UNITED NATIONS

Food and Agriculture Organization of the United Nations

RC

UNEP/FAO/RC/CRC.13/13

Distr.: General
7 July 2017

English only

Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade

Chemical Review Committee
Thirteenth meeting
Rome, 23–27 October 2017
Item 5 (b) (xi) of the provisional agenda*

Technical work: review of notifications of final regulatory action: phorate

Phorate: notifications of final regulatory action

Note by the Secretariat

I. Introduction

1. In accordance with paragraph 5 of Article 5 of the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade, the Secretariat had previously received two notifications of final regulatory action for phorate that meet the requirements of Annex I to the Convention from Parties in the following two prior informed consent regions:

(a) Asia: Thailand (pesticide);[1]

(b) North America: Canada (pesticide).[2]

2. At its fifth meeting, the Chemical Review Committee reviewed the notifications and agreed that only the one received from Canada met the criteria set out in Annex II to the Convention. The notification from Canada and the rationale for the conclusion are set out in document UNEP/FAO/RC/CRC.13/INF/28.[3]

3. The Secretariat has since received an additional notification for phorate that meets the requirements of Annex I from a Party in the Latin America and Caribbean prior informed consent region: Brazil (pesticide).[4]

4. The notification received from Brazil is set out in the annex to the present note. Supporting documentation is set out in document UNEP/FAO/RC/CRC.13/INF/29.

II. Proposed action

5. The Committee may wish:

(a) To review the information provided in the latest notification and supporting documentation from Brazil related to phorate in accordance with the criteria set out in Annex II to the Convention;

* UNEP/FAO/RC/CRC.13/1.

[1] See PIC Circular XIV, December 2001.

[2] See PIC Circular XXVIII, December 2008.

[3] For supporting documentation, see UNEP/FAO/RC/CRC.5/9/Add.1.

[4] See the PIC Circular XLV, June 2017.

(b) If it concludes that the notification meets the criteria set out in Annex II to the Convention, to recommend to the Conference of the Parties that the chemical in question be made subject to the prior informed consent procedure and, accordingly, be listed in Annex III to the Convention, and to agree on a workplan for the preparation of a draft decision guidance document on phorate.

Annex

Notification of final regulatory action for phorate in the pesticide category submitted by Brazil

ROTTERDAM CONVENTION

SECRETARIAT FOR THE ROTTERDAM CONVENTION
ON THE PRIOR INFORMED CONSENT PROCEDURE
FOR CERTAIN HAZARDOUS CHEMICALS AND PESTICIDES
IN INTERNATIONAL TRADE

UNEP

FORM FOR NOTIFICATION
OF FINAL REGULATORY ACTION TO BAN OR SEVERELY RESTRICT A CHEMICAL

Country: Brazil

SECTION 1 IDENTITY OF CHEMICAL SUBJECT TO THE FINAL REGULATORY ACTION

1.1	**Common name**	Phorate
1.2	**Chemical name according to an internationally recognized nomenclature (e.g. IUPAC), where such nomenclature exists**	O,O-diethyl S-ethylthiomethyl phosphorodithioate
1.3	**Trade names and names of preparations**	Granutox and Granutox 150 G

1.4 Code numbers

1.4.1	CAS number	298-02-2
1.4.2	Harmonized System customs code	OSHA IMIS Code Number: 2064
1.4.3	Other numbers (specify the numbering system)	

1.5 Indication regarding previous notification on this chemical, if any

1.5.1 ☐ This is a first time notification of final regulatory action on this chemical.

1.5.2 ☒ This notification replaces all previously submitted notifications on this chemical.

Date of issue of the previous notification: 15 august 2016.

SECTION 2 FINAL REGULATORY ACTION

2.1 The chemical is: ☒ **banned** **OR** ☐ **severely restricted**

2.2 Information specific to the final regulatory action

2.2.1 Summary of the final regulatory action

Prohibition of all technical and formulated products based on phorate active ingredient. So, the production, use, trade, import and export of phorate had been banned.

2.2.2 Reference to the regulatory document, e.g. where decision is recorded or published

Resolution RDC nº 12 of March 13, 2015 issued by National Health Surveillance Agency (ANVISA).

2.2.3 Date of entry into force of the final regulatory action

March, 16, 2015

2.3 Category or categories where the final regulatory action has been taken

2.3.1 All use or uses of the chemical in your country prior to the final regulatory action

Insecticide authorized exclusively for agricultural use for the following crops: cotton, potato, coffee, beans and corn.

