JLCA NEWS ENGLISH EDITION SEP.2013

No.16

JLCA NEWS LETTER

Life-Cycle Assessment Society of Japan

LIME2

Life-cycle Impact assessment Method based on Endpoint modeling

Chapter 2 :
Characterization and Damage Evaluation Methods

2.4 Urban Air Pollution
2.5 Photochemical Oxidant
2.6 Toxic Chemicals (Human Toxicity)

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Chapter 2 Characterization and Damage Evaluation Methods

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Chapter Ⅱ

Characterization and Damage Evaluation Methods

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Chapter II

Characterization and Damage Evaluation Methods

2.4 Urban Air Pollution

Changes in LIME2
- Characterization factors not calculated under LIME1 were calculated.
- With regard to some substances, values were reviewed concerning "the increasing rate of the death rate and the disease rate at each endpoint per unit of pollutant concentration."
- Uncertainty assessment of damage factors was carried out.

2.4.1 What phenomenon is urban air pollution?

The atmosphere contains various substances. The volume of vapor (H_2O) greatly differs according to place and time. However, the composition of the substances other than vapor is almost the same up to 80 km above the ground. Concretely, nitrogen (N_2) and oxygen (O_2) occupy more than 98%, followed by carbon dioxide (CO_2), argon (Ar), etc. (see "Invariant components" in Table 2.4-1).

On the other hand, the concentrations of ozone (O_3), sulfur dioxide (SO_2), and other substances, whose volumes are slight, vary depending on the condition of the source and human activities (see "Variant components" in Table 2.4-1). If a trace of such a substance reaches a level of concentration harmful to human beings and organisms, this is called air pollution (Kawamura et al. 1988).

Air pollution has become more serious as human beings' urban activities and industrial activities have become brisker – especially, as consumption of fossil fuels has increased. For example, in the middle of the 20th century, serious air pollution caused much sufferering and many deaths all over the world. The smog in London in December 1952 was especially famous. Emissions of SO_2 and smoke dust derived from coal burning for manufacturing, heating, and cooking caused smog so serious as to obstruct the view, with the result that about 4,000 persons – mainly, infants and elderly persons – were dead.

In Japan, before World War II, sulfur oxide (SO_x) emitted from refining in Ashio and other copper mines caused damage to surrounding forests and farm products. Around World War II, burning of coal for manufacturing and heating produced smoke dust, causing air pollution.

From the second half of the 1950s, the energy source shifted from coal to petroleum, and many petrochemical complexes were constructed in seaside areas. When petroleum, which contains a lot of sulfur, was burnt, SO_x was emitted from petroleum and caused asthma in Yokkaichi City. From around 1970, nitrogen oxide (NO_x) and volatile organic compound (VOCs) emitted from automobiles and factories produced photochemical oxidant (main component is O_3) and caused photochemical smog.

Table 2.4-1: Composition of the atmosphere

	Substance		Volume	Volume
Invariant components	N_2		78.084±0.004[%]	—
	O_2		20.746±0.002[%]	—
	(CO_2)		0.033±0.001[%]	—
	Ar		0.934±0.001[%]	—
	Ne		—	18.18±0.04[ppm]
	He		—	5.24±0.004[ppm]
	Kr		—	1.14±0.01[ppm]
	Xe		—	0.087±0.001[ppm]
	H_2		—	0.5[ppm]
	CH_4		—	2[ppm]
	N_2O		—	0.5±0.1[ppm]
Variant components	O_3	Photoreaction	—	0～0.07[ppm](Summer) 0～0.02[ppm](Winter)
	SO_2	Factory, volcano, etc.	—	0～1 [ppm]
	NO_2	Factory, automobile, etc.	—	0～0.02
	CH_2O	Acidification of organic CH_4	—	Indetermination
	I_2	Manufacturing	—	Within 10^{-4} [g m^{-3}]
	NaCl	Sea salt	—	10^{-4}[g m^{-3}]
	NH_3	Manufacturing	—	0～Trace
	CO	Manufacturing	—	0～Trace
	H_2O	Evaporation	—	0～35[g m^{-3}]

(Source) Kawamura et al. (1988); data were altered.

After that, air pollution by SO_2 greatly improved because of the government's tightening of regulations and the industrial world's introduction of measures. On the other hand, with the progress of motorization, pollution of roadsides by nitrogen dioxide (NO_2) and diesel emission particles (DEP) have been regarded as problems. Another problem is that air pollution has occurred also by various hazardous chemical substances, such as nitrate salt and sulfate salt produced during long-distance transportation of SO_2 and NO_x as well as benzene, organochloride compounds (trichloroethylene, tetrachloroethylene, etc.), dioxin, etc.

As described above, the sources of air pollutants and the substances that cause air pollution have changed with changes in industries and energy sources, urbanization, and changes in lifestyle. Although some air pollution problems have been solved through efforts by the government, industries, citizens, etc., serious problems still remain. Therefore, it is necessary to prevent the impact of various substances that originate from various sources of emissions.

Under LIME, these air pollution problems were dealt with in "urban air pollution," the impact category covered by this section. However, O_3 is covered by the impact category "photochemical oxidant" (see Section 2.5). Various hazardous chemical substances are covered by the impact categories "hazardous chemical substances" and "eco-toxicity" (see Sections 2.6 and 2.7).

Table 2.4-2 shows the above-described types of air pollution and the measures for the above-described impact categories under LIME. In the table, "primary pollutants" means pollutants directly emitted from factories, automobiles, etc. In addition, "secondary pollutants" means pollutants produced from primary pollutants through chemical reaction.

Table 2.4-2: Types of air pollution and impact categories under LIME

Types of air pollution		Targets of LIME		
		Emitted substances	Atmospheric substances	Impact categories
Pollution by primary pollutants	Pollution by emission of NO_2	NO_2	NO_2	Urban air pollution (this section)
	Pollution by emission of SO_2	SO_2	SO_2	
	Pollution by emission of primary particles*	Primary particles	Primary particles	
Pollution by secondary pollutants	Pollution by O_3 due to primary pollutant emissions (VOCs and NOx)	VOCs	O_3	Photochemical oxidant (Section 2.5)
	Pollution by nitrate salt due to primary pollutant emissions (NO_2)	NO_2	nitrate	Urban air pollution (this section)
	Pollution by sulfate salt due to primary pollutant emissions (SO_2)	SO_2	Sulfate	
Other	Pollution due to emission of hazardous chemical substances	Hazardous chemical substances	Hazardous chemical substances	Hazardous chemical substances (Section 2.6) Eco-toxicity (Section 2.7)

† Particles directly emitted from sources of emissions, such as smoke dust and diesel emission particles

(1) Cause of urban air pollution

After the emission of a primary pollutant, it is transported by the wind (advection), spread out, and chemically changed into a secondary pollutant. In addition, a part of it is removed from the atmosphere through deposition. If human beings and organisms are exposed to these pollutants, they may receive harmful effect, depending on the amount of exposure.

Figure 2.4-1 shows the causation of such urban air pollution (however, for O_3, see Section 2.5).

The first half of the causation will be described herein, while the second half will be described next in (2).

The main physical and chemical phenomena (advection, diffusion, chemical change, deposition, etc.) of air pollution differ according to type and, as a result, the scale of the space over which air pollution spreads.

Primary pollutants spread over the source of emissions and the surroundings through advection and diffusion by the wind.

On the other hand, secondary pollutants are formed though chemical changes, etc. under the influence of insolation, humidity, etc. Because a chemical change takes time, the concentration of secondary pollutants does not necessarily become high around the source of emissions, and the pollution area spreads more widely.

Below, the phenomenon and space scale of air pollution will be described concerning both primary pollutants and secondary pollutants.

a Pollution by primary pollutants

Primary pollutants include NO_x, SO_2, and particulate matter.

NO_x is produced through oxidization of burning nitrogen compounds and nitrogen in the atmosphere during the incineration process at factories, automobiles, etc. There are two

types of NO_x: nitrogen dioxide (NO_2) and nitrogen monoxide (NO). Although Most of NO_x is NO at the time of emission, NO is relatively quickly oxidized into NO_2.

SO_2 is created through oxidation of burning sulfur during the incineration process at factories, etc.

Primary particles include soot created during the incineration process at factories, etc. and DEP created during the combustion of diesel engines (there are also secondary particles, which will be described in b).

After the emission, the above-mentioned primary particles move by the wind and spread horizontally and vertically. When the surface of the ground becomes warmer because of insolation, an air parcel rises, it becomes easier for convection to occur (it becomes easier to be unstable), and it become easier for substances to be mixed with upper and lower substances and diffuse.

As a result of such advection and diffusion, pollution extends from a local scale around the source of emission (a scale of up to about 200 m) to an urban scale (a scale of up to about 20 km).

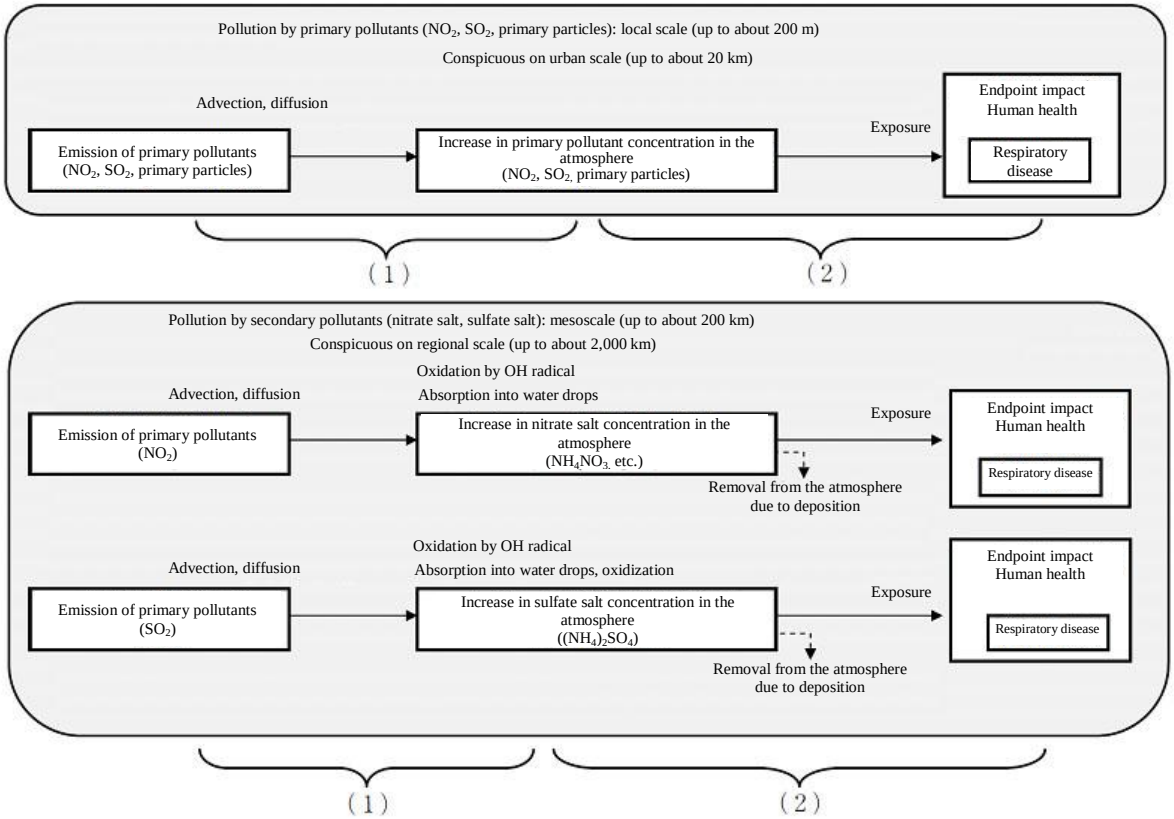


Figure 2.4-1: Cause of urban air pollution

(1) and (2) in the figure correspond to headings in the main text.
(Source: prepared with reference for Ohara et al. (1997); Kasahara (1994))

b Pollution by secondary pollutants

Secondary pollutants include O_3 produced from VOCs and NO_x, and nitrate and sulfate salt produced from NO_x and SO_2. Nitrate and sulfate are described herein (for O_3, see Section 2.5).

Although NO_x and SO_2 are gaseous substances, they are oxidized by OH radical (for the meaning of radical, see Column 2.5-2 "What is a radical"), pass through various processes (chemical reaction with other pollutants, solution in drops of water, etc.), and are changed into particle nitrate (such as NH_4NO_3) and particle sulfate (such as $(NH_4)_2SO_4$) respectively (particles created from emitted substances through chemical changes or phase changes in this way are called secondary particles).

Such pollution by nitrate and sulfate extends to mesoscale (up to about 200 km) and regional scale (up to about 2,000 km). The extent of pollution by nitrate is wider than that by sulfate . This is because the oxidation speed of SO_2 by OH radical is about one-tenth that of NO_2, SO_2 remains gaseous for a long time, and is oxidized into sulfate salt after long-distance transportation.

Nitrate salt and sulfate salt cause oxidization when depositing from the atmosphere to soil and water areas. Because this phenomenon is treated separately in the impact category "acidification," it is excluded from this impact category.

(2) Endpoints for urban air pollution

After air pollutants invade a human being's respiratory system (nasal cavity - pharynx - trachea - bronchi - small bronchi - lung alveoli), they cause deposition, absorption, and injury at various parts according to physical and chemical characteristics.

a Gaseous substances (NO_2, SO_2)

SO_2, a gaseous and highly water-soluble substance, is absorbed in walls of bronchial tubes and causes spasmodic contraction of the respiratory tract. On the other hand, NO_2, having relatively low water solubility, reaches small bronchi and lung alveoli and causes inflammation of mucous membranes.

b Particle substances (primary particles, nitrate salt, and sulfate salt)

It has been pointed out that, although most particles with a diameter of 10 μm or more deposit in the nasal cavity and the pharynx, particles with a smaller diameter deposit deeper in the trachea and the lungs, causing asthma and other medical problems.

2.4.2 Characterization of urban air pollution

Urban air pollution characterization factor (UAF) was calculated in the same way of thinking as the characterization factors for hazardous chemical substances and eco-toxicity (see 2.6.2 (2)).

1) With regard to each air pollutant, calculate an increase in the concentration at the time of emission of a unit amount – that is, the concentration factor [(μg/m^3) / (kg/yr)]. Divide this by the environmental standard value in Japan.

2) Calculate 1) also with regard to the standard substance (SO_2 herein).

3) Divide the result of 1) by 2) to obtain a ratio to the standard substance.

NO_x and SO_2 were adopted as air pollutants, and secondary pollutants transmuted from air pollutants (nitrate salt, sulfate salt) were adopted as substances to which human bodies are exposed.

With regard to calculation of the concentration factor, an increment in the concentration of the secondary pollutants created during the emission of a unit amount of air pollutants was calculated for each prefecture, and increments were totaled in each zone (see 2.4.3 (3)). The daily average threshold of hourly values (0.04 ppm for both NO_x and SO_2) was used as the environmental standard for air pollution.

2.4.3 Damage assessment and uncertainty assessment of urban air pollution

(1) Basic policies for damage assessment and uncertainty assessment

The existing LCA methods that have adopted the endpoint approach for the impact categories of urban air pollution are Eco-indicator 99 (Goedkoop et al. 2000) and EPS (Steen 1999) in Europe. An assessment method based on the endpoint approach was adopted also for LIME.

That is, category endpoints were selected for each type of air pollution, and damage assessment was carried out to calculate damage functions and factors. Moreover, final damage factors were calculated through uncertainty assessment.

The policies for the damage assessment and the uncertainty assessment are as follows:

a Damage assessment

Damage factors were calculated from damage assessment.

The types of air pollution and the zones used for the calculation were as follows (see Figure 2.4-2):

With regard to the types of air pollution, as mentioned in 2.4.1, main physicochemical phenomena and space scale differ by types of air pollution. Therefore, under LIME, damage factors were calculated for primary pollutants and secondary pollutants.

NO_2, SO_2, and primary particles were used as primary pollutants (as shown in Table 2.4-2). The diameters of the primary particles were fixed at PM10 and PM2.5. This is for the following reason: if the diameter is less than 10 μm as described before, primary particles reach deeper parts of the respiratory system and it is easy for them to have influence; in addition, although particles with a diameter of 10 μm are regarded as suspended particulate

matters under the environmental standards in Japan, environmental standards have been established even for particles with a diameter of less than 2.5 μm in the US (Note: however, the definition of particle diameter differs between Japan and the US).

Moreover, two types of sources of emission of the primary pollutants were used: chimneys and automobiles. This is because the pattern of concentration differs due to the differences between the two in the height of the source of emission and the pattern of emissions during a day.

In addition, the types of secondary pollutants are nitrate salt produced from NO_2 and sulfate salt produced from SO_2 (as shown in Table 2.4-2).

After the calculation of damage factors for primary pollutants and secondary pollutants, with regard to NO_2 and SO_2, the damage factors for primary pollutants are added to the damage factors for secondary pollutants (nitrate salt, sulfate salt) (see Figure 2.4-2).

With regard to zones, air pollution is greatly influenced by local weather conditions. Therefore, under LIME, damage factors were calculated for each of the zones (Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, Kyushu/Okinawa) and on average in Japan (see Figure 2.4-2).

The calculation was carried out as follows:

First, the damage factors for primary and secondary pollutants were calculated according causal channels (see Figure 2.4.1) as follows (see Figure 2.4-3):

Step 1:The relation between the amount of primary pollutant emissions and an increase in the atmospheric concentration of primary (or secondary) pollutants was quantified.

Step 2:The relation between the increase in the concentration in 1) and the amount of damage was quantified at each endpoint. Endpoints at which the relation between the concentration of air pollutants and the death/disease rate was assessed quantitatively by epidemiologic surveys were selected as endpoints for human health (see Table 2.4-3). Endpoints for primary production were selected not from the impact category of urban air pollution but from the impact category of "acidification."

Step 3:The damage function for each endpoint was calculated by combining Steps 1 and 2.

Step 4:The damage functions for the endpoints in Step 3 were totaled by the area of protection to obtain damage factors.

Details will be described in 2.4.3 (2) for primary pollutants and 2.4.3 (3) for secondary pollutants.

Next, the obtained damage factors for primary and secondary pollutants were totaled (as shown in Figure 2.4-2).

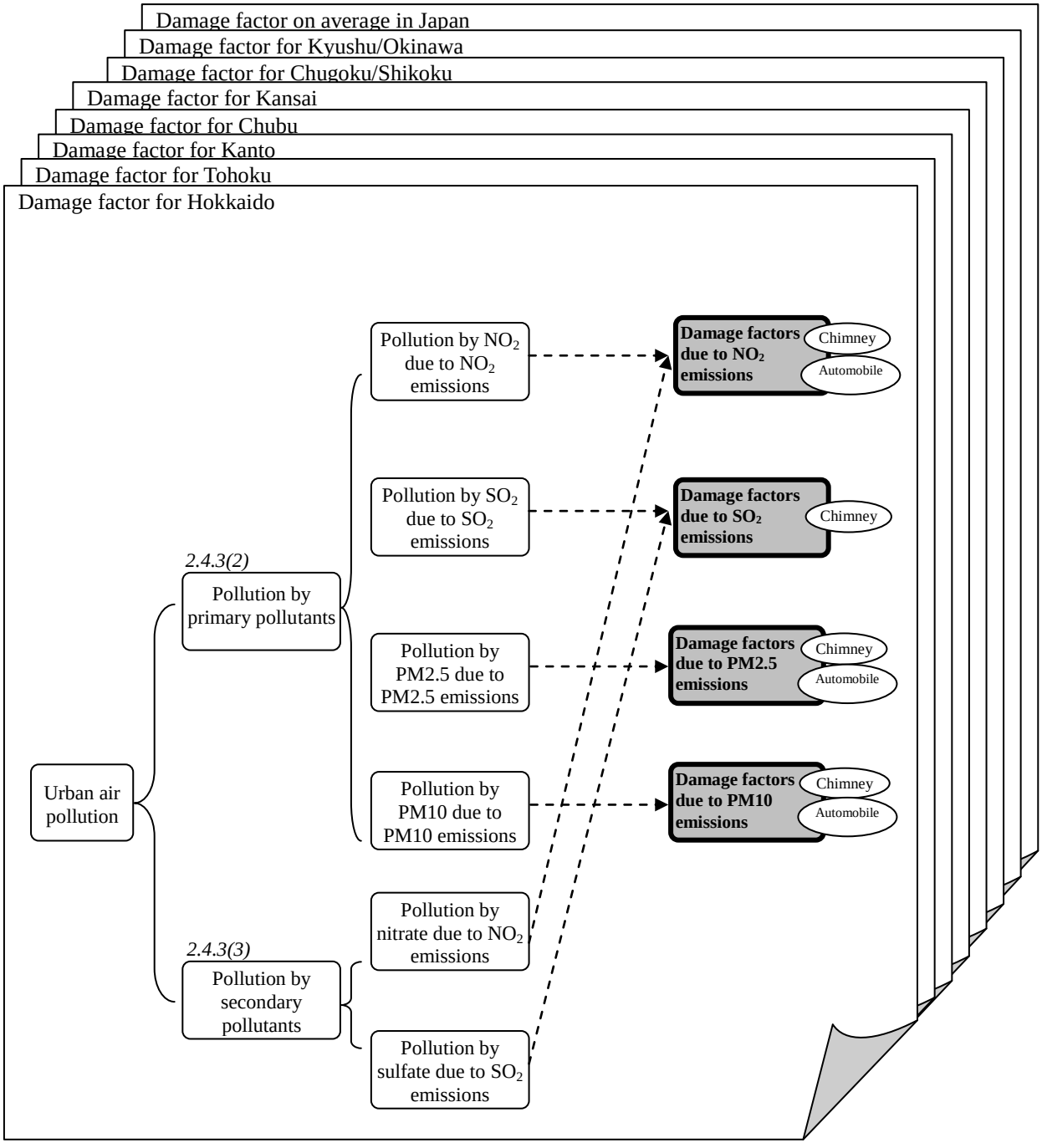


Figure 2.4-2: Objects of calculation for damage factors of urban air pollution

Numbers in italics correspond to headings in the main text.

Table 2.4-3: Category endpoints as objects of calculation for damage functions and factors

Area of protection	Category endpoint		Pollution by primary pollutants • Pollution by NO_2 due to NO_2 emissions • Pollution by SO_2 due to SO_2 emissions	Pollution by primary pollutants • Pollution by PM2.5 due to PM2.5 emissions • Pollution by PM10 due to PM10 emissions Pollution by secondary pollutants • Pollution by nitrate salt due to NO_2 emissions • Pollution by sulfate salt due to SO_2 emissions	Objects of calculation for damage functions	Objects of calculation for damage factors
Human health	Respiratory disease	• Acute death	○	○	Increase in no. of acute deaths (DALY-converted)	○
		• Chronic death	—	○	Increase in no. of chronic deaths (DALY-converted)	
		• Use of bronchodilator	—	○	Increase in no. of sufferers (DALY-converted)	
		• Cough	—	○	Increase in no. of sufferers (DALY-converted)	
		• Lower Respiratory Symptoms	—	○	Increase in no. of sufferers (DALY-converted)	
		• Chronic bronchitis	—	○	Increase in no. of sufferers (DALY-converted)	
		• Chronic cough	—	○	Increase in no. of sufferers (DALY-converted)	
		• Days of behavioral restriction	—	○	Increase in no. of sufferers (DALY-converted)	
		• Hospitalization for respiratory system restriction	○	○	Increase in no. of sufferers (DALY-converted)	
		• Entry into emergency room alization for respiratory system restriction	—	○	Increase in no. of sufferers (DALY-converted)	
		• Entry into emergency room (Asthma)	—	○	Increase in no. of sufferers (DALY-converted)	
		• Entry into emergency room (Inflammation of pharynx/bronchi of preschooler)	—	○	Increase in no. of sufferers (DALY-converted)	
Primary production	Terrestrial ecosystem	Fall in growth due to direct impact to plants	×	×	To be considered in the impact category of acidification	

b Uncertainty assessment

Four types of damage factors were obtained by the procedure described in (a) (damage factors due to NO_2 emissions, SO_2 emissions, PM2.5 emissions, and PM10 emissions). Uncertainty assessment was carried out for each damage factor, and the obtained statistical data were adopted as final damage factors.

Details will be described in 2.4.3 (4).

(2) Pollution by primary pollutants (NO_2, SO_2, primary particles)

Explanations are given according to Steps 1 to 4 in Figure 2.4-3.

a Step 1: Correlation of the amount of primary pollutant emissions with an increase in the concentration of primary pollutants

Simulation was carried out by the use of an atmospheric model to calculate an “increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions” ($\Delta C/\Delta E$).

(a) Cases used for calculation

The cases specified in Table 2.4-4 were used for the calculation.

Table 2.4-4: Case used for calculation of an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE)

	Case	Reason for selecting the case
Zon	7 zones (Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, Kyushu/Okinawa)	• To represent various weather conditions in Japan
Time	1 type (annual average)	• Because the annual average concentration has been assumed as the "concentration" in the "death/disease rate at each endpoint per unit of primary pollutant concentration" (described in b below)
Source of emission	2 types (chimney and automobile)	• Because there are difference between chimneys and automobiles in the height of emission and the daily changing pattern of the amount of emission (see 2.4.3 (2) a (b). However, only chimneys were used for SO_2.

(b) Atmospheric model used for calculation

The concentration of primary pollutants (NO_2, SO_2, and primary particles) is high around the source of emission and becomes lower with increasing distance from the source, due to advection and diffusion. This distribution is influenced by the height of the source, the direction and velocity of the wind, the stability of the atmosphere, etc. To make such a polluting mechanism, a plume model and a puff model were selected for LIME (see Column 2.4-1).

(c) Calculation procedure

A simulation was carried out by the use of revised equations for the plume model and the puff model in (b) above and by the application of weather conditions in each zone. Because the plume model and the puff model assume a linear relation between the amount of emission and the concentration, an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE) was calculated by dividing the concentration obtained from the simulation by the amount of emissions.

Although ΔC/ΔE covers the annual average concentration (see Table 2.4-4), because the relation between weather conditions, such as wind velocity, and concentration is non-linear in the plume model and the puff model, the concentration may not be reproduced accurately if only one case of annual average weather conditions is simulated. To cope with this, under LIME, first the wind direction and velocity and the atmospheric stability were classified into several patterns, and a simulation was carried out for each pattern to calculate ΔC/ΔE. Next, the calculated ΔC/ΔE was weighted with the annual appearance frequency for each pattern to calculate the annual average ΔC/ΔE (see Figure 2.4-4).

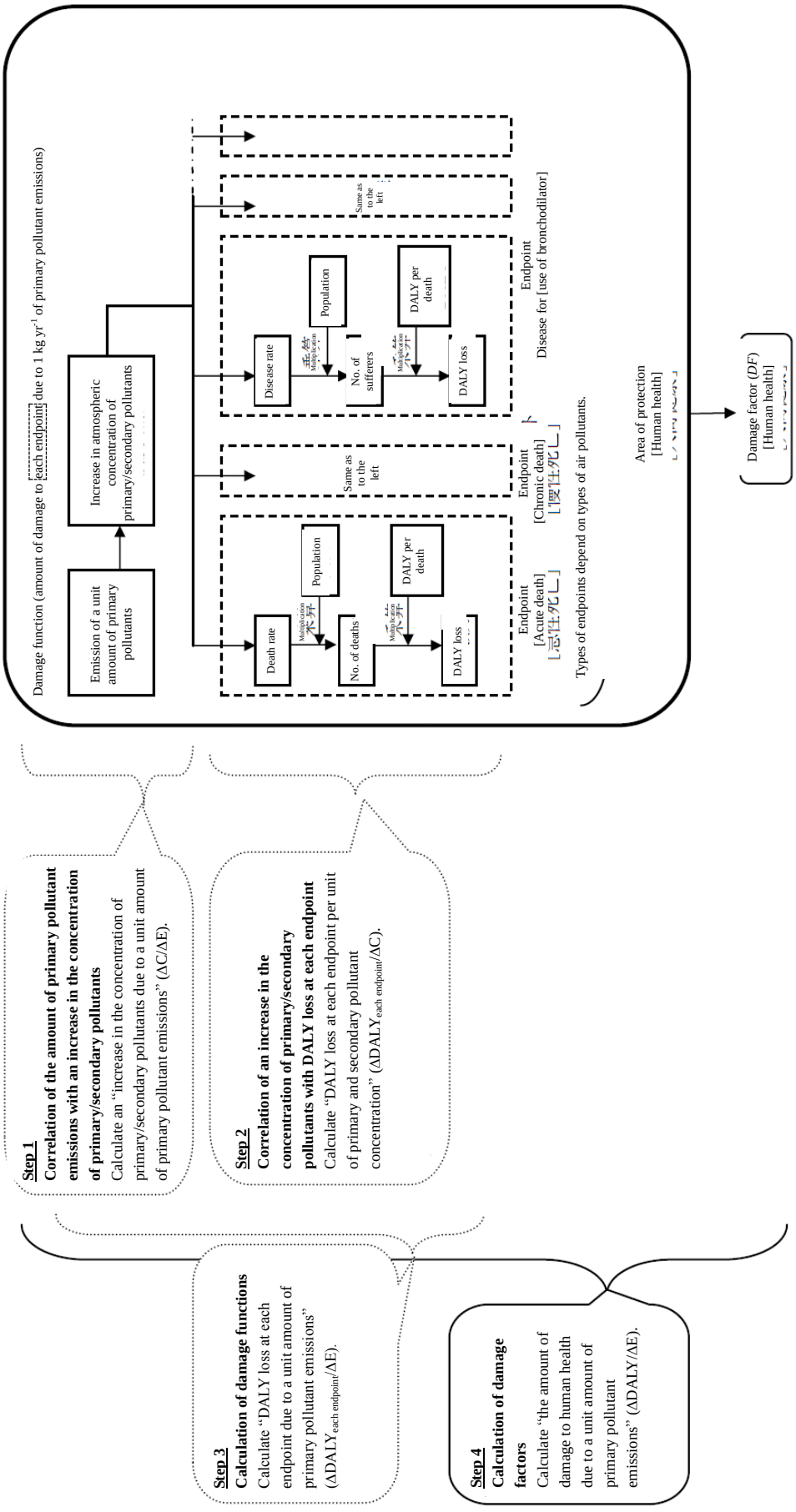


Figure 2.4-3: Flowchart of calculation of damage functions and factors for urban air

Column 2.4-1

Plume model and puff model
The plume model and the puff model are simple models used widely for the calculation of concentration of air pollutants around the source of emission for the purpose of environment assessment. The plume model and the puff model express the concentration downwindunder the lee from the source of emission by the use of a normal distribution. Consideration is given to advection and diffusion, but not to chemical changes (although there are revised equations that take into account chemical changes, they are not minute). Originally, they were models that assumed a normal distribution for not only the downwind direction but also the crosswind direction. From a long-term perspective, because it can be assumed that the probability of wind direction is fixed within a classification of wind direction, the long-term average concentration in the crosswind direction does not change within the classification. To predict such as long-term average concentration, revised equations for the plume model and the puff model were proposed.[1) 2)] The concentration assumed for an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" is expressed as the annual average (see Table 2.4-4), and a revised equation was adopted for LIME. The use of the revised equations for the plume model and the puff model differs depending on wind conditions: windy (1 [m/s] or more); weak wind (0.5 [m/s] to 0.9 [m/s]; no wind (0 [m/s] to 0.4 [m/s]) (see Table 2.4-A). The equations for the plume model and the puff model include an equation that takes into consideration the deposition of particles according to particle diameter. Under LIME, although an attempt was made to use this equation for primary particles (PM2.5, PM10), the result of the calculation was not so different from that of the equations that do not take the deposition into consideration. Because of this, finally, equations that do not take the deposition into consideration were used for NO_2, SO_2, and primary particles (PM2.5, PM10).

Table 2.4-A: Revised equations for the plume model and the puff model used as atmospheric models[1) 2)]

	Basic equation
In a windy condition (1 [m/s] or more)	Use the following equation into which Holland (1953) revised the plume model for the point source: $C(R,z)=\frac{q}{(2\pi)^{\frac{1}{2}}\pi/8\cdot R\sigma_z U}\times\left[\exp\left\{-\frac{(z-H_e)^2}{2\sigma_z^2}\right\}+\exp\left\{-\frac{(z+H_e)^2}{2\sigma_z^2}\right\}\right]$ R, z : R is a downwind distance from the source of emission [m]. z is a vertical distance from the source of emission [m]. (The original point is located at the point source.) C : concentration of substance [kg/m^3]; q: substance emission speed [kg/s]; U: velocity [m/s] σ_z : vertical diffusion parameter [m]; H_e: effective chimney height [m] σ z, the vertical diffusion parameter in the above equation, was calculated based on the Pasquill-Gifford chart (Gifford 1961).
In a weak wind condition (0.5 to 0.9 [m/s])	Use the following equation into which Muto (1979) revised the puff model for the point source: $C(R,z)=\frac{q}{(2\pi)^{\frac{1}{2}}\pi/8\cdot\gamma}\times\left\{\frac{1}{\eta_-^2}\times\exp\left(-\frac{U^2(z-H_e)^2}{2\gamma^2\eta_-^2}\right)+\frac{1}{\eta_+^2}\times\exp\left(-\frac{U^2(z-H_e)^2}{2\gamma^2\eta_+^2}\right)\right\}$ $\eta_-^2=R^2+\frac{\alpha^2}{\gamma^2}(z-H_e)^2$, $\eta_+^2=R^2+\frac{\alpha^2}{\gamma^2}(z+H_e)^2$ α, γ: Use the result of revision based on the diffusion parameter [m/s] and the Turner chart (Turner 1964).
In no wind condition (0 to 0.4 [m/s])	Use the following equation: $C(R,z)=\frac{q}{(2\pi)^{\frac{1}{2}}\gamma}\times\left\{\frac{1}{\eta_-^2}+\frac{1}{\eta_+^2}\right\}$

1) Committee on Suspended Particulate Matters (1997)
2) Environmental Research and Control Center, Committee on Nitrogen Oxide (2000)

Wind direction, wind velocity, and atmospheric stability are classified into the total L pattern, the total M pattern, and the total N pattern, respectively.

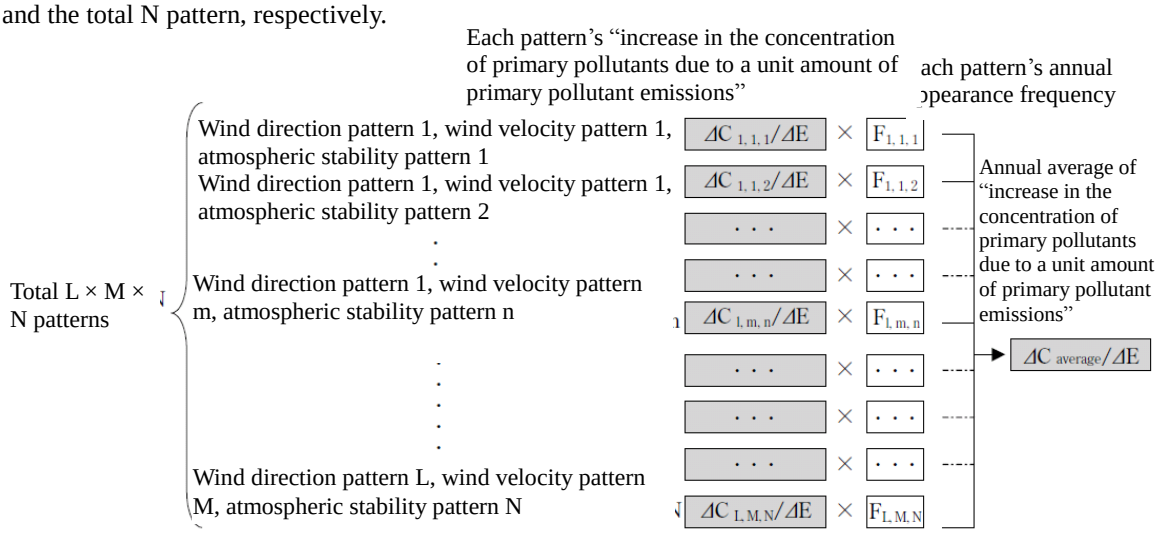


Figure 2.4-4: Procedure for calculation of an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE) concerning annual average concentration

(d) Calculation conditions

The scope of calculation, the amount of emissions, and the height of calculation of concentration were set as shown in Table 2.4-5.

With regard to weather, as described in (c) above, wind direction and velocity and atmospheric stability were classified into several patterns. Table 2.4-6 shows the classification standards.

Weather patterns were classified according to the combination of wind direction and velocity and atmospheric stability specified in the table. Because, as a matter of course, the likelihood of appearance of each weather pattern differs from zone to zone, the annual appearance frequency for each weather pattern was calculated for each of the seven zones specified in "a Cases used for calculation." Data from terrestrial weather observatories around highly populated cities in each zone were used for calculation (see Table 2.4-7).

(e) Calculation results

(i) Concentration distribution

The concentration distribution around sources of primary pollutant emissions was calculated by the above-described method.

Because, as described in (b) above, the same equations (the revised equations for the plume model and the puff model) were used for all the primary pollutants (NO_2, SO_2, primary particles), the calculation results are the same irrespective of the kind of substance.

Of the calculation results in the seven zones – Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, and Kyushu/Okinawa – the results in Kanto (calculated under the weather conditions in Tokyo; see Table 2.4-7) are shown in Figure 2.4-5. The following are the characteristics of the results:

[Emission from chimneys]

The following two points are characteristics common to all the zones:

- Generally, concentration is highest around sources of emissions and becomes lower with increasing distance from them.

