Canadian Water Quality Guidelines for the Protection of Aquatic Life

TRICHLORFON

Trichlorfon (CAS 52-68-6) is an organophosphate insecticide used to control pests such as cockroaches, crickets, silverfish, bedbugs, fleas, cattle grubs, flies, ticks, leafminers and leaf-hoppers (EXTOXNET, 1996). Trichlorfon is manufactured by Bayer CropScience Inc. and is produced in soluble powder, granular, emulsifiable concentrate, and fly bait formulations. It was first registered in Canada in 1980, and is currently registered under the trade names Dylox 420, Dipterex, Neguvon pour-on cattle insecticide, and Dylox 80% (PMRA, 2004).

Production and Uses: Trichlorfon was introduced to the commercial market in 1952 (Lorenz et al., 1955). Dylox 420, containing 420 g active ingredient (a.i.)·L^{-1}, is a liquid insecticide applied aerially to field crops (e.g., alfalfa, grains, corn, tobacco), fruits (e.g., berries), vegetables, and ornamentals (e.g., flowers, shrubs, trees) (PMRA, 2004). Dylox 80% is a soluble powder, containing 80% trichlorfon, which is applied to field crops, vegetables, and ornamentals (PMRA, 2004). Neguvon pour-on cattle insecticide is used to control lice and grubs on cattle. It is a liquid formulation (8% trichlorfon) and is applied directly to the animals' backs (PMRA, 2004). Other applications include the control of pests around domestic areas and on animals and fish, as fly bait, and to treat intestinal and ectoparasites of fish. The amount of trichlorfon in registered products ranges from 1% active ingredient in bait products to 98% active ingredient in technical grade (U.S. EPA, 1997). The Pest Management Regulatory Agency (PMRA) has recently re-evaluated trichlorfon (PMRA, 2008). The re-evaluation eliminated a number of uses, including essentially all residential uses and restricted other uses considerably.

Sources to the environment: Trichlorfon is administered using spray and aerial application methods. These applications to soil and vegetation can expose non-target terrestrial organisms to trichlorfon. Most trichlorfon-containing products used in agriculture (e.g., Dylox 420) are applied multiple times throughout the year, thus prolonging the exposure period to non-target organisms. Although trichlorfon is not to be applied directly to water bodies (PMRA, 2004), the pesticide may enter these systems by leaching, runoff, or spray drift, thus exposing aquatic organisms to residues (U.S. EPA, 1997). Losses as a result of volatilization following application may be significant depending on environmental conditions.

Fate, behaviour and partitioning: Dichlorvos (2,2,-dichlorovinyl dimethyl phosphate) is the major transformation product of trichlorfon (Hofer, 1981; U.S. EPA, 1997). Biotransformation in soil and hydrolysis in water are the primary methods of trichlorfon transformation (HSDB, 1999). The transformation to dichlorvos in water occurs by dehydrochlorination (IPCS, 1992). The rate of transformation increases at greater water alkalinities (IPCS, 1992). Trichlorfon can be transformed to dichlorvos through photolysis. Dichlorvos causes the inhibition of cholinesterase at a rate ≥100 times that of trichlorfon (Hofer, 1981). For freshwater fish, dichlorvos 96h-LC_{50}s ranged from 200 µg a.i.·$L^{-1}$for lake trout (*Salvelinus namaycush*) to 8,900 µg a.i.·$L^{-1}$for walking catfish (*Clarias batrachus*) (IPCS, 1986).

Trichlorfon is highly soluble in water, with a solubility of 154,000 mg a.i.·L^{-1} at 25°C (Mackay et al., 1999). The melting point for trichlorfon has been reported as 83-84°C (Mackay et al.1999).

