1 CAS No.: 141-43-5  Substance: 2-Aminoethanol

Chemical Substances Control Law Reference No.: 2-301
PRTR Law Cabinet Order No.*: 1-20
Molecular Formula: C₂H₇NO  Structural formula:
Molecular Weight: 61.10

*Note: No. in Revised Cabinet Order enacted on October 1, 2009

1. General information

This substance is freely miscible with water, the partition coefficient (1-octanol/water) (log K_{ow}) is –1.31 (19°C), and the vapor pressure is 0.404 mmHg (=53.9 Pa) (25°C). Biodegradability (aerobic degradation) is good.

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). Its main uses are as a detergent and cleanser neutralizer, metal corrosion inhibitor, agricultural chemical solvent, gas absorbent (carbon dioxide and carbon disulfide removal), pH regulator for perming solutions and hair dyes, and raw material for other chemical substances such as ethylene imine and taurine. The production quantity in 2009 was approximately 43,000 t (total for monoaminoethanol, diaminoethanol, and triaminoethanol), and export and import quantities in 2009 were 2,351 t and 3,436 t, respectively (total of monoethanol amine and its salts for both exports and imports). The production and import category under the PRTR Law was ≥100 t.

2. Exposure assessment

Total release to the environment in fiscal 2008 under the PRTR Law was approximately 1,900 t, of which approximately 76 t, or 4% of overall releases, was reported releases. The major destination of reported releases was the atmosphere. Besides this, approximately 3,100 t was transferred to waste. Industry types that reported large releases to the atmosphere were the electrical machinery manufacturing industry and the chemical industry, while those that reported large releases to public freshwater bodies were the electrical machinery manufacturing industry, the petroleum and coal product manufacturing industry, and the chemical industry. Including non-reported releases, releases to water bodies are estimated to have been the greatest. A multi-media model used to predict the distribution into each medium in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment and public freshwater bodies, the proportion distributed to water bodies would be 99.0%, whereas for regions where the largest quantities were estimated to have been released to the atmosphere, the proportion distributed to water bodies would be 95.1%.

Data for setting the predicted maximum exposure to humans via inhalation could not be obtained. Further, albeit past data, general environmental atmospheric data indicated a value of around 0.063 µg/m³. Meanwhile, the annual mean value of atmospheric concentration estimated from reported releases to the atmosphere under the PRTR Law was a maximum of 3.9 µg/m³.

The predicted maximum oral exposure was estimated to be around 0.072 µg/kg/day based on calculations from data for public freshwater bodies. Further, oral exposure was estimated to be around 0.12 µg/kg/day based on calculations from data for public freshwater bodies obtained from an environmental study of a limited area. Meanwhile, oral exposure was estimated to be 8.8 µg/kg/day using the maximum river concentration calculated from reported emissions to public freshwater bodies under the PRTR Law. The risk of exposure to this substance by intake from an environmental medium via food is considered slight based on estimates of oral exposure using estimated concentrations in fish species.
The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was about 1.8 µg/L for freshwater bodies and less than around 0.17 µg/L for seawater. Further, an environmental study of a limited area reported a maximum of 3 µg/L for public freshwater bodies.

The river concentration estimated using reported releases based on the PRTR Law was a maximum of 220 µg/L.

3. Initial assessment of health risk

This substance is corrosive to the skin and eyes. Oral exposure to the substance may show corrosive effects. Symptoms of poisoning via the inhalation route include cough, headache, shortness of breath and sore throat, while those via the oral route include abdominal pain, burning sensation and shock or collapse. Contact with the substance causes redness, pain or burns in the skin or eyes. Exposure to the substance may affect the central nervous system and may possibly cause lowering of consciousness.

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

With regard to oral exposure to the substance, its ‘non-toxic level’ could not be identified. As for inhalation exposure, a LOAEL of 12 mg/m³ (for loss of hair and drowsiness) obtained from mid-term and long-term toxicity tests in rats was divided by 10 as it is always the case with a LOAEL, and was further divided by 10 due to the short test periods. 0.12 mg/m³ derived was deemed as a plausible value for the lowest dose of the substance and was identified as its ‘non-toxic level*’.

