GUIDELINES FOR

CANADIAN DRINKING WATER QUALITY

4-CHLORO-2-METHYL-PHENOXYACETIC ACID (MCPA)

Health Canada Santé Canada

Canada

Health Canada is the federal department responsible for helping the people of Canada maintain and improve their health. Health Canada is committed to improving the lives of all of Canada's people and to making this country's population among the healthiest in the world as measured by longevity, lifestyle and effective use of the public health care system.

Également disponible en français sous le titre :
Recommandations pour la qualité de l'eau potable au Canada Acide (4-chloro-2-méthylphénoxy) acétique (MCPA)

To obtain additional information, please contact:

Health Canada
Address Locator 0900C2
Ottawa, ON K1A 0K9
Tel.: 613-957-2991
Toll free: 1-866-225-0709
Fax: 613-941-5366
TTY: 1-800-465-7735
E-mail: publications-publications@hc-sc.gc.ca

Publication date: February 2022

Cat.: H144-13/24-2022E-PDF
ISBN: 978-0-660-42035-6
Pub.: 210635

GUIDELINE VALUE: A maximum acceptable concentration (MAC) of 0.35 mg/L (350 µg/L) is established for 4-chloro-2-methylphenoxyacetic acid (MCPA) in drinking water.

EXECUTIVE SUMMARY

This guideline technical document was prepared in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water and is based on assessments of MCPA completed by Health Canada's Pest Management Regulatory Agency and supporting documents.

Exposure

MCPA is a phenoxyacetic acid herbicide, registered in Canada for use on agricultural sites, fine turf and lawns, in forestry and at industrial sites. It is among the top 10 pesticides sold in Canada, with more than 1,000,000 kg of MCPA active ingredient sold in 2018, and is used across the country, most extensively in the Prairie provinces. Herbicide formulations can use various forms of MCPA, including the free acid, salts and esters, but all release the acid as the active ingredient. Canadians may be exposed to MCPA through its presence in drinking water, air and food. Certain segments of the population may be exposed in occupational settings related to pesticide use and application.

Data provided by provinces and territories that monitor for MCPA indicate that levels of MCPA in drinking water are mostly below detection limits.

Health effects

Some studies have been conducted on the impacts of chlorophenoxy herbicides, including MCPA, on human health. However, because the subjects were exposed to several pesticides, as well as to other organic compounds, these studies cannot be used to assess the toxicity of MCPA in humans. The MAC of 0.35 mg/L (350 µg/L) was derived based on kidney effects observed in rats.

MCPA is considered by international agencies as either unclassifiable with respect to carcinogenicity, or not likely to be carcinogenic in humans, based on a lack of evidence of carcinogenicity in animal studies.

Analytical and treatment considerations

The development of drinking water guidelines takes into consideration the ability to both measure the contaminant and remove it from drinking water supplies. Several analytical methods are available for measuring MCPA in water at concentrations well below the MAC. At the municipal level, activated carbon, membrane filtration, oxidation, advanced oxidation processes and biological filtration achieved a wide range of removal efficiencies. Although MCPA may be removed using oxidation, water utilities should be aware of the potential for formation of degradation by-products. Pilot- and/or bench-scale testing are recommended prior to full-scale implementation.

For MCPA removal at a small system or household level, for example, in the case of drinking water from a private well, a residential drinking water treatment unit may be an option. Although there are no treatment units currently certified for the removal of MCPA from drinking water, technologies that are expected to be effective include adsorption (activated carbon) and reverse osmosis. When using such a treatment unit, it is important to send samples of water entering and leaving the treatment unit to an accredited laboratory for analysis to ensure that adequate MCPA removal is occurring.

Routine operation and maintenance of treatment units, including replacement of the filter components, should be conducted according to manufacturer specifications.