2.3.2 Final regulatory action has been taken for the category ☐ Industrial

Use or uses prohibited by the final regulatory action

Use or uses that remain allowed (only in case of a severe restriction)

2.3.3 Final regulatory action has been taken for the category ☒ Pesticide

Formulation(s) and use or uses prohibited by the final regulatory action

All uses

Formulation(s) and use or uses that remain allowed

(only in case of a severe restriction)

None

2.4 Was the final regulatory action based on a risk or hazard evaluation? ☒ **Yes**

☐ **No** (If no, you may also complete section 2.5.3.3)

2.4.1 If yes, reference to the relevant documentation, which describes the hazard or risk evaluation

Technical notes on the toxicological revaluation on the active ingredient phorate – prepared by National Health Surveillance Agency (ANVISA) with collaboration of Oswaldo Cruz Foundation (FIOCRUZ).

2.4.2 Summary description of the risk or hazard evaluation upon which the ban or severe restriction was based.

2.4.2.1 Is the reason for the final regulatory action relevant to human health? ☒ Yes

☐ No

If yes, give summary of the hazard or risk evaluation related to human health, including the health of consumers and workers

Phorate was an insecticide authorized in Brazil exclusively for agricultural use in cotton, potato, coffee, beans and corn. In 2008 Brazilian Health Surveillance Agency (ANVISA) initiated the toxicological reassessment of phorate due to evidences of high acute toxicity and neurotoxicity of this active ingredient of pesticides.

Brazilian law predicts that pesticides may have their registrations cancelled in the country when they fall under the following conditions related to human health: when they have no antidote or effective treatment in Brazil; if found teratogenic, mutagenic or carcinogenic; if they cause hormonal disturbances and damage to the reproductive system or if they are

more dangerous to humans than demonstrated in tests with laboratory animals.

Phorate and its metabolites are easily absorbed through skin and mucous membranes and irreversibly block the catalytic activity of acetylcholinesterase (AChE), the enzyme responsible for mediating the hydrolysis of acetylcholine in acetic acid and choline acid. Thus, they interrupt the transmission of nerve impulses in the cholinergic synapses of the central nervous system (CNS), autonomic nervous system (ANS) and neuromuscular junction. Inactivation of AChE causes cholinergic hyperstimulation by acetylcholine accumulation in the synaptic cleft.

Phorate is considered one of the most toxic organophosphate AChE inhibitors, with mean oral LD50 for mice ranging from 1.4 to 10 mg/kg body weight.

Phorate can cause complex neurological clinical manifestations in humans, such as encephalopathy, intermediate syndrome and delayed polyneuropathy, described by various authors (Young, Jung; Ayer, 1979; Kashyap et al., 1984;. WHO / FAO, 1988; Kusic et al., 1991; Dobozy, 1998; Das and Jena, 2000; Thanal, 2001; Jayakumar, 2002; Mission, 2006; Peter; Prabhakar; Pichamuthu, 2008a; 2008b).

However, in laboratory animals that received phorate there were no cases of intermediate syndrome or late polyneuropathy, what shows this pesticide is more toxic to humans than demonstrated in tests with laboratory animals, a prohibitive criterion for registration of pesticides in Brazil.

Besides its neurotoxic effects, phorate demonstrated potential to cause adverse effects to the endocrine regulation processes of steroid hormones in humans (Usmani, 2003), which may contribute to increased cancer cases (Alavanja, et al., 2002; Mahajan et al., 2006; Koutros et al., 2010).

With regard to human exposure, Usha and Harikrishnan (2004) reported several cases of acute poisoning in communities of Kerala, Índia. Among these cases, 5 of them, occurred between 1999 e 2002, are associated to exposure to phorate. According to the authors, in july 1999, about 12 people living in banana crop areas were severely poisoned by phorate. After the product use, it rained on the region, causing the product evaporate quickly and spread to nearby area, reaching the homes. Shortly after application of the product, the symptoms appeared and the affected required hospitalization. In June 2001, a 16 year-old boy died as a result of occupational exposure to phorate for a period of one week. That same year, 40 rural women workers in a tea plantation were intoxicated during harvesting. Symptoms appeared within 30 minutes after exposure, featured by lightheadedness, dizziness, blurred vision, vomiting. Thirty seven women had more severe and remained hospitalized for two days. The authors point out that in July 2002, 31 children from an upper primary school were poisoned by phorate applied in plantation nearby school.