- However, when sources of emissions were surveyed in detail, it was found that the decreasing rate of concentration was lower or the concentration rose slightly at a distance of 50 to 100 m from a source of emission.

The reasons for the latter characteristic are as follows:

When there is no wind, the puff model for the time of no wind is applied for the calculation. In this case, the nearer a source of emission, the higher the concentration. On the other hand, when it is windy and when there is weak wind, the plume model and the puff model for the time of weak wind are respectively applied for calculation. In these cases, at a slight distance from a source of emission, a vertical extent reaches the ground and the highest

concentration appears. Because the calculation results of concentration were an accumulation of the results at the times of no wind and weak wind and at the windy time, the concentration distribution has the characteristics of them all.

[Emission from automobiles]

Because the height of emissions from automobiles is lower than emissions from chimneys, concentration is high around sources of emissions and becomes considerably lower with increasing distance from the sources.

Table 2.4-5: Conditions for calculation of an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE)

Conditions	Content
Scope of calculation	Concentration was calculated until 20 km (radius of 10 km) from the source of emission so that air pollution on a local scale and that on an urban scale (see Figure 2.4-1) could be reproduced.
Amount of emissions	Daily changes in the amount of emissions from chimneys and automobiles were fixed with reference for a survey on daily changes in emission speed.[1] The height of emissions was fixed at 20 m for chimney (the mode of a statistical data[2]) and 1 m for automobiles.
Height for calculation of concentration	Fixed at 1.5 m from the ground with consideration for the height at which human beings breathe

1) FY1999 Survey on the Relation between Environmental Impact and Damage – Preparation of a Pollution Map (March 2000)
2) FY1997 Report on the Results of Operations Entrusted by the Environment Agency: Comprehensive Survey on the Amount of Air Pollutant Emissions (1998)

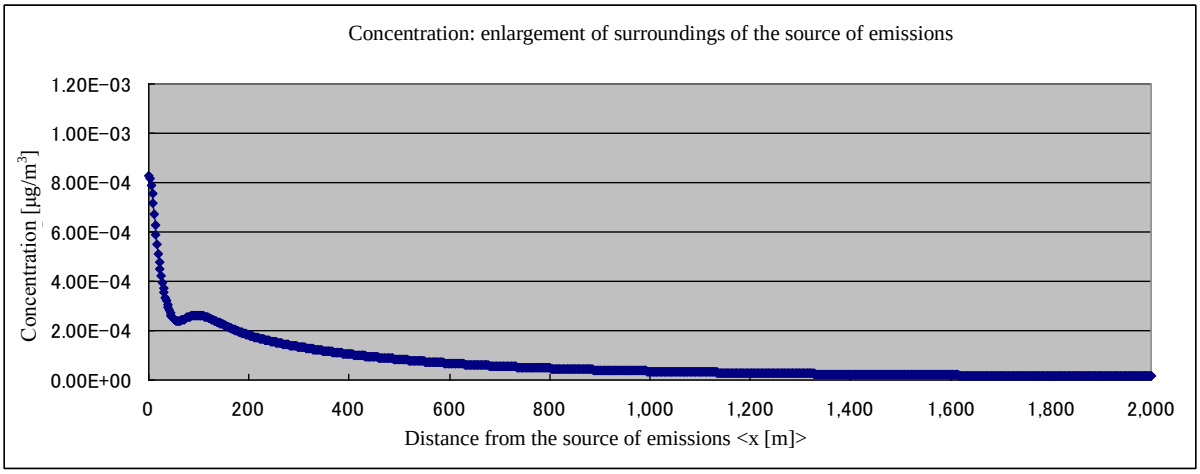
Table 2.4-6: Standards for weather pattern classification

<table>
<tr><td>Weather element</td><td>Classification</td></tr>
<tr><td>Wind direction</td><td>Wind direction was divided into the 16 directions that can be obtained from terrestrial weather observation (Table 2.4-7).</td></tr>
<tr><td>Wind velocity</td><td>Wind velocity was divided as shown in the following table so as to be consistent with the classification for the application of the plume model and the puff model and the classification of wind velocity used for the judgment of atmospheric stability by Pasquill (described below). [1) 2)]
<table>
<tr><td></td><td>Wind velocity [m/s]</td></tr>
<tr><td>No wind</td><td>～0.4</td></tr>
<tr><td>Weak wind</td><td>0.5～0.9</td></tr>
<tr><td rowspan="6">Windy</td><td>1.0～1.9</td></tr>
<tr><td>2.0～2.9</td></tr>
<tr><td>3.0～3.9</td></tr>
<tr><td>4.0～5.9</td></tr>
<tr><td>6.0～7.9</td></tr>
<tr><td>8.0～</td></tr>
</table></td></tr>
<tr><td>Atmospheric stability</td><td>Atmospheric stability was divided into A, A-B, B, B-C, C, C-D, D, E, F, and G based on the following table [2)] in which the atmospheric stability by Pasquill was revised.
<table>
<tr><td rowspan="2">Wind velocity (U) [m/s]</td><td colspan="4">Daytime: amount of insolation cloud cover [kW/m^2]</td><td colspan="3">Nighttime: cloud cover</td></tr>
<tr><td>$T \geqq 0.60$</td><td>$0.60 > T \geqq 0.30$</td><td>$0.30 > T \geqq 0.15$</td><td>$0.15 > T$</td><td>Main cloud (8～10)</td><td>Upper cloud (5～10) Middle/ lower cloud (5～7)</td><td>Cloud cover (0～4)</td></tr>
<tr><td>$U < 2$</td><td>A</td><td>A–B</td><td>B</td><td>D</td><td>D</td><td>G</td><td>G</td></tr>
<tr><td>$2 \leqq U < 3$</td><td>A–B</td><td>B</td><td>C</td><td>D</td><td>D</td><td>E</td><td>F</td></tr>
<tr><td>$3 \leqq U < 4$</td><td>B</td><td>B–C</td><td>C</td><td>D</td><td>D</td><td>D</td><td>E</td></tr>
<tr><td>$4 \leqq U < 6$</td><td>C</td><td>C–D</td><td>D</td><td>D</td><td>D</td><td>D</td><td>D</td></tr>
<tr><td>$6 \leqq U$</td><td>C</td><td>D</td><td>D</td><td>D</td><td>D</td><td>D</td><td>D</td></tr>
</table></td></tr>
</table>

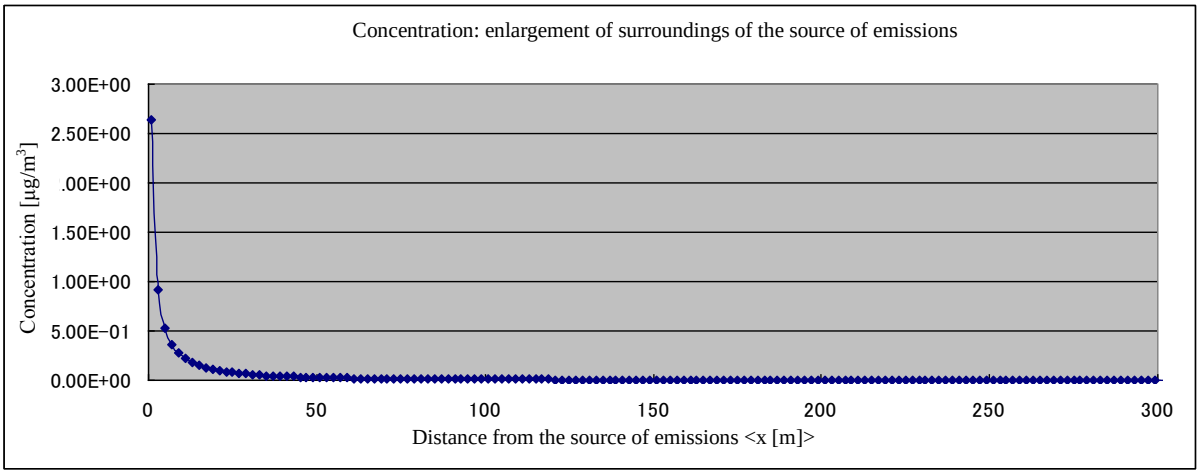
1) Committee on Suspended Particulate Matters (1997)
2) Environmental Research and Control Center, Committee on Nitrogen Oxide (2000)

Table 2.4-7: Observation points of terrestrial weather data used

Zone	Observation points of terrestrial weather data used
Hokkaido	Sapporo District Meteorological Observatory
Tohoku	Sendai District Meteorological Observatory
Kanto	Tokyo District Meteorological Observatory
Chubu	Nagoya Local Meteorological Observatory
Kansai	Osaka District Meteorological Observatory
Chugoku/Shikoku	Hiroshima Local Meteorological Observatory
Kyushu/Okinawa	Fukuoka District Meteorological Observatory



[Emission from a chimney]



[Emission from an automobile]

Figure 2.4-5: Annual average concentration due to 1 kg yr^{-1} of primary pollutant emissions: Tokyo

(ii) Increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions (ΔC/ΔE)

A concentration distribution can be obtained from the plume model and the puff model according to the distance from the source of emission (see (i)). Because of this, when an "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE) was calculated, the products of concentration and areas were summed within a calculation scope of 20 km (radius of 10 km) described in (d). Concretely, the concentration at each calculation point (every 2 m in this case) was multiplied by an inside range of 2 m, including the point, and the resultant products were totaled (Equation 2.4-1).

$$\sum_{n=1}^{500n} \left[\pi \cdot (2n)^2 - \pi \cdot \{2(n-1)\}^2\right] \cdot C(n) \qquad (2.4\text{-}1)$$

n: point that is 1 at a distance of 2 m from the source of emission and increments by 1 with every 2 m distance.

C (*n*): annual average concentration of primary pollutants at the *n*th point [μg/m^3]

The sum of products obtained in this way was divided by the amount of emissions to calculate ΔC/ΔE.

Table 2.4-8 shows the calculation results in each zone.

Table 2.4-8: "Increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" (ΔC/ΔE)

[ug m^{-3} m^2 kg^{-1} yr]

Zone	Chimney	Automobile
Hokkaido	1.302E+03	5.427E+03
Tohoku	1.371E+03	6.151E+03
Kanto	1.454E+03	6.891E+03
Chubu	1.175E+03	5.555E+03
Kansai	1.351E+03	5.807E+03
Chugoku/Shikoku	1.283E+03	5.871E+03
Kyushu/Okinawa	1.311E+03	5.309E+03

b Step 2: Correlation of an increase in the atmospheric concentration of primary pollutants with DALY loss at each endpoint

"DALY loss at each endpoint per unit of primary pollutant concentration" ($\Delta DALY_{each\ endpoint}$ / ΔC) was calculated by Step 2 as shown in Figure 2.4-3.

Figure 2.4-6 shows the calculation flowchart. "(a) Death/Disease rate at each endpoint per unit of primary pollutant concentration" was multiplied by "(b) Population density in each zone" and "(c) DALY of 1 death/1 disease at each endpoint."

The following is an explanation of (a) to (c) in Figure 2.4-6:

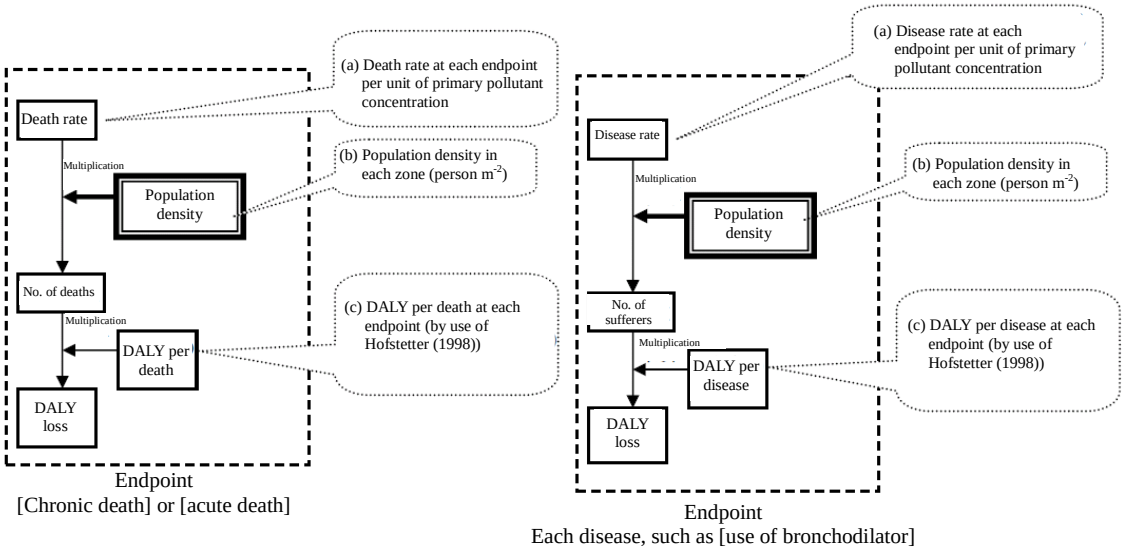


Figure 2.4-6: Flowchart of calculation of "DALY loss at each endpoint per unit of primary pollutant concentration" ($\Delta DALY_{\text{each endpoint}} / \Delta C$)

(a), (b), and (c) in the chart correspond to headings in the main text.

(a) Death/disease rate at each endpoint per unit of primary pollutant concentration

With regard to the "death/disease rate at each endpoint per unit of primary pollutant concentration," the results of an epidemiological survey used for ExternE (European Commission 1999) were used for setting the rate (see Table 2.4-9). However, with regard to chronic death due to PM2.5 or PM10, reexamination was carried out after LIME1, and a rate was fixed based on a new document (Pope et al. 2002). Table 2.4-10 shows values set as described above.

The target population differs among the endpoints (for example, entire population, adult, child, and asthma sufferer). In addition, these "death/disease rates at each endpoint per unit of primary pollutant concentration" are based on the assumption that "concentration" is annual average concentration. Because of this, annual average concentration was used when (a) "death/disease rates at each endpoint per unit of primary pollutant concentration" was calculated in a above (see Table 2.4-4).

Table 2.4-9: Method of setting a "death/disease rate at each endpoint per unit of primary pollutant concentration"

Item	Method of setting
(a) Death rate at each endpoint per unit of primary pollutant concentration	Death rate in Japan by multiplying "increase in death rate at each endpoint per unit of primary pollutant concentration" under ExternE by the current situation
(a) Disease rate at each endpoint per unit of primary pollutant concentration	Disease rate at each endpoint per unit of primary pollutant concentration under ExternE

(a) in the Table corresponds to Figure 2.4-6.

Table 2.4-10: "The increase in death rate and the disease rate at each endpoint per unit of primary pollutant"

Type of respiratory disease (Endpoint)	"Increase in death rate at each endpoint per unit of NO_2/SO_2 concentration" [$(Risk/Risk_{baseline})\ \mu g^{-1}m^3$], Risk=case person^{-1} yr^{-1} "(a) Disease rate at each endpoint per unit of NO_2/SO_2 concentration" [case person^{-1} yr^{-1} μg^{-1} m^3]				(c) DALY of 1 death/1 disease at each endpoint [DALY/case]	Geometric diffusion σ^2	Target population
	NO_2	Geometric diffusion σ^2	SO_2	Geometric diffusion σ^2			
Acute death	Sunyer, et al (1996), Anderson, et al (1996) 0.00034	36.0	Anderson, et al (1996), Touloumi, et al (1996) 0.00072	16	0.75	5.0	Entire population
Hospitalization for respiratory system	Ponce de Leon, et al (1996) 1.400E−06	16.0	Ponce de Leon, et al (1996) 2.040E−06	6	0.011	2.0	Entire population

Type of respiratory disease (Endpoint)	"Increase in death rate at each endpoint per unit of PM10/PM2.5 concentration" Risk=case person^{-1} yr^{-1} "(a) Disease rate at each endpoint per unit of PM10/PM2.5concentration" [case person^{-1} yr^{-1} μg^{-1} m^3]				(c) DALY of 1 death/1 disease at each endpoint [DALY/case]	Geometric diffusion σ^2	Target population
	PM10	Geometric diffusion σ^2	PM2.5	Geometric diffusion σ^2			
Acute death	Spix, et al (1996),Verhorf et al (1996) 0.00040	16.0	Spix, et al (1996), Verhorf, et al (1996) 0.00068	16.0	0.75	5.0	Entire population
Chronic death	Pope, et al (2002) 0.001	4.0E-6	Pope, et al (2002) 0.00593	3.4E-7	6.6	3.0	Aged 30 and over
Use of bronchodilator: adult	Dusseldorp, et al (1995) 0.163	16.0	Dusseldorp, et al (1995) 0.272	16.0	0.00027	2.0	Asthma and adult
Use of bronchodilator: child	Roemer, et al (1993) 0.078	16.0	Roemer, et al (1993) 0.129	16.0	0.00027	2.0	Asthma and child
Cough: adult	Dusseldorp, et al (1995) 0.168	6.0	Dusseldorp, et al (1995) 0.280	6.0	0.00014	2.0	Asthma and adult
Cough: child	Pope, et al (1992) 0.133	6.0	Pope, et al (1992) 0.223	6.0	0.00014	2.0	Asthma and child
Lower respiratory symptoms (stridor): adult	Dusseldorp, et al (1995) 0.061	6.0	Dusseldorp, et al (1995) 0.101	6.0	0.00014	2.0	Asthma and adult
Lower respiratory symptoms (stridor): child	Roemer, et al (1993) 0.103	6.0	Roemer, et al (1993) 0.172	6.0	0.00014	2.0	Asthma and child
Chronic bronchitis: adult	Abbey, et al (1995) 4.900E−05	16.0	Abbey, et al (1995) 7.800E−05	16.0	2	3.0	Adult
Chronic bronchitis: child	Dockery, et al (1989) 1.610E−03	6.0	Dockery, et al (1989) 2.690E−03	6.0	0.025	3.0	Child
Chronic cough	Dockery, et al (1989) 2.070E−03	16.0	Dockery, et al (1989) 3.460E−03	16.0	0.025	3.0	Child
Days of behavioral restriction	Ostro (1987) 0.025	16.0	Ostro (1987) 0.042	16.0	0.00027	2.0	Adult
Hospitalization for respiratory system	Dab, et al (1996) 2.070E−06	6.0	Dab, et al (1996) 3.460E−06	6.0	0.011	2.0	Entire population
Entry into emergency room (chronic obstructive pulmonary disease)	Sunyer, et al (1993) 7.200E−06	36.0	Sunyer, et al (1993) 1.200E−05	36.0	0.00082	3.0	Entire population
Entry into emergency room (asthma)	Schwarts (1993), Bates, et al (1990) 6.450E−06	36.0	Schwarts (1993), Bates, et al (1990) 1.080E−05	36.0	0.00082	3.0	Entire population
Entry into emergency room (inflammation of pharynx/bronchi of preschooler)	Schwarts, et al (1991) 2.910E−05	36.0	Schwarts, et al (1991) 4.860E−05	36.0	0.00082	3.0	Entire population

(a) and (c) in the table correspond to Figure 2.4-6
(Sources) Extern E (European Commission 1999); Pope et al. (2002); the source for the shaded parts is Hofstetter (1998).

(b) Population density in each zone

The population density was fixed for the target population for each endpoint in (a). Population density was adopted rather than population because the dimension of the calculated "death/disease rates at each endpoint per unit of primary pollutant concentration" is the product of the concentration and the area of the ground surface.

(c) DALY of 1 death/1 disease at each endpoint

Values in Hofsterrer (1998) were used (see Table 2.4-10).

c Steps 3 and 4: Calculation of the damage function and the damage factor

Steps 3 and 4 in Figure 2.4-3 were used for calculating the damage function and the damage factor. The concrete procedure is as follows:

- Step 3: The "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" ($\Delta C/\Delta E$) in Step 1 was multiplied by the "DALY loss at each endpoint per unit of primary pollutant concentration" ($\Delta DALY_{\text{each endpoint}}/\Delta C$) in Step 2 to obtain the damage function at each endpoint ($\Delta DALY_{\text{each endpoint}}/\Delta E$).

- Step 4: The damage functions in Step 3 at all the endpoints for respiratory disease were totaled to obtain the damage factor ($\Delta DALY/\Delta E$).

Table 2.4-11 shows examples of the calculated damage functions and factors (pollution due to NO_2 emissions, Tokyo). Table 2.4-12 shows the average prefectural damage factor in each zone and the national average.

Table 2.4-11: The damage function ($\Delta DALY_{\text{each endpoint}}/\Delta E$) and the damage factor ($\Delta DALY/\Delta E$) for human health concerning pollution by NO_2 due to NO_2 emissions: Tokyo

				Tokyo	
Type of source of emissions				Chimney	Automobile
(1) $\Delta C/\Delta E$* [μg m^{-3} m^2 kg^{-1} yr]				1.454E+03	6.891E+03
Endpoint	Acute death		(a) Death rate per unit of NO_2 concentration** (entire population) [case person^{-1} yr^{-1} μg^{-1} m^3]	2.522E−06	
			(b) Population density [person m^{-2}]	5.384E−03	
			(c) DALY for acute death [DALY case^{-1}]	7.500E−01	
		(2) $\Delta DALY_{\text{each endpoint}}/\Delta C$*** [DALY m^{-2}yr^{-1} μg^{-1} m^3] (=(a)×(b)×(c))		1.019E−08	
		(3) $\Delta DALY_{\text{each endpoint}}/\Delta E$ [DALY kg^{-1}] (=(1)×(2))		1.481E−05	7.019E−05
	Hospitalization for respiratory		(a) Disease rate per unit of NO_2 concentration (entire population) [case person^{-1} yr^{-1} μg^{-1} m^3]	1.400E−06	
			(b) Population density [person m^{-2}]	5.384E−03	
			(c) DALY for hospitalization for respiratory system [DALY case^{-1}]	1.100E−02	
		(2) $\Delta DALY_{\text{each endpoint}}/\Delta C$*** [DALY m^{-2}yr^{-1} μg^{-1} m^3] (=(a)×(b)×(c))		8.292E−11	
		(3) $\Delta DALY_{\text{each endpoint}}/\Delta E$ [DALY kg^{-1}] (=(1)×(2))		1.206E−07	5.714E−07
(4) $\Delta DALY/\Delta E$ [DALY kg^{-1}] (=Σ(3))				1.493E−05	7.077E−05

(1) to (4) in the table correspond to Figure 2.4-3; (a) to (c) correspond to Figure 2.4-6.
†1 An increase in NO_2 concentration due to a unit amount of NO_2 emissions
†2 "(a) Acute death rate per unit of NO_2 concentration" is calculated by "the increase rate of acute deaths per unit of NO_2 emissions × the current death rate" (see 2.4.3 (2) b (a)).
†3 DALY loss at each endpoint per unit of NO_2 emissions

Table 2.4-12: The damage factor for human health concerning pollution due to primary pollutant emissions (ΔDALY /ΔE) [ΔDALY kg^{-1}]: the average prefectural damage factor in each zone and the national average

	Pollution by NO_2 due to NO_2 emissions		Pollution by SO_2 due to SO_2 emissions	Pollution by PM2.5 due to PM2.5 emissions		Pollution by PM10 due to PM10 emissions	
	Chimney	Automobile	Chimney	Chimney	Automobile	Chimney	Automobile
Average in Hokkaido	1.803E-07	7.514E-07	3.808E-07	4.377E-05	1.825E-04	1.462E-05	6.094E-05
Average in Tohoku	4.329E-07	1.942E-06	9.143E-07	1.052E-04	4.721E-04	3.510E-05	1.575E-04
Average in Kanto	5.077E-06	2.406E-05	1.072E-05	1.231E-03	5.833E-03	4.118E-04	1.951E-03
Average in Chubu	8.414E-07	3.978E-06	1.777E-06	2.030E-04	9.598E-04	6.798E-05	3.214E-04
Average in Kansai	3.339E-06	1.435E-05	7.054E-06	7.975E-04	3.428E-03	2.687E-04	1.155E-03
Average in Chugoku/ Shikoku	6.222E-07	2.847E-06	1.314E-06	1.518E-04	6.948E-04	5.058E-05	2.314E-04
Average in Kyushu/ Okinawa	9.652E-07	3.909E-06	2.039E-06	2.302E-04	9.320E-04	7.744E-05	3.136E-04
National average	1.637E-06	7.406E-06	3.458E-06	3.946E-04	1.786E-03	1.323E-04	5.987E-04

(Note) Prefectures' damage factors were averaged in each zone.

(3) Pollution by secondary pollutants (nitrate salt and sulfate salt)

The following is an explanation according to Steps 1 to 4 in Figure 2.4-3.

a Step 1: Correlation of the amount of primary pollutant emissions and an increase in the concentration of nitrate/sulfate

An "increase in nitrate/sulfate concentration due to a unit amount of NO_2/SO_2 emissions" (ΔC/ΔE) was calculated based on the result of simulation by the use of an air quality model.

(a) Cases used for calculation

Table 2.4-13 shows the cases used for the calculation.

The "source zone" and the "receptor zone" in the table are types of zones used in the below-described source-receptor matrix formulated by Ikeda and Hagimoto (see Figure 2.4-7).

(b) Air quality model used for calculation

NO_2 and SO_2 are converted into nitrate and sulfate respectively through various channels during long-distant transportation (acidification, chemical reaction with other pollutants, etc.), and they are partially removed from the atmosphere by deposition. To reflect such polluting mechanism, the OPU model formulated by Ikeda (2001) was selected as an air quality model under LIME (see Column 2.4-2).

(c) Calculation procedure

As shown in Figure 2.4-7, a simulation was carried out around Japan, formulated by Ikeda and Hagimoto, with using (b) Air quality model used for calculation. As a result, the annual average concentration of nitrate and sulfate in each grid and the average concentration in each zone were calculated. The source-receptor matrix is a table that shows the relation between the nitrate and sulfate concentration in each zone (= receptor zone) and NO_2/SO_2 emissions in each source zone.

Under LIME, an "increase in nitrate/sulfate salt concentration due to a unit amount of NO_2/SO_2 emissions" was calculated from the source-receptor matrix. Concretely, because the OPU model assumes that the relation between the amount of emissions and concentration is linear, the NO_3^- concentration in each receptor zone was divided by the amount of NO_2 emissions in the source zone, and the SO_4^{2-} concentration in each receptor zone was divided by the amount of SO_2 emissions in the source zone.

(d) Conditions for calculation

The conditions for calculation were set as shown in Table 2.4-14.

Table 2.4-13: Cases used for calculation of an "increase in nitrate/sulfate salt due to a unit amount of NO_2/SO_2 emissions" ($\Delta C/\Delta E$)

	Case	Reason for selecting the case
Zone	6 source zone × 6receptor zone (See Figure 2.4-7)	• See the main text.
Time	1 type (Annual average)	• Because the annual average concentration has been assumed as the "concentration" in the "death/disease rate at each endpoint per unit of nitrate/sulfate"

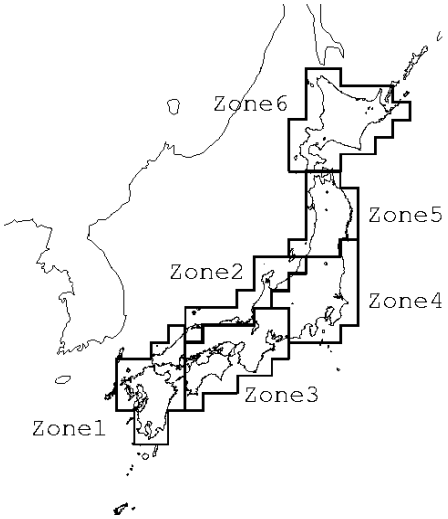


Figure 2.4-7: Division of zones for source-receptor matrix
(Source) Ikeda (2001)

Table 2.4-14: Conditions for calculation of an "increase in nitrate/sulfate concentration due to a unit amount of NO_2/SO_2 emissions" ($\Delta C/\Delta E$)

Condition	Content
Mesh width, layer thickness	1 grid was fixed at 80 km × 80 km. The vertical direction was divided into seven layers: 1st layer between 0 to 100 m; 2nd layer between 100 to 300 m; 3rd layer between 300 to 500 m; 4th layer between 500 to 1,000 m; 5th layer between 1,000 to 2,000 m; 6th layer between 2,000 to 3,000 m; 7th layer between 3,000 and 5,000 m.
Amount of emissions	Values in 1991 calculated by Higashino (1997) were used as the amounts of NO_2/SO_2 emissions.
Initial and boundary values	The initial values for nitrate salt and sulfate were fixed at almost 0.

Column 2.4-2

Column 2.4-2: Ikeda's OPU model

North America, Europe, and East Asia have the problem that air pollutants emitted from industrial districts are transported beyond borders and generate photochemical oxidant and cause oxidization.

The OPU model formulated by Ikeda (2001) is a model for predicting how emitted air pollutants are transported over a long distance and change all over Japan and East Asia. Ikeda used this model to assess the effect of measures for controlling the amount of air pollutant emissions.

This model is a three-dimensional grid model. The transportation, diffusion, chemical changes, atmospheric removal, and other phenomena of air pollutants are calculated. Table 2.4-B shows equations for the calculation.

Table 2.4-B: Equations for the OPU model (Ikeda 2001) used as an air

	Basic equations
SO_2, particulate sulfate ion (SO_4^{2-}), cloud water sulfate ion concentration (cloud- SO_4^{2-})	Use the following equations: $\frac{dC_{1S}}{dt} = -(k_{1S}+k_{2S}+k_{DS}+k_{WS})C_{1S}$ $\frac{dC_{2S}}{dt} = \frac{3}{2}k_{1S}C_{1S} - (k_{3S}+k'_{DS}+k'_{WS})C_{2S}$ $\frac{dC_{3S}}{dt} = \frac{3}{2}k_{2S}C_{1S} + k_{3S}C_{2S} - k_{RS}C_{3S}$ C_{1S} : Atmospheric SO_2 gas concentration [g/m^3] C_{2S} : Atmospheric SO_4^{2-} concentration [g/m^3] C_{3S} : Cloud water $SO4^{2-}$ concentration (clod– SO_4^{2-}) [g/m3] k_{1S} : Reaction speed coefficient from SO_2 to SO_4^{2-} [1/h] k_{2S} : Absorption speed coefficient of SO_2 to cloud water [1/h] k_{3S} : Absorption speed coefficient of SO_4^{2-} to cloud water [1/h] k_{DS} : Dry deposition speed coefficient of SO_2 [1/h] k'_{DS} : Dry deposition speed coefficient of SO_4^{2-} [1/h] k_{WS} : Wet deposition (washout) speed coefficient of SO_2 [1/h] k'_{WS} : Wet deposition speed coefficient of SO_4^{2-} [1/h] k_{RS} : Rainwater absorption (rainout) speed coefficient of SO_4^{2-} [1/h]
NO, NO_2, Nitric acid gas (HNO_3), PAN, particulate sulfate ion (NO_3^-)	Use the following equations: $\frac{dC_{1N}}{dt} = -(k_{DN}+k_{WN})C_{1N}$ $\frac{dC_{2N}}{dt} = -k_{1N}C_{2N} + \frac{46}{121}k_{3N}C_{3N} - (k_{DN}+k_{WN})C_{2N}$ $\frac{dC_{3N}}{dt} = 0.5\left(\frac{121}{46}k_{1N}C_{2N} + k_{2N}C_{3N}\right) - (k_{2N}+k_{3N})C_{3N} - (k'_{DN}+k'_{WN})C_{3N}$ $\frac{dC_{4N}}{dt} = 0.4\left(\frac{63}{46}k_{1N}C_{2N} + \frac{63}{121}k_{2N}C_{3N}\right) - (k''_{DN}+k''_{WN})C_{4N}$ $\frac{dC_{5N}}{dt} = 0.1\left(\frac{62}{46}k_{1N}C_{2N} + \frac{62}{121}k_{2N}C_{3N}\right) - (k'''_{DN}+k'''_{WN})C_{5N}$ C_{1N} : Atmospheric NO gas concentration [g/m^3] C_{2N} : Atmospheric NO_2 gas concentration [g/m^3] C_{3N} : Atmospheric PAN gas concentration [g/m^3] C_{4N} : Atmospheric HNO_3 gas concentration [g/m^3] C_{5N} : Atmospheric particulate NO_3^- ion concentration [g/m^3] k_N : Molar ratio between NO gas and NO_2 gas k_{1N} : Reaction speed coefficient from NO_2 gas to other substances [1/h] k_{2N} : Reaction speed coefficient from PAN gas to other substances (daytime) [1/h] k_{3N} : Reaction speed coefficient from PAN gas to other substances (nighttime) [1/h] k_{DN} : Dry deposition speed coefficient of NO gas and NO_2 gas [1/h] k'_{DN} : Dry deposition speed coefficient of PAN gas [1/h] k''_{DN} : Dry deposition speed coefficient of HNO_3 gas [1/h] k'''_{DN} : Dry deposition speed coefficient of particulate NO_3^- ion [1/h] k_{WN} : Wet deposition speed coefficient of NO gas and NO_2 gas [1/h] k'_{WN} : Wet deposition speed coefficient of PAN gas [1/h] k''_{WN} : Wet deposition speed coefficient of HNO_3 gas [1/h] k'''_{WN} : Wet deposition speed coefficient of particulate NO_3^- ion [1/h]

Table 2.4-15: Source-receptor matrix of NO_2 converted emissions and NO_3^- concentration Average concentration until a vertical height of 100 m

			NO_3^- average concentration at receptor [μg m^{-3}]					
		NO_2 converted emissions [ton yr^{-1}]	zone1	zone2	zone3	zone4	zone5	zone6
source	zone1	260162.8	0.13	0.02	0.05	0.01	0.00	0.00
	zone2	67180.5	0.00	0.03	0.01	0.02	0.01	0.00
	zone3	449830.8	0.03	0.07	0.19	0.07	0.01	0.00
	zone4	448284.0	0.00	0.02	0.02	0.26	0.04	0.00
	zone5	105491.8	0.00	0.00	0.00	0.01	0.05	0.01
	zone6	95667.4	0.00	0.00	0.00	0.00	0.00	0.03

(Source) Personal communication between Ikeda and Hagimoto

Table 2.4-16: Source-receptor matrix of SO_2 converted emissions and SO_4^{2-} concentration Average concentration until a vertical height of 100 m

			SO_4^{2-} average concentration at receptor [μg m^{-3}]					
		SO_2 converted emissions [ton yr^{-1}]	zone1	zone2	zone3	zone4	zone5	zone6
source	zone1	1025230.5	1.36	0.34	0.66	0.23	0.10	0.03
	zone2	49753.0	0.02	0.24	0.07	0.11	0.08	0.01
	zone3	272393.2	0.21	0.43	1.06	0.37	0.11	0.03
	zone4	440573.4	0.05	0.15	0.17	1.50	0.33	0.07
	zone5	62627.9	0.00	0.03	0.01	0.05	0.28	0.05
	zone6	294386.7	0.01	0.02	0.01	0.01	0.06	0.44

(Source) Personal communication between Ikeda and Hagimoto

(e) Calculation results

(i) Concentration distribution

Ikeda and Hagimoto's source-receptor matrix shows the comparison between the average concentration of NO_3^- and SO_4^{2-} in each receptor zone and the amount of NO_2/SO_2 emissions in each source zone (see Tables 2.4-15 and 2.4-16).

The matrix shows that the concentration in the zone where the emission occurred is the highest, and the high concentration area has extended to the surroundings.

(ii) Increase in nitrate/sulfate concentration due to a unit amount of NO_2/SO_2 emissions

Under LIME, the concentration in each receptor zone in (i) is divided by the amount of emissions in each source zone to calculate an "increase in the concentration of NO_3^-/SO_4^{2-} due to a unit amount of NO_2/SO_2 emissions" (see Table 2.4-17 and Table 2.4-18).

As shown in these tables, compared with the NO_3^- concentration due to the emission of 1 [kg yr^{-1}] of NO_2, the SO_4^{2-} concentration has spread further due to the emission of 1 [kg yr^{-1}] of SO_4^{2-}. This indicates that the time necessary for SO_2's change into sulfate salt is longer than the time necessary for NO_2's change into nitrate and that SO_2 changes into sulfate after being transported to a more distant place.

