Executive Summary

1. Introduction

1,4-Dioxane, a synthesized organic compound, is used widely as a solvent for extraction, purification, and reaction. It is produced mainly in Japan, the USA, and Germany. Worldwide production in 1995 was an estimated 8,000 to 10,000 t. Japanese production has been around 4,500 t for the past several years, representing half of the worldwide production. Emission sources other than environmental discharge due to production and use may include by-production due to production of some types of surfactants (mainly alkyl ether sulfates: AES). There is uncertain evidence that this compound has carcinogenic effects in humans, but it is carcinogenic in animals. Therefore, the International Agency for Research on Cancer classifies this compound as “possibly carcinogen to humans (Group 2B),” and the US Environmental Protection Agency classifies it as a “probable human carcinogen (Group B2).”

Contamination with 1,4-dioxane is considered to be problematic mainly in the water environment on account of its physical properties. However, investigation by the Ministry of the Environment in 2001 detected 1,4-dioxane in the air at up to 1.2 μg/m^3. In March 2003, data on chemical emissions from establishments, published under requirements of the Pollutant Release and Transfer Register (PRTR) Law, revealed that 90% of emissions were released to the atmosphere.

1,4-Dioxane was detected at high concentrations in well water piped for tap water in Tokyo and Osaka prefectures in 2002, leading to suspension of the supply of drinking water. In 2003, during the revision of the World Health Organization (WHO) Guidelines for Drinking-water Quality, the reference concentration of 1,4-dioxane in tap water was determined to be 50 μg/L. This criterion was enforced in April 2004. The same guideline value was determined under the requirements for Monitoring Substances in the Environmental Quality Standards for Water Pollution.

Against this background, we quantitatively assessed the risk of 1,4-dioxane to the health of the general public in Japan. The characteristics of this assessment are discussed below.

From existing data, we estimated the exposure by oral, dermal, and inhalational routes. The concentration in detergents was measured and also used to estimate exposure. We assessed exposure among the general public (“general group”) in Japan and among neighbors of highly emitting establishments (“highly exposed group”). Exposure was estimated as the distribution reflecting individual differences, rather than as the mean or worst-case value. Therefore, we dealt qualitatively with variability and individual differences in parameters, including the concentration of 1,4-dioxane in each environmental medium and the uptake of medium, and performed a Monte Carlo simulation.

We assessed hazards from a cross-sectional review of previous hazard assessments and a review of the literature. This assessment enabled us to investigate mechanisms of hazard occurrence objectively and to

derive no observed adverse effect level (NOAEL).

The “margin of exposure” (MOE) is used for risk assessment. The MOE is calculated as the NOAEL divided by the estimated exposure. The MOE value is compared with the uncertainty factor for species or individual differences. MOE > uncertainty factor indicates that the risk is not at a level that is of concern. MOE < uncertainty factor indicates that the risk is at a level that is of concern and there is a need to take countermeasures.

In this assessment, we reviewed previous risk assessments, which showed that 1,4-dioxane has little influence on ecosystems. A series of comprehensive risk assessments by the Research Center for Chemical Risk Management excluded the human health risk of occupational exposure because the nature of the risk is different from that due to exposure through the general environment; therefore, we also excluded it from this assessment.

This risk assessment is based on information available as of July 2004, and could be updated when new data is obtained.

2. Emissions

1,4-Dioxane emission volumes from business establishments were summarized on the basis of PRTR data for the fiscal year (FY) 2001 and FY2002. Also, 1,4-dioxane emissions from nonindustrial production sources (emissions associated with the release of surfactants and from solid-waste landfill disposal sites) are explained.

In FY2001, the total amount of 1,4-dioxane emitted from notified establishments in categories applicable to the PRTR was 183.0 t, which accounted for 4% of the production volume for the year. In FY2002, the total amount of emissions was 248 t. However, statistics for 1,4-dioxane productions for the fiscal year 2002 had not been released at the time of writing, so the ratio to the production volume for 2002 is not known.

In both the FY2001 and FY2002, the total amount of emissions from the Hikari plant of the Takeda Pharmaceutical Company, Ltd. (Plant A) and the Shizuoka plant of the Daito Chemix Corporation (Plant B) accounted for approximately 70% of the nationwide total.

