19	CAS No.: 1763-23-1 (Acid) 29081-56-9 (Ammonium salt) 70225-14-8 (Diethanolamine (DEA) salt) 2795-39-3 (Potassium salt) 29457-72-5 (Lithium salt)	Substance: Perfluorooctane sulfonic acid and its salt forms （Perfluorooctane sulfonate: PFOS）

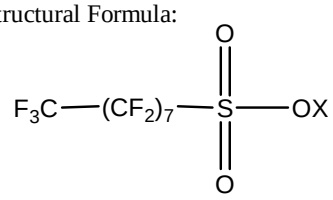
Chemical Substances Control Law Reference No.: 2-1595 (Perfluorooctanesulfonic acid), 2-2810 (Salt (Na,K,Li) of perfluloroalkyl (C4-12) sulfonic acid)

PRTR Law Cabinet Order No.:

Molecular Formula: $C_8F_{17}O_3SX$ (X indicates H, K, etc.)

Molecular Weight: 500.13 (Acid)

Structural Formula:



X indicates H, K, etc.

1. General information

The aqueous solubility of the potassium salt is 519 mg/L (20 ± 0.5°C), and the vapor pressure is 6.4 x 10^{-3} mmHg (= 0.85 Pa) (acid, 25°C; calculated value) and 1.43 x 10^{-11} mmHg (= 1.9 x 10^{-9} Pa) (potassium salt, 25°C; calculated value). Potassium perfluorooctanoate is determinated to be persistent but not to be highly bioaccumulative. In addition, potassium salt is considered not to hydrolyze.

Perfluorooctane sulfonate and its potassium and lithium salts were designated as a Type II Monitoring Chemical Substances under the Low Concerning the Examination and Regulation of Manufacture, etc. of Chemical Substance. PFOS and its analogues are mainly used in semiconductor and photographic industries and for metal plating, photomasks, and bubble extinguishers. There is no other alternative to this substance for use in semiconductors, photomasks, photosensitizers, plating, bubble extinguishers, medical equipment, or electric and electronic parts. In addition, it is indicated that PFOS analogues are degraded by microorganisms and metabolized by larger organisms, resulting in generation of PFOS. The production of PFOS in FY 2005 was 1 to 10 tons per year.

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2. Exposure assessment

As this substance is not 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), no information on release and transfer quantities could be obtained. No reliable log Kow value of this substance was obtained and the distribution ratio by medium was not predicted.

Based on data for the ambient air, the predicted maximum exposure concentration for inhalation exposure to human beings was approximately 0.00003 μg/m^3. The highest estimated oral exposure level was calculated at approximately 0.0067 μg/kg/day from data on drinking water and food. The highest estimated oral exposure level was provisionally calculated at 0.0104 μg/kg/day from groundwater and food data.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was estimated to approximately 11 μg/L for freshwater and approximately 0.028 μg/L for seawater bodies.

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3. Initial assessment of health risk

The potassium salt of this substance irritated eyes of rabbits, but did not irritate their skin. The symptoms which occurred most frequently in rats with oral administration of potassium salt were decrease in motor activities, hypotonia extremities and ataxia. The yellowing of the genitourinary system, gastric distension, hyperemia of gland mucosa and congestion of the lung were observed in the autopsy.

There was insufficient information regarding the carcinogenicity of the substance. For this reason, an initial assessment

of the substance was conducted based on information of non-carcinogenic effects.

A no observed adverse effect level (NOAEL) of 0.00005%(0.015－0.057 mg/kg/day, hypertrophy of hepatic cells in male) was obtained for oral exposure from the medium- and long-term toxicity testing that administered the potassium salt of this substance to rats. As a value of the NOAEL, 0.036 mg/kg/day, the average figure of the dose, was adopted. The value was converted into the dose of this substance from the dose of its potassium salt., A value of 0.03 mg/kg/day was derived as the 'Non-toxic level*'. For inhalation exposure, the 'Non-toxic level*' could not be estimated.

With regard to oral exposure, in case of intakes of drinking water and food, the predicted maximum exposure was approximately 0.0067 μg/kg/day. The margin of exposure (MOE) of 450 was derived from the 'Non-toxic level*' of 0.03 mg/kg/day divided by the predicted maximum dose, and divided by 10, because the 'Non-toxic level*' was established by means of animal testing. For reference, in case of intakes of groundwater and food, the predicted maximum exposure was 0.01 μg/kg/day. The MOE determined in the same way as above was 300.

