Canadian Water Quality Guidelines for the Protection of Aquatic Life

IMIDACLOPRID

Imidacloprid (CAS Registry Number 13826-41-3; IUPAC name 1-(6-chloro-3-pyridylmethyl)-N-nitroimidazolidin-2-ylideneamine) is a synthetic active ingredient used in various insecticide products registered for use in Canada. It is a colourless crystalline solid, has the molecular formula $C_9H_{10}ClN_5O_2$ and a molecular weight of 255.7 (Tomlin 2000). Imidacloprid is very soluble in water, with a solubility of 0.51 to 0.61 $g \cdot L^{-1}$ at 20°C (Krohn 1989; Tomlin 2000), and is relatively non-volatile with reported vapour pressures of 2×10^{-7} to 4×10^{-10} Pa at 20°C (EXTOXNET 1998; Tomlin 2000).

Imidacloprid, produced by Bayer CropScience Inc., was sold and used for the first time in Canada in 1995 for the control of the Colorado potato beetle in eastern Canada (PMRA 2001). Since then, its registered uses have expanded to include: control of various insects on field and greenhouse crops, orchards, and nurseries; flea control on household pets; control of turf pests in urban areas; and others. Formulations of imidacloprid are available as: a slurry for seed treatments, flowable concentrate for seed treatment, granule, wettable powder, soluble concentrate, suspension concentrate, water dispersible granules, and dustable powder (Tomlin 2000). Trade names used for imidacloprid-based pesticides include, but are not limited to, Admire, Advantage, Confidor, Gaucho, Genesis, Impower, Intercept, Maxforce IC, and Merit (PMRA 2005).

In agriculture, imidacloprid is used to control sucking insects such as aphids, leafhoppers, psyllids, thrips, whiteflies and beetles. It is most commonly applied as a soil and foliage treatment, and as a seed dressing (Tomlin 2000). Typical application rates to foliage or soil range from approximately 50 to 320 $g \cdot ha^{-1}$, depending on the crop (PMRA 2005). Imidacloprid is used to treat the seeds of crops such as canola, mustard, and corn (PMRA 2001). On potatoes, the recommended application rate is 6.2 to 9.4 g imidacloprid per 100 kg seed pieces (PMRA 2005). In urban areas, imidacloprid is used to control turf pests in household lawns, parks, athletic fields, golf courses, etc. For treatment of turfgrass to control white grubs, the recommended application rate is approximately 280 g a.i.$\cdot ha^{-1}$ (PMRA 2005). Imidacloprid is also used to control domestic pests such as fleas and cockroaches. For flea control on domestic pets, it is typically available as a solution that can be applied topically once a month to dogs and cats (PMRA 2005). Products contain varying percentages of active ingredient depending on the weight of the animal to which it is intended to be applied.

Based on data available for 7 provinces (NS, NB, PEI, ON, MB, AB, and BC), the total annual quantity of imidacloprid sold or used in Canada has been estimated at approximately 19,600 kg a.i. (Brimble et al. 2005). The actual quantity is probably considerably higher because the data for some provinces only reflected agricultural sales, and did not account for other uses such as flea and tick control on pets and applications in greenhouses and on turfgrass. Also, estimates of quantities sold or used in Saskatchewan were unavailable, but data suggest that imidacloprid is currently among the top 10 pesticides in use in that province (Sam Ferris, Saskatchewan Environment, Regina, personal communication, 2004). Several recent trends also suggest that imidacloprid use is likely to increase in the coming years. In Ontario, licensed pesticide applicators are using imidacloprid on lawns and turf as a replacement for diazinon, which was taken off the market for lawncare use in 2004 (Struger et al. 2002). In Alberta, imidacloprid is expected to replace lindane, which is being phased out, and this could result in increased sales in future years (Byrtus et al. 2002). Since the late 1990s there have also been major increases in imidacloprid useage for flea control in BC (ENKON Environmental Limited 2001).

Direct application of imidacloprid to water bodies is not permitted in Canada. Nonetheless, use of imidacloprid to control terrestrial pests could potentially result in unintended transport to aquatic habitats and indirect contamination through spray drift, atmospheric deposition, soil erosion, and runoff.

Table 1. Water quality guidelines for imidacloprid for the protection of aquatic life (CCME 2007).

Aquatic life	Guideline value (μg a.i.$\cdot L^{-1}$)
Freshwater	0.23*
Marine	0.65*

*Interim guideline.

Several analytical methods exist for measuring imidacloprid in water, but for quantification of low levels, the preferred method is solid-phase extraction with the use of liquid chromatography - mass spectrometry - mass spectrometry (LC-MS-MS). Specific methods using this approach have reported detection limits ranging from 0.1 $\mu g \cdot L^{-1}$ (Culp et al. 2006) to 0.001 $\mu g \cdot L^{-1}$ (Giroux 2003).