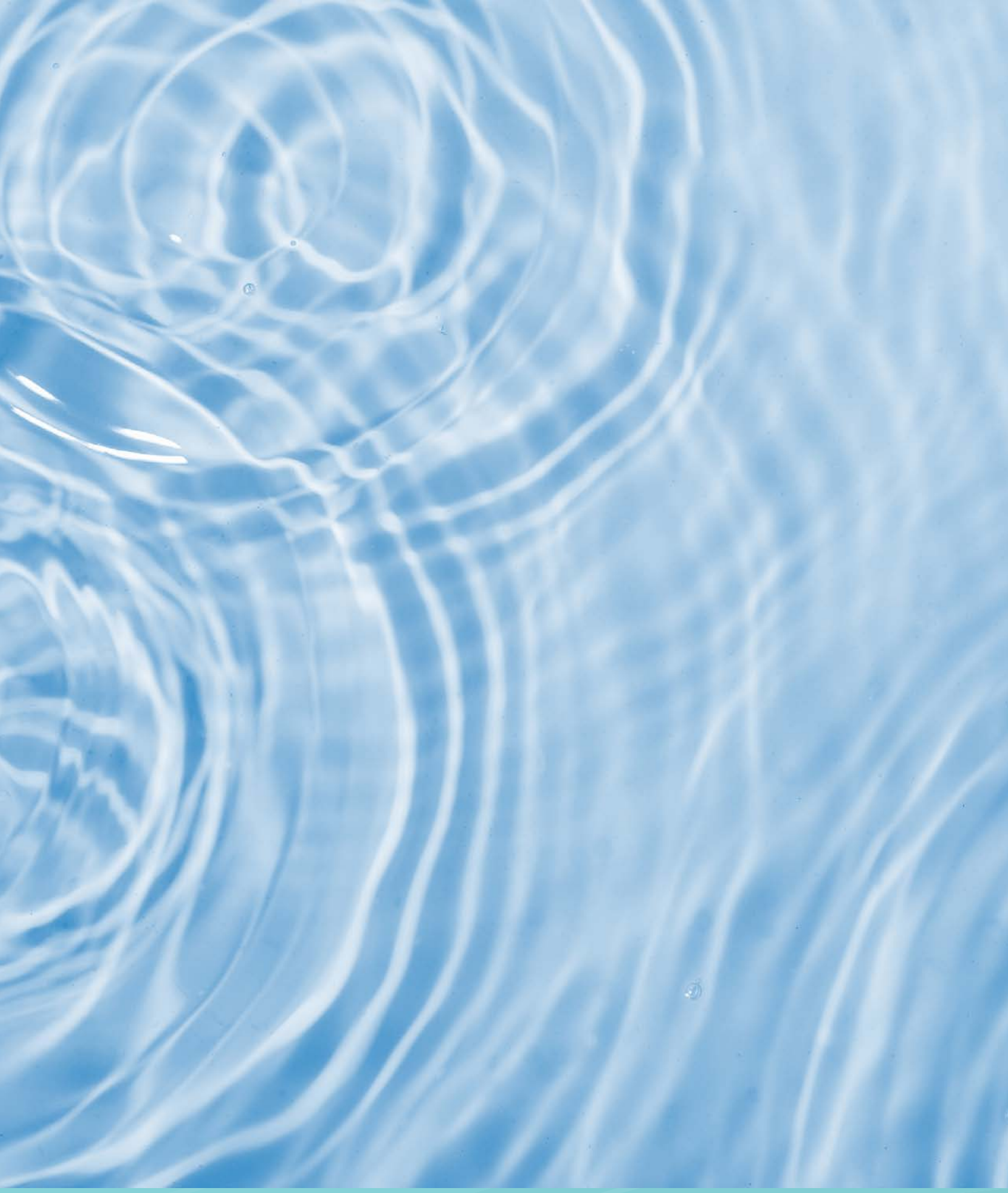
Application of the guidelines

The guidelines are protective against health effects from exposure to MCPA in drinking water over a lifetime. Any exceedance of the MAC should be investigated and followed by the appropriate corrective actions if required. For exceedances in source water where there is no treatment in place, additional monitoring to confirm the exceedance should be conducted. If it is confirmed that source water MCPA concentrations are above the MAC, then an investigation to determine the most appropriate way to reduce exposure to MCPA should be conducted. This may include use of an alternate water supply or installation of treatment. Where treatment is already in place and an exceedance occurs, an investigation should be conducted to verify treatment and determine if adjustments are needed to lower the treated water concentration below the MAC.