1,4-Dioxane in surfactants is a by-product of the sulfation of alcohol ethoxylate (AE), a step in the production of alkyl ether sulfate (AES), a major detergent component of shampoos and skin washes. Therefore, where detergents are used, 1,4-dioxane can be released from households. The PRTR surveys of 1,4-dioxane emissions do not include emissions from households. Because AES production data for the FY1995 to FY1998 were available, 1,4-dioxane byproduct volumes were calculated on the basis of these data.

When the results for FY1998 are taken as an example and the 1,4-dioxane concentrations in AES are

assumed to be 10, 50, 100, 200 and 500 mg/kg, the volumes of 1,4-dioxane produced as a byproduct are 0.7, 3.4, 6.9, 13.8,and 34.4 t, respectively. If similar assumptions are made, the corresponding ratios of the volume of 1,4-dioxane produced as a by-product to the total volume (183 t) of 1,4-dioxane released to the ambient air and to public water areas as reported in the PRTR data for FY2001 are 0.4, 1.9, 3.8, and 18.3%, respectively. Furthermore, the ratios of the volume of 1,4-dioxane produced as a by-product to the total 1,4-dioxane production volume for FY1998 (4294 t) were 0.0, 0.1, 0.2, 0.3, and 0.8%, respectively.

There is a study that suggests that 1,4-dioxane may be generated by a proportion of heat-treated waste plastics in landfill sites. However, sources of 1,4-dioxane that might be generated in solid-waste landfill disposal sites are poorly understood, and there is only fragmentary information on the concentrations of the compound in landfill. Therefore, the current detailed risk assessment does not include a quantitative assessment of this possibility.

3. Environmental Fate

3.1 Degradation

The main pathway for degradation of 1,4-dioxane is considered to be photochemical oxidation in the atmospheric environment, where the half-life is 15 to 36 hours. (The former figure is obtained by assuming that the rate constant for reaction with hydroxyl radicals is 26.4×10^{-12} cm^3/mol/sec and the concentration of hydroxyl radicals is 5×10^5 mol/cm^3; the latter figure is obtained by assuming a rate constant of 1.09×10^{-11} cm^3/mol/sec and a concentration of 5×10^6 mol/cm^3.)

On the other hand, it is known that the substance is stable to hydrolysis by water.

According to an investigation of the degradation of 1,4-dioxane by reaction with ozone to evaluate its removal at water purification plants, the rate constant for degradation was 0.32 mol/s. The half-life of 1,4-dioxane in water was calculated to be 60 h at an ozone concentration of 10–5 mol/L: no data are available on reactions with hydroxyl radicals in environmental waters.

A 2-week aerobic biodegradability test in accordance with the Japanese Chemical Substances Control Law (JCSCL) revealed a degradability of 0% when measured indirectly by biochemical oxygen demand (BOD) and 1% when measured directly by gas chromatography (GC), leading to its evaluation as non-biodegradable.

3.2 Bioaccumulation

A 6-week bioaccumulation test specified by JCSCL revealed the concentration ratio to be 0.2–0.6 and 0.3–0.7 at the preset concentration of 10 mg/L and 1 mg/L, respectively. On the basis of these results, the (former) Ministry of International Trade and Industry announced that 1,4-dioxane accumulates either not at all or only slightly. Moreover, bioaccumulation of 1,4-dioxane would be impossible owing to its high

hydrophilicity and low octanol/water partition coefficient (log KOW = –0.49 to –0.27).

3.3 Distribution

The Henry's law constant of 0.29 Pa·m^3/mol (2.86 × 10^{-6} atm·m^3/mol; experimental value at 20 °C) means that 1,4-dioxane is moderately volatile from water and tends to transit gradually from water to air. The half-lives for bodies of water with depths of 10 m, 10 cm, and 5 cm are 817 days, about 8 days, and about 4 days, respectively.

According to the fugacity model level III, it is estimated that if 1,4-dioxane is emitted into water, 99% or more of the total emitted will remain in waters. Furthermore, if it is emitted into the atmosphere, about 60% of the chemical this will move into waters and soils. If 1,4-dioxane is emitted into soil environment, 40% will move into waters.