Monitoring for imidacloprid has been conducted at various Canadian locations in surface waters, runoff, and groundwater. In surface waters, imidacloprid has rarely been measured above detection limits. For example, analyses of Alberta surface water samples collected in 1999 and 2000 found no imidacloprid above the detection limits of 0.02 – 0.05 $\mu g \cdot L^{-1}$ (Byrtus et al. 2002). Monitoring of the Don and Humber River watersheds in Ontario in 2000-2001 also did not detect imidacloprid, at a detection limit of 1 $\mu g \cdot L^{-1}$ (Struger et al. *in press*). Out of 167 samples collected from approximately 40 different sites in surface waters of Ontario in 2004, again no concentrations of imidacloprid were found, though it should be noted that the detection limit in this study was somewhat high at 4 $\mu g \cdot L^{-1}$ (John Struger, Environment Canada, personal communication, October 2006; Environment Canada 2006). Sampling of surface waters in the Atlantic provinces from 2003 to 2005 did not detect imidacloprid in any of the 82 samples analyzed from PEI (detection limit of 0.2 $\mu g \cdot L^{-1}$), nor any of the 48 samples analyzed from Nova Scotia (Murphy et al. 2006). However, imidacloprid was detected in two out of 57 samples from New Brunswick surface waters, with a maximum concentration of 0.3 $\mu g \cdot L^{-1}$ (Murphy et al. 2006; Environment Canada 2006). Similarly, in a study that looked at imidacloprid in both runoff from potato fields and in surface water of Black Brook, New Brunswick from 2003 to 2005, maximum spike concentrations during rain events were nearing 0.3 $\mu g \cdot L^{-1}$ (Hewitt 2006).

Imidacloprid has more commonly been detected in surface runoff from agricultural fields. For example, runoff collected from potato farms in PEI following rainfall events in 2001 and 2002 had concentrations ranging from below the detection limit of 0.5 $\mu g \cdot L^{-1}$ to 11.9 $\mu g \cdot L^{-1}$ (Denning et al. 2004). Studies in Ontario on tile drains also found low concentrations of imidacloprid in runoff water (PMRA 2001). However, a monitoring effort in Prince Edward Island (45 samples), New Brunswick (42 samples) and Nova Scotia (18 samples), conducted throughout 2003 and 2004, did not detect imidacloprid in any of the runoff water samples, at detection limits of 1.0 to 2.0 $\mu g \cdot L^{-1}$ (Murphy and Mutch 2005).

Only one study that monitored imidacloprid in Canadian groundwater was found. A report from the Ministère de l'Environnement states that imidacloprid (with a detection limit of 0.001 $\mu g \cdot L^{-1}$) and its metabolites (with detection limits of 0.0007 to 0.0009 $\mu g \cdot L^{-1}$) were detected in 35% of groundwater samples collected near potato fields throughout Quebec (Giroux 2003). Samples were collected from shallow wells located close to the treated fields, and therefore represented a worst-case scenario. The maximum concentration of imidacloprid detected was 6.4 $\mu g \cdot L^{-1}$, and maximum concentrations of the metabolites imidacloprid-urea, imidacloprid-guanidine and imidacloprid-olefin were 0.018, 0.4, and 0.0023 $\mu g \cdot L^{-1}$, respectively (Giroux 2003).

Persistence of imidacloprid in soil is affected by various factors, including temperature, organic matter of the soil, and whether the field is cropped or not (Rouchaud et al. 1994; Flores-Cespedes et al. 2002; Krohn and Hellpointer 2002). It is likely that persistence in vegetated areas is decreased through plant (Rouchaud et al. 1994) and microbial (Krohn and Hellpointner 2002) uptake and metabolism. The time required for 50% of the field-applied imidacloprid to dissipate (DT_{50}) can range anywhere from approximately 80 days to 2 years (Mulye 1996; Sabbagh et al. 2002; Krohn and Hellpointer 2002). Assuming typical DT_{50}s of 1 to 2 years, PMRA has classified imidacloprid as persistent in soil based on the classification scheme of Goring et al. (1975).

Adsorption is the main fate process for imidacloprid in soil (Sabbagh et al. 2002). Imidacloprid has a medium to high sorption tendency for soil, with reported soil adsorption coefficients (K_{oc}) ranging from 210 to 262 (Krohn and Hellpointer 2002; Nemeth-Konda et al. 2002; Orme and Kegley 2003). Sorption intensity for imidacloprid and its metabolites is influenced by soil type and depends largely on organic carbon content (Cox et al. 1998). Soil sorption is also influenced by the soil:solution ratio, with lower sorption when the soil contains a higher water content, and it is concentration-dependent, with higher sorption rates when there is a lower initial concentration of imidacloprid present (Cox et al. 1998).

Due to its high water solubility, imidacloprid can leach to depths of at least 105 cm when irrigation conditions are unmatched to water evapotranspiration rates so that the soils become saturated or near-saturated (Felsot et al. 1998). However, there is evidence to suggest that, if used correctly (e.g., at recommended rates, without irrigation, and when heavy rainfall is not predicted), imidacloprid does not characteristically leach into the deeper soil layers

(Rouchaud et al. 1994; Tomlin 2000; Krohn and Hellpointner 2002).

The persistence of imidacloprid in the aqueous environment depends on environmental factors including exposure to light, pH, temperature and microbial community. Photolysis appears to be a major process for degradation. DT_{50} values of 30, 130 and 160 days have been calculated in the absence of light and with variable sediment (Krohn and Hellpointner 2002). Similarly, Spiteller (1993) found that the half-life of imidacloprid under dark conditions was 129 days. Combining metabolic and photolytic processes reduces the DT_{50} values to the range of days (Heimbach and Hendel 2001). The aqueous photolysis half-life for imidacloprid has been determined at approximately 4 hours (Tomlin 2000; Krohn and Hellpointner 2002).

