LIME2

Life-cycle Impact assessment Method based on Endpoint modeling

Chapter 2 (2.7-2.9) Characterization and Damage Evaluation Methods

2.7 Ecotoxicity
2.8 Eutrophication
2.9 Indoor air pollution

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Chapter II

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2.7 Ecotoxicity

Changes between LIME 1 and LIME 2

- Although the data on the ecotoxicity of chemicals in ECOTOX of the United States Environmental Protection Agency (USEPA) were used for LIME 1, because the data have recently been renewed greatly, the data as of 2007 are used for LIME 2. As a result, the number of substances included in the list of damage factors increased greatly, from 89 in LIME 1 to 297 (in 155 groups) in LIME 2.
- Under LIME 1, the data on the ecotoxicity of chemicals harmful to fishes, crustaceans, and algae as groups of organism species were used and three types of mean values were used for toxicity data on the other aquatic organisms. Under LIME 2, however, it was decided to reflect ecotoxicity data in each of the other organism species (amphibians, shellfishes, crustaceans, etc. (other than crustaceans)).
- The number of endangered species in the Red Data Book (RDB) was used for LIME 1. For the purpose of LIME 2, the data were renewed to those as of December 2006.

2.7.1 What phenomenon is ecotoxicity?

(1) Causal relationships of ecotoxicity

When a heavy-metal or organic compound is released from a factory, it, being transformed or decomposed, diffuses into various environmental media, such as the air, water, and soil, causing a change in the concentration of chemicals in the environmental media. How the environment changes greatly differs according to the type of chemical. Some chemicals change their concentration so greatly as to influence individual organisms, sometimes causing a change in the population of species or a change in the regional ecosystem balance and, as a result, becoming a factor for the extinction of species. Such occurrence of an undesirable situation for the ecosystem due to the release of a toxic chemical is herein called "ecotoxicity." Because it is rumored that many chemicals, such as dioxin and poly chlorinated biphenyl (PCB), have toxicity, how to protect the ecosystem from these toxic chemicals has become an extremely important challenge to modern society.

Figure 2.7-1 shows the cause-effect chain of ecotoxicity. The endpoints in the figure are ones that are generally regarded as problematic.
When ecotoxicity was assessed under the LCA so far, risk assessment was carried out by the use of the ratio of the predicted environmental concentration (PEC) estimated by fate analysis to the predicted no-effect concentration (PNEC) estimated from the no observed effect concentration (NOEC) of the species in question. Under LCIA, such an approach has been used for the development of characterization factors as midpoint level assessment. Under LIME, an ecotoxicity assessment method was created with new endpoints by the method to evaluate the extinction probability by the use of not only characterization factors based on the environmental conditions in Japan but also a population dynamics model.

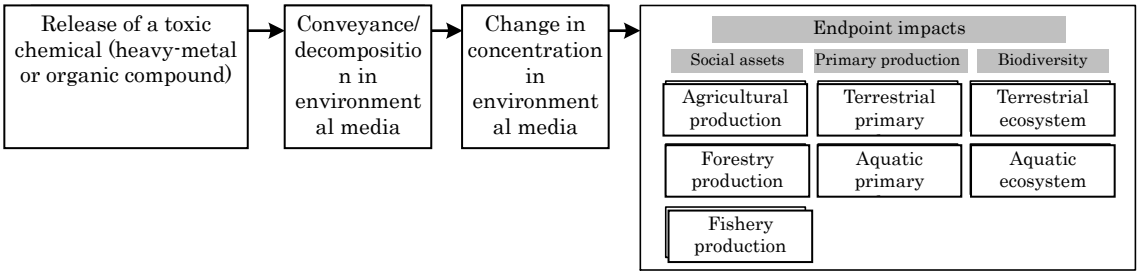

Figure 2.7-1: Causal relationships of ecotoxicity

(2) Endpoints of ecotoxicity

a Impact on biodiversity

If a toxic chemical is released into the environment, the concentration of toxic chemicals in the environment changes and the soundness of individuals of a certain species may receive damage. Impact on the dynamic state of the environment and species differs according to the type of chemical. In the case of some chemicals, even if the amount of released chemicals is extremely small, some species receive serious damage.

If the soundness of individuals of a certain species receives impact, the population may change greatly, resulting in extinction. Because, whether in terrestrial or aquatic areas, all the species in the ecosystem exist by balancing with various other species, a change in the population of a species or the extinction of a species will break down the balance of the biodiversity in the whole ecosystem.

b Impact on primary production and social assets

If a toxic chemical is released into the environment, impact may arise on the primary production (the number of plankton individuals, production of terrestrial plants), which is important for the ecosystem. Moreover, a change in the primary production due to the release of a chemical has impact on zooplankton, which eat phytoplankton. This leads to the extension of the impact to upper stages of the food chain. As a result, fishery and other social assets valuable for human beings receive the impact of the release of the toxic chemical, such as a reduction in yield and a loss of qualification. However, there has so far been hardly any information on changes in plant growth volume due to the release of a chemical.

2.7.2 Characterization of ecotoxicity

(1) Existing characterization factors of ecotoxicity

Because areas are divided into aquatic and terrestrial areas, the aquatic ecotoxicity potential (AETP) and the terrestrial ecotoxicity potential (TETP) are used as the characterization factor of ecotoxicity – that is, the ecotoxicity potential (ETP). Although AETP and TETP are calculated in the same way as the human toxicity potential (HTP), there is no concept of intake as in the case of human beings. Instead, two factors are taken into consideration – the fate and effect of toxic chemicals in environmental media (water and soil). That is, the concentration in environmental media as a result of fate analysis corresponds to the exposure

concentration.

Like the human toxicity characterization factors, the ecotoxicity characterization factors developed early (Heijungs et al. 1992) did not take into consideration the fate of released chemicals in the environment. Recently, however, a lot of those that use results of fate analyses have been developed (Hauschild et al. 1998, Guinée et al. 1996, Huijbregts 1999, Hertwich 1999, Jolliet et al. 1997).

The general form of ETP is as follows (Equation 2.7-1):

$$ETP_{i,ecomp} = \frac{\sum_{fcomp} F_{i,ecomp,fcomp} \times E_{i,fcomp}}{\sum_{fcomp} F_{ref,ecomp,fcomp} \times E_{ref,fcomp}} \qquad (2.7\text{-}1)$$

In this equation, $ETP_{i,ecomp}$ is the ETP when the substance *i* is released into the environmental medium *ecomp*. $F_{i,ecomp,fcomp}$ (fate factor) is the amount of the released *i* that reaches *fcomp*, the final environmental medium just before exposure. $E_{i,fcomp}$ (effect factor) is the toxic effect of the exposure of the organisms growing or living in *fcomp* to *i*. *ref* is the base substance. Concretely, *F* is the increment in the concentration in the environmental medium if a unit amount of the substance *i* is released. *E* is usually the reciprocal of the maximum predicted no-effect concentration (PNEC) of the substance *i* that meets the species conservation target in the ecosystem (usually, conservation of 95%). That is, the stronger the substance's toxicity, the lower the PNEC (effect occurs even if the amount of the chemical is small) and the larger the *E*. Therefore, the higher the exposure efficiency and the stronger the chemical's toxicity, the larger the ETP.

(2) Characterization factors of ecotoxicity under LIME

a Basic policy for calculation of characterization factors

Under LIME, characterization factors were calculated after research and examination of concentration changes in environmental media at the time of release of chemicals and the impact factors of chemicals on each species. Figure 2.7-2 shows the flowchart of estimation of characterization factors.

b Fate analysis of toxic chemicals

Like the toxicity assessment of substances harmful to human health (Section 2.6), the multimedia model developed by École Polytechnique Fédérale de Lausanne (EPFL) was improved and used for fate analysis of released chemicals. This model geographically covers Japan and the surrounding seas and consists of the following environmental media: the air, water, aquatic bottom sediments, the marine surface layer, deep seawater, marine bottom sediments, surface soil, rhizospheric soil, unsaturated layer soil, the water-bearing layer, vegetation regions, and urban regions. This model can be used for considering transportation between environmental media, decomposition within a medium, and the process of external movement. By the use of this model, calculation was made about $PEC_{i,ecomp,fcomp}$ the concentration of the substance *i* in the final environmental medium *fcomp* if *i* is released to the environmental medium *ecomp*. The result was used as the fate factor ($F_{i,ecomp,fcomp}$ in Equation 2.7-1). When characterization factors were calculated, consideration was

given to three media to which the substance is released (*ecomp*): the air, water, and soil. In addition, the final environmental medium, *fcomp*, is water in the case of the aquatic ecosystem, while it is soil in the case of the terrestrial ecosystem.

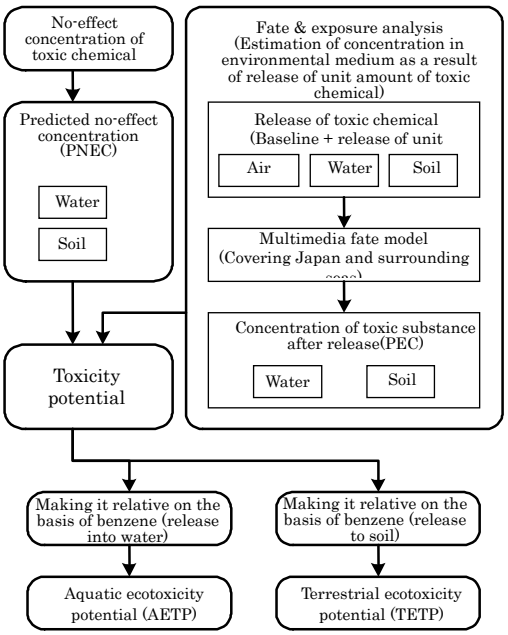

Figure 2.7-2: Flowchart of calculation of characterization factors for ecotoxicity

c Effect factor of the toxic chemical

The effect factor ($E_{i,\,fcomp}$ in Equation 2.7-1) is the reciprocal of PNEC. PNEC ($PNECaqua_i$, $PNECterr_i$) in the aquatic ecosystem or the territorial ecosystem for each chemical was obtained from the existing LCIA study cases (Huijbregts 1999) (Table 2.7-1).

d Calculation of characterization factors of ecotoxicity

The resultant PEC was divided by PNEC to find the base value of toxicity assessment. The base value was divided by the value calculated in the same way concerning the base chemical to make the base value relative and find AETP and TETP. Under LIME, the base chemical was benzene released into water in the case of AETP and benzene released into soil in the case of TETP. In other words, this is the factor for estimating how many times higher the impact becomes compared with the impact when the same amount of benzene is released into the environment (Equations 2.7-2 and 2.7-3).

$$AETP_{i,\,ecomp} = \frac{PEC_{\,i,\,ecomp,\,water} \,/\, PNECaqua_{\,i}}{PEC_{\,benzene,\,water,\,water} \,/\, PNECaqua_{\,benzene}} \qquad (2.7\text{-}1)$$

$$TETP_{i,\,ecomp} = \frac{PEC_{\,i,\,ecomp,\,soil} \,/\, PNECterr_{\,i}}{PEC_{\,benzene,\,soil,\,soil} \,/\, PNECterr_{\,benzene}} \qquad (2.7\text{-}2)$$

Table 2-7-1: Ecotoxicity characterization factors developed under LIME

(a) AETP (aquatic ecotoxicity potential)

Place for release	Predicted no-effect concentration (PNECaqua) [kg/m^3]	Predicted effect concentration (PEC) in surface water [(g/m^3)/ (t/yr)]			Aquatic ecotoxicity potential (AETP) (estimated based on the case where benzene is released into water)		
		Air	Water	Soil	Air	Water	Soil
Benzene	2.40E-03	1.38E-12	1.70E-08	2.95E-11	8.12E-05	1.00E+00	1.73E-03
Arsenic	2.40E-05	9.68E-07	2.65E-06	1.81E-06	5.68E+03	1.55E+04	1.06E+04
Trichloroethylene	2.40E-03	4.52E-12	4.79E-08	1.18E-10	2.66E-04	2.81E+00	6.92E-03
Dichloromethane	2.00E-02	4.45E-11	9.92E-08	3.01E-09	3.13E-04	6.99E-01	2.12E-02
Cadmium	3.40E-07	5.88E-07	1.90E-06	9.08E-07	2.44E+05	7.89E+05	3.76E+05

(b) TETP (terrestrial ecotoxicity potential)

Place for release	Predicted no-effect concentration (PNECterr) [kg/kg (dwt)]	Predicted effect concentration (PEC) in surface soil [(g/m^3)/ (t/yr)]			Terrestrial ecotoxicity potential (TETP) (estimated based on the case where benzene is released into soil)		
		Air	Water	Soil	Air	Water	Soil
Benzene	4.19E-06	4.00E-13	1.64E-13	4.53E-09	8.83E-05	3.61E-05	1.00E+00
Arsenic	3.60E-07	2.97E-05	2.56E-20	1.09E-04	7.64E+04	6.57E-11	2.80E+05
Trichloroethylene	5.67E-06	1.93E-12	9.69E-13	1.79E-08	3.15E-04	1.58E-04	2.92E+00
Dichloromethane	1.70E-05	6.26E-12	3.75E-12	9.18E-08	3.40E-04	2.04E-04	4.99E+00
Cadmium	4.60E-07	2.96E-06	0.00E+00	1.08E-05	5.94E+03	0.00E+00	2.17E+04

In Western countries, Guinée (1996) and Hauschild (1998) developed characterization factors for ecotoxicity. Although these factors are based on the multimedia fate model, they cannot be used for assessment in Japan, because the environmental conditions set for the model were based on data in Western countries. Because the geographical characteristics in Japan were taken into consideration for the fate analysis under LIME, we recommend the ETP developed under LIME as the characterization factor for ecotoxicity. Tables 2.7-1 (a) (b) show the characterization factors of main chemicals.

e Characteristics of characterization factors of ecotoxicity

First, we explain the characteristics of benzene, the base substance. PNEC related to toxicity against aquatic creatures is higher than that against terrestrial creatures, by about three orders of magnitude. On the other hand, PEC in surface water at the time of release into water is higher than PEC in surface soil at the time of release into soil, by about one order. Because of this, the value of PEC/PNEC concerning aquatic ecotoxicity at the time of release into water is lower than the value concerning terrestrial ecotoxicity at the time of release into soil, by about two orders. If they are used as base values and the characterization factors are compared with those in the case of the release of benzene into other environmental media, AETP at the time of release to soil is lower than that at the time of release into water by about

three orders, and AETP at the time of release into the air is lower than that at the time of release into water by about five orders. On the other hand, TETP at the time of release into the air is lower than that at the time of release to soil by about five orders, and TETP at the time of release into water also is lower by about five orders.

With regard to the other chemicals, AETP of trichloroethylene and dichloromethane is almost the same as or slightly lower than that of benzene in the case of any types of release. On the other hand, AETP of arsenic and cadmium tends to be higher by about 3 to 5 orders. Because arsenic and cadmium are heavy metals that are not volatile, PEC becomes higher. This is thought to have increased AETP.

With regard to TETP of the other chemicals, those of trichloroethylene and dichloromethane are almost the same as or slightly lower than that of benzene in the case of any types of release. On the other hand, those of arsenic and cadmium tend to be higher by about 3 to 5 orders in the case of both release into the air and release into soil, but are extremely low in the case of release into water. Even when trichloroethylene or dichloromethane is released into water, its concentration increases to some extent because of volatilization and soil deposition. However, when a heavy metal is released into water, because it does not volatilize, it is hard for its concentration to increase in soil. This seems to be the reason for the result that TETP is high in the case of release to the air and soil, while it is low in the case of release to water.

2.7.3 Assessment of damage from ecotoxicity

(1) Basic policy for calculation of damage factors

Table 2.7-2 shows the category endpoints of ecotoxicity and the object of protection about which the damage function is calculated under LIME. Because at present quantitative information necessary for assessment cannot be fully obtained concerning social assets and primary production, the object of protection about which the damage function was calculated under LIME was limited to biodiversity. In addition, Figure 2.7-3 shows the flowchart of calculating the damage function. As in the case of characterization, the geographical range covered by the damage assessment was limited to Japan.

(2) Damage indices of ecotoxicity

a Index related to the extinction risk of species: EINES

The damage function of ecotoxicity can be expressed by the Expected Increase in Number of Extinct Species (EINES) from an increase in released toxic chemicals in Japan. Equation 2.7-4 shows the total damage amount of all the species if a substance *i* is released into an environmental medium *ecomp*.

$$EINES_{i,ecomp} = \sum_{target species} EINES_{i,ecomp,species} = \sum_{target species} \Delta p_{i,ecomp,species} = \sum_{G} (N_G \times \Delta R_{i,ecomp,G}) \Delta E_{i,ecomp}$$
$$= \sum_{G} (N_G \times FF_{i,ecomp,fcomp} \times EF_{i,fcomp,G}) \Delta E_{i,ecomp} = DF_{i,ecomp} \times \Delta E_{i,ecomp} \quad (2.7\text{-}3)$$

Table 2.7-2: Category endpoints of ecotoxicity and the target of LIME

Object of protection	Category endpoint		Target of damage function calculation	
Social assets	Agricultural production	Decrease in yield of farm products, change in quality	×	Quantitative information is insufficient. However, the influence of intake of chemicals in farm products on health is assessed by the use of toxic chemicals.
	Forestry production	Decrease in forest increment, change in quality	×	Quantitative information is insufficient.
	Fishery production	Decrease in fishery resources through influence on fishes and shellfishes	×	Quantitative information is insufficient. However, the influence of intake of chemicals in fishes on health is assessed by the use of toxic chemicals.
Primary production	Terrestrial primary production	Influence on production of terrestrial plants	×	Quantitative information is insufficient.
	Aquatic primary production	Influence on production of phytoplankton	×	Quantitative information is insufficient.
Biodiversity	Terrestrial ecosystem	Increase in extinction risk through influence on individuals of terrestrial organisms	×	Quantitative information is insufficient.
	Aquatic ecosystem	Increase in extinction risk through influence on individuals of aquatic organisms	○	Algae, crustaceans, fishes, and other aquatic organisms in fresh water

In this equation, target species are all the species covered by the assessment of EINES; species is each group of organisms; ΔE is the amount of release of the toxic chemical i, and Δp is the increment in the extinction risk due to ΔE; G is the category of species (divided into some ranks according to the degree of extinction risk); ΔR is the increment in the extinction risk of a specific species due to a unit release amount of a chemical; FFi, ecomp, fcomp (fate factor) is the amount that reaches the environmental medium fcomp among the unit release amount of i; and EFi,fcomp,G (impact factor) is an increment in the extinction risk when a species belonging to the category G is exposed to a unit amount of the chemical i in the environmental medium fcomp. NG is the number of species included in the category G (such as the endangered class I) among a specific group of species (such as fishes or algae). The damage factor (DFi, ecomp) is the total increment in the extinction risk of each species due to a unit release amount of the chemical i. When this index is used, it is assumed that new extinction of a species is the same damage as an increment in the extinction risk of ten species by 10%, which is expressed as 1 EINES.

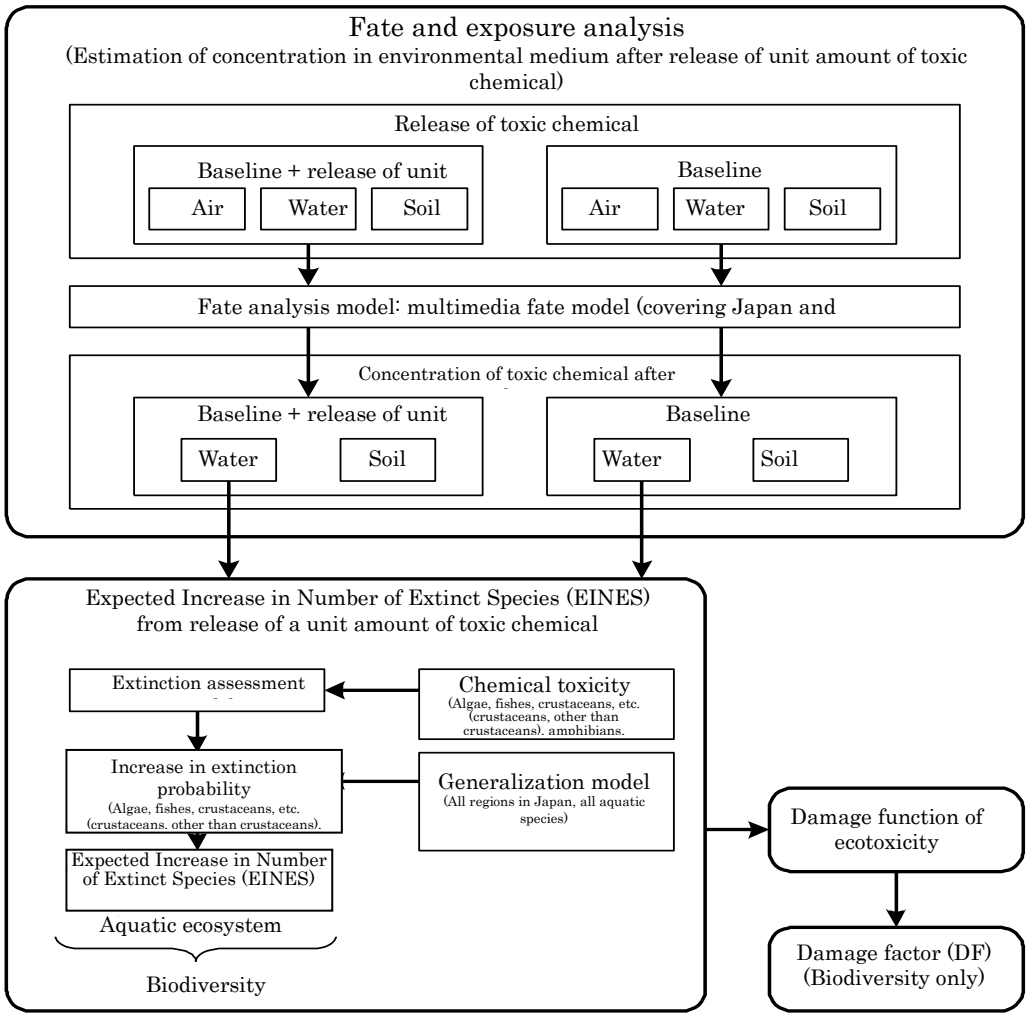

Figure 2.7-3: Flowchart of calculation of damage function of ecotoxicity

b Method of calculating EINES under LIME

As shown in Equation 2.7-4, when the damage factor is calculated, it is appropriate to find an increment in the extinction risk due to exposure to a chemical and accumulate the results. However, because the assessment of the extinction risks of all the existing species is difficult due to limited data, analysis was carried out under LIME for each rank of extinction risk of species written in the Red Data Book (RDB), taking into consideration the availability of models for the assessment of existing extinction risks and data used for the assessment. The assessment was carried out about the species representative of a category (such as the endangered class I). The result was multiplied by the number of specifies that belong to that category to find the assessment result for each category. This is based on the assumption that the assessment result of the extinction risk of the species representative of the category is the mean value of the assessment results of all the species that constitute the category.

c Species covered by assessment

The extinction risk was directly calculated concerning six groups of species: algae, crustaceans, etc. (crustaceans), fishes, shellfishes, crustaceans, etc. (other than crustaceans) and amphibians, all of which are aquatic organisms. They include algae, crustaceans, etc.

(crustaceans) and fishes, because algae, water fleas, and fishes are regarded as important for the field of ecotoxicity and have so far been covered by research studies and there are rich data on them. On the other hand, shellfishes, crustaceans, etc. (other than crustaceans) and amphibians, which had been grouped into "other aquatic organisms" for assessment under LIME 1, were separately covered by assessment under LIME 2 because data on them have been increasing in response to sharp improvement in the ecotoxicity database and because some chemicals have strong toxicity against them.

The characteristics of each aquatic species are described below. With regard to algae, crustaceans, etc. (crustaceans) and fishes in particular, reasons why they are regarded as important in the field of ecotoxicity are added.

1) Algae

Algae are primary producers that grow and increase through photosynthesis, taking nutrient salts. They exist at the bottom of the food chain. Therefore, if the impact of water pollution hinders the growth or propagation of algae, the whole food chain receives great impact. From the viewpoint of experiment, because green unicellular algae are characterized by a short lifecycle, it is possible to assess several-generation impact in a few days. Many assessment results have been attained so far.

2) Crustaceans, etc. (crustaceans)

Water fleas, which are zooplankton, eat plants and are regarded as important organisms that exist in the middle of the food chain and connect primary producers with higher organisms. In addition, because their lifecycle is comparatively short and the first immature flea is released seven to ten days after birth, it is easy to use them for reproductive testing. Water fleas belong to crustaceans and are frequently used for predicting the impact of water pollution on shrimps and other human food.

3) Fishes

Fishes are located at the highest position in the aquatic ecosystem. Because many toxicity test data were reported concerning various types of fishes, they are important as index organisms for environmental pollution.

4) Shellfishes

Mollusks are roughly divided into eight classes. Freshwater shellfishes consist of "gastropod (snail)" and "bivalve." The mollusks that have the largest number of types are gastropods. The total number of types of mollusks is said to be more than 100,000, and gastropods account for about 84% of the total number. In ECOTOX of the United States Environmental Protection Agency (USEPA), the toxicity data on shellfishes are the fourth largest among those on the assessment-covered six types, following those on algae, fishes, and crustaceans.

5) Crustaceans, etc. (other than crustaceans)

According to the Ministry of the Environment's RDB, "crustaceans, etc." consists of six phyla

of animals[*1], and land ones and freshwater ones (including brackish water ones in the case of decapoda) are covered by the assessment. Of them, four types of freshwater phyla – "Porifera," "Cnidaria," "Platyhelminthes," and "Annelida" – are covered by the assessment concerning "crustaceans, etc. (other than crustaceans) under LIME 2. Although, according to USEPA's ECOTOX, the number of toxic data on crustaceans, etc. (other than crustaceans) is the smallest among the covered six types, data on about 70 substances have been obtained among about 320 substances covered.

6) Amphibians

Amphibians usually spawn in freshwater, and larvae live in water. Although adults do not necessarily always live in water, because they cannot live apart from water, they may be influenced by a rise in the concentration of a toxic chemical in water. Therefore, they are covered by the assessment. According to USEPA's ECOTOX, the number of toxic data on amphibians is the fifth largest among the covered six types. However, some chemicals, such as arsenic and lead, have stronger toxicity against amphibians than against other aquatic species.

Damage from toxic chemicals was limited to aquatic organisms, mainly because the data used for the damage assessment were limited to water. Although exclusion of influence on terrestrial organisms may underestimate the assessment results, because the current ecological risk assessment mainly covers aquatic organisms, it can be thought to include the main effects of ecological risk.

Therefore, the development of a damage factor for the assessment of the impact of a toxic chemical on biodiversity requires the finding of N_G, $FF_{i,\,ecomp,\,fcomp}$, and $EF_{i,\,fcomp,\,G}$ in Equation 2.7-4. (3) to (5) below describe the method to calculate these parameters.

(3) Classification of assessment-covered species and the setting of the number of assessment-covered species (NG)

Information on the extinction of wild species has been collected and disclosed by the International Union for Conservation of Nature (IUCN) globally and by the Ministry of the Environment in Japan. The Ministry of the Environment has divided the wild species into the following classes according to probability of extinction: extinct, wild extinct, endangered class I, endangered class II, quasi-endangered, and lack of information. The endangered classes I and II have the following qualitative requirements. If a species meets either of 1) or 2), it falls under that class.

a Endangered class IA

1) The number of mature individuals is less than 50.
2) The probability of extinction is 50% or more in the next 10 years or in the following 3 generations, whichever is longer.

[*1] The six phyla are Porifera (sponge), Platyhelminthes (jellyfish), Cnidaria (triclad), Class Phylactolaemata (bryozoans), Hirudinea (leech, annelid) of Annelida, and Xiphosurida (horseshoe crab), Arachnida (Pseudoscorpiones, harvestman, tick, spider), Crustasia (wood louse, Gammaridea, shrimp, crab) and Diplopod (galleyworm) of Arthropoda.

b Endangered class IB

1) The number of mature individuals is less than 250.
2) The probability of extinction is 50% or more in the next 20 years or in the following 5 generations, whichever is longer.

c Endangered class II

1) The number of mature individuals is less than 1,000.
2) The probability of extinction is 50% or more in the next 100 years.

Table 2.7-3 shows the number of endangered species in Japan that the Ministry of the Environment calculated according to these standards and published. Of them, the number of species for each rank in Table 2.7-4, N_G, was used for the assessment of ecotoxicity.

(4) Calculation for fate analysis of a toxic chemical (FFi, ecomp, fcomp)

By the use of the multimedia fate model used for the characterization, a change in the concentration of each toxic chemical in water was estimated through analysis of the fate of the chemical in the environment when a unit amount of the chemical was released into the environment. Although the multimedia fate model was the same as the model used for the estimation of the amount of exposure to each toxic chemical to human beings, what was used as output was not the amount of exposure of human beings compared with the amount of release but a change in the concentration in water compared with the amount of release, and the index was treated as what indicated the amount of exposure of aquatic organisms. Table 2.7-5 shows the result of fate analysis of main chemicals.

Table 2.7-3: Number of endangered species in Japan

Class / Rank	Animals								Plants			Total
	Mammals	Amphibians	Reptiles	Birds	Brackish water/ freshwater fishes	Insects	Spiders, Crustaceans, etc.	Shellfishes	Vascular plants	Other than vascular plants		
										Algae	Others	
Extinct (EX)	4	0	0	13	3	2	0	25	20	5	32	102
Wild extinct (EW)	0	0	0	1	0	0	1	0	5	1	1	9
Endangered type IA (CR)	12	3	1	21	29	89	17	86	564	34	185	1621
Endangered type IB (EN)	20	10	9	32	29				480			
Endangered type II (VU)	16	18	11	39	18	82	39	165	621	6	103	1118
Quasi-endangered (NT)	16	17	14	18	12	161	40	201	145	24	21	669
Lack of information (DD)	9	5	1	17	5	87	39	71	52	0	71	357

The source is the Biodiversity Information System (http://www.biodic.go.jp/J-IBIS.html). This table is based on the results published through December 2006.

Table 2.7-4: Setting of the number of species (N_G) under LIME

		IA†1	IB†1	II	Sound species
Amphibia		1	9	11	65
Brackish water/freshwater fishes		29	29	18	300
Crustaceans, etc.	Crustaceans	4.5	4.5	17	100
	Other	3	3	14	100
Shellfishes		43	43	165	1000
Algae		17	17	6	5500
Total		97.5	105.5	231	7065
Number of species of "other aquatic organisms" †2		47	55	190	1165

†1 Only the total number of species belonging to the endangered type I is shown concerning algae, crustaceans, etc. and shellfishes. Because the breakdown of classes IA and IB is unknown, half of the total number is allotted between the classes.

†2 Number of species of "other aquatic organisms": the total number of species of amphibia, crustaceans, etc. (other than crustaceans), shellfishes, etc.

(5) Calculation of the effect factor of a toxic chemical (EFi, fcomp, G)

This section describes how to calculate $EF_{i,\ fcomp,\ G}$, the increment in the number of extinct specifies according to the amount of exposure to a toxic chemical. First, the basic data to be used for the model for assessment under LIME are introduced in "a Collection of data on the toxicity of chemicals and the setting of representative values." Some models for the calculation of extinction risk due to exposure to a chemical have been already developed in the field of conservation biology, etc. Main models are explained in "b Existing models for the calculation of extinction risk."

The sensitivity to an increase in the extinction risk due to exposure to a chemical differs among species. Under LIME, different models and parameters were used according to the classes related to the extinction of species (such as the endangered type IA).

Table 2.7-5: Results of fate analysis of main chemicals

Chemical	Concentration in water [(g/m3)/ (t/yr)]					
	Release to the air		Release to water		Release to soil	
	Surface water	Soil	Surface water	Soil	Surface water	Soil
Benzene	1.38E-12	4.00E-13	1.70E-08	1.64E-13	2.95E-11	4.53E-09
Arsenic	9.68E-07	2.97E-05	2.65E-06	2.56E-20	1.81E-06	1.09E-04
Trichloroethylene	4.52E-12	1.93E-12	4.79E-08	9.69E-13	1.18E-10	1.79E-08
Dichloromethane	4.45E-11	6.26E-12	9.92E-08	3.75E-12	3.01E-09	9.18E-08
Cadmium	5.88E-07	2.96E-06	1.90E-06	0.00E+00	9.08E-07	1.08E-05

"c Assessment of *EF* for sound species" and "d Assessment of *EF* for the endangered type II", "e Assessment of *EF* for the endangered type IB" and "f Assessment of *EF* for the endangered type IA" show how to assess *EF* for the respective classes and what parameters were used. Moreover, they also show assessment results.
The damage factor can be gained by applying the results of "c" to "f" to Equation 2.7-4.

a Collection of data on the toxicity of chemicals and the setting of representative values

Under LIME, toxic data from USEPA's ECOTOX were used to clarify the toxicity of chemicals against groups of species (algae, crustaceans, etc. (crustaceans), fishes, crustaceans, etc. (other than crustaceans), shellfishes and amphibians). Data on effective concentration 50% (EC_{50}; the concentration of a chemical or the like that makes the growth increment of the target species 50% in the experiment period) [μg/L] were extracted for algae, while data on lethal concentration 50% (LC_{50}: the concentration of a chemical or the like that makes the death rate of the target species 50% in the experiment period) [μg/L] were extracted for animal groups.

To arrange data, if several sets of data on the toxicity of a chemical exist for the same species, the geometric average was calculated. Next, for each chemical, the geometric average was calculated concerning all data for the same group of organisms (algae, crustaceans, etc. (crustaceans), fishes, crustaceans, etc. (other than crustaceans), shellfishes, and amphibians) and the result was regarded as the evaluated toxicity value of the group. If toxicity data were not obtained concerning a group, the geometric average of the toxicity values of species about which toxicity data were obtained was used. Table 2.7-6 shows the representative values of EC_{50} and LC_{50} of main chemicals.

With regard to species whose extinction risk is high (such as endangered species), toxicity data usually do not exist, due to their rareness. Therefore, evaluated toxicity data obtained from experiment data on the toxicity of sound species were used for such species.

b Existing models for the calculation of extinction risk

Extinction means that the population of a species becomes zero. Therefore, a model for assessing extinction risk needs to express changes in the population. 1) to 3) below explain models for expressing changes in the population, and 4) below compares these models from the viewpoint of application to LIME.

Table 2.7-6: Example of data on ecotoxicity of main chemicals

Chemical	Algae EC_{50} (μg/L)	Crustaceans, etc. (crustaceans) LC_{50} (μg/L)	Fishes LC_{50} (μg/L)	Shellfishes LC_{50} (μg/L)	Crustaceans, etc. (other than crustaceans) LC_{50} (μg/L)	Amphibians LC_{50} (μg/L)
Benzene	7.06E+04	1.38E+03	3.87E+04	3.87E+01	3.24E+04	2.48E+05
Zinc	1.11E+03	5.74E+02	4.67E+03	1.66E+03	2.65E+03	1.00E+04
Trichloroethylene	2.98E+05	4.07E+04	6.56E+04	5.60E+04	7.50E+04	4.58E+04
Dichloromethane	3.15E+05	3.55E+03	4.00E+05	3.14E+04	2.38E+04	2.88E+04
Cadmium	1.68E+02	9.00E+01	2.18E+03	1.97E+03	2.50E+03	4.00E+01

In the case of benzene, for example, because the value of LC_{50} of mollusks (shellfishes) is lower than that of other species by nearly 2 orders of magnitude, benzene is highly toxic especially against shellfishes. In this way, LIME 2 also evaluates ecological effects, which could not be accurately evaluated from the average value for algae, crustaceans, and fishes.

