**Rotterdam Convention**

**Operation of the prior informed consent procedure
for banned or severely restricted chemicals**

**Decision Guidance Document**

**Trichlorfon**



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

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**Introduction**

The objective of the Rotterdam Convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm and to contribute to their environmentally sound use, by facilitating information exchange about their characteristics, by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties. The Secretariat of the Convention is provided jointly by the United Nations Environment Programme (UNEP) and the Food and Agriculture Organization of the United Nations (FAO).

Candidate chemicals[[1]](#footnote-1)1 for inclusion in the prior informed consent (PIC) procedure under the Rotterdam Convention include those that have been banned or severely restricted by national regulatory actions in two or more Parties[[2]](#footnote-2)2 in two different regions. Inclusion of a chemical in the PIC procedure is based on regulatory actions taken by Parties that have addressed the risks associated with the chemical by banning or severely restricting it. Other ways might be available to control or reduce such risks. Inclusion does not, however, imply that all Parties to the Convention have banned or severely restricted the chemical. For each chemical included in Annex III of the Rotterdam Convention and subject to the PIC procedure, Parties are requested to make an informed decision whether they consent or not to the future import of the chemical.

At its eighth meeting, held in Geneva from 24 April to 5 May 2017, the Conference of the Parties agreed to list trichlorfon in Annex III to the Convention and adopted the decision-guidance document with the effect that this chemical became subject to the PIC procedure.

The present decision-guidance document was communicated to designated national authorities on15 September 2017, in accordance with Articles 7 and 10 of the Rotterdam Convention.

**Purpose of the decision guidance document**

For each chemical included in Annex III of the Rotterdam Convention, a decision-guidance document has been approved by the Conference of the Parties. Decision-guidance documents are sent to all Parties with a request that they make a decision regarding future import of the chemical.

Decision-guidance documents are prepared by the Chemical Review Committee. The Committee is a group of government-designated experts established in line with Article 18 of the Convention, which evaluates candidate chemicals for possible inclusion in Annex III of the Convention. Decision-guidance documents reflect the information provided by two or more Parties in support of their national regulatory actions to ban or severely restrict the chemical. They are not intended as the only source of information on a chemical nor are they updated or revised following their adoption by the Conference of the Parties.

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical and others that have not banned or severely restricted it. Risk evaluations or information on alternative risk mitigation measures submitted by such Parties may be found on the Rotterdam Convention website (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Rotterdam Convention website.

Information on the chemical may also be available from other sources.

**Disclaimer**

The use of trade names in the present document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any particular company. As it is not possible to include all trade names presently in use, only a number of commonly used and published trade names have been included in the document.

While the information provided is believed to be accurate according to data available at the time of preparation of the present decision-guidance document, FAO and UNEP disclaim any responsibility for omissions or any consequences that may arise there from. Neither FAO nor UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

| **STANDARD CORE SET OF ABBREVIATIONS** |
| --- |
| < | less than |
| < | less than or equal to |
| > | greater than |
| > | greater than or equal to |
|  |  |
| µg | microgram |
| μm | micrometre |
|  |  |
| ARfD | acute reference dose |
| a.i. | active ingredient |
| AChE | acetylcholinesterase |
| ADI | acceptable daily intake |
| ANVISA | National Health Surveillance Agency of Brazil |
| AOEL | acceptable operator exposure level |
| AR | applied radioactivity |
|  |  |
| b.p. | boiling point |
| bw | body weight |
|  |  |
| °C | degree Celsius (centigrade) |
| CAS | Chemicals Abstracts Service |
| cc | cubic centimetre |
| cm | centimetre |
| CXL | Codex Maximum Residue Limit (Codex Alimentarius MRL) |
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| DAR | Draft Assessment Report |
| DCAA | dichloroacetaldehyde |
| DDVP | 2,2-dichlorovinyl dimethyl phosphate (dichlorvos) |
| DNA | Deoxyribose Nucleic Acid |
| DT50 | dissipation time 50 % |
|  |  |
| EC | European Community |
| EC50 | median effective concentration |
| ED50 | median effective dose |
| EEC | European Economic Community |
| EFSA | European Food Safety Authority |
| EHC | Environmental Health Criteria |
| EPA | Environmental Protection Agency |
| EU | European Union |
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| FAO | Food and Agriculture Organization of the United Nations |
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| g | gram |
| GHS | United Nations Globally Harmonized System of classification and labelling of chemicals |
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| h | hour |
| ha | hectare |
|  |  |
| i.m. | intramuscular |
| i.p. | intraperitoneal |
| IARC | International Agency for Research on Cancer  |
| IC50 | inhibition concentration, 50%; |
| ILO | International Labour Organisation |
| IPCS | International Programme on Chemical Safety |
| IPM | Integrated Pest Management |
| ISO | International Organization for Standardization |
| IUPAC | International Union of Pure and Applied Chemistry |
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| JECFA | Joint FAO/WHO Expert Committee on Food Additives |
| JMPR | Joint FAO/WHO Meeting on Pesticide Residues (Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and a WHO Expert Group on Pesticide Residues) |
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| k | kilo- (x 1000) |
| kg | kilogram |
| Koc | organic carbon normalized partition coefficient (soil/water) |
| Kow | octanol-water partition coefficient |
| kPa | kilopascal |
|  |  |
| L | Litre |
| LC50  | median lethal concentration |
| LD50 | median lethal dose |
| LOAEL | lowest observed adverse effect level |
| LOEL | lowest observed effect level |
| LR50 | median lethal rate |
|  |  |
| m | metre |
| m.p. | melting point |
| mg | milligram |
| ml | millilitre |
| mPa | milliPascal |
| MRL | maximum residue limit |
| MTD | maximum tolerable dose |
|  |  |
| ng | nanogram |
| NOAEC | no-observed-adverse-effect concentration |
| NOAEL | no-observed-adverse-effect level |
| NOEC | no-observed-effect concentration |
| NOEL  | no-observed-effect level |
|  |  |
| OECD | Organisation for Economic Co-operation and Development  |
|  |  |
| PEC | predicted environmental concentration |
| Pow | octanol-water partition coefficient, also referred to as Kow |
| PPE | personal protective equipment |
| ppm | parts per million (used only with reference to the concentration of a pesticide in an experimental diet. In all other contexts the terms mg/kg or mg/L are used). |
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| RfD | reference dose (for chronic oral exposure, comparable to ADI) |
| RMS | Rapporteur Member State |
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| SMR | standardized mortality ratio |
| STEL | short term exposure limit |
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| TER | toxicity exposure ratio |
| TLV | threshold limit value |
| TWA | time weighted average |
| UK | United Kingdom |
| UNEP | United Nations Environment Programme |
| USEPA | United States Environmental Protection Agency |
| UV | ultraviolet |
|  |  |
| VOC | volatile organic compound |
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| w/w | weight per weight |
| WHO | World Health Organization |
| wt | weight |

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| **Decision guidance document for a banned or severely restricted chemical** |

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| ***Trichlorfon*** | **Published: September 2017** |
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| **1.** **Identification and uses (see Annex 1 for further details)**  |
| **Common name** | Trichlorfon |
| **Chemical name and other names or synonyms** | ISO: trichlorfonIUPAC: dimethyl (RS)-2,2,2-trichloro-1-hydroxyethylphosphonateCAS: Phosphonic acid, (2,2,2-trichloro-1-hydroxyethyl)-, dimethyl esterTrichlorfon is a racemic mixture of the R and S enantiomers (The e-Pesticide Manual 2011-2012).Synonyms: trichlorphon, metriphonate, metrifonate, chlorophos, DEP, dipterex (The e-Pesticide Manual 2011-2012)A long list of synonyms is included in IARC (1987). |
| **Molecular formula** | C4H8Cl3O4P |
| **Chemical structure** |  |
| **CAS-No.(s)** | 52-68-6 |
| **Harmonized System Customs Code** | 2931 90 |
| **Other numbers** | CIPAC No: 68EEC No. (EINECS or ELINCS): 200-149-3OPP chemical code: 057901 |
| **Category** | Pesticide |
| **Regulated category** | Pesticide |
| **Use(s) in regulated category** | Trichlorfon was used in the European Union primarily as an insecticide for the control of lepidopterous insects in tomatoes. It also has some acaricidal properties. In Brazil, trichlorfon was registered for pesticidal use in aerial parts of the following crops: avocado, pineapple, squash, lettuce, alfalfa, cotton, prunes, peanuts, rice, banana, eggplant, broccoli, cocoa, coffee, cashew nuts, sugar cane, persimmon, carrot, chicory, citrus, coconut, cauliflower, carnation, peas, beans, figs, custard apple, sunflower, guava, apple, mango, quince, melon, cantaloupe, corn, pastures, cucumber, pear, peach, peppers, cabbage, rose, rubber, soybeans, tomatoes, wheat and grapes. |
| **Trade names** | Trade names listed by EU: Cekufon 80 SPTrade names listed by Brazil: Dipterex Br Técnico, Dipterex 500, Trifonal 500Other trade names listed in the Pesticide Manual are: Saprofon, Susperex, Danex, Dipagrex, Diplox, Dipsol, Ledipex, Dylox, Tugon, Briten, Denkaphon, Ditrifon, Lucavex and Proxol.Additional trade names listed in IPCS / CEC (2005) are: Acrol, DEP and DIMETOX.*This is an indicative list. It is not intended to be exhaustive.*  |
| **Formulation types** | Cekufon 80 SP, a soluble powder (SP) formulation, was registered under different trade names in Europe.Other formulations listed in the Pesticide Manual are: dustable powder (DP), granular bait (GB), granule (GR), pour-on (PO), suspension concentrate (SC), soluble concentrate (SL), soluble powder (SP), ultra-low volume liquid (UL), wettable powder (WP) and coating agent. |
| **Uses in other categories** | There is no reported use as an industrial chemical. |
| **Basic manufacturers** | Bayer CropScience, Cequisa, ChemChina Agrochemical, Dacheng, Hubei Sanonda, Lanxi, Makhteshim-Agan, Nantong Jiangshan, Saeryung Sanonda, Zhengzhou (The e‑Pesticide Manual 2011-2012)*This is an indicative list of current and former manufacturers. It is not intended to be exhaustive.* |