Mesocosm studies suggest that under natural conditions, dissipation times are shorter than those seen in laboratory tests. Moring et al. (1992) determined a half-life for imidacloprid in the water column of 1.4 days in an outdoor microcosm study with four surface applications of the active ingredient, each spaced two weeks apart. Imidacloprid did not appear to persist in the sediment either, with residues below detection limits one month after the last application (Moring et al. 1992). In another mesocosm study, Confidor SL 200 (containing 17.3% imidacloprid) was applied twice, three weeks apart, to artificial ponds at concentrations ranging from 0.6 to 23.5 µg a.i.·L^{-1} (Ratte and Memmert 2003). The calculated mean DT_{50} for imidacloprid in water was 8.2 days. The average DT_{50} for the whole pond system (water and sediment) was 14.8 days (Ratte and Memmert 2003).

The formulation of the imidacloprid product further influences persistence in the aquatic environment. Higher half-life values were found for powder formulations than for liquid (Sarkar et al. 1999). Persistence also increased with increase of application rate (Sarkar et al. 1999). Imidacloprid is generally stable to hydrolysis at environmentally relevant pH, so this is not expected to be a major fate process (U.S. EPA 2005).

The major breakdown products of imidacloprid in water are 6-chloro-3-pyridyl-methylethylendiamine, 6-chloro-nicotinaldehyde, 6-chloro-N-methylnicotinacidamide, 1-[(6-chloro-3-pyridinyl)methyl]-2-imidazolidinone (i.e., imidacloprid urea), and 6-hydroxynicotinic acid. A minor breakdown product is imidacloprid guanidine (Wamhoff and Schneider 1999; Zheng and Liu 1999; Bacey 2000). Under dark, anaerobic conditions, des-nitro imidacloprid is produced. Des-nitro imidacloprid has been found to be more persistent than its parent compound (Fritz and Hellpointner 1991). Both des-nitro imidacloprid and imidacloprid urea are highly water soluble, with solubilities of 180 – 230 g·L^{-1} and 9.3 g·L^{-1} at 20°C, respectively (Krohn 1996a, 1996b).

Imidacloprid has a log K_{ow} of 0.57 (Krohn and Hellpointner 2002; Tomlin 2000) indicating a low potential for accumulation in aquatic species. Imidacloprid does not appear to bioaccumulate in biota (Krohn and Hellpointner 2002; PMRA 2001). The transformation products des-nitro imidacloprid (log K_{ow} < -2 for pH between 4 and 7 and log K_{ow} = -1.7 at pH = 9) and imidacloprid urea (log K_{ow} = 0.46) should also have low bioaccumulation potential (Krohn 1996a,b).

Imidacloprid is a systemic insecticide (Tomlin 2000) meaning that it is taken up by plants, primarily through the roots, and transported within the vascular system of the plant where it can affect plant-feeding pests. Imidacloprid acts as a nicotinic acetylcholine (Ach) agonist (Song and Brown 1998). It binds irreversibly to the nicotinergic receptors in postsynaptic nerves, preventing acetylcholine from binding. Imidacloprid is not degraded by acetylcholinesterase, and therefore this blockage leads to the accumulation of acetylcholine, which ultimately results in paralysis and death (Hovda and Hooser 2002). Imidacloprid has been shown to have a higher binding affinity for insect nerve receptors than for mammalian receptors (Matsuda et al. 2000). Imidacloprid is highly toxic to aquatic insects, as well as some other aquatic invertebrates, but has only low toxicity to fish, algae, amphibians, or mammals (CCME 2007).

Several studies have demonstrated that the transformation products of imidacloprid are considerably less toxic to invertebrates than the parent compound (see Mulye 1997). The toxicity of formulated products relative to technical grade imidacloprid is less clear. Stoughton (2006) compared the toxicities of technical grade imidacloprid and the formulated product Admire® to two freshwater invertebrates, the midge *Chironomus tentans*, and the amphipod *Hyalella azteca*. Different results were observed with the two species. In the case of *H. azteca*, Admire® was considerably more toxic than the technical grade imidacloprid, with 96-h LC_{50}s of 17.44 µg a.i.·L^{-1} for Admire® and 65.43 µg a.i.·L^{-1} for imidacloprid. However, *C. tentans* showed similar responses to the two substances, with 96-h LC_{50}s of 5.40 µg a.i.·L^{-1} for Admire® and 5.75 µg a.i.·L^{-1} for the technical grade imidacloprid. Therefore, it may not be possible to make any general statements on the relative toxicity of

imidacloprid and its formulated products, as this could vary depending on the species.

Water Quality Guideline Derivation

The interim Canadian water quality guidelines for imidacloprid for the protection of freshwater and marine life were developed based on the CCME protocol (CCME 1991). For more information, see the scientific supporting document (CCME 2007).

Toxicity studies that were conducted with formulated products, rather than technical grade imidacloprid, were not considered for use in guideline derivation. Formulants used in pesticide products may augment the toxicity of the active ingredient by making it more bioavailable, or there may be toxicity associated with formulants themselves. Therefore, by not considering toxicity tests with formulated products, it is possible that the guidelines could be underprotective. However, the formulants used will not be the same across all pesticides with the same active ingredient, and potential effects of the formulants themselves may differ among species. For these reasons, it then becomes difficult to make comparisons of toxicity across studies. Therefore, the guidelines are based only on studies with technical grade imidacloprid.