Therefore, a large part of 1,4-dioxane will ultimately end up in water. The limited proportion of 1,4-dioxane in the air at steady state shows that the compound is decomposed in the air or shifted out of the system at a higher rate than out of water into the air.

Moreover, on the basis of the log K_{OW} value, it is estimated that most of the substance will not be adsorbed into soils, but will migrate readily from the soil into subsoil waters.

4. Results of Monitoring of Concentrations in Environment Media

According to a nationwide survey of 1,4-dioxane concentrations in the air conducted by the Ministry of the Environment in 2001, the substance was detected in 22 of 34 samples, with concentrations ranging from 0.015 to 1.2 μg/m^3, the 95 percentile value being 0.15 μg/m^3.

1,4-Dioxane concentrations in public waters were generally below 1 μg/L.

Surveys show that concentrations of 1,4-dioxane in industrial wastewaters are sometimes higher than in public water areas. 1,4-Dioxane was detected in concentrations of more than 100 μg/L in wastewater discharges from some textile, and chemical industry facilities, although the companies did not necessarily recognize the emission of 1,4-dioxane. This suggests underestimation of the amount emitted into waters in the results of the PRTR survey.

In a study of 1,4-dioxane removal rates at sewage-treatment plants, the rates showed a wide range of values from negative ones up to about 60%. This may result partially from the fact that retention time of water at STPs was not taken into consideration.

In a study of water-purification plants, no significant differences were found between tap water sources and purified water. When average concentrations of 1,4-dioxane were sampled, the concentrations varied from 0.05 to 3.9 μg/L, and the geometric mean was 0.26 μg/L. In advanced water-purification treatment plants, no clear differences were found between concentrations in the source water and the purified water. We therefore conclude that 1,4-dioxane is not removed at general water-purification treatment plants.

To investigate 1,4-dioxane concentrations in consumer products, we measured 1,4-dioxane concentrations in shampoos, kitchen detergents, and other detergent products. In products containing AES as a major ingredient, which are frequently claimed to contain 1,4-dioxane residues, the concentrations of 1,4-dioxane ranged from below the detectable limit (< 5 mg/L) at the lowest to 51 mg/L at the highest.

Two studies measured the concentrations of 1,4-dioxane in foods. The concentrations in all food samples were below the detection limit of 0.01 ppm (mg/kg).

Monitoring indicates that air (inhalation), drinking water (oral), and detergent products (inhalation and dermal) should be considered as routes of exposure. Foods as a route of exposure can be neglected. This is summarized in Figure 1.

