| 12 | CAS No.: 100-52-7 | Substance: Benzaldehyde |
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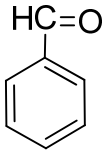
Chemical Substances Control Law Reference No.: 3-1142

PRTR Law Cabinet Order No.: 1-399

Molecular Formula: $C_7H_6O$ Structural Formula:

Molecular Weight: 106.12




**1. General information**

The aqueous solubility of this substance is $3\times10^3$ mg/1,000 g (20°C), the partition coefficient (1-octanol/water) (log $K_{ow}$) is 1.48, and the vapor pressure is 1.27 mmHg (=169 Pa) (25°C). Biodegradability (aerobic degradation) is judged to be good. The substance does not have any hydrolyzable groups.

This substance is designated as a Class 1 Designated Chemical Substance under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (PRTR Law). This substance is almost exclusively used as a raw material for other chemicals, with the majority being used as a raw material for pharmaceuticals (amino acid formulations). The production and import quantity in fiscal 2011 was less than 1,000 t. Fiscal 2012 imports were 318 t while exports were 17 t. The production and import category under the PRTR Law is more than 100 t.

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**2. Exposure assessment**

Total release to the environment in fiscal 2011 under the PRTR Law was approximately 530 t, of which approximately 530 t or more than 99% of overall releases were reported. The major destination of reported releases was the atmosphere. In addition, approximately 4.8 t was transferred to waste materials, and approximately 7.5 t was transferred to sewage. Industry types with large reported releases were the chemical industry and the warehousing industry for the atmosphere, and the chemical industry alone for public freshwater bodies. The largest release among releases to the environment including those unreported was to the atmosphere. A multi-media model used to predict the proportions distributed to individual media in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment overall or to the atmosphere in particular, the predicted proportion distributed to the atmosphere was 82.5%. In regions where the largest estimated releases were to public water bodies, the predicted proportions distributed to water bodies and the atmosphere were 67.9% and 30.7%, respectively.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.44 μg/$m^3$. However, albeit for a limited survey area, a maximum value of 0.49 μg/$m^3$ has been reported based on atmospheric data. In addition, the maximum expected concentration of exposure for indoor air was 117 μg/$m^3$, when study findings for new unoccupied housing were excluded. The mean annual value for the atmospheric concentration in fiscal 2011 was calculated by using a plume-puff model on the basis of releases to the atmosphere reported according to the PRTR Law: this model predicted a maximum level of 0.012 μg/$m^3$.

The maximum expected oral exposure could not be obtained. However, albeit past data, a maximum expected exposure of around 0.012 μg/kg/day was calculated from groundwater data, and a level of less than around 0.04 μg/kg/day was reported in a study of potable water for a limited area. The risk of exposure to this substance by intake from an environmental medium via food is considered slight, given the low bioaccumulation of the substance expected on the basis of its physicochemical properties.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not

be obtained. However, past data yielded around 0.3 μg/L for both public freshwater bodies and seawater.

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**3. Initial assessment of health risk**

This substance may cause irritation to eyes. Its inhalation exposure may cause coughing and sore throat, while its oral exposure may cause sore throat. Contact of the substance with skin may cause redness while its contact with eyes may cause redness and pain.

As sufficient information was not available to evaluate carcinogenicity of the substance, its initial assessment was conducted on the basis of information on its non-carcinogenic effects.

With regard to oral exposure to the substance, a NOAEL of 200 mg/kg/day (for decreased survival rates and hyperplasia of pancreas) obtained from its mid-term and long-term toxicity tests on rats and a NOAEL of 200 mg/kg/day (for hyperplasia of forestomach) obtained from its mid-term and long-term toxicity tests on mice were adjusted for their durations to provide 143 mg/kg/day for its intermittent to continuous exposure. This was considered to be the reliable lowest dose of the substance and identified as its 'non-toxic level*'. As for its inhalation exposure, a LOAEL of 500 ppm (for symptoms such as suppressed body weight increase, hypothermia, and increased liver weight) obtained from its mid-term and long-term toxicity tests on rats, was adjusted for their durations to provide 125 ppm (543 mg/m$^3$) for its intermittent to continuous exposure, and divided by a factor of 10 for their short test periods, and further divided by a factor of 10 for conservative use of the LOAEL. Outcome of 5.4 mg/m$^3$ was identified to be the reliable lowest dose and its 'non-toxic level*'.

With regard to oral exposure to the substance, its health risk could not be assessed as its exposure levels were not known. Its maximum concentration in groundwater was estimated to be below about 0.012 μg/kg/day from historical data (reported in 2000). The MOE (Margin of Exposure) would be over 1,200,000 when calculated from this and its 'non-toxic level*' of 143 mg/kg/day for reference, and divided by a factor of 10 to convert animal data to human data. In addition, the MOE would be over 360,000 when calculated from its maximum concentration of below about 0.04 μg/kg/day in drinking water reported for some locality. As exposure to the substance in the environment through food intakes would be limited, the MOE would not change significantly even when this exposure was included. Therefore, collection of further information would not be required to assess potential health risk from its oral exposure.