1) Canonical model

Iwasa et al. (1997) developed the "canonical model," a basic equation that expresses changes in the population and includes essential elements, as an analysis model for changes in the population. The canonical model takes into consideration the following three elements (the following A, B, and C correspond to A, B, and C in Equation 2.7-5):

A: Multiplication through propagation (see Column 2.7-1)

B: Change in the population according to environmental stochasticity (see Column 2.7-1)
A change in the population according to whether the environmental conditions around organisms, such as temperature and precipitation, are favorable or unfavorable; this change occurs independently from a change in the population according to demographic stochasticity.

C: Change in the population according to demographic stochasticity
This is a change in the gender ratio, the average number of births, or the survival rate due to a limited population. This includes cases where only male individuals exist by chance or where children continue to die by chance. This change occurs independently from a change in the population according to environmental stochasticity. If the population is large, this change has smaller impact than the other elements. However, if the population is small, its impact is not ignorable.

This model can be expressed by the following equation:

$$\frac{dN}{dt} = \underbrace{r(N)N\left(1-\frac{N}{K}\right)}_{\text{A}} + \underbrace{\sigma_e Z_e(t) \circ N}_{\text{B}} + \underbrace{Z_d(t) \bullet \sqrt{N}}_{\text{C}} \qquad (2.7\text{-}4)$$

$N(t)$: Population in the generation t

K: Carrying capacity

$r(N)$: Increase rate of an individual

σ_e: Degree of environmental change

$Z_e(t)$: Random number that expresses environmental change

$Z_d(t)$: Random number that expresses demographic change

$\circ$: Stratonovich integration

$\bullet$: Ito integration

2) Tanaka's calculation method

This model is applicable to cases where the population changes nearly steadily, such as with sound species. Of the elements that the canonical model takes into consideration, this model takes into consideration A and B.

It is all right to assume that sound species are in a steady state, because the population is sufficiently large. In this case, the extinction probability does not change according to the point of time t.

If the probability that a species becomes extinct within a year is 10^{-6}, the cumulative extinction probability is as shown in Figure 2.7-4.

Lande (1993) pointed out that, when the population is steady, the average extinction time gradually becomes close to the exponential function of carrying capacity. Moreover, Lande (1998) found a scaling law of the influence of environmental stochasticity on the average extinction time T and formulated the following equation from the law (see Column 2.7-2):

$$T = c \cdot N^{2\frac{r}{v}-1} \tag{2.7-5}$$

N is the initial population. r and v are the average and dispersion of the intrinsic natural growth rate, respectively. c is a constant. Equation 2.7-6 can be transformed as follows, replacing N with the carrying capacity K:

$$\log T = \log c + \left(2\frac{r}{v} - 1\right)\log K \tag{2.7-6}$$

This shows that, because of exposure to a toxic chemical, if r or K decreases or v increases, T decreases.

If comparison is made in the degree of sensitivity of extinction probability to a change in each element, r and v directly influence log T. However, because K has influence in the form of log K, the influence of a change in the carrying capacity on the extinction time is relatively small. Therefore, under LIME, the influence of a change in the carrying capacity on the extinction time was ignored, and the influence of a change in the intrinsic natural growth rate and the environmental variability were taken into consideration. Below, explanations are given to "the relation between the amount of exposure to a chemical and the intrinsic natural growth rate," "the relation between the amount of exposure to a chemical and changes in the increase rate due to environmental stochasticity" and "estimation of extinction probability."

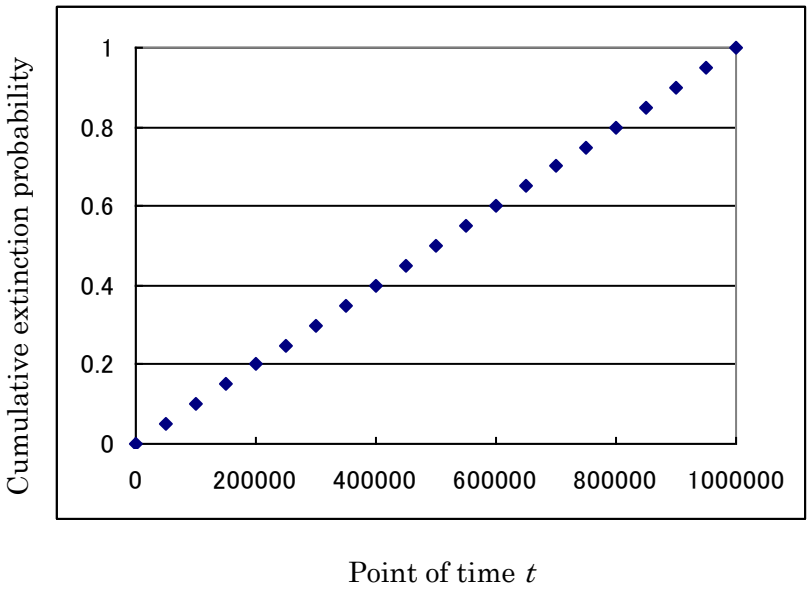

Figure 2.7-4: The cumulative extinction probability until the point of time *t* on the assumption that the population changes steadily and the extinction probability within a year is 10^{-6}

Column 2.7-1

Meanings of carrying capacity, changes in the increase rate according to environmental stochasticity, and the intrinsic natural growth rate

Under LIME, the extinction risk of a species is evaluated by the use of carrying capacity, environmental changes, and the intrinsic natural growth rate as parameters. This column roughly explains these parameters (for details, see Washitani et al. (1996)).
When the population N is changing at the average rate of change r per individual and per time t, the change can be expressed as follows:

$$\frac{dN}{dt} = rN$$

The change rate r is the difference between the birth rate and the death rate. If $r > 0$, the population increases. If $r < 0$, the population decreases. If $r > 0$, the population increases geometrically with the passage of time. In reality, however, the number remains at a certain level. This is because when the population increases, the effect of restraining the increase (which is called the "density effect") works. For example, the living space or the amount of food is limited. This relation can be expressed by the following equation:

$$\frac{dN}{dt} = rN - \frac{r}{K}N^2$$

In this equation, the second item of the right side corresponds to the density effect. In the case of this relation, when the population reaches K, it becomes stable (Figure 2.7-A). This K is called "carrying capacity."

In addition, although r differs according to species and group, it changes also according to various factors. If a change in r is taken into account, the equation above can be changed to the equation below.

$$\frac{dN}{dt} = \{r_0 + x(t)\}N - \frac{r}{K}N^2$$

r_0 is a characteristic of the species and refers to the change rate if there is no influence on changes in the change rate, which is called the "intrinsic natural growth rate." $x(t)$ is a positive or negative function that temporarily and stochastically changes at the average value of 0. If the population is large, the species' extinction risk is low even if the increase rate ($r_0 + x(t)$) changes more or less. However, if the population is small, the species may become extinct if the increase rate continues to be negative for several years. $x(t)$ incorporates demographic stochasticity, catastroph, genetic stochasticity, etc. It also incorporates environmental stochasticity. This refers to a change in the increase rate of a group of individuals due to an environmental factor, such as temperature or precipitation.

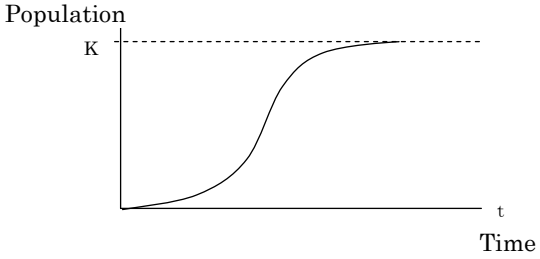

Figure 2.7-A: Changes in the population if the density effect is taken into account

The population increases with the passage of time. However, as the number is nearer to the carrying capacity K, the density effect becomes stronger, and changes in the number remain under the level of K.

Column 2.7-2

Lande's scaling law
Lande's scaling law can be expressed as Equation 2.7-6 under the condition that the population is sufficiently large and is in a steady state. This indicates that, as the intrinsic natural growth rate and the population become higher and the dispersion of the increase rate due to an environmental change becomes smaller, the extinction probability becomes smaller. If the population is sufficiently large and is in a steady state, the average extinction time (temporary scale) can be approximately expressed by the exponential function of the carrying capacity (environmental scale). Therefore, this equation is not applicable to unsteady cases, such as a continuous decrease in the population. Because of this, LIME limits the application of this equation to sound species.

[Relation between the amount of exposure to chemicals and the intrinsic natural growth rate]

Tanaka (Tanaka et al. 2000) carried out statistical fitting of experimental data on several types of chemicals and found that there is the causal relationship between the amount of exposure to chemicals and the intrinsic natural growth rate, as shown in Equation 2.7-8 (Figure 2.7-5). α is defined as the concentration at which the intrinsic natural growth rate is 0.

$$r_x = r_0\{1-(\frac{x}{\alpha})^{\beta}\} \tag{2.7-7}$$

$$\Delta r = r_x - r_0 = -r_0(\frac{x}{\alpha})^{\beta} \tag{2.7-8}$$

$$\beta = 1.82$$

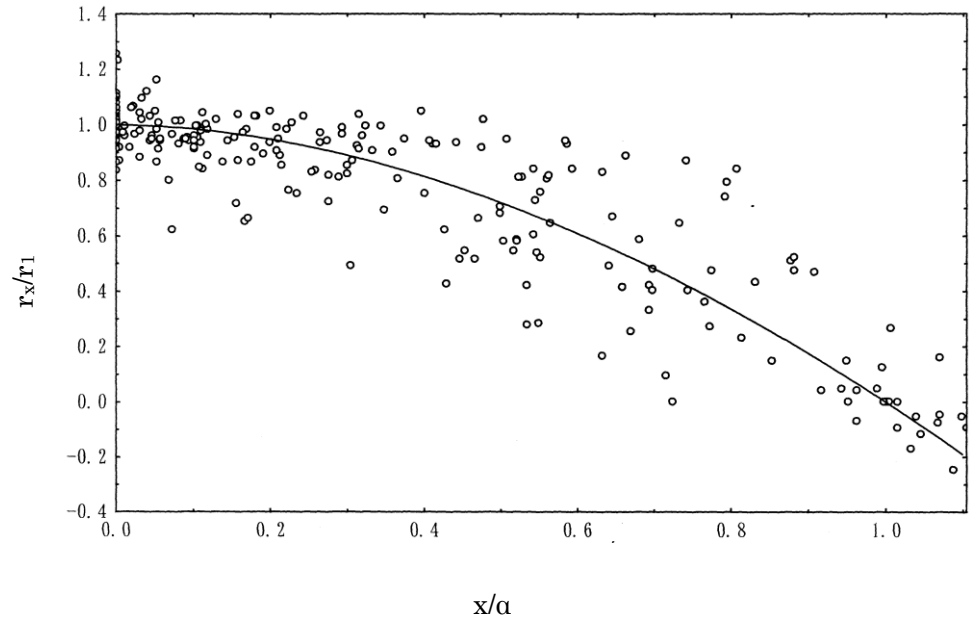

Figure 2.7-5: Relation between the exposure concentration and the intrinsic natural growth rate of species(Tanaka et al. 2001b)

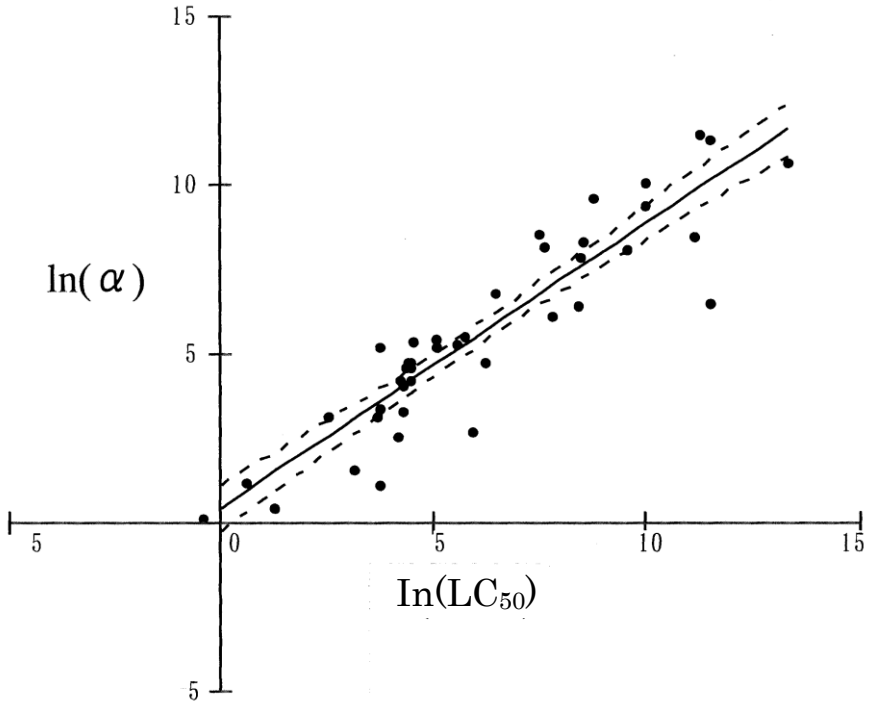

Figure 2.7-6: Relation between the acute toxicity value and the value of α (Tanaka et al. 2001b)

In this equation, r_0 is the initial value of the intrinsic natural growth rate, $r(x)$ is the value at the time of exposure, and x is the exposure concentration. β is the parameter that expresses the slope when response increases as the concentration increases. α is the parameter that determines the density at which toxicity emerges, and is peculiar to each substance and species. Although α can be calculated by the equation below, this equation also has been obtained from statistical fitting of experimental data. Figure 2.7-6 shows the result of fitting of experimental data. In this way, α is highly correlative to LC_{50}.

$$\ln(\alpha) = \ln(LC_{50}) \cdot 0.843 + \ln(1.562) \qquad (2.7\text{-}9)$$

[Relation between the amount of exposure to chemicals and a change in the increase rate due to environmental stochasticity]

This section describes the relation between exposure to chemicals and a change in the increase rate due to environmental stochasticity. Changes in the amount of exposure to chemicals in the general environment were estimated from the items of "Outline of Bottom Sediment Monitoring Results in FY1999" in "Chemicals and the Environment (FY2010 Edition)" published by the Ministry of the Environment. As a result, it was found that the variation coefficient for changes in the amount of exposure is 0.58 on the assumption that the variation coefficient is constant. Because the variation coefficient is assumed to be constant, if the amount of exposure to chemicals (representative value) increases, a change in the amount of exposure (standard deviation) increases accordingly (Figure 2.7-7). By applying this to Equation 2.7-8, the representative value of the intrinsic natural growth rate and the amount of change as a result of a change in the amount of exposure to a chemical were estimated.

$$v(x) = \sigma_{r0}^2 + \sigma_r(x)^2 = v_0 + \sigma_r(x)^2 \quad (2.7\text{-}10)$$

$$\sigma_r(x) = \left| \frac{\partial r_x}{\partial x} \right| \sigma_x = r_0 \beta \left(\frac{x}{\alpha}\right)^{\beta-1} \frac{1}{\alpha} \times \gamma\, x = r_0 \beta \gamma \left(\frac{x}{\alpha}\right)^{\beta} \quad (2.7\text{-}11)$$

$v(x)$: Dispersion of the increase rate due to environmental stochasticity if the amount of exposure is x

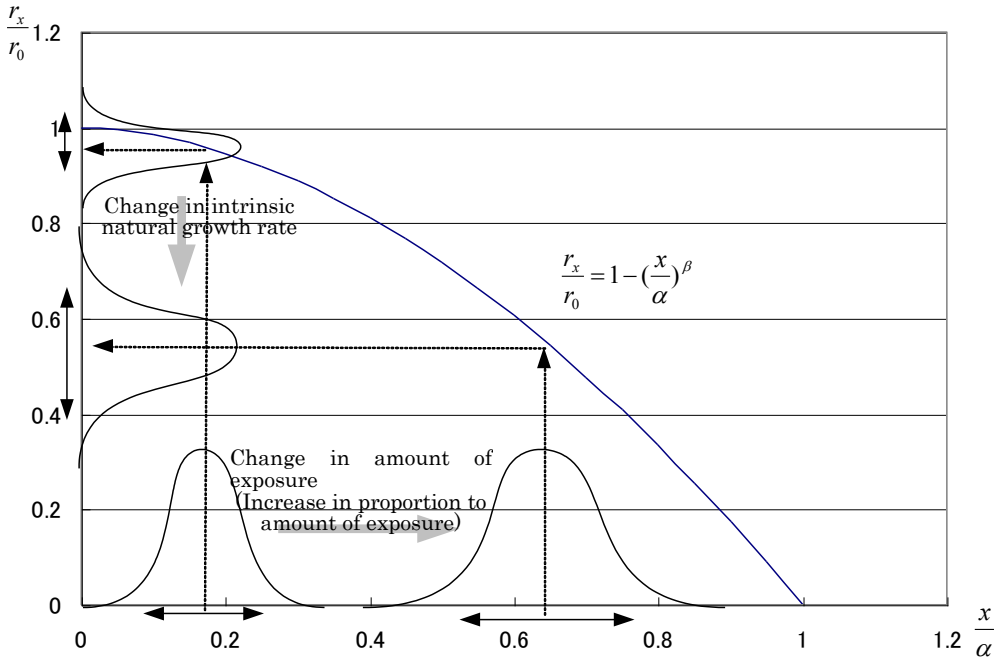

Figure 2.7-7: Changes in the amount of exposure to chemicals and the intrinsic natural growth rate due to environmental stochasticity

If the amount of exposure to chemicals increases, the amount of change in the representative value of the amount of exposure becomes larger. In proportion to this, the amount of change in the intrinsic natural growth rate becomes larger.

$\sigma_r(x)$: Standard deviation of a change in the intrinsic natural growth rate (if the amount of exposure is x)

σ_{r0}: Standard deviation of a change in the intrinsic natural growth rate (if the amount of exposure is 0)

v_0: Dispersion of the increase rate due to environmental stochasticity if the amount of exposure is 0

γ: *Variation coefficient = 0.58*

Figure 2.7-8 shows differences in the extinction probability according to the variation coefficient if environmental variation is taken into consideration in Tanaka's calculation method. Parameters are set in the same way as in the case of the endangered type II crustaceans described below (Table 2.7-9). The initial value of the intrinsic natural growth rate r_0 is 1.5, the initial value of environmental dispersion v_0 is 3.0, the carrying capacity K is 1000, and the extinction probability when the exposure concentration is α is 1.0. This figure shows that, given environmental variability, the extinction probability increases even if the amount of exposure is the same.

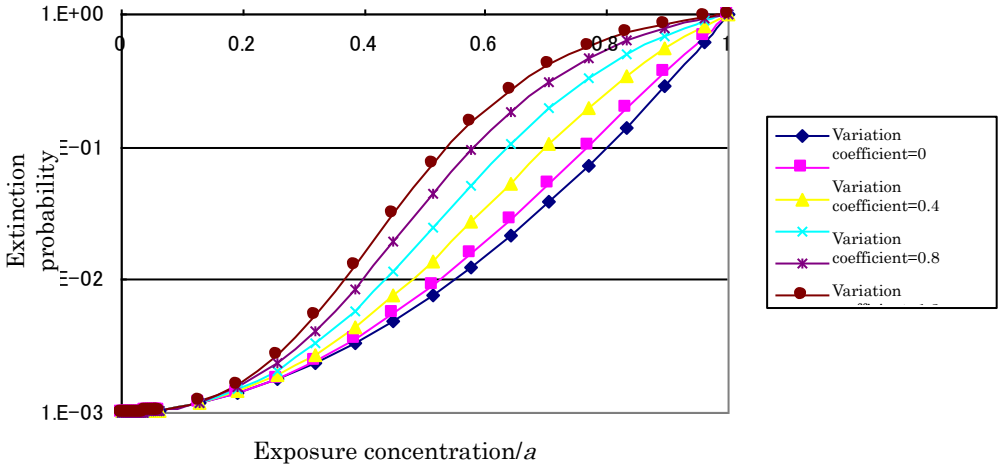

Figure 2.7-8: Change in the relation between exposure concentration and the extinction probability when environmental stochasticity is taken into account (based on Tanaka's calculation method)
If environmental stochasticity is introduced, the extinction probability increases even if the amount of exposure is the same.

[Estimation of extinction probability]

The extinction probability p is defined as the reciprocal of the extinction time T. For example, if T is 10 (years), p is 0.1, which means that the probability of extinction during the next one year is 0.1.

$$p = \frac{1}{T} \tag{2.7-12}$$

The following relation can be obtained from the scaling law (Equation 2.7-6):

$$p = c^{-1} \cdot N^{-\left(2\frac{r}{v}-1\right)} \tag{2.7-13}$$

3) Lande-Orzack model

Lande and Orzack found an analytical solution irrespective of the influence of demographic fluctuation when the population continues to decrease and the density effect can be ignored. The following is the analytical solution:

$$G(t \mid n_0) = \Phi\left[\frac{-n_0 - rt}{\sigma\sqrt{t}}\right] + \exp\left\{-\frac{2n_0 r}{\sigma^2}\right\}\left(1 - \Phi\left[\frac{n_0 - rt}{\sigma\sqrt{t}}\right]\right) \tag{2.7-14}$$

$$\Phi[y] = (2\pi)^{-1/2} \int_{-\infty}^{y} \exp\{-z^2/2\} dz \tag{2.7-15}$$

The variables have the following meanings:

t:	time (unit: year)
n_0:	natural logarithm of the population N_0 when t is 0
$G(t\|n_0)$:	probability of extinction within t years when the natural logarithm of the population is n_0
r:	average of intrinsic natural growth rate
σ^2:	dispersion of intrinsic natural growth rate

Because there are no referable quantitative data concerning the relation between the intrinsic natural growth rate *r* and the amount of exposure *x*, the possibility of extinction within a year is regarded as 1 when *x* is $LC_{50,}$ on the assumption that the relation between the two is linear (Figure 2.7-9).

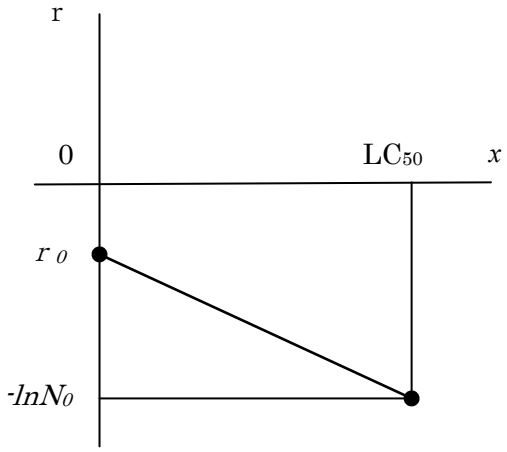

Figure 2.7-9: Assumed relation between *r* and *x* (linear interpolation function)

The following is the equation obtained from this assumption:

$$r(x) = r_0 + \frac{-\ln N_0 - r_0}{\mathrm{LC}_{50}} x \qquad (2.7\text{-}16)$$

As shown below, the Lande-Orzack model also takes into consideration changes in the increase rate due to the amount of exposure to chemicals and environmental stochasticity.

$$v(x) = \sigma_{r0}^2 + \sigma_r(x)^2 = v_0 + \sigma_r(x)^2 \qquad (2.7\text{-}17)$$

$$\sigma_r(x) = \left|\frac{\partial r(x)}{\partial x}\right| \sigma_x = \left|\frac{-\ln N_0 - r_0}{\mathrm{LC}_{50}}\right| \times \gamma_x \qquad (2.7\text{-}18)$$

$v(x)$: Dispersion of the increase rate due to environmental stochasticity when the amount of exposure is x

$\sigma_r(x)$: Variable standard deviation of the intrinsic natural growth rate (if the amount of exposure is x)

σ_{r0}: Variable standard deviation of the intrinsic natural growth rate (if the amount of exposure is 0)

σ_r: Dispersion of the increase rate due to environmental stochasticity when the amount of exposure is 0

γ: Variation coefficient = 0.58

4) Comparison of characteristics of existing models

Although both Tanaka's calculation method and the Lande-Orzack model are based on the canonical model, they add some limited aspects to consider, in order to clarify the relation between extinction probability and its factors. This is because the canonical model cannot be solved analytically. Table 2.7-7 compare the three models. Under LIME, *EF* was

calculated by the use of Tanaka's calculation method for sound species and the endangered type II, the population of which is thought to be almost constant, and by the use of the Lande-Orzack model for the endangered classes IA and IB, the population of which is thought to be decreasing.

Table 2.7-7: Comparison of three models

	Canonical model	Tanaka's calculation method	Lande-Orzack model
Consideration of propagation potential	○	○	○
Consideration of environmental stochasticity	○	○	○
Consideration of demographic stochasticity	○	×	×
Consideration of carrying capacity (density effect)	○	○	×
Treatment of non-steady state	○	×	○
Expressed in the form of function of extinction probability?	×	○	○
Treatment under LIME (Target group of species)	Not used	Used for assessment of sound species and endangered type II	Used for assessment of endangered classes IA and IB

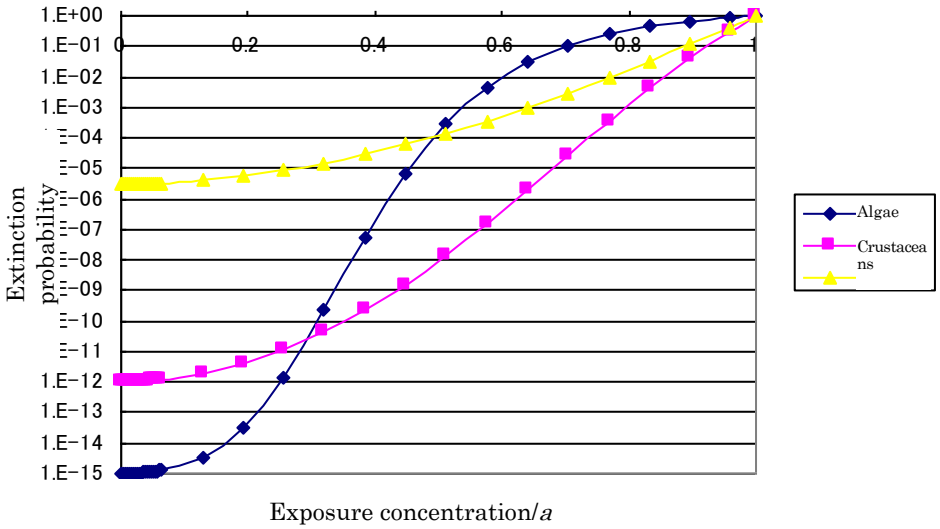

Figure 2.7-10: Relation between exposure concentration and extinction probability (sound species)
Sound species: when the variation coefficient of the intrinsic natural growth rate by environmental stochasticity is 0.58

c Assessment of EF for sound species

The parameters used for the calculation of the extinction risk of sound species were set as follows:

1) Proportionality factor (indicated as c)

The proportionality factor was set so that the extinction probability would become 1 when the exposure concentration is α (α is defined as the degree of concentration at which the intrinsic natural growth rate becomes 0 under LIME).

2) Intrinsic natural growth rate (the initial value is indicated as r0)

The initial value r_0 of the intrinsic natural growth rate of algae, crustaceans, and fishes $r(x)$ was set based on exiting literature (Tanaka et al. 2000, Tanaka et al. 2001a, Tanaka et al. 2001b, Tanaka 2000) and results of interviews with experts (Table 2.7-8). The geometric average value for crustaceans and fishes was used for the other species. The relation expressed by Equation 2.7-8 was used for changes in the amount of exposure x.

3) Dispersion of the increase rate due to environmental stochasticity (the initial value is indicated as ν0)

The initial value v_0 of the dispersion of the increase rate due to environmental stochasticity concerning algae, crustaceans, and fishes $v(x)$ was set based on literature (Tanaka 2000, Weinberger et al. 1982, Hakoyama et al. 2000) and experts' advice (Table 2.7-8). The geometric average value for crustaceans and fishes was used for the other species. The relations expressed by Equations 2.7-11 and 2.7-12 were used for a changes due to the amount of exposure x.

Table 2.7-8: List of parameters used for assessment of sound species

	Algae	Crustaceans, etc. (crustaceans)	Fishes	Shellfishes	Crustaceans, etc. (other than crustaceans)	Amphibians
Initial value of intrinsic natural growth rate r_0	41.2	1.5	0.171	0.506		
Initial value of dispersion of increase rate due to environmental change v_0	82.4	3.0	0.342	1.01		
Carrying capacity K	10^{15}	10^{12}	10^{6}	10^{9}		

4) Carrying capacity (indicated as K)

The value obtained from outdoor data on water fleas in Lake Kasumigaura was used for crustaceans, and the carrying capacities for algae and fishes were calculated based on the value and by reference to the ratio specified in Lande (1993) (Table 2.7-8). The geometric average value for crustaceans and fishes was used for the other species.Figure 2.7-10 shows the extinction probability of each species calculated by application of these parameters to Tanaka's calculation method.This result can be interpreted as the D-R relationship for risk assessment, and makes it possible to calculate *EF*. That is, *EF* is the slope of the curve of extinction probability for the current exposure concentration.
In the case of sound species, because the population is very large, if the amount of exposure

to a chemical is almost zero, the extinction probability becomes very small. In addition, if the amount of exposure is less than α, an increase in the extinction probability due to exposure to the chemical becomes relatively small (for example, even if the exposure concentration increases from zero to 0.2 α, the extinction risk of algae increases by only about 1.0×10^{-14}).

d Assessment of EF for endangered type II

The following parameters were used for the assessment of the extinction risk of endangered type II.

1) Proportionality factor (indicated as c)

The proportionality factor was set so that the extinction probability would become 1 when the exposure concentration disperses at an increase rate with the probability of α.

2) Intrinsic natural growth rate (the initial value is indicated as r0)

The initial value r_0 of the intrinsic natural growth rate of algae, crustaceans, and fishes $r(x)$ was set based on existing literature (Tanaka et al. 2000, Tanaka et al. 2001a, Tanaka et al. 2001b, Tanaka 2000) and results of interviews with experts (Table 2.7-9). The geometric average value for crustaceans and fishes was used for the other species. The relation expressed by Equation 2.7-8 was used for changes in the amount of exposure.

Table 2.7-9: List of parameters used for assessment of endangered type II

	Algae	Crustaceans, etc. (crustaceans)	Fishes	Shellfishes	Crustaceans, etc. (other than crustaceans)	Amphibians
Initial value of intrinsic natural growth rate r_0	41.2	1.5	0.171	0.506		
Initial value of dispersion of increase rate due to environmental change v_0	82.4	3.0	0.342	1.01		
Carrying capacity K	1000	1000	1000	1000		

3) Dispersion of the increase rate due to environmental stochasticity (the initial value is indicated as ν0)

The initial value v_0 of the dispersion of the increase rate due to environmental stochasticity concerning algae, crustaceans, and fishes $v(x)$ was set based on literature (Tanaka at al. 2000, Tanaka et al. 2001a, Tanaka et al. 2001b) and results of interviews with experts (Table 2.7-9). The geometric average value for crustaceans and fishes was used for the other species. The relations expressed by Equations 2.7-11 and 2.7-12 were used for changes according to the amount of exposure.

4) Carrying capacity (indicated as K)

K was set at 1000 by the use of the threshold value of the population condition (mature population should be less than 1000) among the quantitative requirements specified in the

Ministry of the Environment's category definitions (Table 2.7-9). Figure 2.7-11 shows the extinction probability of each species calculated by application of these parameters to Tanaka's calculation method. In this case, because the population is smaller than that for sound species, the extinction risk when the exposure concentration is 0 is higher than that for sound species. In addition, when the exposure concentration is well smaller than α, the increment in the extinction probability due to an increase in the amount of exposure is larger than that for sound species. The slope of the extinction probability at the current exposure concentration is *EF*.

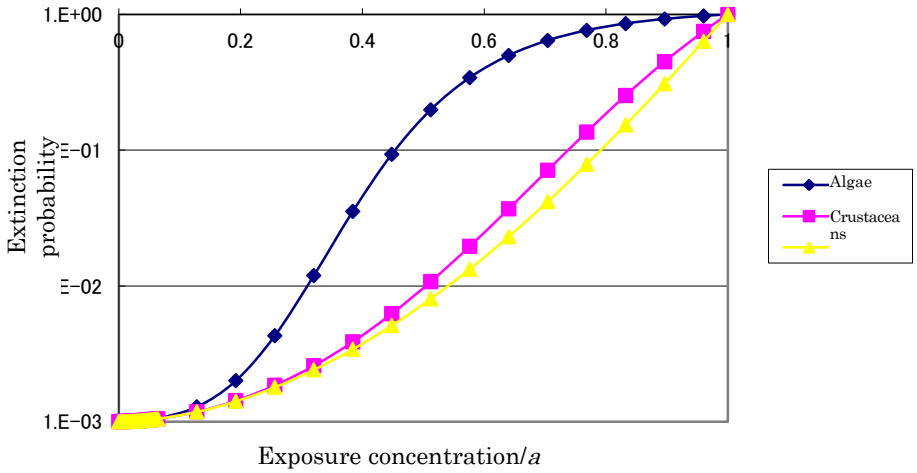

Figure 2.7-11: Relation between exposure concentration and extinction probability (endangered type II)
Endangered type II: when the variation coefficient of environmental changes is 0.58

e Assessment of EF for the endangered type IB

The following parameters were used for the assessment of the extinction risk of the endangered type IB.

1) Intrinsic natural growth rate (the initial value is indicated as r0)

In the state of no exposure (the initial state), the initial value of the intrinsic natural growth rate $r(x)$ was set by the use of the threshold value of the decrease rate condition (the population will decrease by more than 20% in the next five years) among the qualitative requirements specified in the Ministry of the Environment's category definitions (r_0 = -0.045 (Table 2.7-10)). The relation expressed by Equation 2.7-17 was used for changes according to the amount of exposure (Table 2.7-10).

2) Dispersion of the increase rate due to environmental stochasticity (the initial value is indicated as v0)

There are no referential quantitative data. The initial value v_0 of the dispersion of the increase rate due to environmental stochasticity $v(x)$ was set so as to meet the threshold value of the extinction probability condition (the probability that the species will become extinct within 20 years is less than 20%) among the quantitative requirements specified in the Ministry of the Environment's category definitions (G (20|n_0) = 0.2; that is, v_0 = 0.72 (Table 2.7-10)). The relations expressed by Equations 2.7-18 and 2.7-19 were used for changes according to the amount of exposure.

Table 2.7-10: List of parameters used for assessment of the endangered type IB

	Algae	Crustaceans, etc. (crustaceans)	Fishes	Shellfishes	Crustaceans, etc. (other than crustaceans)	Amphibians
Initial value of intrinsic natural growth rate r_0	-0.045					
Initial value of dispersion of increase rate due to environmental stochasticity σ^2_0	0.72					
Carrying capacity K	250					

3) Carrying capacity (indicated as K)

The threshold value of the population condition (mature population should be less than 250) among the quantitative requirements specified in the Ministry of the Environment's category definitions was used (K = 250 (Table 2.7-10)).

4) Period for calculation of extinction probability (indicated as t)

The period was temporarily set at 50 years (which will finally be converted to risk per year). These parameters were assigned in the Lande-Orzack model to calculate the extinction probability. Figures 2.7-12 and 2.7-13 show the time-series cumulative extinction probability if the amount of extinction is 0, and the relation between the exposure concentration and the extinction probability when 50 years pass.
Because the increase rate is negative and the initial population is small in the case of the species belonging to endangered type IB, the extinction probability is high even if there is no exposure to a chemical (the probability that extinction occurs within 50 years is about 50%). An increase in the amount of exposure to chemicals caused an increase in the extinction risk, with the result that the extinction risk has become almost 100% due to about 10% exposure to LC_{50}. In addition, difference in extinction probability according to whether there is consideration for environmental changes is small. After this result was converted into the extinction risk per year to find the slope at the current exposure concentration, *EF* was calculated.