Table 1. Canadian Water Quality Guidelines (CWQG) for trichlorfon for the protection of aquatic life (µg a.i.·L^{-1})

	Long-Term Exposure	Short-Term Exposure
Freshwater	0.009*	1.1**
Marine	NRG	NRG

* **Interim CWQG** - calculated from short-term low-effect data using lowest endpoint approach

** Value calculated from LC_{50} data using the SSD approach

NRG = no recommended guideline

Trichlorfon has a Henry's Law constant of 1.72 x 10^{-9} kPa·m^3 0183·mol^{-1} (HSDB, 1999) and a vapour

pressure of 1.04 x 10^{-6} kPA (Giang et al., 1954; FAO/WHO, 1972; Dedek, 1981; IARC, 1983). These values indicate that trichlorfon is non-volatile

Bioconcentration factors (BCF) are a measure of the tendency of a substance to migrate from water to the body tissues of aquatic organisms where it can accumulate and become concentrated (Rand et al., 1995). An estimated BCF of 3 is reported for aquatic organisms exposed to trichlorfon. This suggests that the potential for bioconcentration is low (HSDB, 1999). This is supported by the rapid rate of transformation and low octanol-water partition coefficient reported for trichlorfon. These factors indicate that trichlorfon will have a low persistence in water and is unlikely to partition to the lipids of aquatic organisms. The results of Lopes et al. (2006) support this, as they found a BCF of 0.41 $L \cdot kg^{-1}$ in the South American ray-finned fish *Piaractus mesopotamicus* (Pacu) and they found a half-life time of 57h (2.5d) for trichlorfon in water. Therefore bioaccumulation and biomagnification of trichlorfon is unlikely to occur.

The high water solubility and low octanol-water partition coefficient of trichlorfon indicate that it will preferentially remain in water rather than partition to organic matter. This is supported by a low soil adsorption coefficient, indicating that trichlorfon does not have an affinity for sediment or suspended solids (HSDB, 1999).

Trichlorfon is considered to be highly mobile in soil, based on its water solubility (154,000 mg/L) and soil adsorption coefficient (log K_{oc} = 0.78-1.90) (HSDB, 1999). These properties suggest that groundwater contamination could occur. However, the rapid transformation of trichlorfon in soil could minimize such contamination (Purdue University, 1987; U.S. EPA, 1997).

Analytical methods: A method to pre-concentrate water samples for the measurement of trichlorfon was reported by Dedek et al. (1987). The standard procedures for residue detection involve extraction with acetonitrile and re-extraction with ether, followed by gas chromatography detection using flame photometric detection or flame thermionic detection. During chromatography, trichlorfon is thermally decomposed to dimethyl phosphate which is then determined (Ferreira and Fernandes, 1980). Acetylation or trimethylsilylation can be used to stabilize trichlorfon for gas chromatography, thus avoiding decomposition (Vilceanu et al., 1973; Bowman and Dame, 1974).

Recent methods have been developed to analyse trichlorfon through liquid chromatography-mass spectroscopy (LC-MS). Trichlorfon has the capacity to catalyze the oxidation of benzidine (4,4′-diamino-biphenyl) to 4-amino-4′-nitro biphenyl in the presence of sodium perborate. The product of the catalyzed reaction can then be measured by LC–MS methods. Reversed-phase high performance liquid chromatography with 365 nm UV detection is then used for separation and quantification of 4-amino-4′-nitro biphenyl. The limit of detection using this method is 2.0 µg a.i.$\cdot L^{-1}$., with recorded recoveries ranging from 67.5 to 82.1%, with relative standard deviations between 4.5 and 7.3% (Zhu et al., 2007).

Ambient concentrations: The National Water Research Institute, Environment Canada recently completed a three year surveillance program of pesticides in each of the five Environment Canada regions (Atlantic, Quebec, Ontario, Prairie & Northern and Pacific Yukon) (Environment Canada, 2011). Trichlorfon was not detected in surface waters sampled in 2003 (n=27 around the Great Lakes. In isolated Ontario lakes, trichlorfon was rarely detected (3 of 163 samples) but had the highest concentration detected relative to 44 other pesticides (0.065 $\mu g \cdot L^{-1}$) (Environment Canada, 2011).

Mode of action: The primary mode of action for organophosphorus pesticides like trichlorfon, is cholinesterase (ChE) inhibition. Cholinesterase is the enzyme that breaks down acetylcholine (AChE), the neurotransmitter responsible for nerve impulses between nerves and their receptors. Inhibition of cholinesterase leads to an accumulation of acetylcholine, causing disruptions in the central nervous system of affected animals. Acetylcholine is an important neurotransmitter across a wide range of taxa. Symptoms of toxicity include hyperactivity, tremors, terminal convulsions and death (IPCS, 1986; Jensen and Gaufin, 1964).