As to oral exposure to the substance, its ‘non-toxic level*’ could not be identified, and its health risk could not be assessed. From the mid-term and long-term toxicity tests on rats administered concentrations at or below 320 mg/kg/day and mid-term and long-term toxicity tests on dogs administered concentrations at or below 97.5 mg/kg/day of the substance contained in hair dye at 22.42%, which indicated the concentrations had no effects on the animals, on the assumption that these concentrations were the NOAELs of the substance, its ‘non-toxic levels*’ would be 32 mg/kg/day for rats after division by 10 due to the short test periods and 22 mg/kg/day (of the substance) for dogs. Furthermore, these values would indicate the maximum exposure to the substance was approximately 0.072 µg/kg/day when intakes through freshwater from public water bodies were assumed. When divided by 10 due to the need to convert the ‘non-toxic level*’ obtained from the animal experiments to a human equivalent dose, MOEs of 44,000 and 31,000 would be derived, respectively. Meanwhile, reproductive/developmental toxicity tests on rats showed effects on fetuses at 50 mg/kg/day which is much lower than those reported elsewhere. When the LOAEL was assumed to be 50 mg/kg/day and was divided by 10 as is always the case with a LOAEL, a MOE of 6,900 would be derived from the LOAEL of 5 mg/kg/day. When calculated from the predicted maximum concentration of 0.12 µg/kg/day for freshwater from the public water bodies, the MOE would be 27,000, 18,000 and 4,200, respectively. Concentrations of the substance in receiving river discharges from the major sources were estimated on the basis of releases into freshwater in public bodies registered for FY2008 under Japanese PRTR. The maximum exposure would be 8.8 µg/kg/day from concentrations in rivers and MOEs of 360, 250 and 57 would be derived. As exposure to this substance through food intakes was estimated minor, even when exposure through groundwater and food were combined, the MOE values would not be greatly affected. Therefore, collection of information on oral exposure to the substance would not be required to assess health risk.

With regard to inhalation exposure to the substance, the absence of information available on exposure concentrations did not allow for a health risk assessment. For reference, however, the maximum concentration of approximately 0.063 µg/m³ in the ambient air was reported in 1994. The MOE would be 190 when calculated from the maximum concentration and the ‘non-toxic level*’ of 0.12 mg/m³ divided by 10 due to the need to convert the ‘non-toxic level*’ obtained from the animal experiments to a human equivalent dose. The maximum annual average concentration of the substance in the ambient air around its major sources would be 3.9 µg/m³ on the basis of emissions reported for FY2008.
under Japanese PRTR, and, thus, the MOE would be 3.1. Therefore, collection of information would be required to assess health risk from inhalation exposure to this substance in the ambient air.

### 4. Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC₅₀ of 2,510 µg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*, a 24-h EC₅₀ of 43,000 µg/L for swimming inhibition in the crustacean *Artemia franciscana*, a 96-h LC₅₀ of more than 100,000 µg/L for the fish species *Oryzias latipes* (medaka), and a 48-h EC₅₀ of 18,170 µg/L for developmental inhibition in the Mediterranean mussel *Mytilus galloprovincialis*. Accordingly, based on these acute toxicity values and an assessment coefficient of 100, a predicted no effect concentration (PNEC) of 25 µg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 1,000 µg/L for growth inhibition in the green algae *P. subcapitata*, a 21-d NOEC of 850 µg/L for reproductive inhibition in the crustacean *Daphnia magna*, and a 41-d NOEC of 1,240 µg/L for growth inhibition in the fish species *O. latipes* (medaka). Accordingly, based on these chronic toxicity values and an assessment coefficient of 10, a predicted no effect concentration (PNEC) of 85 µg/L was obtained. The value of 25 µg/L obtained from the acute toxicity to the algae was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.07 for freshwater bodies and less than 0.007 for seawater, and there is no need considered for further work. However, there are several reports of a ratio to PNEC exceeding 0.1 in public freshwater bodies and freshwater in environmental studies of limited areas. In addition, based on river concentrations estimated from reported releases under the PRTR Law, there is a possibility that locations with higher concentrations than those found in these limited area environmental studies exist.

Accordingly, there is a need to collect more data regarding this substance, and taking into consideration PRTR data, environmental concentration data needs to be augmented.
### 5. Conclusions

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#### 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.