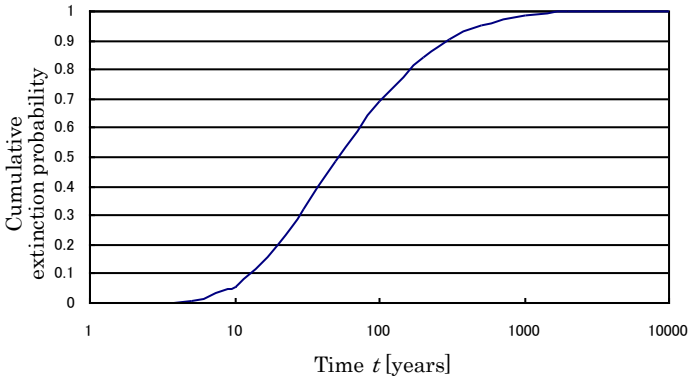

Figure 2.7-12: Endangered type IB's cumulative extinction probability until the point of time *t* (if there is no exposure to a chemical)

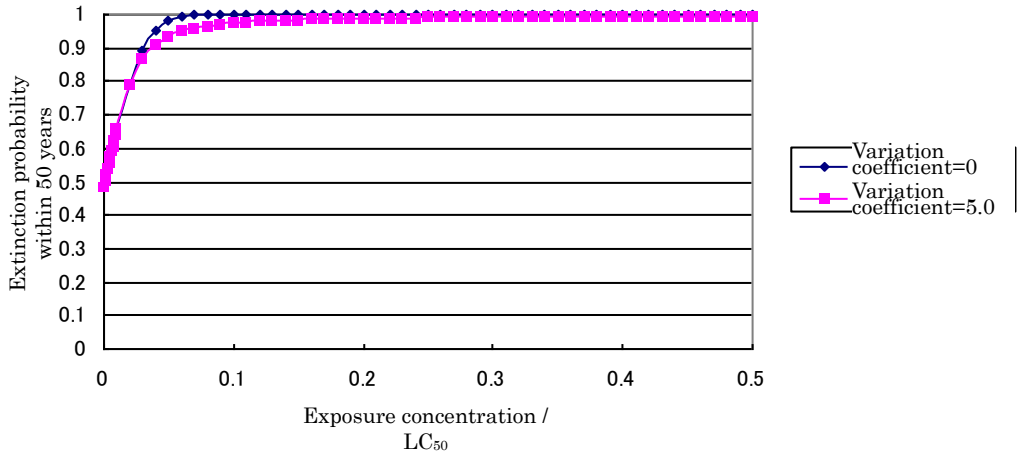

Figure 2.7-13: Relation of exposure concentration and extinction probability concerning endangered type IB(when 50 years pass)

f Assessment of EF for endangered type IA

The following parameters were used for the assessment of the extinction risk of endangered type IA.

1) Intrinsic natural growth rate (the initial value is indicated as r0)

In the state of no exposure (the initial state), the initial value of the intrinsic natural growth rate $r(x)$ was set by the use of the threshold value of the decrease rate condition (the population will decrease by more than 50% in the next 10 years) among the qualitative requirements specified in the Ministry of the Environment's category definitions (r_0 = -0.096 (Table 2.7-11)). The relation expressed by Equation 2.7-17 was used for changes according to the amount of exposure.

2) Dispersion of the increase rate due to environmental stochasticity (the initial value is indicated as v0)

There are no referential quantitative data. The initial value v_0 of the dispersion of the increase rate due to environmental stochasticity $v(x)$ was set so as to meet the threshold value of the extinction probability condition (the probability that the species will become extinct within 3 years is less than 25%) among the quantitative requirements specified in the Ministry of the Environment's category definitions ($G(3|n_0) = 0.25$; that is, $v_0 = 2.5$ (Table 2.7-11)). The relations expressed by Equations 2.7-18 and 2.7-19 were used for changes according to the amount of exposure.

3) Carrying capacity (indicated as K)

The threshold value of the population condition (mature population should be less than 50) among the quantitative requirements specified in the Ministry of the Environment's category definitions was used (K = 50 (Table 2.7-11)).

4) Period for calculation of extinction probability (indicated as t)
The period was temporarily set at 50 years (which, however, will finally be converted to risk per year).

Table 2.7-11: List of parameters used for assessment of endangered type IA

	Algae	Crustaceans, etc. (crustaceans)	Fishes	Shellfishes	Crustaceans, etc. (other than crustaceans)	Amphibians
Initial value of intrinsic natural growth rate r_0	-0.096					
Initial value of dispersion of increase rate due to environmental stochasticity σ^2_0	2.5					
Carrying capacity K	50					

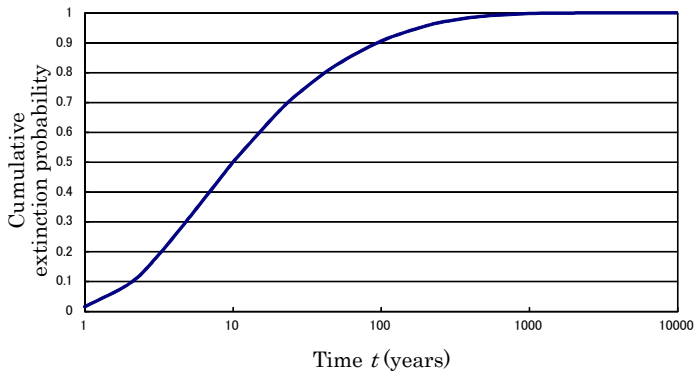

Figure 2.7-14: The endangered type IA's cumulative extinction probability until the point of time *t* (if there is no exposure to a chemical)

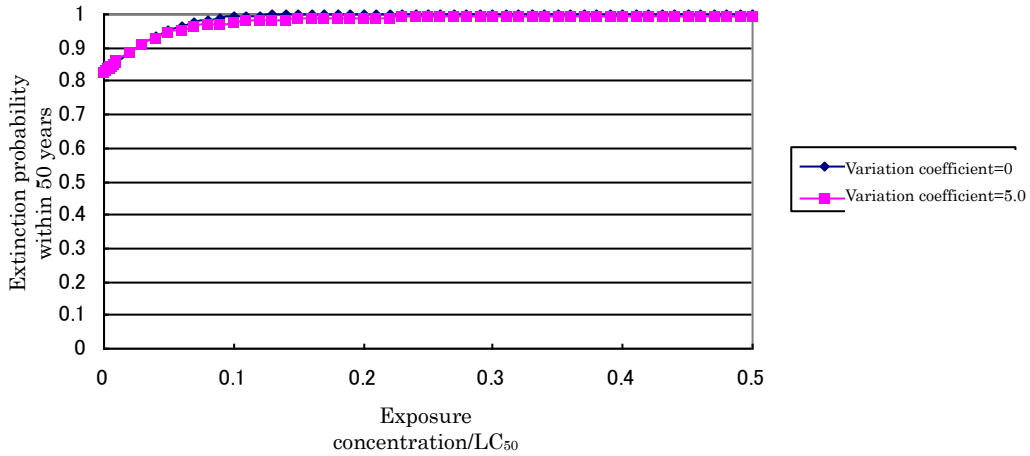

Figure 2.7-15: Relation of exposure concentration and extinction probability concerning endangered type IA (when 50 years pass)

These parameters were assigned in the Lande-Orzack model to calculate the extinction probability. Figures 2.7-14 and 2.7-15 show the time-series cumulative extinction probability if the amount of extinction is 0, and the relation between the exposure concentration and the extinction probability when 50 years pass.

Like endangered type IB, endangered type IA already have very high extinction probability (50 years after, about 80%) even if they are not exposed to chemicals, and the result showed that exposure to about 10% of LC_{50} will lead to extinction almost without fail.
After this result was converted into the extinction risk per year to find the slope at the current exposure concentration, *EF* was calculated for each chemical.

(6) Arrangement of the damage function and damage factor of ecotoxicity

The calculation results and parameters gained so far were entered in Equation 2.7-4 to find the damage factor of ecotoxicity. With regard to some chemicals, Table 2.7-12 shows the parameters used for the calculation of the damage function and the calculation results.

(7) Characteristics of the damage factor of ecotoxicity

The following are characteristics of the damage factors of the five chemical substances shown in Table 2.7-12:

1) Benzene

If the damage factor is compared in terms of the place for release, it is greater in the following order, from highest to lowest: release to water, release to soil, and release to the air. This is because of high efficiency in raising the concentration in surface water.

If EINES is compared among species, EINES of "other species" (the total for shellfishes, crustaceans (other than crustaceans), and amphibians) is the highest. This is because LC_{50} of shellfishes is lower than the other species' toxicity, and shellfishes have strong toxicity. If EINES is compared among endangered classes, EINES of endangered classes IA and IB is higher concerning all the species, while EINES of endangered type II and sound species is 0.

2) Arsenic

If the damage factor is compared in terms of the place for release, it is greater in the following order, from highest to lowest: release to water, release to soil, and release to the air. This is because of high efficiency in raising the concentration in surface water. However, compared with benzene, the damage factor is larger and the differences among the places for release are smaller.

If EINES is compared among species, EINES of algae is highest. This is because EC_{50} of algae is lower than the other species' toxicity, and algae have strong toxicity. If EINES is compared among endangered classes, EINES of endangered classes IA and IB is higher and occupies most of the damage factor concerning all the species. Although EINES of endangered type II and sound species is higher than that of benzene, its contribution to the damage factor is small.

3) Trichloroethylene

If the damage factor is compared in terms of the place for release, it is greater in the following order, from highest to lowest: release to water, release to soil, and release to the air. This is because of high efficiency in raising the concentration in surface water. However, compared with benzene, the damage factor is smaller.

If EINES is compared among species, EINES of "other species" is the highest. This is because LC_{50} of trichloroethylene has strong toxicity against amphibians and the total EINES for shellfishes, crustaceans (other than crustaceans), and amphibians has been entered. If EINES is compared among endangered classes, like benzene, EINES of endangered classes IA and IB is higher concerning all species, while EINES of endangered type II and the sound species is 0.

4) Dichloromethane

If the damage factor is compared in terms of the place for release, it is greater in the following order, from highest to lowest: release to water, release to soil, and release to the air. This is because of high efficiency in raising the concentration in surface water. However, compared with benzene, the damage factor is smaller.

If EINES is compared among species, EINES of "other species" is the highest. This is because LC_{50} of dichloromethane is the smallest for crustaceans, etc. (crustaceans) and has strong toxicity against them and the total EINES for the three other types of species has been entered. If EINES is compared among endangered classes, like benzene, EINES of endangered classes IA and IB is higher concerning all species, while EINES of endangered type II and sound species is 0.

5) Cadmium

If the damage factor is compared in terms of the place for release, it is greater in the following order, from highest to lowest: release to water, release to soil, and release to the air. This is because of high efficiency in raising the concentration in surface water. However, compared with benzene, the damage factor is larger and the differences among the places where the chemical is released are smaller.

If EINES is compared among species, EINES of "other species" is highest. This is because LC_{50} of amphibians is the lowest and has strong toxicity. Moreover, the toxicity against crustaceans and algae is stronger than that against other species. If EINES is compared among endangered classes, EINES of endangered classes IA and IB is higher and occupies most of the damage factor concerning all species. Although EINES of endangered type II and sound species is higher than that of benzene, its contribution to the damage factor is small.

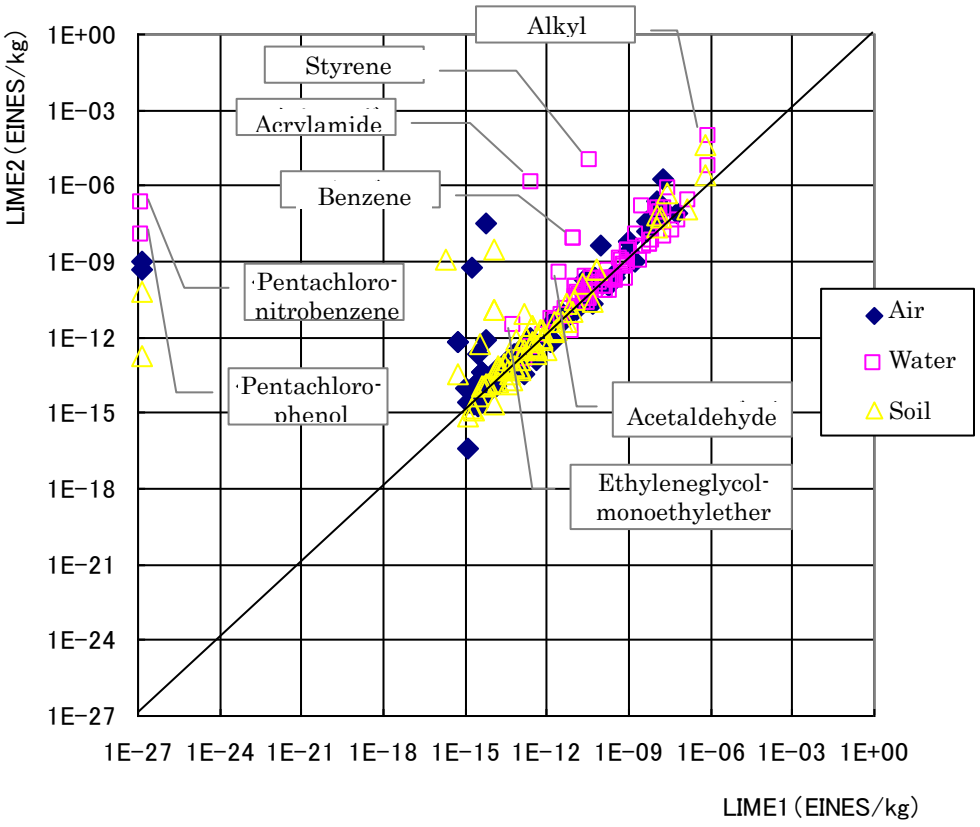

Figure 2.7-16: Comparison between LIME 1 and LIME 2 (ecotoxicity)

The figure shows the chemicals whose damage factor greatly rose at the time of release to water.

Table 2.7-12: Parameters used for calculation of damage factors and calculation results (extracts concerning some chemicals)

Chemical	Efficiency in raising concentration in surface water by place for release [(g/m^3)/ (t/yr)]			Species	Toxicity (Average of EC_{50} or LC_{50} [μg/L]	Division	No. of species	Extinction probability in ordinary (current) time	Expected Increase in Number of Extinct Species (EINES) due to additional release of 1[kg/yr]					
									Additional release to the air		Additional release to water		Additional release to soil	
	Air	Water	Soil						(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)
Benzene	1.38E-12	1.70E-08	2.95E-11	Algae	7.06E+04	Endangered (IA)	17	1.65E-02	0.00E+00	0.00E+00	1.69E-14	2.88E-13	3.12E-17	5.31E-16
						Endangered (IB)	17	9.75E-03	0.00E+00	0.00E+00	7.88E-14	1.34E-12	1.35E-16	2.30E-15
						Endangered (II)	6	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	5500	1.00E-15	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**0.00E+00**		**1.63E-12**		**2.83E-15**
				Crustaceans	1.38E+03	Endangered (IA)	4.5	1.65E-02	6.59E-17	9.89E-17	8.67E-13	1.30E-12	1.50E-15	2.25E-15
						Endangered (IB)	4.5	9.75E-03	3.26E-16	4.89E-16	4.04E-12	6.05E-12	6.98E-15	1.05E-14
						Endangered (II)	17	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	100	1.00E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**5.88E+16**		**7.35E-12**		**1.27E-14**
				Fishes	3.87E+04	Endangered (IA)	29	1.65E-02	0.00E+00	0.00E+00	3.10E-14	8.98E-13	5.20E-17	1.51E-15
						Endangered (IB)	29	9.75E-03	1.39E-17	4.02E-16	1.44E-13	4.18E-12	2.43E-16	7.04E-15
						Endangered (II)	18	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	300	1.00E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**4.02E+16**		**5.07E-12**		**8.55E-15**
				Other†	3.87E+01 3.24E+04 2.48E+05	Endangered (IA)	47		2.32E-15	1.08E-13	2.86E-11	1.33E-09	4.96E-14	2.30E-12
						Endangered (IB)	55		1.02E-14	5.03E-13	1.25E-10	6.19E-09	2.17E-13	1.07E-11
						Endangered (II)	190		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	1165		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**6.11E+13**		**7.52E-09**		**1.30E-11**
				Total (DF^{EcoTox}(*Safe, X, R*))						**6.12E-13**		**7.54E-09**		**1.31E-11**
Arsenic	9.68E-07	2.65E-06	1.81E-06	Algae	3.66E+01	Endangered (IA)	17	1.65E-02	1.86E-09	3.16E-08	5.09E-09	8.65E-08	3.48E-09	5.92E-08
						Endangered (IB)	17	9.75E-03	8.65E-09	1.47E-07	2.37E-08	4.02E-07	1.62E-08	2.75E-07
						Endangered (II)	6	1.00E-03	1.40E-16	8.38E-16	8.66E-16	5.20E-15	4.35E-16	2.61E-15
						Sound species	5500	1.00E-15	6.98E-28	3.84E-24	4.33E-27	2.38E-23	2.17E-27	1.20E-23
						Subtotal				**1.79E-07**		**4.89E-07**		**3.35E-07**

Chemical	Efficiency in raising concentration in surface water by place for release [(g/m^3)/ (t/yr)]			Species	Toxicity (Average of EC_{50} or LC_{50} [μg/L]	Division	No. of species	Extinction probability in ordinary (current) time	Expected Increase in Number of Extinct Species (EINES) due to additional release of 1[kg/yr]					
									Additional release to the air		Additional release to water		Additional release to soil	
	Air	Water	Soil						(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)
Arsenic	9.68E-07	2.65E-06	1.81E-06	Crustaceans	1.14E+03	Endangered (IA)	4.5	1.65E-02	5.95E-11	8.92E-11	1.63E-10	2.44E-10	1.11E-10	1.67E-10
						Endangered (IB)	4.5	9.77E-03	2.77E-10	4.15E-10	7.57E-10	1.13E-09	5.18E-10	7.77E-10
						Endangered (II)	17	1.00E-03	0.00E+00	0.00E+00	3.69E-18	4.79E-17	2.17E-18	2.82E-17
						Sound species	100	1.00E-12	3.03E-27	3.03E-25	1.53E-26	1.53E-24	9.09E-27	9.09E-25
						Subtotal				**5.04E-10**		**1.38E-09**		**9.44E-10**
				Fishes	2.20E+03	Endangered (IA)	29	1.65E-02	3.09E-11	8.95E-10	8.45E-11	2.45E-09	5.78E-11	1.68E-09
						Endangered (IB)	29	9.76E-03	1.44E-10	4.17E-09	3.93E-10	1.14E-08	2.69E-10	7.80E-09
						Endangered (II)	18	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	300	1.00E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**5.06E-09**		**1.38E-08**		**9.48E-09**
				Other†	4.00E+03 7.97E+02 4.00E+01	Endangered (IA)	47		5.78E-11	2.69E-09	1.58E-10	7.35E-09	1.08E-10	5.03E-09
						Endangered (IB)	55		1.53E-09	7.58E-08	4.19E-09	2.07E-07	2.87E-09	1.42E-07
						Endangered (II)	190		7.49E-18	1.33E-15	4.73E-17	8.42E-15	2.38E-17	4.23E-15
						Sound species	1165		1.98E-23	2.30E-20	1.27E-22	1.48E-19	6.17E-23	7.19E-20
						Subtotal				**7.85E-08**		**2.15E-07**		**1.47E-07**
				Total (DF^{EcoTox}(*Safe, X, R*))						**2.63E-07**		**7.19E-07**		**4.92E-07**
Trichloroethene	4.52E-12	4.79E-08	1.18E-10	Algae	2.98E+05	Endangered (IA)	17	1.65E-02	0.00E+00	0.00E+00	1.13E-14	1.92E-13	2.78E-17	4.72E-16
						Endangered (IB)	17	9.75E-03	0.00E+00	0.00E+00	5.25E-14	8.93E-13	1.25E-16	2.12E-15
						Endangered (II)	6	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	5500	1.00E-15	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**0.00E+00**		**1.09E-12**		**2.60E-15**
				Crustaceans	4.07E+04	Endangered (IA)	4.5	1.65E-02	0.00E+00	0.00E+00	8.28E-14	1.24E-13	2.01E-16	3.02E-16
						Endangered (IB)	4.5	9.75E-03	3.47E-17	5.20E-17	3.85E-13	5.78E-13	9.49E-16	1.42E-15
						Endangered (II)	17	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	100	1.00E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**5.20E-17**		**7.02E-13**		**1.73E-15**
				Fishes	6.56E+04	Endangered (IA)	29	1.65E-02	0.00E+00	0.00E+00	5.13E-14	1.49E-12	1.28E-16	3.72E-15
						Endangered (IB)	29	9.75E-03	2.08E-17	6.04E-16	2.39E-13	6.92E-12	5.86E-16	1.70E-14
						Endangered (II)	18	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	300	1.00E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**6.04E-16**		**8.4E-12**		**2.07E-14**

Chemical	Efficiency in raising concentration in surface water by place for release [(g/m^3)/ (t/yr)]			Species	Toxicity (Average of EC_{50} or LC_{50} [μg/L]	Division	No. of species	Extinction probability in ordinary (current) time	Expected Increase in Number of Extinct Species (EINES) due to additional release of 1[kg/yr]					
									Additional release to the air		Additional release to water		Additional release to soil	
	Air	Water	Soil						(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)
Trichloroethene	4.52E-12	4.79E-08	1.18E-10	Other†	5.60E+04 7.50E+04 4.58E+04	Endangered (IA)	47		0.00E+00	0.00E+00	6.01E-14	2.79E-12	1.45E-16	6.77E-15
						Endangered (IB)	55		2.86E-17	1.41E-15	3.18E-13	1.57E-11	7.76E-16	3.84E-14
						Endangered (II)	190		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	1165		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**1.41E-15**		**1.85E-11**		**4.52E-14**
				Total (DF^{EcoTox}(*Safe, X, R*))						**2.07E-15**		**2.87E-11**		**7.02E-14**
Dichloromethane	4.45E-11	9.92E-08	3.01E-09	Algae	3.15E+05	Endangered (IA)	17	1.65E-02	0.00E+00	0.00E+00	2.21E-14	3.76E-13	6.70E-16	1.14E-14
						Endangered (IB)	17	9.75E-03	4.16E-17	7.08E-16	1.03E-13	1.75E-12	3.12E-15	5.31E-14
						Endangered (II)	6	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	5500	1.00E-15	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**7.08E-16**		**2.12E-12**		**6.45E-14**
				Crustaceans	3.55E+03	Endangered (IA)	4.5	1.65E-02	8.78E-16	1.32E-15	1.96E-12	2.95E-12	5.97E-14	8.95E-14
						Endangered (IB)	4.5	9.75E-03	4.09E-15	6.14E-15	9.14E-12	1.37E-11	2.78E-13	4.16E-13
						Endangered (II)	17	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	100	1.00E-12	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**7.45E-15**		**1.66E-11**		**5.06E-13**
				Fishes	4.00E+05	Endangered (IA)	29	1.65E-02	0.00E+00	0.00E+00	1.74E-14	5.05E-13	5.27E-16	1.53E-14
						Endangered (IB)	29	9.75E-03	3.47E-17	1.01E-15	8.10E-14	2.35E-12	2.45E-15	7.11E-14
						Endangered (II)	18	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	300	1.00E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**1.01E-15**		**2.85E-12**		**8.64E-14**
				Other†	3.14E+04 2.38E+04 2.88E+04	Endangered (IA)	47		1.00E-16	4.67E-15	2.29E-13	1.07E-11	6.96E-15	3.24E-13
						Endangered (IB)	55		5.26E-16	2.60E-14	1.18E-12	5.86E-11	3.60E-14	1.78E-12
						Endangered (II)	190		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	1165		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**3.07E-14**		**6.93E-11**		**2.10E-12**
				Total (DF^{EcoTox}(*Safe, X, R*))						**3.99E-14**		**9.09E-11**		**2.76E-12**

Chemical	Efficiency in raising concentration in surface water by place for release [(g/m^3)/ (t/yr)]			Species	Toxicity (Average of EC_{50} or LC_{50} [μg/L]	Division	No. of species	Extinction probability in ordinary (current) time	Expected Increase in Number of Extinct Species (EINES) due to additional release of 1[kg/yr]					
									Additional release to the air		Additional release to water		Additional release to soil	
	Air	Water	Soil						(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)	(1species)	(Whole environment in Japan)
Cadmium	5.88E-07	1.90E-06	9.08E-07	Algae	1.68E+02	Endangered (IA)	17	1.65E-02	2.46E-10	4.19E-09	7.98E-10	1.36E-08	3.81E-10	6.47E-09
						Endangered (IB)	17	9.78E-03	1.15E-09	1.95E-08	3.71E-09	6.31E-08	1.77E-09	3.01E-08
						Endangered (II)	6	1.00E-03	6.29E-18	3.77E-17	4.68E-17	2.81E-16	1.21E-17	7.29E-17
						Sound species	5500	1.00E-15	3.06E-29	1.68E-25	2.34E-28	1.29E-24	6.13E-29	3.37E-25
						Subtotal				**2.37E-08**		**7.67E-08**		**3.66E-08**
				Crustaceans	9.00E+01	Endangered (IA)	4.5	1.65E-02	4.59E-10	6.89E-10	1.49E-09	2.23E-09	7.09E-10	1.06E-09
						Endangered (IB)	4.5	9.83E-03	2.14E-09	3.20E-09	6.91E-09	1.04E-08	3.30E-09	4.95E-09
						Endangered (II)	17	1.00E-03	1.47E-17	1.92E-16	1.20E-16	1.56E-15	3.14E-17	4.09E-16
						Sound species	100	1.00E-12	5.82E-26	5.82E-24	4.78E-25	4.78E-23	1.26E-25	1.26E-23
						Subtotal				**3.89E-09**		**1.26E-08**		**6.01E-09**
				Fishes	2.18E+03	Endangered (IA)	29	1.65E-02	1.89E-11	5.49E-10	6.12E-11	1.78E-09	2.92E-11	8.48E-10
						Endangered (IB)	29	9.75E-03	8.80E-11	2.55E-09	2.85E-10	8.26E-09	1.36E-10	3.94E-09
						Endangered (II)	18	1.00E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Sound species	300	1.00E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
						Subtotal				**3.10E-09**		**1.00E-08**		**4.79E-09**
				Other†	1.97E+03 2.50E+03 4.00E+01	Endangered (IA)	47		4.27E-11	1.99E-09	1.38E-10	6.42E-09	6.60E-11	3.07E-09
						Endangered (IB)	55		9.63E-10	4.77E-08	3.12E-09	1.54E-07	1.49E-09	7.36E-08
						Endangered (II)	190		2.99E-18	5.32E-16	2.56E-17	4.55E-15	6.65E-18	1.18E-15
						Sound species	1165		7.72E-24	8.99E-21	7.06E-23	8.21E-20	1.72E-23	2.00E-20
						Subtotal				**4.96E-08**		**1.61E-07**		**7.67E-08**
				Total (DF^{EcoTox}(*Safe, X, R*))						**8.03E-08**		**2.60E-07**		**1.24E-07**

† The spaces for toxicity values (LC_{50} average) concerning "other" species show the values of LC_{50} for shellfishes, crustaceans (other than crustaceans), and amphibians. With regard to the procedure for calculation of the Expected Increase in Number of Extinct Species (EINES), the extinction probability was first assessed from the increase in the concentration and the toxicity value for each of the three groups of species. After the result was multiplied by the number of species belonging to each group of species, the total was calculated. The spaces for "whole environment in Japan" show the total of EINES of the three groups of species. The spaces for "1 species" shows the total divided by the total number of species.

(8) Comparison of damage factors between LIME 1 and LIME 2

Figure 2.7-16 shows the results of comparison between the newly calculated damage factors under LIME 2 and the existing ones under LIME 1. Although there is generally no great difference between LIME 1 and LIME, damage factors greatly rose concerning some chemicals. This is because LIME 2 greatly reflects the impact on the species other than algae, fishes, and crustaceans. If chemicals have strong toxicity especially against the species other than algae, fishes, and crustaceans (amphibians, shell, etc.), although LIME 1 could not accurately assess toxicity because the average toxicity for algae, fishes, and crustaceans was applied, LIME 2 can accurately assess the toxicity against amphibians and shell. The following are chemicals whose damage factors sharply rose (by a one-or-more-digit percentage).

[Chemicals whose damage factors sharply rose (examples)]

- Alkyl mercury
- Acrylamide
- Acetaldehyde
- Ethyleneglycolmonoethylether
- Styrene
- Benzene
- Pentachloronitrobenzene
- Pentachlorophenol

2.7.4 Procedure for impact assessment of ecotoxicity

Concrete procedures for characterization of ecotoxicity and assessment of damage are as described below.

Users can select characterization, damage assessment, or integration according to purpose and use it for LCA.

In the characterization, the impact of the release of a toxic chemical to the ecosystem is assessed by separating the terrestrial ecosystem from the aquatic ecosystem. The characterization results CI^{Terr} and CI^{Aqua} can be obtained from the inventory Inv (X, R) to R, the place for release of the toxic chemical X, and the characterization factors CF^{Terr} (X, R) and CF^{Aqua} (X, R).

$$CI^{Terr} = \sum_{X}\sum_{R} CF^{Terr}(X,R)\cdot Inv(X,R) \qquad (2.7\text{-}19)$$

$$CI^{Aqua} = \sum_{X}\sum_{R} CF^{Aqua}(X,R)\cdot Inv(X,R) \qquad (2.7\text{-}20)$$

Because the characterization factors CF^{Terr} (X, R) and CF^{Aqua} (X, R) differ according to R, the place for release of the toxic chemical, it is necessary to divide the inventory data by R (air, water, or soil).

With regard to the characterization factors CF^{Terr} (X, R) and CF^{Aqua} (X, R), it is recommendable that LIME should use the characterization factors TETP and AETP obtained from the comparison between fate analysis results based on the environmental conditions in Japan and the maximum permissible amount of toxic chemicals for conservation of species. CI^{Terr} and CI^{Aqua} are regarded as the total amount of released toxic chemicals converted into the amount of benzene released to soil, the typical causative chemical, and the total amount of released toxic chemicals converted into the amount of benzene released to water, respectively.

The damage assessment is carried out concerning the aquatic ecosystem (algae, crustaceans, etc. (crustaceans), fishes, shellfishes, crustaceans, etc. (other than crustaceans) and amphibians). The damage assessment result *DI* (*Safe*) can be obtained from *Inv* (*X, R*) of toxic chemicals and the damage factor DF^{EcoTox} (*Safe, X, R*) of the object of protection *Safe*.

$$DI\,(Safe) \;=\; \sum_{X}\sum_{R} DF^{\,EcoTox}\,(Safe, X, R)\cdot Inv\,(X, R) \qquad (2.7\text{-}21)$$

As in the case of characterization factors, the damage factor DF^{EcoTox} (*Safe, X, R*) differs according to *R*, the place for release of the toxic chemical, it is necessary to divide the inventory data by *R*.

DI (*Safe*) means the amount of potential damage against the object of protection *Safe* due to the release of a toxic chemical. This enables damage assessment concerning biodiversity. If the object of protection is the same, it is possible to compare or integrate it with the amount of damage arising through different impact categories – for example, the impact of land transformation on biodiversity.

The integration factor into which impact on biodiversity is economically converted or non-dimensionalized, the integration factor IF^{EcoTox} (*X, R*), is used for integration. The integration index *SI* can be obtained from each chemical's *Inv* (*X, R*) and integration factor IF^{EcoTox} (*X, R*). The results can be compared directly with or added to assessment results in other impact categories.

$$SI = \sum_{X}\sum_{R}\left(IF^{\,EcoTox}(X,R)\times Inv(X,R)\right) \qquad (2.7\text{-}22)$$

The characterization factor CF^{Terr} (*X, R*) has been attached hereto as A1. The characterization factor CF^{Aqua} (*X, R*) and the damage factor DF^{EcoTox} (*Safe, X, R*) have been attached hereto as A2. The integration factor IF^{EcoTox} (*X, R*) has been attached hereto as A3.

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2.8 Eutrophication

2.8.1 What phenomenon is eutrophication?

The term "eutrophication" is often used to mean an increase in the amount of nutrient salts (mainly nitrogen and phosphorus) flowing into closed water areas, such as inner bays and lakes. Nutrient salts, such as nitrogen and phosphorus, are essential elements for organisms. Usually, the photosynthesis of algae, which is represented by phytoplankton in water areas, is limited by shortage of nutrient salts. Because of this, an increase in the concentration of nutrient salts in water promotes the photosynthesis of algae – that is, the primary production of organic substances. For example, there is a thesis that defines the state of eutrophication by the speed of supply of organic carbon into the ecosystem, using the speed of the primary production of algae in water, which is easier to measure than the inflow amount of nutrient salts (substance of concern) into water (Table 2.8-1) (Nixon 1995).
At the initial stage, eutrophication in water usually means that an increase in the concentration of nutrient salts results in a rise in the speed of primary production, enriching the production of the organisms in the whole ecosystem, including fishery production, through the food chain within the ecosystem. However, if the degree of eutrophication becomes higher, various unfavorable effects arise, including hindrance to the sound circulation of substances in the aquatic ecosystem. Because of this, the term "eutrophication" is usually used as an indicator of not only progress in the nutritional condition in water but also such effects as progress in organic pollution and occurrence of a suboxic phenomenon. In other words, because the effects of eutrophication greatly differ according to water condition, eutrophication may increase fishery production or have unfavorable effects on the aquatic ecosystem.

Table 2.8-1: Trophic levels in the sea (Nixon 1995)

Trophic level	Organic carbon supply (primary production)
	$gCm^{-2}y^{-1}$
Oligotrophic	≤100
Mesotrophic	100-300
Eutrophic	301-500
Hypertrophic	> 500

Column 2.8-1

Eutrophication in Europe

Although eutrophication is regarded as an issue related to water in Japan, the term is also used for the phenomenon of an increase in the amount of nitrogen included in soil in Europe. Soil environments where a shortage of nitrogen limits the growth of plants generally exist. An increase in the amount of nitrogen is drawing attention because it has effects on the plants and algae struggling for existence under a limited amount of nitrogen, such as causing a change in the species composition, increasing productivity, decreasing the diversity of species, or changing tolerance to frost, drought, and other stresses (UNEP/RIVM 1999).
In Japan also, there is the problem that an increase in the amount of nitrogen in soil causes health damage due to groundwater pollution related to nitrate-nitrogen and nitrite-nitrogen. In July 2001, the Ministry of the Environment added ammonia, ammonium compounds, nitrous acid compounds, and nitric compounds to the toxic substances and placed them under the regulation of underground seepage. In Japan, however, an increase in the amount of nitrogen in soil or groundwater is hardly treated as a problem related to eutrophication.

(1) Causal relationships of eutrophication

a Generation of substances of concern

The sources of substances of concern related to eutrophication (nutrient salts and organic substances) can be divided into daily, industrial, livestock, and agricultural water pollutants, as well as water pollutants flowing from urban areas. Generally, there are three types of channels through which substances of concern flow into closed water areas: 1) rivers; 2) direct release from a coast to a closed water area; and 3) the air. In Japan, 1) and 2) occupy a large percentage in light of quantity (Water Pollution Control Division, Water Quality Bureau, Ministry of the Environment 1996). Especially in inner bays where the population and industries concentrate in basins, the amount of substances of concern is great and water pollution due to the inflow of organic substances and eutrophication due to the inflow of nitrogen and phosphorus have great impact on coastal environments. If the circulation of substances is considered from a somewhat wider viewpoint, huge amounts of nitrogen and phosphorus brought in from the outside (domestic and overseas areas) as food, industrial materials, and fertilizer finally flow into seas after being used for human activities, except for those that accumulate in terrestrial areas, are transferred to other basins or are exported to overseas.

As measures for reducing the amount of generated substances of concern and the amount of those flowing into water areas, the regulation of drainage and the improvement of sewerage facilities have been steadily promoted under the Water Pollution Control Act. However, further efforts are still needed for improving water pollution due to the inflow of organic substance and water pollution caused by the inflow of nitrogen and phosphorus, about which the biochemical oxygen demand (BOD) and the chemical oxygen demand (COD) are used as indices. Because of this, in the wide closed sea areas where it is difficult to fulfill the environmental standards only by the regulation of the concentration of wastewater, total volume control has been established to reduce the total volume of pollutants flowing into the areas with consideration for not only the concentration but also the volume of wastewater. At present, the seventh total volume control, whose target year is FY2014, is under consideration.