Trichlorfon is one of the few organophosphates that transforms into a more toxic compound (Howe et al., 1994). Dichlorvos (the break-down product of trichlorfon) inhibits cholinesterase at ≥ 100 times the

rate of trichlorfon (Hofer, 1981). Transformation to the neurotoxin dichlorvos occurs when trichlorfon is hydrolyzed in water, biological fluids, and tissues at pH levels greater than 5.5 (IPCS, 1992).

Uptake of trichlorfon can occur through all routes of exposure (oral, dermal and inhalation), and rapidly permeates to all tissues (IPCS, 1992). Although the primary mode of action is the inhibition of cholinesterase, trichlorfon has also been observed to impede the immune response of fish (Chandrasekara and Pathiratne, 2005; Dunier et al., 1991). Trichlorfon is moderately toxic to fish and generally more toxic to aquatic invertebrates. The primary mode of action of trichlorfon on cyanobacteria is the inhibition of nitrogen metabolism, resulting in the alteration of growth, cell composition, ultrastructure and physiological processes (Marco et al., 1990; Martinez et al., 1991).

***Freshwater Toxicity**:* In the following sections, all concentrations of trichlorfon expressed in μg a.i.·L^{-1} refer to μg of active ingredient (a.i.) per litre. Toxicity tests used in the development of the guideline are based on active ingredient. Formulations in which the percent active ingredient was not sufficiently present (< 90% a.i.) were not used in the development of the guideline, the presence of trichlorfon containing formulations are monitored by the presence of the trichlorfon active ingredient. Trichlorfon containing formulations vary widely in the percentage of trichlorfon in the product depending on use and region.

Trichlorfon is moderately to highly toxic to freshwater fish (IPCS, 1992). Toxicity values available from the literature ranged from a 96h-LC_{50} of 234 μg a.i.·$L^{-1}$for bluegill sunfish (*Lepomis macrochirus*) to a 96h-TLm of 180,000 μg a.i.·L^{-1} for fathead minnow (*Pimephales promelas*) (Mayer and Ellersieck, 1986; Pickering et al., 1961). The lack of information reported in these studies for factors like life stage, exposure conditions, and test type make it difficult to explain why such a large range existed. Differences in species behaviour, feeding ecology, choline receptor sensitivity, and pharmacokinetics likely account for some of the variation between species. Toxicity values were reported for several fish species including goldfish (*Carassius auratus*), carp (*Cyprinus carpio*), rainbow trout (*Oncorhynchus mykiss*), channel catfish (*Ictalurus punctatus*), bluegill sunfish (*Lepomis macrochirus*), and fathead minnow (*Pimephales promelas*).

Little information or data on long-term effects on fish were available. One study by Siwicki et al. (1990) reported a 56d-LOEC for immunological response in carp (*Cyprinus carpio*) of 400,000 μg a.i.·L^{-1}. Two percent active ingredient was used in this study and may explain why the organism was so tolerant. This study was classified as unacceptable.

Trichlorfon is highly toxic to most aquatic invertebrates (IPCS, 1992). Short-term values for freshwater invertebrates ranged from a 48h-EC_{50} of 0.18 μg a.i.·L^{-1} for *Daphnia pulex* (water flea) to a 24h-LC_{50} of 51,970 μg a.i.·L^{-1} for *Brachionus calyciflorus* (rotifer) (Mayer and Ellersieck, 1986; Ferrando and Andreu-Moliner, 1991). Sublethal symptoms of toxicity included hyperactivity, tremors and convulsions (Jensen and Gaufin, 1964). Recovery was observed in organisms placed in clean water following exposure (Jensen and Gaufin, 1964).

A limited number of long-term studies were available for invertebrates. Jensen and Gaufin (1964) investigated long-term effects of trichlorfon on behaviour and survival of stonefly nymphs (*Pteronarcys californica* and *Acroneuria pacifica*) by exposing the species to Dylox in a flow-through system for up to 30 days. The 30d-TLm for *P. californica* was 9.8 μg a.i.·L^{-1}. Similar sensitivity was reported for *A. pacifica* with a 30d-TLm of 8.7 μg a.i.·L^{-1} (Jensen and Gaufin, 1964). Symptoms of toxicity observed in surviving stonefly naiads included hyperactivity, tremors, and convulsions. Water fleas (*Daphnia magna*) exposed to trichlorfon in a flow-through test had a 21d-LOEC of 5.6 μg a.i.·L^{-1} (SSRD, 1997). A 10d-LC_{50} of 2,200 μg a.i.·L^{-1} was reported for adult *Pila globosa* (gastropod) (Singh and Agarwal, 1981).