Freshwater Life

Freshwater fish do not appear to be particularly sensitive to imidacloprid, with toxic effects occurring at concentrations that are at least two orders of magnitude higher than imidacloprid concentrations that have been measured in Canadian waters. In a 60-day exposure of rainbow trout (*Oncorhynchus mykiss*), from newly fertilized eggs to juveniles, a LOEC of 2300 µg a.i.·L^{-1} was reported for effects on growth, while no effects on hatching or survival were observed at the highest test concentration of 19,000 µg a.i.·L^{-1} (Cohle and Bucksath 1991; Gagliano 1992). Acute 96-h LC_{50}s that have been reported for fish typically fall in the range of 200,000 to 300,000 µg a.i.·L^{-1} (e.g., Grau 1987; Grau 1988). A 96-h study on juvenile rainbow trout also reported a LOEC for behavioural effects of 64,000 µg a.i.·L^{-1} (Bowman and Bucksath 1990).

There appears to be a wide range in toxicity of imidacloprid to different invertebrate species, with reported short-term LC_{50} values ranging from 3 to >130,000 µg a.i.·L^{-1} (CCME 2007). Insects and ostracods appear to be particularly sensitive, while cladocerans are relatively insensitive. The most sensitive study is a 10-d LOEC of 1.24 µg a.i.·L^{-1}, based on growth of the larval midge *Chironomus tentans* (Gagliano 1991). However, there are some concerns with this study due to low levels of contamination detected in some of the control samples. The second most sensitive study is a 28-d LOEC (EC_{15}) of 2.25 µg a.i.·L^{-1} for reduced emergence of the midge *Chironomus riparius* (Dorgerloh and Sommer 2001). Several other effect concentrations for technical grade imidacloprid (as well as numerous effect concentrations with formulated products) fall within a factor of 10 of 2.25 µg a.i.·L^{-1}, lending support to this sensitive study. For example, Sánchez-Bayo and Goka (2006) reported 48-h EC_{50}s for immobilization in the ostracod species *Ilyocypris dentifera* and *Cypridopsis vidua* both at 3 µg a.i.·L^{-1}. Stoughton (2006) reported a 96-h LC_{50} for *Chironomus tentans* of 5.75 µg a.i.·L^{-1}. Larvae of another insect species, the black fly *Simulium vittatum*, showed similar acute sensitivity, with 48-h LC_{50} values ranging from 6.75 to 9.54 µg a.i.·L^{-1} (Overmyer et al. 2005).

In general, it appears that algae are at least three orders of magnitude less sensitive to imidacloprid than many insect and ostracod species. The most sensitive algae data reported is a 4-d EC_{50} for growth inhibition in the freshwater diatom *Navicula pelliculosa* of 12,370 µg a.i.·L^{-1} (Hall 1996). A 4-d EC_{50} of 32,800 µg a.i.·L^{-1} was reported for decreased growth by the blue-green alga (*Anabaena flos-aquae*) (Bowers 1996). In a 96-h toxicity test with the green alga *Scenedesmus subspicatus*, no effects on growth rate or biomass were observed at the highest test concentration of 10,000 µg a.i.·L^{-1} (Heimbach 1986). Similarly, in a 5-d test with the green alga *Pseudokirchneriella subcapitata* no effects on growth were observed at the highest test concentration of 119,000 µg a.i.·L^{-1} (Gagliano and Bowers 1991).

A small number of mesocosm and field studies have been conducted with imidacloprid. Moring et al. (1992) reported an overall mesocosm "no significant adverse effect concentration" of 6 µg a.i.·L^{-1}. At the next highest concentration of 20 µg a.i.·L^{-1} there were decreases in overall phytoplankton density and densities of Copepoda, mayflies, caddisflies, and the amphipod *Hyalella azteca* (Moring et al. 1992). Ratte and Memmert (2003) reported an overall mesocosm NOEC of 0.6 µg a.i.·L^{-1} and a LOEC of 1.5 µg a.i.·L^{-1}. The results of this study must be treated with caution, however, because they are based on nominal concentrations and a formulated product was used which contained only 17.3% active ingredient. Therefore, it is unknown what effect the other substances in the formulation had.

Studies that have looked at the effect of pulses versus continuous exposure to imidacloprid suggest that less mortality may occur under pulse exposures, but that short-term pulses may still have long-term impacts (Alexander 2006; Stoughton 2006). Alexander (2006) found that adverse effects were observed at the same concentration for either a 12-hour pulse exposure (followed by 19 days exposure to clean control water), or a 20-day continuous exposure to imidacloprid.

The interim water quality guideline for imidacloprid for the protection of freshwater life is 0.23 µg a.i.·L^{-1}. It was derived by multiplying the 28-d LOEC of 2.25 µg a.i.·L^{-1} for the midge (*C. riparius*) (Dorgerloh and Sommer 2001) by a safety factor of 0.1 (CCME 1991).