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| **2. Reasons for inclusion in the PIC procedure** |
| Trichlorfon is included in the PIC procedure as a pesticide. It is listed on the basis of the final regulatory actions taken by the European Union[[3]](#footnote-3)3 and Brazil to ban trichlorfon as a pesticide.No final regulatory actions relating to industrial chemical uses have been notified. |
| **2.1 Final regulatory action (see Annex 2 for further details)** |
| **European Union**Trichlorfon is not included in the list of authorised active ingredients in Annex I to Directive 91/414/EEC (which has been replaced by Regulation (EC) No. 1107/2009).Complete entry into force of the final regulatory action (Commission Decision 2007/356/EC dated 21 May 2007) was 21 November 2008 since all uses of plant protection products containing trichlorfon were prohibited as of that date. Authorisations for plant protection products containing trichlorfon had to be withdrawn by 21 November 2007 by EU Member States. As of 25 May 2007, no authorisations for plant protection products containing trichlorfon were allowed to be granted or renewed by the Member States. |
| **Reason:** | Human Health and Environment |
| **Brazil**The legal reference for the pesticide management in Brazil is Law Nº 7.802/89 (Pesticide Law), regulated by Decree 4.074/02. The final regulatory action (Resolution-RDC No. 37 of 16 August 2010: Technical regulation on the active ingredient trichlorfon as a result of a toxicological re-evaluation) was based on results of a toxicological re-evaluation and resulted in a ban of all uses of trichlorfon-based products in plant protection (as an agricultural pesticide). The decision was based on the Technical Note of Toxicological Reassessment on trichlorfon commissioned by the National Health Surveillance Agency (ANVISA). The decision entered into force on 18 August 2010 and prevents future registrations of this pesticide. |
| **Reason:** | Human Health |

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| **2.2 Risk evaluation (see Annex 1 for further details)** |

**European Union**

The risk evaluation in the EU took into account the proposed conditions of use within the EU, including the intended uses, the recommended application rates and the good agricultural practices. The conclusion on the peer review was reached on the basis of the evaluation of the representative use in tomatoes in greenhouses in the EU.

For a number of aspects in the evaluation process, final conclusions were not possible due to the lack of reliable data. However, evaluations made by the RMS and during EFSA expert meetings on the basis of the available information demonstrated that the following concerns for human health and the environment were likely to occur under the proposed conditions of use in the EU.

**Human health**

Trichlorfon is classified as harmful during oral exposure and as a skin sensitizer. The most sensitive effect observed during short-term exposure was the reduction in AChE activity.

Taking into account physical and chemical properties of trichlorfon, experts considered the default dermal absorption value of 100 % appropriate for the risk evaluation. Based on the provisional AOEL provided by the RMS in the DAR, together with the dermal absorption value of 100%, exposure models concluded that the operator, worker and bystander exposure estimates exceeded the AOEL to a large extent. The models took into account in their input parameters the conditions prevailing in the EU (*e.g.* maximum applied dose, mode of application).

Further, trichlorfon is metabolized into dichlorvos, which is also an impurity of toxicological concern in trichlorfon. Dichlorvos has been identified as a carcinogen category 2 by IARC in 2004. The potential evaporation of dichlorvos from the plant was shown to be more than 30% of the applied trichlorfon. This could be relevant for worker exposure by inhalation.

**Environment**

The data submitted was insufficient for a conclusive evaluation of fate and behaviour of trichlorfon and its metabolites in the environmental compartments. However, as a result of the evaluation on fate and behaviour of trichlorfon, surface water contamination from glasshouse use could not be excluded. For this reason, an evaluation of the risks to aquatic organisms was considered necessary. EFSA experts on ecotoxicology agreed that *Daphnia magna* was the most sensitive species, being more than one order of magnitude more sensitive than other aquatic species. Based on the existing study with *Daphnia magna*, a high risk for aquatic invertebrates was identified.

**Brazil**

In 2008, a re-evaluation of trichlorfon was initiated because there were concerns about possible risks for human health and the environment.

The environmental part of the re-evaluation could not be completed by the Brazilian Institute of Environmental and Renewable Natural Resources (IBAMA) because no data was provided to allow a conclusion on environmental risks. The Ministry of Agriculture, Livestock and Food Supply (MAPA) thus announced by an administrative act in February 2010 that the registrations of the three trichlorfon-based pesticides were cancelled, because the registrations could not be maintained without a valid environmental evaluation as a necessary element of the registration. However, only upon completion of the toxicological assessment the re-evaluation (which identified concerns for human health) could be concluded. As a consequence, ANVISA finally cancelled the trichlorfon monograph and banned the import of trichlorfon by Resolution RDC 37/2010 of 16 August 2010. This final regulatory action established the definitive prohibition of registration of pesticides containing trichlorfon.

**Human health**

The final regulatory action was based on the results of the toxicological review of trichlorfon, which describes this pesticide as causing acute neurotoxic, genotoxic, immunotoxic, carcinogenic, and teratogenic effects. In addition, trichlorfon affects reproduction and the endocrine system.

Studies on poisoning incidents in Brazil have shown that pesticide poisonings, especially with organophosphorous pesticides, occurred in different regions of Brazil. There is a large underreporting of pesticide poisoning incidents in Brazil. According to a study from the Amazon area of Brazil, agricultural workers were not prepared to use pesticides (including trichlorfon) correctly. They were not sufficiently aware of the risks of pesticides to human health and the environment. This study further concludes that farmers did not use protective clothing or equipment because it was expensive and not suitable for a tropical climate. Due to lack of training and poor knowledge of pesticide hazards, pesticides were handled carelessly during preparation, application and disposal of empty packages. Exposure of farmers, their families, consumers (via residues in food) and the environment was thus high.

Although no poisoning incidents with trichlorfon itself have been reported from Brazil, the decision to ban trichlorfon was taken on the basis of the evaluation of its hazardous properties as well as on expected exposure of agricultural workers to pesticides in general and also to trichlorfon under conditions of use in Brazil.

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| **3. Protective measures that have been applied concerning the chemical**  |

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| **3.1 Regulatory measures to reduce exposure** |
| **European Union** | The ban of trichlorfon as an active ingredient in plant protection products reduces the exposure of operators and the environment.Complete entry into force of the final regulatory action (Commission Decision 2007/356/EC dated 21 May 2007) was 21 November 2008 since all uses of plant protection products containing trichlorfon were prohibited as from that date. Authorisations for plant protection products containing trichlorfon had to be withdrawn by 21 November 2007 by EU Member States. As of 25 May 2007, no authorisations for plant protection products containing trichlorfon were allowed to be granted or renewed by the EU Member States. |
| **Brazil** | The final regulatory action cancelled the trichlorfon monograph and banned the import of trichlorfon by Resolution RDC 37/2010 of 16 August 2010. This established the definitive prohibition of registration of pesticides containing trichlorfon. Use, research in all stages, production, packaging, labelling, transport and export of trichlorfon is also prohibited. |
| **3.2 Other measures to reduce exposure** |

**European Union**

None reported

**Brazil**

None reported

**General**

None

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| **3.3 Alternatives**  |
| *It is essential that before a country considers substituting alternatives, it ensures that the use is relevant to its national needs, and the anticipated local conditions of use. The hazards of the substitute materials and the controls needed for safe use should also be evaluated.***European Union**None reported**Brazil**None reported**General**There are a number of alternative methods involving chemical and non-chemical strategies, including alternative technologies available, depending on the individual crop-pest complex under consideration. Countries should consider promoting, as appropriate, integrated pest management (IPM) and organic strategies as a means of reducing or eliminating the use of hazardous pesticides.Advice may be available through National IPM focal points, the FAO, IFOAM (International Federation of Organic Agriculture Movements), and agricultural research or development agencies. Where it has been made available by governments, additional information on alternatives to trichlorfon may be found on the Rotterdam Convention website [www.pic.int](http://www.pic.int). |
| **3.4 Socio-economic effects** |
| **European Union**No assessment of socio-economic effects was reported. |
| **Brazil**No assessment of socio-economic effects was reported. |

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| **4. Hazards and Risks to human health and the environment** |

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| **4.1 Hazard Classification** |
| **WHO/IPCS** | II - Moderately Hazardous |
| **IARC** | 3 – Unclassifiable (Brazilian notification) |
| **European Union** | **Classification according to Regulation (EC) No 1272/2008 of the European Parliament and of the Council (GHS)****Acute Toxicity (oral) 4 \* - H302** (harmful if swallowed)**Skin Sensitisation 1 - H317** (may cause an allergic skin reaction)**Aquatic Acute 1 - H400** (very toxic to aquatic life)**Aquatic Chronic 1 - H410** (very toxic to aquatic life with long lasting effects)**Classification in accordance with Council Directive 67/548/EEC****Xn**; Harmful**N**; Dangerous for the environmentRisk phrases:**R22**; Harmful if swallowed**R43**; May cause sensitisation by skin contact**R50-53**; Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment |
| **US EPA** | II - Warning - Moderately Toxic (Brazilian notification) |

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| **4.2 Exposure limits** |

**Maximum Residue Limits**

The Codex Alimentarius Commission, following the recommendations of the JMPR, decided to delete all existing CXLs for trichlorfon, as it had been informed that trichlorfon was predominantly used in the non-food area and the manufacturer did not support this compound any more. The latest CXLs for a range of crops before they were deleted are listed in Annex II of the report of the 28th JMPR meeting (FAO/WHO Food Standard Programmes 1997).