Table 2.4-17: "Increase in NO_3^- concentration due to a unit amount of NO_2 emissions" ($\Delta C/\Delta E$) [μg m^{-3} kg^{-1} yr]

		receptor					
		zone1	zone2	zone3	zone4	zone5	zone6
source	zone1	4.997E−10	7.687E−11	1.922E−10	3.844E−11	0.000E+00	0.000E+00
	zone2	0.000E+00	4.466E−10	1.489E−10	2.977E−10	1.489E−10	0.000E+00
	zone3	6.669E−11	1.556E−10	4.224E−10	1.556E−10	2.223E−11	0.000E+00
	zone4	0.000E+00	4.461E−11	4.461E−11	5.800E−10	8.923E−11	0.000E+00
	zone5	0.000E+00	0.000E+00	0.000E+00	9.479E−11	4.740E−10	9.479E−11
	zone6	0.000E+00	0.000E+00	0.000E+00	0.000E+00	0.000E+00	3.136E−10

Table 2.4-18: "Increase in SO_4^{2-} concentration due to a unit amount of SO_2 emissions" ($\Delta C/\Delta E$) [μg m^{-3} kg^{-1} yr]

		receptor					
		zone1	zone2	zone3	zone4	zone5	zone6
source	zone1	1.327E−09	3.316E−10	6.438E−10	2.243E−10	9.754E−11	2.926E−11
	zone2	4.020E−10	4.824E−09	1.407E−09	2.211E−09	1.608E−09	2.010E−10
	zone3	7.709E−10	1.579E−09	3.891E−09	1.358E−09	4.038E−10	1.101E−10
	zone4	1.135E−10	3.405E−10	3.859E−10	3.405E−09	7.490E−10	1.589E−10
	zone5	0.000E+00	4.790E−10	1.597E−10	7.984E−10	4.471E−09	7.984E−10
	zone6	3.397E−11	6.794E−11	3.397E−11	3.397E−11	2.038E−10	1.495E−09

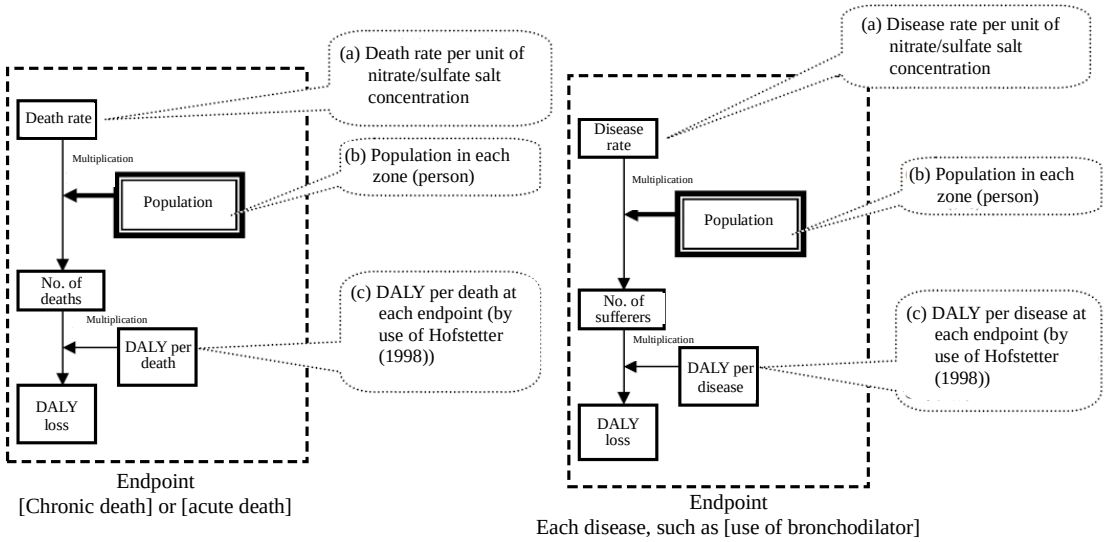


Figure 2.4-8: Flowchart of calculation of "DALY loss at each endpoint per unit of nitrate/sulfate salt concentration" ($\Delta DALY_{\text{each endpoint}}/\Delta C$)

(a), (b), and (c) in the chart correspond to headings in the main text.

Table 2.4-19: "The increasing rate of the death rate and the disease rate at each endpoint per unit of nitrate/sulfate salt concentration"

Type of respiratory disease (endpoint)	"Increasing death rate at each endpoint per unit of nitrate/sulfate salt concentration" [(Risk/$Risk_{baseline}$) $\mu g^{-1}m^3$], Risk=case $person^{-1}$ yr^{-1} "(a) Disease rate at each endpoint per unit of nitrate/sulfate salt concentration" [case $person^{-1}$ $yr^{-1}\mu g^{-1}m^3$]		(c) DALY of 1 death/1 disease at each endpoint [DALY/case]	Geometric diffusion σ^2	Target population
	Nitrate/sulfate †	Geometric diffusion σ^2			
Acute death	Spix et al. (1996),Verhorf et al. (2002) 0.00068	16.0	0.75	5.0	Entire population
Chronic death	(Nitrate) Pope et al. (1995) 0.00643	16.0	6.6	3.0	Aged 30 and over
	(Sulfate) Pope et al. (2002) 0.00769	1.1e－6			
Use of bronchodilator: adult	Dusseldorp et al. (1995) 0.272	16.0	0.00027	2.0	Asthma/adult
Use of bronchodilator: child	Roemer et al. (1993) 0.129	16.0	0.00027	2.0	Asthma/ child
Cough: adult	Dusseldorp et al. (1995) 0.280	6.0	0.00014	2.0	Asthma/ adult
Cough: child	Pope et al. (1992) 0.223	6.0	0.00014	2.0	Asthma/ child
Lower respiratory symptoms (stridor): adult	Dusseldorp et al. (1995) 0.101	6.0	0.00014	2.0	Asthma/ adult
Lower respiratory symptoms (stridor): child	Roemer et al. (1993) 0.172	6.0	0.00014	2.0	Asthma/child
Chronic bronchitis: adult	Abbey et al. (1995) 7.800E-05	16.0	2	3.0	Adult
Chronic bronchitis: child	Dockery et al. (1989) 2.690E-03	6.0	0.025	3.0	Child
Chronic cough	Dockery et al. (1989) 3.460E-03	16.0	0.025	3.0	Child
Days of behavioral restriction	Ostro (1987) 0.042	16.0	0.00027	2.0	Adult
Hospitalization for respiratory system	Dab et al. (1996) 3.460E-06	6.0	0.011	2.0	Entire population
Entry into emergency room (chronic obstructive pulmonary disease)	Sunyer et al. (1993) 1.200E-05	36.0	0.00082	3.0	Entire population
Entry into emergency room (asthma)	Schwarts (1993), Bates et al. (1990) 1.080E-05	36.0	0.00082	3.0	Entire population
Entry into emergency room (inflammation of pharynx/bronchi of preschooler)	Schwarts et al. (1991) 4.860E-05	36.0	0.00082	3.0	Entire population

(a) and (c) in the table correspond to Figure 2.4-8.
† Except for Pope et al. (2002), values for PM2.5 under ExternE were applied.
(Sources) Extern E (European Commission 1999); Pope et al. (2002); the source for the shaded parts is Hofstetter (1998).

Table 2.4-20: The damage function ($\Delta DALY_{each\ endpoint}/\Delta E$) and the damage factor ($\Delta DALY/\Delta E$) for human health concerning pollution by nitrate salt due to NO_2 emissions: pollution by nitrate in Zone 3 due to NO_2 emissions in Zone 4

				source zone / receptor zone	Zone4 / Zone3
(1) $\Delta C/\Delta E$[†1] [$\mu g\ m^{-3}\ kg^{-1}\ yr$]					4.461E−11
Endpoint	Acute death		(a) Death rate per unit of nitrate salt concentration[†2] (Entire population) [case person^{-1} yr^{-1} μg^{-1} m^3]		6.510E−06
			(b) Entire population (person)		37765477
			(c) DALY for acute death [DALY case^{-1}]		7.500E−01
		(2) $\Delta DALY_{each\ endpoint}/\Delta C$[†3] [DALY yr^{-1} μg^{-1} m^3] (= (a)×(b)×(c))			1.844E+02
		(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}] (= (1)×(2))			8.226E−09
	Chronic death	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			6.835E−07
	Use of bronchodilator: adult		(a) Disease rate per unit of nitrate salt concentration (Asthmatic adult) [case person^{-1} yr^{-1} μg^{-1} m^3]		3.512E−01
			(b) Population of asthmatic adults		41256
			(c) DALY for use of bronchodilator [DALY case^{-1}]		2.700E−04
		(2) $\Delta DALY_{each\ endpoint}/\Delta C$[†3] [DALY yr^{-1} μg^{-1} m^3] (= (a)×(b)×(c))			3.912E+00
		(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}] (= (1)×(2))			1.746E−10
	Use of bronchodilator: child	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			1.572E−11
	Cough: adult	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			9.310E−11
	Cough: child	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			1.409E−11
	Lower respiratory symptoms (Stridor): adult	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			3.358E−11
	Lower respiratory symptoms (Stridor): child	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			1.087E−11
	Chronic bronchitis: adult	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			2.850E−07
	Chronic bronchitis: child	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			2.335E−08
	Chronic cough	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			3.003E−08
	Days of behavioral restriction	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			2.072E−08
	Hospitalization for respirator system	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			8.274E−11
	Entry into emergency room (chronic obstructive pulmonary disease)	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			2.139E−11
	Entry into emergency room (asthma)	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			1.925E−11
	Entry into emergency room (inflammation of pharynx/bronchi of preschooler)	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]			8.664E−11
(4) $\Delta DALY/\Delta E$ [DALY kg^{-1}] (= Σ(3))					1.051E−06

(1) to (4) in the table correspond to Figure 2.4-3; (a) to (c) correspond to Figure 2.4-8.

†1 An increase in NO_3^- concentration due to 1kg yr^{-1} of NO_2 emissions

†2 "(a) Acute death rate per unit of nitrate salt concentration" is calculated by "the increase rate of acute deaths per unit of nitrate salt concentration × current death rate." The same applies to chronic death (see 2.4.3 (3) b (a)).

With regard to (a), the values in Table 2.4-19 are converted into values per unit of NO_3^- concentration (NH_4NO_3 is used as nitrate salt and converted by the use of the mass ratio with NO_3^-). This is because (1) in the table expresses an increase in NO_3^- concentration due to 1 kg yr^{-1} of NO_2 emissions.

†3 DALY loss at each endpoint per unit of NO_3^- concentration

b Step 2: Correlation of an increase in the concentration of nitrate/sulfate with DALY loss at each endpoint

According to Step 2 in Figure 2.4-3, "DALY at each endpoint per unit of nitrate/sulfate concentration" ($\Delta DALY_{each\ endpoint}/\Delta C$) was calculated.

The calculation flowchart is as shown in Figure 2.4-8, and is the same as in the case of the primary pollutants in 2.4.3 (2).

The following is an explanation of (a) to (c) in Figure 2.4-8.

(a) Death/disease rates at each endpoint per unit of nitrate/sulfate salt concentration

Like pollution by primary pollutants (see 2.4.3 (2)), the rates were fixed by the use of the results of the epidemiological survey used for ExternE (European Commission 1999) (see Table 2.4-19). However, with regard to chronic death by sulfate salt, reexamination was carried out after LIME1, and a rate was fixed based on a new document (Pope et al. 2002).

(b) Population in each zone

A population is fixed for the target demographic group at each endpoint in (a) (see Table 2.4-19).

(c) DALY of 1 death/1disease at each endpoint

Like the pollution by primary pollutants in 2.4.3 (2), values by Hofstetter (1998) were used (see Table 2.4-19).

c Steps 3 and 4: Calculation of damage functions and damage factors

Steps 3 and 4 in Figure 2.4-3 were used for calculating damage functions and damage factors. The concrete procedure is as follows:

•Steps 3: The "increase in the concentration of nitrate/sulfate due to a unit amount of NO_2/SO_2 emissions" ($\Delta C/\Delta E$) in Step 1 was multiplied by the "DALY at each endpoint per unit of the concentration of nitrate/sulfate" ($\Delta DALY_{each\ endpoint}/\Delta C$) in Step 2 to obtain the damage function at each endpoint ($\Delta DALY_{each\ endpoint}/\Delta E$).

•Step 4: The damage functions in Step 3 at all the endpoints for respiratory disease were totaled to obtain the damage factor ($\Delta DALY/\Delta E$).

Table 2.4-20 shows examples of the calculated damage functions and factors (pollution by nitrate in Zone 3 when NO_2 is emitted in Zone 4). Zones 3 and 4 are located as shown in Figure 2.4-7.

In addition, table 2.4-21 shows the average prefectural damage factor in each zone and the national average.

Table 2.4-21: The damage factor for human health concerning pollution by nitrate/sulfate salt due to NO_2/SO_2 emissions ($\Delta DALY/\Delta E$) [$\Delta DALY\ kg^{-1}$]
The average prefectural damage factor in each zone and the national average

Zone	Pollution by nitrate salt due to emission of 1 kg of NO_2	Pollution by sulfate salt due to emission of 1 kg of SO_2
Average in Hokkaido	1.148E-06	1.088E-05
Average in Tohoku	9.112E-06	8.878E-05
Average in Kanto	1.991E-05	1.479E-04
Average in Chubu	1.732E-05	1.668E-04
Average in Kansai	1.627E-05	1.807E-04
Average in Chugoku/Shikoku	1.541E-05	1.576E-04
Average in Kyushu/Okinawa	1.128E-05	4.582E-05
National average	1.292E-05	1.141E-04

(Note) Prefectures' damage factors were averaged in each zone.

(4) Uncertainty assessment

The damage factors for primary and secondary pollutants obtained in (2) and (3) were totaled to calculate four types of damage factors (damage factors for NO_2 emissions, SO_2 emissions, PM2.5 emissions, and PM10 emissions). Uncertainty assessment of each of these factors was carried out and the obtained statistical data (median, etc.) were used as the final damage factors.

The method for uncertainty assessment was as follows:

・With regard to both primary pollutants and secondary pollutants, the calculation of damage factors had two steps: "Step 1: correlation of the amount of primary pollutant emissions with an increase in the concentration of primary and secondary pollutants" and "Step 2: correlation of an increase in the atmospheric concentration of primary and secondary pollutants with DALY loss at each endpoint." During each of the steps, uncertainty factors were extracted and expressed as a distribution (see Table 2.4-22).

・Although the damage factor for each zone was calculated for the seven zones (Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, Kyushu/Okinawa), because emission zones are often unknown from inventory, the uncertainty factor was used. A distribution was expressed on the assumption that emissions in each of the prefectures in each zone could occur with the same probability.

To examine the distribution for each of the four types of damage factors (damage factors for NO_2 emissions, SO_2 emissions, PM2.5 emissions, and PM10 emissions), a Monte Carlo simulation was carried out by applying random numbers of a normal distribution to the cause of uncertainty and the zones in Table 2.4-22. Figure 2.4-10 shows examples of the obtained distributions, and Table 2.4-24 shows the statistical data.

In addition, to analyze the cause of deviation of damage factor values and the contributions to it, Spearman rank-correlation coefficients were calculated. Table 2.4-25 shows examples.

Moreover, Figure 2.4-11 shows comparison between LIME1 and LIME2 in damage factor.

Table 2.4-22: Method for uncertainty assessment of damage factors for urban air pollution

	2.4.3 (2) Pollution by primary pollutants				*2.4.3 (3)* Pollution by secondary pollutants			
	2.4.3 (2) a Step 1: Correlation of the amount of primary pollutant emissions with an increase in the concentration of primary pollutants		*2.4.3 (2) b* Step 2: Correlation of an increase in the amount of primary pollutants in the atmosphere with DALY loss at each endpoint		*2.4.3 (3) a* Step 1: Correlation of the amount of primary pollutant emissions with an increase in the concentration of secondary pollutants		*2.4.3 (3) b* Step 2: Correlation of an increase in the concentration of secondary pollutants in the atmosphere with DALY loss at each endpoint	
	Uncertainty factor	Assessment method	Uncertainty factor	Assessment method	Uncertainty factor	Assessment method	Uncertainty factor	Assessment method
Damage factors for NO_2 emissions • Chimney • Automobile	The "increase in the concentration of primary pollutants due to a unit amount of primary pollutant emissions" in Table 2.4-8 is an annual average. In reality, the value differs according to weather patterns. The existence of such weather patterns is the uncertainty factor.	Uncertainty was expressed by a standard deviation of values according to the weather patterns mentioned in the left column (Table 2.4-23).	Uncertainty was considered in relation to "(a) the increasing rate of the death rate and the disease rate per unit of primary pollutant concentration" and "(c) DALY for each respiratory disease," components of "relation between an increase in the atmospheric concentration of primary pollutants and DALY at each endpoint."	Uncertainty was expressed by geometric standard deviation in (a) and (c) in the left column by Hofstetter (1998) (see Table 2.4-10).	The uncertainty factor is whether the model has reproduced air pollution concentration.	Uncertainty was expressed by the difference between the calculated values obtained through the verification of the current situation reproducibility of the OPU model (Ikeda 2001; see Figure 2.4-9) and the observed values (standard variation: 2.20).	The same as in the case of pollution by primary pollutants	The same as in the case of pollution by primary pollutants (see Table 2.4-19)
Damage factors for SO_2 emissions • Chimney ・ **Automobile**								
Damage factors for PM2.5 emissions • Chimney • Automobile				-	-	-	-	-
Damage factors for PM10 emissions • Chimney • Automobile				-	-	-	-	-

(*Numbers in italics* correspond to headings of the main text.)

Table 2.4-23: Standard deviation of factors for an increase in primary pollutants according to weather patterns

Zone	Chimney	Automobile
	Standard deviation	Standard deviation
Hokkaido	9.18E+2	5.48E+3
Tohoku	9.40E+2	5.74E+3
Kanto	9.58E+2	6.07E+3
Chubu	8.60E+2	5.02E+3
Kansai	9.18E+2	5.48E+3
Chugoku/Shikoku	8.97E+2	5.31E+3
Kyushu/Okinawa	9.44E+2	5.31E+3

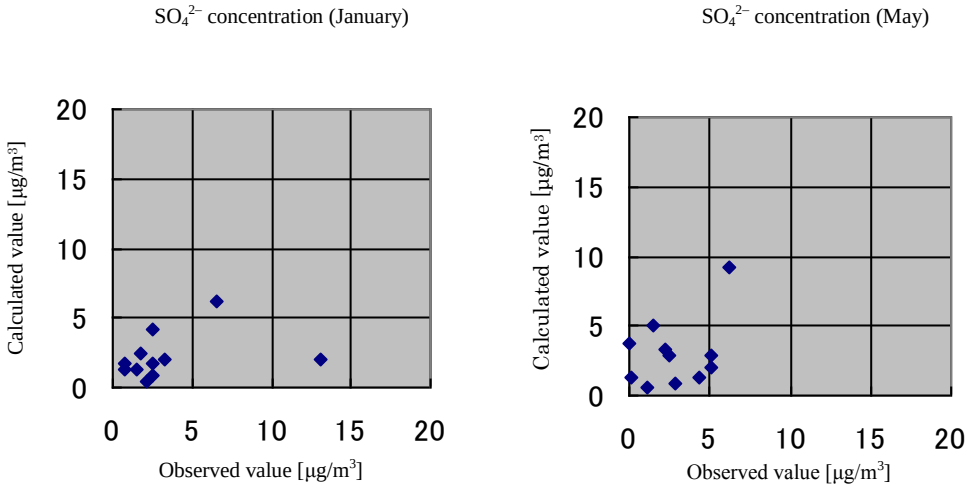
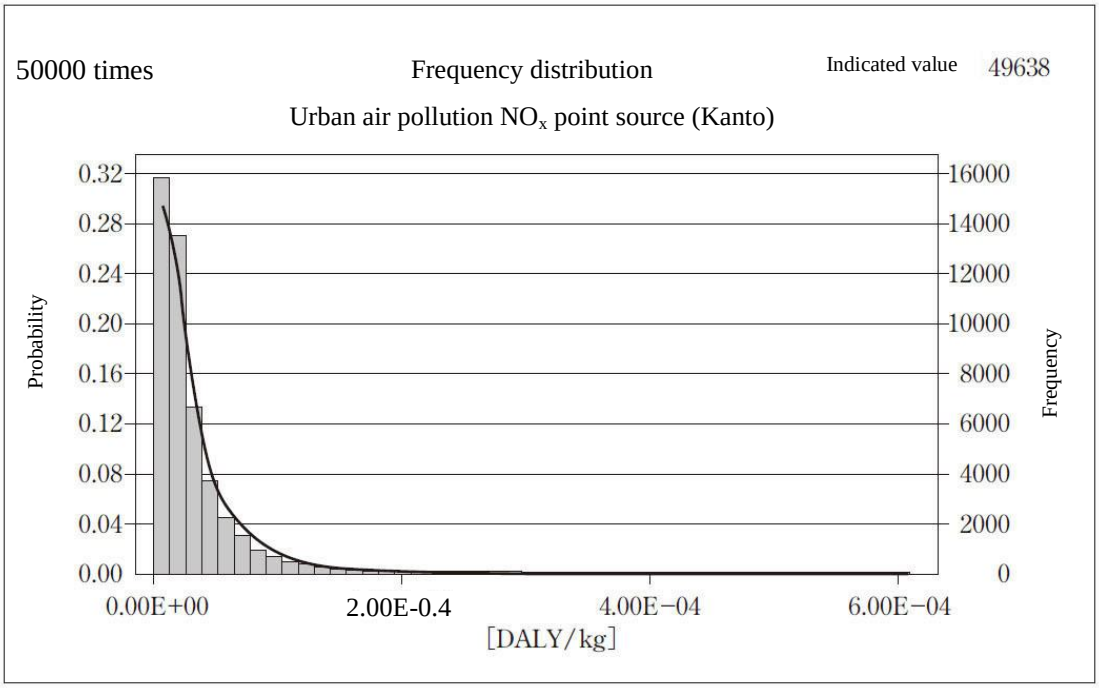
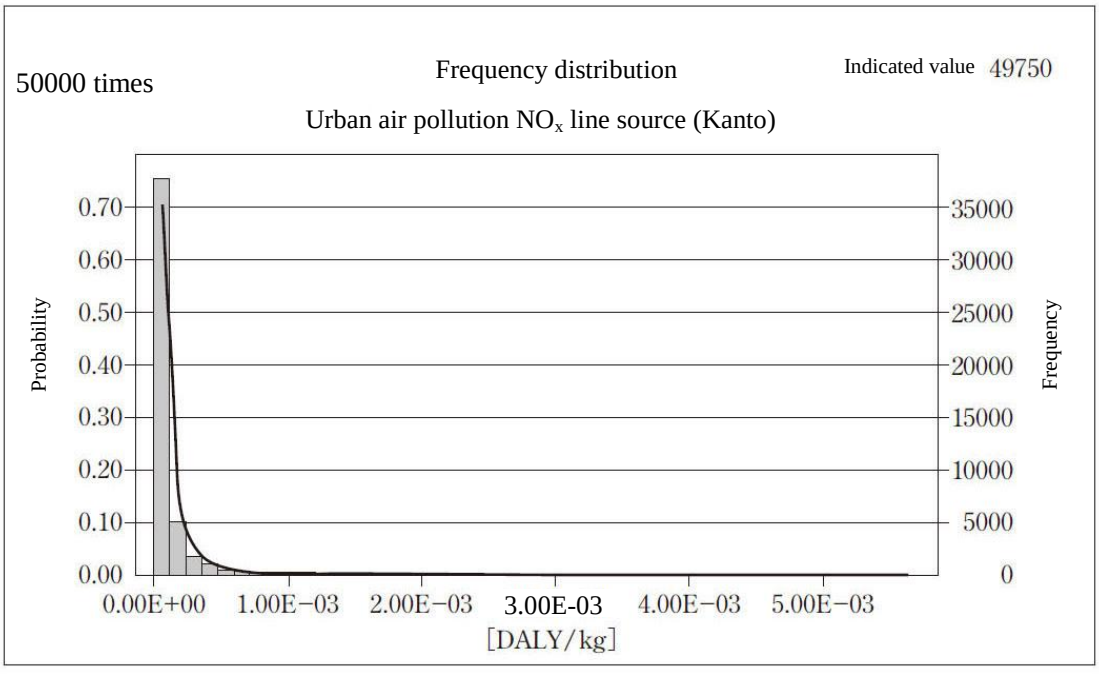


Figure 2.4-9: Verification of the current situation reproducibility of the OPU model (Source) Ikeda (2001)



Chimney



Automobile

Figure 2.4-10: Damage factor distribution obtained from uncertainty assessment of urban air pollution: NO_2, Kanto

Table 2.4-24: Statistical data of damage factors for urban air pollution

		Zone	No. of Calculation	Representative value(Median)	mean value	Standard deviation	dispersion	Kurtosis	Coefficient of variance	10-percentile value	90-percentile value	standard margin of error deviation
NO_2	Chimney	Japan	50000	1.20E-05	2.75E-05	1.07E-04	1.14E-08	2.33E+03	3.88E+00	2.70E-06	5.15E-05	4.77E-07
		Hokkaido	50000	1.21E-06	2.63E-06	8.42E-06	7.09E-11	2.19E+03	3.21E+00	4.06E-07	5.01E-06	3.77E-08
		Tohoku	50000	7.73E-06	1.62E-05	3.97E-05	1.58E-09	1.79E+03	2.45E+00	2.19E-06	3.34E-05	1.78E-07
		Kanto	50000	2.10E-05	5.06E-05	2.02E-04	4.07E-08	1.94E+03	3.99E+00	6.93E-06	9.05E-05	9.02E-07
		Chubu	50000	1.44E-05	2.88E-05	1.06E-04	1.13E-08	9.24E+03	3.69E+00	5.03E-06	5.53E-05	4.75E-07
		Kansai	50000	1.60E-05	3.94E-05	1.75E-04	3.07E-08	2.72E+03	4.45E+00	5.35E-06	6.85E-05	7.84E-07
		Chugoku/Shikoku	50000	1.21E-05	2.24E-05	4.53E-05	2.05E-09	4.28E+02	2.02E+00	4.19E-06	4.39E-05	2.03E-07
		Kyushu/Okinawa	50000	1.06E-05	2.21E-05	6.33E-05	4.00E-09	1.25E+03	2.86E+00	3.68E-06	4.16E-05	2.83E-07
		Tochigi	50000	1.64E-05	3.03E-05	6.12E-05	3.75E-09	6.12E+02	2.02E+00	5.85E-06	5.99E-05	2.74E-07
		Tokyo	50000	3.62E-05	1.31E-04	7.57E-04	5.73E-07	2.66E+03	5.78E+00	1.03E-05	2.14E-04	3.38E-06
	Automobile	Japan	50000	2.13E-05	1.18E-04	1.12E-03	1.25E-06	6.78E+03	9.47E+00	4.81E-06	1.55E-04	5.00E-06
		Hokkaido	50000	1.84E-06	6.72E-06	3.93E-05	1.55E-09	1.63E+03	5.85E+00	5.38E-07	1.06E-05	1.76E-07
		Tohoku	50000	9.12E-06	2.50E-05	1.11E-04	1.24E-08	1.76E+03	4.45E+00	2.59E-06	4.53E-05	4.98E-07
		Kanto	50000	4.15E-05	2.38E-04	1.95E-03	3.81E-06	5.47E+03	8.22E+00	1.05E-05	3.43E-04	8.73E-06
		Chubu	50000	2.08E-05	6.22E-05	3.74E-04	1.40E-07	4.66E+03	6.01E+00	6.61E-06	1.02E-04	1.67E-06
		Kansai	50000	2.85E-05	1.72E-04	1.37E-03	1.87E-06	2.01E+03	7.94E+00	7.65E-06	2.26E-04	6.12E-06
		Chugoku/Shikoku	50000	1.68E-05	4.13E-05	2.45E-04	6.02E-08	9.26E+03	5.94E+00	5.51E-06	7.21E-05	1.10E-06
		Kyushu/Okinawa	50000	1.49E-05	4.97E-05	3.04E-04	9.25E-08	5.95E+03	6.12E+00	4.57E-06	7.81E-05	1.36E-06
		Tochigi	50000	2.21E-05	5.26E-05	1.98E-04	3.92E-08	1.34E+03	3.76E+00	7.32E-06	9.54E-05	8.85E-07
		Tokyo	50000	8.18E-05	5.12E-04	3.56E-03	1.27E-05	3.81E+03	6.96E+00	1.71E-05	8.11E-04	1.59E-05
SO_2	Chimney/Automobile	Japan	50000	1.49E-04	2.64E-04	4.91E-04	2.41E-07	9.80E+02	1.86E+00	2.19E-05	5.76E-04	2.20E-06
		Hokkaido	50000	1.84E-05	2.76E-05	4.02E-05	1.62E-09	2.80E+02	1.45E+00	7.55E-06	5.17E-05	1.80E-07
		Tohoku	50000	1.48E-04	2.49E-04	4.30E-04	1.85E-07	7.97E+02	1.72E+00	5.40E-05	4.97E-04	1.92E-06
		Kanto	50000	2.32E-04	3.86E-04	7.90E-04	6.24E-07	2.50E+03	2.05E+00	8.21E-05	7.57E-04	3.53E-06
		Chubu	50000	2.62E-04	3.91E-04	5.89E-04	3.47E-07	1.71E+03	1.51E+00	1.06E-04	7.45E-04	2.64E-06
		Kansai	50000	2.96E-04	4.46E-04	6.99E-04	4.88E-07	1.29E+03	1.57E+00	1.20E-04	8.46E-04	3.13E-06
		Chugoku/Shikoku	50000	2.29E-04	3.55E-04	5.84E-04	3.41E-07	1.39E+03	1.65E+00	6.53E-05	7.14E-04	2.61E-06
		Kyushu/Okinawa	50000	7.95E-05	1.19E-04	1.81E-04	3.28E-08	1.00E+03	1.52E+00	3.24E-05	2.24E-04	8.11E-07
		Tochigi	50000	2.17E-04	3.54E-04	5.95E-04	3.54E-07	7.26E+02	1.68E+00	7.70E-05	7.01E-04	2.66E-06
		Tokyo	50000	2.95E-04	5.52E-04	1.35E-03	1.83E-06	9.94E+02	2.45E+00	1.02E-04	1.05E-03	6.05E-06

		Zone	No. of Calculation	Representative value(Median)	mean value	Standard deviation	dispersion	Kurtosis	Coefficient of variance	10-percentile value	90-percentile value	standard margin of error deviation
PM 2.5	Chimney	Japan	50000	1.93E-04	5.77E-04	2.46E-03	6.04E-06	8.54E+03	4.26E+00	4.00E-05	1.12E-03	1.10E-05
		Hokkaido	50000	4.70E-05	7.93E-05	1.33E-04	1.78E-08	4.47E+02	1.68E+00	1.48E-05	1.61E-04	5.96E-07
		Tohoku	50000	1.16E-04	2.17E-04	4.11E-04	1.69E-07	4.52E+02	1.90E+00	3.26E-05	4.54E-04	1.84E-06
		Kanto	50000	7.43E-04	1.76E-03	4.86E-03	2.36E-05	1.35E+03	2.75E+00	1.64E-04	3.77E-03	2.17E-05
		Chubu	50000	2.10E-04	4.91E-04	1.17E-03	1.37E-06	9.20E+02	2.39E+00	5.30E-05	1.08E-03	5.24E-06
		Kansai	50000	3.88E-04	1.45E-03	4.20E-03	1.77E-05	4.77E+02	2.91E+00	8.50E-05	3.43E-03	1.88E-05
		Chugoku/Shikoku	50000	1.80E-04	3.14E-04	5.69E-04	3.24E-07	4.06E+02	1.82E+00	5.38E-05	6.37E-04	2.55E-06
		Kyushu/Okinawa	50000	2.89E-04	5.41E-04	1.07E-03	1.14E-06	4.75E+02	1.97E+00	7.52E-05	1.14E-03	4.77E-06
		Tochigi	50000	2.28E-04	3.76E-04	5.81E-04	3.37E-07	1.89E+02	1.55E+00	7.40E-05	7.58E-04	2.60E-06
		Tokyo	50000	3.96E-03	6.65E-03	1.13E-02	1.28E-04	3.09E+02	1.70E+00	1.27E-03	1.34E-02	5.06E-05
PM 2.5	Automobile	Japan	50000	1.33E-03	6.18E-03	2.06E-02	4.24E-04	1.17E+03	3.33E+00	1.92E-04	1.43E-02	9.21E-05
		Hokkaido	50000	1.69E-04	3.34E-04	7.65E-04	5.86E-07	2.38E+03	2.29E+00	4.32E-05	6.97E-04	3.42E-06
		Tohoku	50000	4.40E-04	9.07E-04	2.09E-03	4.38E-06	1.49E+03	2.31E+00	1.08E-04	1.94E-03	9.35E-06
		Kanto	50000	5.26E-03	1.38E-02	3.64E-02	1.33E-03	1.11E+03	2.64E+00	7.50E-04	3.13E-02	1.63E-04
		Chubu	50000	9.84E-04	2.64E-03	7.37E-03	5.43E-05	1.96E+03	2.79E+00	2.02E-04	5.90E-03	3.29E-05
		Kansai	50000	2.55E-03	9.70E-03	2.70E-02	7.31E-04	2.95E+02	2.79E+00	4.04E-04	2.29E-02	1.21E-04
		Chugoku/Shikoku	50000	6.90E-04	1.36E-03	2.94E-03	8.63E-06	1.19E+03	2.15E+00	1.69E-04	2.87E-03	1.31E-05
		Kyushu/Okinawa	50000	9.16E-04	2.24E-03	5.72E-03	3.27E-05	2.48E+03	2.55E+00	1.80E-04	4.98E-03	2.56E-05
		Tochigi	50000	9.86E-04	1.82E-03	3.80E-03	1.44E-05	1.43E+03	2.09E+00	2.73E-04	3.76E-03	1.70E-05
		Tokyo	50000	1.70E-02	3.14E-02	6.19E-02	3.83E-03	4.57E+02	1.97E+00	4.72E-03	6.48E-02	2.77E-04
PM 10	Chimney	Japan	50000	2.38E-05	4.94E-05	1.56E-04	2.42E-08	8.99E+03	3.15E+00	6.99E-06	9.98E-05	6.96E-07
		Hokkaido	50000	2.38E-05	4.94E-05	1.56E-04	2.42E-08	8.99E+03	3.15E+00	6.99E-06	9.98E-05	6.96E-07
		Tohoku	50000	5.40E-05	1.09E-04	2.70E-04	7.29E-08	1.49E+03	2.47E+00	1.60E-05	2.19E-04	1.21E-06
		Kanto	50000	1.81E-04	3.66E-04	1.01E-03	1.03E-06	4.89E+03	2.77E+00	5.47E-05	7.37E-04	4.53E-06
		Chubu	50000	6.99E-05	1.40E-04	3.23E-04	1.04E-07	1.14E+03	2.31E+00	2.02E-05	2.84E-04	1.44E-06
		Kansai	50000	9.41E-05	1.92E-04	4.32E-04	1.87E-07	5.05E+02	2.25E+00	2.70E-05	3.92E-04	1.93E-06
		Chugoku/Shikoku	50000	5.74E-05	1.16E-04	3.07E-04	9.40E-08	6.71E+03	2.65E+00	1.69E-05	2.34E-04	1.37E-06
		Kyushu/Okinawa	50000	3.22E-04	6.60E-04	1.53E-03	2.33E-06	8.57E+02	2.31E+00	9.36E-05	1.34E-03	6.82E-06
		Tochigi	50000	1.16E-04	2.32E-04	5.12E-04	2.63E-07	9.08E+02	2.21E+00	3.49E-05	4.74E-04	2.29E-06
		Tokyo	50000	2.01E-03	4.08E-03	1.05E-02	1.09E-04	3.68E+03	2.56E+00	5.95E-04	8.19E-03	4.67E-05

		Zone	No. of Calculation	Representative value (Median)	mean value	Standard deviation	dispersion	Kurtosis	Coefficient of variancet	10-percentile value	90-percentile value	standard margin of error deviation
PM 10	Automobile	Japan	50000	8.70E-05	2.06E-04	6.98E-04	4.87E-07	9.77E+03	3.39E+00	2.05E-05	4.21E-04	3.12E-06
		Hokkaido	50000	8.70E-05	2.06E-04	6.98E-04	4.87E-07	9.77E+03	3.39E+00	2.05E-05	4.21E-04	3.12E-06
		Tohoku	50000	2.19E-04	4.88E-04	1.49E-03	2.21E-06	3.83E+03	3.05E+00	5.41E-05	1.00E-03	6.65E-06
		Kanto	50000	7.86E-04	1.72E-03	4.36E-03	1.90E-05	7.74E+02	2.54E+00	2.00E-04	3.48E-03	1.95E-05
		Chubu	50000	2.79E-04	6.34E-04	2.95E-03	8.70E-06	2.11E+04	4.65E+00	6.96E-05	1.28E-03	1.32E-05
		Kansai	50000	4.12E-04	9.13E-04	3.05E-03	9.30E-06	1.31E+04	3.34E+00	1.04E-04	1.87E-03	1.36E-05
		Chugoku/Shikoku	50000	2.40E-04	5.28E-04	1.30E-03	1.68E-06	4.72E+02	2.46E+00	6.02E-05	1.09E-03	5.80E-06
		Kyushu/Okinawa	50000	1.15E-03	2.70E-03	8.34E-03	6.96E-05	2.12E+03	3.09E+00	2.75E-04	5.53E-03	3.73E-05
		Tochigi	50000	4.98E-04	1.10E-03	2.62E-03	6.87E-06	8.96E+02	2.38E+00	1.31E-04	2.28E-03	1.17E-05
		Tokyo	50000	8.51E-03	1.92E-02	4.76E-02	2.27E-03	4.53E+02	2.48E+00	2.19E-03	3.96E-02	2.13E-04

Table 2.4-25: Rank correlation coefficients of damage factors for air pollution (example)

NO_2: chimney

Uncertainty item	Rank correlation coefficient
Disease rate per unit of nitrate concentration (chronic bronchitis: adult)	0.541
Disease rate per unit of NO_2 concentration (acute death)	0.429
DALY (acute death)	0.207
Disease rate per unit of nitrate salt concentration (chronic death)	0.204
DALY (chronic bronchitis: adult)	0.202
NO_x emission zone: point source (Kanto)	0.151
Disease rate per unit of nitrate salt concentration (chronic cough)	0.102

NO_2: automobile

Uncertainty item	Rank correlation coefficient
Disease rate per unit of NO_2 concentration (acute death)	0.623
NO_x emission zone: line source (Kanto)	0.303
Disease rate per unit of nitrate salt concentration (chronic bronchitis: adult)	0.297
DALY (acute death)	0.279
DALY (chronic bronchitis: adult)	0.119
Disease rate per unit of nitrate salt concentration (chronic death)	0.100

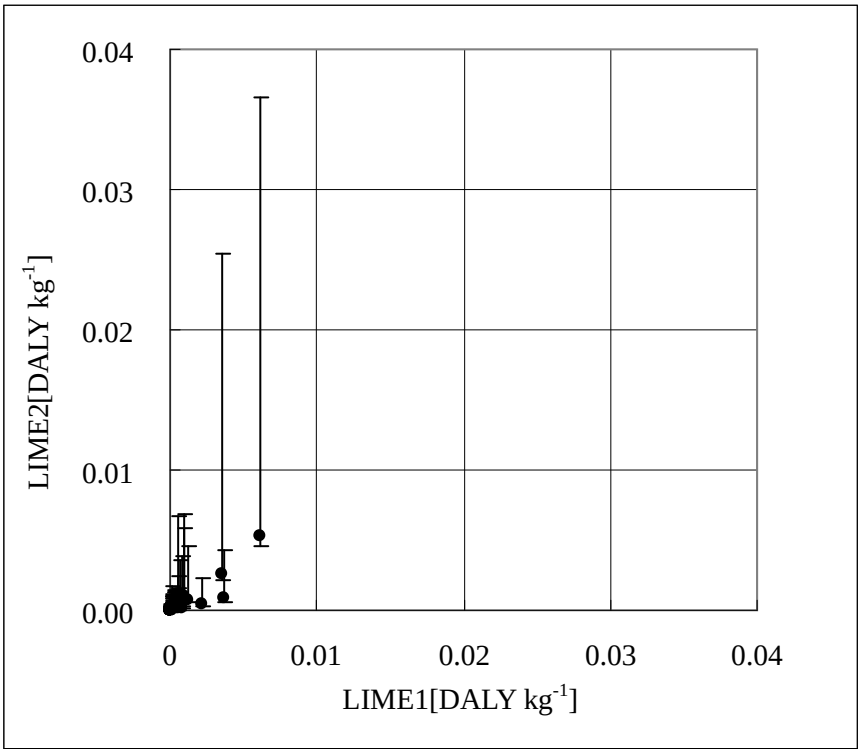


Figure 2.4-11: Comparison between LIME1 and LIME2 in damage factor

The median obtained from uncertainty analysis is used as the damage factor under LIME2 (see Table 2.4-24).