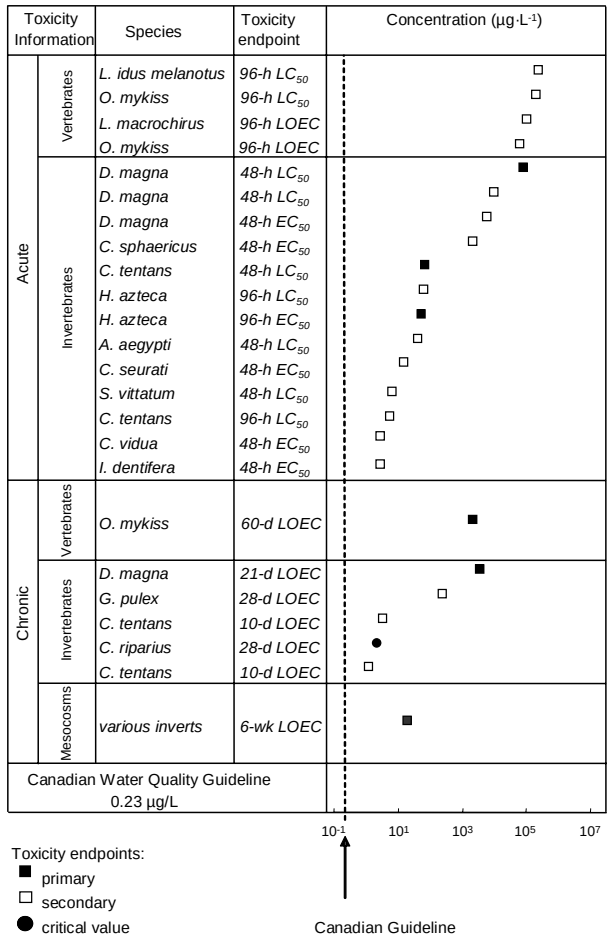

Figure 1. Select freshwater toxicity data for imidacloprid

Marine Life

Only two toxicity studies were available for marine fish. A 7-d LOEC for growth inhibition of 34,000 µg a.i.·L^{-1} was reported for larvae of the marine inland silverside, *Menidia beryllina* (Environment Canada 2005). The 7-d LC_{50} from this same study was 77,500 µg a.i.·L^{-1} (Environment Canada 2005). A study on adult sheepshead minnow, *Cyprinodon variegatus*, reported a 96-hour LC_{50} of 161,000 µg a.i.·L^{-1} (Ward 1990a).

Toxicity data for marine invertebrates were available for three species: the mysid shrimp *Mysidopsis bahia*, the salt marsh mosquito *Aedes taeniorhynchus*, and the brine shrimp *Artemia* sp. The mysid shrimp (*M. bahia*) appears to be very sensitive, with reported 96-h LC_{50} values of 34.1 and 37.7 µg a.i.·L^{-1} (Ward 1990b). For the juvenile salt marsh mosquito (*A. taeniorhynchus*) the 72-h LC_{50} was 21 µg a.i.·L^{-1} (Song and Brown 1998), while the 48-h LC_{50} for the first instar stage was 13 µg a.i.·L^{-1} (Song et al. 1997). Song et al. (1997) reported a 48-h LC_{50} of 361,000 µg a.i.·L^{-1} for adult saltwater brine shrimp (*Artemia* sp.). Juvenile brine shrimp exhibited lower toxicity with approximately 40% of the juveniles dying at 800,000 µg a.i.·L^{-1}, the highest dose administered, after 72 hours (Song and Brown 1998).

No studies were available on the toxicity of imidacloprid to marine algae or plants.

The interim water quality guideline for imidacloprid for the protection of marine life is 0.65 µg a.i.·L^{-1}. It was derived by multiplying the 48-h LC_{50} value of 13 µg a.i.·L^{-1} for *A. taeniorhynchus* (Song et al. 1997) by an acute application factor of 0.05 for nonpersistent substances (CCME 1991).

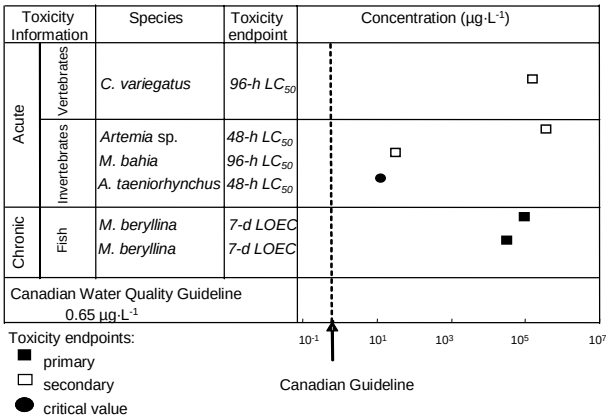

Figure 2. Select marine toxicity data for imidacloprid

References

Alexander, A.C. 2006. Sublethal effects of imidacloprid on mayflies and oligochaetes. M.Sc. thesis. University of New Brunswick, Fredericton.

Bacey, J. 2000. Environmental fate of imidacloprid. Environmental Monitoring and Pest Management Branch. Department of Pesticide Regulation, California. www.cdpr.ca.gov/docs/empm/pubs/fatememo/imid.pdf. Accessed January 2004.

Bowers, L.M. 1996. Toxicity of NTN 33893 2F to the blue-green alga *Anabaena flos-aquae*. Bayer Corporation, Kansas City, Missouri. Report No. 107549. 31 pp. (Reviewed in Mulye 1997)

Bowman, J. and J. Bucksath. 1990. Acute toxicity of NTN 33893 to rainbow trout (*Oncorhynchus mykiss*). Analytical Bio-Chemistry Laboratories, Inc., Columbia, Missouri (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting laboratory). 29 pp. Mobay Project ID: Report No. 100349.