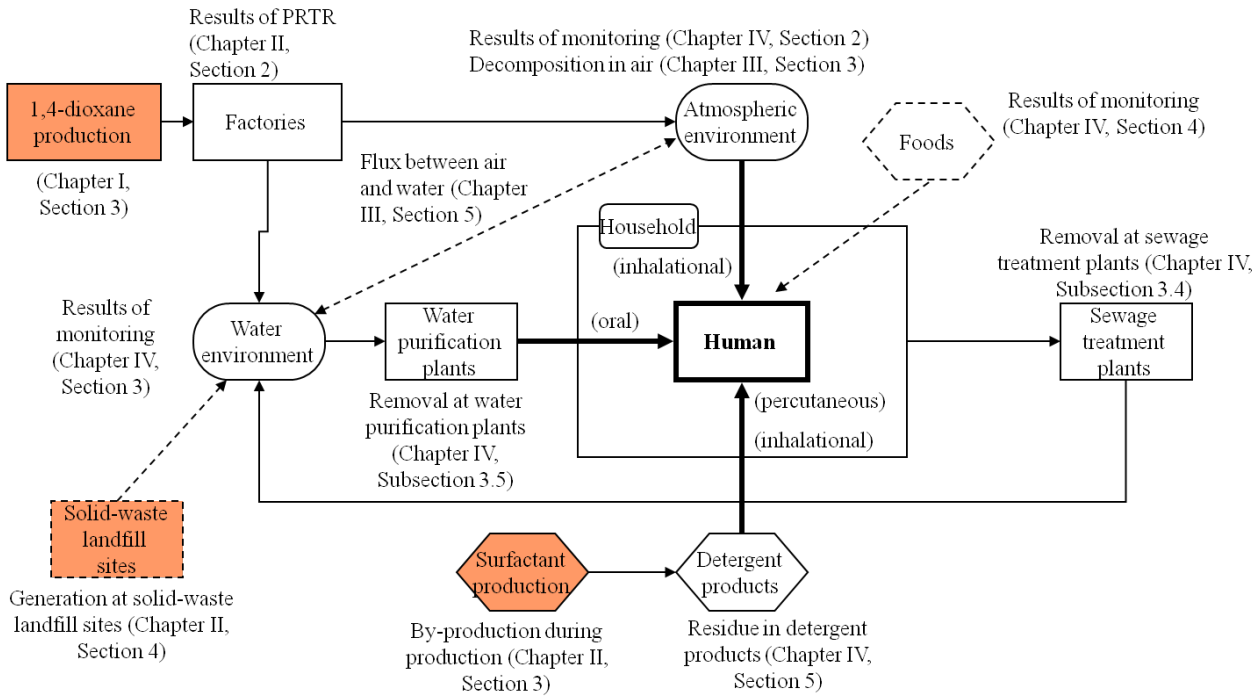

Fig. 1 Summarized scenario of exposure (exposure media and sources)

1) Arrows show the flow of 1,4-dioxane.

2) Bold arrows represent routes of exposure assessed in this study.

3) Dotted arrows represent routes of exposure not assessed in this study (“foods” as exposure medium, “solid-waste landfill sites” as sources, and amount of 1,4-dioxane vaporized from water into air).

4) Shaded boxes represent sources of 1,4-dioxane (incl. “1,4-dioxane production”).

5) The routes of exposure due to the use of detergent products are expressed as both “inhalation” and “dermal” because, for example, using shampoo may cause inhalation of vaporized 1,4-dioxane and absorption through the scalp (see Section 3 in Chapter V).

6) The chapters and sections or subsections under which each theme is described are indicated in parentheses.

5. Exposure Assessment

In assessing exposures of the general population, distributions of individual exposures were estimated for various exposure media and intake routes, and these are presented in terms of per-body weight per-day units. Exposure distributions were estimated by using a Monte Carlo simulation, assuming distributions for 1,4-dioxane concentrations in various media (drinking water, detergent products, and atmosphere) and for other parameters (e.g., human body weight).

For 1,4-dioxane concentrations in drinking water, a log-normal distribution was assumed in which the geometric mean and standard deviation were defined as 0.26 μg/L and 2.8, respectively. Similarly, for 1,4-dioxane concentrations in detergent products, a log-normal distribution was assumed in which geometric mean and standard deviation were set at 8.8 mg/kg, and 2.8, respectively. For both drinking water and detergent products, the geometric mean and geometric standard deviation were calculated from the monitoring data given in Chapter IV. For atmospheric concentrations, a log-normal distribution with a geometric mean of 0.01μg/m^3 and a geometric standard deviation of 3.3 of the estimated annual average concentration were applied; these values were obtained by using the National Institute of Advanced Industrial Science and Technology's Atmospheric Dispersion Model for Exposure and Risk Assessment (AIST–ADMER).

Calculations made by using, as input values, the quantities given in the PRTR summary for emissions of 1,4-dioxane into the atmosphere for FY2001 resulted in estimated atmospheric concentrations of the substance that were less than the actual measured concentrations. Therefore, in assessing exposure, the emission values were adjusted upward to maintain consistency between the estimated concentrations and measured values: the elevated emission values were used as input values and the resulting estimated concentrations were used in the assessment.

The examination of the estimated exposure distribution for each intake route showed that the exposure level for the sum of oral and dermal exposure and that for inhalation were approximately equivalent. The examination for each exposure medium showed that the exposure resulting from the use of detergent products is found to exceed those from drinking water and from the atmosphere. Table 1 shows the resulting estimated exposure distributions.