Table 2.8-2 shows the amount of generated eutrophication substances:

Table 2.8-2: Amount of generated eutrophication substances (UNEP/RIVM 1999)

[tons/day]

	COD		Nitrogen		Phosphorus	
	FY1979	FY1994	FY1979	FY1994	FY1979	FY1994
Tokyo Bay	477	286	365	281	41.4	23.0
Ise Bay	307	246	197	174	26.0	18.9
Seto Inland Sea	1010	746	709	736	66.0	42.6

b Physical process

The impact of eutrophication is closely connected with the flow in the water area, the development of density stratification, formation of a front (a border between different seawaters), and mixing of water masses.

For example, in a water area, if low-density water (light water) is formed above high-density

water (heavy water) (this is called “stratification”), the water area becomes stable in light of density and vertical mixing becomes difficult. This formation of stratification strongly influences eutrophication damage.
The density of seawater depends on water temperature, salinity, and pressure. Because a change in the density due to pressure is small in a shallow coastal area, the density depends on water temperature and salinity. In addition, the strength of stratification in a coastal area depends on the balance between the stability and instability of the density. The factors for stability are phenomena that facilitate a decrease in the density near the surface, such as the inflow of fresh water and the heating of the sea surface, while the factors for instability include a disturbance of seawater caused by tides or wind waves (Figure 2.8-1). The stratification of salt and water temperature, which can be seen in almost all the coastal areas in Japan, is divided into the upper layer for the photosynthesis process (organic substance production process) and the lower layer for the process of mineralizing organic substances (process of consuming oxygen) (Fenchel 1992). Suboxic water mass, the most serious problem among the effects of eutrophication, is not produced only as a result of a rise in the concentration of nutrient salt in a water area. It also occurs under physical conditions where a stationary water mass with little vertical mixing is formed.
Moreover, one of the characteristics of inner bays, where eutrophication is likely to occur, is a flow called estuary circulation. Eutrophication is likely to occur at the mouth of a river, which is characterized by mixing freshwater with seawater and is called an estuary.

Column 2.8-2

Eutrophication substances via the air

Although the amount of substances of concern via the air is not so large in Japan, the supply of nitrogen via the air occupies 20% to 50% of the total load of nitrogen from the outside of water areas (Paerl et al. 1994) and therefore is regarded as important. On the other hand, with regard to phosphorus, substances of concern supplied via the air to seas can be ignored compared with the other supply sources.
Nitrogen compounds that serve as nutrient salts in the air can be classified into the following groups:

- NHx group: gaseous ammonia (NH_3) and particulate ammonia (NH_4^+ aerosol)
- NOv group: Gaseous N_2O_5, HNO_4, NO, NO_2, HNO_3, HNO_2 and PAN, and particulate NO_3^- aerosol and NO_3 radical
- Organic compounds: The concentration of organic nitrogen in the air is very low and there is not much information on their origin.

The supply of nitrogen via the air has two processes: wet deposition, which is caused by rainfall, and dry deposition, which is caused by a disturbance of the air on a sea or land surface. There are two main differences between these two processes: while nitrogen moves from the air to the sea at all the layers where snow or rain falls in the air in the process of wet deposition, nitrogen moves from the air only in the layer very near a sea or land surface; moreover, while wet deposition occurs only when rain falls, dry deposition occurs at any time (Asman et al. 1996).

Because there is a difference between freshwater and seawater in density, a special flow flows toward the bay entrance, and the flow gradually takes in water in the lower layer and becomes saltier. As a supplement flow, water in the lower layer flows toward the mouth of called estuary circulation occurs at an estuary. The upper layer of low-salt water flowing from a

river the river and forms vertical circulation. This flow is called estuary circulation. Estuary circulation is an important flow that greatly influences the transportation of substances and organisms in the inner bay, such as nutrient salt supplied from the river, and is closely connected with the impact of eutrophication.

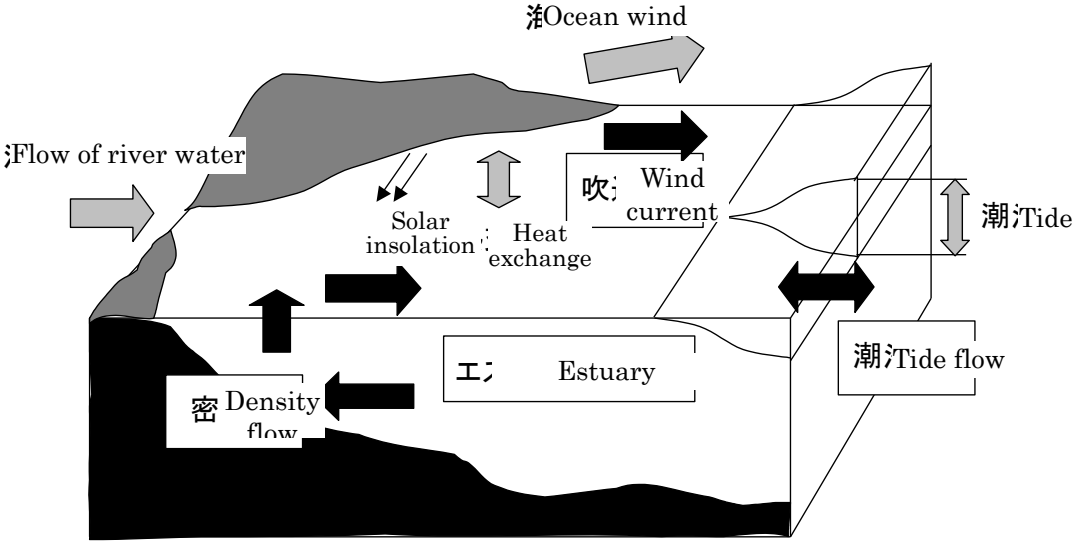


Figure 2.8-1: Physical process in an inner bay

c Biochemical process

In a water area where eutrophication has progressed, the transformation of substances from the dissolved state to the particulate state occurs actively and consecutively. Inorganic nutrient salts (nitrogen and phosphorus) and carbon dioxide in the dissolved state change to organic substances in the particulate state by the photosynthesis of phytoplankton and then become organic again and change to the particulate state through metabolic processes by bacteria and the like. The processes of transforming from the dissolved state to the particulate state include photosynthesis, assimilation of dissolved organic substances by heterotrophic organisms, and transformation of dissolved substances into colloid particles by sedimentation or condensation. The processes of transforming from the particulate state to the dissolved state include dissolution of cells, exudation and leakage of dissolved organic substances from cells of phytoplankton or bacteria, discharge of inorganic dissolved substances that have changed from organic to inorganic due to the metabolism process by heterotrophic organisms, leakage of dissolved substances at the time of breakage of phytoplankton cells during the process of congestion, and leakage of dissolved substances from fecal pellets.

Because, in this way, processes differ between dissolved substances and particulate substances, they create vertically different layers in stratified water. As photosynthesis that produces particulate substances (the process that produces oxygen at the same time) is limited by light, it is carried out in the euphotic layer (the upper layer). In the layer below the euphotic layer (the lower layer), the process of mineralizing submerged phytoplankton and particulate organic substances (oxygen consumption process) is prominent (Figure 2.8-2). The stratification of water and the difference in place (water depth) between the oxygen production process and the oxygen consumption process result in the development of suboxic water under the pycnocline.

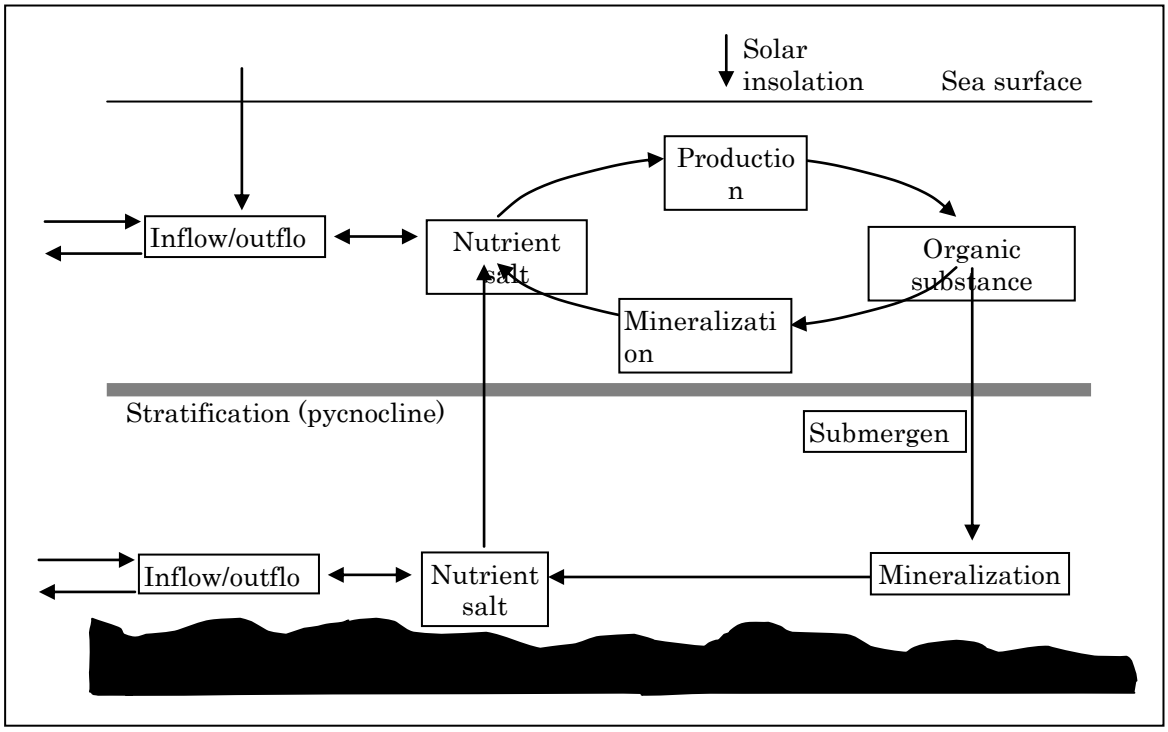


Figure 2.8-2: Nutrient salt circulation under a stratified condition (Revised version of Richardson (1996))

(2) Endpoints of eutrophication

Figure 2.8-3 shows the cause-effect chain of eutrophication. Inorganic nitrogen and phosphorus and organic substances released into water first cause an increase in the current amount of phytoplankton and underwater organic substances and a change in the organization of phytoplankton species, which then create various effects on diversified elements of the ecosystem. This change in the ecosystem has impact on human activities that use water, such as fisheries, and also on social assets. The figure shows endpoints that are usually regarded as problematic.

Of the endpoints of eutrophication, the most serious problem is various effects caused by suboxic water (the concentration of dissolved oxygen (DO) becomes lower than the saturated concentration). Because organisms' systems basically depend on oxygen even in water, a decrease in DO in water greatly influences the whole aquatic ecosystem.

If eutrophication increases plant photosynthesis (production of organic substances), the supply of organic substances to the sea bottom increases. Under the above-mentioned condition stratified by the physical process, oxygen consumption by the decomposition of organic substances and the process of mineralizing them increases in the bottom layer, and the concentration of DO in water decreases. If this progresses, the bottom-layer water may be short of oxygen. Under the condition that no oxygen exists, organic substances may be mineralized in an anaerobic way. This process of mineralization by the use of hydrosulfate may facilitate the production of hydrogen sulfide harmful to living organisms, forming a mat of sulfate reducers on the sea bottom.

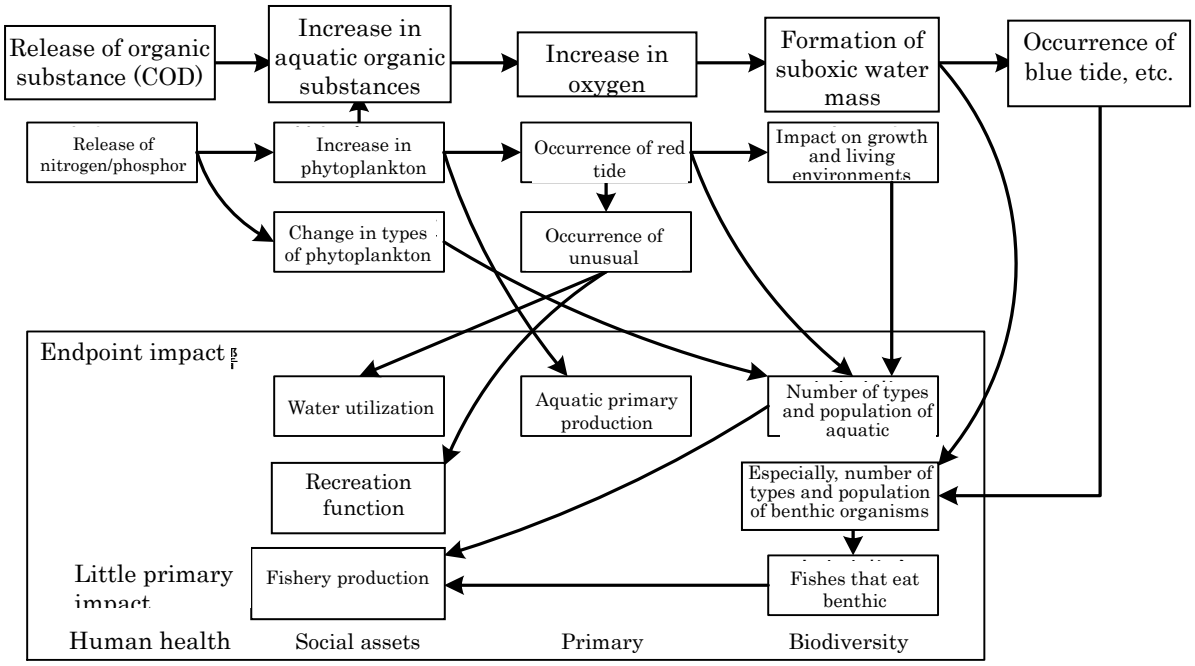


Figure 2.8-3: Cause-effect chain of eutrophication

The important processes of supplying oxygen to water are the supply of oxygen from the air through the sea surface, and the production of oxygen by phytoplankton's photosynthesis. Because both oxygen supply processes occur in the sea surface layer, as described above, suboxic water mass develops in the bottom layer if mixing of water in the surface layer with water in the bottom layer is difficult due to sharp vertical density gradient (density stratification). The processes of supplying oxygen to the bottom-layer water under the stratified condition are usually the supply of oxygen from the surface through pycnocline caused by winds, and the moving of the bottom-layer water that contains oxygen from surrounding sea areas. If pycnocline is destroyed, oxygen is supplied from the surface layer to the bottom layer. Therefore, in a water area, such as Mikawa Bay, where pycnocline is easily destroyed by ocean wind, the amount of organic substances is large because of eutrophication and the speed of oxygen consumption at the bottom layer is high, because the supply of oxygen is frequently replaced with the consumption of oxygen and vice versa, the DO concentration in the bottom layer changes sharply.

Living organisms' first response to a suboxic phenomenon is escape from the suboxic area, as often shown by many kinds of fishes. If oxygen cannot be used in bottom-layer water, shells and other species that have low mobility and low tolerance to lack of oxygen begin to die, the structure of the association of benthic organisms changes, and the decomposition of organic substances centers on the metabolism of microscopic organisms. In serious cases, no oxygen exists in the bottom-layer water and most of the benthic organisms die. According to Denmark's EPA, many types of fishes and benthic organisms try to escape from the water area if the concentration of dissolved oxygen becomes less than 4 mg/L, and animals that have low mobility and cannot escape die in a short time if it becomes less than 2 mg/L (Danish Environmental Protection Agency 1998). The Japan Fisheries Resource Conservation Association has designated 3 mL/L (about 4.2 mg/L) as the minimum concentration of oxygen in the bottom layer that does not have adverse effect on the fishing of benthic fishes (Japan Fisheries Resource Conservation Association 2000).

Figure 2.8-4 shows observed data about the central part of Mikawa Bay as an example of changes in the DO concentration in the bottom layer. The DO concentration shows very

sharp seasonal changes, and decreases to less than 2 mg/L every summer. In a temperate zone, water stratification is a seasonal phenomenon and, in autumn, stratification is destroyed and oxygen is supplied to the bottom layer, with the result that living organisms begin to enter the layer again. However, when suboxic water mass appears again in the next summer, the living organisms die again. In water areas where this pattern is repeated, age structure is not formed in the association of benthic organisms, and the number of kinds of organisms that can live is limited. As a result, serious effects occur on fishery production.

A decrease in the number of benthic organisms due to lack of oxygen has influence on the aquatic ecosystem. Living organisms that play a part in the food chain disappear. Because benthic organisms play a great part in the physical circulation of bottom sediments, the disappearance of benthic organisms transforms the physical circulation of sediments – that is, the process of decomposing organic substances. This frequently worsens the environment further.

With regard to impact on the other endpoints – water utilization and recreation function – although damage that occurred in various areas has been pointed out as a problem, full discussions have yet to be held about how to assess the damage.

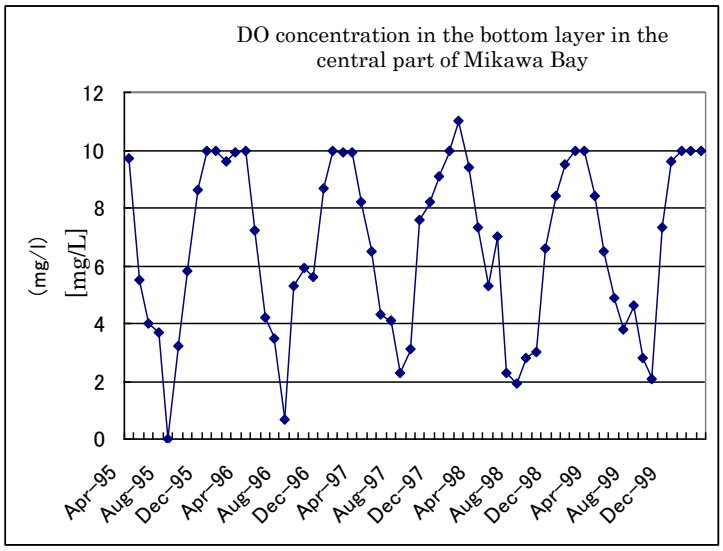


Figure 2.8-4: DO concentration in the bottom layer in the central part of Mikawa Bay (result of a survey on public water areas in Aichi Prefecture)

Column 2.8-3

Process of mineralizing organic substances around the sea bottom
In the process of mineralizing substances around the sea bottom, the main final electronic receptors are oxygen (oxic respiration), nitrate salt (denitrification), manganese oxide (IV), iron oxide, sulfate salt (sulfate reduction) and carbonate (methane production) (Canfield 1993). Mineralization by the use of oxygen has the highest energy efficiency, and the association of benthic organisms first uses oxygen for the mineralization of organic substances. If molecular oxygen disappears, nitrate salt, which has high energy efficiency, is used next, followed by manganese oxide (IV) and iron oxide. Sulfate reduction and methane production have lower energy efficiency than these. Organic substances around the sea bottom are gradually mineralized from processes with high energy efficiency to those with low energy efficiency.

Oxic respiration

$$CH_2O + O_2 \rightarrow CO_2 + H_2O$$ [free energy yields: -475 DG°(KJ mol^{-1} of CH_2O)]

Denitrification

$$5CH_2O + 4NO_3^- \rightarrow 2N_2 + 4HCO_3^- + CO_2 + 3H_2O$$ [-448 DG°(KJ mol^{-1} of CH_2O)]

Mn-oxide reduction

$$CH_2O + 3CO_2 + H_2O + 2MnO_2 \rightarrow 2Mn_2^+ + 4HCO_3^-$$ [-349 DG°(KJ mol^{-1} of CH_2O)]

Fe-oxide reduction

$$CH_2O + 7CO_2 + 4Fe(OH)_3 \rightarrow 4Fe^{2+} + 8HCO_3^-$$ [-114 DG°(KJ mol^{-1} of CH_2O)]

Sulfate reduction

$$2CH_2O + SO_4^{2-} \rightarrow H_2S + 2HCO_3^-$$ [-77 DG°(KJ mol^{-1} of CH_2O)]

Methane production

$$2CH_2O + 2H_2O \rightarrow 2CO_2 + 4H_2$$

$$4H_2 + CO_2 \rightarrow CH_4 + 2H_2O$$ [-58 DG°(KJ mol^{-1} of CH_2O)]

2.8.2 Eutrophication and characterization

Under LIME, the characterization factor for the midpoint approach and the damage factor for the endpoint approach were calculated in the impact category of eutrophication. This part describes the existing characterization factor.

(1) Existing methods for finding the characterization factor of eutrophication

There are three methods for finding the characterization factor of eutrophication (eutrophication potential, EP):

1) Calculation from the average composition ratio of biomass (Heijungs et al. 1992, Wenzel et al. 1997, Kärrman 2001)

2) Inclusion of fate and exposure analysis and regional information (Huijbregts et al. 2000)

3) Paying attention to endpoints (Goedkoop et al. 1999)

The method for finding the characterization factor listed as 1) has been widely used for LCA so far. Each eutrophic substance is weighted based on the average biomass composition ratio of aquatic organisms (Redfield ratio: $C_{106}H_{263}O_{110}N_{16}P$). First, the weights of nitrogen and phosphorus are calculated from the average biomass composition ratio, and the weight of 138 moles of oxygen molecules is calculated as COD because the decomposition of a mole of biomass requires 138 moles of oxygen molecules. Then, phosphoric salt is standardized as the base substance to find the characterization factor.

$$EP = (v_i / M_i) \Big/ (v_{PO_4^{3-}} / M_{PO_4^{3-}}) \quad (2.8\text{-}1)$$

In this equation, v_i is the contribution ratio (P:N : COD = 1 : 1/16 : 1/138) of the substance i (P, N, COD) as an eutrophication substance, and M_i is the molecular weight of the substance i.

The method listed as 2) calculates the fate factor FF of nitrogen compounds released into the air in the whole of Europe. FF was calculated for each European country. Based on the average, FF was calculated also for Western Europe, Eastern Europe, and the Whole of Europe. FF can be calculated by the following equation:

$$FF_{x,i,direct,air\rightarrow marine,Europe} = \frac{\sum_{j\in Europe} t_{i,j,x} \times E_{x,i} \times A_j \times K_{j,marine}}{E_{x,i}} \qquad (2.8\text{-}2)$$

In this equation, $t_{i,j,x}$ is the transport coefficient – that is, the ratio of the substance x that was released from the territory i and deposited in the territory j [mgNm^{-2}kgN^{-1}]. $E_{x,i}$ is the amount of the released substance x in the territory i [kgN yr^{-1}]. A_j is the area of the grid cell j [km^2]. $K_{j,marine}$ is the ratio of the ocean in the grid cell j [–]. The transport coefficient t is from the European Monitoring and Evaluation Programme (EMEP).

Based on this result, it was estimated that an average of 16% directly deposit in the ocean in the whole of Europe (Western Europe: 24%, Eastern Europe: 7.2%).

The method listed as 3) calculates the damage factor, taking into consideration the vulnerability of deposition areas. The disappearance rate of plant species (PDF) is used as the index for damage to the ecosystem. This is advantageous in that it enables comparison with other impact categories, such as land use and acidification. However, the method has some problems. For example, because it is based on data in Holland, it is difficult to expand it across the whole of Europe. In addition, it only takes into consideration terrestrial impact, and not impact on aquatic organisms.

(2) Characterization factors of eutrophication under LIME

Although the characterization factor calculated by the Equation 2.8-1 has been adopted widely in Europe, it is impossible to consider the characteristic that the impact of eutrophication greatly differs depending on the condition of water into which a substance of concern is released. The following problems exist in relation to eutrophication: there are various processes related to environmental response to the substance of concern; and the interrelationships among the physical, biological, and chemical processes that form the ecosystem in a closed water area influence damage in an extremely complicated way and have highly regional characteristics. Therefore, in the impact assessment of eutrophication, it is necessary to identify damage and assess the impact, taking into consideration the main processes between the generation of a substance of concern and the occurrence of damage and the interaction among the processes in relation to the environmental characteristics of the place for release. In addition, with regard to the processes between the generation of a substance of concern and its inflow into water, if Equation 2.8-1 is applied to the eutrophication substances released into the air (nitrogen oxide (NO_x) and ammonia (NH_3)), all the released substances are regarded as substances of concern and it becomes impossible to consider the fate of substances that are thought to differ among countries and regions. In addition, the characterization factor about which the fate analysis in Equation 2.8-2 is taken into consideration is not very useful in Japan because it is based on backgrounds in Europe. Under LIME, because eutrophication is an impact category where regional characteristics have strong influence, the development of the following characterization factors was tried with consideration for regional characteristics in Japan:

1) With regard to the eutrophication substances released into the air (nitrogen oxide and ammonia), add the fate in the air to the value calculated from the average biomass composition ratio [eutrophication potential by fate analysis (EPF)].

2) Use the DO concentration as the midpoint for the impact of eutrophication and add the fate in the air in the case of the substances released into the air [eutrophication potential by material circulation (EPMC)].

With regard to EPF, the source-receptor relationship (SRR) of NO_x and NH_3 calculated during the development of the characterization factors of acidification under this project was used. SRR of acidification gives the ratio of substances that deposit in Japan to the substances released into the air in Japan. On the assumption that the remaining substances deposit in water areas, the characterization factor was calculated on the basis of phosphoric salt by multiplying it by the Redfield ratio. This is based on the assumption that substances that deposited in land areas do not flow into water areas. Among the eutrophication substances, the organic substances' amount of terrestrial deposition through the air is much lower than their amount on the ground. Regarding phosphorus, the amount that is deposited through the air is small, and the outflow in a dissolved state is low because it is strongly absorbed in soil. With regard to phosphorus, the outflow of phosphorus that is deposited into the air is low because, although the substances that are deposited through the air can flow out to water areas as in the case of groundwater pollution by nitrate-nitrogen, nitrogen controls the plant growth rate in general forests.
With regard to EPMC, for the purpose of the assessment of the DO concentration, which is used for the calculation of the damage function of eutrophication, a change in the bottom-layer DO concentration due to a substance of concern was calculated by the below-described flow model and material circulation model. The results of the calculation concerning representative closed sea areas in Japan (Tokyo Bay, Ise Bay, Mikawa Bay, and Osaka Bay) (see Table 2.8-3) were averaged, weighting the amount of each eutrophication substance released in the existing four closed water areas. As a result, the value of a change in the DO concentration due to the release of a unit amount of eutrophication substances was found. After that, characterization factors were calculated by taking into account SRR concerning the substances released in the air (NO_x and NH_3). Table 2.8-4 shows the resultant characterization factors.
With regard to EPF, compared with the value calculated from the average biomass composition ratio (EP), only the substances released into the air were taken into consideration for the ratio of substances depositing in water areas, with the result that the impact became smaller.
The characteristics of the water area to which a substance of concern is released, which are very important for the impact of eutrophication, were taken into consideration for EPMC. The comparison between EPF and EPMC shows that the impact of COD was smaller in the case of EPMC than in the case of EPF, while the impact of nitrogen and phosphorus was larger, which indicates that the impact of organic substances produced in the target closed water area by the use of nutrient salt is larger than the impact of organic substances flowing in from the outside of the water area. Moreover, the comparison between nitrogen and phosphorus shows that the impact of phosphorus is larger in the case of EPMC than in the case of EPF. This reflects the situation where phosphorus's control of photosynthesis by phytoplankton, which produces organic substances in water areas, is stronger than nitrogen's.
Under LIME, the value for which consideration is given to atmospheric fate analysis based on information about Japan and the material circulation characteristics in the target water area (EPMC) is recommendable as the characterization factor of eutrophication.

Table 2.8-3: Results of calculation of a change in the bottom-layer DO concentration due to a change in the load

Tokyo Bay	Bottom-layer DO concentration [mg/L]						
	30%	20%	10%	Current	10%	20%	30%
Case of change in N	2.98000	2.98000	2.98000	2.98	2.98000	2.98000	2.98000
Case of change in P	3.35683	3.24017	3.11380	2.98	2.84233	2.70464	2.57008
Case of change in NP	3.35683	3.24017	3.11380	2.98	2.84233	2.70464	2.57008
Case of change in COD	2.98168	2.98111	2.98056	2.98	2.97943	2.97885	2.97829

Ise Bay	Bottom-layer DO concentration [mg/L]						
	30%	20%	10%	Current	10%	20%	30%
Case of change in N	1.40934	1.40150	1.40073	1.40	1.39926	1.39847	1.39772
Case of change in P	1.42116	1.41446	1.40742	1.40	1.39217	1.38390	1.37799
Case of change in NP	1.42303	1.41580	1.40811	1.40	1.39138	1.38217	1.37244
Case of change in COD	1.40000	1.40000	1.40000	1.40	1.40000	1.40000	1.40000

Mikawa Bay	Bottom-layer DO concentration [mg/L]						
	30%	20%	10%	Current	10%	20%	30%
Case of change in N	2.36290	2.36119	2.36001	2.36	2.35999	2.35998	2.35997
Case of change in P	2.36409	2.36268	2.36131	2.36	2.35872	2.35800	2.35800
Case of change in NP	2.36415	2.36272	2.36133	2.36	2.35872	2.35747	2.35627
Case of change in COD	2.36000	2.36000	2.36000	2.36	2.36000	2.36000	2.36000

Osaka Bay	Bottom-layer DO concentration [mg/L]						
	30%	20%	10%	Current	10%	20%	30%
Case of change in N	2.53274	2.53182	2.53093	2.53	2.52907	2.52818	2.52725
Case of change in P	2.58499	2.56596	2.54799	2.53	2.51200	2.49928	2.47489
Case of change in NP	2.55059	2.54322	2.53641	2.53	2.52353	2.52188	2.50892
Case of change in COD	2.53107	2.53072	2.53035	2.53	2.52965	2.52928	2.52894

Table 2.8-4: Characterization factors of eutrophication

	EP	EPF	EPMC
ammonia (air)	0.35	0.15	0.092
ammonia (water)	0.35	0.35	0.21
ammonium	0.33	0.33	0.20
nitrate	0.1	0.1	0.059
nitrogen	0.42	0.42	0.26
nitrogen dioxide (air)	0.13	0.018	0.011
nitrogen monoxide (air)	0.2	0.028	0.017
phosphate	1.0	1.0	1.0
phosphorus	3.06	3.06	3.06
chemical oxygen demand	0.022	0.022	0.0015

EP: calculated from the average composition ratio of biomass (Heijungs et al. 1992)
EPF: calculated from the average composition ratio of biomass with consideration for SRR
EPMC: calculated from decrease in the DO concentration in closed inner bays in Japan with consideration for SRR

2.8.3 Eutrophication damage assessment

This part describes the damage factors (damage functions) calculated for the endpoint approach.

(1) Basic policy for calculation of damage factors

The calculation of eutrophication damage requires assuming a specific water area, because, as described above, the impact of eutrophication greatly differs among water areas according to their regional characteristics. Under LIME, a damage function was calculated for four typical closed water areas in Japan – Tokyo Bay, Ise Bay, Mikawa Bay, and Osaka Bay.

Table 2.8-5 shows the category endpoints of eutrophication and the objects of the calculation of the damage functions under LIME. In addition, Figure 2.8-5 shows a flowchart of the calculation of damage functions.

Although the impact of eutrophication varies, under LIME, attention was paid to lack of oxygen as an effect of eutrophication, and the number of kinds of benthic species, which are directly influenced by lack of oxygen the most, and a decrease in the current amount of benthic species was dealt with as concrete damage. Moreover, the impact on fishery production was estimated from a decrease in the current amount of benthic species. Because effects on the other endpoints – recreation function and water utilization function – have not been fully discussed yet, they are not dealt with herein.

Under LIME, when these estimations were carried out, the effects were calculated by dividing a series of processes where complicated interactions are thought to exist into the following stages, for the sake of convenience: the process from the generation of a substance of concern to its flowing into a water area; changes in material dynamics in the water area and the DO concentration in water; and the response of benthic organisms to the change.

Table 2.8-5: Category endpoints of eutrophication and the objects of calculation under LIME

Object of protection	Category endpoint		Object of calculation of damage function	
Human health	Temporary impact on human health is small.		—	No object
Social assets	Water utilization	Obstacle to water utilization due to unusual smell/taste caused by red tide, etc.	—	Quantitative assessment is difficult.
	Recreation function	Obstacle to hydrophilic function due to decreasing transparency, unusual taste, etc.	—	Quantitative assessment is difficult.
	Fishery production	Damage to fisheries and culture due to a decrease in fishery resources through influence of generation of suboxic water mass on benthic organisms	○	Fishery production in closed water areas as a result of lack of oxygen
Primary production	Aquatic ecosystem	Change in primary production of phytoplankton (usually, an increase)	△	Impact on the current amount of benthic organisms (impact on higher-order production)
Biodiversity	Aquatic ecosystem	Impact on the structure of aquatic species	△	Impact on the number of kinds of aquatic species

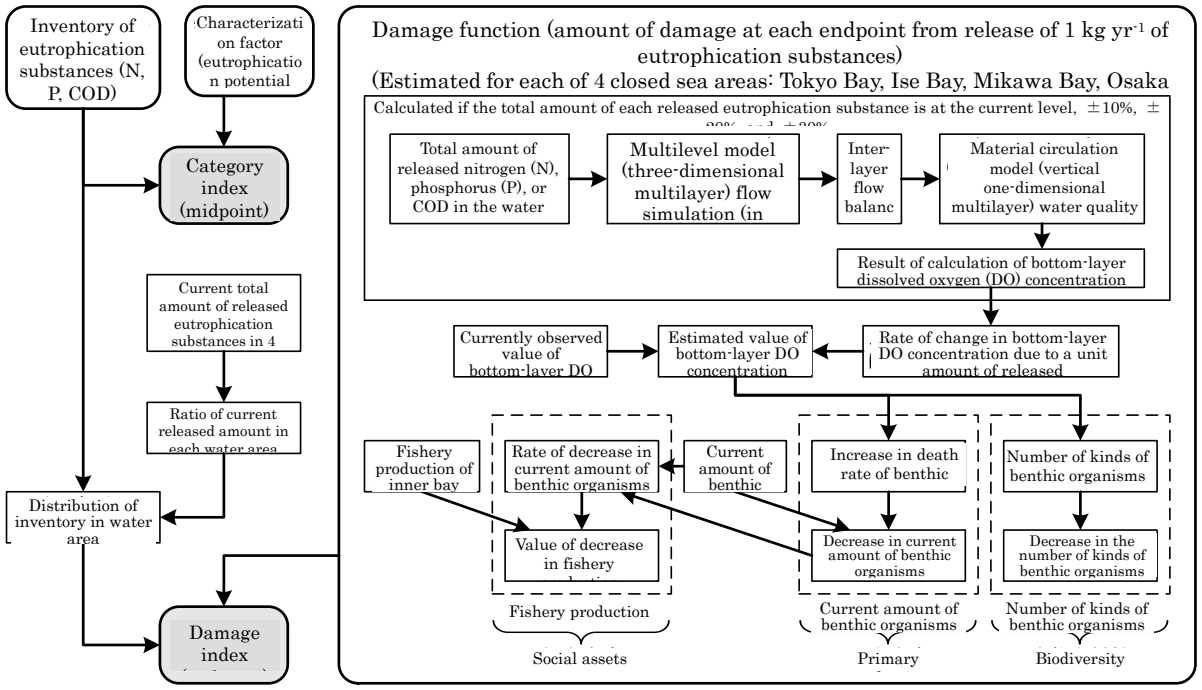


Figure 2.8-5: Flowchart of estimation of eutrophication damage functions

(2) Process from the generation of a substance of concern to its flowing into a closed water area

In the process from the generation of a substance of concern to its flowing into a closed water area, the amount of organic substances, for which COD is used as the index, changes according to production, decomposition, mineralization, etc., and the forms of nitrogen and phosphorus also change in various manners. The ratio of the amount of a substance of concern generated in land to the amount of the substance of concern flowing into water is called the reaching rate. It is thought that the reaching rate greatly changes according to geographical conditions and weather conditions, such as rainfall. However, there are almost no data for examination of such changes. Therefore, in this study, the reaching rate adopted by the Ministry of the Environment is used for COD. With regard to nitrogen and phosphorus, no consideration is given to the reaching rate, because if their forms change, the whole amount rarely changes like COD, and most of the amount reaches water sooner or later.