Brimble, S., P. Bacchus, and P.-Y. Caux. 2005. Pesticide utilization in Canada: a compilation of current sales and use data. Environment Canada, Ottawa.

Byrtus, G., A. Anderson, K. Saffran, G. Bruns, and L. Checknita. 2002. Determination of new pesticides in Alberta's surface waters (1999-2000). The Water Research User Group, Alberta Environment. Accessed: September 2004. http://www3.gov.ab.ca/env/water/reports/NewPesticidesInSurfaceWaters_1999_2000.pdf

CCME (Canadian Council of Ministers of the Environment). 1991. A Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life. Canadian Environmental Quality Guidelines, Task Force on Water Quality Guidelines, Winnipeg, Manitoba.

CCME (Canadian Council of Ministers of the Environment). 2007. Canadian water quality guidelines: Imidacloprid. Scientific Supporting Document. Winnipeg, Manitoba.

Cohle, P. and J. Bucksath 1991. Early Life Stage Toxicity of NTN 33893 Technical to Rainbow Trout (*Oncorhynchus mykiss*) in a Flow-Through System. Analytical Bio-Chemistry Laboratories, Inc., Columbia, Missouri (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting company). Report Number 101214. 85 pp.

Cox, L., W.C. Koskinen, and P.Y. Yen. 1998. Influence of soil properties on sorption-desorption of imidacloprid. J. Environ. Sci. Health B33(2): 123-134.

Culp, J.M., K. Liber, A. Cessna, and K. Doe. 2006. Improved ecological risk assessment of imidacloprid in aquatic ecosystems: PSF Final Report – February 2006. Pesticide Science Fund, Environment Canada.

Denning, A., W.R. Ernst, G.R. Julien, K.G. Doe, A. Cook, M. Bernier, P. Jackman, and C. Loiser. 2004. An assessment of buffer zone effectiveness in reducing pesticide runoff from potato fields in Prince Edward Island (2001-2002). Environment Canada, Environmental Protection Branch, Atlantic Region. Surveillance Report EPS-5-AR-04-05.

Dorgerloh, M. and H. Sommer. 2001. Influence of imidacloprid (tech.) on development and emergence of larvae of Chironomus riparius in a water-sediment system. Bayer AG, Crop Protection Development, Institute for Environmental Biology & Institute of Metabolism Research and Residue Analysis, Leverkusen, Germany. 74 pp.

ENKON Environmental Limited. 2001. Survey of Pesticide Use in British Columbia : 1999. Environment Canada; BC Ministry of Environment, Lands and Parks. Surrey, British Columbia.

Environment Canada. 2005. Toxicity testing using imidacloprid: inland silverside test report. Prepared by Stantec Inc. for the National Guidelines and Standards Office, Environment Canada. Ottawa.

Environment Canada. 2006 (Draft). Presence, levels and relative risks of priority pesticides in selected Canadian aquatic ecosystems. Summary of 2003-2005 surveillance results. Prepared by Cantox Environmental for the National Water Quality Monitoring Office, Environment Canada, Ottawa.

EXTOXNET (Extension Toxicology Network). 1998. Imidacloprid – Pesticide Information Profile. University of California-Davis, Oregon State University, Michigan State University, Cornell University, and the University of Idaho: Extension Toxicology Network (EXTOXNET). http://www.extoxnet.orst.edu/. Accessed January 2004.

Felsot, A.S., W. Cone, J. Yu, and J.R. Ruppert. 1998. Distribution of imidacloprid in soil following subsurface drip chemigation. Bull. Environ. Contam. Toxicol. 60: 363-370.

Flores-Cespedes, F., E Gonzalez-Pradas, M. Fernandez-Perez, M. Villafranca-Sanchez, M. Socias-Viciana, and M.D. Urena-Amate. 2002. Effects of dissolved organic carbon on sorption and mobility of imidacloprid in soil. J. Environ. Qual. 31:880-888.

Fritz, R., and E. Hellpointner. 1991. Degradation of pesticides under anaerobic conditions in the water/sediment system: imidacloprid. Miles Report No. 101346. Bayer AG, Leverkusen, Germany. 69 pp. (Reviewed in Mulye 1995)

Gagliano, G.G. 1991. Growth and survival of the midge (*Chironomus tentans*) exposed to NTN 33893 technical under static renewal conditions. Mobay Corporation, Stilwell, Kansas (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting company). 43 pp. Mobay Report No. 101985.

Gagliano, G.G. 1992. Raw data and statistical analysis supplement for early life stage toxicity of NTN 33893 to rainbow trout (Oncorhynchus mykiss). ABC Laboratories, Inc., Columbia, Missouri (performing laboratory). Miles Incorporated, Agriculture Division, Kansas City, Missouri (submitting company). Miles Report No. 101214-1. 295 pp.

Gagliano, G.G., and L.M. Bowers. 1991. Acute toxicity of NTN 33893 to the green alga (*Selenastrum capricornutum*). Mobay Corporation, Stilwell, Kansas (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting company). 30 pp. Mobay Report No. 101986.