Concerning inhalation exposure, because its 'Non-toxic level*' is not determined, its health risk can not be identified. For reference, assuming that the absorption rate is 100%, the 'Non-toxic level*' for the oral exposure is converted to the 'Non-toxic level*' for the inhalation exposure. The resulting value is 0.1 mg/m^3. The MOE determined from this figure and the predicted maximum exposure concentration of the ambient air is exceeding 330,000.

Toxicokinetics and metabolism of this substance largely depend on animal species and sex. Especially, the half-life of this substance in human serum(5.4yrs) is much longer than those in laboratory animals. Accordingly, it would be appropriate to assess the health risk of this substance based on the body burden instead of exposure dose or concentration. Since the MOE based on the body burden was greatly different from the MOE above and little is known about the toxicity mechanisms of this substance, it is difficult to identify its health risk. Accordingly, it would be required to collect information on inhalation exposure to this substance for its health risk assessment.

Information of toxicity					Exposure assessment			Result of risk assessment			Judgment
Exposure Path	Criteria for risk assessment		Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure quantity and concentration					
Oral	'Non-toxic level*'	0.03 mg/kg/day	Rats	hypertrophy of hepatic cells in male	drinking water, food	0.0067 μg/kg/day		MOE	450	×	(▲)
					groundwater, food	－ μg/kg/day		MOE	－	×	
Inhalation	'Non-toxic level*'	－ mg/m^3	－	－	Ambient air	0.00003 μg/m^3		MOE	－	×	(▲)
					Indoor air	－ μg/m^3		MOE	－	×	×

Non-toxic level *

- When a LOAEL is available, it is divided by 10 to obtain a level equivalent to NOAEL.
- 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, reliable information of a 96-hour median effective concentration (EC_{50}) growth inhibition value of 48,200 μg/L was found for the algae *Pseudokirchneriella subcapitata*, a 96-hour median lethal concentration (LC_{50}) of 3,340 μg/L was found for the crustacea Mysidae, *Americamysis bahia*, a 96-hour LC_{50} of 8,810 μg/L was found for the fish *Pimephales promelas* (fathead minnow), and a 10-day EC_{50} growth inhibition value of 87.2 μg/L was found for the other organism *Chironomus tentans* (chironomus). Accordingly, an assessment factor of 100 was used, and a predicted no effect concentration (PNEC) of 33 μg/L was obtained based on the acute toxicity values. With regard to chronic toxicity, a 96-hour no observed effect concentration (NOEC) for green algae *P. subcapitata* growth inhibition was 5,300 μg/L, a 35-day NOEC for reproduction of Mysidae *A. bahia* was 232 μg/L, a 47-day NOEC for mortality of fish (fathead minnow) *P. promelas* was 278 μg/L, and a maximum 63-day NOEC for inhibition of emergence of Chironomus *C. tentans* was less than 2.3 μg/L. Based on these reliable chronic toxicity data, a PNEC was calculated at 23 μg/L with an assessment factor of 10. As the PNEC for the substance, a value of 23 μg/L obtained from the chronic toxicity for the crustacea was used.

The PEC/PNEC ratio was 0.5 for freshwater bodies and 0.001 for seawater bodies. Accordingly, efforts to gather information are thought to be necessary. As for this substance, some efforts are exerted to reduce releases to the environment; however, changes in environmental concentrations should be widely understood given the results of previous investigations.

Hazard assessment (basis for PNEC)			Assessment factor	Predicted no effect concentration PNEC (μg/L)	Exposure assessment		PEC/ PNEC ratio	Result of assessment
Species	Acute / chronic	Endpoint			Water body	Predicted environmental concentration PEC (μg/L)		
Crustacea (Mysidae)	Chronic	NOEC reproduction	10	23	Freshwater	11	0.5	▲
					Seawater	0.028	0.001	

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Risk cannot be identified. However, it would be required to collect information.	(▲)
	Inhalation exposure	Risk cannot be identified. However, it would be required to collect information for the ambient air.	(▲)
Ecological risk	Efforts to gather information are required. Changes in environmental concentrations should be widely understood.		▲

［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.