6Da	**3.00**	0.98	19.0	8.4	--
1,2,3,6,7,8-HxCDD	6Db	**2.05**	0.97	more than 70.0	13.1	3.5
1,2,3,7,8,9-HxCDD	6Dc	**1.32**	0.96	8.5	4.9	--
1,2,3,4,6,7,8-HpCDD	7D	**0.92**	0.86	6.6	3.7	3.2
OCDD	8D	**0.69**	0.76	5.6	6.7	5.7
2,3,7,8-TCDF	4F	**0.06**	0.97	0.4	--	--
1,2,3,7,8-PeCDF	5Fa	**0.14**	0.99	0.9	--	--
2,3,4,7,8-PeCDF	5Fb	**1.56**	0.98	9.9	19.6	4.7
1,2,3,4,7,8-HxCDF	6Fa	**0.89**	0.97	5.7	6.2	2.9
1,2,3,6,7,8-HxCDF	6Fb	**0.97**	0.97	6.2	6.0	3.5
1,2,3,7,8,9-HxCDF	6Fc	**ND**	0.95	--	--	--
2,3,4,6,7,8-HxCDF	6Fd	**0.37**	0.96	2.4	5.8	--
1,2,3,4,6,7,8-HpCDF	7Fa	**0.36**	0.87	2.6	3.0	6.5
1,2,3,4,7,8,9-HpCDF	7Fb	**0.52**	1.00	--	3.2	--
OCDF	8F	**0.03**	0.95	less than 0.2	--	1.8

Using the product of TEF and YNF instead of TEF, we were successful in relating the congener profile of dietary dioxins to that of dioxins in breast milk, as shown in Figure 5 (Hashimoto). First we estimated the yearly trends of the amounts of respective dioxin congeners in daily diet from 1977 to 1996, based on the report regarding dioxins in samples archived for total diet study.[27] Next, the body burden of

respective congeners in 1998 due to dietary intake for two decades was calculated, considering the YNF. Third, the congener profile of breast milk dioxins calculated on the basis of the body burden dioxins was compared to that of breast milk dioxins surveyed.[22] Without introducing the YNF, the congener profile of dioxins in breast milk cannot be explained by that of dioxins in diet.

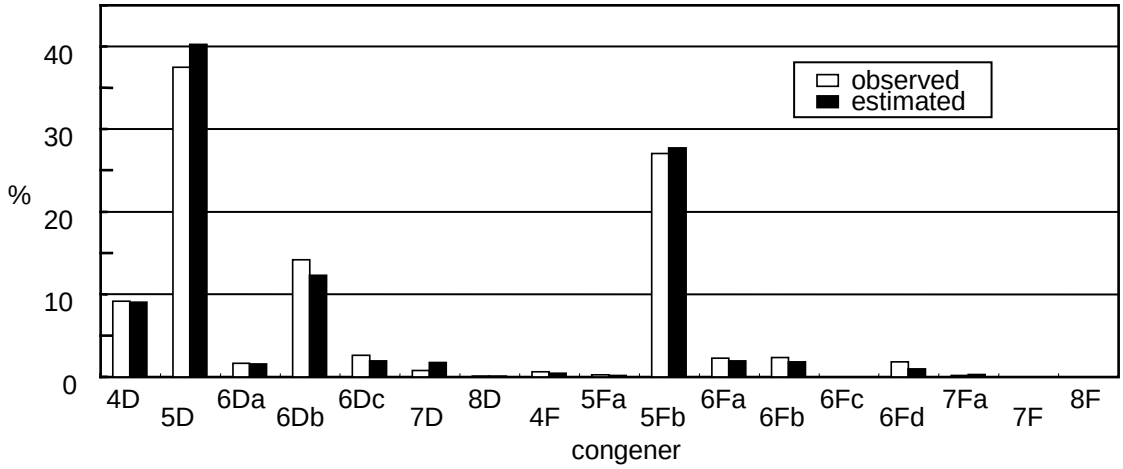

Table 5 Dioxin congener profile in breast milk (Hashimoto)

Table 1 indicates that generally, the YNF values of PCDDs are larger than those of PCDFs. Furthermore, HxCDDs and PeCDD pose a greater risk than estimated from their TEF values, while PCDFs other than 2,3,4,7,8-PeCDF pose a smaller risk than estimated from their TEF values. In addition, it should be noted that the YNF of 1,2,3,7,8-PeCDF is larger than that of 2,3,4,7,8-PeCDF by more than one order.