Giroux, I. 2003. Contamination de l'eau souterraine par les pesticides et les nitrates dans les régions en culture de pommes de terre. Campagne d'échantillonnage de 1999-2000-2001. Direction de suivi de l'état de l'environnement, Ministère de l'Environnement, Gouvernement du Québec, Québec. www.mddep.gouv.qc.ca/pesticides/pomme_terre/Pesticides_pomme_terre.pdf

Goring, C.A.I., D.A. Laskowski, J.H. Hamaker, and R.W. Meikle. 1975. Principles of pesticide degradation in soil. *In*: Haque, R. and V.H. Freed, eds. *Environmental dynamics of pesticides*. pp 135 – 172. Plenum Press, New York. (Cited in Mulye 1995)

Grau, R. 1987. The acute toxicity of NTN 33893 Techn. to golden orfe (*Leuciscus idus melanotus*) in a static test. Final Report FO-1042, Study No. E 2820089-9. Bayer AG, Leverkusen, Germany. 11+ pp.

Grau, R. 1988. The acute toxicity of NTN 33893 technical to rainbow trout (*Salmo gairdneri*) in a static test. Bayer AG, Leverkusen-Bayerwerk, Germany (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting company). 18 pp. Report No. 101303.

Hall, A.T. 1996. Toxicity of NTN 33893 2F to the freshwater diatom *Navicula pelliculosa*. Bayer Corporation, Kansas City, Missouri. Report No. 107658. 31 pp. (Reviewed in Mulye 1997)

Heimbach, F. 1986. Growth inhibition of green algae (*Scenedesmus subspicatus*) caused by NTN 33893 (technical). Bayer AG, Leverkusen, Germany Report HBF/A1 27. 16 pp.

Heimbach and Hendel. 2001. Unpublished study. Bayer CropScience AG, Monheim am Rhein, Germany. (Cited in Krohn and Hellpointner 2002)

Hewitt, M. 2006. Characterizing potato pesticide impacts in aquatic systems of Atlantic Canada. Project Report submitted under the Pesticide Science Fund, Environment Canada.

Hovda, L.R. and S.B. Hooser. 2002. Toxicology of newer pesticides for use in dogs and cats. The Veterinary Clinics: Small Animal Practice. 32(2):455-467.

Krohn, J. 1989. Water solubility of NTN 33893. Miles report No. 99859. Bayer AG, Wuppertal-Elberfeld, Germany. 10 pp. (Reviewed in Mulye 1995)

Krohn, J. 1996a. Water solubility and partition coefficient of desnitro compound. Bayer Corporation, Kansas City, Missouri. Report No. 107676. (Reviewed in Mulye 1997)

Krohn, J. 1996b. Water solubility and partition coefficient of urea compound. Bayer Corporation, Kansas City, Missouri. Report No. 107677. 15 pp. (Reviewed in Mulye 1997)

Krohn, J. and E. Hellpointner. 2002. Environmental fate of imidacloprid. Pflanzenschutz Nachrichten Bayer. Volume 55, Special Edition. www.bayercropscience.com/bayer/cropscience/cscms.nsf/id/studies/$file/Gaucho_Studien_IV_2_engl.pdf

Matsuda K., M. Shimomura, Y. Kondo, M. Ihara, K. Hashigami, N. Yoshida, V. Raymond, N.P. Morgan, J.C. Freeman, K. Komai, and D.B. Sattelle. 2000. Role of loop D of the α 7 nicotinic acetylcholine receptor in its interaction with the insecticide imidacloprid and related neonicotinoids. Br. J. Pharmacol.130:981-6. (Cited in Hovda and Hooser 2002)

Moring, J.B., J.H. Kennedy, and J. Wiggins. 1992. Assessment of the potential ecological and biological effets of NTN 33893 on aquatic ecosystems as measured in fibreglass pond systems. Report prepared by Miles Inc., Agriculture Division. Bayer CropScience Report No. 102600.

Mulye, H.S. 1995. Environmental evaluation of imidacloprid insecticide and the end-use product Admire 240F. Submission Numbers: 94-1706 and 94-1705. Pesticides Division, Commercial Chemicals Evaluation Branch, Environmental Protection Service, Environment Canada.

Mulye, H.S. 1996. Supplementary review of imidacloprid technical and the end-use product, Admire 240F insecticide. Submission Numbers: 94-1706 and 94-1705. Environmental Evaluation Division, Pest Management Regulatory Agency, Health Canada.

Mulye, H.S. 1997. Supplementary review of imidacloprid technical and the end-use product, Admire 240F insecticide. Submission Numbers: 95-1229 and 96-2186. Environmental Assessment Division, Pest Management Regulatory Agency, Health Canada.

Murphy, C and Mutch, J.. 2005. Multimedia Pesticide Monitoring Programs in Prince Edward Island, New Brunswick and Nova Scotia. Year 1 and Year 2 (2003/04, 04/05) Progress Report, Environment Canada and Prince Edward Department of Environment, Energy and Forestry.

Murphy,C., J.P. Mutch, D. Reeves, T. Clark, S. Lavoie, H. Rees, L. Chow, L-A. Nunn, and D. Hebb. 2006. Multi-media pesticide monitoring programs in Prince Edward Island, New Brunswick and Nova Scotia, Final Project Report of 3-year monitoring program, 2003/04 – 2005/06. Environment Canada, Environmental Protection Branch, Charlottetown.

Nemeth-Konda, L., G. Füleky, G. Morovjan, and P. Csokan. 2002. Sorption behaviour of acetochlor, atrazine, carbendazim, diazinon, imidacloprid and isoproturon on Hungarian agricultural soil. Chemosphere 48: 545-552.