According to the Ministry of the Environment's data, the ratio of the amount of reaching COD to the amount of generated COD was 80.2% in Tokyo Bay, 94.9% in Ise Bay, and 96.4% in Osaka Bay on average for the 14 years from 1979 to 1992.

(3) Changes in material dynamics in a closed water area and the DO concentration in water

Prediction of the DO concentration in a closed water area requires expression of material dynamics with consideration for physical, biological, and chemical processes within the above-mentioned water areas. Because of this, the flow balance in the inner bays was calculated by a flow simulation, using a numerical simulation model that combined the "flow model" for expressing the physical process with the "material circulation model (water quality model)" for expressing the biological and chemical processes (Nakata 1993). Based on the result, the bottom-layer DO concentration was calculated by a material circulation simulation.

a Flow simulation

The “multilevel model” was adopted as the flow model. It took into consideration the flow driven by wind above sea level, difference in water density, and tides, all of which are important for the flow in an inner bay. This model consists of a group of equations that describe 1) the movement of fluids in an inner bay and the mouth of a river; 2) continuity of fluids; 3) changes in the tidal level; 4) dispersion of chlorinity; 5) dispersion of heat; and 6) relationships among seawater density, chlorinity, and water temperature. The model was partitioned by 1 km × 1 km horizontally and by intervals of several meters vertically.

With regard to each water area, geographical features, water depth, tides, and weather conditions were inputted and flow simulation was carried out for the summer season, when the DO concentration seems to decrease more greatly than in the other seasons. Based on the result of the simulation, the interlayer flow balance was calculated.

Figure 2.8-6 shows the result of the flow simulation in Tokyo Bay in summer. The figure reproduces the estuary circulation – that is, water flowing from rivers flows out to the mouth of the bay from the upper layer and flows into the closed-off section of the bay from the lower layer around the line between Kawasaki/Yokohama and Kisarazu.

b Water quality simulation

The ecosystem material circulation model was adopted as the water quality model. It expresses the dynamics of carbon, nitrogen, and phosphorus among phytoplankton, zooplankton, detritus (non-living particulate organic substances), dissolved organic substances, and nutrient salt in a water area, taking into consideration the relations with oxygen generation and consumption. The water quality model was not partitioned horizontally, and consideration was given only to the vertically divided layers (box multilayer model).

The initial value was the observed value of water quality in each layer. A water quality simulation was carried out by inputting the flow balance between layers and between the inside and outside of each bay through a flow simulation and the load of eutrophication substances in each water area into the water quality model. The validity of the simulation was examined by comparison between the simulation results and the observed values.

Recently, there has been developed a model that can assess the interaction between the material circulation in neritic waters, including tidelands and seaweed bends, and the material circulation in inner bay sediments and the inner bay floating material circulation and can assess changes in time (Sohma et al. 2001, Sohma et al. 2002, Sohma et al. 2004). In this study, because of lack of data, consideration was not given to the interaction between the material circulation in neritic waters and that in sediments and changes in time.

Figure 2.8-7 shows the result of the water quality simulation in Tokyo Bay together with observed data during the target calculation period. It was judged from the comparison between the calculation results and the observed values that although detailed examination is difficult due to wide fluctuation in observed values, the calculation results show a trend in vertical changes and reproduce the current water quality trends.

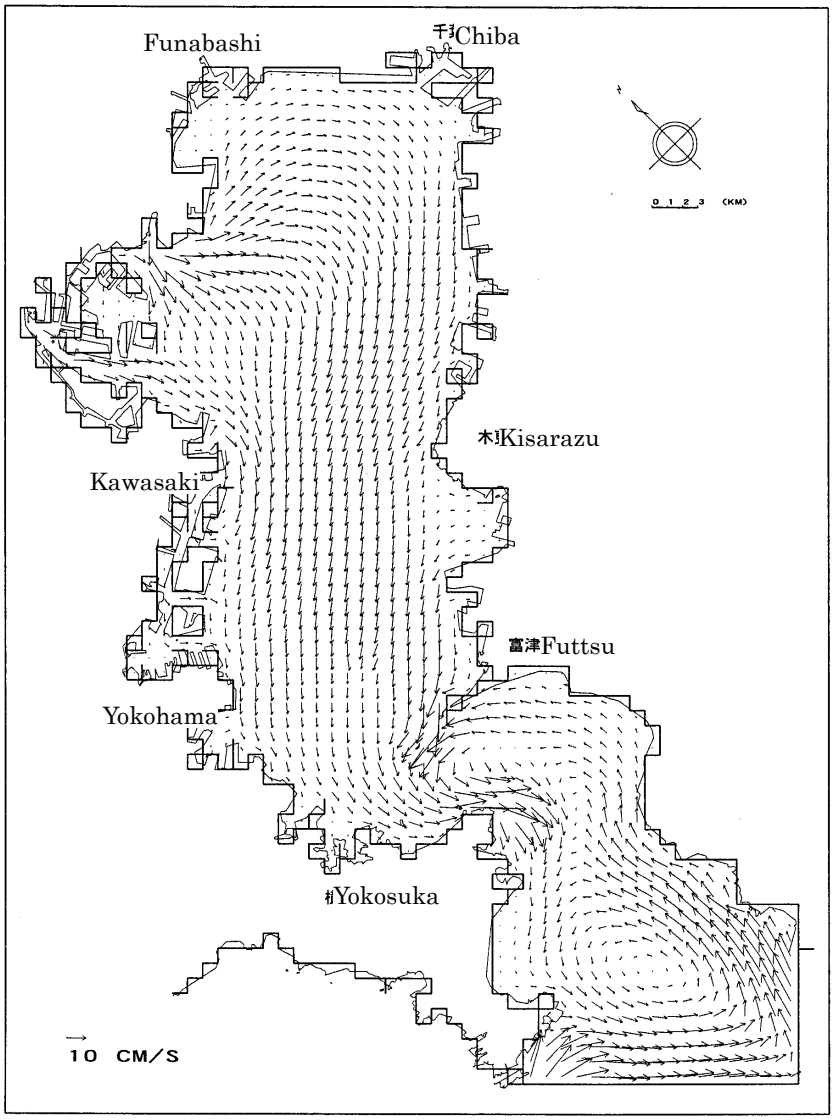


Figure 2.8-6 (1) Flow simulation result (Tokyo Bay, surface layer)
The direction and size of a vector show the direction and speed of the average flow in summer.

c Relation between the amount of released eutrophication substances and the bottom-layer DO concentration

To calculate the DO concentration in each layer, a simulation was carried out to change the load of nitrogen, phosphorus, and COD to the current level, ±10%, ±20%, and ±30%. Based on the result, the rate of a change in the bottom-layer DO concentration due to release of a unit amount of eutrophication substances was calculated.

However, conditions for the layer division of the water qualification model differ in reality, although they are the same for the purpose of calculation. For example, some layers are in contact with the sea bottom, while other layers are not in contact with it, depending on geographical features. Moreover, because the result of calculation of the DO concentration by the water quality model is the calculated average value within each layer, it is different from the actual value at places where layers are in contact with the sea bottom and therefore the impact of a DO decrease on benthic organisms becomes a problem.

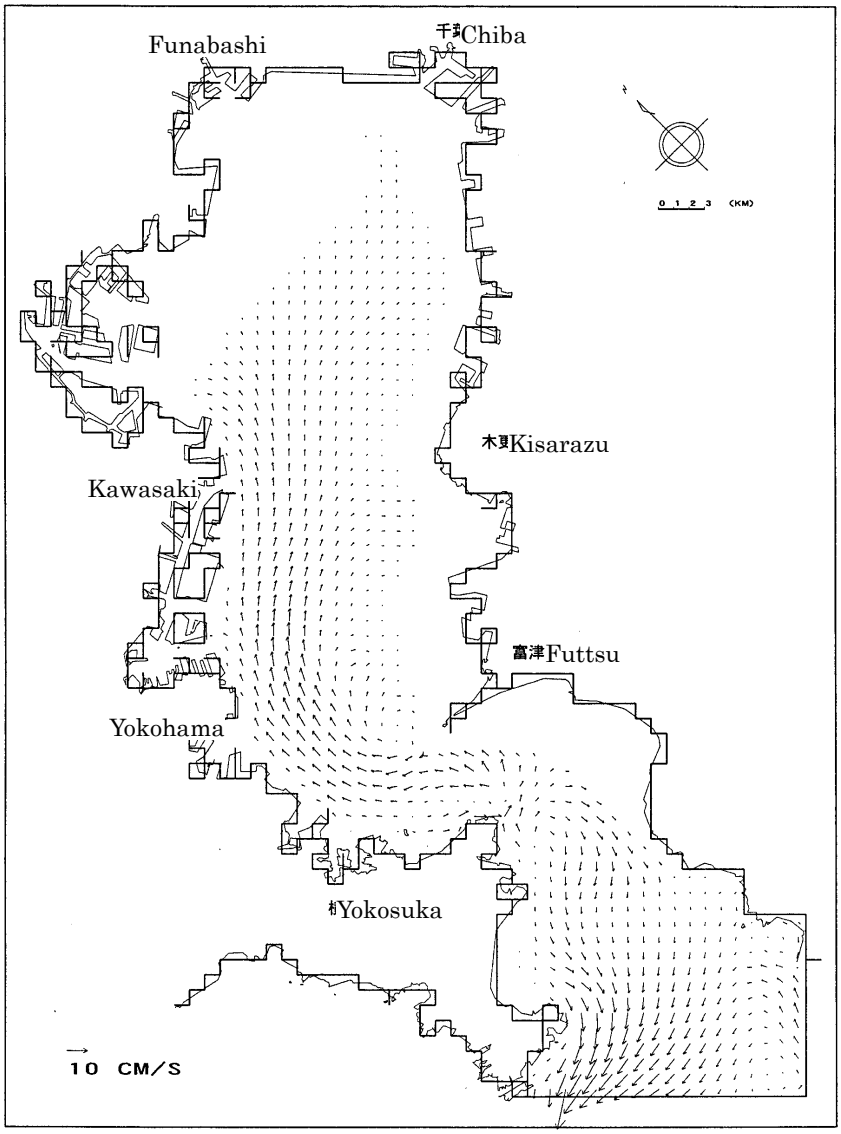


Figure 2.8-6 (2) Flow simulation result (Tokyo Bay, bottom layer)
The direction and size of a vector show the direction and speed of the average flow in summer.

Because of this, a change in the bottom-layer DO concentration due to release of eutrophication substances was calculated by multiplying the currently observed value of the DO concentration in the water just above the sea bottom by the DO concentration change rate found from the water quality simulation (result of DO simulation at the time of a change in the load/result of Do simulation in the case of the current load).

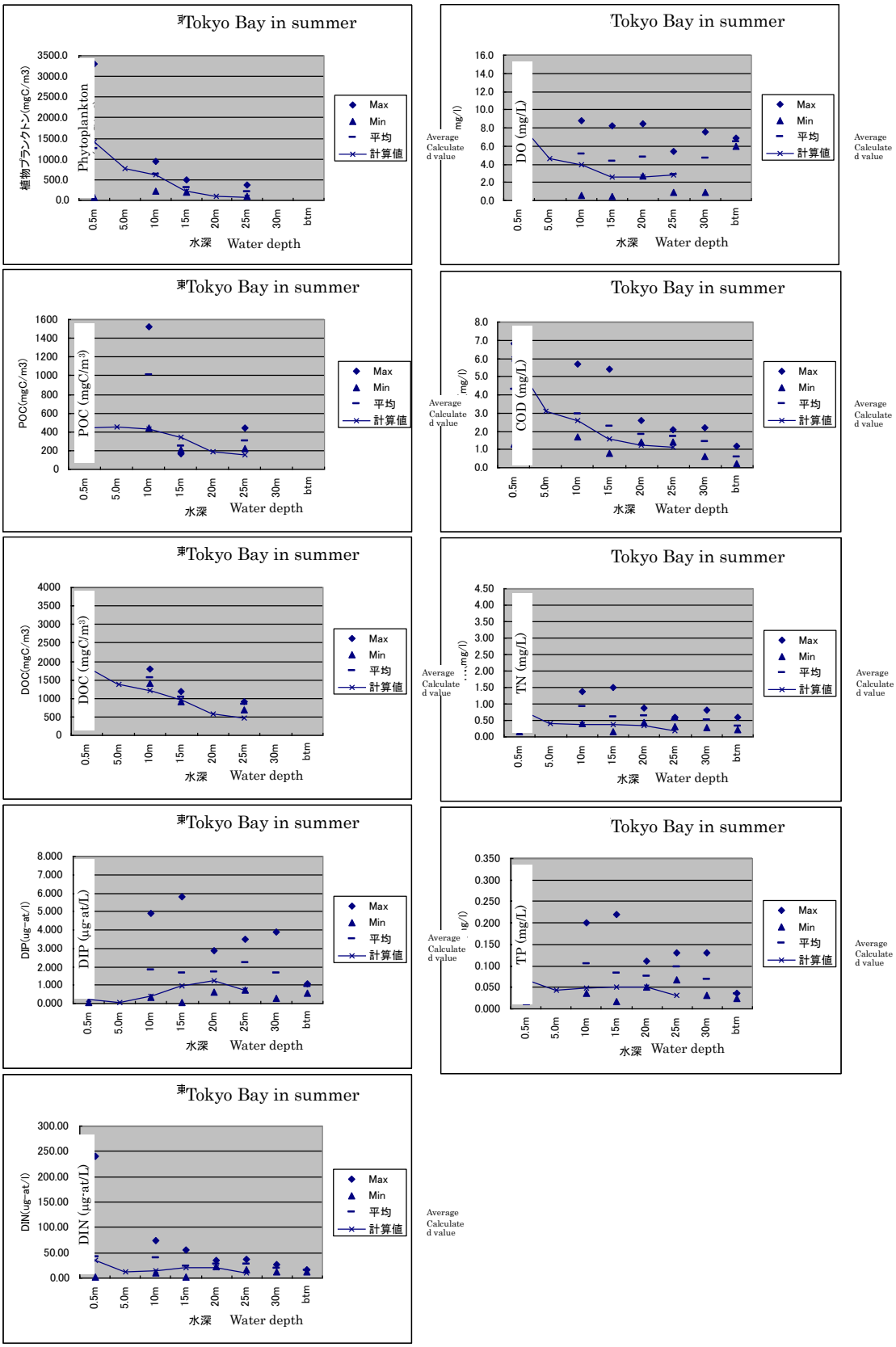


Figure 2.8-7: Results of water quality simulation in Tokyo Bay and observed values

POC: particulate organic carbon; DOC: dissolved organic carbon; DIP: dissolved inorganic phosphorus; DIN: dissolved inorganic nitrogen; DO: dissolved oxygen; COD: chemical oxygen demand; TN: total nitrogen; TP: total phosphorus

Column 2.8-4

Flow model

The movement and mixing of water and substances in the sea are expressed by motion equations and continuity equations that include items that express momentum, heat, and transport of substances. The multilevel barocline flow model adopted here is widely used for various kinds of assessment in coastal areas.
The basic equations for the multilevel barocline flow model are the below-described six equations concerning state quantities (momentum, flow, mass, heat conservation law, and state equation). The coordinate system of these equations consists of the *x*-*y* axis for mean sea level and the *z* axis for the vertical direction.
Because the vertical scale for an inner bay area is sufficiently smaller than the horizontal scale, it is assumed that the acceleration and the viscosity in the vertical movement equation are ignorable compared with gravitational acceleration (hydrostatic approximation). In addition, because changes in density are sufficiently small, the Boussinesq approximation is assumed, which ignores changes in density other than buoyancy and enables the application of a continuity equation for an incompressible fluid.

Motion equation for direction *x*

$$\frac{\partial u}{\partial t} = -\frac{\partial}{\partial x}(u^2) - \frac{\partial}{\partial y}(uv) - \frac{\partial}{\partial z}(uw) + f_0 v - g\frac{\partial \zeta}{\partial x} - \frac{g}{\rho}\int_z^0 \frac{\partial \rho}{\partial x}dz - \frac{1}{\rho}\frac{\partial P_0}{\partial x} + \frac{\partial}{\partial x}(N_x\frac{\partial u}{\partial x}) + \frac{\partial}{\partial y}(N_y\frac{\partial u}{\partial y}) + \frac{\partial}{\partial z}(N_z\frac{\partial u}{\partial z})$$

Motion equation for direction *y*

$$\frac{\partial v}{\partial t} = -\frac{\partial}{\partial x}(uv) - \frac{\partial}{\partial y}(v^2) - \frac{\partial}{\partial z}(vw) + f_0 u - g\frac{\partial \zeta}{\partial y} - \frac{g}{\rho}\int_z^0 \frac{\partial \rho}{\partial y}dz - \frac{1}{\rho}\frac{\partial P_0}{\partial y} + \frac{\partial}{\partial x}(N_x\frac{\partial v}{\partial x}) + \frac{\partial}{\partial y}(N_y\frac{\partial v}{\partial y}) + \frac{\partial}{\partial z}(N_z\frac{\partial v}{\partial z})$$

Continuity equation

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} + \frac{\partial w}{\partial z} = 0$$

Heat balance equation (temperature diffusion equation)

$$\frac{\partial T}{\partial t} = -\frac{\partial}{\partial x}(uT) - \frac{\partial}{\partial y}(vT) - \frac{\partial}{\partial z}(wT) + \frac{\partial}{\partial x}(k_x\frac{\partial T}{\partial x}) + \frac{\partial}{\partial y}(k_y\frac{\partial T}{\partial y}) + \frac{\partial}{\partial z}(k_z\frac{\partial T}{\partial z})$$

Chlorine balance equation (chlorine diffusion equation)

$$\frac{\partial Cl}{\partial t} = -\frac{\partial}{\partial x}(uCl) - \frac{\partial}{\partial y}(vCl) - \frac{\partial}{\partial z}(wCl) + \frac{\partial}{\partial x}(K_x\frac{\partial Cl}{\partial x}) + \frac{\partial}{\partial y}(K_y\frac{\partial Cl}{\partial y}) + \frac{\partial}{\partial z}(K_z\frac{\partial Cl}{\partial z})$$

State equation

$$\rho = \rho(Cl, T)$$

The following are the variables and signs used in these equations:
u, v, w: components of flow speed in directions *x, y, z* [cm/sec]
ζ: displacement between average water surface and free water surface (tide level) [cm]
H: height between average water surface and sea bottom [cm]
ρ: fluid density [g/cm^3]
fo: Coriolis parameter [sec^{-1}]

g: gravitational acceleration [cm/sec^2]
P_0: atmospheric pressure [g/cm·sec^2]
T: water temperature [°C]
Cl: chlorinity
N_x, N_y, N_z: eddy viscosity coefficients in directions x, y, z
k_x, k_y, k_z: temperature diffusion coefficients in directions x, y, z
K_x, K_y, K_z: substance diffusion coefficients in directions x, y, z

Column 2.8-5

Floating material circulation model

Because many organisms floating in water are transported by flow or diffused by disturbance, their distribution greatly depends on the processes of fluid dynamics. In addition, the distribution of nutrient salts necessary for the propagation of phytoplankton also greatly depends on the process of diffusive mixing, a process of fluid dynamics. Because the processes of fluid dynamics greatly influence the ecosystem, it is necessary to formularize the interaction between the two systems. To describe temporal changes in the current volume B of a compartment (a component of the model) at an arbitrary point in a water area (x, y, z), the material circulation model shows a material balance equation as follows:

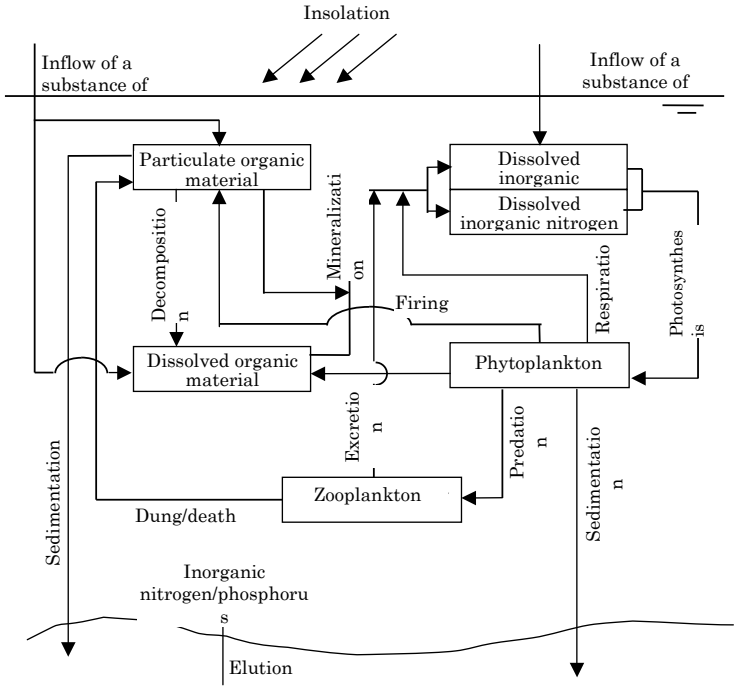
$$\frac{\partial B}{\partial t} = -u\frac{\partial B}{\partial x} - v\frac{\partial B}{\partial y} - w\frac{\partial B}{\partial z} \quad \text{(Advection)}$$

$$+ \frac{\partial}{\partial x}(K_x\frac{\partial B}{\partial x}) + \frac{\partial}{\partial y}(K_y\frac{\partial B}{\partial y}) + \frac{\partial}{\partial z}(K_z\frac{\partial B}{\partial z}) \quad \text{(Diffusion)}$$

$$+ (\frac{\partial B}{\partial t}) \quad \text{(Biological/chemical process)}$$

In this equation,
B: current volume of each compartment (see the table below)
u, v, w: components of flow speed in directions x, y, z
K_x, K_y, K_z: substance diffusion coefficients in directions x, y, z

Compartment	Sign	Unit
Phytoplankton	P	mgC/m^3
Zooplankton	Z	mgC/m^3
Particulate organic	POM	mgC/m^3
Dissolved organic	DOM	mgC/m^3
Dissolved inorganic	DIN	μg–atm/L
Dissolved inorganic	DIP	μg–atm/L
Dissolved oxygen	DO	mg/L
Chemical oxygen	COD	mg/L



Biological and chemical processes have been formularized for each compartment. If the coefficient for the speed of a change process is v ( ), the coefficient for limitation on the speed is μ ( ), and a factor function is entered in the parentheses, the change process for phytoplankton, for example, can be expressed as described below. For detailed formularization of each elementary process, see Nakata K. (1993), etc.

$$\frac{dP}{dt} = v_1(T)\cdot\mu_1(DIN,DIP)\cdot\mu_2(I,P)\cdot P \ [\text{propagation by photosynthesis}]$$
$$-\mu_3(P)\cdot\{v_1(T)\cdot\mu_1(DIN,DIP)\cdot\mu_2(I,P)\cdot P\} \ [\text{extracellular secretion}]$$
$$-v_3(T)\cdot P \ [\text{respiration}] - v_4(T,P)\cdot Z \ [\text{predation by zooplankton}]$$
$$-v_5(T)\cdot P \ [\text{firing}] \qquad -w_P\frac{\partial P}{\partial z}[[\text{sedimentation}]$$

In this equation,

P: current amount of phytoplankton
T: Water temperature
DIN, DIP: dissolved inorganic nitrogen, dissolved inorganic phosphorus
I: light intensity
Z: current amount of zooplankton
w_p: sedimentation speed

(4) Biodiversity: damage function to the number of kinds of benthic organisms

To quantify the impact of suboxidation on the number of kinds of benthic organisms, the Tokyo Metropolitan Government carried out a survey on benthic organisms (from FY1988 to FY1997). Based on the resultant data, it was examined how the bottom-layer DO concentration is related with the number of kinds of generated benthic organisms. Regression analysis was carried out by a quadratic polynomial, using the data on the bottom-layer DO concentration of less than 6.0 mg/L (Figure 2.8-8). The analysis excluded four tidal lands where the impact of a decrease in the aquatic DO concentration is small

because they are exposed to the air at regular intervals. Moreover, consideration was given to the fact that if the DO concentration is more than 6 mg/L, no living organisms receive impact (Matsukawa 1992).

$$y = 0.3746\,x^2 + 2.0068\,x + 0.9831 \quad (R^2 = 0.4669,\ \mathrm{n} = 184) \qquad (2.8\text{-}3)$$

In this equation, y and x are the number of kinds of generated benthic organisms and the bottom-layer DO concentration [mg/L], respectively.

The damage function that indicates the rate of decrease in the number of kinds of benthic organisms was calculated by assigning the rate of change in the bottom-layer DO concentration due to release of a unit amount of eutrophication substances to Equation 2.8-3.

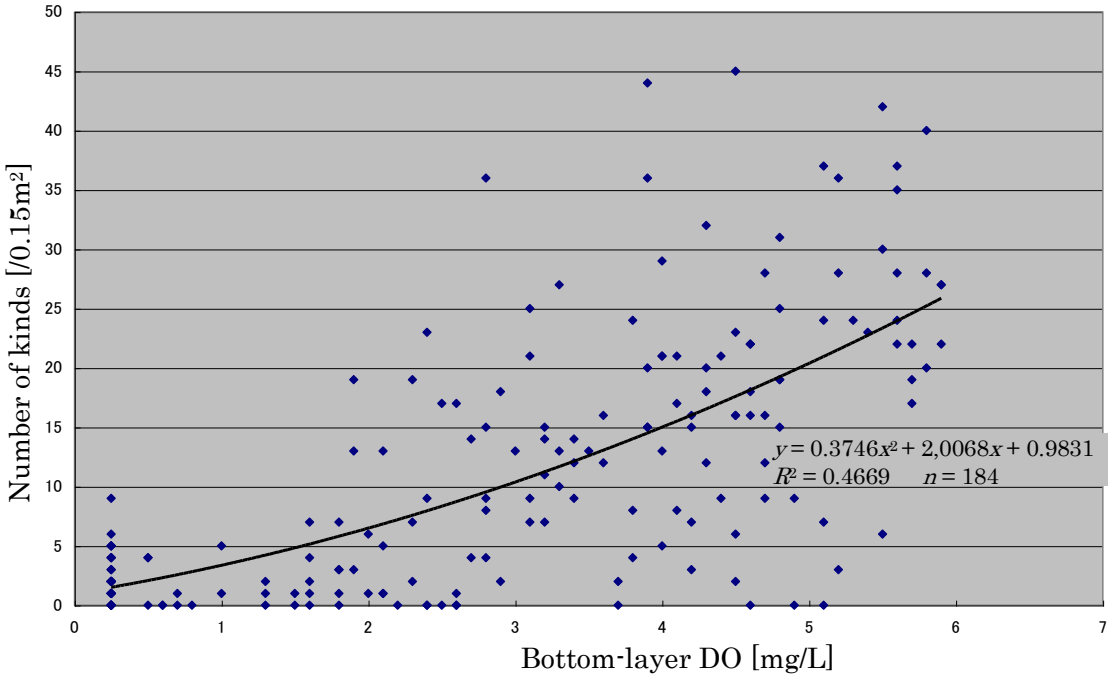


Figure 2.8-8: Relation between bottom-layer DO and the number of kinds of benthic organisms according to the Tokyo Metropolitan Government's data
Data for May and September during the decade from 1988 to 1997

(5) Primary production: damage function for the current amount of benthic organisms

The relation between the release of eutrophication substances and the damage to the current amount of benthic organisms was estimated as the damage function for biological production.

As the basis for quantifying the impact of suboxidation on the current amount of benthic organisms, a case in Holland (Baretta et al. 1988) was selected where the DO saturation (the ratio of the actual amount of dissolved oxygen to the potential amount) and the death speed of each category of benthic organisms were formularized.

$$W / B = mor \cdot (1 - \min(1.0, ROX/qox)) \qquad (2.8\text{-}4)$$

In this equation, *W, B, mor, ROX,* and *qox* are the amount of dead benthic organisms, the amount of existing benthic organisms, the relative death speed of benthic organisms, the DO saturation, and the level of Do saturation under which death occurs, respectively.

Under LIME, the model for which Suzuki et al. formularized *qox* (Suzuki et al. 1998) was used. Macrobenthos, meiobenthos, and aerobic layer bacteria were taken into consideration as categories of benthic organisms.

$$qox = 0.7 \cdot \left\{ (T / ks)^{a} \,/\, (1 + (T / ks)^{a}) \right\} \qquad (2.8\text{-}5)$$

In this equation, *T*, *ks,* and *a* are the water temperature [°C], the half-value constant (=25°C), and the index around the half-value constant, respectively. The value *a* for each category is 20 for macrobenthos, 5 for meiobenthos, and 10 for aerobic layer bacteria. Moreover, *mor* is 1.0 day^{-1} for all categories (If the DO concentration is zero, this means that all organisms die in one day).

The damage function was calculated by assigning the rate of change in the bottom-layer DO concentration due to release of a unit amount of eutrophication substances to Equations 2.8-4 and 2.8-5 to find the rate of decrease in the current amount of benthic organisms of each category and summing up the products of the results and the current amount of benthic organisms of each category.

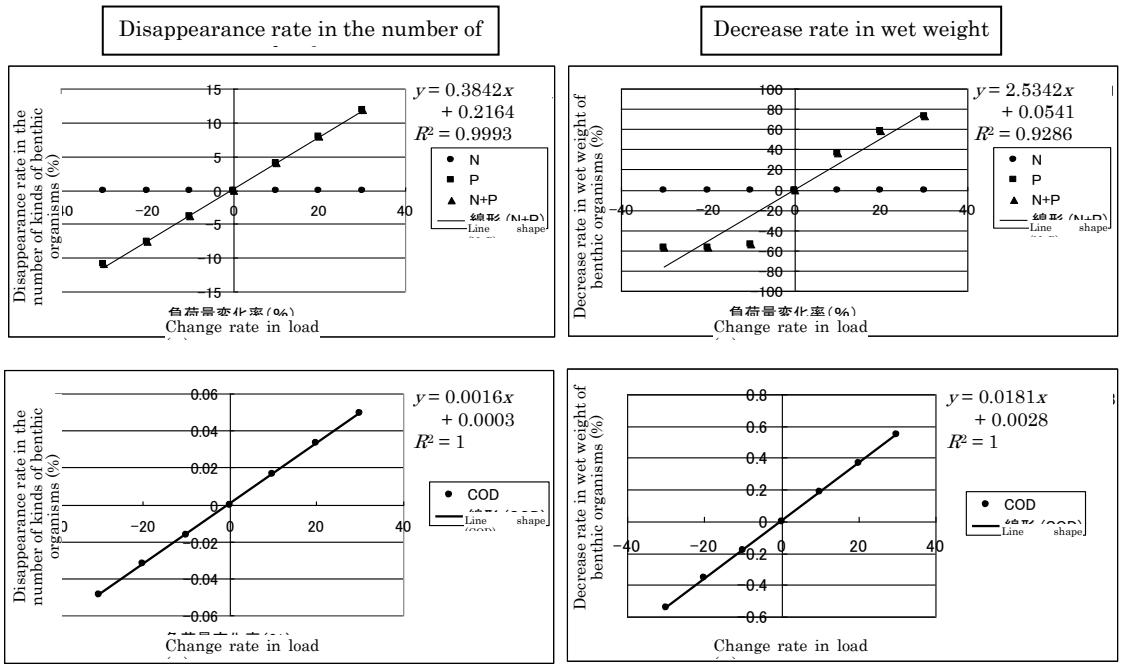


Figure 2.8-9: Changes in the number of kinds and wet weight of benthic organisms according to load change (Tokyo Bay)
N: change in nitrogen only; P: change in phosphorus only;
N+P: simultaneous change in nitrogen and phosphorus; COD: change in COD only

Figure 2.8-9 shows the results of calculation in Tokyo Bay concerning changes in the number of kinds and wet weight of benthic organisms on the assumption that the load of nitrogen, phosphorus, and COD is at the current level, ±10%, ±20%, and ±30%. The results of the

calculation show that an increase in the load results in a decrease in both the number of kinds and the current amount of benthic organisms and that the inflow of phosphorus has stronger impact than the inflow of nitrogen.

(6) Social assets: damage function for fishery production

Of the fishery species caught in Tokyo Bay, Ise Bay, Mikawa Bay, and Osaka Bay, damage to benthic organisms and fishes for which inner bays are important habitats was calculated.

It was assumed that the impact of lack of oxygen on benthic organisms is almost the same as the impact on fishes that strongly depend on the inner bay environment, because the impact strongly influences the ecosystem in the whole inner bay. That is, the degree of damage to fishery production due to release of a unit amount of eutrophication substances was regarded as the same as the decrease rate calculated by the damage function for the current amount of benthic organisms.

The target of calculation of damage function was fishery species caught in the inner bays. However, fishes that temporarily flow into an inner bay and eat plankton, such as sardines, are excluded from the target, in view that they do not depend on the inner environment much and are unlikely to receive the impact of eutrophication. The following fishery species seem to receive the impact:

> flatfish, sea bream, striped mullet, sea bass, conger, greenling, sand borer, gizzard shad, grunt, goby, synodontidae, conger pike, Spanish mackerel, sand lance, puffer, deep-sea smelt, halfbeak, shrimp, crab, shellfish, squid, octopus, sea cucumber, squilla, and other fishery species

With regard to these fishery species, information on catches and fishery values was obtained from the fishery statistical data (catches, fishery value, and unit price of each kind of fish) of the Kanto, Chubu, and Kinki Agricultural Administration Offices. Because statistical data on catches are divided not according to water area but according to area to which fishery businesses that caught aquatic organisms belong (catch accounting for production unit), it was assumed, based on data about each fishery business or municipality, that aquatic organisms were caught in surrounding areas.

The damage function for fishery production was calculated by multiplying the damage function, which indicates the decrease rate in the current amount of benthic organisms due to release of a unit amount of eutrophication substances, by catches.

(7) Arrangement of damage functions of eutrophication

Damage functions were obtained for each of the four closed sea areas. The damage functions of eutrophication are results of weighting the damage functions for the four areas by the current amount of release of each eutrophication substance in the four areas. Figure 2.8-10 shows the damage functions for fishery production (social assets). With regard to the substances released into the air, the SSR of the NO_x and NH_3 were calculated for the development of the characterization factors of acidification under LIME. With regard to biodiversity and primary production, because they were consistent with the other impact categories in terms of the calculated number of kinds and current amount of benthic organisms, they are not reflected in the damage functions of eutrophication.