**EU risk evaluation**

During the EU risk evaluation, the reference values were not confirmed by the experts during the EU peer review. The provisional values proposed by the Rapporteur Member State in the DAR are:

Acceptable Daily Intake (ADI): 0.045 mg/kg bw/day (based on a NOAEL of 4.5 mg/kg bw/day from a 2-year rat study with a safety factor of 100).

Acceptable Operator Exposure Level (AOEL): 0.09 mg/kg bw/day (based on a LOAEL of 45 mg/kg bw/day from a 90-day oral rat study with a higher safety factor of 500).

Acute Reference Dose (ARfD): 0.1 mg/kg bw (based on a NOAEL of 10 mg/kg bw/day from the acute oral neurotoxicity study in rats with a safety factor of 100).

**FAO/WHO**

The FAO/WHO Joint Meeting on Pesticide Residues (JMPR) established an ADI of 0-0.01 mg/kg bw, based on the fact that the following levels cause no toxicological effects:

 Rat: 50 mg/kg in the diet equivalent to 2.5 mg/kg bw.

 Dog: 50 mg/kg in the diet equivalent to 1.25 mg/kg bw. (JMPR 1978)

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 2003 amended the ADI for trichlorfon from

0 – 0.02 mg/kg bw to 0 – 0.002 mg/kg bw, based on a LOEL of 0.2 mg/kg bw per day for inhibition of erythrocyte acetylcholinesterase activity in humans treated orally and a safety factor of 100.

**US EPA**

A chronic Reference Dose (RfD) of 0.002 mg/kg/day was established based on the results of a ten year chronic feeding study in monkeys in which a NOEL was not determined and a LOEL of 0.2 mg/kg/day was established. The inhibition of plasma red blood cells and brain cholinesterase in the monkeys at 0.20 mg/kg/day were considered to be a marginal response. It was concluded that the LOEL could be used for risk assessment purposes if an uncertainty factor of 100 was considered when establishing the RfD. This uncertainty factor was chosen due to the lack of a NOEL and to account for inter-species extrapolation and intra-species variability (US EPA, 1997).

**WHO drinking water guideline**

Trichlorfon is excluded from guideline value derivation, as a review of the literature on occurrence or credibility of occurrence in drinking water has shown evidence that trichlorfon does not occur in drinking water (WHO, 2011).

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| **4.3 Packaging and labelling** |
| The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemical in:  |
| **Hazard Class and Packing Group** | For trichlorfon (pure substance):Hazard class: UN: 6.1Packing group: UN: III(United Nations, 2011) |
| **International Maritime Dangerous Goods (IMDG) Code** | For trichlorfon (pure substance):UN 2783Organophosphorous pesticide, solid, toxic (trichlorfon)Class 6.1Marine pollutant(IMO, 2010) |
| **Transport Emergency Card** | TEC (R)-61GT7-III (IPCS / CEC 2005) |

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| **4.4 First aid** |

**NOTE:** The following advice is based on information available from the World Health Organisation and the notifying countries and was correct at the time of publication. This advice is provided for information only and is not intended to supersede any national first aid protocols.

Trichlorfon can be absorbed into the body by inhalation of its aerosol, through the skin and by ingestion. Evaporation at 20°C is negligible; a harmful concentration of airborne particles can, however, be reached quickly on dust forming. Trichlorfon may cause effects on the nervous system by inhibiting the cholinesterase activity, resulting in convulsions, respiratory failure, and death. Exposure at high level may result in death.

Symptoms of (acute) ingestion and/or inhalation are: nausea, sweating, vomiting, dizziness, weakness, abdominal cramps, diarrhoea, pupil constriction, muscle cramps, excessive salivation, laboured breathing, or unconsciousness. The symptoms of acute intoxication do not become manifest until some hours have passed.

In all cases of exposure, consult a doctor! If breathing has stopped, apply artificial respiration.

In case of inhalation, remove to fresh air and provide rest. In case of ingestion, induce vomiting (ONLY IN CONSCIOUS PERSONS, NOT IN CASE OF AN EMULSIFIABLE CONCENTRATE!); obtain medical attention immediately. If skin contact occurs, remove and wash contaminated clothes. Rinse and then wash skin with water and soap. Eyes should be rinsed with plenty of clean water for at least 15 minutes (remove contact lenses if easily possible), then obtain medical attention immediately.

If the victim is unconscious or convulsing, do NOT give anything by mouth and do NOT induce vomiting.

Depending on the degree of exposure, periodic medical examination is suggested. Specific treatment is necessary in case of poisoning with trichlorfon; the appropriate means with instructions must be available (WHO/IPCS, 1991; IPCS / CEC, 2005).

***If trichlorfon is formulated with solvent(s), also consult the International Chemical Safety cards (ICSC) of the solvent(s). Carrier solvents used in commercial formulations may change physical and toxicological properties.***

**Further information may be found on the website of the IPCS/WHO at** [**www.inchem.org**](http://www.inchem.org)

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| **4.5 Waste management**  |
| Regulatory actions to ban a chemical should not result in creation of a stockpile requiring waste disposal. For guidance on how to avoid creating stockpiles of obsolete pesticides the following guidelines are available: *FAO Guidelines on Prevention of Accumulation of Obsolete Pesticide Stocks (1995), The Pesticide Storage and Stock Control Manual (1996)* and *Guidelines for the management of small quantities of unwanted and obsolete pesticides (1999).*The European Union avoided creating stockpiles of trichlorfon by taking a stepwise approach to the phase-out of permitted uses. The risk was considered manageable during this phase-out period.In all cases waste should be disposed in accordance with the provisions of the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and Their Disposal (1996), any guidelines thereunder, and any other relevant regional agreements.It should be noted that the disposal/destruction methods recommended in the literature are often not available in, or suitable for, all countries; *e.g.*, high temperature incinerators may not be available. Consideration should be given to the use of alternative destruction technologies. Further information on possible approaches may be found in *Technical Guidelines for the Disposal of Bulk Quantities of Obsolete Pesticides in Developing Countries (1996).***Trichlorfon-specific guidance:**Technical trichlorfon and its formulations should be stored in locked, well-ventilated buildings, preferably buildings specifically used for insecticide storage. Do not expose to direct sunlight. Keep products out of reach of children and unauthorized personnel. Do not store near animal feed or foodstuffs.Absorb spilled liquid, and cover contaminated areas with a 1:3 mixture of sodium carbonate crystals and damp sawdust, lime, sand, or earth. Sweep up and place the sweepings in a closeable, impervious container. Ensure that the container is tightly closed and suitably labelled before transfer to a safe place for disposal.Prevent liquid from spreading and contaminating other cargo, vegetation, or waterways with a barrier of the most suitable material available, *e.g.*, earth or sand. If the spill occurs into a waterway and the trichlorfon-containing material is immiscible with water and sinks, dam the waterway to stop the flow and to retard dissipation by water movement. Use a bottom pump, dredging, or underwater vacuum equipment to remove undissolved material.Empty any of the product remaining in a damaged/leaking container into a clean empty container, which should then be tightly closed and suitably labelled.Decontaminate emptied leaking containers with a 10% sodium carbonate solution added at the rate of at least 1 litre per 20-litre drum. Swirl round to rinse walls, empty, and add rinsings to sawdust, etc. Puncture empty containers to prevent re-use.Contaminated absorbents, containers, surplus product, etc. should be burnt in a proper incinerator, at high temperatures, with effluent gas scrubbing. When no incinerator is available, bury in an approved dump, or in an area where there is no risk of contamination of surface or ground water. Before burying, liberally mix with sodium carbonate (washing soda) crystals to help neutralize the product and mix with soil rich in organic matter. Comply with any local legislation (WHO/IPCS, 1991). |

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| **Annexes** |

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| **Annex 1** | **Further information on the substance** |
| **Annex 2** | **Details on Final regulatory action** |
| **Annex 3** | **Address of designated national authorities** |
| **Annex 4** | **References** |

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| **Annex 1 Further information on the substance** |

The information presented in this Annex reflects the conclusions of the notifying parties: the European Union and Brazil. The notification from the European Union was published in PIC Circular XXX of December 2009. The notification from Brazil was published in PIC Circular XXXIV of December 2011.

Where possible, information on hazards provided by the notifying parties has been presented together, while the evaluation of the risks, specific to the conditions prevailing in the notifying Parties are presented separately. This information has been taken from the documents referenced in the notifications in support of their final regulatory actions to ban trichlorfon and includes the conclusion of the European Food Safety Authority (EFSA) on the peer review of trichlorfon, the review report for the active substance trichlorfon finalised in September 2006 by the European Commission, the technical note on the toxicological review of trichlorfon prepared by the Brazilian National Health Surveillance Agency (ANVISA) and the US EPA Reregistration eligibility decision for trichlorfon (1997), which was cited in the supporting documentation to the notification from Brazil.

Furthermore, information from the FAO/WHO Joint Meeting on Pesticide Residues (JMPR) monographs of toxicological evaluation of trichlorfon in 1971, 1975 and 1978, as well as the IARC summary and evaluation for trichlorfon from 1983 and the toxicological evaluation from the Joint FAO/WHO Expert Committee on Food Additives (JECFA) from 2000 and 2003 has been taken into account.