Few studies have been performed on the toxicity of trichlorfon to algae or aquatic plants. Trichlorfon alters growth, cell composition, ultrastructure and physiological processes in cyanobacteria, with toxicity attributed to the inhibition of nitrate uptake (Marco et al., 1990). However, available studies have shown the alga *Chlorella vulgaris* to be highly tolerant of trichlorfon with 72h-LOECs ranging from 25,000 to 100,000 μg a.i.·L^{-1} for effects to photosynthesis and physiological processes, respectively (Martinez et al., 1991).

One study reported effects on amphibians from exposure to trichlorfon. Szubartowska et al. (1990)

reported 1-, 2- and 3-week LOECs of 4,000 µg a.i.·L^{-1} for effects on hematocrit (the percentage of whole blood that is composed of red blood cells) and mean corpuscular volume in the green frog (*Rana esculenta*).

Marine Toxicity*:* No acceptable marine toxicity studies for trichlorfon were found.

Toxicity Modifying Factors: There are insufficient data regarding the effects of pH, temperature, hardness, and UV radiation on the toxicity of trichlorfon to reliably identify patterns of toxicity modifying effects or to normalize toxicity data. However, as pH increases, so does toxicity (Howe et al., 1994, Woodward and Mauck, 1980), due to the more rapid transformation of trichlorfon to dichlorvos. Similarly, the pH of exposure media significantly influences the toxicity of trichlorfon. Temperature also increases the rate of transformation of trichlorfon to dichlorvos by increasing metabolism, and increasing the rate of trichlorfon uptake (Howe et al., 1994; Woodward and Mauck, 1980).

Water Quality Guideline Derivation: The water quality guidelines for trichlorfon are adopted from the Ideal Performance Standards developed for the National Agri-Environmental Standards Initiative (Environment Canada 2006), based on CCME (1991) for the long-term guidelines and CCME (2007) for the short-term benchmark concentration. The short-term benchmark concentration was developed using the statistical approach with a species sensitivity distribution (SSD). The long-term freshwater quality guideline was developed using the lowest-endpoint and an application of a safety factor. There were no marine studies available, so no marine guidelines were developed.

Short-term Freshwater Benchmark Concentration: Short-term benchmark concentrations are derived using severe effects data (such as lethality) of defined short-term exposure periods (24- to 96-h). These values estimate severe effects to the aquatic ecosystem and are intended to give guidance on the impacts of severe, but transient, situations (e.g., spill events to aquatic receiving environments and infrequent releases of short-lived/non-persistent substances). Short-term benchmark concentrations *are not* protective of aquatic life.

A total of 26 data points were used in the derivation of the short-term benchmark concentration. Toxicity studies meeting the requirements for primary and secondary data, according to CCME (2007) protocol, were considered in the derivation of the short-term SSD. Each species for which appropriate short-term toxicity was available was ranked according to sensitivity, and its centralized position on the SSD was determined using the Hazen plotting position (estimate of the cumulative probability of a data point). Intra-species variability was accounted for by taking the geometric mean of the studies considered to represent the most sensitive lifestage and endpoint. Table 2 presents the final dataset that was used to generate the fitted SSD for trichlorfon. For detailed information, including which studies were used to calculate the geometric means for the various species, refer to Table 8.1 and 8.2 of the supporting document. Aquatic toxicity studies reported by the USEPA (Mayer and Ellersieck, 1986; SSRD, 1997) and Health Canada's Pest Management Regulatory Agency (PMRA) were generally classified as primary data, but downgraded if warranted during evaluation.

The normal cumulative distribution function (CDF) model provided the best fit of the models tested (Anderson-Darling Statistic = 0.334). The equation of the fitted normal model is of the form:

$$f(x) = \frac{1}{2}\left(1 + erf\left(\frac{x-\mu}{\sigma\sqrt{2}}\right)\right)$$

where μ = 2.2481 and σ = 1.3447, are the location and scale parameters of the model, x is the concentration metameter, and the functional response, f(x), is the proportion of taxa affected.