Note: Specific guidance related to the implementation of drinking water guidelines should be obtained from the appropriate drinking water authority.

TABLE OF CONTENTS

1.0 EXPOSURE CONSIDERATIONS . . . 1
1.1 Sources and uses . . . 1
1.2 Substance identity . . . 3
1.3 Exposure . . . 4
2.0 HEALTH CONSIDERATIONS . . . 9
2.1 Kinetics . . . 9
2.2 Health effects . . . 12
2.3 Effects in humans . . . 12
2.4 Effects in animals . . . 15
2.5 Genotoxicity and carcinogenicity . . . 19
2.6 Mode of action . . . 19
2.7 Selected key study . . . 20
3.0 DERIVATION OF THE HEALTH-BASED VALUE . . . 21
4.0 ANALYTICAL AND TREATMENT CONSIDERATIONS . . . 23
4.1 Analytical methods to detect MCPA . . . 23
4.2 Treatment considerations . . . 24
4.2.1 Municipal scale . . . 25
4.2.1.1 Conventional treatment . . . 25
4.2.1.2 Activated carbon adsorption . . . 26
4.2.1.3 Membrane filtration . . . 29
4.2.1.4 Oxidation and hydrolysis . . . 32
4.2.1.5 Advanced oxidation processes . . . 35
4.2.1.6 Biological filtration . . . 36
4.2.2 Residential scale . . . 41
5.0 MANAGEMENT STRATEGIES . . . 43
5.1 Monitoring . . . 43
6.0 INTERNATIONAL CONSIDERATIONS . . . 45
7.0 RATIONALE . . . 47
8.0 REFERENCES . . . 48
APPENDIX A: LIST OF ABBREVIATIONS . . . 62
APPENDIX B: CANADIAN WATER QUALITY DATA . . . 65



1.0 EXPOSURE CONSIDERATIONS

1.1 Sources and uses

4-Chloro-2-methylphenoxyacetic acid (MCPA) is a post-emergent chlorophenoxy herbicide used to control broadleaf weeds and woody plants. It is registered for use in Canada on agricultural sites, on fine turf (parks, playgrounds, golf courses, zoos, botanical gardens and athletic playing fields), lawns (public and commercial buildings) and sod (grown in sod farms harvested for transplanting), as well as in forestry (spruce seedlings for reforestation) and at industrial sites (vegetation control) (Health Canada, 2005). MCPA is among the top 10 active ingredients sold in Canada for agricultural uses; in 2018, more than 1,000,000 kg of MCPA active ingredient was sold (Health Canada, 2018).

MCPA acts as a plant growth regulator and is used to control post-emergent broadleaf weeds in agriculture and in urban, forestry and aquatic environments (IARC, 1983; Weed Science Society of America, 1989; HSDB, 2010). Pesticides like MCPA control weeds once they have emerged through absorption by the leaves and roots and distribution throughout the plant. By stimulating nucleic acid and protein synthesis, MCPA affects enzyme activities, respiration and cell division. As a result, treated plants exhibit malformed leaves, stems and roots (US EPA, 1990).

Environmental fate data indicate that all forms of MCPA will revert to the acid, thus the physicochemical characteristics of different forms of MCPA in the environment will be those associated with the acid (US EPA, 2004a).

MCPA has been detected in lakes, rivers and reservoirs (dugouts), which may serve as sources for drinking water. Migration from soil to water is due to direct or indirect transport mechanisms, including non-target drift from aerial or ground boom spraying (vaporization in air), deposition in rain, erosion of soil particles by wind or water, surface runoff and leaching. MCPA may also be found in water as a result of spills, deliberate dumping of tank residues or equipment washing operations (Caux et al., 1995; Murray et al., 2004). Based on its vapour pressure and its Henry's law constant, MCPA is not expected to volatilize from water (see Table 1). MCPA 2-ethylhexyl ester (MCPA-EHE), however, is expected to volatilize from water (Health Canada, 2007). In water, biological degradation (under aerobic conditions) is an important process affecting MCPA's environmental fate (Soderquist and Crosby, 1975; Sattar and Paasivirta, 1980; Smith

and Hayden, 1981; Health Canada, 2006). However, in anaerobic aquatic systems (sediment/water), the biodegradation of MCPA is negligible (Health Canada, 2006). Hydrolysis and photodegradation are not important routes in the degradation of MCPA in water (Health Canada, 2006). During periods of cold weather and low light, the degradation of MCPA via biological degradation or photolysis is limited (Byrtus et al., 2004). MCPA has not been shown to bind significantly to sediments (Caux et al., 1995). The derivatives of MCPA have been shown to dissociate in water to MCPA acid.