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| **1.** | **Physico-Chemical properties**  |
| **1.1** | **Identity** | ISO: trichlorfonIUPAC: dimethyl (RS)-2,2,2-trichloro-1-hydroxyethylphosphonateCAS: Phosphonic acid, (2,2,2-trichloro-1-hydroxyethyl)-, dimethyl ester |
| **1.2** | **Formula** | C4H8Cl3O4P |
| **1.3** | **Colour and Texture** | Between white and pink waxy solid (90.1-94.1% purity) (EU notification)colourless crystals (WHO/IPCS 1992, Pesticide Manual) with a weak, characteristic odour (Pesticide Manual) |
| **1.4** | **Decomposition temperature** | > 100°C; decomposition before boiling (99.4 % purity) (EU notification) |
| **1.6** | **Density (g/cm3)** | 1.68 g/mL at 20ºC (99.4% purity) (EFSA 2006)1.73 g/mL (Pesticide Manual, Brazilian notification and WHO/IPCS 1992) |
| **1.7** | **Resistance to acids** | Trichlorfon appears to be stable under acid conditions, but unstable in neutral and basic solutions (EFSA 2006).Trichlorfon is slowly hydrolysed in acid media; the half-life is 526 days at pH 1-5 and 20°C. Cleavage of one of the methyl ester groups takes place by acid hydrolysis. In alkaline media, at pH 8 and 37.5°C, it hydrolyses initially to the more toxic compound dichlorvos, but is essentially 100% hydrolysed in 24 h to less toxic products, such as dimethyl hydrogen phosphate, dichloroacetaldehyde, and glyoxal (WHO/IPCS, 1991 and 1992). |
| **1.8** | **Resistance to alkalis** |
| **1.9** | **Tensile strength (103 kg/cm2)** | No information available. |
| **2** | **Toxicological properties**  |
| **2.1** | **General**  |  |
| **2.1.1** | **Mode of Action** | Depression of plasma, red blood cell and brain AChE activities and neurotoxicological signs (EU notification) |
| **2.1.2** | **Symptoms of poisoning** | Signs and symptoms of trichlorfon poisoning are characteristic of AChE inhibition and may include exhaustion, weakness, confusion, excessive sweating and salivation, abdominal pains, vomiting, pinpoint pupils, and muscle spasms. In severe cases of poisoning, unconsciousness and convulsions may develop and death may result from respiratory failure. A delayed polyneuropathy, associated with weakness of the lower limbs, may sometimes occurr a few weeks after exposure (WHO/IPCS, 1992). |
| **2.1.3** | **Absorption, distribution, excretion and metabolism in mammals** | The absorption of trichlorfon is rapid in all species tested, including humans, irrespective of the route of administration. Peak blood concentrations were achieved within 1-2 h but decreased quickly thereafter; the half-time of trichlorfon in human plasma is approximately 2 h. It is widely distributed. Trichlorfon was detected in the milk of lactating cows, and the compound and its metabolites were found in fetal tissue in treated guinea pigs. Trichlorfon undergoes conversion to dichlorvos via a dehydrochlorination reaction that occurs spontaneously at pH values above 5.5. Although little dichlorvos was recovered, it is generally believed to be responsible for the anticholinesterase effects of trichlorfon. The metabolism of trichlorfon in mammals also occurs through O-demethylation and cleavage of phosphorus-carbon bonds. Therefore, the major metabolites are desmethyl trichlorfon, desmethyl dichlorvos, dimethyl hydrogen phosphate, methyl dihydrogen phosphate, and phosphoric acid. Trichlorfon and its metabolites are excreted primarily in the urine (JECFA, 2000). Trichlorfon is rapidly and completely absorbed (80-90% within 24 hours). The highest plasma levels were reported 0.5 and 5 hours after administration, indicating enterohepatic recirculation. Trichlorfon is widely distributed, with the highest concentrations occurring in the liver and kidneys. The main metabolic pathway of trichlorfon involves glucuronidation and further dehydrochlorination. A minor pathway involving the conversion of trichlorfon to dichlorvos has also been identified. In rats, approximately 50% of an administered dose is excreted via the urine, 20% is excreted via the faeces, and 20% is expired as carbon dioxide. In rabbits, greater than 95% is excreted via the urine (EU notification).  |
| **2.2** | **Toxicology studies** |  |
| **2.2.1** | **Acute toxicity** | The acute toxicity of trichlorfon in rats and mice was similar when it was administered orally, intraperitoneally, or subcutaneously. It was less toxic to rats and rabbits after dermal application than when given by the other routes (JECFA, 2000).Oral LD50 values in laboratory animals range from 160 to 950 mg/kg bw.Dermal LD50 values for rat and mouse are greater than 5000 mg/kg bw.(WHO/IPCS, 1992; JECFA, 2000).LD50 (male rat, oral) 258 mg/kg bwLD50 (female rat, oral) 212 mg/kg bwLD50 (rat dermal) >5000 mg/kg bwLD50 (rat inhalation) >0.533 mg/L (highest concentration tested).Trichlorfon is non-irritating to the skin and eyes according to EU criteria and a slight ocular irritant (JECFA, 2000). It is sensitising to the skin (Magnusson and Kligman's test) (EU notification). Skin sensitization potential was demonstrated in guinea pigs (WHO/IPCS, 1992). |
| **2.2.2** | **Short term toxicity** | Short-term, oral toxicity studies were carried out on rats, dogs, monkeys, rabbits, and guinea pigs. In a 16-week study on rats, a 4-year study on dogs, and a 26-week study on monkeys, no-observed-effect levels (NOELs) of 100 mg/kg diet, 50 mg/kg diet, and 0.2 mg/kg bw (based on plasma, erythrocyte, or brain AChE activity), respectively, were determined. Inhalation exposure of rats, over 3 weeks, indicated a NOEL of 12.7 mg/m3, based on the inhibition of plasma, erythrocyte, and brain AChE activity (WHO/IPCS, 1992).Critical effects: Depression of plasma, red blood cells and brain AChE activities and neurotoxicological signs.Target organs: Increased weight, liver, kidney, spleenRat (oral, 90 days, male): NOAEL = 135 mg/kg bw/day (RBC and brain AchE levels were not determined)Rat (oral, 90 days, female): LOAEL = 45 mg/kg bw/day (RBC and brain AChE levels were not determined)Rabbit (dermal, 3 weeks): NOAEL = 100 mg/kg bw/dayRat (inhalation, 3 weeks): NOAEL = 3.43 mg/kg bw/day (EU notification)In a 21-day toxicity study, trichlorfon was administered dermally to rabbits for 15 days (5 days a week for 3 weeks) at doses of 0, 100, 300 or 1000 mg/kg/day. The systemic NOEL was greater than the highest dose tested. The NOEL for cholinesterase inhibition was 100 mg/kg/day. The LOEL for cholinesterase inhibition was 300 mg/kg/day based on depression in red blood cell activity (US EPA, 1997).A single dose **human clinical trial** conducted in 1990 was reported for the evaluation of the use of trichlorfon in the treatment of Alzheimer's disease in the WHO/IPCS 1992 monograph. A single oral dose of 0, 2.5, 5.0, 7.5 or 15 mg/kg/day was administered to humans. The NOEL was 2.5 mg/kg/day and the LOEL was 5.0 mg/kg/day based upon the inhibition of plasma and red blood cell cholinesterase and clinical signs of vomiting, nausea and diarrhoea (WHO/IPCS, 1992, in US EPA, 1997). The results of a double blind, placebo-controlled, single-centre study of the safety, tolerability and pharmacokinetics of trichlorfon in patients with Alzheimer disease were used to calculate the NOEL for inhibition of erythrocyte cholinesterase activity. A total of 27 patients were given an oral loading dose of trichlorfon by capsule containing 1.5, 2.5, 4 or 4 mg/kg bw per day for 6 days, followed by a daily oral maintenance dose of 0.25, 0.4, 0.65 or 1 mg/kg bw for 21 days. The mean inhibition of erythrocyte cholinesterase activity at the end of the treatment was 14%, 35%, 66%, 77% and 82% with the placebo and the four treatments, respectively. A linear extrapolation of the data on inhibition of erythrocyte cholinesterase activity resulted in an estimated NOEL of 0.1–0.2 mg/kg bw (JECFA, 2003). |
| **2.2.3** | **Genotoxicity (including mutagenicity)** | Under physiological conditions, trichlorfon has been reported to have a DNA-alkylating property. The trichlorfon mutagenicity results have been both positive and negative. Dichlorvos may be responsible, either in part or in full, for the effects observed. Most of the *in vitro* mutagenicity studies on both bacterial and mammalian cells were positive while few of the *in vivo* studies produced a positive result (WHO/IPCS, 1992).Trichlorfon has been tested in a large number of studies for genotoxicity covering a wide range of end-points, with considerable variation in the results for most end-points. Both positive and negative results were obtained in tests for bacterial mutations and for gene mutation in mammalian cells in vitro, but the results of studies of effects on chromosomes in mammalian cells in vitro (chromosomal aberrations or sister chromatid exchanges) were uniformly positive. Mostly negative results were found in assays in mammals in vivo, comprising tests for somatic cell mutations in bone marrow (sister chromatid exchange, negative result in a single study), micronucleus formation (negative results in five of six studies) and chromosomal aberrations (negative results in three of five studies). Mostly negative results were also found in assays for germ cell mutagenicity in vivo, comprising dominant lethal mutations (negative results in six of nine studies) and chromosomal aberrations in spermatogonia or spermatocytes (negative results in three of four studies). JECFA at its sixtieth meeting received further data on mutagenicity, comprising positive results in studies of sister chromatid exchange in vivo but not in vitro. Trichlorfon was a germ cell aneugen in laboratory animals in vivo. There was also limited evidence from observations in poisoned humans that trichlorfon caused aneuploidy and chromosome damage in lymphocytes. A study involving pregnant women suggested that exposure to uncertain concentrations of residues of trichlorfon in fish may have caused trisomy 21 (Down syndrome) in their offspring as a result of germ cell aneugenicity (JECFA, 2003).Equivocal results have been reported in *in vitro* gene mutation assays conducted in Chinese hamster lung cells. Positive results have been reported in *in vitro* chromosomal aberration assays conducted in human lymphocytes, with and without metabolic activation.However the clastogenicity could not be confirmed *in vivo* for somatic cells (micronucleus test) or germ cells (dominant lethal assay) since the studies were considered as non-acceptable due to major deviations from the guidelines (EU notification). |
| **2.2.4** | **Long term toxicity and carcinogenicity** | The available data are insufficient to evaluate the carcinogenicity of trichlorfon to humans (IARC 1987).Long-term toxicity/carcinogenicity studies were carried out on mice, rats, monkeys, and hamsters after oral, intraperitoneal, or dermal administration. An adverse effect on the gonads was seen following the oral exposure of mice and rats at dose levels of 30 mg/kg bw and 400 mg/kg diet, respectively. In a 24-month study on rats and a 10‑year study on monkeys, NOAELs of 50 mg/kg diet and 0.2 mg/kg bw, respectively, were determined. Available data do not provide evidence of carcinogenicity following the long-term exposure of test animals by several routes of administration (WHO/IPCS, 1992).Rat (oral, 2 years): NOAEL = 4.5 mg/kg bw/day (brain AChE depression, hypercholesterolemia and renal calcification (in males)).Mouse (oral, 2 years): LOAEL = 49.21 mg/kg bw/day (AChE depression) (EU notification).Incidences of adrenal pheochromocytomas and mononuclear cell leukaemia were increased in male rats; however, incidences were not increased in females to the same extent, and were not increased in a second study at higher doses. Adrenal pheochromocytoma is reported to be common in this strain of rats. No carcinogenic effects in mouse were observed.Based on the available data in rats and mice, it was concluded that trichlorfon is not a carcinogenic compound (EFSA, 2006).The US EPA has classified trichlorfon “as a Group E chemical, no evidence of carcinogenicity for humans” (US EPA, 1997).JECFA at its sixtieth meeting concluded that the weight of the evidence from the assays for mutagenicity in vivo indicated that trichlorfon residues in animal-derived foods would not present a carcinogenic hazard to consumers (JECFA, 2003). |
| **2.2.5** | **Effects on reproduction** | Studies on mice, rats, and hamsters indicate that trichlorfon produces a teratogenic response in rats at doses high enough to produce maternal toxicity. Exposure of rats to 145 mg trichlorfon/kg diet, during gestation, caused fetal malformations. A gavage dose of 400 mg/kg bw in hamsters also produced both maternal toxicity and a teratogenic response. The lowest dose by gavage that produced teratogenic effects in rats was 80 mg/kg bw. The effects appear to be time specific in the gestation period. A NOEL of 8 mg/kg was determined in this gavage study.NOELs of 8 mg/kg bw and 200 mg/kg bw were demonstrated for rats and hamsters, respectively. Teratogenic responses involving the central nervous system have also been reported for the pig and guinea pig.However, no teratogenic effects were observed in a 3-generation reproduction study on rats, in which high dose levels induced adverse reproductive effects. The NOEL in this study was 300 mg/kg diet (WHO/IPCS, 1992).JECFA evaluated studies of developmental toxicity with trichlorfon conducted in four animal species. In these studies, teratogenic effects were seen only at very high, maternally toxic doses. In addition, as multigeneration studies of reproductive toxicity did not provide evidence of paternally transmitted teratogenicity, the Committee considered that the effect on exposed males had been assessed. JECFA later reviewed the assessment by the JMPR in 1993 (WHO, 1994) of dichlorvos, the major metabolite of trichlorfon. That Meeting concluded that dichlorfos was not teratogenic in mice, rats or rabbits, even at doses that were toxic to maternal animals. In addition, at 12 mg/kg bw per day, dichlorvos had no reproductive effects in rats in a three-generation study. On the basis of these considerations, JECFA concluded that the information from the study in humans would not significantly affect its risk assessment of trichlorfon.Chromosomal effects were studied in the lymphocytes of 31 people who had attempted suicide by taking unknown doses of trichlorfon. There appeared to be an increase in per cent aneuploidy in blood samples collected 3–6, 30 and 180 days after the incidents. An increase was also found in the rate of chromatid and chromosome‑type aberrations. It was concluded that the intake that had resulted in these effects far exceeded the ADI established for trichlorfon by the JECFA at its fifty-fourth meeting (JECFA, 2003).The toxic potential of trichlorfon on mammalian reproduction was assessed in a three-generation study in the rat, as well as in teratology studies in the rat and rabbit. Based on the poor quality of the reproductive studies, a data requirement has been set for a multigeneration study in rats. However, the available teratology studies were sufficient to demonstrate that trichlorfon has no developmental toxicity.Critical effect: No evidence of foetotoxicity in rats and rabbits.Rabbit (teratology study): Maternal NOAEL 15 mg/kg bw/day; Developmental NOAEL 45 mg/kg bw/day (EU notification) |
| **2.2.6** | **Neurotoxicity/ delayed neurotoxicity, Special studies where available** |