Yoshida estimated the contribution of origins to three major congeners in breast milk: the contribution of aerial deposition, CNP and PCP to 1,2,3,7,8-PeCDD was 21 %, 70 % and 9 %, respectively, that to 1,2,3,6,7,8-HxCDD was 53 %, 40 % and 7 % and that to 2,3,4,7,8-PeCDF was 84 %, 8 % and 8%. Taking the YNF into consideration, the contribution of CNP to toxic effects is greater than that expected on the basis of the TEFs.

6. Risk Comparisons

Now we discuss our main oncern, in other words, how we should manage the risks arising due to dioxins. We have believed and stressed that the use of chemicals should be managed based on risk-benefit analysis. However, risk management should proceed in the following steps to the final risk-benefit analysis from a practical viewpoint: 1) Comparisons with other risks and a target risk level, 2) risk evaluation of alternatives to reduce risks, and 3) risk-benefit analysis or cost-effectiveness analysis.

6.1 A tool for risk comparisons - LLE

In the previous sections, risk is represented in terms of the probability of occurrence of an adverse event which is called an endpoint. However, using this method, it is difficult to compare risks of different endpoints. To overcome this difficulty, we proposed a method for evaluating various types of risks on a single scale, referred to as Loss of Life Expectancy (LLE),[15),16),17),18)] which will be discussed in more detail by M. Gamo in this Proceedings (The Proceedings of the 3rd International Workshop). Since what we are interested in is the magnitude of impact on public health, the risk should be represented by the product of the probability of an adverse event and the consequences of the adverse event. We

proposed that the consequences of the adverse event should be evaluated in terms of LLE. Thus various types of risks are represented in terms of LLE and can be compared to each other. In Table 2, the risks represented in terms of the probability of the specific endpoints and the LLE.

Table 2 Risk Comparisons

Receptors	Causes	Endpoint	Consequences (LLE in years)	Risk in terms of probability	RISK in terms of LLE (days)	Data source
Adults (Average general)	PCDD/Fs	Cancer	12.6 [18]	1.3×10^{-4}	0.6	Japan
Fetuses	PCDD/Fs	Immunological indicators		1×10^{-3}		Japan
Fetuses	PCDD/Fs+ Co-PCBs	Immunological indicators		2×10^{-2}		Japan
Breast-fed infants	PCDD/Fs+ Co-PCBs	Immunological indicators		less than the risk for fetuses		Japan
	PCDD/Fs	Cancer	12.6 [18]	1.5×10^{-4}	0.6	Canada
	15 POPs	Cancer	12.6 [18]	2.0×10^{-4}	0.9	Canada
Fetuses	Methyl mercury	Neurological effects	1.85 [15]	3×10^{-3}	2.0	Japan
Infants	Not breast-feeding	Mortality	70	2.6×10^{-3}	67.1 [32]	Great Britain

6.2 Comparisons with other similar risks

Risk management starts with risk comparisons. Two examples of calculated risks are introduced for risk comparisons. The first example is the excess cancer risk due to chlorination byproducts in tap water: the risk was estimated to be 1.2×10^{-4} in Kanamachi Water Works in Tokyo in the latter half of the 1980s [16] and on average 1.1×10^{-4} in the 1980s in the United States.[28] We can understand that the adult cancer risk due to PCDD/Fs for the average general population of 1.3×10^{-4} is similar to the risk due to the consumption of tap water.

The second one is the risk to fetuses due to methylmercury. There are striking similarities between dioxins and methylmercury. Both chemicals are mainly ingested via fish consumption and fetuses exposed transplacentally represent a sensitive subpopulation. NOAEL of 1.1 μg/kg/day was determined for methyl mercury from two human epidemiological studies. The endpoint is a combination of numerous childhood neurological effects, such as delayed walking and talking, neurological scores more than 3, mental disorders and seizures.[29] The following were assumed: the interhuman variation regarding individual sensitivity to dioxins follows a log-normal distribution with a geometric standard deviation of 1.7, the average Japanese daily intake of methylmercury is 0.16 μg/bw kg/day[30] with geometric standard deviation of 1.5.[31] The resultant risk was calculated to be 3×10^{-3}. This risk is between the risks due to PCDD/Fs and due to the sum of PCDD/Fs and Co-PCBs. Considering that the severity of the endpoint for the risk due to methylmercury is greater, the risks due to PCDD/Fs and Co-PCBs are comparable to or less than the risks due to methylmercury.