MCPA is not persistent in soil (US EPA, 2004a), with a half-life varying between 15 and 50 days (Soderquist and Crosby, 1975; Sattar and Paasivirta, 1980). The rate of degradation depends upon several factors, such as soil type, soil pH, soil moisture, concentration of MCPA, climatic conditions and organic matter content (Sattar and Paasivirta, 1980). 2-Methyl-4-chlorophenol has been identified as the main soil degradation product (University of Hertfordshire, 2019). Degradation of MCPA occurred within 5 to 9 weeks in acidic soil compared with 1 week in neutral soil (pH 6.3 and above) (Sattar and Paasivirta, 1980). Microbial degradation is the most important transformation process for MCPA in soil (Caux et al., 1995) with the presence of both oxygen and proper moisture being important (Sattar and Paasivirta, 1980). In the absence of oxygen, the biotransformation of MCPA in soil is negligible (Health Canada, 2006). Photodecomposition and hydrolysis in soil are not important degradation processes for MCPA (US EPA, 2004a, 2004b; Health Canada, 2006).

Field studies indicate that MCPA does not leach appreciably below soil depths of 15 cm (Health Canada, 2007). Mobility appears to be related to the soil's organic matter content, increasing as the organic matter content decreases (WHO, 2003). Contamination of surface water may occur through spray drift and runoff, whereas groundwater may become contaminated via leaching (US EPA, 2004a). The mobility and leaching of the non-acid forms of MCPA (i.e., amine and sodium salts, esters) have not been determined. Field studies with MCPA-EHE have shown that, under normal conditions, a large proportion converts to MCPA acid on the day of application, with near complete conversion by day 3 (US EPA, 2004a). Under dry conditions, MCPA-EHE was found to persist for days, with greater than 90% present after 48 hours (Smith and Hayden, 1980).

Little information is available on the atmospheric fate of MCPA. Waite et al. (2005) demonstrated, by high-volume air sampling at various heights above ground level in the Canadian Prairies, that atmospheric concentrations of MCPA are strongly influenced by regional atmospheric transport and that its primary transport mechanism is via adsorption to solid particles in the atmosphere. The half-life of MCPA due to photo-oxidation has been estimated to be 2.2 days (Caux et al., 1995).

1.2 Substance identity

MCPA is a chlorophenoxy herbicide that is available in various formulations including the free acid; a dimethylamine salt (MCPA-DMAS); a sodium salt; and an ester (MCPA-EHE) (US EPA, 2004b, 2004d). Although MCPA may be applied in various forms, a single common functional group (the parent acid) is the active portion of the herbicide formulation (US EPA, 2004c, 2004e; Health Canada, 2006). It has been reported that some phenoxy herbicides, such as 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), can be contaminated by dioxins and furans during production, however, MCPA has not been shown to be contaminated with dioxin (Wiklund et al., 1988; Eriksson et al., 1990; Mannetje et al., 2005).

The physicochemical properties of MCPA that are relevant to its presence in drinking water are summarized in Table 1.

Table 1. Physicochemical properties[a] of MCPA relevant to its presence in drinking water

Property	MCPA	Interpretation
CAS RN	94-74-6	-
Molecular weight (g/mol)	200.6	-
Water solubility (g/L)[b]	26.2 at pH 5; 293 at pH 7; 320 at pH 9	Varying solubility
Vapour pressure (volatility)	8.18×10^{-5} to 1.36×10^{-4} Pa at 20°C	Low potential to volatilize
Henry's Law constant	7.46×10^{-5} Pa·m^3/mol	Not expected to volatilize from water or moist surfaces
Octanol: water partition coefficient (Log Kow)	1.43–2.82	Low potential to bioaccumulate

[a] From Worthing and Hance (1991); USDA (2001); Struger et al. (2004); US EPA (2004a, 2004d); Health Canada (2006; 2007).