| Species / Study | End point | Effects |
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| Rat(oral gavage, acute) | NOAEL 10 mg/kg bw | Clinical signs of toxicity, alterations in Field Observation Battery (FOB), decreased motor activity, and significant inhibition of plasma, RBC and brain AChE |
| Rat(diet, 90 day) | NOAEL 6.08 mg/kg bw/day | Decreased bodyweight, motor and locomotor activity, inhibition of all types of AChE, myelin degeneration (EU notification) |
| Hen(acute delayed neurotoxicity) | LD50 167 mg/kg bw | Typical signs of AChE inhibition were observed, however, no delayed neurotoxicity and no inhibition of neurotoxic esterase (NTE) were observed. This study is of poor quality, but is considered acceptable as additional information. |
| Hen (90 day delayed neurotoxicity) | NOAEL 9 mg/kg bw/day | Inhibition of whole blood AChE activity and associated clinical symptoms, slight axonal degeneration of the spinal cord |

Trichlorfon is not classified as an immunotoxicant (WHO/IPCS, 1992). |
| **2.2.7** | **Summary of mammalian toxicity and overall evaluation** | WHO has classified trichlorfon as moderately hazardous (WHO 2009). The oral LD50 of trichlorfon is between 212 (EU notification) and 800 mg/kg bw (WHO/IPCS, 1992). Trichlorfon is harmful if swallowed and has low acute dermal toxicity (EU notification, EFSA, 2006). Clinical signs of acute intoxication includenausea, vomiting, weakness, abdominal cramps, diarrhoea, pupil constriction, muscle cramps, excessive salivation, laboured breathing, or unconsciousness*.* The symptoms may be delayed. Trichlorfon is non-irritating to the skin and eyes, but sensitising to the skin (EU notification)*.* Trichlorfon is neither genotoxic nor classified as carcinogenic according to effects observed in studies on mice, rats, monkeys, and hamsters. The available teratology studies in rats and rabbits were sufficient to demonstrate that trichlorfon has no developmental toxicity (EFSA, 2006).In contrast to these findings, the results of the national risk evaluation as laid down in the supporting documentation to the notification from Brazil are: Trichlorfon has genotoxic, immunotoxic, teratogenic, and neurotoxic characteristics, causes cerebellar hypoplasia, adverse effects on reproduction and on the hormonal system (endocrine deregulation). Trichlorfon is also considered as having the potential to cause neurological damage higher for humans than for animals, as demonstrated on the delayed neuropathy (ANVISA, 2009).The following exposure limits were derived:**EU**From the EU risk evaluation, the following exposure limits were proposed by the RMS but not confirmed at EU level:Acceptable Daily Intake (ADI): 0.045 mg/kg bw/day (based on a NOAEL of 4.5 mg/kg bw/day from a 2-year rat study with a safety factor of 100).Acceptable Operator Exposure Level (AOEL): 0.09 mg/kg bw/day (based on a LOAEL of 45 mg/kg bw/day from a 90-day oral rat study with a higher safety factor of 500).Acute Reference Dose (ARfD): 0.1 mg/kg bw (based on a NOAEL of 10 mg/kg bw/day from an acute oral neurotoxicity study in rats with a safety factor of 100).**US EPA (1997)**[[4]](#footnote-4)4Acute RfD = 0.002 mg/kg/day (based on the results of a ten year chronic feeding study in monkeys in which a NOEL was not determined, a LOEL of 0.2 mg/kg/day and an uncertainty factor of 100 for inter-species extrapolation and intra-species variability).Chronic RfD = 0.002 mg/kg/day (based on the same study endpoints as for the acute RfD).**JECFA (2000)**Acceptable Daily Intake (ADI): 0 – 0.02 mg/kg bw/day (based on a NOAEL of 0.2 mg/kg bw/day from inhibition of erythrocyte acetylcholinesterase activity in humans treated orally and a safety factor of 10).**JECFA (2003)**The ADI for trichlorfon was amended from 0 – 0.02 mg/kg bw to 0 – 0.002 mg/kg bw (based on a LOEL of 0.2 mg/kg bw per day for inhibition of erythrocyte acetylcholinesterase activity in humans treated orally and safety factor of 100).Trichlorfon is excluded from drinking-water guideline value derivation, as a review of the literature on occurrence or credibility of occurrence in drinking water has shown evidence that trichlorfon does not occur in drinking water (WHO, 2011). |
| **3** | **Human exposure/Risk evaluation**  |
| **3.1** | **Food** | Food of plant or animal origin was the main source of exposure of the general population to trichlorfon. Trichlorfon was also used as a public health insecticide, a veterinary drug for the control of endo- and ectoparasites in/on cattle, sheep, goats, pigs, horses, poultry, dogs, cats and fish, as well as an anthelmintic in medicine (JECFA, 2000). In the 1970s these non-agricultural pesticide uses together accounted for less than 10% of the total use in the Western world (JMPR 1972).In the meantime, the situation has changed: The Codex Alimentarius Commission, following the recommendations of the JMPR, decided to delete all existing CXLs for trichlorfon, as it had been informed that trichlorfon was predominantly used in the non-food area and the manufacturer did not support this compound any more. The latest CXLs for a range of crops before they were deleted are listed in Annex II of the report of the 28th JMPR meeting (FAO/WHO Food Standard Programmes, 1997).Following application to green plant material, a half-life of about 1-2 days was obtained for trichlorfon, as shown by residue studies on cotton leaves, grass, cabbage, clover, alfalfa and lettuce. Crops treated 2-4 weeks before harvest are practically free of residues at harvest time (maize, soybeans, rape, flax). Following application to bananas, oranges and groundnuts, the bulk of the trichlorfon residue is contained in the peel and shells, respectively. Within a few days after the application only slight residues are to be found in the pulp and nuts, respectively. Food processing further reduces residues in food.The behaviour of trichlorfon is characterized by its hydrophilic properties. Its decomposition is brought about by splitting the P-C bond and by hydrolysis of the P‑OCH3 bonds. In addition, in tissues it can be converted in trace amounts to dichlorvos.As a result of trichlorfon use, residues may occur in animal feed. When the applications are made in accordance with good agricultural practice the residues are considered to be no hazard to the animal health and no detectable contamination of the foods derived from these animals is to be expected (JMPR, 1972).The concentration of trichlorfon in various organs after direct application to animals largely depends on the treatment procedure and the formulation used. The residue level in organs and tissues reached its maximum within 12 hours of dosing in cows and pigs. Only low residues were detectable after 24 hours. The fat of milk contains much lower residue than the whole milk since trichlorfon is hydrophilic. Trichlorfon was detected in organs and tissues of sheep 7 days after aerosol treatment (JMPR, 1979). |
| **3.2** | **Air** | Exposure via air is not considered relevant, since the available data indicates a low volatility of trichlorfon from soil surface. However, occupational exposure where air concentration exceeded 0.5 mg/m3, resulted in a reversible decrease in plasma AChE activity (see also 3.4) (EFSA, 2006). |
| **3.3** | **Water** | Exposure via water is not considered relevant, since “a review of the literature on occurrence or credibility of occurrence in drinking water has shown evidence that [trichlorfon] does not occur in drinking water” (WHO, 2011, p. 181). |
| **3.4** | **Occupational exposure**  | **European Union**Due to an inadequate database (short term, reproductive toxicity and genotoxicity), the risk evaluation for operators, workers and bystanders could not be finalised in the EU peer review process. For transparency, the provisional risk evaluation provided by the rapporteur Member State is presented below. It is important to note that the dermal absorption value used with the estimates in the DAR was 10%. As the experts agreed for a dermal absorption value of 100%, this will result in exposure estimates much higher than the provisional AOEL for operators, workers and bystanders.The models used for the exposure assessment are not appropriate for greenhouse uses. Furthermore, in relation with worker exposure, the potential evaporation of the metabolite dichlorvos from the plant was discussed. Since this was shown to be more than 30% of the applied trichlorfon, this could be relevant for worker exposure by inhalation. The representative plant protection product Cekufon 80 SP is a soluble powder containing 800 g trichlorfon/kg for use on tomatoes in greenhouses.**Operator exposure**According to the intended uses submitted by the notifier the maximum applied dose is 2.4 kg a.i./ha and the minimum volume 1000 L of water/ha. The applications were made using tractor-mounted sprayers, handsprayer (gun) and knapsack. For the tractor-mounted application, the work rate taken into account is 10 ha/day and 3 h of spraying/day. For the hand held application, the work rate taken into account is 0.4 to 4 ha/day and 2 h of spraying/day.The UK POEM model (“Predictive Operator Exposure Model”) gave estimated exposures below the AOEL for gun and knapsack application methods if gloves are worn. With the German BBA model (operator exposure model developed at the former “Biologische Bundesanstalt”), only the knapsack application gave estimated exposures below the AOEL (79% and lower) with the use of gloves. For all the uses and when standard PPE, plus mask and protection for the head are used, the estimated exposition is under the AOEL (74% and lower).