Damage per load of 1 kg [× 10^{-6}%]

		Disappearance rate in the number of kinds of benthic				Decrease rate in the wet weight of benthic			
		Tokyo Bay	Ise Bay	Mikawa	Osaka Bay	Tokyo Bay	Ise Bay	Mikawa	Osaka Bay
NH_3	ammonia (air)	0.133	0.025	0.010	0.008	0.88	0.15	0.09	0.08
NH_3	ammonia (water)	0.308	0.059	0.023	0.019	2.03	0.35	0.20	0.18
NH_4^+	ammonium	0.291	0.055	0.021	0.018	1.92	0.33	0.19	0.17
NO_3^-	nitrate	0.085	0.016	0.006	0.005	0.56	0.10	0.06	0.05
T–N	nitrogen	0.375	0.071	0.027	0.023	2.47	0.42	0.25	0.22
NO_2^-	nitrogen dioxide (air)	0.016	0.003	0.001	0.001	0.11	0.02	0.01	0.01
NO	nitrogen monoxide (air)	0.024	0.005	0.002	0.002	0.16	0.03	0.02	0.01
PO_4^{3-}	phosphate	1.493	0.214	0.082	0.112	9.85	1.27	0.74	1.08
T–P	phosphorus	4.577	0.655	0.252	0.344	30.19	3.89	2.26	3.30
COD	chemical oxygen demand	0.00153	0.00000	0.00000	0.00154	0.0173	0.0000	0.0000	0.0146

Model calculation results

Damage functions of eutrophication (fishery production)

	Eutrophication-contributing	yen/kg
NH_3	ammonia (air)	2.93E+01
NH_3	ammonia (water)	6.79E+01
NH_4^+	ammonium	6.41E+01
NO_3^-	nitrate	1.86E+01
T–N	nitrogen	8.25E+01
NO_2^-	nitrogen dioxide (air)	3.51E+00
NO	nitrogen monoxide (air)	5.39E+00
PO_4^{3-}	phosphate	3.18E+02
T–P	phosphorus	9.74E+02
COD	chemical oxygen demand	6.40E−01

Total annual fishery production of target species likely to receive damage from eutrophication (millions of yen)

Tokyo	Ise Bay	Mikawa Bay	Osaka
6784	14308	11643	6236

Estimated from data of each Regional Agricultural Administration Office

Estimated damage in each water area

Weighting the results for the four water areas by the current ratio of generated load

Figure 2.8-10: Damage functions of eutrophication (fishery production)

The source-receptor relationship (SRR) of NO*x* and NH_3 calculated during the development of the characterization factors of acidification was applied to substances released to the air.

2.8-4 Procedures for impact assessment of eutrophication

The concrete procedures for characterization of eutrophication and assessment of damage are as follows. With regard to characterization, Equation 2.8 is used to find the category index *CI* from the inventory *Inv* (*X*) of each eutrophication substance *X* and $CF^{Eutrophication}$ (*X*):

$$CI^{Eutrophication} = \sum_{X} CF^{Eutrophication}(X) \cdot Inv(X) \qquad (2.8\text{-}6)$$

Several suggestions have so far been made concerning $CF^{Eutrophication}$ (*X*). Under LIME, among them, the characterization factor EPMC was recommended, which is based on the environmental conditions in Japan and gives consideration to atmospheric fate analysis and aquatic material circulation characteristics.

The index for the endpoint approach – that is, the damage index *DI* (*Safe*) – can be obtained from *Inv* (*X*) of each eutrophication substance *X* and the $DF^{Eutrophication}$ (*Safe, X*) for each object of protection *Safe* (only social assets by fishery production herein) (the Equation 2.8-7).

Moreover, the integrated index *SI* can be obtained by the use of the integration $IF^{Eutrophication}(X)$ into which the impact on social assets is economically converted or non-dimensionalized (the Equation 2.8-8).

$$DI(Safe) = \sum_{X} DF^{Eutrophication}(Safe, X) \cdot Inv(X) \quad (2.8\text{-}7)$$

$$SI = \sum_{X} IF^{Eutrophication}(X) \times Inv(X) \quad (2.8\text{-}8)$$

The characterization factor $CF^{Eutrophication}(X)$, the damage factor $DF^{Eutrophication}(Safe, X)$, and the integration factor $IF^{Eutrophication}(X)$ have been attached hereto as A1, A2, and A3, respectively.

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2.9 Indoor air pollution

2.9.1 What phenomenon is indoor air pollution?

(1) Background factors to the increasing appearance of indoor air pollution

There are three main background factors to indoor air pollution. One of them is problems in houses. Japanese houses have traditionally been devised to be ventilated well, because importance has been placed on the summer season. Therefore, the amount of ventilation is extremely large, resulting in almost no indoor air pollution or sick house problem. Recently, however, the improvement of indoor environments and energy conservation has required super-insulated houses. As a result, although indoor environments have been sharply improved, drafts have decreased, and wood, paper, and soil have been replaced with new construction materials. This has resulted in the production of a large quantity of pollutants. In addition, because of a decrease in the amount of ventilation, the pollutants produced in open combustion appliances and mold caused by moisture have become tangible problems.

The second background factor is lifestyle changes. If a house is next to another house or faces a noisy road, it is difficult to open windows even in the summer season. In addition, chemicals are often used for living ware.

The third background factor is problems in human bodies. While in the past few people suffered hay fever or allergies, recently many people have suffered them. It is said that such diseases occur if the amount of pollutants accumulated in the body through respiration or food exceeds a certain level. Because, as described above, a large amount of chemicals has been brought into our lives, every time people breathe or have a meal, they accumulate pollutants in their bodies. With the worsening of air pollution, the amount of chemical pollutants accumulated in bodies has been increasing. Moreover, when houses are built, people inhale a lot of chemical pollutants, which may trigger the occurrence of disease. In addition, pollutants accumulated in human bodies are thought to be concentrated between generations. If so, a child born from a mother who has accumulated pollutants in her body has a certain amount of pollutants even at the birth. The number of people suffering allergies has recently increased as a result of an increasing number of people who have become sensitive to chemicals because they had already accumulated pollutants at birth or have accumulated a lot of pollutants due to their proneness to pollution by chemicals around them.

(2) Causes of indoor air pollution damage

The main causes of indoor air pollution are not only volatile organic compounds, formaldehyde, and other toxic chemicals generated from construction materials and furniture but also carbon dioxide (CO_2) generated from human bodies, nitrogen oxides (NO_x) generated from open combustion appliances, sulfur oxides (SO_x), and cigarette smoke. In rooms, there are various other sources of pollution that pollute indoor air.

People take in substances through various activities, such as eating and drinking. The amount of indoor air taken in through respiration is the largest and accounts for nearly 60% of the total amount of substances taken in. The amount of indoor air taken in is overwhelmingly larger than the amount of food and water taken in. That is, it can be said that indoor air has great influence on human health. However, while people are sensitive to muddy water and rotten food, they tend to be indifferent to dirty air. Because, unlike the

case of water, changes in the properties of air cannot be seen, people tend to be careless toward worsening of air quality, which is said to be one of the serious causes of indoor air pollution damage.

(3) Endpoints of indoor air pollution

The object of protection from indoor air pollution is limited to "human health." Although damage to some "social assets" can be imagined – for example, a decrease in the monetary value of a building where indoor air pollution occurs – because such damage is not general, it is not examined herein.
As there are various indoor air pollutants, various types of health damage occur due to exposure to these pollutants, such as sick house syndrome, allergies, and respiratory diseases. With regard to some pollutants, the cause-effect relationship with health damage has still not been clarified. In this study, an assessment method was developed concerning health damage that is caused by main pollutants and about which sufficient quantitative information can be obtained. Figure 2.9-1 shows the substances and health damage covered by the assessment.

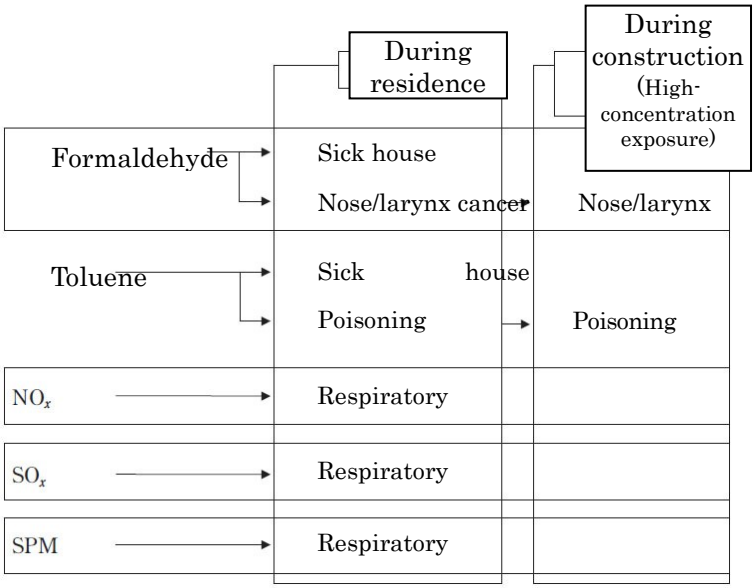


Figure 2.9-1: Target indoor pollutants and health damage (endpoints)

a Health damage by formaldehyde

Although formaldehyde exists in the natural world, such as in food, it is mainly used as glue for construction materials and causes health damage through indoor diffusion. Conspicuous symptoms caused by exposure to formaldehyde are usually acute irritation in the eyes, the nose, and the pharynx. In addition, formaldehyde is suspected causing cancer and is known to give pathological and cellular genetic influence to the mucous membrane of the nasal cavities. With the spread of the sick house problem, sick house syndrome, which has symptoms such as dizziness, nausea, and eye and nose pain, has become known to more people. There are similarities between sick house syndrome and chemical hypersensitivity. However, sick house syndrome is different from chemical hypersensitivity in that symptoms, such as throat irritation or pain, headache, and difficulty in concentration, completely disappear as a result of leaving the house in question. Although a person suffers symptoms when he is living in a room, his physical condition becomes better outside of the room

because he does not make contact with the causative pollutant – this is sick house syndrome, which is caused by indoor environmental pollution. The symptoms of sick house syndrome greatly differ from person to person. Usually, however, people have the following symptoms: 1) headache; 2) reduction in concentration, memory, or thinking power; 3) discomfort in the eyes; 4) dizziness and nausea; 5) nasal mucus, nosebleed, difficulty in respiration, or asthmatic attack; 6) migraine; 7) skin itching; 8) bowel movement disturbance; 9) menstruation disorder; 10) muscle or joint ache; 11) fatigue or wariness; and 12) depression, excitement, and sleep disorder. These symptoms can be roughly divided into mucosa symptoms and mental symptoms.

b Health damage by toluene

Because information on health damage by toluene greatly differs between treatises on occupational exposure, there are many reports on health damage by high-concentration exposure. Toluene diffuses when used as glue for housing materials or as solvent for paint. It is easily absorbed in the human body and stimulates the eyes, the throat, the trachea, etc. In the case of relatively low-concentration or short-time exposure, people suffer sick house syndrome or chemical hypersensitivity, both of which involve mild mental disorder. It has been pointed out that chronic high-concentration exposure has influence on the central nervous system. If exposure is extremely high-concentration, blackout or death may occur. The evidence proving cancer-causing property is insufficient, and toluene is said to be unlikely to cause cancer.

c Health damage by NOx

NO_x is generated when nitrogen and oxygen in the air react to combustion. It is generated indoors by an open combustion appliance or cigarette smoke and causes respiratory health damage, such as bronchitis or pneumonia. In addition, because it stimulates the respiratory tract, asthma patients may respond to lower-concentration exposure than healthy people.

d Health damage by SOx

Absorption of SO_x generated by oil or coal combustion may cause a respiratory disease. Like the symptoms in the case of NO_x, it is known that a respiratory disease, such as bronchitis or pneumonia, may occur.

e Health damage by SPM

Suspended particulate matter (SPM) is generated indoors mainly by cigarette smoke. SPMs may flow into a room from an outside factory or an automobile. The occurrence of health damage depends on the components, concentration, and diameter of particles. SPMs mainly cause a respiratory disease. Although there are substances that cause cancer in their chemical structure, such as cigarette smoke and radon, respiratory diseases caused by general SPMs are covered herein. If the diameter is large, SPMs easily deposit in the upper respiratory tract and have effect on the pharynx. In addition, they have effect on health by reacting to allergens. SPMs with a small diameter reach the pulmonary alveoli and deposit in them. Because SPMs that deposit in the pulmonary alveoli exist for several hundred days, they may cause various disorders because of their chemical and physical properties. In addition, the smaller the diameter, the higher the ratio of SPMs that deposit in the pulmonary alveoli and the more likely they are to cause serious health damage.

2.9.2 Characterization and damage assessment of indoor air pollution

(1) Basic policies for calculation of damage factors and analysis of uncertainty

a Selection of indoor air pollutants

We decided to deal mainly with the indoor pollutants that fulfill the following requirements:

1) The probability of existing indoors is relatively high.
2) The substance causes serious health damage.
3) Quantitative and highly reliable data can be collected.
4) Necessity for a quantitative assessment method is socially high.

Sick house syndrome is an important object of research, and formaldehyde is the worst among the substances that cause sick house syndrome. However, the research is still at the stage of development, and few data are available data. With regard to toluene and volatile organic compounds (VOC) also, the Ministry of Health, Labour and Industry has fixed standard values for indoor concentration as in the case of formaldehyde. However, because quantitative data on VOC are fewer and the rate of VOC existing indoors is lower than in the cases of formaldehyde and toluene, this study covers only formaldehyde and toluene. In addition to chemicals generated from construction materials, there are pollutants generated from products that residents bring in rooms. In Japan especially, NO_x and SO_x are generated from open combustion appliances used in winter and may cause respiratory diseases. In Japan, open combustion appliances have been used in houses for many years. In addition, because of economic efficiency, many families are consuming oil, which generates SO_x. Because NO_x and SO_x have been regulated severely from the viewpoint of air pollution, research on them has been carried out for many years and sufficient quantitative data can be collected. Because of this, NO_x and SO_x are covered by this study. It is reported that NO_x and SO_x react to water outdoors and change into nitric acid and sulfuric acid, secondary pollutants, causing serious health damage. Indoors, however, they hardly react with water and change into such pollutants. Therefore, secondary pollutants are not covered by this study. One of the main causes of indoor air pollution is smoking, which causes countless SPMs indoors. Such respiratory diseases due to particles have been reported and researched frequently. Because, like NO_x and SO_x, SPMs are regulated outdoors also, it is easy to collect a lot of quantitative data. Because of this, SPMs also were included in the objects of this study. Because impact on heath damage differs according to the diameter of SPM, PM 10 (with a diameter of 10 μm) and PM 2.5 (with a diameter of 2.5μm) were used for the research.

In addition to the sick house problem, concern for health damage due to exposure to toxic chemicals at the stage of construction of houses has grown into a social problem. Although the number of people suffering occupational diseases due to chemicals has been decreasing, a considerable number of people still suffer them every year. According to the result of special health checkup of workers handling chemicals, the number of people who show symptoms of such diseases is increasing. During the research, two models were prepared concerning the sick house problem and the stage of construction of houses. Damage assessment was conducted for each of the models. The target chemicals were formaldehyde and toluene, both of which were known to become highly concentrated during construction.

b Selection of category endpoints (symptoms)

Formaldehyde is known to cause the symptoms of sick house syndrome, such as dizziness, headache, and nausea. In the case of sick house syndrome in particular, because symptoms differ greatly among people, the symptoms of sick house syndrome were roughly divided into mucosa symptoms and mental symptoms according to medical specialists' knowledge. Moreover, formaldehyde is suspected of causing cancer by worldwide environmental protection organizations, such as the U.S. Environmental Protection Agency (EPA). It is pointed out that high-concentration exposure or long-term exposure – especially, chronic exposure during construction – may cause nose or larynx cancer. Because of this, nose and larynx cancers are covered by this study.

Toluene also is known to cause the symptoms of sick house syndrome, such as headache and nausea. Because, like the symptoms of sick house syndrome caused by formaldehyde, those caused by toluene can be roughly divided into mucosa syndromes and mental syndromes, these two types of symptoms were covered by the assessment. In addition, it has been medically recognized that high-concentration or chronic exposure to toluene during construction causes serious poisoning symptoms.
It is medically clear that NO_x and SO_x cause respiratory disease. Because of this, hospitalization and acute death due to respiratory disease were covered by the assessment.

Because SPMs are used as a general term for various pollutants, they are related to many diseases because of the chemical structure of the substance itself. Although the cancer-causing property of cigarette smoking is frequently discussed, it is necessary to limit discussions to "cigarette smoking." Because general SPMs are covered herein, we decided that this study should include respiratory diseases whose causal relationship with SPMs has been medically recognized.

c Flowchart of damage assessment of indoor air pollutants

Each of the items necessary for calculation for damage assessment is examined according to the flow shown in Figure 2.9-2.

[Equation for calculation of damage function of indoor air pollution]

$$DF^{\text{Indoorairpollution}} = \Delta C \times DR \times P \times D_S \qquad (2.9\text{-}1)$$

$DF^{\text{Indoorairpollution}}$: damage function of indoor air pollution [DALY/kg]
ΔC: increment in the indoor concentration to the open air due to release of a unit amount of indoor pollutant [μg/m^3] …… **(first step)**
DR: dose-response relationship [case/person/year/(μg/m^3)] …… **(second step)**
P: number of target patients
D_S: degree of damage from disease (amount of damage per case) [DALY/case]

ΔC (increment in the indoor concentration) in Equation 2.9-1 is examined according to the fate and exposure analysis as the first step in Figure 2.9-2. Based on the result, the characterization of indoor air pollution is also examined. In addition, *DR*, *P,* and *Ds* are calculated through examination of the "dose-response relationship" in the damage/effect analysis as the second step, the examination of the "number of target patients," and the examination of the "degree of damage from disease" respectively. This can be summarized as in Table 2.9-3.

Table 2.9-1: Indoor air pollutants and category endpoints [during residence]

Formaldehyde		NO_x		Suspended particulate matter (SPM) (Right column: condition for each symptom)		
1	Sick house syndrome Mucosa symptoms	1	Respiratory disease Acute death	1	Respiratory disease Acute death	
2	Sick house syndrome Mental symptoms	2	Hospitalization for Respiratory disease	2	Respiratory disease Chronic death	30 year of age or older
3	Nose/larynx cancer			3	Hospitalization for Respiratory disease	
				4	Use of bronchodilator	Asthma sufferer
Toluene		SO_x		5	Cough	Asthma sufferer
				6	Stridor	Asthma sufferer
1	Sick house syndrome Mucosa symptoms	1	Respiratory disease Acute death	7	Chronic bronchitis	
				8	Chronic cough	
2	Sick house syndrome Mental symptoms	2	Hospitalization for Respiratory disease	9	Day of movement restriction	
3	Poisoning symptoms			10	Emergency room hospitalization due to chronic closed lung disease	
				11	Emergency room hospitalization due to asthma	
				12	Preschooler's emergency room hospitalization due to pharyngitis/bronchitis	

Table 2.9-2: Indoor air pollutants and category endpoints [during construction]

Formaldehyde		Toluene	
1	Sick house syndrome mucosa symptoms	1	Sick house syndrome mucosa symptoms
2	Sick house syndrome mental symptoms	2	Sick house syndrome mental symptoms
3	Nose/larynx cancer	3	Poisoning symptoms

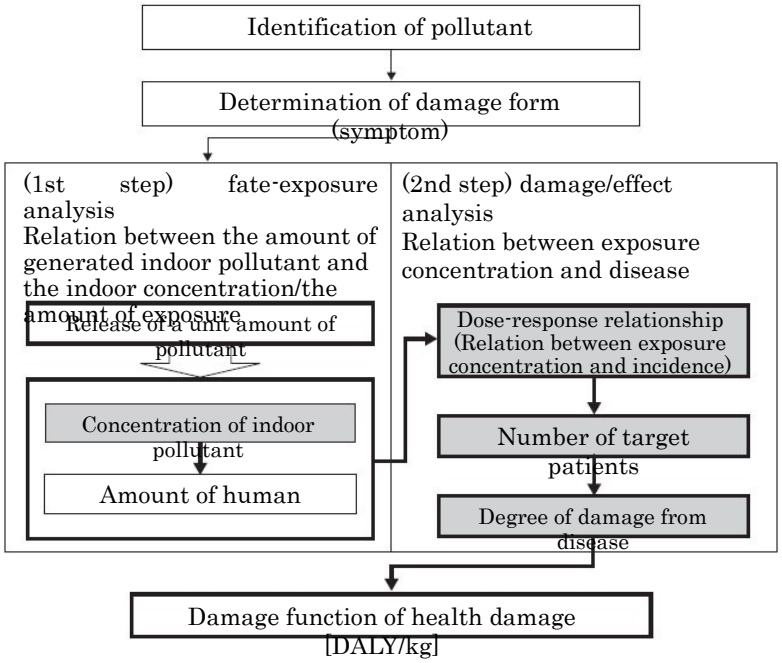


Figure 2.9-2: Flowchart of calculation of damage function of indoor air pollution
(Shaded parts are elements to which uncertainty analysis is applied)

Table 2.9-3: Relation between the flowchart of calculation of damage function and each item

Calculation flowchart	Item to be calculated	Item corresponding to damage function equation
1) Fate analysis	Increment in indoor concentration due to release of a unit amount of pollutant	ΔC
2) Exposure analysis	Predicted daily intake, characterization factor	—
3) Damage analysis	Dose-response relationship	DR
4) Impact analysis	Number of target patients	P
	Degree of damage from disease	D_S

d Uncertainty analysis of indoor air pollution

Limited data were used for damage assessment of indoor air pollution. If a judgment was needed, consultation was held with experts. Because of this, when a parameter (representative value) was fixed, an average value or an appropriate value was selected from among uncertain values that contain individual variability in data (amount of intake, metabolic capacity, and sensitivity) and measurement errors. It can be said that the resultant damage function also contains such uncertainties. Although damage assessment requires estimation of safety (division by a safety coefficient or an uncertainty coefficient), because excessive estimation may result in overestimation, consideration was given to assuming the uncertainty of each parameter and showing the degree of uncertainty as a whole.

(2) Characterization of indoor air pollution (fate analysis and exposure analysis)

a Modeling of indoor air pollution

First, general indoor air pollution was modeled. The general house and living environment for Japanese, including construction, were modeled as described below.

[Housing model]

In the field of architecture, “Architectural Institute of Japan’s Standard Specifications for Wooden Houses” are usually used for houses, while the Institute for Building Environment and Energy Conservation’s “New Energy Saving and Standards for Housing” are usually used for apartment houses. In this study, however, importance was placed also on highly reliable models and representation of Japanese average houses. Because of this, a survey was carried out about the distribution of total floor areas of houses in Japan (Architectural Institute of Japan, housing macro model). According to the result, the average total floor area for a house is 129.03 m^2, almost the same as 125.86 m^2, the total floor area specified in the Architectural Institute of Japan’s Standard Specifications. Therefore, it will be no problem if the Architectural Institute of Japan’s Standard Specifications is used as the representative model of houses in Japan. On the other hand, the average total floor area for an apartment house in Japan is 47.06 m^2 (National Census 2000). If compared with 80.99 m^2, the total floor area of the house model by the Institute for Building Environment and Energy Conservation, it is found that the size of the house model is far larger than the size of an actual apartment house. The national average total floor area for an apartment house, 47.06 m^2, was adopted herein and was combined with the floor height examined separately to determine a representative value for room volume. Because both the Architectural Institute of Japan’s Standard Specifications and the Institute for Building Environment and Energy

Conservation specify that the floor height is 2.4 m, it was assumed that the floor height of the apartment house model is 2.4 m.

[Frequency of ventilation]

The frequency of ventilation expresses the number of times of the indoor air's entire replacement in a certain time. As a sick house measure, the Building Standards Act specifies that the frequency of ventilation for a house should be 0.5 times/h. This means that it is necessary to secure an amount of fresh air that makes it possible to replace half of the amount of indoor air in one hour.

With regard to the frequency of ventilation for general houses, we could not find any studies in Japan that show a value appropriate as the representative value. Although actually measured values can be found in some studies, because they cover only a certain region or houses where no one lives, they do not have sufficiently decisive grounds to show a representative value in Japan. Because of this, after ventilation was divided into three types – 1) opening ventilation, 2) machinery ventilation, and 3) open window ventilation – we estimated a value for each type and then estimated the average frequency of ventilation. As a result, the average frequency of ventilation for a house was found to be 1.07 times/h. For the purpose of calculation, the fraction was cut off and 1.0 time/h was adopted.

Table 2.9-4: Characteristics of single-family and apartment house models

Type of house	Single-family	Apartment
House area [m^2]	125.86	47.06
Floor height [m]	2.4	2.4
Frequency of ventilation [times/h]	1.0	0.7

Generally, apartment houses are more airtight than single-family houses. As a result of a consultation with specialists engaged in construction, the frequency of ventilation for an apartment house was fixed at 0.7 times/h, compared with 1.0 time/h, the representative value for a single-family house.

Because sufficient data on the frequency of ventilation during construction could not be found, it was assumed that the frequency of ventilation is 1.0 time/h for single-family houses and 0.7 times/h for apartment houses. Although windows and the like are usually open during interior finishing, it was assumed herein that the minimum essential ventilation is maintained.

b Fate analysis

Results of fate and exposure analysis in relation to the human body's inhalation of pollutants vary according to type of pollutant, form of room, ventilation method, interior furniture arrangement, positioning of people indoors, human postures and human activities (static or moving), duration of staying in the room, etc. It is difficult to analyze the properties immediately and comprehensively. Representative pollutants, rooms, ventilation, human position, human posture and duration of residence, etc, were modeled and general properties were examined. In addition, with regard to the exposure of the human body to an indoor pollutant, first the properties of indoor air pollution were clarified by the use of computational fluid dynamics (CFD). Formaldehyde was used as the pollutant, because rich data have been accumulated data on it. A 6-tatami room, which is popular in Japan, was assumed as shown in Figure 2.9-3. The room capacity was about 22 m^3. Because only natural ventilation was

assumed to observe indoor air properties, the frequency of ventilation was set at 0.5 times/h, although this is different from the above-described assumption model (usually, ventilation is carried out through window opening and machinery ventilation in addition to natural ventilation). Opening ventilation was adopted: fresh air flows into the room through the baseboards on all sides of the floor and flows out from all sides of the ceiling. The room temperature is fixed at 23 degrees centigrade and there is no heat exchange with the outside on the surface of each wall. Formaldehyde, the pollutant, is generated from the floor, the walls, and the ceiling in the model room in a unified way. Interior material with a diffusion grade of F☆☆☆ (20 μg/(m^2·h) herein) is used for the floor, and interior material with a diffusion grade of F☆☆☆☆ (5 μg/(m^2·h) herein) is used for the walls and the ceiling. The amount of formaldehyde generated from the interior materials is 8.4 mg/day.

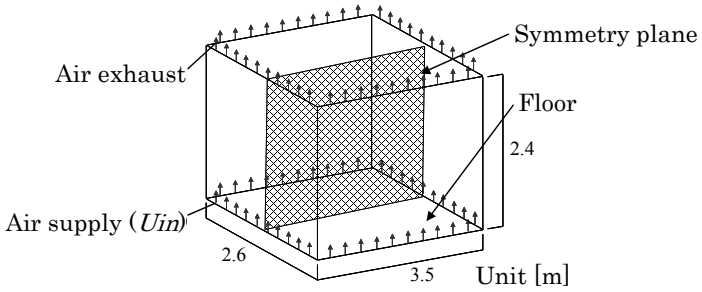


Figure 2.9-3: Indoor model for CFD analysis

Although the amount of human respiration is usually fixed at 15 m$^{3/}$day for the purpose of risk analysis, because it varies according to the human body's activities, it was fixed at 8 L/min (11.5 m^3/day) during sitting and at 6 L/min (8.6 m^3/day) during lying (sleeping). With regard to the duration of residence, it was assumed that people lived in their house for 75% of the lives (18 hours per day), and the number of sleeping hours was fixed at 7.5 according to a survey on the actual condition (Data book 2000 National Time Use Survey). The position for breathing was lying down during sleeping (height of face: 30 cm above the floor) and sitting during the hours other than sleeping hours (10.5 hours) (height of face: 120 cm above the floor). Table 2.9-5 shows a summary of the numerical analysis. It was assumed that fresh air flowed into the room at a wind velocity of 0.25 m/s through the baseboards on all sides of the floor. The strength of disturbance was 5%. Each wall's coordinates y^+ were 0.02 or less.

After the flow-field analysis, boundary conditions were given to the surface of formaldehyde diffusion to analyze the field of formaldehyde diffusion. It was assumed that formaldehyde diffused into the room was exhausted from all sides of the ceiling by ventilation without adsorption on the walls and the ceiling. In addition, it was assumed that fresh air flowed into the room through the baseboards and contained formaldehyde with a concentration of 0 μg/m^3.

Figures 2.9-4 and 2.9-5 show the results of analysis of wind velocity vectors on the symmetry plane in the model room and analysis of the place of formaldehyde diffusion.

Although Figure 2.9-4 shows that the wind velocity (*Uin*) around the inflow openings of fresh air on all sides of the floor is 0.25 m/s, which is a little high, the wind velocity in most parts of the room is low. Therefore, the room is extremely calm on the whole. The velocity of winds along the walls is about 1×10^{-2} m/s. The velocity of downward winds around the center of the room is less than 5×10^{-3} m/s.

Table 2.9-5: Summary of numerical analysis

Room size	3.5 × 2.4 × 2.6 m
Number of meshes	About 170,000
Frequency of ventilation	0.5 times/h
Wind velocity	*Uin* = 0.25 m/s
Room temperature	23°C (isothermal)
Turbulence model	Low Re type k-ε model (Abe-Nagano model)
Difference scheme	Primary upwind difference (motion equation, scalar equation)
Entrance boundary	*Uin*=0.25 m/s, *lin*=1/7・Lin, k=3/2(*Uin*×0.05)^2, $\varepsilon=C\mu \cdot k^{3/2}/lin$, $C\mu$=0.09, *Lin*=0.001 m
Exit boundary	*Uout*: *kout* by mass conservation, εout: free slip
Amount of diffused formaldehyde*	Floor: 20μg/(m^2•h,) Wall/ceiling: 5μg/(m^2•h)
Diffusion coefficient of formaldehyde in the air	Da=1.53×10^{-5} m^2/s (23°C)

* The highest amount of diffusion at each diffusion class is used.

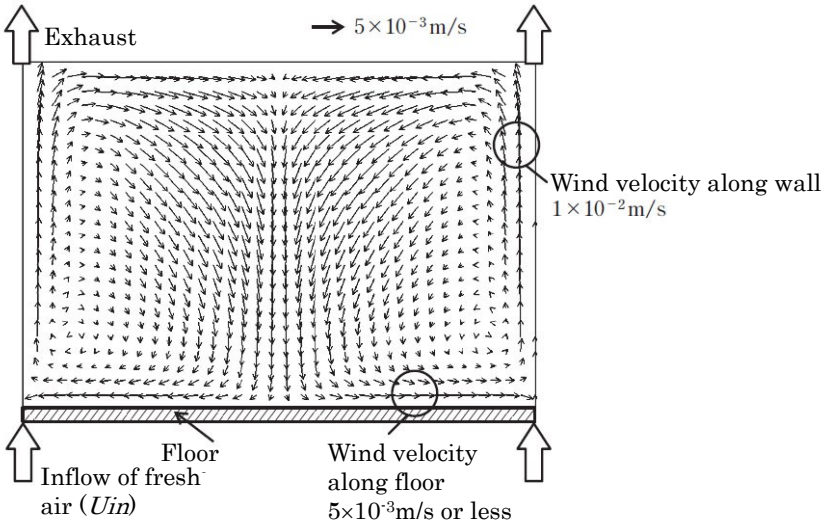


Figure 2.9-4: Wind velocity vectors

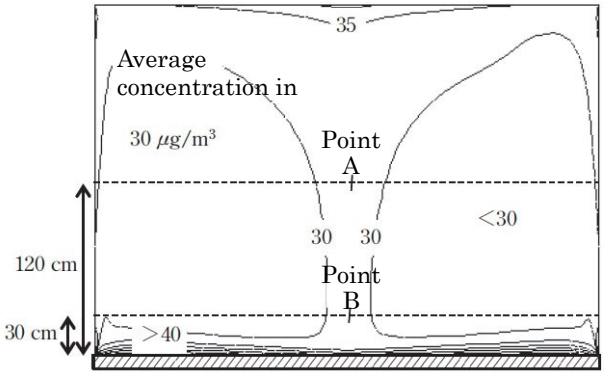


Figure 2.9-5: Formaldehyde concentration distribution
The numbers in the figure indicate concentration [μg/m^3]

According to Figure 2.9-5, although formaldehyde is distributed all over the room almost equally, the concentration is the highest near the floor. Although formaldehyde moves and diffuses in the room because of an indoor air current, its concentration tends to become high near the floor because the wind velocity is extremely low there and the amount of formaldehyde generated from the floor. The average indoor concentration was 30 μg/m^3.

c Exposure analysis

Usually, pollutants enter the human body through the mouth, inhalation, and the skin. This study covers only inhalation. According to existing research (Shu et al. 2001; Kasahara et al. 2002), the human body inhales air from the area below the mouth, because of an upward air current caused by human heat generation. In this study, it was assumed that the human body inhales the average concentration of formaldehyde in the area up to 120 cm above the floor when sitting (Point A in Figure 2.9-5) and in the area up to 30 cm above the floor when lying down (Point B in Figure 2.9-5). As shown in Figure 2.9-5, if formaldehyde of 8.4 mg/day is generated in the room, the average concentration of formaldehyde is 30 μg/m^3 in the inhalation area when sitting and 34 μg/m^3 in the inhalation area when lying down. If the relation between the amount of generated formaldehyde and the indoor concentration is proportional in the room and 1 kg of formaldehyde is generated per year (= 2.74 g/day), the concentration of formaldehyde is 9.79×10^3 μg/m^3 in the inhalation area when sitting and 1.11×10^4 μg/m^3 in the inhalation area when lying down.

The result of CFD analysis found that the wind velocity in most of the room for the analysis model was low and the room was extremely calm and that, in such as case, the concentration of chemicals was almost the same all over the room. Because of this, in the case of analysis models similar to this model, it is all right to assume uniform diffusion in the room and calculate simply by the use of the indoor concentration uniform diffusion equation (Equation 2.9-2) used for architectural environment engineering. The following is the result of calculation by the use of the indoor concentration uniform diffusion equation (Equation 2.9-2) concerning an analysis model similar to an actual CFD analysis model:

[Indoor concentration uniform diffusion equation]

$$C = C_0 + \frac{M}{Q} \qquad (2.9\text{-}2)$$

C: indoor concentration [mg/m^3]
C_0: outdoor concentration [mg/m^3]
M: amount of generated pollutant [mg/h]
Q: amount of ventilation [m^3/h] (= frequency of ventilation (no. of times/h) × room capacity V [m^3])

According to Equation 2.9-2, if C_0, the initial indoor concentration for CFD analysis, is = 0 mg/m^3, the calculation result becomes as follows:

$$\mathrm{C} = \text{Initial indoor concentration } 0\mathrm{mg/m^3} + \frac{8.4\,\mathrm{mg,\ total\ amount\ of\ generated\ formaldehyde\ per\ day}}{262.08\mathrm{m^3,\ total\ amount\ of\ ventilation\ per\ day\ (=0.5times/h\times 21.8\,m^3\times 24\,h/day)}}$$

$$= 0.03205\mathrm{mg/m^3} = 32\mu\mathrm{g/m^3}$$

The results of the estimation of indoor concentration distribution as a result of the CFD analysis were the average concentration of formaldehyde 30 μg/m^3 and 34 μg/m^3 in the inhalation area when sitting and when lying down, respectively. This is almost the same as 32 μg/m^3, the result of calculation of indoor uniform diffusion. The calculations below will be carried out by the use of the indoor concentration uniform diffusion equation.

d Calculation of an increment in the concentration due to release of a unit amount of a pollutant

ΔC in the Equation 2.9-1, the equation for the calculation of the damage function, is an increment in the indoor concentration due to release of a unit amount of a pollutant. It can be calculated by the use of the "indoor concentration uniform diffusion equation," based on the result of indoor concentration analysis by CFD. For the purpose of calculation of an increment, based on the Equation 2.9-2, ΔC can be expressed as follows:

$$\Delta C = C - C_0 = \frac{M}{Q} \qquad (2.9\text{-}3)$$

Because this depends only on the amount of generation and the amount of ventilation, if calculation is carried out by assuming a unit amount of release (1 kg), the calculation result becomes the same irrespective of the type of substance if the house model and the frequency of ventilation are the same. In this study, because the house model is the same for both living and construction, consideration is given only to the difference between single-family houses and apartment houses.