2.4.4 Procedure for impact assessment of urban air pollution

LCA users can select what meets their purpose from among characterization, damage assessment, and weighting and use it for LCA, etc.

(1) Characterization

The result of characterization $CI^{AirPollution}(X)$ can be obtained from the inventory $Inv(X)$ of the atmospheric emissions of the primary pollutant X (NO_2 or SO_2) and the characterization factor $CF^{AirPollution}(X)$ (see Equation 2.4-2).

$$CI^{AirPollution} = \sum_{X} IF^{AirPollution}(X) \cdot Inv(X) \qquad (2.4\text{-}2)$$

The characterization factor $CI^{AirPollution}(X)$ is shown in Appendix A1.

(2) Damage assessment, weighting

a Damage assessment

The result of damage assessment $DI(Safe)$ can be obtained from $Inv(X, Region, Source)$ of urban air pollutants and the damage factor for each area of protection $Safe\ DF^{AirPollution}(Safe, X, Region, Source)$ (see Equation 2.4-3). $DI(Safe)$ means the amount of potential damage to *Safe*, the area of protection from air pollutant emissions.

$$DI(Safe) = \sum_{X} \sum_{Region} \sum_{Source} DF^{AirPollution}(Safe, X, Region, Source) \cdot Inv(X, Region, Source) \qquad (2.4\text{-}3)$$

When an LCA user carries out damage assessment for urban air pollution, inventory data must be matched with the form of damage factor as follows:

<Division by the source of emissions and the particle diameter>

・The sources of emissions of NO_x and PM should be divided into chimneys and automobiles.

・Inventory should be calculated in the form of PM2.5 or PM10. An inventory database may be presented in the form of soot or dust. In such a case, it is necessary to convert the inventory data into PM2.5 or PM10 before applying the data to LIME.

<Division by zone>

・It is desirable to divide inventory data by emission zone. However, if it is impossible to divide inventory data by emission zone, assessment can be made by the use of the average damage factor in Japan $DF^{AirPollution}(Safe, X, Average, Source)$ (see Equation 2.4-4).

$$DI(Safe) = \sum_{X} \sum_{Source} DF^{AirPollution}(Safe, X, Average, Source) \cdot Inv(X, Source) \qquad (2.4\text{-}4)$$

b Integration

As in the case of a, in this impact category "urban air pollution," damage assessment can be carried out for human health. If an area of protection is common to two or more impact categories, comparison and integration are possible.

When integration is carried out, the integration factor $IF^{AirPollution}(X)$ is used after economic conversion or non-dimensionalization of the impact on human health. The single index *SI* can be obtained from each pollutant's *Inv* (*X*) and the integration factor $IF^{AirPollution}(X)$ (see Equation 2.4-5). The obtained result can be compared directly or added to assessment results for other impact categories.

$$SI = \sum_{X} IF^{AirPollution}(X) \cdot Inv(X) \qquad (2.4\text{-}5)$$

Appendix A2 shows the damage factors $DF^{AirPollution}$ (*Safe, X, Region, Source*) and $DF^{AirPollution}$ (*Safe, X,* A*verage, Source).* Appendix A3 shows the integration factor $IF^{AirPollution}(X)$.

Acknowledgement

For the purpose of the development of this LCIA method, the source-receptor matrix for nitrate and sulfate became available through the kindness of Mr. Yuko Ikeda, Professor Emeritus of Osaka Prefecture University, and Mr. Hagimoto (Osaka Prefecture University at that time). We are very grateful for their kindness.

[References for Section 2.4]

Busch, Buschbom RL, Cannon WC, Lauhala KE, Miller FJ, Graham JA, Smith LG (1986): Effects of ammonium nitrate aerosol exposure on ling structure of normal and elastase – impaired rats and guinea pigs, Environ. Res., 39: 237 - 252

European Commission (1999): ExternE: Externalities of Energy, Vol. 7 Methodology 1998 update, Office for Official Publications of the European Communities

Gifford FA (1961): Use of Routine Meteorological Observations for Estimating Atmospheric Dispersion, Nuclear Safety, 2 (4)

Goedkoop, Spriensma (2000): The Eco─indicator 99, A Damage Oriented Method for Life Cycle Impact Assessment, Methodology Report 2nd edition

Hofstetter P (1998): Perspectives in Life Cycle Impact Assessment; A Structured Approach to Combine Models of the Technosphere, Ecosphere, and Valuesphere, Kluwers Academic Publishers

Holland (1953): A Meteorological Survey of the Oak Ridge Area, Atomic Energy Comm. Report ORO─99, Washington D.C.

Loscutoff SM, Cannon WC, Buschbom RL, Busch RH, Kirkland BW (1985): Pulmonary function in elastase-treated guinea pigs and rats exposed to ammonium sulfate or ammonium nitrate aerosols, Environ. Res., 36:170 - 180

Pepelko WE, Mattox JK and Cohen AL (1980): Toxicology of ammonium sulfate in the lung. Bull. Environm. Contam. Toxicol. 24, pp.156-160.

Pope, CA, Burnett, RT, Thun, MJ, Calle, EE, Krewski, D, Ito, K & Thurston, GD (2002): Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution, Journal of the American Medical Association, vol. 287, no. 9, pp. 1132-1141.

Steen B (1999): A Systematic Approach to Environmental Priority Strategies in Product Development (EPS) Version 2000 - Models and Data of the Default Method, CPM Report 1999:5, Chalmers University of Technology, Göteborg, p. 312

Turner (1964): A diffusion Model for an Urban Area, J. of Appl. Met., 3 (1)

Ikeda Y (2001): Establishment of Comprehensive Measures for Control of the Amount of Air Pollutant Emissions in the Whole East Asia, Report on Research Results for FY1997 to FY2000 Scientific Research Subsidies (Basic Research (B) (1)) (Mar. 2001)

Ohara T, Uno I (1997): Numerical Simulation of Mesoscale Air Pollution, Journal of Japan Society for Atmospheric Environment, 32 (3): A39 - A57

Kasahara M (1994): Behavior of Particles in the Atmosphere – Formation, Transformation, Removal, Journal of Japan Society for Atmospheric Environment, 29 (6): A93 - A101

Kawamura T, Iwaki H (ed.) (1988): 2 Atmospheric Environment, Environmental Science I, Natural Environment System, Asakura Publishing

Environmental Agency, Air Quality Bureau, Air Pollution Control Bureau (supervision), Committee on Suspended Particulate Matters (1997): Suspended Particulate Matter Pollution Prediction Manual, Toyokan Publishing

Environmental Research and Control Center, Committee on Nitrogen Oxide [new edition] (2000): Nitrogen Oxide Control Manual

Higashino H (1997): Research on Emission and Transportation of Air Pollutants in East Asia, doctoral thesis at Osaka Prefecture University

Muto N (1979): Long-term average puff model for urban pollution prediction, Journal of Japan Society for Atmospheric Environment, 14 (8): 340 - 352

1999 Survey on the Relation between Environmental Impact and Damage – Preparation of a Pollution Map (March 2003)

1997 Report on the Results of Operations Entrusted by the Environment Agency: Comprehensive Survey on the Amount of Air Pollutant Emissions (1998)

2.5 Photochemical Oxidant

2.5.1 What phenomenon is photochemical oxidant?

Although photochemical oxidant is a part of urban air pollution described in the preceding section (2.4), under LIME it is treated as an independent impact category.

Photochemical oxidant is a main component of photochemical smog, which occurs mainly in summer, and gives impact on the human respiratory system and plant leaves.

Photochemical smog first occurred in Los Angeles around 1940 and has become a problem since around 1970 in Japan. It has become a serious air pollution problem not only in advanced countries but also in major cities of developing countries, such as Mexico City. In Japan, during the 1970s, there was a year when photochemical smog warnings were issued on a total of 328 days. Although the number of such days has recently decreased, it has still been around 100 every year.

The main components of photochemical oxidant are ozone (O_3) and peroxyacetyl nitrate (PAN), with ozone occupying the majority. From the outset, ozone has existed in the natural world. If there are volatile organic compounds (VOCs) and nitrogen oxide (NO_x) in the atmosphere, a large quantity of ozone will be produced from them through photochemical reaction and have impact on organisms.

(1) Causation of photochemical oxidant

After emissions, VOCs and NO_x (both are called ozone "precursor substances") cause photochemical reaction by ultraviolet rays and produce ozone. If human beings and other organisms are exposed to ozone, they may receive harmful impact according to the amount of exposure. Figure 2.5-1 shows the causation of ozone. The first half of the causation will be described herein, while the second half will be described in (2) below.

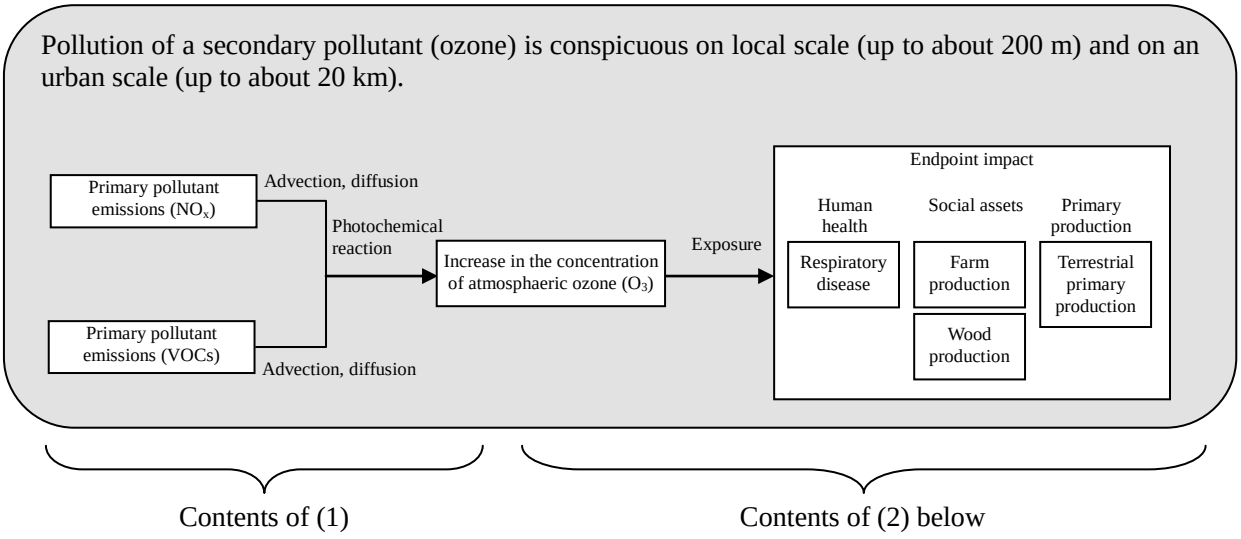


Figure 2.5-1: Cause effect chain of photochemical oxidant
(1) and (2) in the figure correspond to headings in the main text.

a Emission of ozone precursor substances

Because, among ozone precursor substances, NO_x has already described in 2.4.1 (1) a, an explanation herein is given about VOCs.

VOCs are organic compounds that mostly move as gas in the atmosphere when being emitted into the environment (Sakurai 2000). The number of types is more than 100 and there are an extremely wide range of types (see Table 2.5-1).

Column 2.5-1

Tropospheric ozone and stratospheric ozone

The ozone covered by this impact category is the tropospheric ozone existing between the surface of the ground and about 12 km above the ground.

"Ozone layer depletion" (Section 2.1) covers the destruction of ozone existing in the stratosphere (about 12 to 50 km above the ground), the layer just above the troposphere (see Figure 2.5-A).

Tropospheric ozone and the stratospheric ozone are the same chemical substance. However, while stratospheric ozone absorbs harmful ultraviolet rays from the sun and enables organisms to live, tropospheric ozone gives harmful impact to organisms as described in this section.

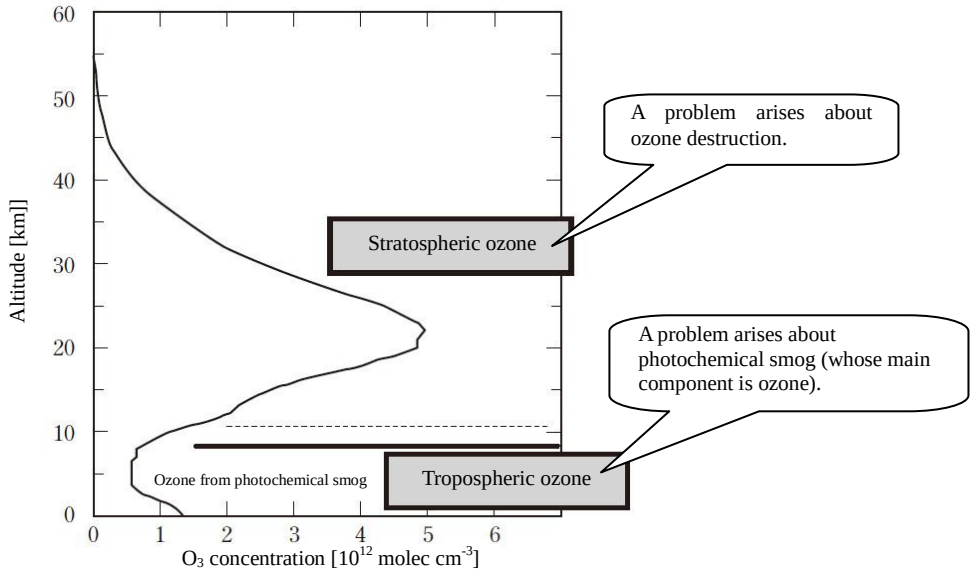


Figure 2.5-A: Photochemical oxidant ozone and ozone in the destructed ozone layer
(Source) Prepared based on Imamura (2001)

Table 2.5-1: Examples of VOCs

	Concrete examples
Alkanes (hydrocarbons of methane series)	Methane, ethane, propane, isopentane, etc.
Alkenes (hydrocarbons of ethylene series)	Ethylene, propylene, isoprene, 1,3-butadiene, etc.
Alkynes (hydrocarbons of acetylene series)	Acetylene, etc.
Aromatic series	Benzene, toluene, *o*-xylen, *m*-xylen, *p*-xylen, ethyl benzene, etc.
Aldehydes	Formaldehyde, acetaldehyde, etc.
Ketones	Acetone, methyl ethyl ketone, etc.
Ethers	Dimethyl ether, diethyl ether, etc.
Alcohols	Methyl alcohol, ethyl alcohol, butyl alcohol, etc.

(Sources) Prepared by Mizuho Information and Research Institute with reference to National Institute for Environmental Studies (2001) and Andersson-Skold et al. (1992).

There are two processes of occurrence of VOCs: artificial processes and natural processes (plants).

Artificial processes include evaporation from fuels, solvents, paints, and other petrochemical products, and emission during burning. Concrete sources are oil refineries, oil tank facilities, oil stations (evaporation from fuels), factories (evaporation from solvents, burning of fuels), outdoor painting (evaporation from paints, etc.), automobiles (burning of and evaporation from fuels), etc.

b Creation of ozone from the ozone precursor substances

This part describes the process of ozone creation, the influencing factors, and the characteristics of ozone concentration.

a) Process of creation of ozone

Ozone has existed in the natural world from the outset and has repeated formation and disappearance as described in 1) to 3) below.

1) Photodissociation of NO_2 by ultraviolet rays

Atmospheric NO_2 is dissociated into NO and O (oxygen atom) by ultraviolet rays.

$$NO_2 + h\nu \rightarrow NO + O$$

2) Creation of ozone

The oxygen atom in 1) is combined with O_2 (oxygen molecule) and creates ozone.

$$O + O_2 + M \rightarrow O_3 + M$$

3) Consumption of ozone by NO oxidation (NO_2 production)

The ozone produced in 2) disappears through oxidation of NO.

$$NO + O_3 \rightarrow NO_2 + O_2$$

If only NO_x is present in the atmosphere, ozone does not increase, because the balance between the production and disappearance of ozone is maintained through the cycle described in 1) to 3) above. However, if VOCs are present in the atmosphere, a large quantity of ozone is typically produced by the processes described in 4) and 5) below (see Figure 2.-5-2).

4) Resolution of VOCs and production of radicals

VOCs are resolved by attacks from ozone and OH radicals and produce radicals.

Because radicals are very highly reactive, a chain reaction occurs and results in the production of various additional radicals (hydroperoxyl radical (HO_2・) (・ is a sign that indicates a radical), alkyl radical (R・), alkoxy radical (RO・), alkylperoxy radical (RO_2・), etc.).

The number of carbons in VOCs decreases through the creation of the above-mentioned radicals. This cycle is repeated until VOCs disappear.

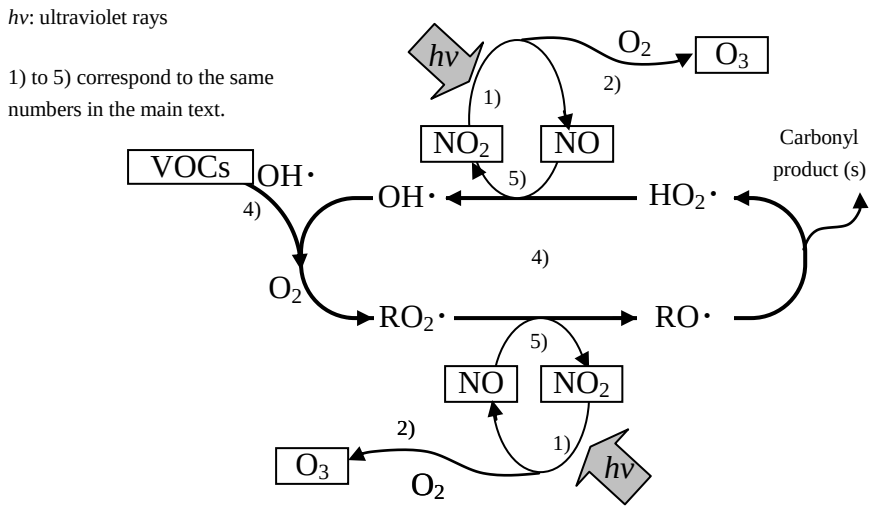


Figure 2.5-2: Pattern diagram of the process of ozone creation through resolution of VOCs and oxidation of NO

(Sources) Prepared based on USEPA (1971), Jenkin et al. (1999), and Wakamatsu et al. (2001)

5) Oxidation of NO by radicals

NO is oxidized by the radicals produced through process 4) above and becomes NO_2.

Unlike process 3), this process 5) produces NO_2 without consuming ozone. NO_2 produces ozone in the reaction 1) and 2) above. Because of this, the production of ozone is accelerated.

Column 2.5-2

What is a radical?
"A molecule contains an even number of electrons, pairs of which distribute around the atomic nucleus and contribute to the chemical binding of molecules. If the number of electrons is odd, one electron does not form a pair – that is, it is an unpaired electron. If a chemical combination has an unpaired electron, it is called a "free radical," or simply a "radical." [……] Because radicals are unstable and responsive, they promptly respond to each other or to stable molecules and cease to be radicals. [……]" (Source) Ito et al. (2000)

b) Actors that influence ozone creation

Ozone creation is influenced by the status of pollution by VOCs and NO_x. With regard to weather conditions, it is easier for ozone to be created when insolation is strong and the temperature is high (see Table 2.5-2).

Table 2.5-2: Factors that influence ozone creation

Factor		What influence does the factor cause?
Status of air pollution	Type of VOCs	• Because the speed of response to OH radicals and degraded products differ among types of VOCs, they differ in the easiness of creating ozone.
	Concentration ratios of NO_x and VOCs in the atmosphere	• The easiness of creating ozone differs according to the concentration ratios of NO_x and VOCs in the atmosphere. As the case may be, the emission of NO_x or VOCs may reduce ozone concentration (Shinozaki et al. (1984a, 1984b), etc.).
Weather		• If insolation or temperature becomes higher, many responses related to ozone creation increase in speed. • Atmospheric movement facilitates the diffusion of NO_x and VOCs. Concretely, when the sun rises, vertical mixture becomes active because of convection and the stable layer near the ground is destroyed, with the result that the so-called mixed layer grows. In the mixed layer, it is easier for NO_x and VOCs to be mixed uniformly. • As a result of growth of the mixed layer, stratospheric ozone is taken in from the upper layer. This is because stratospheric ozone may reach the ground during the period between the end of winter and the beginning of spring because of the jet stream, the passing of a cold front, and the passing of migratory anticyclone (Reference: Wakamatsu et al. (2001)) (Utsunomiya et al. (1994)).

As described above, the process of ozone creation is very complicated. Because of this, the relation betrween NO_x and VOCs emissions and ozone concentration is not linear. In addition, because weather conditions and air pollution differ, easiness of ozone creation differs among zones.

c) Characteristics of ozone concentration

If there is a change in the amount of precursor substance emissions or the amount of insolation during a day, the ozone concentration also changes. The typical change pattern is as follows: the concentration is low early in the morning and, with the passage of time, gradually rises because of an increasing amount of precursor substance emissions from factories and automobiles and an increase in insolation; the concentration reaches a peak in the afternoon and decreases afterwards.

After precursor substance emissions, ozone is created through reaction that lasts several hours to about one day. Because of this, ozone concentration does not necessarily rise near sources of emissions, and pollution extends on a mesoscale (up to 200 km) (Ohara et al. 1997).

(2) Endpoints of photochemical oxidant

a Human health

Because ozone is strongly oxidative and highly responsive, it has harmful impact on the human body. If ozone is absorbed in the respiratory system, it is likely to reach deep parts of the lungs. It causes symptoms such as a stimulus to the nose or the throat, asthma, chronic

bronchitis, and a decline in lung function. In addition, it causes a stimulus to the mucous membrane of the eyes.

b Plants

Ozone brings about various types of damage to plants. Damage can be roughly divided into visible damage and invisible damage.

Visible damage is leaves' whitening, browning, and blackening. If damage worsens from pigment disorder to cellular necrosis, the function of leaves greatly declines.

In addition, invisible damage occurs when ozone is taken in from pores on leaves and influences respiration, photosynthesis, transpiration, enzymatic activity, etc. Even if visible damage does not appear, invisible damage may appear.

These types of damage hinder the growth of plants.

2.5.2 Characterization of photochemical oxidant

(1) Existing characterization factors for photochemical oxidant

Indices for the power to form oxidant have been used as characterization factors for photochemical oxidant, such as the photochemical oxidant creation potential (POCP) (UNECE1990; Derwent et al. 1991) and the incremental hydrocarbon reactivity (IR) (Carter et al. 1989).

Of them, POCP is an index developed for the assessment of VOCs emission scenarios. POCP is calculated by dividing the ozone concentration at the time of a change in the amount of emission of a certain type of VOC by the ozone concentration at the time of a change in the amount of ethylene emissions.

$$POCP_i = (a_i / b_i) \,/\, (a_{C_2H_4} / b_{C_2H_4}) \quad (2.5\text{-}1)$$

In this equation, *a* is an increase in the oxidant concentration at the time of emission of substance i or ethylene (C_2H_4), and *b* is the amount of emissions of substance *i* or ethylene.

POCP, which was developed in 1990, was adopted for the UN Economic Commission for Europe's Convention on Long-range Transboundary Air Pollution. It has been widely used in the field of LCA, too (Heijungs et al. 1992). Although POCP was an average-type index at that time, Derwent et al. adopted a marginal-type index for assessing an increment in the ozone concentration at the time of additional emissions of ozone precursor substances to the current emission scenario since 1996. There are also other POCPs developed by researchers other than Derwent et al.

On the other hand, because IR indicates a change in the ozone concentration at the time of emission of a certain type of VOC, it does not effect standardization from ethylene and other specific substances like POCP. Because of this, IR cannot be used as a characterization factor as it is.

$$IR_i = \Delta[O_3] \,/\, \Delta m_i \quad (2.5\text{-}2)$$

In this equation, $\Delta[O_3]$ is an increase in the ozone concentration with an increase in the amount of emissions of the type *i* of VOC, and Δm_i is an increase in the amount of emissions of the type *i* of VOC. IR is a marginal-type index that indicates to what extent a change in the amount of emission of a certain type of VOC influences a change in the ozone concentration under the baseline scenario.

POCP and IR have been calculated for the whole of Europe and urban areas in the US, respectively.

(2) Characterization factor for photochemical oxidant under LIME

When the damage factor *DF* is calculated in the following Section 2.5.3, "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE) will be calculated (see 2.5.3 (2)). The characterization factor for photochemical oxidant is calculated by dividing each VOC's ΔC/ΔE by ethylene's ΔC/ΔE.

Under LIME, this characterization factor is called the "ozone conversion equivalency factor" (OCEF).

As described in Section 2.5.3, photochemical oxidant is a highly regional environmental problem. Unlike POCP and IR in (1) above, OCEF under LIME is a characterization factor that reflects weather and air pollution in Japan and is calculated for each zone in Japan.

In addition, while POCP and IR are calculated under some weather and air pollution conditions (for example, average conditions in the summer season, conditions whereby the ozone concentration becomes maximum, etc.), OCEF are obtained by averaging OCEFs under various conditions concerning weather in each zone, etc (see Section 2.5.3). Because of this, it can be said that OCEF is a characterization factor that fully represents the conditions in each zone.

2.5.3 Damage assessment of photochemical oxidant

(1) Basic policies for calculation of damage functions and factors

Eco-indicator 99 (Goedkoop et al. 2000) and the Environmental Priority Strategies (EPS) (Steen 1999) in Europe have adopted an endpoint approach for the impact category of photochemical oxidant by the existing LCA method. LIME also has adopted an endpoint approach for the assessment method.

Photochemical oxidant is a very regional environmental problem because it is greatly influenced by regional weather and air pollution conditions. Because of this, under LIME, damage functions and factors are calculated for each zone.

The object of calculation and the calculation method are described as follows:

a Object of calculation

The calculation covers the following category endpoints:

1) Category endpoints of human health

Endpoints were selected where the relation between the air pollutant concentration and the death/disease rates has been assessed quantitatively based on epidemiological surveys, etc.

2) Category endpoints of social assets

Decreases in the values of farm production and wood production due to ozone were selected.

3) Category endpoint of primary production

A decrease in terrestrial net primary productivity (NPP) due to ozone was selected.

Table 2.5-3 shows details of these category endpoints. The amount of damage calculated for each endpoint in the table is used as the damage function. The total of damage functions in each area of protection is the damage factor.

Table 2.5-3: Category endpoints covered by the calculation of damage functions and factors

Area of protection	Category endpoint		Object of calculation of damage function	Object of calculation of damage factor
Human health	Respiratory disease	• Acute death	Increase in the number of acute deaths (DALY-converted))	○
		• Asthma spasm	Increase in the number of sufferers (DALY-converted)	
		• Day of slight behavioral restriction	Increase in the number of sufferers (DALY-converted)	
		• Hospitalization for respiratory system	Increase in the number of sufferers (DALY-converted)	
		• Symptom days	Increase in the number of sufferers (DALY-converted)	
		• Entry into emergency room (asthma)	Increase in the number of sufferers (DALY-converted)	
Social assets	Agri. production	• Impact of damage to farm products	Decrease in the value of farm production	○
	Wood production	• Impact of damage to trees	Decrease in the value of wood production	○
Primary production	Terrestrial eco-system	• Impact of damage to plants	Decrease in terrestrial NPP	○

b Assessment method

Damage functions and factors were calculated according to the causal channels (see Figure 2.5-1) by the following Steps 1 to 4 (see Figures 2.5-3 to 2.5-5).

Step 1: The relation between the amount of VOCs emissions and an increase in the ozone concentration in the atmosphere was quantified.

Step 2: The relation between the increase in the concentration in Step 1 and the amount of damage at each endpoint was quantified.

Step 3: The damage function at each endpoint was calculated by combination of Steps 1 and 2.

Step 4: The total of damage functions in Step 3 for each area of protection was used as the damage factor.

Of these steps, Step 1 will be explained in (2) below. Steps 2 to 4 will be explained in (3) to (5) below concerning each area of protection – human health, social assets, and primary production.

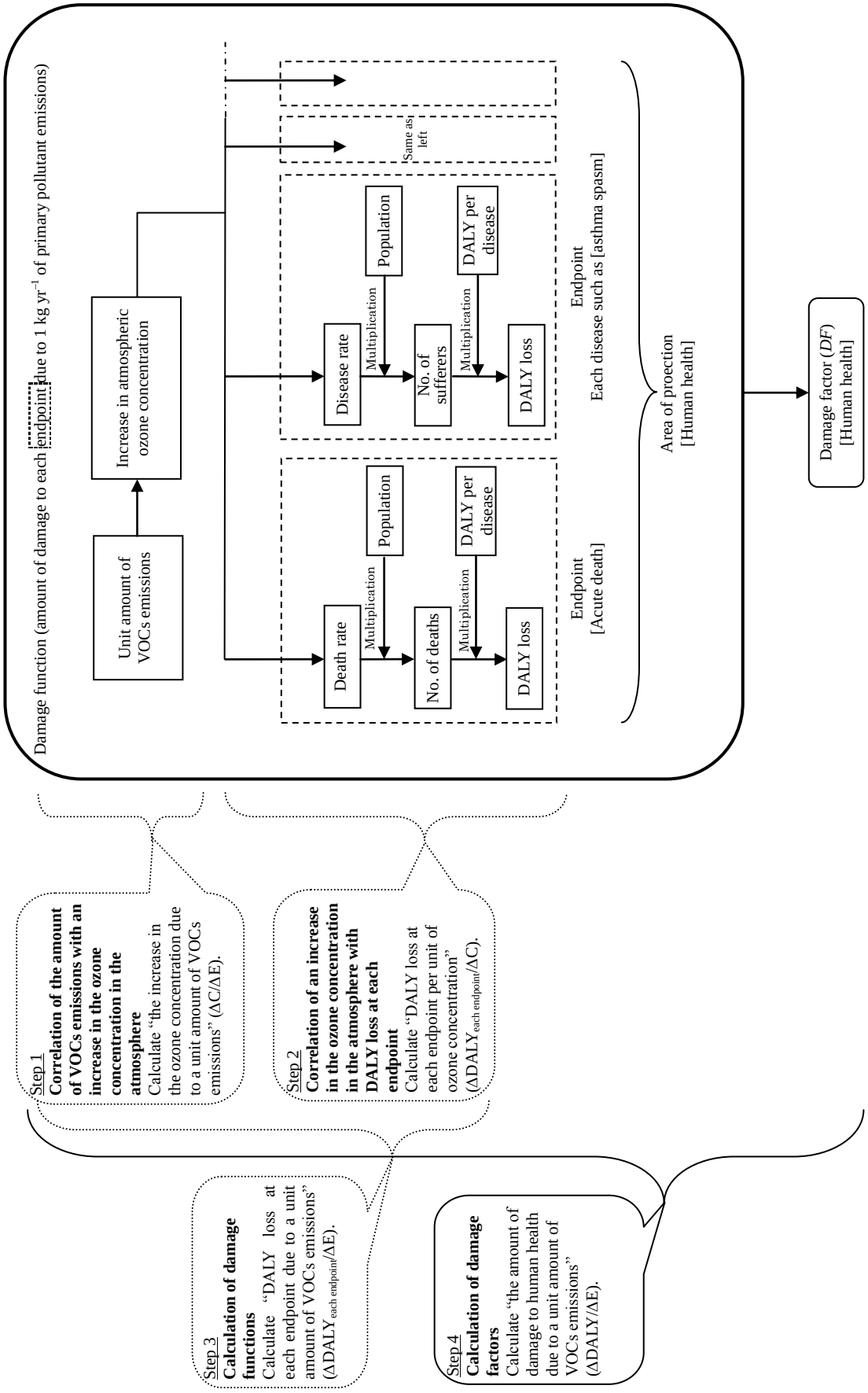


Figure 2.5-3: Flowchart of calculation of damage functions and damage factors for photochemical oxidant (area of protection: human health)

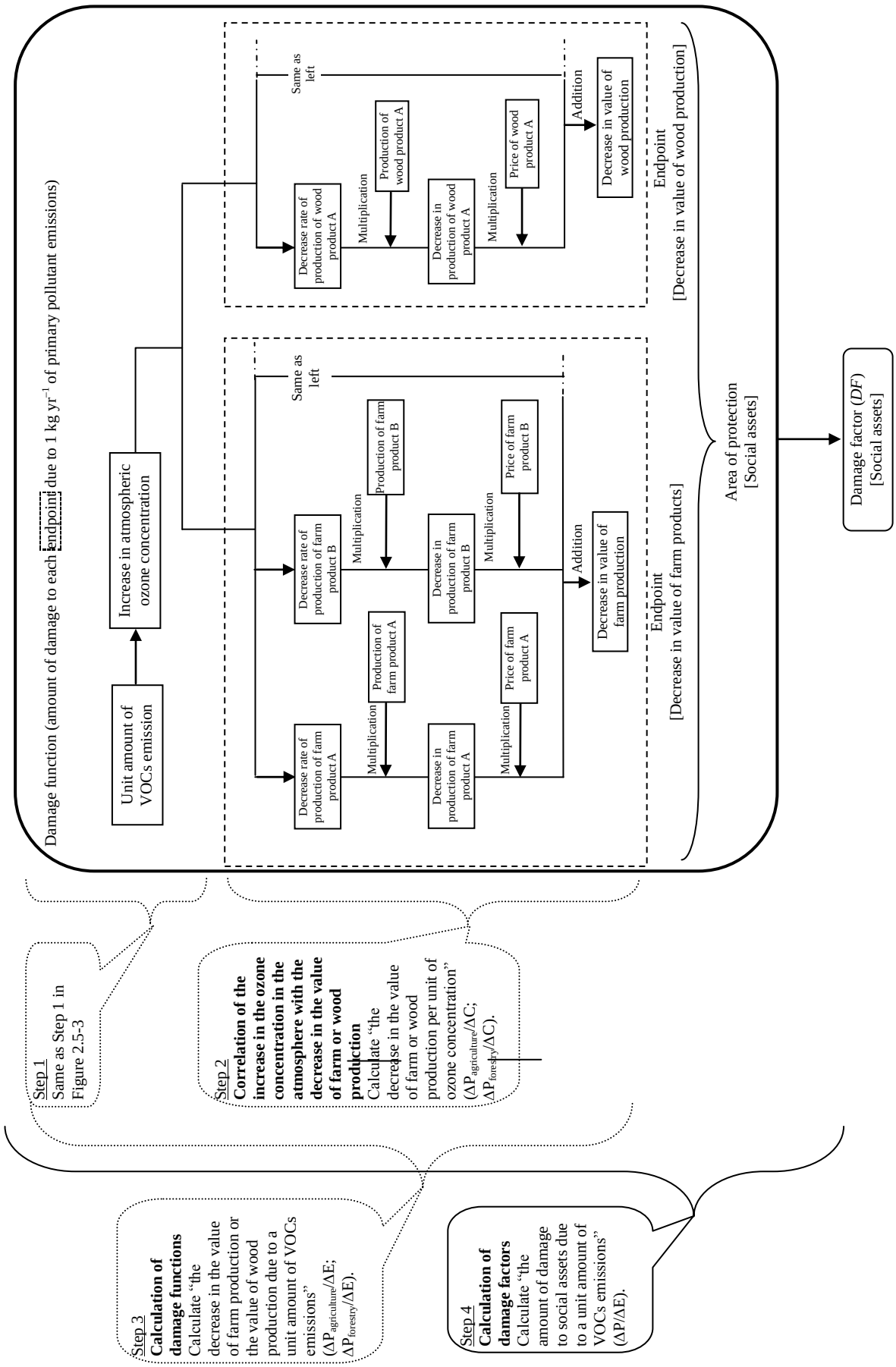


Figure 2.5-4: Flowchart of calculation of damage functions and damage factors for photochemical oxidant (area of protection: social assets)

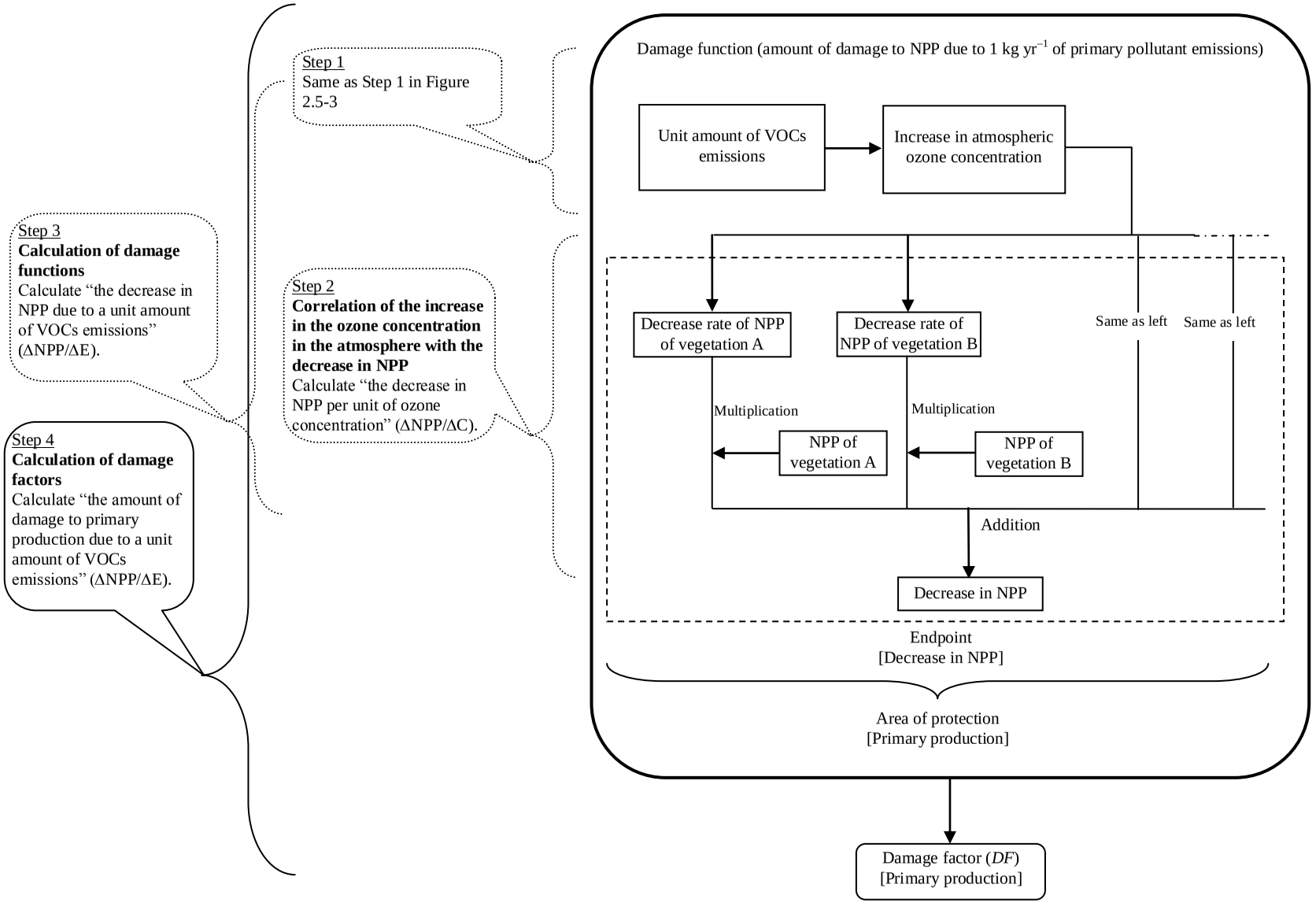


Figure 2.5-5: Flowchart of calculation of damage functions and damage factors for photochemical oxidant (area of protection: primary production)

(2) Correlation of the amount of VOCs emissions with an increase in the ozone concentration in the atmosphere

As Step 1 in Figures 2.5-3 to 2.5-5, a simulation was carried out by the use of an air quality model to calculate an "increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE).

a Object of assessment

The calculation covered the case shown in Table 2.5-4.