Summary statistics for the short-term SSD (Figure 1) are presented in Table 3.

Therefore, the short-term exposure benchmark concentration indicating the potential for severe effects (e.g., lethality or immobilization) to sensitive freshwater life during transient events is 1.1 µg a.i.·L^{-1} for trichlorfon.

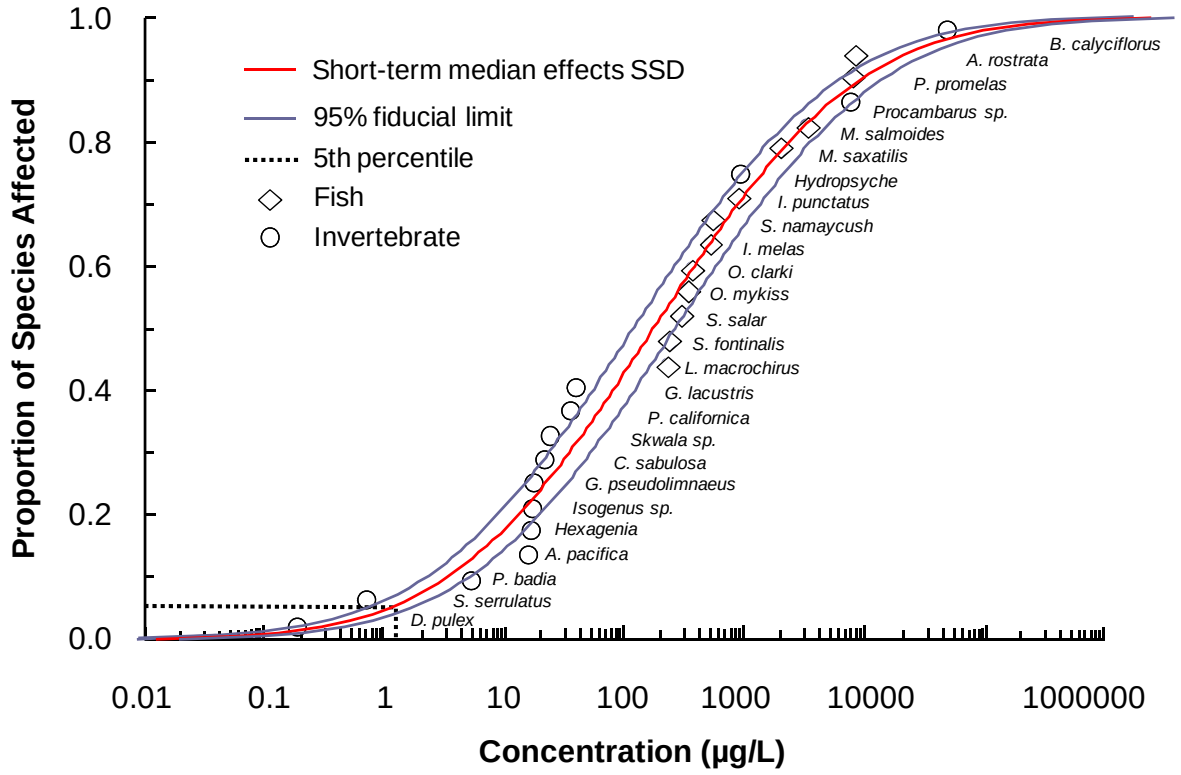

Figure 1. Short-term SSD representing the toxicity of trichlorfon in freshwater consisted of acceptable short-term LC_{50}s, EC_{50}s, and TL_ms of 26 aquatic species versus proportion of species affected.