**Worker exposure**Workers re-entering fields treated beforehand with Cekufon 80 SP are exposed to 45.5% of the AOEL when PPE are used or >900% of theAOEL when PPE are not used.**Bystander exposure**The risk evaluation for bystanders was based on the estimation of Ganzelmeier *et al* 1995, taking into account a default drift rate of 8% of application. According to this, the worst-case applications (gun application use without PPE) led to possible bystander exposure of about 76% of the AOEL.**Brazil**The notification refers to several studies that have shown that pesticide poisonings, especially with organophosphorous pesticides, occurred in different regions of Brazil. In addition, the technical note (ANVISA, 2009) indicates that many pesticide poisoning incidents were not reported in Brazil. According to a study from the Amazon area of Brazil, agricultural workers were not prepared to use pesticides (including trichlorfon) correctly. They were not sufficiently aware of the risks of pesticides to human health and the environment. This study further concludes that farmers did not use protective clothing or equipment because it was expensive and not suitable for a tropical climate. Due to lack in training and poor knowledge of pesticide hazards, pesticides were handled carelessly during preparation, application and disposal of empty packages. Exposure of farmers, their families, consumers (via residues in food) and the environment was thus high. Although no poisoning incidents with trichlorfon itself have been reported from Brazil, the decision to ban trichlorfon was taken on the basis of evaluation of its hazardous properties as well as on expected exposure of agricultural workers to pesticides in general and also to trichlorfon under conditions of use in Brazil.**Other reported (occupational) exposure**Several cases of acute poisoning from intentional (suicide) or accidental exposure have occurred. Signs and symptoms of intoxication included characteristics of AChE inhibition, such as exhaustion, weakness, confusion, excessive sweating and salivation, abdominal pains, vomiting, pinpoint pupils, and muscle spasms. In severe cases of poisoning, unconsciousness and convulsions developed and death usually resulted from respiratory failure. In cases where victims survived because of medical intervention, a delayed polyneuropathy associated with weakness of the lower limbs occurred a few weeks after exposure. In fatal cases, autopsy findings have shown ischaemic changes in the brain, spinal cord, and vegetative ganglia, damage to the myelin sheath in the spinal cord and brain peduncles, and structural changes in the axons of peripheral nerves.A few cases of occupational poisoning have occurred, mainly because safety precautions were neglected. Occupational exposure at a workplace where air concentrations exceeded 0.5 mg/m3 resulted in decreased levels of plasma cholinesterase and changes in the EEG pattern. However, these effects were completely reversible on cessation of exposure. No cases of skin sensitization have been reported (WHO/IPCS, 1991). |
| **3.5**  | **Medical data contributing to regulatory decision** | Trichlorfon has been used for the treatment of intestinal parasites and Alzheimer Disease. Acute poisonings have shown dose-related clinical signs of AChE inhibition (and delayed neuropathy in some cases) (EFSA, 2006).Trichlorfon has been extensively used for the treatment of schistosomiasis in humans. Administration of a single dose (7-12 mg/kg) resulted in 40-60% inhibition of cholinesterase in the plasma and erythrocytes, without the manifestation of any cholinergic symptoms. However, mild symptoms were observed in cases with a repeated dose regimen. A high dose level (24 mg/kg) caused severe cholinergic symptoms (WHO/IPCS, 1991). |
| **3.6** | **Public exposure**  | None reported. |
| **3.7** | **Summary-overall risk evaluation** | The **European Union** has conducted a risk evaluation of the human health effects of trichlorfon. An assessment of the potential exposure of operators, workers and bystandes, despite limitations of the underlying data, led to the conclusion that operators, workers and bystanders may be exposed to levels of trichlorfon above the acceptable operator exposure level (AOEL)**Brazil** has conducted a risk evaluation of the human health effects of trichlorfon. Based on the hazardous properties of trichlorfon as well as on conditions of use in Brazil, the expected risks resulting from the exposure of agricultural workers, bystanders and the general population to trichlorfon were considered too high. |
| **4** | **Environmental fate and effects**  |
| **4.1** | **Fate** |  |
| **4.1.1** | **Soil** | **EU risk evaluation**The available data from aerobic soil degradation studies, which were not completely accepted by experts (a new study was requested), suggested that degradation of trichlorfon in aerobic soil is pH dependent. In non-sterile aerobic soil at pH 5, following application of radiolabelled trichlorfon, approximately 30% of the applied radioactivity (AR) was present in soil as non-extractable residues after 67 days.Desmethyl-dichlorvos accounted for 37.55% AR and dichlorovinylphosphate accounted for 40.68% AR. At pH 7, 9-21% AR was present in soil as non-extractable residues after 33 days. In sterile aerobic soil at pH 5, 25% AR was present in the soil as non-extractable residues after 47 days. Due to the difficulty to derive experimentally a reliable Koc value for trichlorfon, a worst-case value of zero was used in the risk evaluation. The Koc value for two metabolites was set at zero due to missing data and for the metabolite dichlorvinyl phosphate the Koc was 10.2 mL/g.**Brazilian risk evaluation**Trichlorfon is not persistent in soil. Biologic degradation is the most important route in the process of mineralization. Hydrolysis contributes for the degradation in neutral to acidic conditions (Brazilian notification).**US EPA risk evaluation**It appears that hydrolysis and aerobic metabolism are the main routes of dissipation in both soil and water. The major degradation product in both soil and water is dichlorvos, with desmethyl dichlorvos also reported as a degradation product in soil. Dichlorvos is itself a registered pesticide active ingredient.Trichlorfon was found to degrade rapidly in non-sterile aerobic soils (half-life ~ 1 to 27 days) but was stable in a sterile soil (half-life over 40 days).Potential to leach is suggested by findings of high mobility in soil: Dissociation constant (Kd) estimates of 0.25 to 0.50 were obtained for soils with texture varying from sand to silty clay and organic matter content of 0.5% to 5.1%. For more details see US EPA (1997). |
| **4.1.2** | **Water** | **EU risk evaluation**The degradation of trichlorfon in water is pH dependent. In a sterile buffer solution at pH 5, following application of radiolabelled trichlorfon, approximately 80% of the AR was identified as parent compound after 34 days.Approximately 10% AR was identified as desmethyl-DDVP, and 7.7% AR was identified as dichloroacetaldehyde (DCAA). At pH 7, after 48 hours, 40% AR was identified as trichlorfon, 25.5% AR was identified as DDVP, 22.7% AR as DCAA, 22.7% AR as DCAA and 12% AR as desmethyl-DDVP. At pH 9, 10.5% AR was present as parent compound after 45 minutes, 52.3% AR was detected as DDVP and 10.5% AR was detected as desmethyl-DDVP.DT50 values were calculated to be 117 days at pH 5, 38 hours at pH 7 and 31 minutes at pH 9. However, it should be noted that a data requirement for the accurate identification of the metabolites hydrolytically produced was established during the risk evaluation.Trichlorfon is not expected to undergo photodegradation and is not readily biodegradable.**Brazilian risk evaluation**Studies of abiotic degradation in water (hydrolysis and photolysis) indicate that trichlorfon and its main degradation product dichlorvos exhibit characteristics of high mobility. Trichlorfon has a high potential for mobility due to its high water solubility and low adsorption in soil. It is therefore likely to contaminate ground water, but is considered not persistent in aquatic environments.**US EPA risk evaluation**Potential for contamination of surface and ground water by trichlorfon and trichlorfon degradation product (particularly DDVP) cannot be adequately assessed because acceptable field dissipation data is not available. Potential to leach is suggested by findings of high mobility in soil.Risk of contamination of surface and ground water may be moderated by rapid degradation of trichlorfon in soil and water. It appears that hydrolysis and aerobic metabolism are the main routes of dissipation in both soil and water. The major degradation product in both soil and water is DDVP. DDVP is itself a registered pesticide active ingredient.Studies in pond water and sterile water indicate more rapid degradation at lower pH (higher acidity). In pond water, trichlorfon degraded rapidly at pH 8.5 and room temperature (99% of applied active ingredient degraded in 2 hours), but was stable when held at pH 5.0 for 2 hours. In sterile water trichlorfon hydrolyzed rapidly at pH 7 and 9 (half-life 31 minutes at pH 9 and 34 hours at pH 7) but at pH 5 the half-life was 104 days.The available data, from field studies that were not completely acceptable, suggested that trichlorfon and DDVP may have little potential to contaminate ground water because they degrade rapidly in soil. Acceptable field studies are needed (US EPA, 1997). |