Table 2.5-4: Case used for calculation of "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE)

	Case	Reasons for selecting the case
Zone	7 zones (Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, Kyushu/Okinawa)	• To represent various weather conditions and air pollution situations in Japan
Time	Summer (June to August)	• Because summer is the season when insolation and temperature are high and creation of ozone is the most likely
Ozone concentration index	Daytime 7-hour average concentration Daytime 12-hour average concentration	• Because the "concentration" in the "the death/disease rates at each endpoint per unit of ozone concentration" is based on the assumption of the daytime 6-hour average concentration (described below in 2.5.3 (3)) • Because the "concentration" in the "the decrease rate of farm production per unit of ozone concentration" is based on the assumption of the daytime 7-hour average concentration (described below in 2.5.3 (4)) • Because the "concentration" in the "the decrease rate of wood production per unit of ozone concentration" is based on the assumption of the daytime 12-hour average concentration (described below in 2.5.3 (4)) • Because the "concentration" in the "the decrease rate of NPP per unit of ozone concentration" is based on the assumption of the daytime 7-hour average concentration and the daytime 12-hour average concentration (described below in 2.5.3 (5))

b Air quality model used for calculation

As described above, ozone creation is very complicated, because many kinds of pollutants, such as VOCs and NO_x, are involved and insolation and temperature have impact. To reflect such a polluting mechanism, a model that Uno et al. (1992) developed by correcting the photochemical box model (PBM) developed by the US Environmental Protection Agency (EPA) (Schere et al. 1984) was selected for LIME (see Column 2.5-3).

c Assessment procedure

Simulation was carried out by the use of the model in b and by the application of weather conditions in each zone.

Because, as described above, the relation between the amount of VOCs emissions and the amount of created ozone is nonlinear, the division of the concentration obtained from a simulation by the amount of emissions is inappropriate for the calculation of "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE). Because of this,

under LIME, a simulation (base case) was carried out with the current amount of VOCs emissions first, and then a simulation (sensitivity analysis) was carried out with a small increase in the amount of emissions of one of the eight components of CBM-IV (see Column 2.5-3). Lastly, the difference between the concentrations obtained from the two simulations was divided by the difference in the amount of emissions to calculate ΔC/ΔE (see Figure 2.5-6).

Although what can be gained from a base case simulation is the ozone concentration attributable to the current amount of VOCs emissions, the air quality model was verified by comparison with actually observed values.

Although ΔC/ΔE in summer is calculated this time (see Table 2.5-4), because the creation of ozone receives complicated impact from weather conditions and air pollution, the concentration may not be reproduced accurately if a simulation is carried out concerning only one case of average summer weather and air pollution conditions.

Under LIME, with regard to the Kanto and Kansai zones, which are urban areas where the formation of ozone is conspicuous, weather conditions and air pollution situations in summer are classified into several patterns, for each of which ΔC/ΔE was calculated in detail. That is, ΔC/ΔE was calculated for each pattern by the method specified in Figure 2.5-6 and weighted with the frequency of appearance of each pattern to obtain the average value in summer (see Figure 2.5-7).

On the other hand, with regard to the zones other than Kanto and Kansai, ΔC/ΔE was calculated concerning one case of average summer weather and air pollution conditions.

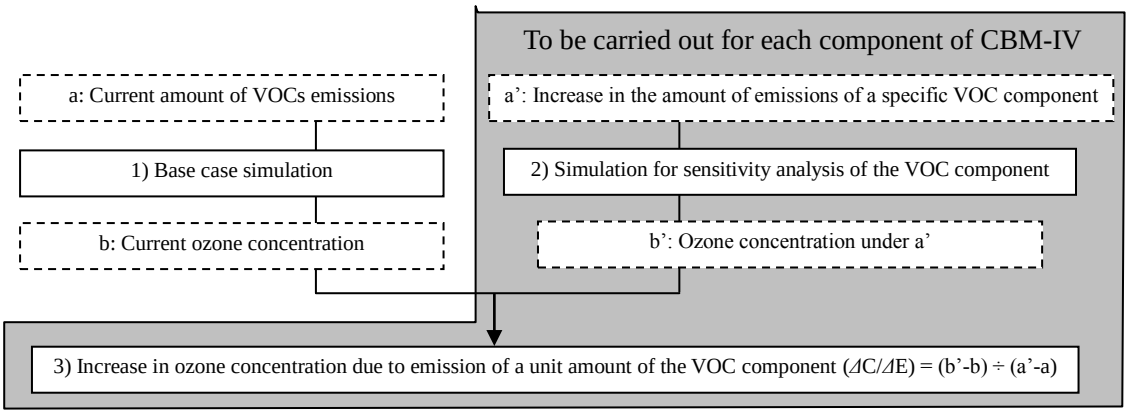


Figure 2.5-6: Procedure for calculation of "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE)

Column 2.5-3

PBM model (Schere et al. (1984) corrected by Uno et al. (1992))
PBM is a model that indicates the photochemical reaction of VOCs and NOx on an urban scale or on a mesoscale. PBM approximates altitudes up to the mixed layer in the calculation area by one box. It is assumed that, within the mixed layer, substances are well mixed vertically through convection and are distributed homogenously within the mixed layer. PBM takes into account the following phenomena: emission of substances, dilution of

substances by the wind, the growth of the mixed layer and resultant intake of substances from the upper layer, and various chemical reactions (see Figure 2.5-B).

Table 2.5-A shows the basic equation for the model.

Uno et al. (1992) developed the chemical reaction model by improving CBM-IV (Carbon-Bond Mechanism – IV) (Gery et al. 1989).

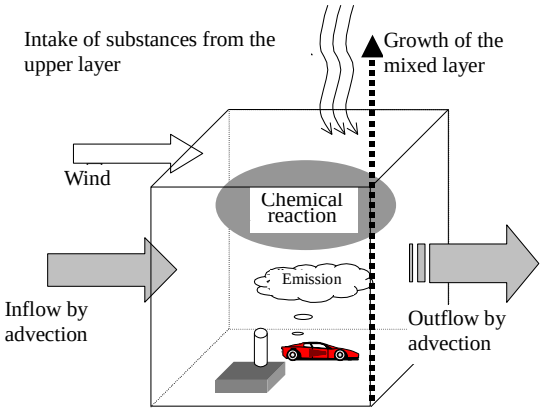


Figure 2.5-B: Conceptual diagram of PBM

(Source) Prepared based on Shere et al. (1984)

Table 2.5-A: Basic equation of PBM

Basic equation	How to introduce the basic equation
$\frac{\partial C_i}{\partial t} = U\frac{\partial C_i}{\partial x} + \frac{\partial z}{\partial t}\frac{\partial C_i}{\partial z} + \frac{Q_i}{z} + R_i(C_1, C_2, \cdots C_i, \cdots C_n)$ Temporal change in concentration of substance; Advection of substance by wind; Intake of substance from upper layer as result of growth of mixed layer; Emission of substance; Occurrence and disappearance of substance through chemical reaction C_i: Average concentration of substance i in the area [ppm] U: Average velocity in the area [m/s] z: Altitude of mixed layer [m] Q_i: Intensity of emission of substance i [ppm – m/S] R_i (C_1, C_2, …, C_i, …C_n): speed of occurrence/disappearance of substance i through chemical reaction [ppm/s]	On the assumptions described below, integrate the substance conservation equation in and below the mixed layer. • Substances are always mixed well and the concentration of each substance is homogeneous in the box. • Sources of emissions are uniformly distributed on the surface of the ground. • Intake of air from outside of the box is caused by advection by the wind horizontally and by growth of the mixed layer vertically.

Table 2.5-B: Correspondence in volume between each component of VOC and 8 components of CBM-IV (example)

Each VOC component	PAR	OLE	ETH	TOL	XYL	FORM	ALD2	ISOP
ETHYLENE			1.0					
PROPENE	1.0	1.0						
FORMALDEHYDE						1.0		
M-XYLENE					1.0			
ETHYL ALCOHOL	0.4							

Some components are extracted from USEPA (1989).

Because, as described above, the number of VOC components is more than 100, if each their chemical reactions them are modeled separately, the calculation would consume enormous quantities of data and time. To avoid this, VOC components are expressed by the following eight components (carbon bond) in CBM-IV (see Table 2.5-B).

- Single bond carbon atoms (PAR)
- Ethylene (ETH)
- Double bond carbon atoms other than ethylene (OLE)
- Reactive aromatic ring (toluene TOL and Xylen XYL)
- Carbonyl group (formaldehyde FORM and other aldehydes ALD2)
- Terpenes originated from plants (isoprene ISOP)

Chemical reaction equations are described not by each component of VOCs but by the eight components in Table 2.5-B. That is, the reactions of more than 100 VOC components are combined together into the reactions of the eight components, thereby saving calculation time.

The model takes into account 33 types of components, including the eight components and NO_2. The total number of chemical reaction equations is 82.

The PBM explained above includes all the physical and chemical processes necessary for the prediction of pollutants and, if a polluting phenomenon occurs under the mixed layer, can simulate the characteristics well (Uno et al. 1994). In addition, calculation time is reduced by making the calculation area one box as described above and by combining together the VOCs components into eight, whereby it becomes possible to carry out many simulations as shown in Figure 2.5-7 below.

The amount of insolation, maximum temperature, NO_x concentration, and the ratio between VOCs concentration and NO_x concentration are classified into the total I pattern, the total J pattern, the total L pattern, and the total M pattern,

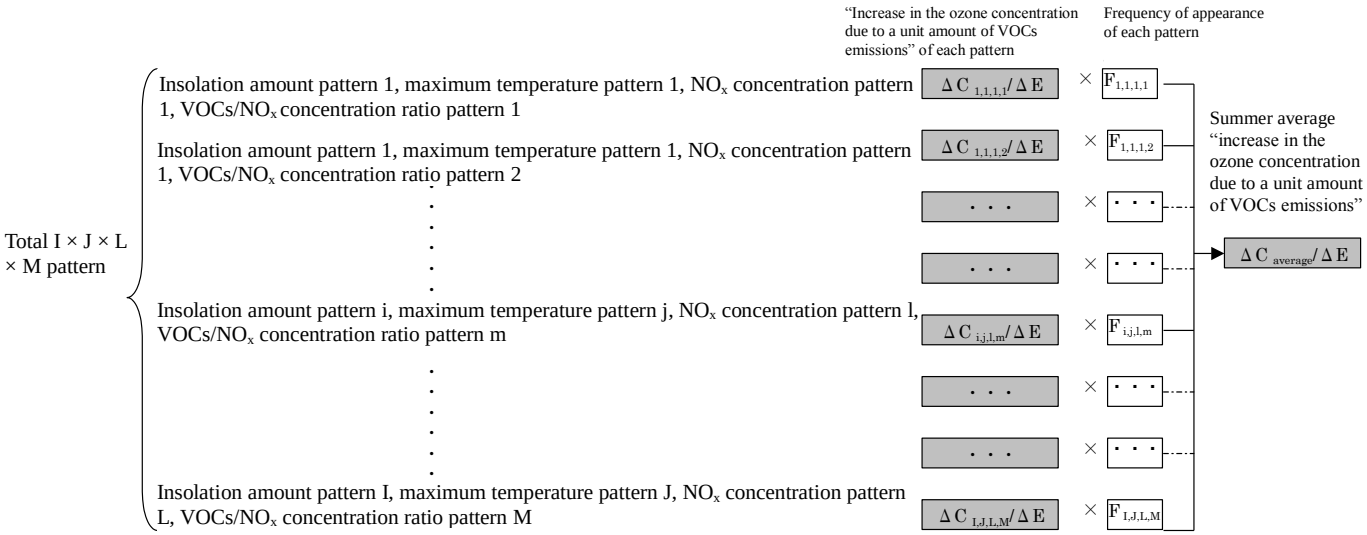


Figure 2.5-7: Procedure for calculation of "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE) concerning the summer average concentration

d Conditions for calculation

Conditions for calculation were set as described in Table 2.5-5.

Table 2.5-5: Conditions for calculation of "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE)

Conditions for calculation		Contents of setting
Condition for amount of emissions	Amount of emissions by source and time	Kanto: Environmental Agency (1998) Kansai: Kondo et al. (1999) Other than Kanto and Kansai: "FY2009 Survey on Environmental Impact and Damage – Preparation of a Pollution Map" (2000)
Initial conditions	VOCs, NO_x, O_3	The concentration at 5:00 a.m., the time of beginning of calculation, was set from each zone's observed values of concentration by time (environment data file (National Institute for Environmental Studies)). The following are the observation points used (observation points where observed values of ozone concentration by time existed were selected so that the air quality model could be verified through comparison between calculated values and observed values): Kanto (whole Kanto zone), Kansai (all of Osaka and Hyogo Prefectures), Hokkaido (National Sapporo), Tohoku (National Sendai), Chubu (Aichi Prefecture), Chugoku/Shikoku (National Kurashiki), Kyushu/Okinawa (National Kita Kyushu)
Conditions for air boundaries	VOCs, NO_x	1/10 of the initial values
	O_3	Concentration at each time was set from ozone observation data at Happo-One (Nagano Research Institute for Health and Pollution (now Nagano Environmental Conservation Research Institute), National Institute for Environmental Studies).
Conditions for lateral boundaries	VOCs, NO_x	1/10 of the initial values
	O_3	Conditions for lateral boundaries for O_3 + 0.9 × conditions for lateral boundaries for NO_2 (=PO_{bg}) = background O_3 concentration 0.004 ppm (Ohara et al. 1997)
Conditions for weather (Insolation, temperature, wind velocity)		Japan Meteorological Agency's terrestrial weather observation data The following are observatories used (selected from among those near to the observation points selected for "Initial conditions" in this table): Kanto (whole Kanto zone), Kansai (Kobe Marine Observatory, Osaka District Meteorological Observatory), Hokkaido (Sapporo District Meteorological Observatory), Tohoku (Sendai District Meteorological Observatory), Chubu (Nagoya District Meteorological Observatory), Chugoku/Shikoku (Okayama District Meteorological Observatory), Kyushu/Okinawa (Fukuoka District Meteorological Observatory)

e Calculation results

By the above-described method, the base case simulation in Figure 2.5-6 was carried out to calculate the ozone concentration that corresponds to the current amount of VOCs emissions. In addition, a sensitivity analysis simulation was carried out and was compared with the base case simulation to calculate "the increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE).

Of results in Hokkaido, Tohoku, Kanto, Chubu, Kansai, Chugoku/Shikoku, and Kyushu/Okinawa, the results in Kanto are as follows:

(a) Results of the base case simulation

Weather conditions and air pollution situations in summer were divided into 25 patterns (see Figure 2.5-7) and a simulation was carried out for each of the patterns.

The concentration obtained from the simulation was compared with the value observed on the day related to each pattern. The comparison was made concerning 1) daily changes in the concentration and 2) the daytime 7-hour and 12-hour average concentrations.

With regard to 1) daily changes in the concentration, Figure 2.5-8 shows the pattern that appears the most frequently among the 25 patterns. On the whole, the calculated values have

reproduced actual daily changes in the ozone concentration well.

With regard to 2) the daytime 7-hour and 12-hour average concentrations, comparison was made between the calculated values and the observed values concerning all 25 patterns to calculate correlation coefficients.

Table 2.5-6 shows the results in relation to correlation coefficients. Because the coefficients for the daytime 7-hour and 12-hour average concentrations exceeded 0.8, it was judged that the air quality model's capability to reproduce the current situation was high.

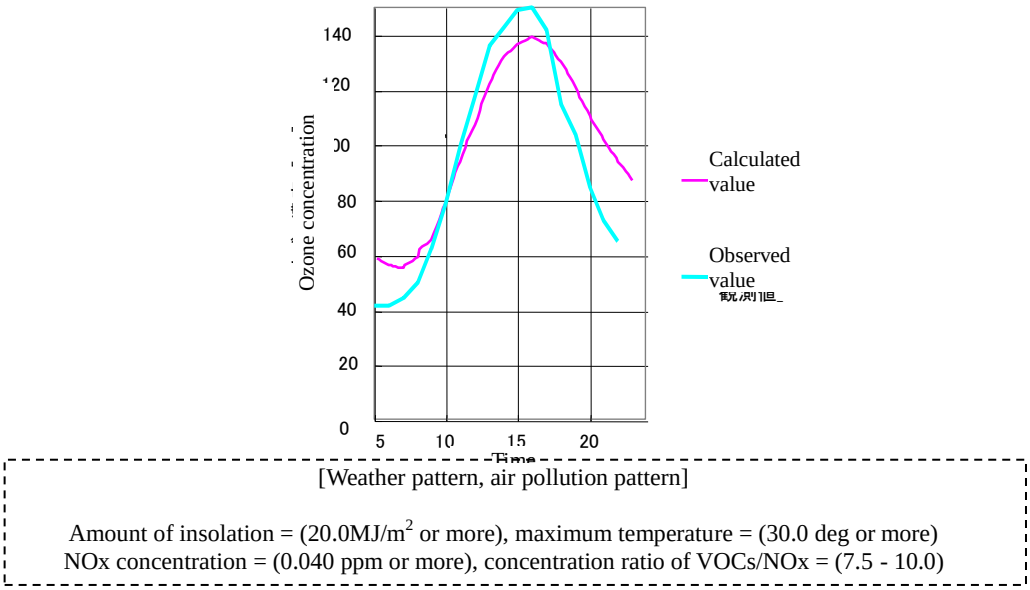


Figure 2.5-8: Daily changes in the ozone concentration obtained from base case simulation: Kanto
Observed value: observation points in Kanto in the National Institute for Environmental Studies' environmental database file (air environmental time values)

Table 2.5-6: Correlation coefficients between the base case simulation of ozone concentration and observed values: Kanto

		[Summer] (25 patterns)
Daytime 7-hour average concentration	Correlation coefficient	0.859
Daytime 12-hour average concentration	Correlation coefficient	0.838

(b) Increase in the ozone concentration due to a unit amount of VOCs emissions (ΔC/ΔE)

A sensitivity analysis simulation was carried out by increasing a unit of emissions of each of the eight components of CBM-IV. "Increase in the ozone concentration due to the emission of a unit amount" (ΔC/ΔE) of the eight components of CBM-IV was calculated. Using this ΔC/ΔE and the correlation between the eight components of CBM-IV and each VOCs component (see Table 2.5-B in Column 2.5-3), the ΔC/ΔE of each VOCs component was calculated.

Table 2.5-7 shows examples of the calculation results (Kanto).

Table 2.5-7: "Increase in the ozone concentration due to the emission of a unit amount of each VOC component" (ΔC/ΔE): Kanto, summer

[ppm kg^{-1} yr]

Daytime 7-hour average concentration	ETHYLENE	4.19E-11
	PROPENE	6.32E-11
	FORMALDEHYDE	3.23E-11
	M-XYLENE	1.77E-11
	ETHYL ALCOHOL	1.04E-12
Daytime 12-hour average concentration	ETHYLENE	2.92E-11
	PROPENE	4.61E-11
	FORMALDEHYDE	2.55E-11
	M-XYLENE	1.51E-11
	ETHYL ALCOHOL	7.92E-13

(3) Damage functions and factors for human health

With regard to damage functions and factors for human health, an explanation is given for Steps 2 to 4 in Figure 2.5-3.

a Step 2: Correlation of an increase in the ozone concentration in the atmosphere with DALY loss at each endpoint

By Step 2 in Figure 2.5-3, "DALY loss at each endpoint per unit of ozone concentration" (ΔDALYeach endpoint/ΔC) was calculated.

Figure 2.5-9 shows the flowchart of calculation, and it is as in the case of pollution by primary pollutants in 2.4.3 (2).

The following is an explanation about (a) to (c) of Figure 2.5-9.

(a) Death/disease rates at each endpoint per unit of ozone concentration

Like pollution by the primary pollutants in 2.4.3 (2), rates were set as shown in Table 2.5-8 by the use of the epidemiologic survey results used for ExternE (EC 1999).

Table 2.5-9 shows "the increase rate of the death rate and the disease rate at each endpoint per unit of ozone concentration" under ExternE, which was used for the setting in the above table.

The "concentration" in "the increase rate of the death rate and the disease rate at each endpoint per unit of ozone concentration" under ExternE is the daytime 6-hour average concentration. Because of this, when "an increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE) was calculated in 2.5.3 (2), the daytime 7-hour average concentration was used as an approximate value to the daytime 6-hour average concentration (see Table 2.5-4).

(b) Population in each zone

Population was set for the target population group at each endpoint in (a) (see Table 2.5-9).

(c) DALY per death/disease at each endpoint

As in the case of pollution by primary pollutants in 2.4.3 (2), the values from Hofstetter (1998) were used (see Table 2.5-9).

b Steps 3 and 4: Calculation of damage functions and factors

Steps 3 and 4 in Figure 2.5-3 were carried out as follows:

• Step 3: "The increase in the ozone concentration due to a unit amount of VOCs emissions" (ΔC/ΔE) in 2.5.3 (2) was multiplied by "DALY loss at each endpoint per unit of ozone concentration" (ΔDALYeach endpoint/ΔC) to obtain the damage function for each endpoint (ΔDALYeach endpoint/ΔE).

• Step 4: The damage functions calculated as described in Step 3 were added up at all the endpoints of respiratory disease to obtain the damage factor (ΔDALY/ΔE).

Table 2.5-10 shows examples (ethylene, Kanto) of the calculated damage functions and factors.

In addition, Table 2.5-11 shows the average regional damage factor in each zone and the national average.

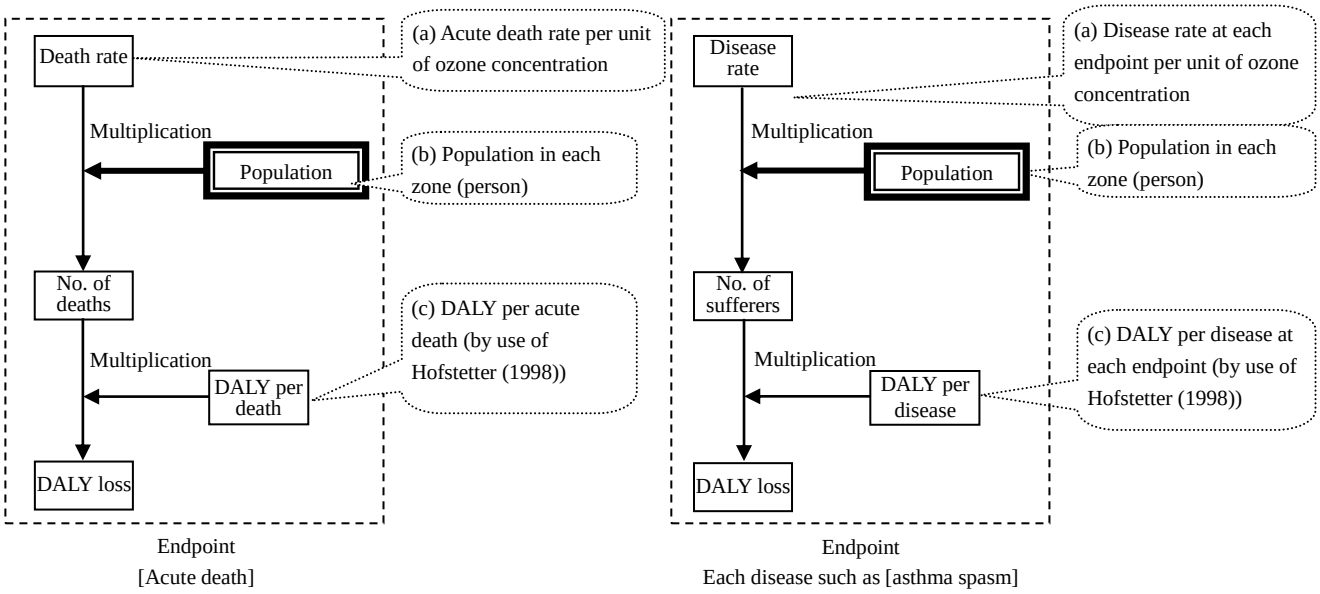


Figure 2.5-9: Flowchart of calculation of "DALY loss at each endpoint per unit of ozone concentration" ($\Delta DALY_{each\ endpoint}/\Delta C$)

Table 2.5-8: Method of setting "death/disease rates at each endpoint per unit of ozone concentration"

Item	Setting method
(a) Death rate at each endpoint per unit of ozone concentration	The following equation was used for the calculation: "Increasing rate of the death rate at each endpoint per unit of ozone concentration" under ExternE × current death rate in Japan
(a) Disease rate at each endpoint per unit of ozone concentration	"Disease rate at each endpoint per unit of ozone concentration" under ExternE was used as it was.

(a) in the table corresponds to Figure 2.5-9.

Table 2.5-9: The increase rate of the acute death rate and the disease rate per unit of ozone concentration

Type of respiratory disease (Endpoint)	"Increase rate of the acute death rate per unit of ozone concentration" [(Risk/Risk$_{baseline}$) ppm^{-1}], Risk＝case person^{-1} yr^{-1} "(a) Disease rate at each endpoint per unit of ozone concentration" [case person^{-1} yr^{-1} ppm^{-1}]			(c) DALY per death/disease at each endpoint [DALY/case] [DALY/case]	Geometric diffusion σ^2	Target population
	Ozone		Geometric diffusion σ^2			
Acute death	Sunyer et al.(1996)	1.178E+00	16.0	0.75	5.0	Entire pop.
Asthma spasm	Whittemore et al.(1989)	8.565E+00	36.0	0.00027	2.0	Asthma
Day of slight behavioral restriction	Ostro et al.(1989)	1.949E+01	16.0	0.00014	2.0	Adult
Hospitalization for respiratory system	Pounce de Leon et al.(1996)	7.067E-03	6.0	0.011	2.0	Entire pop.
Symptom days	Krupnick et al.(1990)	6.588E+01	6.0	0.00014	2.0	Entire pop.
Entry into emergency room (asthma)	Cody et al.(1992), Bates et al.(1990)	2.635E-02	36.0	0.00082	3.0	Entire pop.

(a) and (c) in the table correspond to Figure 2.5-9.
(Source) ExternE (EC1999); Hofstetter (1998) is used for shadowed parts.

Table 2.5-10: Damage function ($\Delta DALY_{each\ endpoint}/\Delta E$) and damage factor ($\Delta DALY/\Delta E$) for human health due to ozone pollution by ethylene emissions: Kanto

			Kanto
(1) $\Delta C/\Delta E$[†1] [ppm kg^{-1} yr] <Daytime 7-hour average concentration>			4.191E-11
Endpoint	[Acute death]	(a) Death rate per unit of ozone concentration [†2] (entire population) [case person^{-1} yr^{-1} ppm^{-1}]	8.739E-03
		(b) Population [person]	39,520,058
		(c) DALY of acute death [DALY case^{-1}]	7.500E-01
		(2) $\Delta DALY_{each\ endpoint}/\Delta C$ [†3] [DALY yr^{-1} ppm^{-1}] (=(a)×(b)×(c))	2.590E+05
		(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}] (=(1)×(2))	1.086E-05
	[Asthma spasm]	(a) Disease rate per unit of ozone concentration (asthma sufferers) [case person^{-1} yr^{-1} ppm^{-1}]	8.565E+00
		(b) Number of asthma sufferers [person]	51,376
		(c) DALY of asthma spasm [DALY case^{-1}]	2.700E-04
		(2) $\Delta DALY_{each\ endpoint}/\Delta C$[†3] [DALY yr^{-1} ppm^{-1}] (=(a)×(b)×(c))	1.188E+02
		(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}] (＝(1)×(2))	4.980E-09
	[Day of slight behavioral restriction]	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]	3.843E-06
	[Hospitalization for respiratory system]	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]	1.288E-07
	[Symptom days]	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]	1.528E-05
	[Entry into emergency room (asthma)]	(3) $\Delta DALY_{each\ endpoint}/\Delta E$ [DALY kg^{-1}]	3.580E-08
(4) $\Delta DALY/\Delta E$ [DALY kg^{-1}] (=Σ (3))			3.015E-05

(1) to (4) in the table correspond to Figure 2.5-3; (a) to (c) correspond to Figure 2.5-9.
[†1] Increase in the ozone concentration due to a unit amount of ethylene emissions
[†2] "(a) Acute death rate per unit of ozone concentration" is calculated by "the increase rate of the acute death rate per unit of ozone concentration × the current death rate" (see 2.5.3 (3) a (a))
[†3] DALY loss at each endpoint per unit of ozone concentration

Table 2.5-11: Damage factor for human health due to ozone pollution by ethylene emissions (ΔDALY/ΔE) [DALY kg^{-1}]
Average regional damage factor in each zone and national average

Average in Hokkaido	3.106E-06
Average in Tohoku	4.392E-06
Average in Kanto	3.015E-05
Average in Chubu	3.867E-06
Average in Kansai	2.543E-05
Average in Chugoku/Shikoku	6.196E-06
Average in Kyushu/Okinawa	4.394E-05
National average	1.673E-05

(4) Damage functions and factors for social assets

As shown in Table 2.5-3, the endpoints of social assets include farm production and wood production.

In a) below, a damage function was calculated by Steps 2 and 3 in Figure 2.5-4 concerning the endpoint of farm production. In the following b), a damage function was calculated also concerning the endpoint of wood production.

In the last c), Step 4 in the figure was carried out to add up the damage functions at both endpoints and obtain the damage factor.

a Damage function for farm production

a) Step 2: Correlation between the increase in the ozone concentration in the atmosphere and the value of decrease in farm production

Step 2 in Figure 2.5-4 was carried out to calculate "the value of decrease in farm production per unit of ozone concentration" ($\Delta P_{agriculture}/\Delta C$).

Figure 2.5-10 shows the flowchart of calculation. "(a) Decrease rate of production of each farm product per unit of ozone concentration" was multiplied by "(b) Production of each farm product" and "(c) Price of each farm product." The results were added up by type of farm product.
The following is an explanation about (a) to (c) in Figure 2.5-10.

(a) Decrease rate of production of each farm product per unit of ozone concentration

The rate was fixed as follows:

Decrease rate of production per unit increase in ozone concentration
= Decrease rate of yield of each farm product per unit increase in ozone concentration
= (Relative yield at current ozone concentration – relative yield at time of unit increase in ozone concentration) ÷ relative yield at current ozone concentration

The yield in the equation was calculated by the use of the equation obtained through experiments by Kobayashi et al. (1995) and Lesser at al. (1990) (see Table 2.5-12). These equations for calculation of yield are based on the daytime 7-hour average ozone concentration. Because of this, when "the increase in the ozone concentration due to a unit

amount of VOCs emissions" was calculated in 2.5.3.(2), the daytime 7-hour average ozone concentration was used (see Table 2.5-4).

(b) Production of each farm product

It was set by the use of the yield data in the "Statistics on Crops" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries).

(c) Price of each farm product

The price was set by the use of the "Statistical Yearbook of Ministry of Agriculture, Forestry and Fisheries" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries). The selected prices were not wholesale prices, retail prices, or other prices downstream in the distribution channel but producers' prices upstream. This is because the downstream of the distribution channel is far from the endpoint, and it was estimated that the influence of factors other than the natural environment, the target of LCA, would become greater.