The children showed persistent headache, chest pain, breathing difficulty, nausea, giddiness, blurring of vision and stomach pain, and one of them showed uncontrolled muscle twitching and convulsions even after 24 hours of treatment.

On 21 July 2006, 20 residents of Salkiana village, district Jalandhar, India, had to be rushed to a hospital when neurotoxic symptoms of acute exposure to phorate were observed. The

product was used in a nearby sugarcane field. The worst affected were the school children of an Elementary School. Teachers and students started complaining of a strange smell and breathlessness. Suddenly one student fell unconscious and then students started to faint. Within ten minutes, 16 students fainted after inhaling something that was toxic. In addition to difficulty breathing, the most frequent symptoms were poorly being, headache, eye irritation, dizziness, nausea, vomiting, lacrimation, salivation excessive, muscle cramps and pain. Six days after exposure to phorate, several patients still had symptoms such as eye irritation, dermal reactions and general uneasiness. (Mission, 2006).

Several studies show that agricultural workers exposed to phorate are victims of poisonings and deaths related to toxicity characteristics of the active ingredient. The exposure becomes even more dangerous due to the difficulties relating to the availability and/or inefficiency of PPE. Moreover, this various social issues (low education, low income) and biological (age and gender) are factors that increase the risk and severity of poisoning caused by this organophosphate.

Therefore, from the reevaluation of the health effects of phorate, completed in 2015, ANVISA concluded this active ingredient of pesticides has the potential to cause hormonal disturbances in humans and is more toxic to humans than demonstrated in tests with laboratory animals, which are prohibitive criteria for registration of pesticides in Brazil.

Phorate was banned in Brazil on March 16, 2014, where it was no longer marketed since 2011.

References

- ALAVANJA, M.C.R. *et. al.* Use of Agricultural Pesticides and Prostate Cancer Risk in the Agricultural Health Study Cohort. American Journal of Epidemiology, v.157, n.9, 2002.

- DAS, B.; JENA, R. K. Encephalopathy, intermediate syndrome and delayed polyneuropathy in acute black danadar (Phorate 10 CG) poisoning. JAPI, v. 48, n.5, p. 540-541, 2000.

- DOBOZY, V. Phorate - Review of pesticide poisoning incident data. 29 jul. 1998. Memorandum. 07 p. <http://www.epa.gov/pesticides/foia/reviews/057201/057201-031.pdf>.

- JAYAKUMAR, U. Children in Kerala poisoned by banana pesticide. Pesticides News, n. 57, p. 19, 2002. <http://www.pan-uk.org/pestnews/Issue/pn57/pn57p19b.htm>

- KASHYAP, S. K.; JANI, J. P.; SAIYED, H. N.; GUPTA, S. K. Clinical effects and cholinesterase activity changes in workers exposed to phorate (Thimet). J Environ Sci Health B, v. 19, p. 479-489, 1984.

- KOUTROS S. *et al.* Pesticide Use Modifies the Association Between Genetic Variants on Chromosome 8q24 and Prostate Cancer. Cancer Res, v.70, p.9224-9233, 2010.

- KUSIC, R.; JOVANOVIC, D.; RANDJELOVIC, S.; JOKSOVIC, D.; TODOROVIC, V.; BOSKOVIC, B.; JOKANOVIC, M.; VOJVODIC. V. HI-6 in man: efficacy of the oxime in poisoning by organophosphorus insecticides. Hum Exp Toxicol, v. 10, n. 2, p.

113-118, 1991.
- MAHAJAN, R, *et al.* Phorate exposure and incidence of cancer in the agricultural health study.Environ Health Perspect. v. 114, n. 8, p. 1205-9, 2006.
- MISSION, K. V. Pesticide Spray Proves Disastrous In Salkiana Village, Jalandhar. 2006. <http://www.worldproutassembly.org/archives/2006/08/pesticide_spray.html>
- PETER, J. V.; PRABHAKAR, A. T.; PICHAMUTHU, K. Delayed-onset encefalopathy and coma in acute organophosphate poisoning in humans. NeuroToxicology, v. 29, p. 335-342, 2008a.
- PETER, J. V.; PRABHAKAR, A. T.; PICHAMUTHU, K. In-laws, insecticide - and a mimic of brain death. The Lancet, v.371, p. 622, 2008b.
- THANAL. Phorate poisoning in India. Pesticides News, n. 53, p. 5, 2001. <http://www.pan-uk.org/pestnews/Issue/pn53/pn53p5.htm>
- USMANI, K. A.; ROSE, R.L.; HODGSON, E. Inhibition and activation of the human liver microsomal and human cytochrome P450 3A4 metabolism of testosterone by deployment-related chemicals. Drug Metab Dispos, v.31, n.4, p.384-91, 2003.
- YOUNG, R. J.; JUNG, F. P.; AYER, H. E. Phorate intoxication at an insecticide formulating plant. Am Ind Hyg Assoc J, v. 40, n. 11, p. 1013-1016, 1979.
- WORLD HEALTH ORGANIZATION/FOOD AND AGRICULTURE ORGANIZATION (WHO/FAO) (EUA). Phorate. Data sheets on pesticides n. 75. 1988.