Table 2. Endpoints used to determine the short-term freshwater benchmark concentration for trichlorfon

Species	Endpoint	Concentration (µg a.i.·L^{-1})
Fish		
Lepomis macrochirus	96-h LC_{50}	234**
Salvelinus fontinalis	96-h LC_{50}	240**
Salmo salar	96-h LC_{50}	300**
Oncorhynchus mykiss	96-h LC_{50}	330*
Oncorhynchus clarki	96-h LC_{50}	375**
Ictalurus melas	96-h LC_{50}	515
Salvelinus namaycush	96-h LC_{50}	550
Ictalurus punctatus	96-h LC_{50}	880
Morone saxatilis	96-h LC_{50}	2000
Micropterus salmoides	96-h LC_{50}	3,450
Pimephales promelas	96-h LC_{50}	7,900
Anguilla rostrata	96-h LC_{50}	8,570
Invertebrates		
Daphnia pulex	48-h EC_{50}	0.18
Simocephalus serrulatus	48-h LC_{50}	0.7
Pteronarcella badia	96-h LC_{50}	5.3
Acroneuria pacifica	96-h LC_{50}	16.5
Hexagenia	24-h TL_m	17
Gammarus pseudolimnaeus	96-h LC_{50}	17*
Isogenus sp.	96-h LC_{50}	17*
Claasenia sabulosa	96-h LC_{50}	22
Skwala sp.	96-h LC_{50}	24
Pteronarcys californica	96-h LC_{50}	35
Gammarus lacustris	96-h LC_{50}	40
Hydropsyche	24-h TL_m	910
Procambarus sp.	96-h LC_{50}	7,800
Brachionus calyciflorus	24-h LC_{50}	49,423*

* value shown is the geometric mean of comparable values
** only the most sensitive value of the acceptable data was used in the SSD due to the wide range of pH values used in the toxicity tests

Table 3. Short-term freshwater benchmark concentration for trichlorfon resulting from the SSD method.

	Concentration (µg a.i.·L^{-1})
SSD 5th percentile	1.1
SSD 5th percentile, LFL (5%)	0.7
SSD 5th percentile, UFL (95%)	1.7

Long-term Freshwater interim CWQG: Long-term exposure CWQGs identify benchmarks in the aquatic ecosystem that are intended to protect the most sensitive species and life stage for indefinite exposure periods.

The long-term endpoints identified from the primary and secondary studies consisted of three invertebrate species. The available long-term toxicity data were insufficient to derive a guideline using the SSD approach. Therefore, the Ideal Performance Standard (Environment Canada 2006) was adopted as the long-term freshwater guideline. The IPS derivation followed CCME (1991) protocol for interim guideline development due to the paucity of long-term data. The interim CWQG was thus calculated by applying a safety factor of 20 to the short-term EC_{50} of the most sensitive species, namely a 48-h EC_{50} of 0.18 µg a.i.·L^{-1} for *D. pulex* (Table 2). The recommended application factor for trichlorfon is 20 because trichlorfon is considered relatively non-persistent in water.

The CWQG is calculated as follows:

$$CWQG = EC_{50} \div AF$$
$$= 0.18 \text{ µg a.i.·L}^{-1} \div 20$$
$$= 0.009 \text{ µg a.i.·L}^{-1}$$

where AF = application factor

Therefore, the long-term freshwater interim CWQG for the protection of freshwater life is 0.009 µg a.i./L, for trichlorfon.

Short-term Benchmark Concentrations and Long-term Marine CWQGs: No acceptable marine studies were found, therefore there were insufficient data to derive short- or long-term guidelines for the protection of marine life.

Implementation and other considerations: The above guideline was developed using only toxicity data derived using the trichlorfon active ingredient. In regions of high trichlorfon use, additional site-specific guidance and sampling may be required to determine

the effects of formulated products which include trichlorfon. Furthermore, due to the rapid transformation of trichlorfon to dichlorvos, additional site specific guidance may be required to ensure aquatic life is not being impacted, especially in regions elevated in pH and/or temperature.

References

Bowman, P.B. and P.W. Dame. 1974. Gas-liquid chromatography of trichlorfon in soluble powder formulations. Journal Association of Analytical Chemists 57:1128-1131.

CCME (Canadian Council of Ministers of the Environment). 1991. Appendix IX - A protocol for the derivation of water quality guidelines for the protection of aquatic life (April 1991). *In*: Canadian water quality guidelines. Canadian Council of Resource and Environment Ministers, 1987. [Updated and reprinted CCME, 1999, Winnipeg.].

CCME (Canadian Council of Ministers of the Environment). 2007. A Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life. Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Chandrasekara, H.U., and A. Pathiratne. 2005. Influence of low concentrations of trichlorfon on haematological parameters and brain acetylcholinesterase activity in common carp, *Cyprinus carpio* L. Aquaculture Research 36:144-149.