Table 1 Summary of exposure assessment in general population [μg/kg/day]

Mean	Median	S.D.[1)]	Percentiles		Exposure media		Routes of intake	Mean	Median	S.D.[1)]	Percentiles	
			5%	95%							5%	95%
0.0072	0.0031	0.014	0.00039	0.025	Air	→	Inhalational	0.023	0.013	0.035	0.00027	0.072
0.025	0.015	0.036	0.0030	0.077	Detergent products	→	Oral [2)]	0.024	0.013	0.039	0.0025	0.079
0.015	0.0052	0.035	0.00050	0.056	Drinking water	→	Dermal [2)]					

1) S.D. = standard deviation

2) Oral and dermal exposure was estimated as total.

Next, for high-exposure groups, the atmospheric concentrations near the high-emission source establishments A and B were estimated by using the Ministry of Economy, Trade, and Industry's Low-Rise Industrial Source Dispersion Model (METI-LIS). For this estimation, meteorological data are required. Two candidate meteorological stations for supplying input data were available for each of the facilities: the Shimomatsu and Yanai stations for Establishment A and the Fukuda and Omaezaki stations for Establishment B. However, insufficient data were available to permit the selection of one of the stations in each case, and therefore data from both the stations were input for each facility to make estimates. Similarly, two cases were assumed in terms of the height of the exhaust vent (5 m and 10 m), and the height data were input to make corresponding estimates. The results are shown in Tables 2 and 3.

Table 2 Summary of METI-LIS estimates (Establishment A)

Assumed emission height	Kudamatsu monitoring station		Yanai monitoring station	
Estimate	5 m	10 m	5 m	10 m
Max. concentration [1)] [μg/m^3]	855	512	551	223
Max. concentration in residential area off-site [μg/m^3]	8.6	5.1	275	111

Table 3 Summary of METI-LIS estimates (Establishment B)

Assumed emission height	Fukuda monitoring station		Omaezaki monitoring station	
Estimate	5 m	10 m	5 m	10 m
Max. concentration [μg/m^3]	294	75	72	15
Max. concentration in residential area off-site [μg/m^3]	15	3.7	3.6	1.5

When the highest concentrations for off-site residential areas are converted into exposure levels per day per body weight, assuming that the body weight is 50 kg, and the air inhalation rate is 15 m^3/day, the resulting values are 1.5–82.5 μg/kg/day near Establishment A and 0.45–4.5 μg/kg/day near Establishment B. These values are considerably higher than the estimated exposure levels per day per body weight shown in Table 1.

6. Hazard Assessment

Comprehensive hazard assessments of 1,4-dioxane have been conducted by the World Health Organization (WHO), the European Union (EU), the Austrian government, the US government, the Gesellschaft Deutscher Chemiker (GDCh), and the Ministry of Economy, Trade, and Industry of Japan. Also, carcinogenicity assessments have been conducted by the US government, the German government, and the WHO. In addition to these assessments, the hazardous effects of 1,4-dioxane have been assessed by several advisory bodies in Japan: the Water Quality Management Committee of the Life and Environment Tap-Water Section of the Health, Welfare, and Science Advisory Body assessed 1,4-dioxane when formulating tap-water quality reference values, and the Environment Reference Health Items Specialist Committee of the Water Environment Chapter of the Central Environment Health Items Advisory Body examined the substance when formulating guideline values for water quality environment health items.

Based on these materials and some literature sources on hazardous effects of 1,4-dioxane, we conducted an assessment of the hazards of 1,4-dioxane to human health.

6.1 Noncarcinogenic effects

In humans, short-term exposure at high concentrations of 1,4-dioxoane in atmospheric air causes mucosal irritation and hazardous effects on the kidney, liver, and brain that could lead to death in severe cases. Also, in acute toxicity studies in animals, either by the inhalation route or the drinking-water route, a short-term exposure caused hazardous effects on the kidneys and the liver.

In a study on rats or mice of the effects of continuous administration of 1,4-dioxane in drinking water, serious effects were observed on the nasal cavity, lung, liver, and kidney; the NOAEL for chronic toxicities was 10 mg/kg/day.