Expected effect of the final regulatory action

Eliminate the risks posed by phorate

2.4.2.2 Is the reason for the final regulatory action relevant to the environment?

☐ Yes

☒ No

If yes, give summary of the hazard or risk evaluation related to the environment

Expected effect of the final regulatory action

2.5 Other relevant information regarding the final regulatory action

2.5.1 Estimated quantity of the chemical produced, imported, exported and used

	Quantity per year (MT)	Year
produced	Formulated Product (Final Product): 153,9 t	2009
imported	Active Ingredient: 17,15 t	2009

exported	Active Ingredient: 35,96 t	2011
used	2009: Active Ingredient Sells: 26,49 t 2009: Formulated Product (Final Product) Sells: 272,58 t 2010: Formulated Product (Final Product) Sells: 6,72 t 2011: Formulated Product (Final Product) Sells: 0,01 t 2012, 2013, 2014 and 2015: no production, import, export and sells.	

2.5.2 Indication, to the extent possible, of the likely relevance of the final regulatory action to other states and regions

Similar health and environmental problems are likely to be encountered in other countries where the substance is used.

2.5.3 Other relevant information that may cover:

2.5.3.1 Assessment of socio-economic effects of the final regulatory action

2.5.3.2 Information on alternatives and their relative risks, e.g. IPM, chemical and non-chemical alternatives

The alternatives to phorate applied in cotton crops in Brazil are: acephate, acetamiprid, benfuracarb, methidathion, esfenvarelate, imidacloprid, thiacloprid, permethrin, cypermethrin, azadirachtin, cyfluthrin, pymetrozine, methomyl, beta-cyfluthrin, flonicamid, chlorpyrifos, bifenthrin, deltamethrin, dimethoate, carbosulfan, clothianidin, zeta-cypermethrin, triazophos, fenthion, malathion, diafenthiuron, furathiocarb, thiodicarb, fenvarelate and fenitrothion. The alternatives to phorate applied in potato crops in Brazil are: acephate, acetamiprid, benfuracarb, esfenvarelate, imidacloprid, thiacloprid, alfa-cypermethrin, pymetrozine, methomyl, beta-cyfluthrin, chlorpyrifos, bifenthrin, deltamethrin, carbosulfan, beta-cypermethrin, piridafenthion, diafenthiuron, fipronyl, clorantraniliprole, cadusafos, tebupirinfos, lambda cyalotrine, gama-cyalotrine and chlorphenapir. The alternatives to phorate applied in coffee crops in Brazil are: esfenvarelate, imidacloprid, permethrin, cypermethrin, azadirachtin, cyfluthrin, beta-cyfluthrin, chlorpyrifos, zeta-cypermethrin, alfa-cypermethrin, beta-cypermethrin, novaluron, abamectin, clorantraniliprole, teflubenzuron, lufenuron, cyantraniliprole, pyriproxyfen, fenpropathrin, gamma-cyhalothrin, lambda-cyihalotrin and fluvarinate. The alternatives to phorate applied in bean crops in Brazil are: thiodicarb, imidacloprid, malathion, chlorpyriphes, esfenvarelate, acetate, acetamiprid, bifenthrin, beta-cyfluthrin, thiacloprid, phenopopation, clothanidine, carbosulfan, permethrin and etofenprox. The alternatives to phorate applied in corn crops in Brazil are: chlorpyriphes, fipronyl,

bifenthrin and imidacloprid.

2.5.3.3 Basis for the final regulatory action if other than hazard or risk evaluation

2.5.3.4 Additional information related to the chemical or the final regulatory action, if any

SECTION 3 PROPERTIES

3.1 Information on hazard classification where the chemical is subject to classification requirements

International classification systems e.g. WHO, IARC, etc.