6.3 Setting targets for risk level to manage risks

To understand as the first step, the need for and extent of measures that must be taken for reducing the risks involved, it will be convenient if we can compare the risks in terms of criteria which are common and available to all activities. The criteria are tentative, which we refer to as targets, because

we do not think that all risks should be controlled based on the equal-risk principle. Here, we consider what should be appropriate as targets both to indicate the need for action and to evaluate the effects of the policies adopted. The equal-risk principle is fundamentally based on the concept that we should not inherently take any risks and should be free from any risks. However, as it is not practical to realize a risk-free society, a risk level which corresponds to risk-free or negligibly small from a practical viewpoint has been sought. Through long-term debates and trials, the level of excess cancer risk for a lifetime of one in a million or ten in a million has been established as being negligibly small, in other words, as being free from risk. In Japan, the level of cancer risk for a lifetime of ten in a million was used to establish criteria for cancer-causing chemicals in drinking water and for benzene in air.

Therefore, it is appropriate to set a target for the cancer risk level in drinking water and in air to limit the level to ten in a million. Next, we extend the concept of this target level to noncancer risks and risks from foods. We propose values of the target for noncancer risks and for the risks from foods listed in Table 3. The proposed target levels of noncancer risks were determined so that the LLE of the risks would correspond to the LLE of a cancer risk of ten in a million. The risk of 3×10^{-6} of a fatal disease, the consequences of which correspond to the LLE of 35 years, is the same as a risk of LLE of 0.04 days and is approximately equal to the cancer risk of ten in a million, 0.05 days in terms of the LLE.

Table 3. Targets for risk level

Media	Endpoint	Consequences (LLE, years)	Target risk level	
			Probability	LLE, days
Drinking water Ambient air	cancer	12.6	10^{-5}	0.05
	fatal	35	3×10^{-6}	0.04
	deadly	1	10^{-4}	0.04
	change in indicators	(less than) 1	(more than) 10^{-4}	
Food	cancer	12.6	10^{-4}	0.5
	fatal	35	3×10^{-5}	0.4
	deadly	1	10^{-3}	0.4
	change in indicators	(less than) 1	(more than) 10^{-3}	

The target levels for risks due to food consumption proposed are tenfold more than the respective target levels for drinking water and for ambient air, because it is more difficult to control risks arising due to food consumption than those due to drinking water consumption.

The endpoints which were chosen to establish the TDI of dioxinlike compounds are not directly related to a specific disease or type of injury, but are rather indications of the possible occurrence of immunological dysfunctions. Certainly, their clinical significance is not clear. In such a case, it is difficult to determine the target risk level. However, we propose here to set the target risk level at 10^{-4} and 10^{-3} due to drinking water and due to food, respectively, from the viewpoint of protection.

Comparisons of the risks estimated due to dioxins with targets listed in Table 3 indicates the following: 1) the cancer risk estimated on the basis of the activator-mechanism for adults is 1.3×10^{-4} and that for breast-fed infants is 1.5×10^{-4}, which exceeds by a small amount the target, though the cancer risk estimated on the basis of the promoter-mechanism is nearly zero, and 2) the immunological risk to fetuses due to PCDD/Fs is close to the target of 10^{-3}, while that due to the sum of PCDD/Fs and Co-PCBs exceeds it.

Since the target is generally determined for the risk due to a single chemical, application of this value to the risk due to a mixture of PCDD/Fs and Co-PCBs is disputable. However, taking a

conservative standpoint, we thought that the risk due to the sum of PCDD/Fs and Co-PCBs should be compared with the target of 10^{-3}. Therefore, it is concluded that the risks due to dioxinlike compounds should be reduced.

7. Evaluation of alternatives

Next, the effectiveness of the following risk reduction alternatives were evaluated: 1) not to breast-feed, 2) to regulate dioxins in food and 3) to regulate dioxin emission from municipal garbage incinerators.