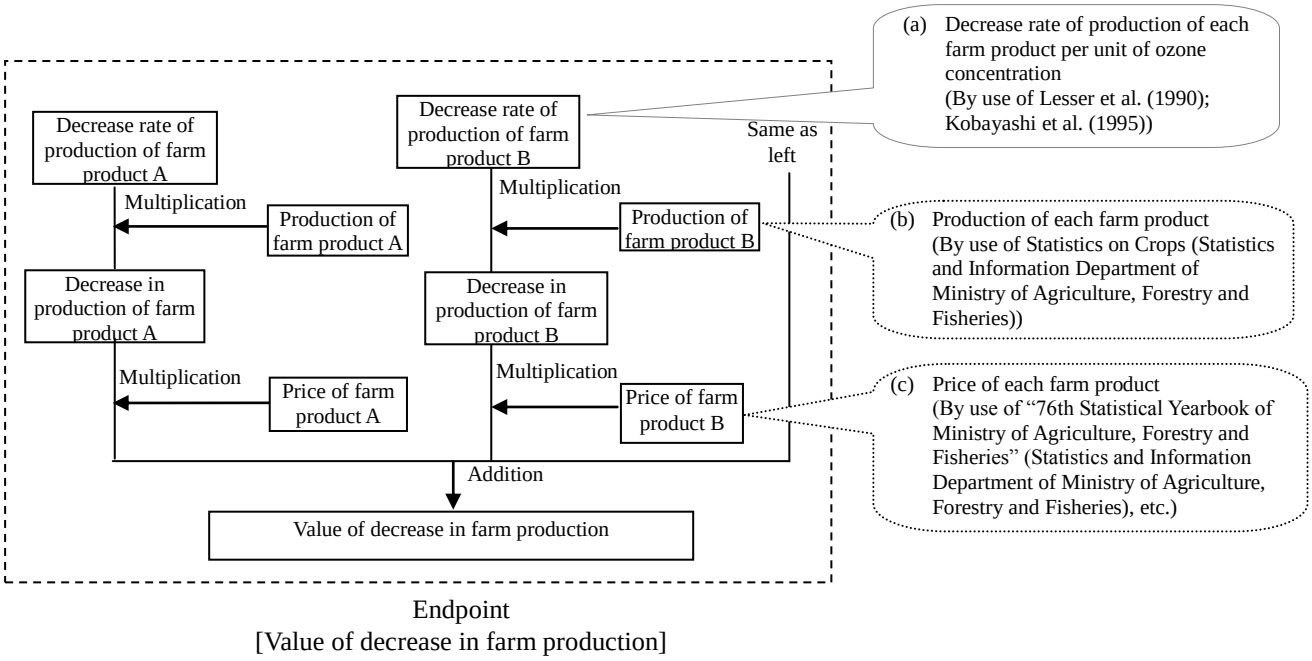


Figure 2.5-10: Flowchart of calculation of "the value of decrease in farm production per unit of ozone concentration" ($\Delta P_{agriculture}/\Delta C$)

Table 2.5-12: Yield equation used for "the decrease rate of production of each farm product per unit of ozone concentration"

Basic equation	Crop	Equation
Kobayashi et al. (1995)	Rice	$RY = \exp[C(O_3-20)]$ *RY*: Relative yield on the assumption that the standard ozone concentration is 20 ppb C=-0.001822 O_3[ppb] Daytime 7-hour average concentration
Lesser et al. (1990)	7 kinds, such as alfalfa	$RY = \exp\{-(O_3/\beta)^m\}/\exp\{-(0.02/\beta)^m\}$ *RY*: Relative yield on the assumption that the standard ozone concentration is 0.02 ppm O_3 [ppm] Daytime 7-hour average concentration *β, m*: Weibull model parameter (see Table 2.5-13)

Table 2.5-13: Weibull model parameter for each farm product in Lesser at al. (1990)

	Weibull Model (see Table 2.5-12)	
	β (ppm)	m
Alfalfa	0.178	2.07
Corn	0.124	2.83
Cotton	0.111	2.06
Forage	0.139	1.95
Kidney Bean	0.279	1.35
Soybean	0.107	1.58
Winter Wheat	0.136	2.56
Lettuce	0.120	9.76
Peanut	0.109	2.27
Sorghum	0.314	2.07
Tobacco	0.145	1.66
Tomato	0.204	1.67
Turnip	0.093	2.70

Table 2.5-14: Damage factors for farm production due to ozone pollution by ethylene emissions ($\Delta P_{agriculture}/\Delta E$): Kanto

			Kanto
(1) $\Delta C/\Delta E^{\dagger}$ [ppm kg^{-1} yr] ＜daytime 7-hour average concentration＞			4.191E-11
	[Paddy rice]	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]	1.82E+00
		(b) Production [kg yr^{-1}]	1464250000
		(c) Price [yen kg^{-1}]	243
		(2) Value of decrease in production per unit of ozone concentration [yen yr^{-1} ppm^{-1}] (= (a) × (b) × (c))	649467538422
		(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}] (= (1) × (2))	2.72E+01
	[Wheat]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	7.13E+00
	[Soybean]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	3.30E+00
	[Peanut]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	6.94E+00
	[Corn]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	0.00E+00
	[Tomato]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	4.13E+00
	[Immature corn]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	3.88E-01
	[Lettuce]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	6.42E+00
	[Leaf tobacco]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	2.92E+00
	[Early harvested corn]	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	5.79E+00
(3)$\Delta P_{agriculture}/\Delta E$ [Yen kg^{-1}] (＝Σ (3)')			6.42E+01

(1) to (3) in the table correspond to Figure 2.5-4; (a) to (c) correspond to Figure 2.5-10.
† Increase in ozone concentration due to a unit amount of ethylene emissions

b) Step 3: Calculation of damage function

Step 3 in Figure 2.5-4 was carried out to calculate the damage function.

"The increase in the ozone concentration due to a unit amount of VOCs emissions" ($\Delta C/\Delta E$) in 2.5.3.(2) was multiplied by "the value of decrease in the production of each farm produce per unit of ozone concentration" ($\Delta P_{agriculture}/\Delta C$) in a) above, to calculate the damage function ($\Delta P_{agriculture}/\Delta E$).

Table 2.5-14 shows the results. Finally, the farm products covered by the calculation of the damage function ($\Delta P_{agriculture}/\Delta E$) were the farm products about which all data on "(a) Decreasing rate of production of each farm product per unit of ozone concentration," "(b) Production of each farm product," and "(c) Price of each farm product" in Figure 2.5-10 were collected.

b Damage function for wood production

Wood is processed from the material (logs) to wood products, pulp, chips, etc. LIME covered the material at the upstream stage of the processing. This is for the same reason as in the case of the farm products in a above.

Wood is used as materials (for sawing, pulp/chips, plywood, etc.), fuel wood and charcoal, wood for cultivating mushrooms, etc. Of them, wood for sawing and pulp/chips, which occupies a large percentage of supply and demand, was used for LIME.

a) Step 2: Correlation of the increase in the atmospheric ozone concentration with the value of decrease in wood production

Step 2 in Figure 2.5-4 was carried out to calculate "the value of decrease in wood production per unit of ozone concentration."

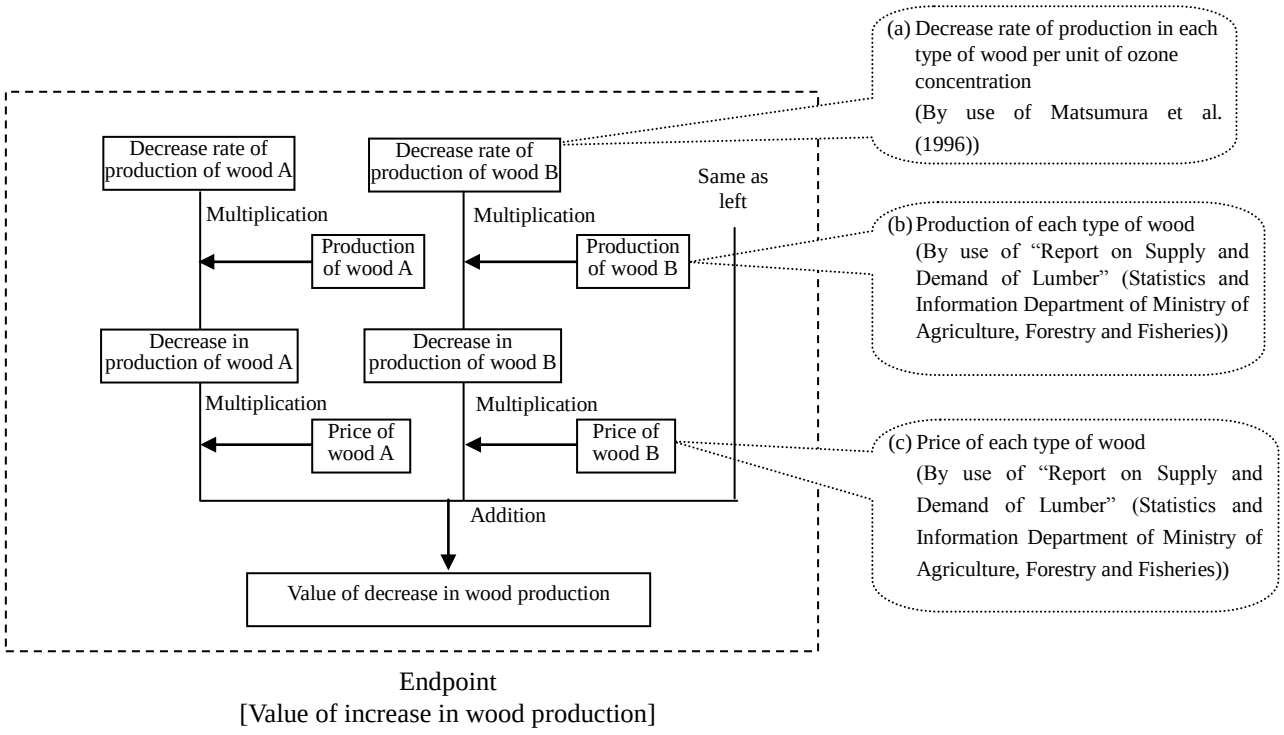


Figure 2.5-11: Flowchart of calculation of "the value of decrease in wood production per unit of ozone concentration"

Figure 2.5-11 shows the flowchart of calculation. "(a) Decrease rate of production in each type of wood per unit of ozone concentration" was multiplied by "(b) Production of each type of wood" and "(c) Price of each type of wood" and the results for all the types of wood were added up.

The following is an explanation about (a) to (c) in Figure 2.5-11.

(a) Decrease rate of production of each type of wood per unit of ozone concentration

The decrease rate was set as follows on the assumption that a decrease in the growth of trees leads to a decrease in wood production:

Decrease rate of production of each type of wood per unit increase in ozone concentration
= decrease rate of the dry growth rate of each type of tree per unit increase in ozone concentration
= (dry growth rate at current ozone concentration – dry growth rate per a unit increase in ozone concentration) ÷ dry growth rate at current ozone concentration

The dry growth rate in the equation was calculated by the calculation equation prepared based on experimental data from Matsumura et al. (1996) (see Table 2.5-15). The prepared equation for calculation of dry growth rate is based on the assumed daytime 12-hour average ozone concentration. Because of this, when "the increase in the ozone concentration due to a unit amount of VOCs emissions" was calculated in 2.5.3 (2), the daytime 12-hour average concentration was used (see Table 2.5-4).

The dry growth rate for cedar in Table 2.5-15 was used for conifer wood. The dry growth rate for zelkova in the table was used for broadleaf wood.

Table 2.5-15: Equation for calculation of the dry growth rate used for "the decrease rate of the dry growth rate of each tree per unit of ozone concentration"

Basic equation	Crop	Equation
Matsumura et al. (1996)	Cedar	Y＝-0.0926X＋46.765 (R_2=0.9738) Y : Dry growth rate [g/yr] X [ppb]: Daytime 12-hour average concentration
	Zelkova	Y＝-0.6777X＋164.09 (R_2=0.9762) X and Y are the same as those for cedar.

The above equation was obtained as follows: first, among studies that quantitatively assessed the impact of ozone on the growth of trees constituting forests (Matsumura et al. (1996); Matsumura et al. (1998); Kono et al. (1999)), the data from Matsumura et al. (1996) were selected because they enabled the calculation of the growth rate and showed a trend for the growth rate to decrease consistently as the ozone concentration becomes higher within the extent of ozone concentration observable in the environment; next, the data were used to obtain the regression equation for the prediction of the dry growth rate from the ozone concentration.

(b) Production of each type of wood

Data on production of materials were prepared based on the "Report on Supply and Demand of Lumber" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries).

(c) Price of each type of wood

The prices of sawing materials and pulp materials were set by the use of the "Statistical Yearbook of Ministry of Agriculture, Forestry and Fisheries" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries) and the "Report on Supply and Demand of Lumber" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries). The price of chip materials was set by the use of "Lumber Prices" (Statistics and Information Department of Ministry of Agriculture, Forestry and Fisheries).

Step 3: Calculation of damage function

Step 3 in Figure 2.5-4 was carried out to calculate the damage function.

"The increase in the ozone concentration due to a unit amount of VOCs emissions" (see 2.5.3 (2)) was multiplied by "the value of decrease in production of each type of wood per unit of ozone concentration" (see a) above) to calculate the damage function for each endpoint. Table 2.5-16 shows the results.

Table 2.5-16: Damage function for wood production due to ozone pollution by ethylene emissions ($\Delta P_{forestry}/\Delta E$): Kanto

				Kanto
(1) $\Delta C/\Delta E^{\dagger}$[ppm kg^{-1} yr] <daytime 12-hour average concentration>				2.916E-11
Red pine, black pine	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]			4.167E+00
	Sawing material		(b) Production [m^3 yr^{-1}]	19,399
			(c) Price [yen m^{-3}]	20,200
		(2) Value of decrease in production per unit of ozone concentration [yen yr^{-1} ppm^{-1}] (= (a) × (b) × (c))		1,632,747,931
		(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}] (= (1) × (2))		4.762E-02
	Wood chip material		(b) Production [m^3 yr^{-1}]	11,532
			(c) Price [yen m^{-3}]	4,950
		(2) Production per unit of ozone concentration [yen yr^{-1} ppm^{-1}] (= (a) × (b) × (c))		237,832,761
		(3)'Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}] (= (1) × (2))		6.936E-03
	Pulp material		(b) Production [m^3 yr^{-1}]	6,787
			(c) Price [yen m^{-3}]	6,583
		(2) Value of decrease in production per unit of ozone concentration [yen yr^{-1} ppm^{-1}] (= (a) × (b) × (c))		186,161,358
		(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}] (= (1) × (2))		5.429E-03
Cedar	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]			4.167E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		1.186E+00
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		2.188E-02
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		9.866E-04
Japanese cypress	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]			4.167E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		9.193E-01
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		3.535E-03
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]		6.814E-04

Larch	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]		4.167E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	2.190E-02
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	2.045E-03
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	4.250E-05
Conifer Other	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]		4.167E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	3.449E-02
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	2.857E-03
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	0.000E+00
Oak	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]		7.973E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	1.436E-02
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	2.000E-03
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	6.377E-04
Beech	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]		7.973E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	3.384E-03
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	5.167E-04
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	1.613E-04
Broad leaf tree Other	(a) Decrease rate of production per unit of ozone concentration [ppm^{-1}]		7.973E+00
	Sawing material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	9.794E-02
	Wood chip material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	1.778E-01
	Pulp material	(3)' Value of decrease in production due to 1 kg of ethylene emissions [yen kg^{-1}]	1.781E-03
(3) $\Delta P_{forestry}/\Delta E$ [Yen kg^{-1}] (=Σ (3)')			2.552E+00

(1) to (3) in the table correspond to Figure 2.5-4; (a) to (c) correspond to Figure 2.5-11.

† Increase in the ozone concentration due to a unit amount of ethylene emissions

Table 2.5-17: Damage factor for social assets due to ozone pollution by ethylene emissions (ΔP/ΔE) [Yen kg^{-1}]

Average regional damage factor in each zone and national average

	Damage factor for farm products ($\Delta P_{agriculture}/\Delta E$)	Damage factor for wood production ($\Delta P_{forestry}/\Delta E$)	Damage factor for social assets ($\Delta P/\Delta E$)
Average in Hokkaido	2.506E+01	2.916E+00	2.798E+01
Average in Tohoku	3.817E+01	3.638E+00	4.181E+01
Average in Kanto	6.425E+01	2.552E+00	6.680E+01
Average in Chubu	1.512E+01	1.582E+00	1.670E+01
Average in Kansai	1.034E+01	2.632E+00	1.297E+01
Average in Chugoku/Shikoku	2.219E+01	5.538E+00	2.773E+01
Average in Kyushu/Okinawa	2.268E+02	4.062E+01	2.674E+02
National average	5.741E+01	8.498E+00	6.591E+01

c Calculation of damage factors for social production

The damage factors for farm products and wood production calculated in a and b above ($\Delta P_{agriculture}/\Delta E$; $\Delta P_{forestry}/\Delta E$) were added up to obtain the damage factor for social assets ($\Delta P/\Delta E$).

Table 2.5-17 shows the average regional damage factor in each zone and the national average.

(5) Primary production: Damage functions and factors for terrestrial NPP

As shown in Table 2.5-3, the endpoint of primary production is terrestrial NPP.

The calculation of the damage function and damage factor for terrestrial NPP can be explained according to Steps 2 to 4 in Figure 2.5-5.

a Step 2: Correlation of the increase in the atmospheric ozone concentration with the amount of decrease in NPP

Step 2 in Figure 2.5-5 was carried out to calculate "the amount of decrease in NPP per a unit of ozone concentration" ($\Delta NPP/\Delta C$).

Figure 2.5-12 shows the flowchart of calculation. "(a) Decrease rate of NPP of each type of vegetation per unit of ozone concentration" was multiplied by "(b) NPP of each type of vegetation." After that, the results for all types of vegetation were added up.

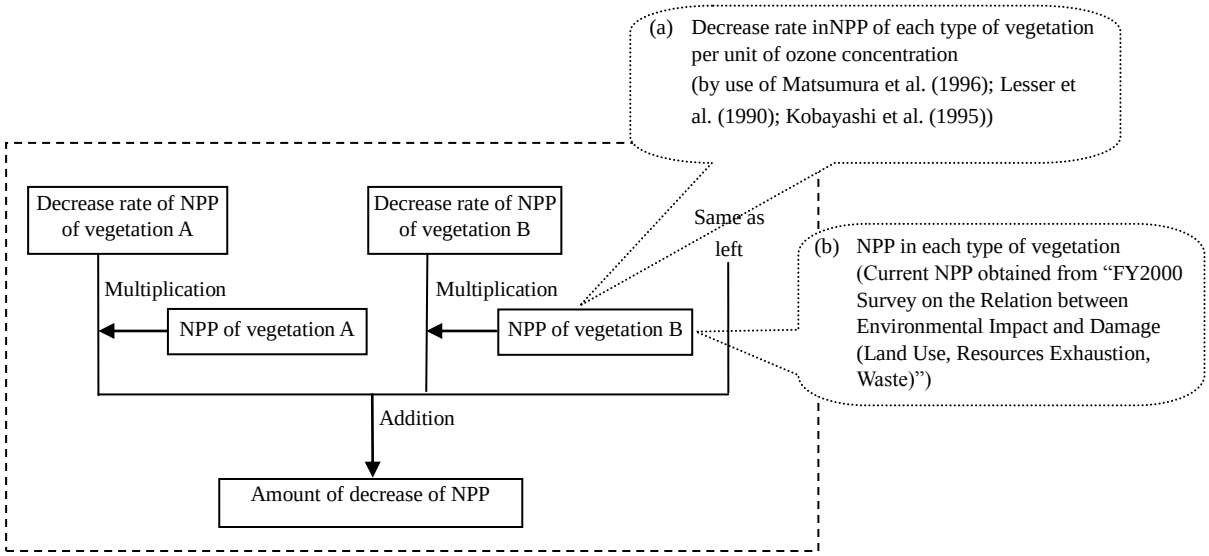


Figure 2.5-12: Flowchart of calculation of "the amount of decrease in NPP per unit of ozone concentration" ($\Delta NPP/\Delta C$)

The following is an explanation of (a) and (b) in Figure 2.5-12:

(a) Decrease rate of NPP of each type of vegetation per unit of ozone concentration

Types of vegetation were classified according to the "FY2000 Survey on the Relation between Environmental Impact and Damage (Land Use, Consumption Exhaustion, Waste)" (2001) as shown in Table 2.5-18.

Table 2.5-18: Classification of vegetation

Evergreen broadleaf forest, Beech forest, Birch forest, Oak forest, Natural conifer forest, Pine forest, Cedar and cypress forest, Deciduous conifer forest, Alpine scrub forest, Subtropical scrub forest, Montane evergreen scrub forest, Montane deciduous scrub forest, Bamboo forest, Evergreen orchard, Tea plantation, Deciduous orchard, Mulberry field, Dry field, Paddy field, Fallow field, Bamboo grass field, Rice field, Amphibious field, Aquatic field, special field, Large plant group, Small plant group, Artificial plant, Urban green space, etc.

With regard to the decreasing rate of NPP of each type of vegetation, "the decrease rate of the dry growth rate of each type of wood per unit of ozone concentration" (see 2.5.3 (4) b) was used for the vegetation of trees. In addition, "the decrease rate of production of each farm product per unit of ozone concentration" (see 2.5.3 (4) a) was used for the types of vegetation other than trees.

(b) NPP of each type of vegetation

It was decided that the current NPP obtained from the "FY2000 Survey on the Relation between Environmental Impact and Damage (Land Use, Resources Consumption, Waste)" (2001) should be used.

b Steps 3 and 4: Calculation of damage function and damage factor

Steps 3 and 4 were carried out as shown in Figure 2.5-3.

In Step 3, "the increase in the ozone concentration due to a unit amount of VOCs emissions" ($\Delta C/\Delta E$) in 2.5.3 (2) was multiplied by "the amount of decrease in NPP per unit of ozone concentration" ($\Delta NPP/\Delta C$) in a above to obtain the damage function ($\Delta NPP/\Delta E$). Because the number of endpoints is one, it is used as the damage factor in Step 4 as it is.

Table 2.5-19 shows the average prefectural damage factor in each zone and the national average.

Table 2.5-19: Damage factor for primary production due to ozone pollution by ethylene emissions ($\Delta NPP/\Delta E$) [ton kg^{-1}]
Average regional damage factor in each zone and the national average

Average in Hokkaido	6.842E-03
Average in Tohoku	4.783E-03
Average in Kanto	6.819E-03
Average in Chubu	2.743E-03
Average in Kansai	2.893E-03
Average in Chugoku/Shikoku	6.466E-03
Average in Kyushu/Okinawa	3.009E-02
National average	8.662E-03

2.5.4 Procedure for the impact assessment of photochemical oxidant

Concrete procedures for the characterization and impact assessment of photochemical oxidant are as follows:

Users can select what meets their purpose from among characterization, damage assessment, and weighting and use it for LCA.

With regard to characterization, the characterization result $CI^{PhotoOxidant}$ can be obtained from the inventory of the photochemical oxidant formation substance X *Inv* (*X*, *Region*) and the characterization factor $CF^{PhotoOxident}$ (*X*, *Region*) (see Equation 2.5-3). $CI^{PhotoOxidant}$ is regarded as the total amount of emissions of ethylene (C_2H_4), a typical VOC, into which each precursor VOC to photochemical oxidant is converted.

$$CI^{PhotoOxidant} = \sum_{X} \sum_{\text{Region}} CF^{PhotoOxidant}(X, \text{Region}) \cdot Inv(X, \text{Region}) \quad (2.5\text{-}3)$$

There are various lists of characterization factors $CF^{PhotoOxidant}$ (*X*, *Region*). Under LIME, OCEF, which is described in 2.5.2 (2), is recommended as a characterization factor that is based on the weather conditions in Japan and enables assessment with consideration for the weather conditions in each zone.

Because the formation of photochemical oxidant greatly differs according to the weather conditions in emission areas, Japan was divided into seven zones, and OCEF was presented for each zone ($CF^{PhotoOxidant}$ (*X*, *Region*)).

Therefore, if inventory data are expressed by zone, characterization can be carried out by zone. If there is no inventory classified by zone, calculation can be carried out by the use of the national average characterization factor $CF^{PhotoOxidant}$ (*X*, *Average*) (see Equation 2.5-4).

$$CI^{PhotoOxidant} = \sum_{X} CF^{PhotoOxidant}(X, Average) \cdot Inv(X) \quad (2.5\text{-}4)$$

With regard to damage assessment, the damage assessment result *DI* (*Safe*) can be obtained from *Inv* (*X*, *Region*), the inventory of the precursor substance of photochemical oxidant, and $DF^{PhotoOxidant}$ (*Safe*, *X*, *Region*), the damage factor by area of protection *Safe* (see Equation 2.5-5).

$$DI(Safe) = \sum_{X} \sum_{Region} DF^{PhotoOxidant}(Safe, X, Region) \times Inv(X, Region) \quad (2.5\text{-}5)$$

DI (Safe) means the amount of latent damage to each area of protection *Safe* due to emission of a photochemical oxidant formation substance.

Because, like the characterization factor, the damage factor *DF* has been obtained for each zone, if inventory data are classified by zone, it is possible to carry out damage assessment with consideration for differences in regional conditions. If inventory has no information on emission areas, damage assessment can be carried out by the use of the national average damage factor $DF^{PhotoOxidant}$ (*Safe*, *X*, *Average*) (see Equation 2.5-6).

$$DI(Safe) = \sum_{X} DF^{PhotoOxidant}(Safe, X, Average) \times Inv(X) \quad (2.5\text{-}6)$$

In this impact category of "photochemical oxidant," damage assessment can be carried out concerning human health, social assets, and primary production. If there is an area of protection common to two or more impact categories, comparison and integration are

possible.

In the case of integration, $IF^{photoOxidant}(X)$ is used as a factor that integrates human health, social assets, and primary production. The single index *SI* can be obtained from *Inv* (*X*) of each photochemical oxidant formation substance and the integration factor $IF^{PhotoOxidant}(X)$. The result can be compared directly with or added to assessment results in other impact categories (see Equation 2.5-7).

$$SI = \sum_{X} \left(IF^{PhotoOxidant}(X) \times Inv(X) \right) \qquad (2.5\text{-}7)$$

Appendix A1 shows the characterization factors $CF^{PhotoOxidant}(X, Region)$ and $CF^{PhotoOxidant}(X, Average)$. Appendix A2 shows the damage factors $DF^{PhotoOxidant}(Safe, X, Region)$ and $DF^{PhotoOxidant}(Safe, X, Average)$. Appendix A3 shows the integration factor $IF^{PhotoOxidant}(X)$.

Acknowledgment

For the purpose of the development of this LCIA method, ozone observation data at Happo-One were provided through the kindness of Takao Katsuno, team leader of the Nagano Environmental Conservation Research Institute, and Kentaro Murano, Research Director of the National Institute for Environmental Studies. We express our heartfelt thanks for their kindness.

[References for Section 2.5]

Andersson ― Skold Y,Grennfelt P, Pleijel K (1992): Photochemical Ozone Creation Potentials: A study of Different Concepts, J. Air Waste Manage. Assoc., 42(9): 1152 - 1158

Carter WPL, Atkinson R (1989): Computer modeling study of incremental hydrocarbon reactivity, envir. Sci.Technol, 23 : 864 - 880

Derwent RG, Jenkin ME (1991): Hydrocarbons and the long ― range transport of ozone and pan across Europe, Atomos. Environ, 25: 1661 - 1678 D

EC (1999): ExternE: Externalities of Energy, Vol.7 Methodology 1998 update, Office for Official Publications of the European Communities

Gery MW, Whitten GZ, Killus JP, Dodge MC (1989): A photochemical kinetics mechanism for urban and regional scale computer modeling J. Geophy. Res., 94 (D10): 12925 - 12956

Goedkoop M, Spriensma R (2000): The Eco ― indicator 99, A Damage Oriented Method for Life Cycle Impact Assessment, Methodology Report 2nd edition

Heijungs R, Guinée JB, Huppes G, Lankreijer RM, Udo de Haes HA, Wegener Sleeswijk A, Ansems AMM, Eggels PG, Diun R van, Goede HP dé (1992): Environmental Life Cycle Assessment of Products; Backgrounds & Guide, Leiden

Hofstetter P (1998): Perspectives in Life Cycle Impact Assessment; A Structured Approach to Combine Models of the Technosphere, Ecosphere and Valuesphere, Kluwers Academic Publishers

Jenkin ME, Hayman GD (1999): Photochemical ozone creation potentials for oxygenated volatile organic compounds: sensitivity to variations in kinetic and mechanistic parameters, Atomos. Environ., 33: 1275 - 1293D

Kobayashi K, Okada M, Nouchi I (1995): Effects of ozone on dry matter partitioning and yield of Japanese cultivars of rice (Oryza sativa L.): Agriculture, Ecosystems & Environment, 53 : 109 - 122

Lesser VM, Rawlings JO, Spruill SE, Somerville MC (1990): Ozone Effects on Agricultural Crops : Statistical Methodologies and Estimated Dose ― Response Relationships, Crop Science, 30: 148 - 155

Schere KL, Demerjian KL (1984): User's guide for the photochemical box model (PBM), EPA ― 600/8 ― 84 ―022a

Steen B (1999): A Systematic Approach to Environmental Priority Strategies in Product Development (EPS) Version 2000-Models and Data of the Default Method, CPM Report 1999: 5, Chalmers University of Technology, Göteborg, p.312

UNECE (1990): Draft technical annex on classification of volatile organic compounds based on their photochemical ozone creation potential, United Nations Economic Commission for Europe, Geneva

USEPA (1971): Requirements for preparation, adoption, and submittal of implementation plans, Fed. Regist, 36: 15486 - 15506, August 14

USEPA (1989): User's Manual for OZIPM – 4 (ozone isopleth plotting with optional mechanisms), Vol. I

Ito H, Iwamura H, Saito T, Watanabe N (2000): Small Encyclopedia of Chemical Substances, Iwanami Junior Shinsho

Imamura T (2001): Basic Knowledge on Ozone and the Ozone Layer, News of the National Institute for Environmental Studies, 20 (3)

Utsunomiya A, Doi T, Mizoguchi T (1994): Changes in the Ozone Concentration in Unpolluted Mountainous Areas, Journal of Japan Society of Air Pollution, 29 (6): 332 - 339

Uno I, Wakamatsu S (1992): Analysis of Winter High-Concentration NO_2 Pollution by the Photochemical Box Model, Journal of Japan Society of Air Pollution, 27 (5): 246 - 257

Uno I, Mitsumoto S (1994): Numerical Simulation of Air Pollution, Journal of Japan Society of Air Pollution, 29 (3): A43 - A54

Ohara T, Uno I (1997): Numerical Simulation of Mesoscale Air Pollution, Journal of Japan Society for Atmospheric Environment, 32 (3): A39 - A57

Operations Entrusted to the Institute of Behavioral Sciences by the Environment Agency (1998): FY1997 Survey for Development of an Acid Rain Forecast Method

Kono Y, Matsumura H (1999): Multiple Impact of Ozone and Artificial Acid Rain on the Growth of Cedar, Japanese Cypress, and Sawara Cypress, Journal of Japan Society for Atmospheric Environment, 34 (2), 74 - 85

National Institute for Environmental Studies (2001): Clarification of the Dynamic State of VOC in Urban Areas and Research on Assessment of Impact on Air Quality (FY1998 to FY2000), Report of Special Research from National Institute for Environmental Studies (SR-42-2001)

Kondo A, Yamaguchi K, Nishikawa E, Hara T, Okazaki R (1999): Examination of Impact of Emissions from Ships on the Air Pollution Concentration in the Osaka Bay Area, Journal of Kansai Shipbuilding Association, (231): 101 - 109

Sakurai T (2000): Tunnel Survey on VOCs Emitted from Automobiles, News of the National Institute for Environmental Studies, 19 (1)

Shinozaki M, Yoshida T, Katatani N, Mizuno T (1984a): Examination of Impact of Intake of Emitted NO_x on Atmospheric O_3 Formation Reaction, Environmental Pollution, 19 (3): 113 - 120

Shinozaki M, Yoshida T, Katatani N, Mizuno T (1984b): Examination of Impact of Intake of Emitted NMHC on

Atmospheric O_3 Formation Reaction, Environmental Pollution, 19 (6): 327 - 334

FY1999 Survey on the Relation between Environmental Impact and Damage – Preparation of Pollution Map (2000)

FY2000 Survey on the Relation between Environmental Impact and Damage (Land Use, Resources Exhaustion, Waste) (2001)

Matsumura H, Aoki H, Kono Y, Izuta T, Totsuka T (1996): Impact of Ozone on the Dry Growth and Gas Exchange Rates of Cedar, Japanese Cypress, and Zelkova Seedlings, Journal of Japan Society for Atmospheric Environment, 31 (6): 247 - 261

Matsumura H, Kobayashi T, Kono Y (1998): Single and Multiple Impact of Ozone and Artificial Acid Rain on the Dry Growth and Gas Exchange Rates of Cedar, Abies Homolepis, and Zelkova Seedlings, Journal of Japan Society for Atmospheric Environment, 33 (1): 16 - 35

Wakamatsu S, Shinozaki M (2001): Extensive Air Pollution – Mechanism and Impact on Plants, Popular Science 199, Shokabo

2.6 Toxic Chemicals (Human Toxicity)

Changes under LIME 2

- Under LIME2, damage functions were renewed because the International Agency for Research on Cancer (IARC) reviewed the carcinogenesis risk of some substances since LIME 1.

- Although the substances covered by the Pollutant Release and Transfer Register (PRTR) under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management were assessed under LIME1, the database on the Integrated Risk Information System (IRIS) of the Environmental Protection Agency (EPA) specifies the unit carcinogenesis risk of other substances. As a result of an increase in the number of covered substances by the use of the database, the number of substances in the damage factor list increased from 135 under LIME 1 to 168 under LIME 2.

- Under LIME 1, the D-R factor for chronic diseases was estimated from the no observable adverse effect level (NOAEL) and the lowest observed adverse effect level (LOAEL). However, with regard to heavy metals, because quantitative information on the D-R relationship was obtained from ample examples of epidemiological surveys and risk assessment documents, the damage functions for chronic disease from heavy metals were renewed based on the information.

2.6.1 What phenomenon is the human toxicity of toxic chemicals?

(1) What is the human toxicity of toxic chemicals?

At present, more than 100,000 types of chemicals are produced and used for various purposes all over the world, greatly contributing to the realization of healthy, safe, and rich lives. On the other hand, some chemicals have intrinsic "hazards." They have a "toxic risk" to human beings and the ecosystem if human beings and other organisms are exposed to them through air, water, food, etc. Under LIME, chemicals that may have toxic impact on human health are called "toxic chemicals with human toxicity." Human toxicity appears when human beings take toxic chemicals into their bodies under the circumstances where they are exposed to them. However, how human toxicity appears differs according to type of chemical (such as cancer-causing substances and asthma-causing substances). Moreover, because the intensity of human toxicity differs according to the degree of toxicity of chemicals and the degree of exposure, exposure may lead to slight sickness or fatal cases.

Under LIME, "impact on human health in the case of emission of toxic chemicals with human toxicity into general environments, such as the atmosphere, water areas, and soil" was assessed. The following are important points concerning the development of LIME.

- Because, as described in the beginning, there are various types of chemicals, it is extremely difficult to grasp the toxicity of and exposure to all substances quantitatively. Therefore, LIME only covers the substances whose impact can be estimated quantitatively from the existing information.

• With regard to the exposure route of toxic substances, it was assumed that exposure occurred through general environments, such as the atmosphere, water areas, and soil. Although there are cases where human beings are exposed to toxic substances directly through working environments and the use of products, such cases were excluded.

• The "hazard" of toxic substances is classified into the hazard that may have impact if the threshold level is exceeded (such as acute toxicity and developmental toxicity) and the hazard that has no threshold level and may have impact even if the substance is taken in slight amounts (such as carcinogenesis). If there is a threshold level, whether or not impact exists differs below and above the level. Because the purpose was to obtain a factor that indicates "risk increment per unit amount of emissions," an index for the hazard without a threshold level was estimated.

• Although chemicals move through environmental media, such as the atmosphere, the hydrosphere, and the biosphere, movability differs among substances (such as highly vaporizable substances and highly soluble substances). In addition, chemicals are changed through decomposition and oxidation-reduction reaction. Because of this, a box model that takes into consideration tis complicated material balance and changes (Figure 2.6-1) was used to estimate the fate of chemicals in the environment ("fate" means chemicals' emission into environmental media, movement through them, transformation/decomposition, and final distribution in each medium).

• The routes through which chemicals are taken into human bodies are roughly classified into transbronchial (intake by the respiratory system through respiration), oral (intake by the digestive system through food and drinking water), and endemic (penetration through the skin). The transtracheal route and the oral route were assessed this time.

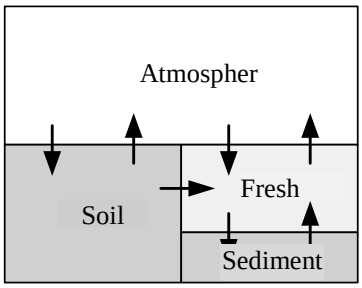


Figure 2.6-1: Concept of the box model

The box model expresses the environmental media (air, water, soil, etc.) in the spatial sphere (region, continent, etc.) as compartments, and it is assumed that various conditions are homogeneous within each compartment. Based on the material balance between compartments (transportation between media) and the material balance within a compartment (emission and decomposition), the substance concentration in each compartment is estimated from the equilibrium condition or the initial value to a certain point of time. There are also a model that divides an environmental medium into two or more compartments and a model that takes into consideration material balance with the outside of the system.

(2) Endpoints of human toxicity of toxic chemicals

The toxic chemicals dealt with herein are those covered by the Pollutant Release and Transfer Register (PRTR) under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (type-1 designated chemicals), and the area of protection was limited to human health (impact on the

ecosystem is not assessed herein, but will be assessed in Section 2.7).

First, endpoints for human toxicity should be defined from among human diseases.

Under PRTR, carcinogenicity, mutagenicity, oral chronic toxicity, inhalant chronic toxicity, reproductive/developmental toxicity, and sensitizing property were assessed as toxicity items against human health. Substances that have any of these types of toxicity have been selected for assessment (I. (1) of Table 2.6-1). It is said that about 95% of the 354 chemical substances selected as Type-1 designated chemicals may have toxicity against human health (under the cabinet order revised in 2008, the number of substances was increased to 462 in April 2010).

Table 2.6-1: Assessment items and criteria for selection of type-1 designated chemicals under PRTR

	Assessment content	Item	Criterion by item	Criterion
I. Toxicity	(1) Risk of having damage to human health	Carcinogenicity	Class 1 or 2	Chemicals classified into any of them
		Mutagenicity	Existence of reliable data	
		Oral chronic toxicity	Class 1 or 2 or 3	
		Inhalant chronic toxicity	Class 1 or 2 or 3	
		Reproductive/developmental toxicity (including teratogenesis)	Class 1 or 2 or 3	
		Sensitizing property	Existence of reliable data	
		Acceptable concentration in working environment	Class 1 or 2 or 3	
	(2) Inhabitation or growth of animals and plants	Eco-toxicity against aquatic organisms (alga, water flea, fishes)	Class 1 or 2 or 3	
	(3) Damage to human health through ozone layer destruction	Ozone depleting substances	Substances specified in Montreal Protocol	
II. Amount of exposure	"Continuous existence in a considerably wide local environment"	Amount of manufacture/import	10 or more tons of annual manufacture/ import (Cumulative amount of manufacture/import in the case of ozone depleting substances)	Substances designated in I. and falling under either
		Detection	Detection from two or more zones during the past decade	

• Prepared based on "Designation of Type-1 Designated Chemicals and Type-2 Designated Chemicals under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (Report)" (February 2000).

• With regard to the selection of type-2 designated chemicals, although the items and criteria for toxicity assessment are the same as in the case of type-1 designated chemicals, there are differences in the criteria for the amount of exposure.

• "Class 1" and "Class 2": "Classes" have been defined according to the intensity of toxicity for each assessment item in "Concrete Criteria for Selection of Chemicals Covered by PRTR and MSDS," an annex to "Designation of Type-1 Designated Chemicals and Type-2 Designated Chemicals under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (Report)."