	Hazard class
WHO	Ia - Extremely hazardous

Other classification systems e.g. EU, USEPA

	Hazard class
USEPA	I - Highly toxic

3.2 Further information on the properties of the chemical

3.2.1 Description of physico-chemical properties of the chemical

Molecular Formula	C7H17O2PS3
Molecular Weight	260,38 *g moL*$^{-}$

Water solubility	50 mg/l @ 25 deg C
Melting point	-42,9ºC
Boiling point	125-127 deg C @ 2.0 mm Hg

Reference

PubChem- https://pubchem.ncbi.nlm.nih.gov/

3.2.2 Description of toxicological properties of the chemical

Summary of critical end-points for phorate

Absorption, distribution, excretion and metabolism in animals

Rate and extent of oral absorption: Rapid, approximately 90% within 24 h

Dermal absorption: Extensive based on acute toxicity

Distribution: Rapid and extensive

Potential for accumulation: None

Rate and extent of excretion: 89% within 24 h; urinary excretion predominated (77%); faecal excretion (12%).

Metabolism in animals - Major pathway: cleavage of phosphorus–sulfur bond, methylation of the liberated thiol group and oxidation of the resulting divalent moiety to the sulfoxide and sulfone.

Toxicologically significant compounds (plants, animals and the environment): Parent, phorate sulfoxide and phorate sulfone

Acute toxicity

Rat, LD50, oral 3.7 mg/kg bw in males, 1.4 mg/kg bw in females

Rat, LD50, dermal 9.3 mg/kg bw in males, 3.9 mg/kg bw in females

Rat, LC50, inhalation 0.06 mg/l of air in males (1 h), 0.011 mg/l of air (1 h) in females

Rabbit, dermal irritation Highly toxic by skin contact—could not be tested

Rabbit, ocular irritation Highly toxic by eye contact—could not be tested

Dermal sensitization Highly toxic by skin contact—could not be tested

Short-term studies of toxicity

Target/critical: effect Brain and erythrocyte acetylcholinesterase activity, and miosis (rats)

Lowest relevant oral NOAEL: 0.07 mg/kg bw per day

Lowest relevant dermal NOAEL: No data

Lowest relevant inhalation NOAEC: No data

Genotoxicity Negative results in vivo and in vitro

Long-term studies of toxicity and carcinogenicity

Target/critical effect: Inhibition of erythrocyte and brain cholinesterase activity

Lowest relevant NOAEL: 0.07 mg/kg per day (rat)

Carcinogenicity: Not carcinogenic in mice and rats

Reproductive toxicity

Reproduction target/critical effect: Reduced pup growth at maternally toxic dose

Lowest relevant reproductive NOAEL: 2 ppm, equivalent to 0.17 mg/kg bw per day

Developmental target/critical effect: Decreased pup weights and delayed ossification at maternally toxic doses (rats)

Lowest relevant developmental NOAEL: 0.3 mg/kg bw per day (rats)

Neurotoxicity/delayed neurotoxicity

Single dose study of neurotoxicity

Target/critical effect: Signs consistent with acetylcholinesterase inhibition; no neuropathological effects

Relevant NOAEL: 0.25 mg/kg bw

Delayed neuropathy: No delayed neurotoxicity in hens

Medical data: Findings consistent with inhibition of acetylcholinesterase activity; no record of permanent sequelae

Reference

JMPR/FAO Report 2004

3.2.3 Description of ecotoxicological properties of the chemical

Ecological Effects:

Effects on birds: Phorate is very highly toxic to birds. The reported acute oral LD50 values are 12.8 mg/kg in chukar, 7.5 mg/kg in starlings, 0.6 to 2.5 mg/kg in mallards, 7 to 21 mg/kg in northern bobwhite quail, 1 mg/kg in red-winged blackbirds, and 7 mg/kg in ring-neck pheasants. The 5- to 8-day dietary LC50 values are reported as 370 to 580 ppm in Japanese quail, mallard, northern bobwhite quail, and ring-neck pheasant.