Dedek, W. 1981. Guanine N7-alkylation in mice *in vivo* by metrifonate-discussion of possible genotoxic risk in mammals. Acta Pharmacologica et Toxicologica Supplemental 49:40-50.

Dedek, W., K.D. Wenzel, F. Luft,H. Oberländer, and B. Mothes. 1987. Preconcentration of hydrophilic pesticides from aqueous solutions and extraction of residues using the polymeric sorbent Wofatil Y77. I. Preconcentration of hydrophilic pesticides from water. Fresenius Z. Anal. Chem., 328: 484-486.

Dunier, M., A.K. Siwicki, and A. Demani. 1991. Effects of organophosphorus insecticides: effects of trichlorfon and dichlorvos on the immune response of carp (*Cyprinus carpio*). Ecotoxicology and Environmental Safety 22:79-87.

Environment Canada 2006. Ideal Performance Standards (IPS) for trichlorfon. National Agri-Environmental Standards Initiative. Report No. 2-37

Environment Canada. 2011. Presence, levels and relative risks of priority pesticides in selected Canadian aquatic ecosystems. Water Science and Technology Directorate, Environment Canada. Burlington, ON. March 2011.

EXTOXNET (Extension Toxicology Network). 1996. Pesticide Information Profiles Trichlorfon. Cornell University, Ithaca, NY. http://extoxnet.orst.edu/pips/trichlorfon.htm.

FAO/WHO. 1972. Trichlorfon. In: 1971 *Evaluations of some pesticide residues in food,* Geneva, World Health Organization, pp. 183-230 (WHO Pesticide Residues Series, No. 1).

Ferrando, M.D., and E. Andreu-Moliner. 1991. Acute lethal toxicity of some pesticides to *Brachionus calyciflorus* and *Brachionus plicatilis*. Bulletin of Environmental Contamination and Toxicology 47:479-484.

Ferreira, J.R. and A.M.S.S. Fernandes. 1980. Gas-liquid chromatographic determination of organophosphorus insecticide residues in fruits and vegetables. Journal Association of Analytical Chemists 63:517-522.

Giang, P.A., W.F. Barthel and S.A. Hall. 1954. Colorimetric determination of O,O-dialkyl 1-hydroxyphosphonates derived from chloral. Journal of Agriculture and Food Chemistry 2:1281-1284.

Hofer, W. 1981. Chemistry of metrifonate and dichlorvos. Acta Pharmacologica Scandinavian*,* 49(suppl.5):7-14.

Howe, G.E., L.L. Marking, T.D. Bills, J.J. Rach., and F.L. Mayer, Jr. 1994. Effects of water temperature and pH on toxicity of terbufos, trichlorfon, 4-nitrophenol and 2,4-dinitrophenol to the amphipod Gammarus pseudolimnaeus and rainbow trout (*Oncorhynchus mykiss*). Environmental Toxicology and Chemistry 13:51-66.

HSDB (Hazardous Substances Data Bank). 1999. Trichlorfon. National Library of Medicine, Bethesda, MD. URL: http://toxnet.nlm.nih.gov/

IARC (International Agency for Research on Cancer). 1983. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: 1972-PRESENT. World Health Organization, International Agency for Research on Cancer, Geneva.

IPCS (International Programme on Chemical Safety). 1986. Environmental Health Criteria No. 63. Organophosphorus Insecticides: A general introduction. World Health Organization, Geneva. URL: http://www.inchem.org/documents/ehc/ehc/ehc63.htm

IPCS (International Programme on Chemical Safety). 1992. Environmental Health Criteria No. 132. Trichlorfon. World Health Organization, Geneva. URL: http://www.inchem.org/documents/ehc/ehc/ehc132.htm

Jensen, L.D. and A.R. Gaufin. 1964. Long-Term Effects of Organic Insecticides on Two Species of Stonefly Naiads. Transactions of the American Fisheries Society 93: 357-363.

Lopes, R.B., L.C. Paraiba, P.S. Ceccarelli, and V.L. Tornisielo. 2006. Bioconcentration of trichlorfon insecticide in pacu (*Piaractus mesopotamicus*). Chemosphere 64:56-62.