For example, with regard to carcinogenicity, the criteria for classification into each class are as follows:

• Class 1: Substances classified into a category equivalent to the assessment "carcinogenic to human beings" by any of the institutes (IARC, EPA, EU, NTP, ACGIH, Japan Society for Occupational Health)

• Class 2: Substances classified into 2A or 2B by IARC as substances "highly suspected of being carcinogenic to human beings" or substances classified into a category equivalent to "highly suspected of being carcinogenic to human beings" by other two or more institutes

Risk assessment requires quantitative values concerning dose-response (D-R). When substances were selected under PRTR, quantitative values were used as a criterion for the existence of toxicity concerning only carcinogenicity and oral/inhalant chronic toxicity among the toxicity items to be assessed. With regard to the other assessment items, information sufficient for quantitative assessment has not been obtained. For example, quantitative information on "positive" or "negative" is used for judging sensitizing property. As for inhalant chronic toxicity, because the quality of data on substances is not integrated, it was excluded from assessment.

Therefore, carcinogenicity and oral chronic toxicity were selected as the endpoints of human toxicity to be assessed under LIME (however, with regard to heavy metals newly assessed under LIME 2, because a D-R factor was obtained based on epidemiological research cases, inhalant chronic toxicity was assessed. See 2.6.3).

Figure 2.6-2 shows the causality of human toxicity. Toxic chemicals emitted into the environment were transformed or decomposed, moving through environmental media, such as the air, water, and soil. Toxic chemicals in the air are taken into human bodies through air inhalation, and toxic substances in water areas and soil are taken into human bodies through oral intake of drinking water and food. These induce illness, such as cancer and chronic diseases.

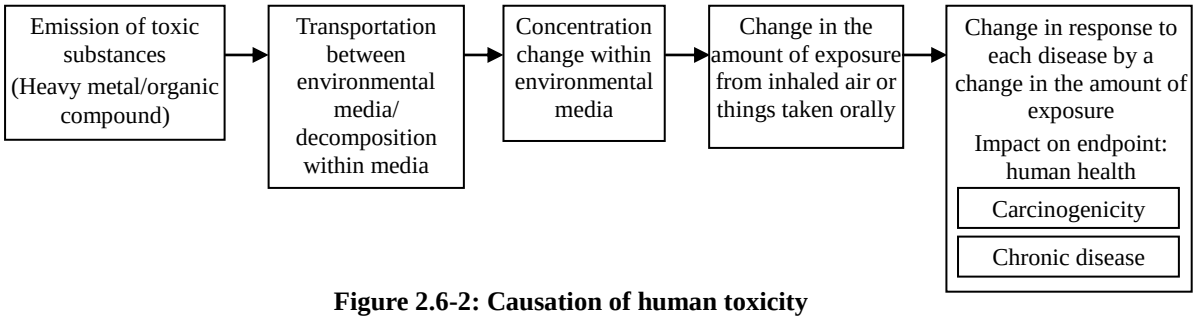


Figure 2.6-2: Causation of human toxicity

2.6.2 Characterization factor for toxic chemicals

(1) Existing characterization factor for toxic chemicals

The characterization factor for toxic substances is called "human toxicity potential" (HTP). Generally, HTP is calculated with consideration for three processes: fate, exposure/intake, and effect. Many studies on LCA point out that the fate of emitted toxic substances in the environment – especially, "transportation between media" and "decomposition within media" – is important for calculation of a characterization factor (CML 2001; Heijungs et al. 1992; Hauschild et al. 1998; Guinée et al. 1996; Jolliet et al. 1997; Lindfors et al. 1995; Udo de Haes et al. 1996). An early characterization factor developed in 1992 (Heijungs et al. 1992) did not take into consideration the fate of emitted chemicals in the environment. At present, however, three types of HTP, which differ from each other in how the fate in the environment is treated, have been developed:

1) HTP that semi-quantitatively expresses transportation between media and

decomposition within media (Hauschild et al. 1998)

2) HTP that expresses transportation between media and decomposition within media by a numerical model (Guinée et al. 1996, Huijbregts 1999, Hertwich 1999)

3) HTP that expresses transportation between media and decomposition within media by a numerical model and a rule of thumb (Jolliet et al. 1997)

Moreover, in many cases, HTP has been defined as shown in the equation below. However, the names of items differ among cases.

$$HTP_{i,ecomp} = \frac{\sum_{fcomp}\sum_{r} F_{i,ecomp,fcomp} \cdot T_{i,fcomp,r} \cdot I_r \cdot E_{i,r}}{\sum_{fcomp}\sum_{r} F_{ref,ecomp,fcomp} \cdot T_{ref,fcomp,r} \cdot I_r \cdot E_{ref,r}} \qquad (2.6\text{-}1)$$

In this equation:

$HTP_{i,ecomp}$:	HTP when the toxic chemical *i* is emitted into the compartment *ecomp*
$F_{i,ecomp,fcomp}$:	Fate factor – ratio of *i* emitted into *ecomp* and having reached the final compartment *fcomp* just before exposure
$T_{i,fcomp,r}$:	Transportation factor – ratio of *i* distributed to *r*, two or more exposure routes between *fcomp* and human beings (air inhalation, oral intake of drinking water and food, etc.)
I_r:	Intake factor – ratio of *i* taken in through *r* by human beings (such as the ratio of the amount of human beings' inhalation to the amount of air)
$E_{i,r}$:	Effect factor – ratio of the effect of exposure of *i* on human toxicity through *r*. $E_{i,r}$ is often given as the reciprocal of the acceptable daily intake (ADI).
ref:	Reference substance – the substance that is selected as the reference substance differs among cases.

The numerator of Equation 2.6-1 quantitatively expresses the impact of the emission of a unit amount of a toxic chemical on human toxicity and is called "toxicity potential." The characterization factor for human toxicity can be obtained by dividing the toxicity potential of each toxic chemical by the toxicity potential of the reference substance (the denominator of Equation 2.6-1).

The following is an overview of two cases where the characterization factor for human toxicity was obtained by expressing transportation between media and decomposition within media by a numerical model (Guinée et al. 1996, Huijbregts 1999).

Guinée et al. (1996) regarded four elements – the air, water, agricultural soil, and industrial soil – as the environmental media and regarded six routes – the air, seafood, drinking water, grain, beef, and dairy products – as the routes of human exposure. The largest difference with the method so far (Hauschild et al. 1998) is that consideration was given to decomposition within environmental media and immobilization (sedimentation on the sea bottom had been neglected because it takes a lot of time to move to another medium), and

they were expressed as numerals concerning each substance so that fate analysis could be modeled more realistically and comprehensively. The potential daily intake (PDI) of a toxic chemical is expressed as a total estimate of daily intake for each exposure route and is divided by ADI to obtain toxicity potential. The reference substance for obtaining the characterization factor is 1.4-dichlorobenzene (air emission). HTP was calculated by the following equation:

$$HTP_{i,\,ecomp} = \frac{PDI_{\,i,\,ecomp} \cdot E_{\,i}}{PDI_{\,1,4-dichlorobenzene,\,air} \cdot E_{\,1,4-dichlorobenzene}} \qquad (2.6\text{-}2)$$

In this equation:

$HTP_{i,\,ecomp}$:	HTP when the toxic chemical *i* is constantly emitted into the compartment *ecomp* at a rate of 1,000 kg/day
$PDI_{i,\,ecomp}$:	PDI when *i* is emitted to *ecomp*
E_i:	Effect factor – the reciprocal of ADI of *i*, which is fixed irrespective of exposure route

Guinée et al. (1996) calculated HTP on the assumption that a toxic chemical is constantly emitted. However, this is unsuitable for impact assessment when an additional amount of a substance over the baseline emission is emitted. Moreover, although transportation between media was expressed by a numerical model, they did not take into full consideration exposure routes and intake.

Huijbregts (1999) calculated a characterization factor for human toxicity, improving the method developed by Guinée et al (1996). The following are main improvements:

1) It became possible to carry out fate analysis of toxic chemicals emitted inconstantly.

2) An assessment period was adopted for fate analysis so that HTP of substances likely to remain for a long time (such as heavy metal chemicals) would be weighted comparatively highly.

3) Fate analysis was modeled globally and a nested model was adopted so that space sizes – regional, continental, and global – could be taken into consideration.

4) The temperature dependencies of steam pressure, solubility, Henry's constant, and decomposition speed were taken into consideration.

5) Water areas were classified into freshwaters and salt waters in the case of regional and global sizes.

HTP is calculated by the following equation:

$$HTP_{i,\,ecomp} = \frac{\sum_{r}\sum_{s} PDI_{\,i,\,ecomp,\,r,\,s} \cdot E_{\,i,\,r} \cdot N_{\,s}}{\sum_{r}\sum_{s} PDI_{\,1,4-dichlorobenzene,\,air,\,r,\,s} \cdot E_{\,1,4-dichlorobenzene,\,r} \cdot N_{\,s}} \qquad (2.6\text{-}3)$$

In this equation:

$HTP_{i,}\ ecomp_{:}$	HTP when the toxic chemical *i* is constantly emitted into the compartment *ecomp*
$PDI_{i,}\ ecomp_{,r,s:}$	PDI at a space size of *s* through the exposure route *r* when *i* is emitted to *ecomp* – estimation of PDI for each of four types of assessment periods (20 years, 100 years, 500 years, and an indefinite period after emission)
$E_{i,r:}$	*Effect* factor – the reciprocal of ADI of *i* through the exposure route *r*
$N_{s:}$	*Weighting* factor of the space size *s* set from population density

Although the method developed by Huijbregts (1999) could more realistically model the fate analysis of toxic chemicals, it has a problem of having considerable uncertainty. This is mainly because of many model parameters. With regard to heavy metal chemicals especially, HTP greatly differs according to the assessment period. However, pointing out many problems, Leiden University's Institute of Environmental Sciences (CML) (2001) has recommended Huijbregts (1999)'s global HTP with an indefinite assessment period as the first choice for the characterization factor.

(2) Characterization factor of toxic chemicals under LIME

Under LIME also, HTP was developed by the use of a numerical model to fate analysis. Figure 2.6-3 shows a flowchart of the calculation of the characterization factor. Procedures for calculating the characterization factor can be roughly divided into the estimation of PDI by fate and exposure analysis and the estimation of the impact factor.

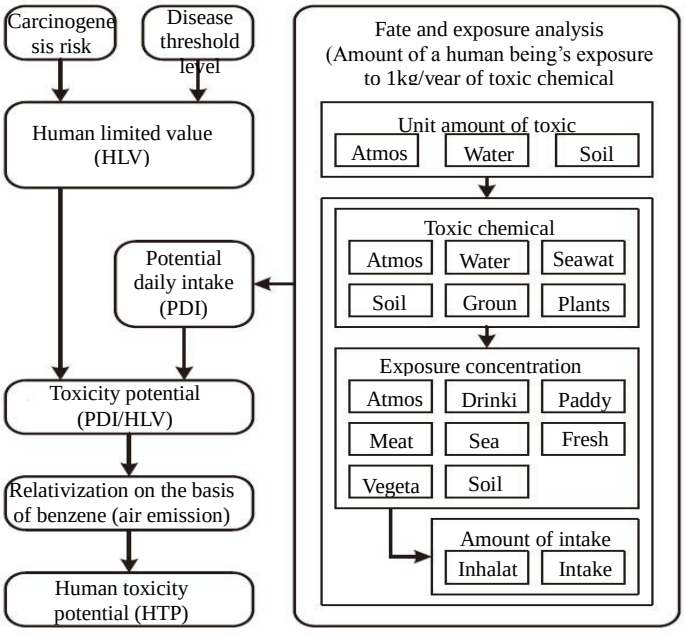


Figure 2.6-3: Flowchart of calculation of human toxicity potential

a Fate and exposure analysis

Under LIME, the fate analysis of toxic chemicals in the air was carried out by the use of the multimedia fate model developed by École Polytechnique Fédérale de Lausanne (EPFL). When the fate analysis was carried out, consideration was given to conditions in Japan in

relation to geographical features, population, the amount of intake by type of food, etc. The following are main characteristics of the model:

- The geographical extent of the model is Japan and surrounding sea areas.
- The compartments are the atmospheric boundary layer (lower tropospheric layer), surface water (rivers, freshwater), surface water (lakes, freshwater), freshwater substratum, marine surface layer, deep seawater, marine substratum, topsoil, rhizosphere soil, unsaturated layer, vegetation, and urban areas.
- The model takes into consideration transportation between media and decomposition within media.
- The compartments where toxic chemicals are artificially emitted are the terrestrial atmospheric boundary layer, surface water, and topsoil (Figure 2.6-4).
- As human exposure routes, oral intake of air inhalation, drinking water, seafood, farm products, beef, and dairy products was taken into consideration (Figure 2.6-5).

By the use of this model, the amount of intake through inhalation and the mouth in the cause of the emission of a unit amount of a substance into a compartment was calculated as PDI [mg/kg/day] (the unit means the amount of intake per kilogram of weight per day).

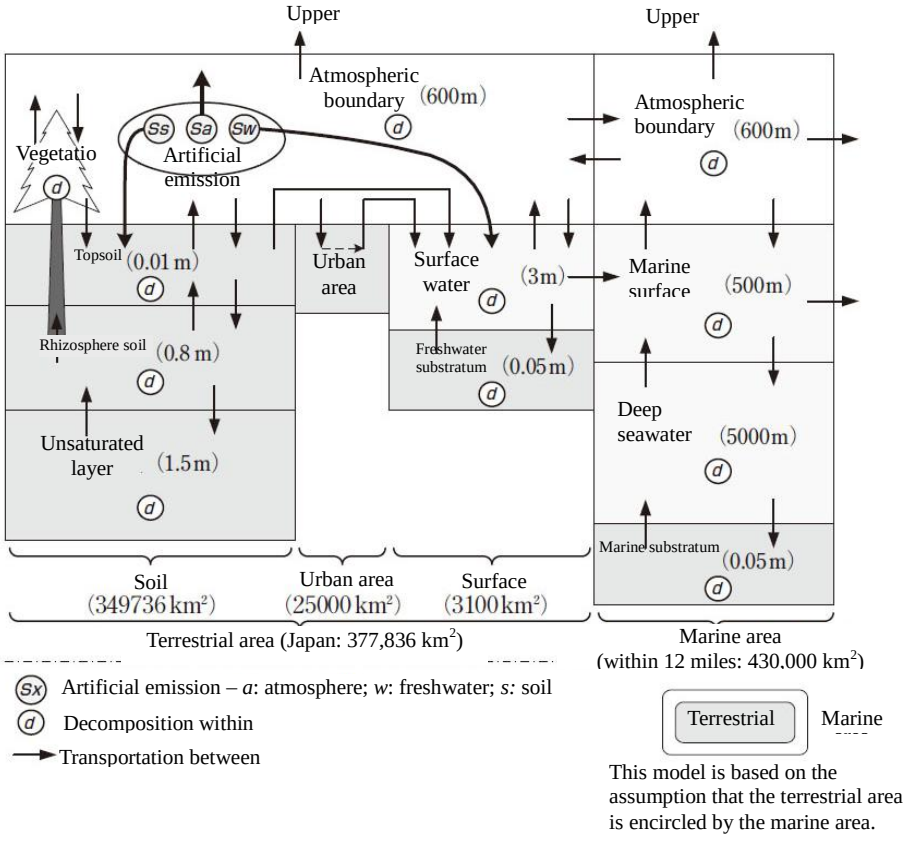


Figure 2.6-4: Pattern diagram of the fate analysis model under LIME

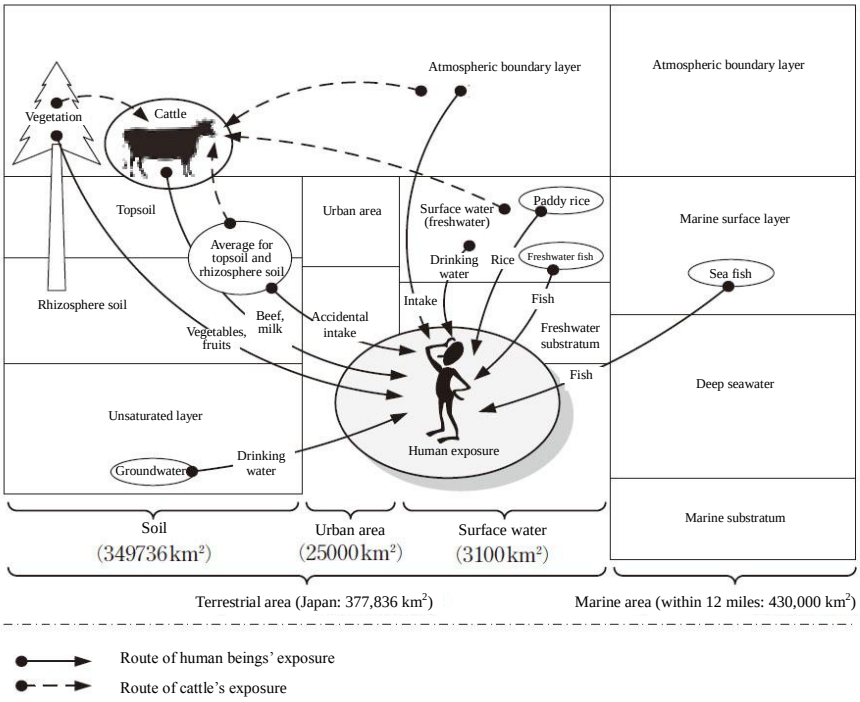


Figure 2.6-5: Exposure routes to be considered by the fate analysis model under LIME

b Impact factor for toxic chemicals

The maximum limit until which exposure concentration (inhalation) or intake (oral intake) is unlikely to have impact on human health (the limit is called the "human limited value" (HLV)) was used for the calculation of an impact factor, which was supposed to be the reciprocal of HLV.

If, like chronic diseases, a threshold level of pathogenesis exists concerning exposure concentration or amount, the threshold level is supposed to be HLV. The ADI value calculated from the no observed adverse effect level (NOAEL) was used as HVL (Equations 2.6-4 to 2.4-7).

$$NOAEL_h = NOAEL_a / UF_{a \rightarrow h} / UF_{sub\ Chronic \rightarrow Chronic} \quad (2.6\text{-}4)$$

$$ADI = NOAEL_h / SF \quad (2.6\text{-}5)$$

$$E_{inh} = 1 / HLV = 1 / ADI_{inh} \quad (2.6\text{-}6)$$

$$E_{oral} = 1 / HLV = 1 / ADI_{oral} \quad (2.6\text{-}7)$$

In this equation:

$NOAEL_a$:	NOAEL obtained from animal testing
$NOAEL_h$:	Human NOAEL
$UF_{a \rightarrow h}$:	Uncertainty factor concerning extrapolation from animals to human beings (species difference)
$UF_{sub\ Chronic \rightarrow Chronic}$:	Uncertainty factor concerning extrapolation from sub-chronic to chronic
SF:	Safety factor concerning individual difference

E_{inh}, E_{oral}: Effect factor of inhalation and oral intake of a toxic chemical
$ADI_{inh,}$ ADI_{oral}: ADI [mg/kg/day] of inhalation and oral intake of the chemical

On the other hand, if there is no threshold level of pathogenesis such as cancer, the amount of intake of a toxic chemical in the case of an increase in the lifetime carcinogenesis risk by 10^{-6} was supposed to be HLV and was calculated from the unit risk (Equations 2.6-8 and 2.6-9).

$$E_{inh} = 1 /(10^{-6} / UR_{inh}) \quad (2.6\text{-}8)$$

$$E_{oral} = 1 /(10^{-6} / UR_{oral}) \quad (2.6\text{-}9)$$

In this equation:

UR_{inh}, UR_{oral}: Unit risk of inhalation and oral intake of the substance [risk/ (mg/kg/day)] (for how to obtain each value, see 2.6.3 (3))

c Calculation of characterization factors for toxic chemicals

Toxicity potential is the total of the products of PDI of inhalation and oral intake of each toxic chemical and the effect factor (reciprocal of HLV). The total was divided by the value calculated in the same way concerning the reference substance to obtain HTP.

Under LIME, benzene emitted into the air was chosen as the reference substance, and the characterization factor was calculated for oral chronic toxicity and carcinogenesis each ($HTP_{i,ecomp}^{Chronic}$, $HTP_{i,ecomp}^{Cancer}$) (Equations 2.6-10 and 2.6-11).

$$HTP_{i,comp}^{Chronic} = \frac{\sum_r PDI_{i,comp,r} \cdot E_{i,r}}{\sum_r PDI_{benzene,air,r} \cdot E_{benzene,r}} = \frac{\sum_r PDI_{i,comp,r} / ADI_{i,r}}{\sum_r PDI_{benzene,air,r} / ADI_{benzene,r}} \quad (2.6\text{-}10)$$

$$HTP_{i,comp}^{Cancer} = \frac{\sum_r PDI_{i,comp,r} \cdot E_{i,r}}{\sum_r PDI_{benzene,air,r} \cdot E_{benzene,r}} = \frac{\sum_r PDI_{i,comp,r} /(10^{-6} / UR_{i,r})}{\sum_r PDI_{benzene,air,r} /(10^{-6} / UR_{benzene,r})} \quad (2.6\text{-}11)$$

Table 2.6-2 shows some of the calculated characterization factors. If there is sufficient information about the chronic toxicity of a substance, the substance is excluded from assessment (the substance is indicated by "–"). Note that, because the denominator differs between the chronic toxicity and the characterization factor, they cannot be compared simply.

With regard to the characterization factors for carcinogenesis, the substances that show large characterization factors are hexavalent chromium compound (air emission), ethylene oxide (water emission), and acrylamide (water emission). Although it has been recognized that hexavalent chromium compound has the possibility of causing cancer through inhalation, it has not been well recognized that it has the possibility of causing cancer through oral intake. Therefore, the characterization factor for air emission, which causes a large quantity of inhalation exposure, is large, while the characterization factors for water emission and soil emission are small. On the other hand, it has been recognized that acrylamide and ethylene

oxide are carcinogenic both through inhalation and through the oral route, and the result showed a high characterization factor for water emissions whereby the amount of exposure is the largest.

With regard to the characterization factors for chronic toxicity, acrylamide (water emission), benezene (water emission), and methacrylic acid (water emission) showed especially large values. Because the amount of oral exposure is the largest in the case of water emission, the result showed that the characterization factors for water emission is high.

Table 2.6-2: Characterization factors for human toxicity (partial)

	Characterization factors for carcinogenesis			Characterization factors for chronic toxicity		
Substance	(Air emission)	(Water emission)	(Soil emission)	(Air emission)	(Water emission)	(Soil emission)
Acrylamide	5.52E+02	2.24E+03	1.98E+00	6.68E+00	2.66E+01	2.35E-02
Ethyl acrylate	2.31E+00	1.12E+01	1.48E-01	-	-	-
Acrylonitrile	2.96E+01	5.67E+02	4.08E+00	-	-	-
Acetaldehyde	2.11E-01	4.88E-02	8.09E-03	-	-	-
Aniline	7.39E-01	9.54E+00	4.63E-02	1.17E-02	4.74E-01	1.91E-03
Ethyl acrylate	7.35E+01	4.10E+03	3.20E+01	-	-	-
Hexavalent chromium compound	5.14E+03	3.45E-15	3.44E-14	3.08E-01	9.99E-01	5.30E-01
Lead	7.71E+01	2.14E+02	1.38E+02	2.62E+00	7.52E+00	4.86E+00
Benzene	1.00E+00	2.95E-01	2.63E-01	1.00E+00	1.30E+01	2.30E-01
Methacrylic acid	-	-	-	7.52E-01	8.66E+00	4.81E-02

2.6.3 Damage assessment of toxic chemicals

(1) Basic policies for calculation of damage factors

Table 2.6-3 shows the category endpoints of human toxicity and the objects of calculation of damage factors under LIME. Although toxic chemicals cause various diseases, as described above, the number of assessment items that enable the obtainment of quantitative information is limited. Therefore, under LIME, the objects of assessment were limited to carcinogenesis and oral chronic diseases. However, with regard to the heavy metals newly reassessed under LIME 2,[1] because quantitative information can be obtained based on epidemiological surveys, inhalant chronic diseases were included in the objects of assessment. Main policies for and characteristics of the calculation of damage factors are as follows:

- As in the case of characterization factors, the geographical extent of damage factors is Japan and surrounding sea areas.

- With regard to carcinogenesis, 16 types of cancer, including lung cancer and uterus cancer, were covered.

- With regard to oral chronic diseases, among the non-communicable diseases (NCDs), 39 diseases, excluding congenital anomaly and oral diseases, were defined as oral chronic diseases.[2] Except for heavy metals, the average for all the diseases was

[1] Seven types of heavy metals were included in the objects of calculation: lead, cadmium, mercury, hexavalent chromium, arsenic, nickel, and antimony.

[2] It is hard to think that oral diseases are caused by exposure to chemicals. In addition, because DALY for a disease is very small and the incidence is very large (75% of the total incidence of non-communicable diseases), the average DALY for a chronic disease is very small if oral diseases are taken into consideration. For these reasons, oral diseases were excluded. In addition, because diseases that have

used for the calculation of "DALY per disease." With regard to heavy metals, the value calculated for each category of the International Classification of Diseases in ICD-10 codes was used.

- With regard to inhalant chronic diseases, because quantitative and unified data cannot be obtained, they were not included in the objects of assessment under LIME 1. However, they were included in LIME 2 because quantitative information concerning heavy metals could be obtained based on rich epidemiological surveys.

- Among the PRTR type-1 designated chemicals, the number of chemicals selected partially by reason of carcinogenesis is 74, while the number of chemicals selected partially by oral chronic toxicity is 151 (before the revision of the relevant cabinet order in 2008).

Figure 2.6-6 shows the flowchart of the calculation of the damage function and the damage factor.

Table 2.6-3: Category endpoints and damage functions included in the objects of calculation concerning human toxicity

Area of protection	Category endpoint		Objects of calculation of damage functions	
Human health	Carcinogenesis	Cancer caused by intake of carcinogenic toxic chemicals	○	16 types of cancer
	Oral chronic diseases	Diseases caused when a toxic chemical is repeatedly taken into a human body through intake of food and drinking water for a long period	○	Setting of "chronic diseases" with consideration for 39 types of diseases
	Inhalant chronic diseases	Diseases caused when a toxic chemical is repeatedly taken into a human body through inhalation for a long period	△	Setting of "chronic diseases" with consideration for 39 types of diseases * Limited to heavy metals
	Other diseases	Other diseases caused through intake of a toxic chemical	×	Quantitative information is poor.

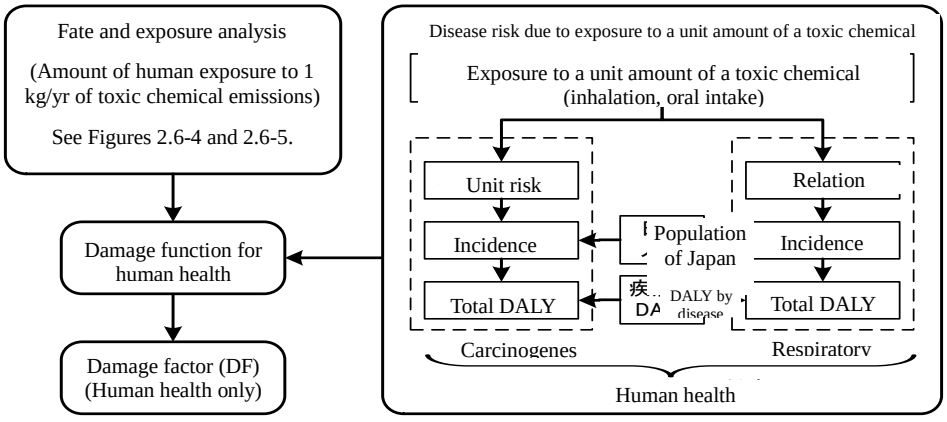


Figure 2.6-6: Flowchart of calculation of damage function and factor for human toxicity

not been caused by toxic chemicals may be included in the 39 types of diseases, it is necessary to examine this closely.

(2) Fate analysis of toxic chemicals

Fate analysis results obtained from the calculation of the characterization factor was used.

(3) Impact factor for toxic chemicals

The intensity of human toxicity in the area of protection of human health is quantitatively indicated by disability-adjusted life years (DALY). DALY, which was proposed by Murray et al. (1996a), indicates the amount of damage as loss of life expectancy. DALY is calculated by the following equation, based on YLL, which is the number of years lost due to death from disease, and YLD, which is the number of years lost due to survival with disability.

$$DALY = YLL + YLD \tag{2.6-12}$$

YLL and YLD are calculated from disease information, such as epidemiological statistical data and disability weight. With regard to such disease information, sequences to "Global Health Statistics" (GHS) (Murray et al. 1996a) and "Global Burden of Diseases" (GBD) (Murray et al. 1996b) are available on the website of the World Health Organization (WHO) and include epidemiological statistical data that can be used for the calculation.

If r is the discount rate, YLL and YLD can be calculated according to age and sex as in the following equation (the resultant values are not weighted by age):

$$YLL_{age,sex} = \frac{1-\exp(-rL_{age,sex})}{r} \quad (\text{if r=0, } YLL_{age,sex} = L_{age,sex}) \tag{2.6-13}$$

$$YLD_{age,sex} = T_{dur,sex}\left\{R_{treat}DW_{treated,age} + (1-R_{treat})DW_{untreated,age}\right\} \tag{2.6-14}$$

Therefore, the total loss of life expectancy at each age group, $DALY_{age}$ [DALYs], can be calculated by the following equation:

$$DALY_{age,sex} = N_{death,age,sex}YLL_{age,sex} + N_{inc,age,sex}YLD_{age,sex} \tag{2.6-15}$$

Therefore, the average DALY per incidence [DALYs/incidence] was calculated by the following equation:

$$DALY = \frac{\sum_{sex}\sum_{age} DALY_{age,sex}}{\sum_{sex}\sum_{age} N_{inc,age,sex}} \tag{2.6-16}$$

Table 2.6-4 summarizes the meanings of the variables in the equations and the concrete sources of values.

Under this method, to calculate DALY suitable for the conditions in Japan, available information on diseases in Japan was used. If such information was unavailable, GBD, GHS, and other statistical information (such as WHO) were used, selecting data on conditions similar to those in Japan (such as data in advanced countries) (Table 2.6-4).

Table 2.6-4: Variables used for the calculation of DALY and sources of values

Variable	Name of variable	Unit	Source (cancer)	Source (chronic disease)
$R_{inc,age,sex}$	Age-specific disease rate	[incidence /100000 persons]	Cancer statistics in Japan (National Cancer Center)	Global Health Statistics' classification for EME
$R_{death,age,sex}$	Age-specific death rate (ASDR)	[deaths /100000 persons]	Cancer statistics in Japan (National Cancer Center)	Global Health Statistics' classification for EME
$T_{dur,age,sex}$	Age-specific disease duration	[years]	Global Health Statistics' classification for EME	Same as left
$DW_{treated,age}$	Disability weight (treated)	NO unit	Global Burden of Disease's classification for EME	Same as left
$DW_{untreated,age}$	Disability weight (untreated)	NO unit	Global Burden of Disease's classification for EME	Same as left
R_{treat}	Treated rate	NO unit	Global Burden of Disease's classification for EME (Necessary only in the case of $DW_{treated,\ age} \neq DW_{untreated,\ age}$)	Same as left
$P_{age,sex}$	Age-specific population	[person]	Ministry of Health, Labour and Welfare's Vital Statistics FY2002 (Ministry of Health, Labour and Welfare's Information and Statistics Department 2001)	Global Burden of Disease's classification for EME
$L_{age,sex}$	Average life expectancy	[years]	Interpolation of WHO's Life Table of Japanese Men and Women	Same as left
$N_{inc,age,sex}$	Number of incidents	[person]	$= P_{age,sex} * R_{inc,age,sex}/100000$	Same as left
$N_{death,age,sex}$	Number of deaths	[person]	$= P_{age,sex} * R_{death,age,sex}/100000$	Same as left

DALY and the average DALY (applied to cases where body parts could not be identified) was calculated concerning cancers about which the unit risk of carcinogenesis could be obtained – mouth and oropharynx cancer, lung cancer, oesophagus cancer, stomach cancer, spleen cancer, liver/gallbladder cancer, rectum cancer, colon cancer, breast cancer, uterus cancer, ovary cancer, prostate cancer, bladder cancer, leukemia, lymphoma/myeloma, and skin cancer (Table 2.6-5). Although cancer and chronic disease are lifetime risks, the discount ratio for future impact *r* was fixed at 0.

On the other hand, with regard to chronic disease, except for heavy metals, DALY was calculated for the 36 types of diseases (8 categories) in Table 2.6-6. The resultant DALYs were averaged with the weight of incidence to obtain DALY for chronic disease. With regard to heavy metals, DALY in Japan was estimated for each of the 16 large categories of ICD-10 based on WHO's DALY for each country and patient statistics.

(4) Human health: cancer damage analysis

Under LIME, the incidence of cancer during the lifetime, which increases with a unit amount of toxic chemical emissions, is expressed as a unit risk (UR [risk/(mg/kg/day)]) (WHO 1987). The inhalation unit risk (IUR) and the oral slope factor (OSF) were obtained from the database (USEPA, IRIS Database) of the Integrated Risk Information System (IRIS) of the Environmental Protection Agency (EPA) and existing studies (Hofstetter 1998; Crettaz et al.

Table 2.6-5: DALY for cancer

Name of disease	DALYs/incidence [DALYs/inc.]	Incidences in Japan [inc.]	DALYs (Japan) [DALYs]
Oesophagus cancer	12.757	1.54E+04	1.96E+05
Stomach cancer	8.087	1.13E+05	9.11E+05
Colon cancer	7.138	6.43E+04	4.59E+05
Rectum cancer	7.413	3.49E+04	2.59E+05
Liver cancer	10.283	6.07E+04	6.24E+05
Pancreas cancer	16.199	2.07E+04	3.36E+05
Lung cancer	12.587	7.02E+04	8.84E+05
Breast cancer	8.282	3.43E+04	2.84E+05
Uterus cancer	7.033	1.83E+04	1.29E+05
Ovary cancer	15.279	7.05E+03	1.08E+05
Prostate cancer	5.271	1.85E+04	9.77E+04
Leukemia	18.992	8.47E+03	1.61E+05
Mouse and oropharynx cancer	8.078	9.51E+03	7.68E+04
Melanoma and other skin cancers	4.368	7.75E+03	3.39E+04
Bladder cancer	3.586	1.48E+04	5.32E+04
Lymphomas and multiple myeloma	11.535	1.63E+04	1.88E+05
Cancer	9.339	5.14E+05	4.80E+06

Table 2.6-6: DALY for chronic diseases

Category of chronic disease	Number of subcategories (typical diseases)	DALY [DALY/ incidence]	Incidences in EME [inc.]	DALYs (EME) [DALYs]
Diabetes	4 (leg gangrene, retinopathy, amputation, etc.)	1.384	4.40E+06	6.08E+06
Nervous/psychiatric symptom	11 (Parkinson's disease, epilepsy, melancholia, etc.)	0.563	4.46E+07	2.51E+07
Sensory disease	2 (glaucoma, cataract)	1.441	1.92E+05	2.77E+05
Cardiocirculatory disease	8 (myocardiosis, angina, congested heart failure, etc.)	11.096	4.31E+06	4.79E+07
Respiratory disease	2 (asthma, chronic obstructive pulmonary disease)	1.742	4.26E+06	7.43E+06
Digestive disease	3 (digestive ulcer, cirrhosis, appendicitis, etc.)	1.999	2.03E+06	4.05E+06
Genital/urinary disease	3 (nephritis, benign prostatic hyperplasia, etc.)	0.682	2.74E+06	1.87E+06
Musculoskeletal disease	3 (rheumatoid arthritis, osteoarthritis, etc.)	2.781	2.63E+06	7.32E+06
Chronic disease	―	1.535	6.51E+07	1.00E+08

Of the 39 types of diseases, the table shows the 36 types about which DALY could be obtained.

lungs through inhalation and indicates the incidence of cancer per unit concentration (1 μg/m^3) of the toxic chemicals contained in the inhaled air. IUR is multiplied by the daily amount of inhalation per kg of weight to obtain UR_{inh} [risk/ (mg/kg/day)]. OSF covers the toxic chemicals included in water, food, and other things taken orally into the digestive system and indicates the incidence of cancer per mg of intake per kg of weight per day. This was expressed as UR_{oral} [risk/ (mg/kg/day)].

If the unit risk cannot be obtained, supplement was made based on the carcinogenic class of PRTR. Concretely, if substances whose unit risk is already known and whose carcinogenic class is the same exist in the chemical group to which the type of chemical in question belongs (the type number under PRTR is the same as the cabinet order number), the average of their unit risks was applied.

In addition, because the carcinogenic risk list of the International Agency for Research on Cancer (IARC) was partially updated after LIME 1, the carcinogenic classes of the related substances were changed and the average of the unit risks was recalculated for supplement under LIME 2. Moreover, because unit risks of chemicals not covered by LIME 1 were established in EPA's chemical database IRIS, the number of substances to be assessed was increased.

(5) Human health: damage analysis of chronic diseases

Under LIME, the probability of suffering a chronic disease due to exposure to a unit amount of a toxic chemical was expressed by the D-R factor obtained based on epidemiological surveys. The D-R factor indicates the relation between the amount of intake and the incidence rate. Although chronic diseases are not defined clearly, they are defined as the non-communicable diseases other than congenital anomaly and oral diseases for the purpose thereof. The damage factor for chronic diseases is calculated by two methods. One of them was used for LIME 1; it is the method whereby the damage factor is calculated through estimation of the D-R factor from NOAEL and the lowest observed adverse effect level concerning most chemicals whose detailed epidemiological information is hard to obtain. The other method was used for heavy metals under LIME 2; it is the method whereby the damage factor is calculated by reference to the D-R factor based on rich epidemiological surveys and risk assessment documents. The following are explanations for the methods:

a Chemicals other than heavy metals

Only the oral chronic diseases are dealt with herein. With regard to the inhalant chronic diseases, because substances differ in uncertainty factor, they cannot be dealt with uniformly and are excluded here. The D-R factor for chronic diseases due to oral intake of a toxic substance of 1 mg/kg/day was estimated from EPA's IRIS Database (USEPA, IRIS Database) and existing studies (Hofstetter 1998; Crettaz et al. 2004a; Crettaz et al. 2004b).