| **4.1.3** | **Air** | Trichlorfon is considered as non-volatile. It is not expected that both trichlorfon and dichlorvos (which is considered volatile) be transported for long distances or persist in the air for a long time (Brazilian notification).The half-life of trichlorfon in the troposphere due to the reaction with hydroxyl radicals has been calculated to be 1.73 days (EU notification). |
| **4.1.4** | **Bioconcentration** | Bioconcentration in fish has not been evaluated because its log Kow is less than 2 (Brazilian notification) / below 3 (EFSA, 2006) and hence, the risk for bioconcentration in fish is considered to be low. |
| **4.1.5** | **Persistence** | Trichlorfon is not persistent in soil or the aquatic environment (Brazilian notification).**EU risk evaluation**The data package submitted on the degradation rate in soil was not sufficient to derive a reliable DT50 for trichlorfon to be used in the assessment. The degradation rate of dichlorvos was investigated in a separate study under aerobic conditions in two biologically active soils and one sterile soil (22ºC, 40% maximum water holding capacity). Dichlorvos degraded very rapidly in soil, with 59-61% AR recovered as 14CO2 after only 2 days of incubation. The unextracted fraction of radioactivity was approximately the same in all soils (< 22%). The first order DT50 was estimated to be < 1 day.Field dissipation studies were not submitted as they were not required.PECsoil were recalculated using a DT50lab, pH 6.5 value of 13.184 days as a realistic worst case for tomatoes, a crop with the optimal yield in the pH range 6.5-6.9. However, this DT50 value was derived from a degradation study considered not reliable during the peer review. |
| **4.2** | **Effects on non-target organisms** |  |
| **4.2.1** | **Terrestrial vertebrates** | Trichlorfon is moderately to highly toxic to birds (LD50 single dose for *Coturnix coturnix japonica* = 110.1 mg/kg bw). The acute toxicity through diet varies from 720 mg a.i./kg in feed (ppm) (*Colinus virginianus*) to more than 5000 mg a.i./kg in feed (ppm) (*Anas platyrhynchos*) (Brazilian notification).**US EPA risk evaluation for birds (US EPA 1997)****Acute toxicity**Based on acute oral and subacute dietary testing, trichlorfon is highly toxic to practically non-toxic to birds. LD50 values of 22.4 to 123 mg/kg bw, based on the technical material, were obtained representing seven species; LC50 values of 720 to >5000 mg/L were obtained representing four species. An LD50 value of 99 mg/kg bw was determined for bobwhite quail using a formulated product (42.4% active ingredient), indicating moderate toxicity.**Chronic/reproductive toxicity**Two avian reproduction studies show that there will be effects on reproduction at levels of trichlorfon (99.8% a.i.) as low as 30 ppm i.a. in feed.The avian reproduction LOELs for trichlorfon are 30 ppm for the bobwhite quail and 78 ppm for the mallard duck. |
| **4.2.2** | **Aquatic species** | **Algae**Green algae (*Scenedesmus subspicatus*): 120 hours EC50 = 10 mg/L (technical trichlorfon, 98.1% a.i.) (EU notification).Green algae (*Scenedesmus subspicatus*): 96 hours EC50 = 1367 mg a.i./L (Brazilian notification).**Aquatic invertebrates**Water flea (*Daphnia similis*): 46 hours EC50 (46h) = 0.00045 mg/L (Brazilian notification).Based on the available studies, a high risk to aquatic invertebrates was identified in the EU risk evaluation, although the existing study on the most sensitive species, *Daphnia magna*, was considered to be of poor quality (EU notification).EC50 values range from 0.18 μg/L for *Daphnia pulex* to 7800 μg/L for crayfish; however, ten of eleven studies resulted in EC50 estimates indicating very high toxicity (EC50 ≤ 0.1 mg/L). A life cycle study with *Daphnia magna* showed that growth, survival, and reproduction were impaired from trichlorfon at levels greater than 5.6 ng/L (US EPA 1997).**Fish**Rainbow trout (*Oncorynchus mykiss*): 96 hours LC50 = 0.7 mg/L (technical trichlorfon, 98.1% a.i.) (EU notification).Zebrafish *(Brachydanio rerio):* 96 hours LC50 = 759 mg/L (Brazilian notification).Acute toxicity tests have been conducted with trichlorfon technical on 12 species of freshwater fish and 10 species of freshwater invertebrates. For fish, LC50 estimates ranged from 0.23 mg/L for bluegill sunfish to 110 mg/L for fathead minnow, indicating that trichlorfon technical ranges from highly toxic to practically non- toxic to freshwater fish.A freshwater fish early life stage test showed that trichlorfon technical causes adverse effects to rainbow trout growth and survival at levels greater than 110 μg/L.For marine and estuarine species, studies using trichlorfon technical resulted in LC50 values ranging from 0.36 μg/L for pink shrimp to >1.0 mg/L for spot (*Leiostomus xanthurus*), indicating very high to moderate toxicity (US EPA, 1997). |
| **4.2.3** | **Honeybees and other arthropods** | **Honeybees**Trichlorfon is considered very toxic to bees (LD50 *=* 3.6 µg a.i./bee) (Brazilian notification).The estimated LD50 is 59.8 μg/bee, indicating that trichlorfon technical has low toxicity to honey bees (US EPA, 1997).**Other arthropods**Aphid parasitoid (*Aphidius rhopalosiphi*): LR50 = 0.519 g a.i./haPredatory mite (*Typhlodromus pyri*): LR50: 90% mortality was observed at 1.2 kg a.i./ha Low risk is assumed due to indoor use (EU notification). |
| **4.2.4** | **Earthworms** | Subchronic toxicity: (*Eisenia foetida*, 14 day study): LC50 =140 mg a.i./kg soil (EU notification).Trichlorfon is considered to be non-toxic to earthworms (Brazilian notification). |
| **4.2.5** | **Soil microorganisms** | The effects of trichlorfon were tested on soil microbial respiration and nitrogen transformation. Effects were less than 25 % at day 28 at 9.6 mg a.i./kg dry weight soil (7200 g a.s/ha). (EFSA, 2006). The formulation containing trichlorfon can cause effects in the soil microorganisms involved in the carbon and nitrogen cycle (Brazilian notification). |
| **4.2.6** | **Terrestrial plants** | No effects reported. For summary of overall risk evaluation see chapter 5.6. |
| **5** | **Environmental Exposure/Risk Evaluation**  |
| **5.1** | **Terrestrial vertebrates** | No studies on the toxicity to birds or mammals are available and none have been considered necessary in the EU risk evaluation, as the representative use is in glasshouses to which birds and mammals have limited access and this will hence limit exposure. Therefore the risk to birds and mammals from the representative use evaluated is regarded as low (EU notification). |
| **5.2** | **Aquatic species** | The test concentrations were not analytically verified during the available studies with trichlorfon on fish, *Daphnia magna* and algae. Therefore it was considered that these studies could only be used as additional information. Nevertheless, based on the available studies, a high risk to aquatic invertebrates was identified. The study on the most sensitive species, *Daphnia magna*, was considered to be of poor quality and should be repeated as *Daphnia magna* was the most sensitive species by more than one order of magnitude. Based on the existing study the risk to aquatic invertebrates can provisionally already be considered as high (EFSA, 2006). |
| **5.3** | **Honey bees and other arthropods** | Due to the proposed use in the EU (indoor use in glasshouses on tomatoes where pollinators will be present), the risk to bees must be addressed. Since no toxicity studies on bees had been submitted by the applicant, the risk to bees could not be evaluated.A standard laboratory toxicity study on *Aphidius rhopalosiphi* with the lead formulation CEKUFON 80 SP and on *Typhlodromus pyri* with the formulation DIPTEREX (50% trichlorfon) are available. Both these studies on indicator species indicate a very high toxicity to non-target arthropods. However, as the use is in-doors, the risk to non-target arthropods was considered to be low. In case of integrated pest management, a further risk evaluation will be necessary (EFSA, 2006). |
| **5.4** | **Earthworms** | The Toxicity Exposure Ratio (TER) is a ratio of the toxicity of a chemical to a test organism (LD50 or NOEL), and the predicted exposure to the substance.Based on a provisional PECsoil of 2.733 mg a.s./kg soil, the resulting acute TER of 51 is above the appropriate trigger value of 10, indicating a low risk to earthworms from the representative uses of trichlorfon evaluated (EFSA, 2006). |
| **5.5** | **Soil microorganisms** | No effects were seen in a study where the tested concentration of trichlorfon exceeds the application rate of the representative use evaluated. Therefore, the risk to soil non-target microorganisms is considered to be low (EFSA, 2006). |
| **5.6** | **Summary – overall risk evaluation** | The risk to birds, other terrestrial vertebrates, non-target arthropods, earthworms, other soil non-target macro-organisms, soil microorganisms and non-target plants from the representative indoor use of trichlorfon in greenhouses on tomatoes is considered to be low. Based on the available studies, which were considered to be of poor quality, a high risk to aquatic invertebrates from trichlorfon use was provisionally identified (EU notification). |