[b] Solubility as reported in Health Canada (2006). Note that other literature sources report "practically insoluble"; these sources do not provide the pH at which the values were measured. Given that solubility changes with pH, these values are not reported in the table.

1.3 Exposure

Canadians may be exposed to MCPA through its presence in drinking water, air and food. In addition, certain segments of the population may be exposed in occupational settings related to pesticide use and application. Media-specific exposure data are available for water (surface and drinking), air and food.

Canadian water monitoring data was obtained from the provinces and territories (municipal and non-municipal supplies), the open literature and Environment Canada (Environment Canada, 2011) (Appendix B).

The exposure data provided reflect different method detection limits (MDL) of accredited laboratories used within and amongst the jurisdictions, as well as their respective monitoring programs. The data provided by the provinces and territories do not indicate the timing of monitoring in relation to the pesticides application and runoff events. As a result, the exposure data and its statistical analysis provide only a limited picture. Data provided by the provinces and territories indicate that MCPA levels are below the method reporting limit (MRL) or MDL in most samples collected. These collected samples were from a variety of water supplies in Canada including surface water and groundwater, as well as treated and distributed water where monitoring occurred (British Columbia Ministry of Health, 2019; Ontario Ministry of the Environment, Conservation and Parks, 2020; Indigenous Services Canada, 2019; Manitoba Sustainable Development, 2019; Ministère de l'Environnement et de la Lutte contre les changements climatiques, 2019; Nova Scotia Environment, 2019; Saskatchewan Water Security Agency, 2019; PEI Department of Communities, Land and Environment, 2019). Table 2 summarizes the monitoring data for jurisdictions in which all samples were below the MDL. Table 3 summarizes the data for jurisdictions in which MCPA detections were reported. No monitoring data were available for New Brunswick, Newfoundland and Labrador, or Yukon (New Brunswick Department of Environment and Local Government, 2019; Newfoundland and Labrador Municipal Affairs and Environment, 2019; Yukon Environmental Health Services, 2019).

Table 2. Summary of non-detect monitoring data for MCPA

Jurisdiction (MDL µg/L)	Monitoring Period	Type of Water System	Water Type (Municipal: ground/surface—raw, treated, distributed)	# Detects/samples
British Columbia (2)	2015–2017	Municipal	Surface—raw	0/9
FNIHB Ontario Region (0.12–0.2)	2004–2018	Public Water Systems	Ground—raw	0/1
			Ground—treated	0/35
			Ground—distribution	0/1
			Surface—treated	0/65
			Surface—distribution	0/4
		Semi-Public Water Systems	Ground—raw	0/1
			Ground—treated	0/1
			Ground—distribution	0/14
			Surface—treated	0/2
			Surface—distribution	0/1
		Private Water Systems	Surface—treated	0/1
FNIHB Atlantic Region (2.0)	2004–2018	Public Water Systems	Ground—treated	0/3
			Ground—distribution	0/4
			Surface—treated	0/1
FNIHB Quebec Region (0.05–0.5)	2004–2018	Not available	Community well	0/13
			Private well	0/24
			System, drinking water	0/32
Nova Scotia (0.5–5.0)	2012–2018	Municipal	Ground—raw	0/53
			Ground—treated	0/24
			Surface—raw	0/19
			Surface—treated	0/22
Saskatchewan (0.0001–1.0)	2014–2019	Municipal	Ground—raw (municipal)	0/9
			Ground and surface—treated (municipal)	0/4
			Ground & surface—distributed (municipal)	0/29
Prince Edward Island (0.5–1.0)	2004–2017	Municipal	Ground—raw	0/362

FNIHB—First Nations and Inuit Health Branch; MDL—method detection limit.