Under PRTR, the following are used as criteria for judging whether a chemical has oral chronic toxicity:

1) Water quality standard

2) ADI, an index of oral toxicity of agricultural chemicals

3) NOAEL of repeated oral administration

4) LOAEL of repeated oral administration

These qualitatively indicate the intensity of toxicity and can be inferred to have a connection with the D-R factor. Because of this, a method for calculating a D-R factor was developed and applied from these indices.

The standard value and the index value were converted into a D-R factor by the following five-step procedure:

1) Calculation of NOAEL/LOAEL

If an index value of NOAEL/LOAEL for animals serves as a criterion, the value is used. If the water quality standard or the agricultural chemical standard serves as a criterion, NOAEL for animals is estimated from the standard value or the ADI of the agricultural chemical (Equations 2.6-17 and 2.6-18).

- ADI of agricultural chemical

$$NOAEL_a = ADI/SF \quad (2.6\text{-}17)$$

- Standard value of water quality

$$NOAEL_a = WQC/UF \times V_{DW} / BW \times r \quad (2.6\text{-}18)$$

In this equation:

NOAELa:	NOAEL for animals
SF:	Safety factor for agricultural chemical standard (100)
WQC:	WHO's or Japan's standard value of water quality
UF:	Uncertainty factor (100)
V_{DW}*:*	Volume of drinking water (2 L/day)
BW:	Weight of human body (WHO: 60 kg; Japan: 50 kg)
r:	Contribution rate of drinking water (WHO and Japan: 10%)

2) Correction of species difference

NOAEL (LOAEL) for animals is converted into NOAEL (LOAEL) for human beings with consideration for the species difference between human beings and animals. The factor for the conversion of species difference differs among documents; there is no uniform factor. The factor has been fixed at 10 for the purpose hereof.

3) Revision of exposure duration

NOAEL (LOAEL) for sub-chronic toxicity is converted to NOAEL (LOAEL) for chronic toxicity so that the results of short-term and mid/long-term experiments can be applied to lifelong exposure. Concretely, the empirical value obtained by Lewis et al. (1990) was adopted (correction factor: 3.3).

4) Estimation of ED_{10h} by correlating equation

ED_{10h} is estimated from NOAEL (LOAEL). ED_{10h} is 10% effect level [mg/kg/day]. Crettaz et al. (2004a, 2004b) obtained correlating equations, $ED_{10h} = 1.6 \cdot NOAEL$ and $ED_{10h} = 0.3 \cdot LOAEL$, from chemicals about which NOAEL/LOAEL and ED_{10h} were found. These equations were adopted herein.

5) Calculation of D-R factor

D-R factor is calculated from ED_{10h}, as expressed by the following equation:

$$DR \equiv \frac{0.1}{ED_{10h} - Thr} \qquad (2.6\text{-}19)$$

Although *Thr* is the threshold level, as shown in Figure 2.6-7, interpretation can be made in two ways according to the existence of a threshold level. Under this method, irrespective of background dose (concentration), it was decided that a D-R factor should be calculated, assuming that *Thr* = 0, taking into consideration the purpose of obtaining a factor that indicates a risk increment at every range of low dose (concentration).

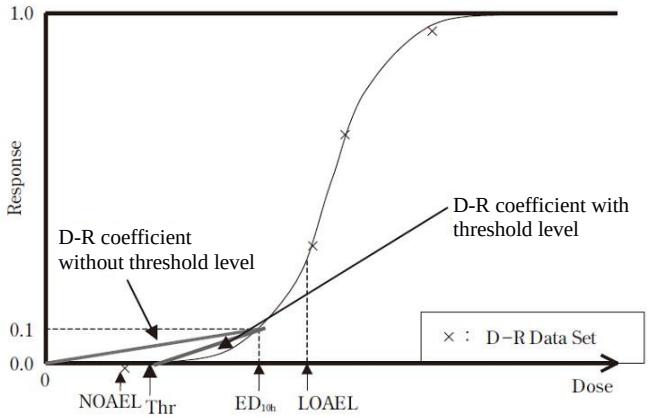


Figure 2.6-7: Difference in interpretation of D-R coefficient according to existence of threshold level

b Heavy metals

Epidemiological documents concerning dose-response relationships were examined for each type of heavy metal, and a slope of the straight line (hereinafter referred to as the "D-R factor") was calculated on the assumption that the dose-response curve can be approximated to a straight line. Not only oral chronic diseases but also inhalant chronic diseases were included in the objects of calculation.

1) Heavy metals covered

Lead, cadmium, mercury, hexavalent chromium, arsenic, nickel, and antimony

2) Documents examined

Under LIME 2, examination was carried out not about each treatise on an epidemiological survey of heavy metals but about various risk assessment documents. The following is the list of assessment documents:

- International Program on Chemical Safety (IPCS) INCHEM: Environmental Health Criteria Monographs (EHCs)
- Agency for Toxic Substances and Disease Registry (ATSDR): Toxicological Profile
- Environmental Protection Agency (EPA): IRIS
- National Institute of Advanced Industrial Science and Technology, Research Center

for Chemical Risk Management: Risk Assessment Documents

- Ministry of the Environment: Environmental Risk Assessment of Chemicals
- New Energy and Industrial Technology Development Organization (NEDO), National Institute of Technology and Evaluation: Initial Risk Assessments

3) Arrangement of D-R factors

The D-R factors obtained from the documents were arranged by type of disease and by exposure route. If D-R factors were different between the sexes and between adults and children, the information about them was arranged as well (Table 2.6-7).

Table 2.6-7: D-R factors for heavy metals used for LIME (partial extracts)

Substance		Disease covered by damage analysis		Damage analysis result			Source
		Disease		Exposure route	Target	D-R factor	
Lead		Anemia		Inhalation, oral intake	Adult	7.75E-03 (1/(μg/100mL))	a
					Child	3.33E-03 (1/(μg/100mL))	b
		Reduction in IQ points		Inhalation, oral intake	Child (up to 1 year)	1.74E-01 (points/(μg/100mL))	b
		Disorder of reproduction	Natural abortion	Inhalation, oral intake	Pregnant woman	3.60E-03 (1/(μg/100mL))	a, c
		Hypertension	Ischemic heart disease	Inhalation, oral intake	Hypertension incidence	2.07E-05 (1/(μg/100mL))	d
			Apoplexy	Inhalation, oral intake	Hypertension incidence	3.12E-06 (1/(μg/100mL))	d
Cadmium		Renal tubular disorder		Oral intake	Man	1.37E-03 (1/(μg/day))	b
				Oral intake	Woman	1.97E-03 (1/(μg/day))	b
Hexavalent chromium		Renal tubular disorder	Septonasal ulcer	Inhalation	-	3.82E-03 (1/(μg/day))	b, e
			Septonasal perforation	Inhalation	-	1.43E-03 (1/(μg/day))	b, e
Mercury	Inorganic mercury	Limb sensory organ disorder		Inhalation	-	1.53E-04 (1/(μg/day))	b
		Autonomic function disorder		Inhalation	-	4.60E-05 (1/(μg/day))	b
	Methyl mercury	Developmental nerve disorder		Oral intake	Pregnant woman	3.31E-04 (1/(μg/day))	d
		Abnormal perception		Oral intake	Child	3.08E-04 (1/(μg/day))	d

The unit of the denominator of the D-R factor for lead indicates not the amount of exposure but an increase in serum lead concentration. As for the other substances, the denominator indicates the amount of exposure per day.

[Sources]

a Agency for Toxic Substances and Disease Registry (ATSDR): Toxicological Profile Information Sheet

b IPCS INCHEM: Environmental Health Criteria Monographs（EHCs）

c Borja-Aburto VH, Hertz-Picciotto I, Lopez MR, et al. 1999. Blood lead levels measured prospectively and risk of spontaneous abortion. Am J Epidemiol 150: 590-597.

d the Empire State Electric Energy Research Corporation (ESEERCO): NEW YORK STATE ENVIRONMENTAL EXTERNALITIES COST STUDY, 1995

e LINDBERG E, HEDENSTIERNA G, (Swedish National Board of Occupational Health and safety) (1983) Chrome plating: Symptoms, findings in the upper airways, and effects on lung function. Arch Environ Health, 38 (6): 367-374

(6) Damage factor for toxic chemicals

A damage factor for human toxicity is a factor that indicates an increase in the amount of damage to human health due to additional emission of a toxic chemical. Two damage factors are examined herein concerning carcinogenesis and chronic diseases. Under LIME, it is assumed that the amount of damage increases linearly with emission of a chemical. Because of this, calculation of a damage function means calculation of its inclination.

a Calculation of the carcinogenic damage function

As shown in Equation 2.6-20, the number of sufferers from each type of cancer was estimated from PDI (daily amount of intake per kg of weight) of inhalation and oral intake gained from the result of fate analysis, the unit risk of carcinogenesis (Inhalation: UR_{inh}; oral intake: UR_{oral}), and the population of Japan. After that, the number is converted to DALY to obtain the damage function for carcinogenesis (Equation 2.6-20).

$$Canc_{i,ecomp} = \left(PDI_{i,ecomp,inh} \cdot \sum_{can} UR_{inh,i,can} \cdot DALY_{can} + PDI_{i,ecomp,oral} \cdot \sum_{can} UR_{oral,i,can} \cdot DALY_{can} \right) \cdot Pop_{Japan} \quad (2.6\text{-}20)$$

In this equation:

$Canc_{i, ecomp}$:	Factor for the carcinogenic damage function when toxic chemical *i* is emitted into compartment *ecomp* [DALYs]
$PDI_{i, ecomp, r}$:	PDI through the exposure route *r* (oral or inhalant) of toxic chemical *i* emitted into compartment *ecomp* [mg/kg/day]
$UR_{inh, i, can}$:	Unit risk of a type of cancer *can* though inhalation of a toxic chemical *i* [risk/ (mg/kg/day)] (conversion based on ($IUR_{i,can}$ [risk/ ($\mu g/m^3$)] and the daily amount of inhalation per 1 kg)
$UR_{oral, i, can}$:	Unit risk of a type of cancer *can* through oral intake of a toxic chemical *i* [risk/ (mg/kg/day)] (equal to $OSF_{i,cam}$)
$DALY_{can}$:	DALY of a type of cancer *can* [DALYs/incidence]
Pop_{Japan}:	Population of Japan (persons)

b Calculation of damage functions for chronic diseases

As shown in Equation 2.6-21, damage functions for chronic diseases were calculated by the use of PDI (daily amount of intake per kg of weight) of oral intake obtained from the fate analysis result, D-R factors for chronic diseases, DALY for chronic diseases estimated from the incidence of each disease classified as chronic disease; and the population of Japan beyond the threshold level.

<Other than heavy metals>

$$Chronic_{i,ecomp} = PDI_{i,ecomp,oral} \cdot \left(DR_i \cdot DALY_{chronic} \right) \cdot Pop_{Japan} \quad (2.6\text{-}21)$$

<Heavy metals>

$$Chronic_{i,ecomp} = \{ PDI_{i,ecomp,inh} \cdot (DR_{i,inh} \cdot DALY_{chronic}) + PDI_{i,ecomp,oral} \cdot (DR_{i,oral} \cdot DALY_{chronic}) \} \cdot Pop_{Japan}$$

In this equation,

$Chronic_{i, ecomp}$:	Factor of the damage function for chronic diseases when toxic chemical *i* is emitted into compartment *ecomp* [DALYs]
$DR_{i,oral}$:	Risk of suffering a chronic disease through oral intake of toxic chemical *i* [risk/ (mg/kg/day)]
$DR_{i,inh}$:	Risk of suffering a chronic disease through inhalant exposure of toxic chemical *i* [risk/ (mg/kg/day)]
$DALY_{chronic}$:	DALY for chronic disease [DALYs/incidence]
Pop_{Japan}:	Population of Japan (persons)

[Method of estimating the population that has the risk of suffering a chronic disease]

In the case of a chronic disease, if the amount of exposure exceeds the threshold level, there is a risk of suffering it.

With regard to substances other than heavy metals, because, as described above, the purpose was to obtain a factor that indicates a risk increment to every extent of low dose (concentration), a D-R factor was fixed on the assumption that *Thr* = 0 without consideration for the threshold level. Because of this, the population that has the risk of suffering a chronic disease is the population of Japan itself.

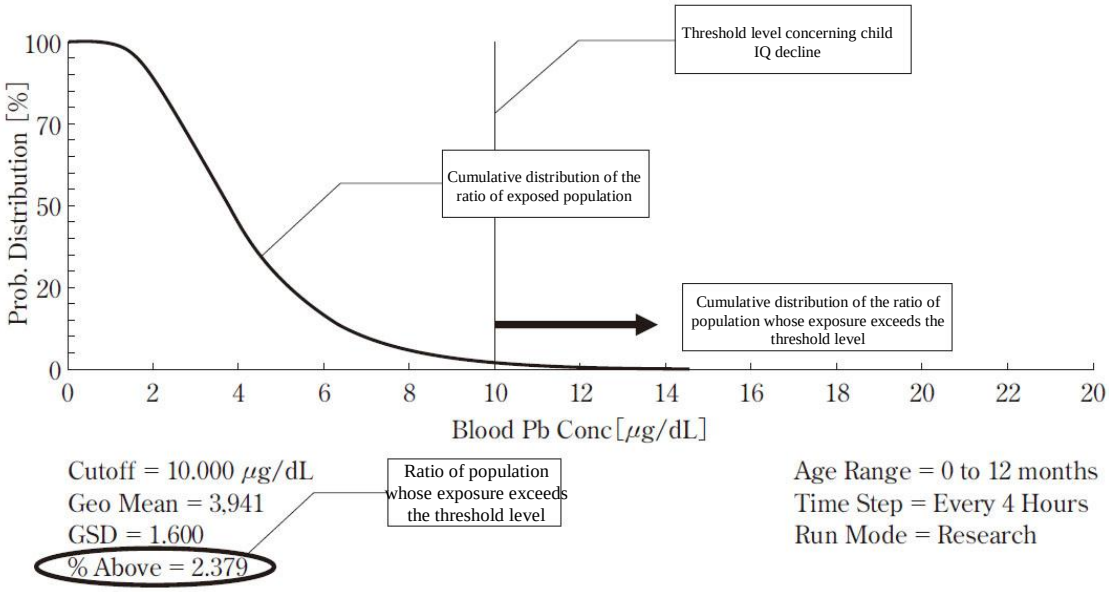


Figure 2.6-8: Exposed population distribution and the threshold level concerning decline in IQ of infants less than 1 year old due to exposure to lead (example of estimation by the IEUBK model)

On the other hand, with regard to heavy metals, because it was relatively easy to obtain data on the threshold level for each disease and the daily amount of intake, assessment was carried out in as much detail as possible. Concretely, by reference to the "Risk Assessment Documents: Lead" (National Institute of Advanced Industrial Science and Technology, Research Center for Chemical Risk Management 2006), a probability density distribution was estimated concerning the daily amount of exposure to heavy metals, collecting information on the amount of exposure via air, water, food, and soil, and carrying out a Monte Carlo simulation by the use of the statistics software "Crystal Ball" (Kozo Kaikaku Engineering) and an analysis by the biokinetic model of lead "IEUBK" (USEPA) (Figure 2.6-8). Based on the estimated probability density distribution, the ratio of the population beyond the threshold level for each disease was estimated and the population that has the risk of suffering a chronic disease in Japan was estimated (Equation 2.6-22).

[Population with the risk of suffering a chronic disease in Japan]
= [total population of Japan] × [ratio of population whose daily amount of exposure exceeds the threshold level] (2.6-22)

c Calculation of the damage factor (DF)

Based on the results of a and b, the damage factor DF was calculated as the total of the factors of the damage functions for carcinogenesis and oral chronic diseases as shown in the Equation 2.6-23:

$$DF_{i,ecomp} = Canc_{i,ecomp} + Chronic_{i,ecomp} \quad (2.6\text{-}23)$$

Table 2.6-8 shows examples of intake efficiency, unit risks (cancer), D-R factors (oral chronic diseases), and endpoints. Table 2.6-9 shows examples of the damage factor DF for human toxicity.

Of the substances whose damage factor has been entered, the following substances have relatively high damage factors: lead, hexavalent chromium compound, and acrylamide (in the case of air emission); lead, acrylamide, and benzene (in the case of water emission); and lead, hexavalent chromium compound, and ethylene oxide (in the case of soil emission). Therefore, it is necessary to examine the characteristics of lead, hexavalent chromium, acylamide, and benzene closely, as follows:

With regard to lead, the contribution of the damage function for chronic toxicity is large in all cases – air, water, and soil emission. When comparison is made among the media to which the substance is emitted, the damage factor is the highest in the case of water emission. This is because, if there is a possibility of suffering a disease due to both inhalant exposure and oral exposure, the amount of oral exposure is larger than the amount of inhalant exposure and therefore the damage factor is higher in the case of water emission.

With regard to hexavalent chromium, the contribution of the damage function for carcinogenesis through inhalant exposure is large in the case of air emission, while the contribution of the damage function for chronic toxicity is large in the case of water and soil emission. When comparison is made among the media to which the substance is emitted, the damage factor is the highest in the case of air emission. This is because it has been pointed out that hexavalent chromium has inhalant carcinogenicity and therefore the damage

factor becomes large at the time of air emission, which increases the amount of inhalant emission.

With regard to acrylamide, irrespective of the medium to which the substance is emitted, the contributions of the damage functions for oral carcinogenesis and oral chronic toxicity are high. When comparison is made among the media to which the substance is emitted, the damage factor is the highest in the case of water emission. This is because the amount of oral exposure is higher in the case of water emission.

With regard to benzene, the contribution of the damage function for carcinogenesis through inhalant exposure is high in the case of air emission, while the contribution of the damage functions for oral chronic diseases is high in the case of water and soil emission. When comparison is made among the media to which the substance is emitted, the damage factor is the largest in the case of water emission. This is because the value of damage function due to oral exposure is large, and therefore the amount of oral exposure is higher in the case of water emission.

(7) Comparison between LIME 1 and LIME 2 in damage factors

Figure 2.6-9 shows the results of comparison of the damage factors newly calculated under LIME 2 with those under LIME 1. Damage factors greatly increased concerning some substances, while they greatly decreased concerning several substances. Generally, there is no great difference between LIME 1 and LIME 2. Some of the substances whose damage factors greatly increased or decreased are listed below, together with reasons.

1) Substances whose damage factors greatly increased

Air emission: Cadmium, arsenic, hexavalent chromium compound, etc.
Water and soil emission: cadmium, arsenic, etc.

< Cause >
All of them are heavy metals, because epidemiological literature was examined closely to have the impact of chronic toxicity reflected in assessment results.

2) Substances whose damage factors greatly decreased

Air emission: bis (2-ethylhexyl) phthalate, methacrylic acid
Water and soil emission: 1,3-butadiene, acetaldehyde, formaldehyde, pentachlorophenol, nickel, nickel compound, hexavalent chromium compound

< Cause >
Under LIME 1, to prevent underestimation, damage functions were calculated concerning both substances with an inhalant carcinogenetic risk and those with an oral carcinogenetic risk. Under LIME 2, to make the judgment more strictly, damage functions were not calculated concerning substances that are still not recognized as having an inhalant carcinogenetic risk or an oral carcinogenetic risk. In addition, with regard to bis (2-ethylhexyl) phthalate, because the literature value adopted for LIME 1 as the no observed adverse effect level (NOAEL) was considerably high and the reliability of the value was doubtful, the value entered in IRIS was newly adopted.

Table 2.6-8: Intake efficiency, unit risks (cancer), D-R factors (oral chronic diseases), and endpoints (partial)

Substance	Inhalant intake efficiency [mg-chem intake/mg-chem emitted/Japanese pop.]			Oral intake efficiency [mg-chem intake/mg-chem emitted/Japanese pop.]			Intake efficiency†1	IUR [risk/(μg/m³)]†2	Cancer by inhalation	OSF [risk/(mg/kg-day)]†2	Oral carcinogenesis	D-R factor [risk/(mg/kg-day)]†3	Chronic toxicity
	Air emission	Water emission	Soil emission	Air emission	Water emission	Soil emission							
Acrylamide	1.08E-06	1.13E-11	7.10E-11	5.32E-06	2.55E-05	2.24E-08		1.30E-03 α	Av. for cancer	4.50E+00 α	Av. for cancer + uterus cancer + oral/pharynx cancer	1.24E+02 α	Chronic disease
Ethyl acrylate	1.43E-06	2.63E-07	8.16E-08	4.40E-09	2.63E-05	3.89E-08		2.74E-05 ε	Av. for cancer	2.10E+02 γ	Av. for cancer		
Acrylonitrile	7.19E-06	2.86E-06	4.41E-07	8.73E-08	5.25E-05	2.19E-07		6.80E-05 α	Lung cancer	5.40E-01 α	Av. for cancer + melanoma and other skin cancer + stomach cancer		
Acetaldehyde	1.03E-06	3.77E-07	6.26E-08	1.87E-08	3.08E-05	4.45E-08		2.20E-06 α	Av. for cancer				
Aniline	1.56E-06	8.70E-08	2.45E-08	5.30E-07	8.51E-05	3.19E-07		7.40E-06 β	Av. for cancer	5.70E-03 α	Av. for cancer	6.60E+01 γ	Chronic disease
Ethylene oxide	1.08E-05	5.10E-06	8.47E-07	1.41E-07	5.83E-05	3.86E-07		1.00E-04 β	Av. for cancer	3.57E+02 ε	Av. for cancer		
Hexavalent chromium compound	7.28E-06	4.90E-24	4.88E-23	2.81E-05	1.15E-04	6.10E-05		1.20E-02 α	Lung cancer			2.39E+02	Septonasal perforation†4
Benzene	4.40E-06	1.68E-06	8.40E-07	2.07E-09	5.18E-05	1.04E-07	α	4.14E-06 α	Leukemia	2.87E-02 α	Leukemia	2.89E+01 α	Chronic disease
Methacrylic acid	3.47E-06	5.08E-08	4.53E-08	5.17E-06	9.95E-05	5.07E-07						1.03E+01 δ	Chronic disease

†1 Note on intake efficiency:
α: The intake efficiency of the same metal element was used as a substitute.

†2 About carcinogenic OSF (oral slope factor) and IUR (inhalation unit risk)
α: Cited from the database of IRIS
β: Cited from Hofstetter's literature
γ: Cited from Crettaz's literature
δ: The same carcinogenesis class and the same chemical species were used as substitutes.
ε: The same cancer class geometric average under PRTR

†3 Sources of original data for calculation of chronic disease D-R factors (other than heavy metals)
α: Water qualification standard (WHO)
β: Long-term NOAEL (IRIS)
γ: Long-term LOAEL (IRIS)
δ: Irregular NOAEL (assessment sheet)
ε: Agricultural chemical ADI
* With regard to heavy metals, D-R factors have been cited directly from various epidemiological survey cases.

†4 There are several types of chronic diseases to be assessed concerning heavy metals. One of them has been picked up herein.

Table 2.6-9: Damage factors for human toxicity (partial)

Substance	Damage factor for human carcinogenesis through inhalant exposure			Damage factor for human carcinogenesis through oral exposure			Damage factor for chronic toxicity through oral exposure			Damage factors for carcinogenesis through inhalant and oral exposure and chronic toxicity through oral exposure		
	[DALYs/kg]			[DALYs/kg]			[DALYs/kg]			[DALYs/kg]		
	(Air emission)	(Water emission)	(Soil emission)	(Air emission)	(Water emission)	(Soil emission)	(Air emission)	(Water emission)	(Soil emission)	(Air emission)	(Water emission)	(Soil emission)
Acrylamide	2.58E-05	2.70E-10	1.69E-09	3.82E-04	1.83E-03	1.61E-06	6.59E-04	3.15E-03	2.78E-06	1.07E-03	4.98E-03	4.39E-06
Ethyl acrylate	7.19E-07	1.32E-07	4.09E-08	5.63E-10	3.37E-06	4.97E-09				7.09E-07	3.50E-06	4.59E-08
Acrylonitrile	1.20E-05	4.79E-06	7.39E-07	6.70E-07	4.03E-04	1.68E-06				1.27E-05	4.08E-04	2.42E-06
Acetaldehyde	6.55E-08	1.52E-08	2.52E-09							6.55E-08	1.52E-08	2.52E-09
Aniline	2.11E-07	1.18E-08	3.32E-09	1.84E-08	2.95E-06	1.11E-08	3.50E-07	5.62E-05	2.11E-07	5.80E-07	5.92E-05	2.25E-07
Ethylene oxide	1.98E-05	9.32E-06	1.55E-06	3.06E-06	1.27E-03	8.39E-06				2.29E-05	1.28E-03	9.94E-06
Hexavalent chromium compound	2.15E-03	1.45E-21	1.44E-20				2.90E-05	1.19E-04	6.29E-05	2.18E-03	1.19E-04	6.29E-05
Lead	3.65E-06	0.00E+00	4.85E-23	2.03E-05	6.65E-05	4.30E-05	1.98E-02	4.75E-02	3.60E-02	1.98E-02	4.76E-02	3.60E-02
Benzene	6.32E-07	2.59E-07	1.29E-07	7.36E-10	1.84E-05	3.70E-08	5.98E-08	1.50E-03	3.01E-06	6.92E-07	1.51E-03	3.17E-06
Methacrylic acid							5.33E-05	1.03E-03	5.24E-06	5.33E-05	1.03E-03	5.24E-06

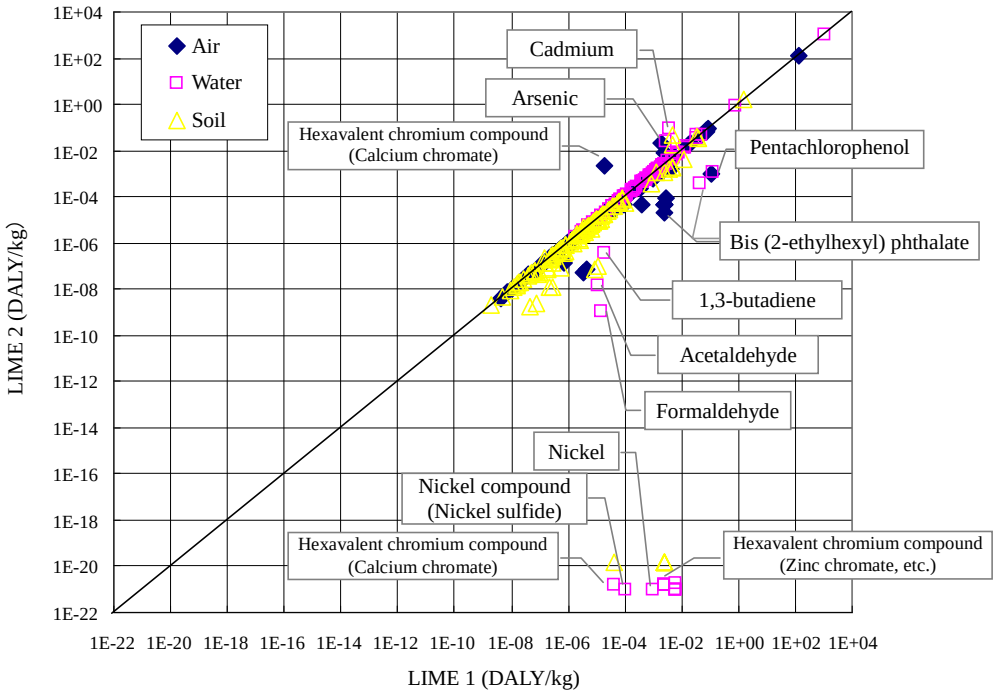


Figure 2.6-9: Comparison between LIME 1 and LIME 2 (toxic chemicals)

2.6-4 Procedure for impact assessment of a toxic chemical

This part concretely describes the procedure for impact assessment of a toxic chemical, including characterization, damage assessment, and weighting. Although the toxic chemical and the medium to which the substance is emitted were so far described as *i* and *r* respectively, they are described as *X* and *R* respectively herein to coordinate with the other impact categories (other sections).

Users can select what is suitable for their purpose from among characterization, damage assessment, and weighting and use it for LCA.

The characterization results $CI^{HumanTox\_Cancer}$ and $CI^{HumanTox\_Chronic}$ can be obtained from *Inv* (*X*,*R*), the amount of emission (inventory) of the toxic chemical *X* to the medium to which the substance is emitted (air, water, soil) *R*, and the characterization factors for cancer and chronic toxicity $CF^{HumanTox\_Cancer}(X,R)$ and $CF^{Humantox\_Chronic}(X,R)$ (Equations 2.6-24 and 2.6-25).

$$CI^{HumanTox\_Cancer} = \sum_X \sum_R CF^{HumanTox\_Cancer}(X,R) \cdot Inv(X,R) \qquad (2.6\text{-}24)$$

$$CI^{HumanTox\_Chronic} = \sum_X \sum_R CF^{HumanTox\_Chronic}(X,R) \cdot Inv(X,R) \qquad (2.6\text{-}25)$$

Because the characterization factor $CF^{HumanTox}(X,R)$ differs according to *R*, the medium to which the substance is emitted, it is necessary to divide inventory data according to it.

Column 2.6-1

Relation between [damage factor] and [damage factor × amount of emissions]

A damage factor indicates the amount of damage due to a unit amount of chemical emissions. Given the actual amount of emissions in the whole Japan, how does the amount of damage by each substance change? In this column, the relation between [damage factor] and [damage factor × PRTR amount of emissions] was analyzed by the use of the average of the past five years' PRTR data on the amount of chemical emissions in the whole Japan (FY2002 to FY2006). Figure 2.6-A shows the result.

Substances can be classified into three groups according to changes in ranking.

(1) Substances that hold a high rank in both [damage factor] and [damage factor × PRTR amount of emissions]

(2) Substances that hold a considerably lower rank in [damage factor × PRTR amount of emissions] than the rank in [damage factor]

(3) Substances that hold a considerably higher rank in [damage factor × PRTR amount of emissions] than the rank in [damage factor]

Substances falling under (1) cause both a large amount of damage per unit amount and a large amount of damage in the whole of Japan. Substances falling under (2) cause a relatively small amount of damage due to a relatively small amount of emissions in Japan, although the amount of damage per unit amount is high. Substances falling under (3) cause a large amount of damage due to a large amount of emissions in Japan, although the amount of damage per unit amount is small.

Some of the substances falling under each of the three groups are listed below. Heavy metals fall under (1), and dioxin falls under (2). In addition, agricultural chemicals fall under (3) because the amount of emissions not reported is relatively large. However, attention must be paid to the fact that uncertainty is high because the amount of emissions not reported is an estimate.

[(1) Substances that hold high ranks in both [damage factor] and [damage factor × PRTR amount of emissions]]

Lead: [damage factor] 4th place → [damage factor × PRTR amount of emissions] 1st place
Cadmium: [damage factor] 3rd place → [damage factor × PRTR amount of emissions] 7th place
Arsenic: [damage factor] 8th place → [damage factor × PRTR amount of emissions] 8th place

[(2) Substances that hold a high rank in [damage factor] but a low rank in [damage factor × PRTR amount of emissions]]

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (dioxin group): [damage factor] 1st place → [damage factor × PRTR amount of emissions] 27th place
Berylium: [damage factor] 12th place → [damage factor × PRTR amount of emissions] 72nd place

[(3) Substances that hold a low rank in [damage factor] but a high rank in [damage factor × PRTR amount of emissions]]

1,3-Dichloropropene: [damage factor] 40th place → [damage factor × PRTR amount of emissions] 3rd place
2-Thioxo-3,5-dimethyltetrahydro-2*H*-1,3,5-thiadiazine: [damage factor] 56th place → [damage factor × PRTR amount of emissions] 6th place

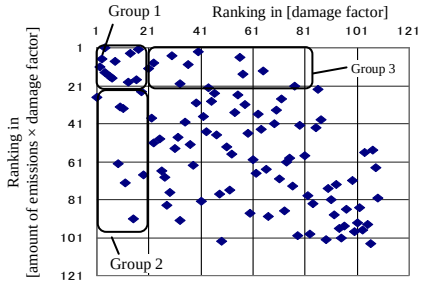


Figure 2.6-4A: Relation between [damage factor] and [damage factor × amount of emissions]

There are several methods for calculating a characteristic factor. LIME recommends that HTP, characterization factors obtained from the ratio between the result of fate and exposure analysis and the permissible amount of intake based on the environmental conditions in Japan, be used as a list of characterization factors. Because HTP is a set of factors based on the air emission of benzene, $CI^{HumanTox}$ can be regarded as the total emissions of toxic chemicals converted into the amount of air emissions of benzene, the reference substance. Given that the meaning of the threshold level differs between carcinogenic substances and chronic toxicity, it became possible to carry out assessment after distinguishing carcinogenesis from chronic toxicity.

In addition, the damage assessment result *DI* (*Safe*) can be obtained from each toxic chemical's *Inv* (*X*, *R*) and the damage factor for *Safe*, each area of protection, $DF^{HumanTox}$ (*Safe*, *X*, *R*) (Equation 2.6-26):

$$DI(Safe) = \sum_{X}\sum_{R} DF^{HumanTox}(Safe, X, R) \cdot Inv(X, R) \qquad (2.6\text{-}26)$$

Because, as in the case of $CI^{HumanTox}$, the damage factor $DF^{HumanTox}$ (*Safe*, *X*, *R*) differs according to *R*, the medium to which the toxic chemical is emitted, inventory data must be divided according to it.

DI (*Safe*) means the amount of latent damage to the area of protection *Safe* due to toxic chemical emissions. It is possible to carry out damage assessment of toxic chemicals against human health. *DI* (*Safe*) can be compared and integrated with the amount of damage to the area of protection common to different impact categories – that is, the amount of damage to human health through the impact categories other than toxic chemicals.

The integration factor converted economically from impact on human health or converted into zero dimension $IF^{HumanTox}$ (*X*, *R*) are used for integration. The single index *SI* can be obtained from each toxic chemical's *Inv* (*X*, *R*) and the integration factor $IF^{HumanTox}$ (*X*, *R*).

The result can be compared directly with or added to assessment results in other impact categories (Equation 2.6-27).

$$SI = \sum_{X} \left(IF^{HumanTox}(X) \times Inv(X) \right) \qquad (2.6\text{-}27)$$

Appendices A1, A2, and A3 show the characterization factor $CF^{HumanTox}(X,R)$, the damage factor $DF^{HumanTox}(Safe, X, R)$, and the integration factor $IF^{HumanTox}(X, R)$, respectively.

[References for Section 2.6]

CML (2001): Life cycle assessment. An operational guide to the ISO standards. Final report, CML Leiden University, Leiden

Crettaz P et al. (2004a): Assessing Human Health Response in Life Cycle Assessment using ED10s and DALYs - Part 1: Cancer Effects

Crettaz P et al. (2004b): Assessing Human Health Response in Life Cycle Assessment using ED10s and DALYs - Part 2: Non - cancer Effects

Guinée J et al. (1996): USES, Uniform System for the Evaluation of Substances. Inclusion of fate in LCA characterization of toxic releases applying USES 1.0. Generic modeling of fate, exposure and effect for ecosystems and human beings with data for about 100 chemicals, Int. J. LCA, 1:133-138

Hauschild M, Wenzel H (1998): Environmental Assessment of Products. Volume 2: Scientific background, Chapman & Hall, London

Heijungs R et al. (1992): Environmental Life Cycle Assessment of Products. Guide and Backgrounds, CML Leiden University, Leiden

Hertwich E (1999): Toxic Equivalency: Addressing Human Health Effects in Life Cycle Impact Assessment. Thesis, University of California, Berkeley

Hofstetter P (1998): Perspective in Life Cycle Impact Assessment, Kluwer Academic Publishers, Dordrecht

Huijbregts MAJ (1999): Priority Assessment of Toxic Substances in LCA. Development and Application of the Multi-media Fate, Exposure and Effect Model USES-LCA. IVAM environmental research, University of Amsterdam, Amsterdam

Jolliet O, Crettaz P (1997): Critical surface-time 95. A life cycle impact assessment methodology including fate and exposure. Swiss Federal Institute of Technology, Institute of Soil and Water Management, Lausanne

Lindfors LG et al. (1995): LCA-NORDIC Technical Reports No. 10. Nordic Council of Ministers, Copenhagen

Murray CLJ et al. (1996a): Global Health Statistics, Harvard University Press, Boston

Murray CLJ et al. (1996b): The Global Burden of Diseases Vol. I. WHO/Harvard School of Public Health/World Bank, Harvard University Press, Boston

Udo de Haes HA et al. (1996): Practical Guidelines for Life Cycle Assessment for the EU Ecolabeling Programme, Leiden

USEPA: IRIS Database

WHO (1987): Air Quality Guidelines for Europe, World Health Organization, Geneva

Chemical Products Council (2000): Designation of Type-1 Designated Chemicals and Type-2 Designated Chemicals under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting. Improvements in Their Management (Report)

Ministry of Health, Labour and Welfare's Information and Statistics Department (2001): Vital Statistics of Japan, Vol. 2

National Cancer Center (2001): Cancer Statistics

National Institute of Advanced Industrial Science and Technology's Research Center for Chemical Risk Management (2006): Risk Assessment Documents 9: Lead