First, whether the avoidance of breast-feeding is appropriate was examined from the perspective of risk reduction. The risk as a result of avoiding breast-feeding was estimated, subsequently the risk are compared with the risks due to breast-feeding estimated in section 3.2. Rogan et al. estimated the increase in life expectancy attributable to breast-feeding to be 67.1 days,[32] based on an intervention study in Great Britain which is the only large data set that includes sufficient details of mortality, infant feeding and covariables to enable a reasonable estimate of the adjusted effect of breast-feeding on mortality in developed countries. This estimate was interpreted here as the minimum risk arising from not breast-feeding, because possible beneficial effects other than a decrease in mortality due to breast-feeding were not considered. The risk due to the avoidance of breast-feeding is at least tenfold greater than the risks due to breast-feeding. Although there is no room here to discuss the evaluation of the policy that regulates dioxin levels of food, in short, the policy is inappropriate for the same reason.

Regarding the regulation of dioxin emission from municipal incinerators, the cost per life-year saved for urgent countermeasures directed by the Ministry of Health and Welfare was calculated to be 79 million yen and this policy is judged to be appropriate . On the other hand, the cost per life-year saved for the final program directed by the MHW is 540 million yen and the final program is judged to be inappropriate in terms of cost-effectiveness.[13),14)]

The temporal trends of the dioxin body burden of a 27-year-old woman were estimated from 1970. Although the estimation failed to reproduce the peak in the beginning of the 1970s, it well reproduced the decreasing tendency after the middle of the 1970s. Using this model, the effects of countermeasures were evaluated. Assuming that dioxin emission from incinerators decreased to one-fifth the current level in 1999, it was estimated that the dioxin levels in breast milk will be one-half the current levels 17 years after 1999, and that the contribution of the herbicide impurities will increase to 79%. If the emission decreased to one-tenth in 1999, the dioxin levels in breast milk will decrease by one-half after 15 years. The contribution of herbicide impurities will increase to 81%.

These results indicate that even though strict regulations were enforced against emissions from incinerators, the effects on reducing human risks are small and it would be a long time before the effects will be felt. This is why the current dioxin pollution is mainly a residual from the past and the major exposure pathway is fish consumption. In such a case, we must seek more cost-effective ways to reduce risks, even though the risk level is higher than target levels. Considering exposure levels and pathways, we must spend more time before making a decision on which way to proceed in terms of formulating the final program for municipal incinerators.

Furthermore, it must be noted that many issues remain to be investigated. In addition, a study of measures for reducing the effects of dioxins in paddy fields has not yet been undertaken. In addition, measures to treat garbage and wastes other than incineration have not been investigated.

8. The need for a study for identification of possible emission sources

Lastly, I would like to stress the importance of identifying emission sources in risk management. We found that the impurities in the two herbicides were the largest sources of dioxin emissions. Our research regarding the sediment core in Lake Shinji suggests that there may have been other emission sources in the past which are still unknown. We would like to point out that other than our work, sincere and thorough investigations to determine possible emission sources have not yet been conducted and that scientific methodology to locate and identify emission sources based on the monitored data remains undeveloped. Our research project has aimed at the development of such methodologies, not only in the case of dioxins but also in other cases, such as for benzene by Fushimi and Kajihara and for ethylene by Okazaki.

Until now, I have discussed problems related to PCDD/Fs of dioxin-like compounds; however, the study of Co-PCBs may be more important, considering that approximately 60 % of the average Japanese dietary intake in terms of TEQ is Co-PCBs and the remaining portion is PCDD/Fs. Also, with regard to Co-PCBs, it is of utmost importance to identify its possible sources. By analyzing the congener profile, Masunaga estimates that 50-60 % of CoPCBs originate from incineration and remaining portion from manufactured PCB.

I have discussed our achievements regarding dioxins from the perspective of human health risk evaluation. Our research project has also yielded results in the study of ecological effects due to dioxins. For instance, Kwan studied the relationship between dioxin accumulation in aquatic organisms and their positions in the food web, and Iseki studied the effects on Common Cormorants, Phalacrocorax carbo.

Furthermore, Nakamaru has carried out a advanced study on ecological effects due to DDT in which a new and original methodology to evaluate the ecological risk was developed.

Our research project will be completed within the next year. A more comprehensive report will be presented at the Workshop held in 2001.

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9. References

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