Effects on aquatic organisms: Phorate is very highly toxic to fish. Reported 96-hour LC50 values range from 2 to 13 ug/L in cutthroat trout, bluegill sunfish and largemouth bass. Other 96-hour LC50 values are 110 ug/L in northern pike and 280 ug/L in channel catfish. Reported 96-hour LC50 values for the compound in freshwater invertebrates such as stoneflies and scuds are 4 ug/L, also indicating very high toxicity. Other LC50 values are 0.006 ug/L for amphipods and 0.11 to 1.9 ug/L in other freshwater invertebrates. The acute oral LD50 of phorate is 85 mg/kg in bullfrogs.

Effects on other organisms: Phorate is toxic to bees, with a reported topical application LD50 of 10 ug per bee.

Environmental Fate:

Breakdown in soil and groundwater: Phorate is of moderate persistence in the soil environment, with reported field half-lives of 2 to 173 days. A representative value may be approximately 60 days. Actual residence times may be influenced by soil clay and organic matter content, rainfall, and soil pH. Soil treatments often leave more residues in plants than foliar treatments, because the compound persists in the soil and is readily taken up by plant roots. Phorate binds moderately well to most soils and is slightly soluble in water. It should therefore not be highly mobile in most soils, and should mainly be transported with runoff via sediment and water. Phorate has minimal potential to leach through the soil and contaminate groundwater. This is most likely where soils are sandy and

aquifers are shallow. Field studies indicate that leaching is very low in soils high in clay and organic matter content, and lower in sandy soils.

Breakdown in water: The half-life of phorate in acidic water solutions is between a few days and a few weeks, depending on temperature; the half-life in alkaline (basic) water may be much shorter. Phorate is degraded by waterborne microorganisms and hydrolysis. As it breaks down in water, nontoxic, water-soluble products are formed.

Breakdown in vegetation: Phorate itself is not persistent in plants, but plants metabolize phorate to very potent anticholinesterase agents such as the sulfoxide and sulfone derivatives of the compound. This activity will usually peak several days following application before decreasing. Phorate and its soil metabolites are absorbed from the soil by plant roots and are translocated to above-ground portions of the plant. Following treatment with a 10% granular formulation at 1 pound a.i./acre, phorate residues persisted at very low levels for 28 days in the kernels, cobs, or husks. After 83 days, there were no detectable residues of phorate or breakdown products.

Reference

http://extoxnet.orst.edu/pips/phorate.htm

SECTION 4 DESIGNATED NATIONAL AUTHORITY

Institution	Ministry of Environment
Address	Department of Environmental Quality in Industry. Secretariat of Climate Change and Environmental Quality Esplanada dos Ministérios, Bloco B, 8º andar, Gabinete
Name of person in charge	Letícia Reis de Carvalho
Position of person in charge	Director
Telephone	+55 61 2028 2073
Telefax	+55 61 2028 2908
E-mail address	leticia.carvalho@mma.gov.br, gsq@mma.gov.br

Date, signature of DNA and official seal: _____ 19/12/16

Letícia Reis de Carvalho
Diretora do Departamento de
Qualidade Ambiental na Indústria

PLEASE RETURN THE COMPLETED FORM TO:

Secretariat for the Rotterdam Convention
Food and Agriculture Organization
of the United Nations (FAO)
Viale delle Terme di Caracalla
00153 Rome, Italy
Tel: (+39 06) 5705 2188
Fax: (+39 06) 5705 6347
E-mail: pic@pic.int

OR

Secretariat for the Rotterdam Convention
United Nations Environment
Programme (UNEP)
11-13, Chemin des Anémones
CH – 1219 Châtelaine, Geneva, Switzerland
Tel: (+41 22) 917 8296
Fax: (+41 22) 917 8082
E-mail: pic@pic.int

Definitions for the purposes of the Rotterdam Convention according to Article 2:

(a) 'Chemical' means a substance whether by itself or in a mixture or preparation and whether manufactured or obtained from nature, but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial;

(b) 'Banned chemical' means a chemical all uses of which within one or more categories have been prohibited by final regulatory action, in order to protect human health or the environment. It includes a chemical that has been refused approval for first-time use or has been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process and where there is clear evidence that such action has been taken in order to protect human health or the environment;

(c) 'Severely restricted chemical' means a chemical virtually all use of which within one or more categories has been prohibited by final regulatory action in order to protect human health or the environment, but for which certain specific uses remain allowed. It includes a chemical that has, for virtually all use, been refused for approval or been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process, and where there is clear evidence that such action has been taken in order to protect human health or the environment;

(d) 'Final regulatory action' means an action taken by a Party, that does not require subsequent regulatory action by that Party, the purpose of which is to ban or severely restrict a chemical.