Orme, S. and S. Kegley. 2003. PAN Pesticide Database, Pesticide Action Network. Imidacloprid. www.pesticideinfo.org. Accessed: January 2004.

Overmyer, J.P., B.N. Mason, and K.L. Armbrust. 2005. Aquatic toxicity of imidacloprid and fipronil to a nontarget aquatic insect, *Simulium vittatum* Zetterstedt cytospecies IS-7. Bull. Environ. Contam. Toxicol. 74: 872-879.

PMRA (Pest Management Regulatory Agency). 2005. Electronic Dossier, Delivery, and Evaluation System (EDDE): Electronic Labels: Search and Evaluation (ELSE). http://eddenet.pmra-arla.gc.ca/4.0/4.01.asp

PMRA (Pest Management Regulatory Agency). 2001. Imidacloprid. Regulatory Note. REG2001-11. Ottawa: Health Canada, Pest Management Regulatory Agency. Available at http://www.pmra-arla.gc.ca/english/pdf/reg/reg2001-11-e.pdf

Ratte, H.T., and U. Memmert. 2003. Biological effects and fate of imidacloprid SL 200 in outdoor microcosm ponds. Bayer AG, Crop Protection, Leverkusen, Germany. Report No. 811776.

Rouchaud, J., F. Gustin, and A. Wauters. 1994. Soil Biodegradation and Leaf Transfer of Insecticide Imidacloprid Applied in Seed Dressing in Sugar Beet Crops. Bull. Environ. Contam. Toxicol. 53: 344-350.

Sabbagh, G.J., M.F. Lenz, J.M. Fisher, and E.L. Arthur. 2002. Significance of binding on imidacloprid degradation in soils, and effects of soil characteristics on imidacloprid adsorption capacity. Report No. 200327. Bayer CropScience, Stilwell, Kansas.

Sánchez-Bayo, F., and K. Goka. 2006. Influence of light in acute toxicity bioassays of imidacloprid and zinc pyrithione to zooplankton crustaceans. Aquat. Toxicol. 78: 262-271.

Sarkar, M.A., P.K. Biswas, S. Roy, R.K. Kole, and A. Chowdhury. 1999. Effect of pH and type of formulation on the persistence of imidacloprid in water. Bull. Environ. Contam. Toxicol. 63:604-609.

Song, M.Y., J.D. Stark, and J.J. Brown. 1997. Comparative toxicity of four insecticides, including imidacloprid and tebufenozide, to four aquatic arthropods. Environ. Toxicol. Chem. 16 (12): 2494-2500.

Song, M.Y. and J.J. Brown. 1998. Osmotic effects as a factor modifying insecticide toxicity on *Aedes* and *Artemia*. Ecotoxicol. Environ. Safety. 41: 195-202.

Spiteller, M. 1993. Aerobic metabolism of imidacloprid, ^{14}C-NTN 33893, in an aquatic model ecosystem. Bayer AG, Institute for Metabolism Research, Leverkusen-Bayerwerk, Germany. Report No. PF 3950.

Stoughton, S.J. 2006. Toxicity of imidacloprid to two aquatic invertebrates, *Chironomus tentans* and *Hyalella azteca*, under different exposure conditions. M.Sc. Thesis. University of Saskatchewan, Saskatoon.

Struger, J., T. Fletcher, P. Martos, B. Ripley, and G. Gris. 2002. Pesticide concentrations in the Don and Humber River Watersheds (1998-2000). Environment Canada, Ontario Ministry of the Environment, and City of Toronto. 21 pp.

Struger, J., T. Fletcher, and G. Gris. *In press.* Occurrence of lawn care and agricultural pesticides in the Don and Humber River watersheds (1998-2002). Joint publication of Environment Canada and Ontario Ministry of Environment.

Tomlin, C.D.S. (Ed.). 2000. The Pesticide Manual, 12th Edition. British Crop Protection Council, Surrey, United Kingdom.

U.S. EPA (United States Environmental Protection Agency). 2005. Pesticide Fate Database. Environmental Fate and Effects Division of the Office of Pesticide Programs. Accessed: October 2005 http://cfpub.epa.gov/pfate/home.cfm

Wamhoff, H. and V. Schneider. 1999. Photodegradation of Imidacloprid. Journal of Agriculture, Food and Chemistry. 47(4):1730-1734.

Ward, G.S. 1990a. NTN-33893 technical: acute toxicity to sheepshead minnow, *Cyprinodon variegatus*, under static conditions. Toxicon Environmental Sciences, Jupiter, Florida (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting laboratory). 36 pp. Mobay Project ID: Report No. 100354.

Ward, G.S. 1990b. NTN 33893 technical: acute toxicity to the mysid, *Mysidopsis bahia*, under flow-through test conditions. Toxicon Environmental Sciences, Jupiter, Florida (performing laboratory). Mobay Corporation, Kansas City, Missouri (submitting laboratory). 46 pp. Mobay Report No. 100355.

Zheng, W. and W. Liu. 1999. Kinetics and mechanism of the hydrolysis of imidacloprid. Pesticide Science 55(4): 482-485.

Reference listing:

Canadian Council of Ministers of the Environment. 2007. Canadian water quality guidelines for the protection of aquatic life: Imidacloprid. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

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