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| **Annex 2 – Details on final regulatory actions reported**  |

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| **European Union** |

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| **1** | **Effective date(s) of entry into force of actions** | 21 November 2008: all uses of plant protection products containing trichlorfon were prohibited as from that date at the latest. Authorisations for plant protection products containing trichlorfon had to be withdrawn by 21 November 2007 by EU Member States. |
|  | **Reference to the regulatory document** | Commission Decision 2007/356/EC of 21 May 2007 concerning the non-inclusion of trichlorfon in Annex I to Council Directive 91/414/EEC and the withdrawal of authorisations for plant protection products containing that substance (Official Journal of the European Union L 133 of 25.5.2007, p.42-43), available at:<http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2007:133:0042:0043:EN:PDF> |
| **2** | **Succinct details of the final regulatory action(s)** | Trichlorfon is not included in the list of authorised active ingredients in Annex I to Directive 91/414/EEC. The authorisations for plant protection products containing trichlorfon had to be withdrawn by 21 November 2007. From 25 May 2007, no authorisations for plant protection products containing trichlorfon could be granted or renewed. |
| **3** | **Reasons for action** | Human health: unacceptable risks for operators, workers and bystanders.Environment: high risk for aquatic invertebrates. |
| **4** | **Basis for inclusion into Annex III** | The final regulatory action to ban trichlorfon was based on a risk evaluation taking into consideration local conditions in the EU Member States. |
| **4.1** | **Risk evaluation** | During the evaluation of trichlorfon a number of areas of concern have been identified. The review concluded that the risk evaluation for operator, workers, bystanders and consumer exposure could not be finalised. Trichlorfon is classified as harmful during oral exposure and as a skin sensitizer. Notably, the most sensitive effect observed during short-term exposure is reduction in acetylcholinesterase (AChE) activity. Due to lack of data it was not possible to establish an AOEL and the risk evaluation was performed on the basis of a provisional AOEL. In the absence of dermal absorption studies and taking into account physical and chemical properties, the default dermal absorption value of 100 % was considered appropriate for the risk evaluation. This resulted in exposure estimates that were much higher than the provisional AOEL for operators, workers and bystanders. In addition, concerns were identified with regard to the level of relevant impurities (*e.g.* dichlorvos) in the technical material and the risk for aquatic organisms. The use of trichlorfon examined during the risk evaluation includes the use of a permanent structure that protects the plants (*e.g.* a glasshouse). Therefore, the risk to birds and mammals was regarded as low based on limited exposure to trichlorfon via tomatoes grown under protection. The risk to non-target arthropods, earthworms, other soil non-target macro-organisms and non-target plants was also considered to be low. However, although the aquatic toxicity data are inadequate, the assessment on the existing study suggests that the risk to aquatic organisms can already be considered as high, and the risk to bees could not be assessed due to the lack of data. Moreover, due to the lack of information, a sound assessment of the route and rate of degradation of trichlorfon in soil could not be concluded. For similar reasons, potential for contamination of surface and groundwater by trichlorfon could not be adequately assessed. There is also an outstanding data gap for a study on the effects of trichlorfon on sewage treatment plants. |
| **4.2** | **Criteria used** | Risks to human health and the environment |
|  | **Relevance to other States and Region** | Similar concerns to those identified are likely to be encountered in other countries where the substance is used, particularly in developing countries. |
| **5** | **Alternatives** | None reported. |
| **6** | **Waste management** | None reported. |
| **7** | **Other** | None reported. |

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| **Brazil** |

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| **1** | **Effective date(s) of entry into force of actions** | 18 August 2010 (date of publication of Resolucion-RDC No. 37 of 16 August 2010) |
|  | **Reference to the regulatory document** | Resolution-RDC No. 37 of16 August 2010: Technical regulation on the active ingredient Trichlorfon as a result of Toxicological Revaluation. Available at <http://www.brasilsus.com.br/legislacoes/rdc/105101-37.html> (in Portuguese) or in Document UNEP/FAO/RC/CRC.8/5/Add.2 (p. 8) (English translation) |
| **2** | **Succinct details of the final regulatory action(s)** | All uses of trichlorfon as a pesticide for agricultural purposes are prohibited. Registrations of all technical products and formulations based on trichlorfon active ingredient, including its domestic usage, were cancelled. Therefore, the production, trade and import of trichlorfon had all been banned. |
| **3** | **Reasons for action** | Health risks for agricultural workers, bystanders and the general population. |
| **4** | **Basis for inclusion into Annex III** | The final regulatory action to ban trichlorfon was based on a risk evaluation taking into consideration local conditions in Brazil. |
| **4.1** | **Risk evaluation** | Brazilian Law No 7.802 /89, establishes in Article 3 that a pesticide may be banned when “(...), (c) it is teratogenic, mutagenic or carcinogenic according to updated results or experiences of the scientific community; (d) when it causes hormonal disorders, damages to the reproductive system, according to updated procedures and experiences in the scientific community; (f) when it causes damage to the environment”.Trichlorfon is an organophosphorous insecticide that has high potential to cause neurotoxic effects (neurobehavioral and neurochemical features), anatomical and cell damage in humans. The main mechanism of neurotoxicity of trichlorfon is the acetylcholinesterase inhibition. It can overstimulate the nervous system causing nausea, dizziness, confusion, and at very high exposure, respiratory paralysis and death.Trichlorfon is also genotoxic, immunotoxic, carcinogenic, teratogenic, causes adverse effects on reproduction and on the endocrine system. Experimental studies indicate that trichlorfon, as well as dichlorvos, its main metabolite, lead to depletion of the immune response. These immunosuppressive effects may increase the susceptibility of individuals exposed to trichlorfon on infections by pathogens and increase the cases of neoplasms.Many cases of intoxication of farm workers are reported and population living nearby the areas with extensive use. Comparative studies between intoxicated humans and animals after acute exposure to trichlorfon have shown that the neurotoxic effect is more aggressive in humans than in animals, thus conforming to a situation susceptible to ban this active ingredient in Brazil. |
| **4.2** | **Criteria used** | Risks to human health |
|  | **Relevance to other States and Region** | None reported. |
| **5** | **Alternatives** | None reported. |
| **6** | **Waste management** | None reported. |
| **7** | **Other** | Hazard classification of trichlorfon in Brazil:ANVISA: Class II Highly toxicIBAMA: Class III Dangerous to the environment |

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| **Annex 3 – Addresses of designated national** **authorities**  |

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| **European Union** |
| European CommissionDG EnvironmentBU-9, 06/164B-1049 BrusselsBelgiumDr. Juergen HelbigPolicy Officer | **Phone**  +32 2 298 8521**Fax**  +32 2 296 7617**E-mail** Juergen.Helbig@ec.europa.eu  |

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| **Brazil** |
| Ministry of EnvironmentDepartment of Environmental Quality in Industry,Secretariat of Climate Change and Environmental QualityEsplanada dos Ministérios, Bloco B, 8o andar, GabineteSérgia de Souza OliveiraDirector | **Phone**  +55 61 20018 1244**Fax**  +55 61 2028 1759**E-mail** sergia.oliveira@mma.gov.br, gsq@mma.gov.br |

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| **Annex 4 – References**  |

**Regulatory actions**

**European Union**

Commission Decision 2007/356/EC of 21 May 2007 concerning the non-inclusion of trichlorfon in Annex I to Council Directive 91/414/EEC and the withdrawal of authorisations for plant protection products containing that substance (*Official Journal of the European Union* L 133 of 25.5.2007, p.42-43). Available at:
<http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2007:133:0042:0043:EN:PDF>

**Brazil**

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<http://www.brasilsus.com.br/legislacoes/rdc/105101-37.html> (in Portuguese) or in
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1. 1 According to the Convention, the term “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. [↑](#footnote-ref-1)
2. 2 According to the Convention, the term “Party” means a State or regional economic integration organization that has consented to be bound by the Convention and for which the Convention is in force. [↑](#footnote-ref-2)
3. 3As indicated by the Depositary of the Convention in a notification dated 31 March 2010 (reference: C.N.182.2010.TREATIES-2), which was in turn based on a communication from the Council of the European Union dated 8 March 2010, following the entry into force of the Treaty of Lisbon amending the Treaty on European Union and the Treaty establishing the European Community, with effect from 1 December 2009 the European Union replaced the European Community (Article 1, third paragraph, of the Treaty of Lisbon) and took over all rights and obligations of the European Community. The former European Community has accordingly been replaced by the European Union in respect of all conventions or agreements for which the Secretary-General of the United Nations is the depositary and to which the European Community is a signatory or a contracting party. [↑](#footnote-ref-3)
4. 4 For revised endpoints and other factors for acute and chronic dietary exposure see US EPA (2006): Report on FQPA Tolerance Reassessment Progress and Interim Risk Management Decision (TRED) for Trichlorfon, p. 11. Available at: www.epa.gov/pesticides/reregistration/REDs/trichlorfon\_red.pdf [↑](#footnote-ref-4)