Lorenz, W, A. Henglein and G. Schrader. 1955. The new insecticide *O,O*-dimethyl 2,2,2-trichloro-1-hydroxy ethylphosphonate. Journal of the American Chemistry Society 77:2554-2556.

Mackay, D., W.Y. Shiu, and K.C. Ma. 1999. Physical-chemical Properties and Environmental Fate Handbook. CRC Press LLC, Boca Raton, FL.

Marco, E., F. Martinez, and M.I. Orus. 1990. Physiological alterations induced by the organophosphorus insecticide trichlorfon in Anabaena PCC 7119 grown with nitrates. Environmental and Experimental Botany 30(2):119-126.

Martinez, F., J.A. Jarillo, and M.I. Orus. 1991. Interactions between trichlorfon and three Chlorophyceae. Bulletin of Environmental Contamination and Toxicology 46:599-605.

Mayer Jr., F.L. and M.R. Ellersieck. 1986. Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemicals and 66 Species of Freshwater Animals. Resource Publication No. 160, U.S.Department of Interior, Fish Wildlife Service, Washington, DC.

Pickering, Q.H., C. Henderson, and A.E. Lemke. 1961. Toxicity of Organic Phosphorus Insecticides to Different Species of Warmwater Fishes. Transactions of the American Fisheries Society 90/91:175-184.

PMRA (Pest Management Regulatory Agency). 2004. Evaluation Delivery Dossier Electronic (EDDENet). Electronic Labels: Search and Evaluation. Pest Management Regulatory Agency, Ottawa, Ontario. URL: http://eddenet.pmra-arla.gc.ca/4.0/4.01.asp.

PMRA (Pest Management Regulatory Agency). 2008. Re-evaluation Decision RVD2008-27: Trichlorfon. Pest Management Regulatory Agency, Ottawa, Ontario.

Purdue University. 1987. National Pesticide Information Retrieval System. Accessed March, 2006. http://ppis.ceris.purdue.edu/

Rand, G.M., P.G. Wells and L.S. McCarty. 1995. Fundamentals of Aquatic Toxicology, Second Edition. Taylor & Francis, Washington, DC. ISBN 1-506032-091-5.

Singh, O. and R.A. Agarwal. 1981. Toxicity of certain pesticides to two economic species of snails in northern India. Journal of Economic Entomology 74:568-571.

Siwicki, A.K., M. Cossarini-Dunier, M. Studnicka and A. Demael. 1990. In Vivo Effect of the Organophosphorus Insecticide Trichlorphon on Immune Response of Carp (*Cyprinus carpio*). Ecotoxicology and Environmental Safety 19:99-105.

Sundaram, K.M.S. and I.W. Varty. 1989. Distribution and persistence of trichlorfon in a forest environment. Journal of Environmental Science and Health B24(6):647-659.

Szubartowska, E., K. Gromysz-Kałkowska, and K. Wójcik. 1990. Behavior of the formed blood elements in *Rana esculenta* L. after repeated contacts of the animal with a therapeutic dose of Foschlor. Bulletin of Environmental Contamination and Toxicology 45:796-803.

U.S. EPA (United States Environmental Protection Agency). 1997. Reregistration Eligibility Decision (RED). Trichlorfon. EPA 738-R-98-017. US Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Washington, DC.

Vilceanu, R., P. Schulz, R. Draghici and P. Soimu. 1973. Gas chromatography of (1-hydroxy-2,2,2-trichlorethyl)-phosphonic acid esters. Journal of Chromatography 82:285-290 (in German).

Woodward, D.F. and W.L. Mauck. 1980. Toxicity of five forest insecticides to cutthroat trout and two species of aquatic invertebrates. Bulletin of Environmental Contamination and Toxicology 25: 846-853.

Zhu, H.-Z., Y.-M. Cui, X.-W. Zheng, H.-R. Han, and M.-M. Yang. 2007. Determination of trace trichlorfon by high performance liquid chromatography with UV detection based on its catalytic effect on sodium perborate oxidizing benzidine. Analytica Chimica Acta 584(1): 166-171.

Reference listing:

Canadian Council of Ministers of the Environment. 2012. Canadian water quality guidelines for the protection of aquatic life: Trichlorfon. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

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