Although an animal study suggests that 1,4-dioxane may have neurotoxic effects, the study was not appropriate for detecting neurotoxicity and therefore the conclusion was suspended. Also, although a human study has suggested possible immunotoxicity, the study was not originally designed to investigate effects on the immune system and therefore the results cannot stand as evidence for the claim. Moreover, in an animal toxicity study, no findings that suggest effects on the immune system were presented. Therefore,

at present, the immunotoxicity of 1,4-dioxane should not be included in a risk assessment.

6.2 Carcinogenic effects

In human epidemiological research, no relationships between 1,4-dioxane and carcinogenicity have been identified.

In animal studies, administration of 1,4-dioxane in drinking water led to an increased number of liver tumors in rats, mice, and guinea pigs. In rats and mice, tumors in nasal cavity tissues have also been observed.

In a two-year inhalation chronic toxicity and carcinogenicity study, no effects were observed at 400 mg/m^3.

According to animal studies, after a high-dose administration, 1,4-dioxane temporarily accumulates in the liver and kidneys. It is assumed that metabolism from 1,4-dioxane to β-hydroxyethoxyacetic acid (HEAA) reaches a saturation point at high doses. The threshold for the saturation is estimated to occur at a plasma concentration of 100 μg/mL for rats. Also, in analysis using the physiologically based pharmacokinetics (PBPK) model, the concentration of 1,4-dioxane showed a nonlinear increase in the liver in the range where saturation of its metabolism is expected. Moreover, the rate of incidence of liver tumors is well correlated with concentrations of unchanged 1,4-dioxane in the liver, but not with concentrations of metabolites in the liver. Consequently, it is rational to conclude that the causative agent of the liver tumors produced by 1,4-dioxane is unchanged 1,4-dioxane.

It is generally believed that unchanged 1,4-dioxane is not genotoxic. On the other hand, genotoxicity of its metabolites is still a controversial subject and cannot be excluded. However, it is considered that is not a problem because there is a good correlation between the incidence of liver tumors and hepatic concentrations of the unchanged substance, and the metabolite suspected of carcinogenicity is believed to occur only in high-concentration exposures.

If the oncogenic mechanism of 1,4-dioxane is nongenotoxic, the most probable mechanism is that of a compensatory proliferative reactions to the cytotoxicity (or tissue toxicity), and many assessment organizations take this view. Although some test results show that no cell proliferation is induced by 1,4-dioxane, there is general agreement that cell proliferation is triggered by exposure to the chemical.

Although some study reports claim an absence of cell proliferation in oncogenesis in the nasal cavity tissues, acute suppurative inflammation has been observed, indicating the occurrence of local tissue damage. This tissue damage is thought to induce cell proliferative reactions. Also, it is suspected that the cause of the tissue damage is related to tissue-damaging reactions or a direct stimulus from drinking 1,4-dioxane-containing water.

Tumors occur in nasal cavity tissues in rats and mice, but not in guinea pigs. It is highly probable that occurrence of the tumors is related to the characteristic shapes of the nasal cavities of rats and mice, which

supports the idea that the tumors develop by local stimulation by 1,4-dioxane or from cytotoxic reactions.

Based on the above, the tumors in the nasal cavity tissues observed in animal studies are judged to originate from local exposures that are specific to rats and mice subjected to drinking-water administration. The tumors are believed to have developed from compensative cell proliferation, which is based on cell proliferation. However, tumors in nasal cavity tissues caused by 1,4-dioxane are considered not to be equivalent to human tumors, and therefore they are not used as an endpoint for human carcinogenicity assessment.

6.3 Quantitative hazard assessment

The liver tumors observed in animal studies are caused by carcinogenic promotion based on compensatory proliferation triggered by the cytotoxicity of 1,4-dioxane, and a similar mechanism may cause tumors in humans. On the basis of this mechanism, we adopt the position of assuming that there is a threshold level for carcinogenicity by 1,4-dioxane that can be adopted in evaluating its carcinogenicity hazard.

For an oral exposure NOAEL, a value of 10 mg/kg/day is judged to be appropriate from the toxicity study results and the metabolic profile.