Increment in indoor concentration ΔC [single-family house]
= Annual unit amount of released pollutant (1 kg) ÷ annual amount of ventilation [m^3]
= 1 kg/year ÷ (1.0 times/h × 302 m^3 × 24 h/day × 365 days/year)
= 3.78×10^{-7} kg/m^3 =3.78×10^{2} μg/m^3
(Room capacity (single-family house): 302 m^3 (Architectural Institute of Japan's Standard model), frequency of ventilation: 1.0 time/h)

Increment in indoor concentration ΔC [apartment house]
= Annual unit amount of released pollutant (1 kg) ÷ annual amount of ventilation [m^3]
= 1 kg/year ÷ (0.7 times/h × 111 m^3 × 24 h/day × 365 days/year)
= 1.47×10^{-6} kg/m^3 =1.47×10^{3} μg/m^3
(Room capacity (single-family house): 111 m^3 (national average in Japan), frequency of ventilation: 0.7 times/h)

e Characterization

The characterization of indoor air pollution is calculated in the same way as that of human toxicity and air pollution. First, predicted daily intake (*PDI*) is calculated from the indoor concentration at the time of a unit amount of release (1kg/year) obtained from "c Exposure analysis." The result is divided by acceptable daily intake (*ADI*). The substances about which the standard indoor concentration value has been specified in the Building Standards Act are examined. In this study, calculation is carried out concerning formaldehyde and toluene as examples. The results are divided by *DPI/ADI* of formaldehyde to find the characterization factor.

[Predicted daily intake (PDI)]

As shown in Equation 2.9-4, *PDI* of an indoor chemical can be expressed by dividing the product of the indoor chemical concentration in the inhaled air and the amount of respiration by the body weight. Outdoor inhalation is not included in the object of calculation. Because a daily air intake of 15 m^3 and a weight of 50 kg are generally used for risk analysis, they are used.

Predicted daily intake of indoor pollutant [μg/kg bw/day]

$$= \frac{\text{Indoor pollutant concentration } [\mu g/m^3] \times \text{intake } [m^3/day]}{\text{Weight [kg bw]}} \qquad (2.9\text{-}4)$$

PDI was calculated by the use of an increase in the indoor concentration for single-family houses. 1 of Table 2.9-1 shows the calculation results. Because indoor air pollution depends only on the amount of generation and the amount of ventilation, the value is the same, irrespective of the type of substance.

Table 2.9-6: Calculation results of characterization factors of indoor pollutants

1	**Predicted daily intake (PDI)**
	3.78×10^2 [μg/m^3] ×15 [m^3/day] ÷ 50 [kg bw] = 1.13×10^2 [μg/kg bw/day]
	(*bw: body weight)
2	**Acceptable daily intake (ADI)**
	Formaldehyde *ADI* = 100 [μg/m^3] ×15 [m^3/day] ÷ 50 [kg bw] = 30 [μg/kg bw/day]
	Toluene *ADI* = 260 [μg/m^3] ×15 [m^3/day] ÷ 50 [kg bw] = 78 [μg/kg bw/day]
3	**PDI/ADI**
	Formaldehyde = 1.13×10^2 [μg/kg bw/day] ÷30 [μg/kg bw/day] = 3.78
	Toluene = 1.13×10^2 [μg/kg bw/day] ÷78 [μg/kg bw/day] = 1.45
4	**Characterization factor**
	Formaldehyde = 3.78÷3.78 = 1 (standard)
	Toluene = 1.45÷3.78 = 0.38

[Acceptable daily intake (ADI)]

With regard to formaldehyde and toluene, the standard indoor concentration has been fixed under the Building Standards Act. The standard indoor concentration is 100 μg/m^3 for formaldehyde and 260 μg/m^3 for toluene. Like the calculation of *PDI*, assuming that the amount of intake is 15 m^3/day and the weight is 50 kg, the calculation is carried out as in 2 of Table 2.9-6.

In this way, after *PDI/ADI* of each substance is calculated, the characterization factor is calculated based on formaldehyde (*PDA/ADI* of each substance ÷ *PDA/ADI* of formaldehyde).

(3) Health impact: damage function of sick house syndrome

With regard to sick house syndrome, there is no research on dose-response relationships in the world. Sakabe (2007), a co-researcher who is a medical expert in sick house syndrome, pointed out that it is difficult to determine a dose-response relationship simply concerning

sick house syndrome, which is characterized by great differences among individuals. This is because if the same dose is given, some people show response, while other people do not.

An ordinary dose-response relationship indicates the response rate to the amount of exposure (or concentration). Because of this, in many cases, if the dose increases, the response rate also increases. The calculation method examines the relationship after comparing differences in the response rate when different doses are given to several test groups. Because of this, usually, animal experience is employed and the result is extrapolated to find human beings' dose-response relationship.

Symptoms of sick house syndrome are often light ones, such as dizziness or abnormality in the nose, or ones peculiar to human beings. In addition, because it is difficult to reproduce disease by experiment – for example, disease occurs only in a room – experiment by the use of animals is very difficult. For the same reason, experiment on human beings also is difficult. As a result, we found it necessary to examine the dose-response relationship concerning sick house syndrome by ourselves.

a "QEESI," a diagnosis sheet for sick house syndrome

In the case of sick house syndrome, "QEESI (Quick Environmental Exposure Sensitivity Inventory)" is used to inquire patients about their health conditions to check symptoms. In addition, because the situation in which exposure occurred is very important for investigating the cause, we request patients to measure the concentration of pollution and bring the result to us (upon request, a health center is ready to measure it). The Kitasato Institute Hospital, to which the co-researcher Sakabe belongs, has a lot of such data. We herein examine the dose-response relationship based on these valuable data by ourselves.

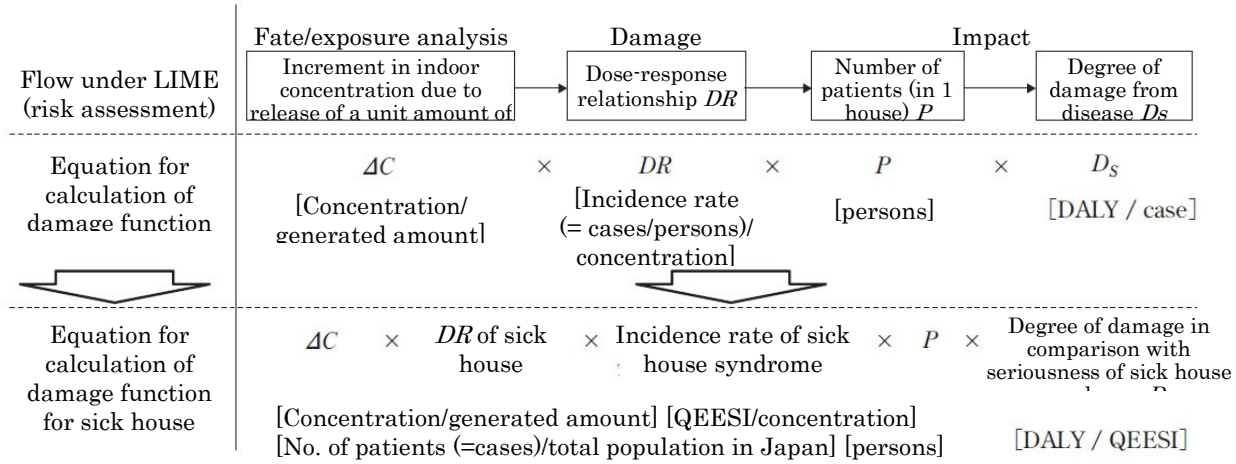
b Seriousness of symptoms and the incidence rate of disease

QEESI consists of five questions. One of them concerns the seriousness of symptoms. Focusing on them, we decided to examine the relation between the indoor concentration and the seriousness of symptoms. In this case, however, because of the definition of the response rate (the incidence rate), the dose-response relationship has becomes useless. This is because QEESI is used only for sick house syndrome patients and therefore the incidence rate is 100%. In addition, the dose-response relationship of ordinary chemicals does not contain information to the effect that with an increase in the concentration, symptoms become heavier. The basic idea is simple: as the concentration increases, the number of people who respond to it increases. Worsening of the health conditions of patients is not examined concerning the dose-response relationship. Because of this, we decided to review the structure from the viewpoint of the whole flow of risk assessment.

c Incidence rate of sick house syndrome

Because the incidence rate of sick house syndrome does not depend on the degree of concentration, it is difficult to express it as the number of incidences per degree of concentration. Because of this, we examined whether the incidence rate could be replaced with another means. As a result, we decided to use the ratio of sick house syndrome patients to the total population of Japan as the incidence rate of sick house syndrome. This makes it possible to estimate the incidence of sick house syndrome for average Japanese. Although no large-scale survey on the number of sick house syndrome patients has been conducted in

Japan, Prof. Uchiyama, of Kyoto University, reported in FY2003 that the ratio of chemical hypersensitivity patients in Japan is 0.74% of the population of Japan (based on a survey of about 4,000 people). The medical expert Sakabe estimated that this value included the number of sick house syndrome patients and chemical hypersensitivity patients. Because this study concerns sick house syndrome, chemical hypersensitivity patients should be excluded. Because of this, the value of 0.74% was adjusted with consideration for the ratio between chemical hypersensitivity patients and sick house syndrome patients. Of the outpatients of the Kitasato Institute Hospital, 40% to 50% suffer sick house syndrome and about 14% suffer chemical hypersensitivity. Based on this, we fixed the ratio of sick house syndrome patients at 0.56% (= 0.74% × (45 / (45 + 14)).



* Case is a unit that expresses one case of disease
* QEESI is a unit that expresses the degree of seriousness in sick house syndrome

Figure 2.9-6: Equation for calculation of damage function for sick house syndrome

d Device of equation for calculation of damage function

The dose-response relationship of sick house syndrome is expressed by the degree of seriousness in symptoms in comparison with the concentration by the use of QEESI (QEESI is used as a unit herein). Therefore, the structure of the calculation equation as a whole was reviewed as shown in Figure 2.9-6. Because the dose-response relationship is a datum on the incidence rate of 100% limited to sick house syndrome patients, the ratio of patients to the population as examined in 3) above was regarded as the incidence rate and was used as the multiplier for the equation. In addition, because the degree of seriousness of symptoms QEESI is used also for the degree of damage from disease in impact analysis (the last *Ds*), it is necessary to adjust the degree of damage to QEESI. By this method, the equation for calculation of damage function for sick house syndrome was newly defined as shown in the lower part of Figure 2.9-6.

e Dose-response relationship of sick house syndrome

Although the sick house syndrome patients of the Kitasato Institute Hospital request health centers to measure the indoor concentration in their houses, the method to indicate the concentration differs among measuring agencies. Because of this, the data dealt with herein consist of detailed and rough data. To give preference to utilization of all the data, we divided degrees of concentration into three levels, such as below and above the standard

indoor concentration value. Medical inspections sometimes adopt the "concept of doubling." For example, because the standard indoor concentration value is 0.08 ppm in the case of formaldehyde, inspection is carried out at 0.04 ppm, half the concentration, and at 0.16 ppm, double the concentration. We adopted this method and divided concentration by doubling the standard value. In addition, because 0.25 ppm is used as the threshold of poisoning due to high-concentration exposure, this value was regarded as the maximum concentration (if a person is exposed to higher concentration, not sick house syndrome but acute poisoning is applied). As a result, the method to express the concentration of formaldehyde and toluene is as shown in Table 2.9-7.

Table 2.9-7: Division of concentration to three levels concerning formaldehyde and toluene

Chemical	Standard value	Double standard value	Boundary value with acute poisoning
Formaldehyde	0.08 ppm	0.16 ppm	0.25 ppm
Toluene	0.07 ppm	0.14 ppm	0.28 ppm

Because sick house syndrome is roughly divided into 1) mucosa symptoms and 2) mental symptoms, QEESI data also are arranged as follows:

Of the five main items of QEESI, the item questioning the "level of symptoms" consists of 10 questions about 10 body parts. The degree of seriousness is divided into ten grades (0 for no symptom; the higher the grade, the higher the degree of seriousness). 1) Mucosa symptoms are those related to a) tracheas and mucosa, b) the skin, and c) digestive organs. 2) Mental symptoms are symptoms related to a) cognition, b) affection, and c) nervous mechanism. QEESI makes it possible to grasp immediately what symptom emerges in what part of the body. Figure 2.9-7 shows how the degree of seriousness of symptoms is grasped by QEESI. The highest score for the ten questions concerning 1) mucosa symptoms and 2) mental symptoms is 30 (10 × 3 symptoms). In addition, QEESI data only cover the following persons: those who have clearly suffered the disease by exposure in their houses (excluding those who live in newly built houses or houses rebuilt within three years or who have received occupational exposure); those who do not have another chronic disease (to exclude disorders by causes other than sick house syndrome); and those aged 15 years and over (to exclude answers from young people because the data are based on answers from individuals). Figure 2.9-8 shows the relation between the Kitasato Institute Hospital's concentration data and the seriousness of symptoms of sick house syndrome (QEESI score).

Regression analysis of these data was carried out to prepare a relation equation. First, consideration was given to setting a threshold. The Building Standards Act has set the standard indoor concentration value as the value at which a person will not suffer sick house syndrome if exposed for his or her lifetime. Because of this, if the actual value is less than the standard value, no person suffers sick house syndrome. That is, it is desirable to set the threshold at the standard concentration value. According to actual data on patients, however, some people suffered sick house syndrome even at less than the standard value. To cope with this, regarding the Kitasato Institute Hospital's data, relation equations were created for the case where a threshold is set and for the case where no threshold is set, and the degree of fitting (prediction accuracy of the data of the relation equations) was judged by another index.

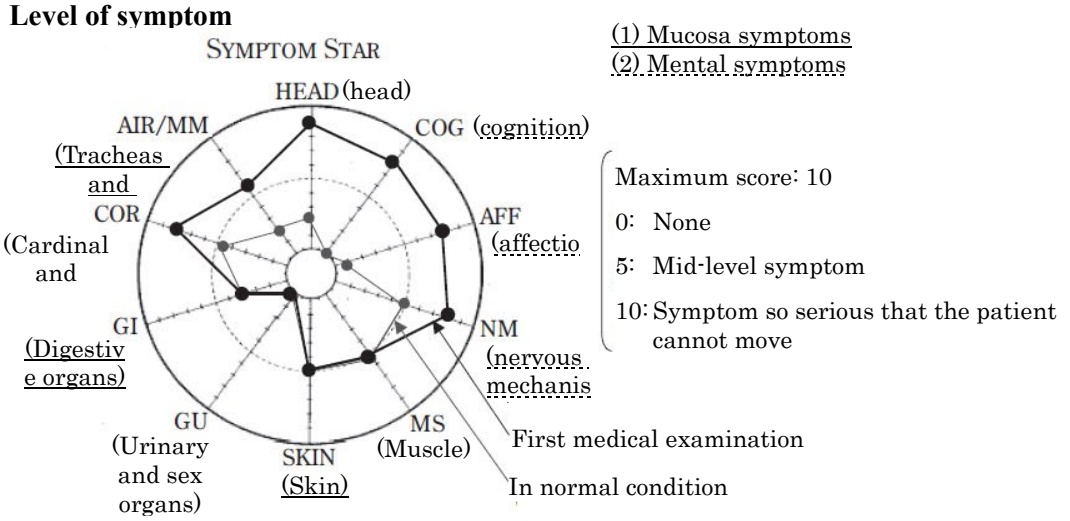
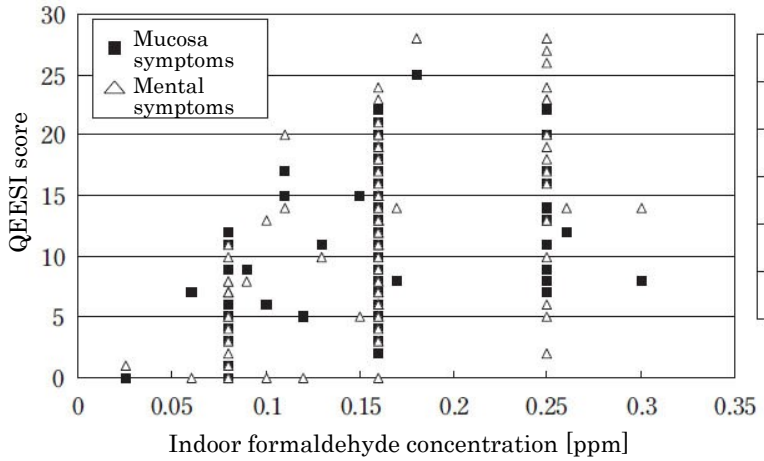
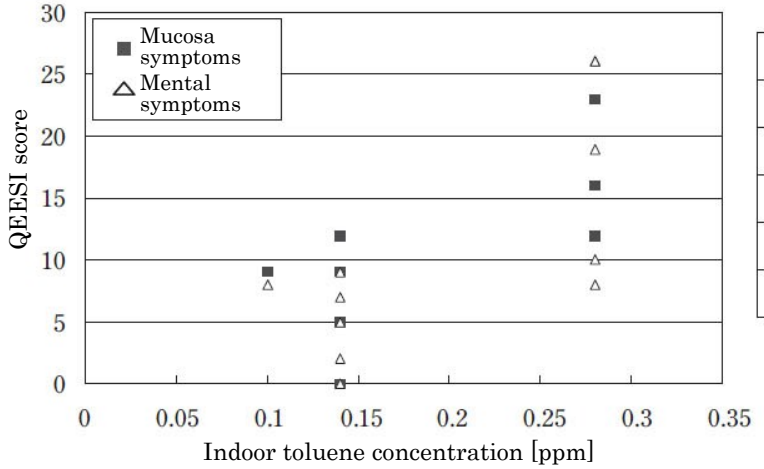


Figure 2.9-7: "Level of symptom" by QEESI (to check each symptom and the seriousness)



Correlation analysis	Mucosa symptoms	Mental symptoms
No. of data	97	97
Correlation coefficient *r*	0.40	0.52
P value	<0.01	<0.01
Significance test	Significant	Significant
Strength of correlation	Mid-level	Mid-level

Figure 2.9-8 (1): Indoor formaldehyde and seriousness of symptoms of sick house syndrome (QEESI score)



Correlation analysis	Mucosa symptoms	Mental symptoms
No. of data	10	11
Correlation coefficient *r*	0.66	0.72
P value	<0.01	<0.01
Significance test	Significant	Significant
Strength of correlation	Strong	Strong

Figure 2.9-8 (2): Indoor toluene and seriousness of symptoms of sick house syndrome (QEESI score)

The Akaike Information Criteria (AIC) was used for the judgment. The purpose of this index is to evaluate the accuracy of statistical models. It is frequently called *AIC*. Because the judgment was made on the equation that passes through the origin (no threshold) and on the equation that does not pass through the origin (a threshold exists; y has a negative value section), *AIC* suitable for the judgment was used. However, AIC was used not for judging the degree of fitting but for comparing several equations. The following is the *AIC* Equation:

$$AIC = n \times \ln\left[\frac{\Sigma(y-\hat{y})^2}{n}\right] + 2k + 1 \qquad (2.1\text{-}4)$$

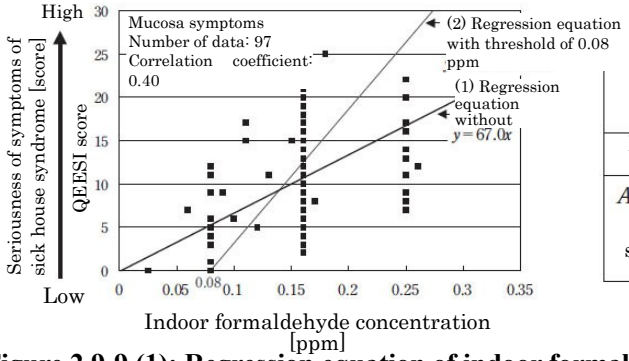
[in the case of a linear relation equation (equation derived by the least-square method)]

k: number of parameters
n: number of data
y: observed value
$\hat{y}$: predicted value

Because the standard concentration value is used as the threshold, the threshold was fixed at 0.08 ppm for formaldehyde and 0.07 ppm for toluene. Figures 2.9-9 and 2.9-10 show relation equations for toluene and sick house syndrome, respectively. The figures show that AIC of the equation without a threshold that passes through the origin is smaller than AIC of the equation with a threshold in both equations (regression equations). It was found from comparison of both equations that **(1) the equation without a threshold** can accurately predict data on patients.

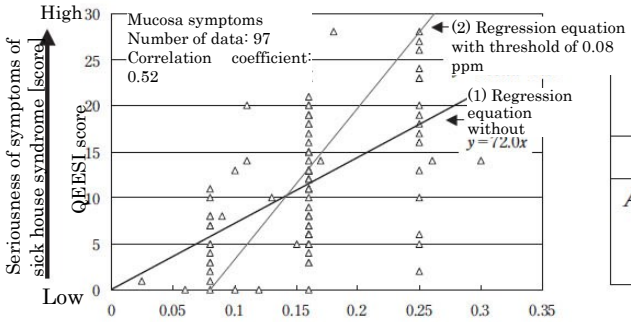
Based on this result, we decided to adopt **the dose-response relationship equation without a threshold**. The inclination of (1) the equation without a threshold in Figures 2.9-9 and 2.9-10 is multiplied by the above-described incidence of sick house syndrome to find the dose-response relation (Tables 2.9-8 and 2.9-9).

Tables 2.9-8 and 2.9-9 show that the dose-response relationship of formaldehyde is almost the same as that of toluene. It is known that the risk of toluene is usually lower than the risk of formaldehyde. The reason why the dose-response relationship of toluene is higher is that there are persons who have suffered from exposure to both formaldehyde and toluene, with the result that toluene-related results increase due to the influence of formaldehyde. Although it is essential to investigate the influence of each substance separately, this is difficult with the patients data used for this study. Because this study is characterized by the use of data on actual patients, we respect and use the results gained from the study.



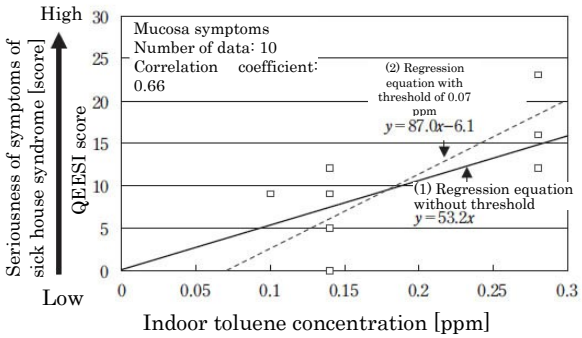
	(1) Without threshold	(2) With threshold (0.08 ppm)
Regression equation	$y=67.0x$	$y=156.3x-12.5$
AIC (the smaller the	325	419

Figure 2.9-9 (1): Regression equation of indoor formaldehyde concentration and mucosa symptoms of sick house syndrome



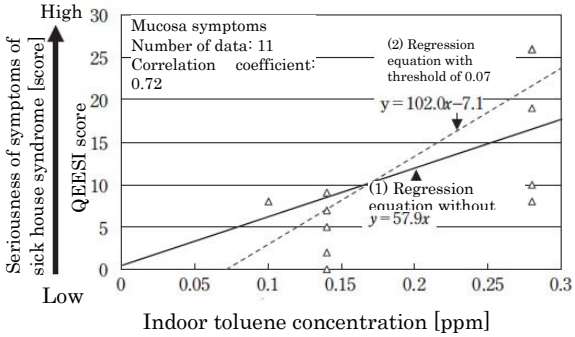
	(1) Without threshold	(2) With threshold (0.08 ppm)
Regression equation	$y=72.0x$	$y=163.9x-13.1$
AIC (the smaller the	355	417

Figure 2.9-9 (2): Regression equation of indoor formaldehyde concentration and mental symptoms of sick house syndrome



	(1) Without threshold	(2) With threshold (0.07 ppm)
Regression equation	$y=53.2x$	$y=87.0x-6.1$
AIC (the smaller the	33	39

Figure 2.9-10 (1): Regression equation of indoor toluene concentration and mucosa symptoms of sick house syndrome



	(1) Without threshold	(2) With threshold (0.07 ppm)
Regression equation	$y=57.9x$	$y=102.0x-7.1$
AIC (the smaller the	44	47

Figure 2.9-10 (2): Regression equation of indoor toluene concentration and mental symptoms of sick house syndrome

Table 2.9-8: Dose-response relationships DR of indoor formaldehyde and sick house syndrome

DR relationship of formaldehyde [QEESI /ppm] (With consideration for the incidence of 0.56%)	
(1) Mucosa symptoms	$66.98 \times 0.0056 = 3.78 \times 10^{-1}$
(2) Mental symptoms	$71.96 \times 0.0056 = 4.06 \times 10^{-1}$

Table 2.9-9: Dose-response relationships DR of indoor toluene and sick house syndrome

Dose-response relationship of toluene [QEESI /ppm] (With consideration for the incidence of 0.56%)	
(1) Mucosa symptoms	$53.16 \times 0.0056 = 3.00 \times 10^{-1}$
(2) Mental symptoms	$57.88 \times 0.0056 = 3.27 \times 10^{-1}$

f Impact analysis of sick house syndrome

We will examine the number of target persons. An actual house has rooms with a strong impact of indoor air pollution and rooms with little impact of it. The residents move from room to room with the passage of time. For the purpose of accuracy, it may be necessary to take into consideration the number of persons in each room and the time of their staying in each room. However, if the residents stay somewhere in the house, a calculation method can be devised for one house as a unit. This method was adopted for the study. Although not all the residents stay home 24 hours a day, as the number of persons who stay home daily we adopted the average number of household members, 2.58, according to National Census FY2005. That is, the average number of household members was used as the number of persons exposed to indoor air pollution. This value was used irrespective of whether the house is a single-family house or an apartment house.

Next, we will calculate *DALY* of sick house syndrome. Under LIME, *DALY*, disability-adjusted life-years, is used as an index of human health damage. *DALY* can be calculated by the sum of years lived with a disability (YLD) and years of life lost (YLL).

Because there seems no existing research on *DALY* of sick house syndrome, a new calculation method was developed. Sick house syndrome rarely causes death directly and it is difficult to prove a causal relationship. Because of this, consideration was given only to the sum of years lived with a disability *YLD*. *YLD* can be calculated by multiplying the degree of seriousness of a disability by the duration of the disability.

When *YLD* is calculated, it is necessary to calculate for (1) mucosa symptoms, (2) mental symptoms, and the degree of seriousness and duration of each disability. The degree of seriousness of a disability is estimated from similar symptoms, based on the method of classification that divides the degree into six levels according to typical symptoms and troubles. The six-level classification is as shown in Table 2.9-10. Table 2.9-11 shows the degrees of seriousness in symptoms similar to the mucosa symptoms and mental symptoms of sick house syndrome. Based on this, medical experts determined the degree of seriousness in disability caused by sick house disease as shown in Table 2.9-12.

The representative value of duration of disability was fixed at two years, because it takes about two years to cure ordinary sick house syndrome (Table 2.9-13). Moreover, to assign the value in the equation for sick house syndrome (Figure 2.9-7), *DALY Ds* for each case

[DALY/case] was substituted with *DALY* for each point of QESSI [DALY/QEESI] by dividing *DALY Ds* for each case by the average QEESI for each case [QEESI/case]. The average score of patients' data used in this study was used as the average QEESI for each case.

Table 2.9-10: Classification of level of disability (6 levels)

Level	Seriousness	Health condition
1	0.096	Disability exists in at least one activity in one of the fields of amusement, education, reproduction, and work.
2	0.220	Disability exists in most of the activities in one of the fields of amusement, education, reproduction, and work.
3	0.400	Disability exists in activities in two or more of the fields of amusement, education, reproduction, and work.
4	0.600	Disability exists in most of the activities in most of the fields of amusement, education, reproduction, and work.
5	0.810	Assistance is necessary for daily life, such as preparation of meals, shopping, and housework.
6	0.920	Assistance is necessary for basic activities, such as meal, sanitation, and evacuation.

Table 2.9-11: Symptoms similar to those of sick house syndrome and the level of seriousness

Symptoms similar to mucosa symptoms		Symptoms similar to mental symptoms	
Disease/symptoms	Seriousness of disability	Disease/symptoms	Seriousness of disability
Asthma	0.099	Depressive disorder	0.600
Peptic ulcer	0.115	Panic disorder	0.173
Liver cirrhosis	0.330	Drug-related disorder	0.251
Appendicitis	0.463	Poisoning	0.609

Table 2.9-12: Seriousness of disability caused by sick house syndrome

	Average	Maximum
Mucosa symptoms	0.096	0.220
Mental syndromes	0.220	0.400

Table 2.9-13: DALY D_S of disability caused by sick house syndrome

		(1) Mucosa symptoms	(2) Mental symptoms
(a) Seriousness of disability		0.096	0.220
(b) Duration of disability		2 (years)	
(c) (= (a)×(b)) *DALY* D_S [DALY/case]		0.192	0.440
(d) Average QEESI by case [QEESI/case]	Formaldehyde	11.19	11.56
	Toluene	11.00	10.91
(e) (= (c)/(d)) *DLAY* for each point of QEESI [DALY/QEESI]	Formaldehyde	1.72×10^{-2}	3.81×10^{-2}
	Toluene	1.75×10^{-2}	4.03×10^{-2}

(4) Health impact: damage function of nose/larynx cancer

a Dose-response relationship of nose/larynx cancer

Although various models were devised concerning carcinogenesis, EPA's unit risk is well known in the world and is used for many epidemiological studies. The unit risk expresses carcinogenesis probability when a person is exposed to a carcinogenic material in his or her lifetime as the value per unit concentration in the medium. Under LIME, it was already used for the assessment of the carcinogenic risk when a chemical is generated outdoors. In this study, by following it, we calculated a dose-response relationship when formaldehyde is generated indoors. Because the unit risk expresses the response rate when ordinary life is maintained for one's lifetime, it is necessary to divide it by the number of years in the lifetime to calculate the dose-response relationship per year. In addition, when the dose-response relationship in the working environment during construction is examined, it is necessary to convert exposure during ordinary life into exposure in the working environment. We calculated this by estimating the conversion coefficient from an average life expectancy of 75 years and 45 working years, which are used for ordinary risk assessment. Tables 2.9-14 and 2.9-15 show the dose-response relationship during residence and during construction, respectively.

Table 2.9-14: Dose-response relationship DR between indoor formaldehyde and nose/larynx cancer [during residence]

Dose-response relationship *DR*
1.30×10^{-2} (case/(mg/m^3)/ lifetime years) ÷ 75 (years) = 1.73×10^{-4} (case/(mg/m^3))
Unit risk: 1.30×10^{-2} (case/(mg/m^3)/lifetime years)
Lifetime years: 75 (years)

Table 2.9-15: Dose-response relationship DR between indoor formaldehyde and nose/larynx cancer [during construction]

Dose-response relationship *DR*
1.30×10^{-2} (case/(mg/m^3)/lifetime years) ÷ 75 (years) × conversion coefficient 0.13 = 2.25×10^{-5}(case/(mg/m^3))
Unit risk: 1.30×10^{-2} (case/(mg/m^3)/lifetime years)
Lifetime years: 75 (years), working years: 45 (years)
Conversion coefficient from ordinary exposure to occupational exposure =
8 (h)/24 (h) × 5 (days)/7 (days) × 48 (weeks)/52 (weeks) × 45 (years)/75 (years) = 0.13

b Impact analysis of nose/larynx cancer

The average number of residing people is 2.58, the same number as in the case of sick house syndrome. With regard to construction, the number of persons who suffer damage from occupational exposure to an indoor air pollutant is calculated. The total number of workers engaged in house construction is about 1.88 million (in 2000) and the number of cases where the construction of a house begins is about 1.23 million. Therefore, the number of persons who receive occupational exposure at the construction site of a house is 1.53.

Under LIME 1, in the method to assess health damage from toxic chemicals, the value of *DALY* was examined concerning nose/larynx cancer caused by formaldehyde. Therefore, the already-calculated *DALY Ds* of nose/larynx cancer caused by formaldehyde (*YLL* = 4.5; *YLD* = 0.4; *DALY* = 4.9) were used.

(5) Health impact: damage function of acute poisoning

a Dose-response relationship of toluene and acute poisoning

With regard to poisoning caused by toluene, by reference to Nakanishi et al. (2005), the already-calculated concentration increment ΔC was used to calculate the value of the dose-response relationship. Nakanishi et al. (2005) examined the relation between the exposure concentration and the incidence rate based on epidemiological data. Because this relation concerns general exposure, it is necessary to change the relation to that for construction. Therefore, we considered the conversion coefficient from general exposure to occupational exposure (similar to Table 2.9-15) and defined the dose-response relationship of poisoning caused by toluene (Tables 2.9-16 and 2.9-17). In addition, because the relationship depends on ΔC (indoor concentration increment), the difference between a single-family house and an apartment house was taken into consideration.

Table 2.9-16: Dose-response relationship DR between indoor toluene and poisoning [during residence]

Dose-response relationship *DR* [single-family house]
Response = $\{1/[1+\exp(-(-3.09722)-0.00909961\times\text{Dose})-0.043\} = 3.65\times10^{-4}$ (case/(mg/m^3))

Response is an increment in the incidence, and Dose is the exposure concentration for a single-family house. (= $\Delta C = 3.78\times10^{-1}$(mg/m^3))

Dose-response relationship *DR* [apartment house]
Response = $\{1/[1+\exp(-(-3.09722)-0.00909961\times\text{Dose})-0.043\} = 7.77\times10^{-4}$ (case/(mg/m^3))

Response is an increment in the incidence, and Dose is the exposure concentration for an apartment house. (= $\Delta C = 1.471$(mg/m^3))

Table 2.9-17: Dose-response relationship DR between indoor toluene and poisoning [during construction]

Dose-response relationship *DR* [single-family house]
Response = $\{1/[1+\exp(-(-3.09722)-0.00909961\times\text{Dose})-0.043\}$ × conversion coefficient 0.13 = 4.81×10^{-5} (case/(mg/m^3))

Response is an increment in the incidence, and Dose is the exposure concentration for a single-family house. (= $\Delta C = 3.78\times10^{-1}$(mg/m^3))

Dose-response relationship *DR* [apartment house]
Response = $\{1/[1+\exp(-(-3.09722)-0.00909961\times\text{Dose})-0.043\}$ × conversion coefficient 0.13= 1.02×10^{-5} (case/(mg/m^3))

Response is an increment in the incidence, and Dose is the exposure concentration for an apartment house. (= $\Delta C = 1.47$(mg/m^3))

b Impact analysis of acute poisoning

The average number of residing people is 2.58, the same number as in the case of sick house syndrome. With regard to construction, the number of persons who receive occupational exposure at the construction site of a house is 1.53 as examined above.

For the purpose of this study, acute poisoning is mainly caused by inhalation of toluene. Because acute poisoning caused by toluene rarely results in death, *YLL* is omitted and only *YLD* was examined. *YLD* can be calculated by multiplying the level of seriousness of the disability by the duration of the disability. The level of seriousness was calculated from a decrease in the quality of life (QOL) due to poisoning. *QOL* is 0 in the state of death and 1 in the state of ordinary health (or complete health). The object of assessment is scored 0 to 1 in terms of health condition. Because the level of seriousness of disability is equivalent to 1 minus *QOL*, the level was calculated at 0.08 by the use of the value of *QOL*, 0.92, which corresponds to a decline in the muscular strength of the limbs, hearing ability, and power of concentration according to Nakanishi et al. (2005). The duration of disability due to poisoning was fixed at 2 years, the ordinary duration of disease based on medical judgment. As a result, *DALY* of poisoning was calculated at 0.16.

(6) Health impact: damage function of respiratory diseases

a Dose-response relationship of respiratory diseases

Respiratory diseases were divided into hospitalization and acute death. Because the health impact caused by a substance after exposure in the inside of the body is the same irrespective of exposure method and channel if the substance is the same, a similar dose-response relationship was cited from ExternE (Holland et al. 1999), epidemiological data examined concerning air pollution under LIME 1. The dose-response relationship is a relationship between the substance and the health impact and is common irrespective of whether indoors or outdoors.