With regard to inhalation exposure to the substance in the ambient air, its mean exposure concentration was estimated to be below about 0.23 μg/m$^3$, while its maximum exposure concentration was predicted to be about 0.44 μg/m$^3$. The MOE would be 1,200 when calculated from its 'non-toxic level*' of 5.4 mg/m$^3$ and its maximum exposure concentration predicted from animal experiments, and divided by a factor of 10 to convert animal data to human data. In addition, its maximum (annual mean) concentration in the ambient air near the operators discharging the substance in high concentrations was calculated to be 0.012 μg/m$^3$ from its emissions reported in FY 2011 under the PRTR Law. The MOE would be 45,000 when calculated from this for reference. As for its concentrations in the indoor air, its mean exposure concentration was calculated to be 8.3 μg/m$^3$, while its maximum exposure concentration was predicted to be 117 μg/m$^3$. The MOE would be 5 when calculated from its 'non-toxic level*' of 5.4 mg/m$^3$ and its predicted maximum exposure concentration, and divided by a factor of 10 to convert from animal data to human data. Therefore, no further action would be required at this moment to assess health risk from its inhalation exposure in the ambient air, while the substance would be subject to further research to identify health risk from its inhalation in the indoor air.

| Toxicity | | | | Exposure assessment | | Result of risk assessment | | | Judgment |
|---|---|---|---|---|---|---|---|---|---|
| Exposure Path | Criteria for risk assessment | Animal | Criteria for diagnoses (endpoint) | Exposure medium | Predicted maximum exposure dose and concentration | | | | |
| Oral | 'Non-toxic level*' 143 mg/kg/day | Rat<br>Mouse | Decreased survival rates, etc.<br>Hyperplasia of forestomach | Drinking water<br>Freshwater | —<br>— | μg/kg/day<br>μg/kg/day | MOE<br>MOE | —<br>— | ×<br>× | (○) |
| Inhalation | 'Non-toxic level*' 5.4 mg/m$^3$ | Rat | Suppressed body weight increase, hypothermia, increased liver weight, etc. | Ambient air<br>Indoor air | 0.44<br>117 | μg/m$^3$<br>μg/m$^3$ | MOE<br>MOE | 1,200<br>5 | ○<br>■ | ○<br>■ |

Non-toxic level *

- When a LOAEL is available, it is divided by 10 to obtain a NOAEL-equivalent level.
- When an adverse effect level for the short-term exposure is available, it is divided by 10 to obtain a level equivalent to an adverse effect level for the long-term exposure.

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**4. Initial assessment of ecological risk**

With regard to acute toxicity, the following reliable data were obtained: a 72-h $EC_{50}$ of 32,000 μg/L for growth inhibition in the green alga *Pseudokirchneriella subcapitata*, a 96-h $LC_{50}$ of more than 15,800 μg/L for the crustacean *Orconectes immunis* (North American freshwater crayfish), a 96-h $LC_{50}$ of 1,070 μg/L for the fish species *Lepomis macrochirus* (bluegill), and a 96-h $LC_{50}$ of more than 15,800 μg/L for the freshwater snail *Aplexa hypnorum*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 11 μg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 2,000 μg/L for growth inhibition in the green alga *P. subcapitata*, and a 48-h NOEC of 26,000 μg/L for reproductive inhibition in the ciliate protozoan *Tetrahymena thermophila*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 20 μg/L was obtained.

The value of 11 μg/L obtained from the acute toxicity to the bluegill was used as the PNEC for this substance.

The predicted environmental concentration (PEC) of this substance could not be obtained. As such, a judgment on ecological risk could not be made. However, past data yielded values of less than 0.3 μg/L for both freshwater bodies and seawater, resulting in a ratio to PNEC of less than 0.027. The likelihood of a large increase in the concentration of this substance in public water bodies is considered low, based on its production and import quantity and transfers under the PRTR Law. Accordingly, the need to collect further data on this substance is considered to be minimal.

| Hazard assessment (basis for PNEC) | | | Assessment factor | Predicted no effect concentration PNEC (μg/L) | Exposure assessment | | PEC/PNEC ratio | Judgment based on PEC/PNEC ratio | Assessment result |
|---|---|---|---|---|---|---|---|---|---|
| Species | Acute/ chronic | Endpoint | | | Water body | Predicted environmental concentration PEC (μg/L) | | | |
| Fish (bluegill) | Acute | $LC_{50}$ mortality | 100 | 11 | Freshwater | — | — | × | ○ |
| | | | | | Seawater | — | — | | |

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**5. Conclusions**

| | Conclusions | | Judgment |
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| Health risk | Oral exposure | Although risk to human health could not be confirmed, collection of further information would not be required. | (○) |
| | Inhalation exposure (atmosphere) | No need of further work at present. | ○ |
| | Inhalation exposure (room air) | Candidates for further work. | ■ |
| Ecological risk | No need of further work at present. | | ○ |

［Risk judgments］ ○: No need for further work ▲: Requiring information collection

■: Candidates for further work ×: Impossibility of risk characterization

（○）: Though a risk characterization cannot be determined, there would be little necessity of collecting information.

（▲）: Further information collection would be required for risk characterization.