For an inhalation exposure NOAEL, the ambient concentration of 400 mg/m^3 (111 pm) is used: this is the value at which no non-neoplastic changes or increases in tumors occurred in a carcinogenicity study involving inhalation administration. Although no inhalation toxicity studies subjected to animals other than this study have been conducted in test animals, the results were considered appropriate because concentrations of 1,4-dioxane in the target organ (the liver) at this exposure level are far below the carcinogenic threshold according to the PBPK model analysis, and also because when the exposure concentration is low, the area under the curve (AUC) of the human liver concentration curve is expected to be always lower than the expected values for rats or mice. Therefore, the value of 400 mg/m^3 is adjusted by the exposure time (7 hours/day), and by the number of days (5 days/week), and the resulting value of 83 mg/m^3 is used as the inhalation NOAEL in this assessment.

An uncertainty factor of 1000 is used (species difference: 10; individual difference: 10; neoplastic change: 10).

Noncarcinogenic effects following oral exposure are believed to be caused by cytotoxicity of the unchanged substance (1,4-dioxane), as is the case with carcinogenic effects. Therefore, an assessment of noncarcinogenic effects in humans should be completed by conducting an assessment for carcinogenic effects, and therefore in this assessment, a quantitative risk assessment for noncarcinogenic effects is not conducted.

7. Risk Assessment

In this chapter, the health risk to the Japanese population from exposure to 1,4-dioxane through various exposure routes is assessed on the basis of results of exposure assessment and hazard assessment. Health risk is assessed by comparing the MOE to the uncertainty factor, specifically for 1) inhalation exposure in the general population; 2) oral and dermal exposures in the general population; 3) inhalation exposure near Establishment A; and 4) inhalation exposure near Establishment B.

In this assessment, a value of 10 mg/kg/day was used as an oral exposure NOAEL, as indicated from the hazard assessment above, and 83 mg/m^3 was used as an inhalation exposure NOAEL. When the latter value is converted into a value in terms of body weight per day, it becomes 25 mg/kg/day. A value of 1,000 was used as an uncertainty factor for both oral and inhalation exposures.

For inhalation exposure in the general population, the MOE is calculated as 350,000 using the 95th percentile (0.072 μg/kg/day). This greatly exceeds the uncertainty factor (1,000), and is interpreted as “the risk is not at a level that is of concern and no need to take countermeasures”.

In oral and dermal exposures in the general population, the MOE is calculated as 130,000 using the 95th percentile of oral and dermal exposure (0.079 μg/kg/day). This greatly exceeds the uncertainty factor (1,000), and is interpreted as “the risk is not at a level that is of concern and no need to take countermeasures”.

For the highly exposed groups near Establishments A and B, the highest dose in the off-site residential area was compared with the inhalation exposure NOAEL, since exposures by any other routes can be ignored, judged from the above calculations.

Near Establishment A, the highest exposure in the off-site residential area reaches its highest value of 275 μg/m^3 (Table 2) when the emission height is set at 5 m and meteorological data from the Yanai station are used: in this case, the MOE is calculated to be 300. Similarly, when the emission height is set at 10 m, and meteorological data from Yanai are used, the MOE is calculated by the same method to be 750. Therefore, if the meteorological conditions near Establishment A are similar to those around the Yanai station, the MOE is below the uncertainty factor of 1,000, irrespective of the emission height: from this, the risk is judged as “the risk is at a level that is of concern and there is a need to take countermeasures”.

In this article, we will not show specific countermeasures, but the following consideration may be done. To derive a conservative estimate, we selected meteorological conditions and emission heights that favored overestimation of exposure, and a high uncertainty factor. However, the MOE is not greatly different from (smaller than) the uncertainty factor. In addition, Establishment A has used 1,4-dioxane for several years (according to management), so the populace has been exposed for that time, whereas the animal study on which NOAEL was based assumed chronic or lifelong exposure. Therefore, immediate emergency measures are not necessary at Establishment A, and mid-term reduction measures will be enough to reduce

the risk.

On the other hand, for Establishment B, the highest concentration for the off-site residential area (15 μg/m^3) is obtained when the emission height is set at 5 m and meteorological data are taken for the Fukuda meteorological station. The MOE in this case is calculated to be 5,530. In this case, the MOE exceeds the uncertainty factor 1,000, from which it is judged as “the risk is not at a level that is of concern and no need to take countermeasures”.