With regard to respiratory hospitalization, the incidence rate per unit of NO_x or SO_x concentration is shown in ExternE (Holland et al. 1999). With regard to acute death, the dose-response coefficient obtained on the assumption that the current death rate increases according to the concentration of a pollutant is applied. This coefficient is expressed as the rate of increase in the death rate due to an increase in the unit of NO_x concentration [risk/$risk_{baseline}$/($\mu g/m^3$)]. The death rate after an increase in the unit of NO_x can be calculated by multiplying the current death rate [$risk_{baseline}$/year] by the coefficient. Table 2.9-18 shows the dose-response relationships of NO_x and SO_x.

The epidemiological data ExternE (Holland et al. 1999) is applied also to SPM. Table 2.9-19 shows the dose-response relationship of SPM. The death rate per SPM concentration unit is used for acute death and chronic death, while the incidence rate per SPM concentration unit is used for the dose-response relationships of the other respiratory diseases. Because the incidence rate may differ between adults and children, depending on symptoms, different dose-response relationships should be used in such cases.

b Impact analysis of respiratory diseases

The average number of residing people is 2.58, the same number as in the case of sick house syndrome. With regard to SPM, however, the difference in the dose-response relationship between adults and children and the diseases suffered only by asthmatics were assessed. This required consideration of the ratio between adults and children and the ratio of asthmatics to make them target populations. The ratios were calculated based on the result of examination of statistical data, and multiplication was performed. Table 2.9-20 shows the calculated ratios and Table 2.9-21 shows the calculated target populations for each disease.

DALY Ds of respiratory diseases was examined based on Hofstetter (1998). Table 2.9-22 shows *DALY Ds* of all the respiratory diseases assessed in this study.

Table 2.9-18: Dose-response relationships DR of NO_x and SO_x

			NO_x	SO_x
Respiratory hospitalization	Incidence rate per unit of concentration [case/person/year/(μg/m^3)]		1.400×10^{-6}	2.040×10^{-6}
Acute death		(i) Rate of increase in death rate per unit of concentration	3.400×10^{-4}	7.200×10^{-4}
		(ii) Current death rate	814.7×10^{-3}	7.418×10^{-3}
	(i) × (ii) Death rate per unit of concentration [case/person/year/(μg/m^3)]		2.522×10^{-6}	5.341×10^{-6}

Table 2.9-19: Dose-response relationship DR of SPM and respiratory diseases

			Death rate or incidence per DR concentration unit [cases/persons/years/(□g/m^3)]	
			PM10	**PM2.5**
1	Acute death		2.97×10^{-6}	5.05×10^{-6}
2	Chronic death		7.42×10^{-6}	4.40×10^{-5}
3	Respiratory hospitalization		2.07×10^{-6}	3.46×10^{-6}
4	Use of bronchodilator	(Adult)	1.63×10^{-1}	2.72×10^{-1}
		(Child)	7.80×10^{-2}	1.29×10^{-1}
5	Cough	(Adult)	1.68×10^{-1}	2.80×10^{-1}
		(Child)	1.33×10^{-1}	2.23×10^{-1}
6	Stridor	(Adult)	6.10×10^{-2}	1.01×10^{-1}
		(Child)	1.03×10^{-1}	1.72×10^{-1}
7	Chronic bronchitis	(Adult)	4.90×10^{-5}	7.80×10^{-5}
		(Child)	1.61×10^{-3}	2.69×10^{-3}
8	Chronic cough		2.07×10^{-3}	3.46×10^{-3}
9	Day of movement restriction		2.50×10^{-2}	4.20×10^{-2}
10	Chronic occlusive lung disease		7.20×10^{-6}	1.20×10^{-5}
11	Asthma		6.50×10^{-6}	1.08×10^{-5}
12	Inflammation in pharynx/bronchi of preschool child		2.91×10^{-5}	4.86×10^{-5}

Table 2.9-20: Ratio of persons with a respiratory disease-related condition to the target population

Condition	Ratio to the whole population
Adult (aged 20 and over)	0.795
Child (aged 0 to 19)	0.205
Aged 30 and over	0.652
Asthma (adult) patient	5.03×10^{-3}
Asthma (child) patient	3.57×10^{-3}

Table 2.9-21: Target population for respiratory diseases

			Target population
1	Acute death		2.58
2	Chronic death	Aged 30 and over	1.68
3	Respiratory hospitalization		2.58
4	Use of bronchodilator	(Adult) (asthmatic)	0.00130
		(Child) (asthmatic)	0.00920
5	Cough	(Adult) (asthmatic)	0.00130
		(Child) (asthmatic)	0.00920
6	Stridor	(Adult) (asthmatic)	0.00130
		(Child) (asthmatic)	0.00920
7	Chronic bronchitis	(Adult)	2.05
		(Child)	0.530
8	Chronic cough	(Child)	0.530
9	Day of movement restriction	(Adult)	2.05
10	Chronic occlusive lung disease		2.58
11	Asthma		2.58
12	Inflammation in pharynx/bronchi of preschool child		2.58

Table 2.9-22: DALY Ds of respiratory diseases

	Respiratory disease		$DALY\,D_S$ per death or incidence
1	Acute death		0.75
2	Chronic death		6.6
3	Respiratory hospitalization		0.011
4	Use of bronchodilator	(Adult)	0.00027
		(Child)	0.00027
5	Cough	(Adult)	0.00014
		(Child)	0.00014
6	Stridor	(Adult)	0.00014
		(Child)	0.00014
7	Chronic bronchitis	(Adult)	2
		(Child)	0.025
8	Chronic cough		0.025
9	Day of movement restriction		0.00027
10	Chronic occlusive lung disease		0.00082
11	Asthma		0.00082
12	Inflammation in pharynx/bronchi of preschool child		0.00082

(7) Arrangement of damage factors of indoor air pollution

All the items necessary for the equation for calculation of damage function were examined as described above. Equation 2.9-1 shown in the basic policies at the beginning, the equation for calculation of damage function of indoor air pollution, and the equation for calculation of damage function of sick house syndrome are calculated by the new equations suggested in Figure 2.9-6. Table 2.9-23 (during residence) and Table 2.9-24 (during construction) show calculated values and calculation results for each pollutant and disease.

Table 2.9-23 (1): Damage functions of indoor air pollution [during residence] (formaldehyde)

Formaldehyde								
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC	(G) Dose-response relationship *DR*	(H) No. of target patients *P*	(I) Degree of damage from disease $DALY\,D_S$	(F)×(G)×(H)×(I) Damage function for each disease [DALY/kg]	Damage function of sick house syndrome	Damage function
Single-family	Sick house syndrome mucosa symptoms	3.06×10^{-1} [ppm]	3.78×10^{-1} [QEESI/ppm]	2.58	1.72×10^{-2} [DALY/QEESI]	5.12×10^{-3}	1.73×10^{-2}	1.81×10^{-2}
	Sick house syndrome mental symptoms	3.06×10^{-1} [ppm]	4.06×10^{-1} [QEESI/ppm]		3.81×10^{-2} [DALY/QEESI]	1.22×10^{-2}		
	Nose/larynx cancer	3.78×10^{-1} [mg/m^3]	1.73×10^{-4} [case/person/year/(mg/m^3)]		4.9 [DALY/case]	8.28×10^{-4}	-	
Apart-ment	Sick house syndrome mucosa symptoms	1.19 [ppm]	3.78×10^{-1} [QEESI/ppm]	2.58	1.72×10^{-2} [DALY/QEESI]	1.98×10^{-2}	6.71×10^{-2}	7.03×10^{-2}
	Sick house syndrome mental symptoms	1.19 [ppm]	4.06×10^{-1} [QEESI/ppm]		3.81×10^{-2} [DALY/QEESI]	4.73×10^{-2}		
	Nose/larynx cancer	1.47 [mg/m^3]	1.73×10^{-4} [case/person/year/(mg/m^3)]		4.9 [DALY/case]	3.21×10^{-3}	-	

Table 2.9-23 (2): Damage functions of indoor air pollution [during residence] (Toluene, NO_X, SO_X)

Toluene								
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC	(G) Dose-response relationship *DR*	(H) No. of target patients *P*	(I) Degree of damage from disease $DALY\,D_S$	(F)×(G)×(H)×(I) Damage function for each disease [DALY/kg]	Damage function of sick house syndrome	Damage function
Single-family	Sick house syndrome mucosa symptoms	3.06×10^{-1} [ppm]	3.00×10^{-1} [QEESI/ppm]	2.58	1.75×10^{-2} [DALY/QEESI]	4.13×10^{-3}	1.45×10^{-2}	1.46×10^{-2}

	Sick house syndrome mental symptoms	3.06×10^{-1} [ppm]	3.27×10^{-1} [QEESI/ppm]		4.03×10^{-2} [DALY/QEESI]	1.04×10^{-2}		
	Nose/larynx cancer	3.78×10^{-1} [mg/m^3]	3.65×10^{-4} [case/person/ year/(mg/m^3)]		0.16 [DALY/case]	5.69×10^{-4}	-	
Apart-ment	Sick house syndrome mucosa symptoms	1.19 [ppm]	3.00×10^{-1} [QEESI/ppm]	2.58	1.75×10^{-2} [DALY/QEESI]	1.60×10^{-2}	5.63×10^{-2}	5.68×10^{-2}
	Sick house syndrome mental symptoms	1.19 [ppm]	3.27×10^{-1} [QEESI/ppm]		4.03×10^{-2} [DALY/QEESI]	4.03×10^{-2}		
	Nose/larynx cancer	1.47 [mg/m^3]	7.77×10^{-4} [case/person/ year/(mg/m^3)]		0.16 [DALY/case]	4.70×10^{-4}	-	

NO_X							
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC [□g/m^3]	(G) Dose-response relationship *DR* [case/person / year/□□g/m^3)]	(H) No. of target patients *P*	(I) Degree of damage from disease *DALY* D_S [DALY/case]	(F)×(G)×(H)×(I) Damage function for each disease [DALY/kg]	Damage function
Single-family	Respiratory disease Acute death	3.78×10^{2}	2.52×10^{-6}	2.58	0.75	1.84×10^{-3}	1.86×10^{-3}
	Respiratory disease hospitalization		1.40×10^{-6}		0.011	1.50×10^{-5}	
Apart-ment	Respiratory disease Acute death	1.47×10^{2}	2.52×10^{-6}	2.58	0.75	7.15×10^{-3}	7.21×10^{-3}
	Respiratory disease hospitalization		1.40×10^{-6}		0.011	5.82×10^{-5}	

SO_X							
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC [□g/m^3]	(G) Dose-response relationship *DR* [case/person / year/□□g/m^3)]	(H) No. of target patients *P*	(I) Degree of damage from disease *DALY* D_S [DALY/case]	(F)×(G)×(H)×(I) Damage function for each disease [DALY/kg]	Damage function
Single-family	Respiratory disease Acute death	3.78×10^{2}	5.34×10^{-6}	2.58	0.75	3.90×10^{-3}	3.93×10^{-3}
	Respiratory disease hospitalization		2.04×10^{-6}		0.011	2.19×10^{-5}	
Apart-ment	Respiratory disease Acute death	1.47×10^{2}	5.34×10^{-6}	2.58	0.75	1.51×10^{-3}	1.52×10^{-3}
	Respiratory disease hospitalization		2.04×10^{-6}		0.011	8.49×10^{-5}	

Table 2.9-23 (3): Damage functions of indoor air pollution [during construction] (SPM)

SPM									
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC [□g/m^3]	(G) Dose-response relationship DR [case/person/year/□□g/m^3)]		(H) No. of target patients P	(I) Degree of damage from disease $DALY\,D_S$ [DALY/case]	(F)×(G)×(H)×(I) Damage function for each disease [DALY/kg]		Damage function
			PM10	PM2.5			PM10	PM2.5	PM10/PM2.5
Single-family	Acute death	3.78×10^{2}	2.97×10^{-6}	5.05×10^{-6}	2.58	0.75	2.17×10^{-3}	3.69×10^{-3}	PM10 1.33×10^{-1}
	Chronic death		7.42×10^{-6}	4.40×10^{-5}	1.68	6.6	3.11×10^{-2}	1.85×10^{-1}	
	Respiratory hospitalization		2.07×10^{-6}	3.46×10^{-6}	2.58	0.011	2.22×10^{-5}	3.71×10^{-5}	
	Use of bronchodilator (adult)		1.63×10^{-1}	2.72×10^{-1}	1.30×10^{-3}	0.00027	2.16×10^{-5}	3.60×10^{-5}	
	Use of bronchodilator (child)		7.80×10^{-2}	1.29×10^{-1}	9.20×10^{-3}	0.00027	7.32×10^{-5}	1.21×10^{-4}	
	Cough (adult)		1.68×10^{-1}	2.80×10^{-1}	1.30×10^{-3}	0.00014	1.15×10^{-5}	1.92×10^{-5}	
	Cough (child)		1.33×10^{-1}	2.23×10^{-1}	9.20×10^{-3}	0.00014	6.47×10^{-5}	1.09×10^{-4}	
	Stridor (adult)		6.10×10^{-2}	1.01×10^{-1}	1.30×10^{-3}	0.00014	4.19×10^{-6}	6.94×10^{-6}	
	Stridor (child)		1.03×10^{-1}	1.72×10^{-1}	9.20×10^{-3}	0.00014	5.01×10^{-5}	8.37×10^{-5}	PM2.5 3.49×10^{-1}
	Chronic bronchitis (adult)		4.90×10^{-5}	7.80×10^{-5}	2.05	2	7.59×10^{-2}	1.21×10^{-1}	
	Chronic bronchitis (child)		1.61×10^{-3}	2.69×10^{-3}	5.30×10^{-1}	0.025	8.06×10^{-3}	1.35×10^{-2}	
	Chronic cough (child)		2.07×10^{-3}	3.46×10^{-3}	5.30×10^{-1}	0.025	1.04×10^{-2}	1.73×10^{-2}	
	Day of motion restriction (adult)		2.50×10^{-2}	4.20×10^{-2}	2.05	0.00027	5.23×10^{-3}	8.79×10^{-3}	
	Chronic occlusive lung disease		7.20×10^{-6}	1.20×10^{-5}	2.58	0.00082	5.76×10^{-6}	9.59×10^{-6}	
	Asthma		6.50×10^{-6}	1.08×10^{-5}	2.58	0.00082	5.16×10^{-6}	8.63×10^{-6}	
	Inflammation in pharynx/bronchi of preschool child		2.91×10^{-5}	4.86×10^{-5}	2.58	0.00082	2.33×10^{-5}	3.89×10^{-5}	
Apart-ment	Acute death	1.47×10^{2}	2.97×10^{-6}	5.05×10^{-6}	2.58	0.75	8.42×10^{-3}	1.43×10^{-2}	PM10 5.16×10^{-1}
	Chronic death		7.42×10^{-6}	4.40×10^{-5}	1.68	6.6	1.21×10^{-1}	7.16×10^{-1}	
	Respiratory hospitalization		2.07×10^{-6}	3.46×10^{-6}	2.58	0.011	8.61×10^{-5}	1.44×10^{-4}	
	Use of bronchodilator (adult)		1.63×10^{-1}	2.72×10^{-1}	1.30×10^{-3}	0.00027	8.38×10^{-5}	1.40×10^{-4}	
	Use of bronchodilator (child)		7.80×10^{-2}	1.29×10^{-1}	9.20×10^{-3}	0.00027	2.84×10^{-4}	4.70×10^{-4}	
	Cough (adult)		1.68×10^{-1}	2.80×10^{-1}	1.30×10^{-3}	0.00014	4.48×10^{-5}	7.46×10^{-5}	
	Cough (child)		1.33×10^{-1}	2.23×10^{-1}	9.20×10^{-3}	0.00014	2.51×10^{-4}	4.21×10^{-4}	
	Stridor (adult)		6.10×10^{-2}	1.01×10^{-1}	1.30×10^{-3}	0.00014	1.63×10^{-5}	2.69×10^{-5}	
	Stridor (child)		1.03×10^{-1}	1.72×10^{-1}	9.20×10^{-3}	0.00014	1.94×10^{-4}	3.25×10^{-4}	PM2.5 1.35
	Chronic bronchitis (adult)		4.90×10^{-5}	7.80×10^{-5}	2.05	2	2.95×10^{-1}	4.69×10^{-1}	
	Chronic bronchitis (child)		1.61×10^{-3}	2.69×10^{-3}	5.30×10^{-1}	0.025	3.12×10^{-2}	5.22×10^{-2}	
	Chronic cough (child)		2.07×10^{-3}	3.46×10^{-3}	5.30×10^{-1}	0.025	4.02×10^{-2}	6.71×10^{-2}	
	Day of motion restriction (adult)		2.50×10^{-2}	4.20×10^{-2}	2.05	0.00027	2.03×10^{-2}	3.41×10^{-2}	

	Chronic occlusive lung disease		7.20×10^{-6}	1.20×10^{-5}	2.58	0.00082	2.23×10^{-5}	3.72×10^{-5}	
	Asthma		6.50×10^{-6}	1.08×10^{-5}	2.58	0.00082	2.00×10^{-5}	3.35×10^{-5}	
	Inflammation in pharynx/bronchi of preschool child		2.91×10^{-5}	4.86×10^{-5}	2.58	0.00082	9.02×10^{-5}	1.51×10^{-4}	

Table 2.9-24: Damage functions of indoor air pollution [during construction]

Formaldehyde						
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC [mg/m^3]	(G) Dose-response relationship DR [case/person/year/(mg/m^3)]	(H) No. of target patients P	(I) Degree of damage from disease $DALY\ D_S$ [DALY/case]	(F)×(G)×(H)×(I) Damage function [DALY/kg]
Single-family	Nose/larynx cancer	3.78×10^{-1}	2.29×10^{-5}	1.53	4.9	6.49×10^{-5}
Apart-ment	Nose/larynx cancer	1.47	2.29×10^{-5}	1.53	4.9	2.52×10^{-4}

Toluene						
House	Health condition (Disease)	(F) Calculation of increase in concentration due to release of unit amount of pollutant ΔC [mg/m^3]	(G) Dose-response relationship DR [case/person/year/(mg/m^3)]	(H) No. of target patients P	(I) Degree of damage from disease $DALY\ D_S$ [DALY/case]	(F)×(G)×(H)×(I) Damage function [DALY/kg]
Single-family	Poisoning	3.78×10^{-1}	4.81×10^{-5}	1.53	0.16	4.45×10^{-6}
Apart-ment	Poisoning	1.47	1.02×10^{-4}	1.53	0.16	3.68×10^{-5}

2.9-3 Procedure for impact assessment of indoor air pollution

The procedure for damage assessment of indoor air pollution can be described concretely as follows. First, assumptions are made about whether during residence or during construction and whether a single-family house or an apartment house. If damage is assessed about the period between construction and residence, the results of assessment of damage during construction and during residence are totaled. After assumptions are made, an equation for assessment of a pollutant is selected. For example, if the impact of formaldehyde is assessed during residence in a single-family house, the damage function 1.81×10^{-2} [DALY/kg] for [during residence] – (formaldehyde) – single-family house in Table 2.9-23 (1) is used. If the amount of generated formaldehyde is found, it is possible to express potential damage from indoor air pollution as a value of *DALY*.

To know damage from sick house syndrome only, use the damage function 1.73×10^{-2} DALY/kg for sick house in the column of damage function for each disease in the same table. The same is applicable to other substances and symptoms.

For example, if the construction period for a single-family house is 3 months and formaldehyde of 1 kg/year is released from construction materials for the two years after the construction, the generated amount is 0.25 kg during construction and 2 kg during residence.

Such damage can be calculated by the following equations:

1) Table 2.9-24 [construction] - formaldehyde – damage function for single-family house → 6.49×10^{-5} [DALY/kg]
   $6.49 \times 10^{-5} \times 0.25 = 1.63 \times 10^{-5}$ [DALY]
2) Table 2.9-23 [residence] - formaldehyde – damage function for single-family house → 1.81×10^{-2} [DALY/kg]
   $1.81 \times 10^{-2} \times 2 = 3.62 \times 10^{-2}$ [DALY]
3) Add 1) with 2).
   $1.63 \times 10^{-5} + 3.62 \times 10^{-2} \fallingdotseq 3.62 \times 10^{-2}$ [DALY]
4) The damage from the generation of formaldehyde from this constriction material is 3.62×10^{-2} [DALY].

2.9.4 Uncertainty analysis of damage assessment of indoor air pollution

The parameters used for the calculation of typical damage functions of formaldehyde can be arranged as described herein (Table 2.9-25).

How to prepare probability distributions for the fluctuation of these parameters was first examined (Table 2.9-26). Quantitative data for all the probability distributions could be obtained through the calculation of damage functions. However, judging that a probability distribution cannot be given to every parameter and that great impact is given unless uncertainty is high, probability distributions were given by placing priority on items shared with other substances.

Table 2.9-25: Parameters taken into consideration in the calculation of Equation 2.9-1

Analysis process	Item	Data used	Data source (Original source)	Number of original data	Causes for uncertainty
Increase in concentration ΔC	Total floor area	Single-family house standard specifications (referring also to single-family house average floor area by type of family)	1) Architectural Institute of Japan, single-family house standard specifications 2) Architectural Institute of Japan, housing macro model (National Census 2000, Housing and Land Statistics)	1 (235)	Unevenness in Japan (difference between urban and rural areas, etc.)
		Apartment house average floor area by type of family		235	
	Highness	1) Single-family house standard specifications (referring also to IBEC apartment model)	1) Architectural Institute of Japan, single-family house standard specifications 2) IBEC, New Energy-Saving Standards and Guidelines for Houses	1	Errors in representative values
	Frequency of ventilation	Simplified prediction sheet for relation between airtightness and amount of leaked air	IBEC, New Energy-Saving Standards and Guidelines for Houses (Yoshino H: Plan of Air Tightness and Ventilation Equipment for a Single-Family House)	1	Errors in model Incomplete information
		Corresponding opening area (Survey of actually measured airtightness)	IBEC, New Energy-Saving Standards and Guidelines for Houses (Yoshino H: Trend Survey on Air	12 (3 regions × 4 insulation standards)	Data in limited years and regions

			Tightness of Houses and Standards)		
		Temperature, wind velocity	Meteorological Agency, data in 2005	6 (3 regions × 2)	Data in limited years and regions
		National sharing of housing insulation standards	Architectural Institute of Japan, housing macro model (National Census 2000, housing and land statistics)	24 (3 regions × 4 insulation standards × 2 single-family houses or apartment houses)	Data in limited years and regions, incomplete information
		Frequency of machinery ventilation	Society of Heating, Air Conditioning and Sanity Engineers, ventilation standards	4	Ambiguity, incomplete information
		Heating and cooling periods	Air-conditioning load calculation (SMASH) default data	3 (by region)	Incomplete information
		Time of residence	Society of Heating, Air Conditioning and Sanity Engineers, SHASE schedule	6 (by type of family)	Ambiguity, incomplete information
		Frequency of ventilation through open window	Empirical value	1	Unknown
	Model	Indoor instant uniform diffusion equation	Empirical equation	1	Errors in model
Dose-response relationship DR (sick house syndrome)		Relation between indoor concentration and seriousness of symptoms	Kitasato Institute Hospital's data on patients	97 (by disease)	Uncertainty of model
		Incidence rate	National survey Kitasato Institute Hospital's data on patients	1 2 (ratio between SHS and MCS)	Incomplete information
(Cancer)		Unit risk	US EPA unit risk	1	Differences among species, lower concentration
Target population P		Average household population	National Census 2005	47 (prefectures)	Unevenness in Japan
		Working population	Ministry of Internal Affairs and Communications: Input-Output Table, Data Report (2) in 2000 Employment Table	1	Unevenness among years
Total $DALY\ D_S$ for each disease		Amount of damage per case	1) Medical experts' judgment 2) Table of level of disorder 3) Data from existing research	14	Unknown, uncertainty of model, incomplete information, ambiguity
		Average QEESI	Kitasato Institute Hospital's data on patients	97 (by disease)	Unevenness among patients

Table 2.9-26: Uncertainty of damage functions of indoor air pollution: details of setting of probability distributions

No.	Item	Unit	Probability distribution	Value (representative)	Parameter (standard deviation, etc.)	Data used	How to set parameter
1	Total floor area of single-family house	m^2	Student *t* Distribution	126.46 (midpoint)	Standard: 19.96 Latitude: 1.077	Architectural Institute of Japan, housing macro model	Based on distribution of all data on the left
2	Relation between formaldehyde concentration and mucosa symptoms	QEESI/ person/year/ppm	Normal distribution	66.98 (average)	Standard deviation: 3.28	Kitasato Institute Hospital's data on patients	Regression analysis of data on the left and regarding standard error as standard deviation
3	Relation between formaldehyde concentration and mental symptoms	QEESI/ person/year/ppm	Normal distribution	71.96 (average)	Standard deviation: 3.70	Kitasato Institute Hospital's data on patients	Regression analysis of data on the left and regarding standard error as standard deviation
4	Target population	Person	Beta distribution	2.58 (average)	α:14.32 β:6.05	National Census 2005	Based on distribution of all data on the left
5	Amount of damage from mucosa symptoms	DALY/case	Normal distribution	0.096 (average)	Upper limit of 95% (2σ): 0.220	Value of medical specialist's diagnosis	Setting maximum value at upper limit of 95%
6	Amount of damage from mental symptoms	DALY/case	Normal distribution	0.220 (average)	Upper limit of 95% (2σ): 0.400	Value of medical specialist's diagnosis	Setting maximum value at upper limit of 95%

(1) Total floor area of single-family house [m2]

Although damage functions are calculated by the use of the Architectural Institute of Japan's standard specifications model, the national distribution of total floor areas of single-family houses written in the housing macro model (Architectural Institute of Japan) was referred to for the estimation of probability distribution. Because the Architectural Institute of Japan's standard specifications model was selected by reference to the national distribution, the national distribution was used without any change and was defined from the viewpoint of conformity with the most suitable distribution by Crystal Ball (Figure 2.9-11).

Indication of amount of statistics	Student *t* distribution
Average	129.03
Standard deviation	28.40

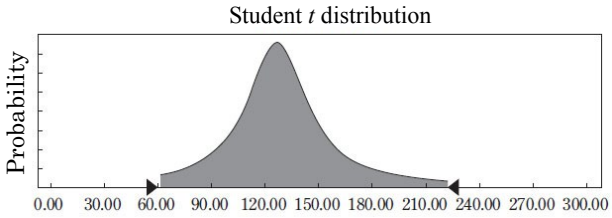


Figure 2.9-11: Probability distribution of total floor areas of single-family houses

(2) Relation of formaldehyde concentration and level of mucosa symptoms of sick house syndrome [QEESI/person/year/ppm]

The relation was estimated by the use of the Kitasato Institute Hospital's data on patients. Because uneven data were integrated into an equation, the equation seems to contain many

uncertainties, such as personal difference (sensitivity and metabolism) and errors in measure of concentration. We examined how to define the degree of the uncertainties. Under LIME 1, regression analysis of observed data was carried out to find the dose-response relationship of global warming, and the standard error in the slope of the resultant equation was used as the standard deviation to define the normal probability distribution. In this study, therefore, the same method was used to define the probability distribution. Figure 2.9-12 shows the result of the regression analysis (Excel Sheet).
As a result of the regression analysis, the standard error in the slope (concentration coefficient) was found to be 3.28. Based on this, a normal distribution with an average of 67.0, the slope, and with a standard deviation of 3.28 was assumed (Figure 2.9-13).

	Coefficient	Standard deviation	*t*	Value of *P*	Lower limit of 95%	Upper limit of 95%
Concentration	66.97806	3.282682	20.40346	1.11E−36	60.46199	73.49413

Assumed to be a normal distribution with standard deviation of 3.28

Figure 2.9-12: Result of regression analysis (coefficient and standard deviation)

Indication of amount of statistics	Normal distribution
Average	66.98
Standard deviation	3.28

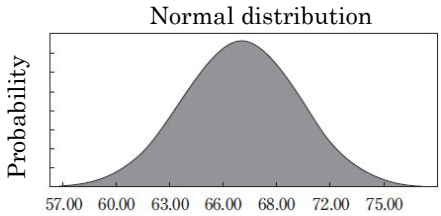


Figure 2.9-13: Probability distribution of relation between formaldehyde and the seriousness of mucosa symptoms

(3) Relation of formaldehyde concentration and the seriousness of mental symptoms of sick house syndrome [QEESI/person/year/ppm]

In the same way, probability distribution was defined concerning formaldehyde concentration and mental symptoms of sick house syndrome. As a result of the regression analysis described in (2), normal distribution was assumed by using the standard error as the standard deviation (Figure 2.9-14).

Indication of amount of statistics	Normal distribution
Average	71.96
Standard deviation	3.70

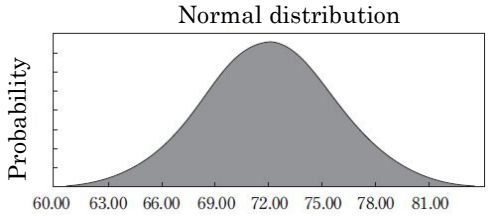


Figure 2.9-14: Probability distribution of relation between formaldehyde and the seriousness of mental symptoms

(4) Target population

With regard to the target population, the average was calculated from the National Census 2005's data on the average number of household members in each prefecture. Because of this, the distribution was defined from the viewpoint of conformity with the most suitable distribution by Crystal Ball (Figure 2.9-15).

Indication of amount of statistics	Beta distribution
Average	2.72
Standard deviation	0.20

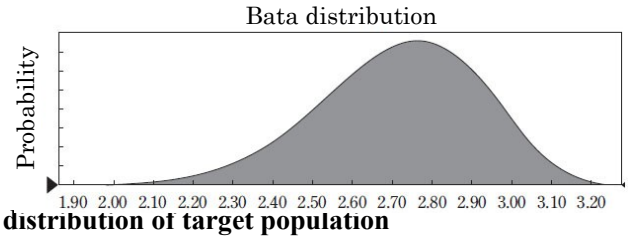


Figure 2.5-15: Probability distribution of target population

(5) Amount of damage from mucosa symptoms of sick house syndrome [DALY/case]

Although a new value was generally discussed by a panel of two or more doctors, because it was difficult to hold such a panel this time, one medical specialist determined a value from existing research. Uncertainties predicted concerning this item include one doctor's prejudiced judgment and fluctuation in the seriousness of symptoms themselves. This study used the average and maximum values of the seriousness of disorder used for the determination. Assuming that there were fluctuations between the average value as the center and the maximum value, we dealt with this by the 95% upper limit of normal distribution. That is, the larger of 2σ (σ standard deviation) added to the average μ was used as the maximum value. Figure 2.9-16 shows the probability distribution. The average and maximum values were set at 0.096 and 0.220, respectively as the 95% upper limit.

Indication of amount of statistics	Normal distribution
Average	9.60×10^{-2}
Standard deviation	7.54×10^{-2}

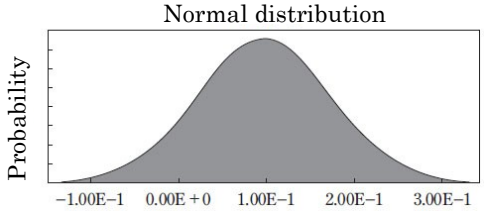


Figure 2.9-16: Probability distribution of the amount of damage from mucosa symptoms of sick house syndrome

Indication of amount of statistics	Normal distribution
Average	2.20×10^{-1}
Standard deviation	1.09×10^{-1}
Diffusion	1.20×10^{-2}
Skewness	0.00
Kurtosis	3.00
Variation coefficient	0.04974

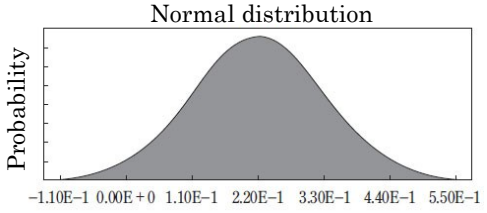
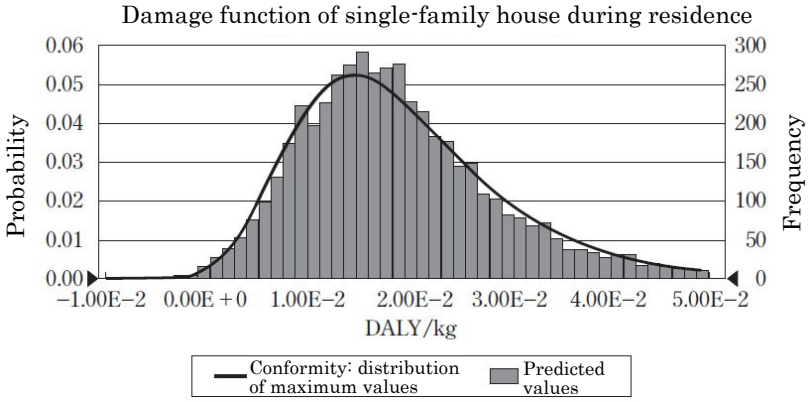


Figure 2.9-17: Probability distribution of the amount of damage from mental symptoms of sick house syndrome

(6) Amount of damage from mental symptoms of sick house syndrome [DALY/case]

In the same way, 0.220, probability distribution was established based on a normal distribution in which the average value of the seriousness of disorder from mental symptoms determined by medical specialists, and 0.400, the maximum value of the seriousness, were set as the 95% upper limit (Figure 2.9-17).



Indication of amount of statistics	Conformity: distribution of maximum values	Predicted value
Average	1.96×10^{-2}	1.96×10^{-2}
Median	1.78×10^{-2}	1.79×10^{-2}
Mode	1.47×10^{-2}	'---
Standard deviation	1.08×10^{-2}	1.08×10^{-2}
Diffusion	1.17×10^{-4}	1.18×10^{-4}
Skewness	1.14	1.27
Kurtosis	5.40	6.04
Variation coefficient	0.5524	0.5544
Lower limit	$-\infty$	-9.22×10^{-3}
Upper limit	$+\infty$	8.54×10^{-2}
Average standard error	'---	1.53×10^{-4}

Figure 2.9-18: Prediction graph of formaldehyde during residence (single-family house)

(7) Result of uncertainty analysis of damage functions of formaldehyde (single-family house)

Simulation was performed 5,000 times by the use of Crystal Ball, calculation software (Figures 2.9-18 and 2.9-19).

The results show that the maximum value distribution of damage functions is for single-family houses during residence. Because the standard deviation and the variation coefficient are considerably small, the impact of the distribution seems low.

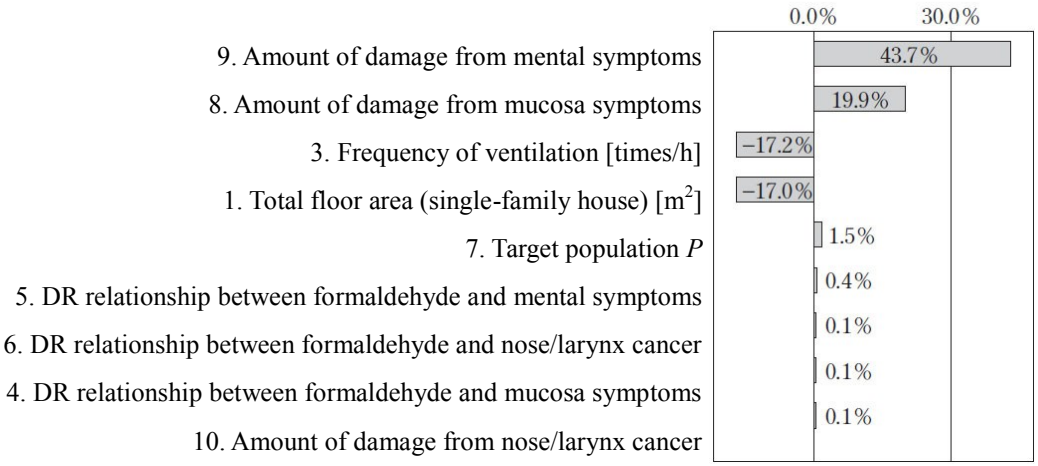


Figure 2.9-19: Graph of sensitivity to formaldehyde during residence (single-family house)

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