Table 3. Summary of MCPA detections in select provinces in Canada

Jurisdiction (MDL µg/L)	Monitoring Period	Water Type (Municipal: ground/surface—raw, treated, distributed and Non-Municipal: ground/not specified)	# Detects/ samples	Max Value (µg/L)
Ontario (0.0015–0.01)	2016–2020	Surface—treated (municipal)	8/1698	69
		Ground—treated (municipal)	6/1728	401
		Distribution (municipal)	0/51	-
Quebec (0.1–3.0)	2012–2018	Ground—distribution (municipal)	1/322	2.4
		Surface—distribution (municipal)	0/1005	-
		Ground—raw[a] (municipal)	0/46	-
		Ground—treated[a] (municipal)	0/17	-
		Ground—distribution[a] (municipal)	0/5	-
		Ground—raw[b] (municipal)	0/83	-
		Ground—raw[b] (non-municipal)	0/19	-
Manitoba (0.025)	2012–2018	Surface—ambient	75/428	1.1

[a] Potato Project 2017–2018: During the period covered, analysis results for MCPA pesticide found in raw, treated or distributed groundwater were obtained by the Department from 9 drinking water supplies.

[b] Small Systems Project 2012–2018: During the period covered, analysis results for MCPA found in raw groundwater were obtained by the Department from 25 drinking water supplies.

MDL—method detection limit

Additional Canadian water monitoring data were also obtained from the literature. In British Columbia, MCPA was detected in 1/13 samples of runoff in the Lower Fraser Valley in 2003, with a maximum reported concentration of 110 ng/L (Tuominen et al., 2005).

In Alberta, groundwater samples were collected over a three-year sampling period (2013–2015) from regions with dryland and irrigated agriculture to examine the occurrence of pesticide mixtures including MCPA (Munira et al., 2018). MCPA was most frequently detected (n = 436 samples) during the summer (85% of detections) with less frequent detections in the spring and fall (15%). Levels of MCPA ranged from 26 to 1,293 ng/L in southern Alberta and 32 to 42 ng/L in central Alberta. In Alberta, aside from one detection at 1,293 ng/L, MCPA was detected at levels ≤ 342 ng/L.

In a study by Environment and Climate Change Canada (2015), monitoring of 47 pesticides in three rivers in the Prairies from 2006–2011 revealed that MCPA was one of the top three detected pesticides. MCPA was detected in 39/74 samples (53%) from the Carrot River at a median level of 1.5 ng/L; in 57/74 samples (77%) from the Assiniboine River at a median level of 10.45 ng/L; and in 53/77 samples (69%) from the Red River at a median level of 5.07 ng/L.

A study of 15 surface drinking water reservoirs (fed primarily from cropland snowmelt and occasional rainfall runoff) in Alberta, Manitoba and Saskatchewan detected MCPA in 99% of 206 reservoir samples taken between 2003 and 2004, with mean and maximum concentrations of 57 ng/L and 374 ng/L, respectively (detection limit of 0.58 ng/L) (Donald et al., 2007). The authors observed that MCPA was consistently present in water samples from the 15 water reservoirs. MCPA was detected in higher mean concentrations in July samples (89.1 ± 13.8 ng/L) after herbicide application (May to early July) as compared to April/May samples (36.5 ± 8.7 ng/L) after the snowmelt runoff. In central Saskatchewan, Donald et al. (2018) carried out monitoring for 29 herbicides, including MCPA, for five years in 16 wetlands on minimum-tillage farms and in 7 wetlands on organic farms. MCPA was detected at a frequency of > 50% from the wetlands on both minimum-tillage and organic farms. MCPA was detected in all 96 samples, with a maximum concentration of 5,980 ng/L for the minimum-tillage farms and 257 ng/L for the organic farms. The mean concentrations were 276 ng/L (70 samples) for the 16 wetlands on the minimum-tillage farms and 119 ng/L (26 samples) for the 7 wetlands on the organic farms.

A 2003 spring/summer pesticide surveillance study reported MCPA detections in 15 small dugouts sampled in the three Prairie provinces (Murray et al., 2004). Mean MCPA concentrations ranged from 13 to 108 ng/L in most of the dugouts, with two reservoirs having mean levels ranging from 200 to 320 ng/L, possibly due to commercial formulations (which include MCPA) being applied in the watershed or catchment.

In Ontario, the Drinking Water Surveillance Program (DWSP) monitors water quality at selected municipal drinking water systems for scientific and research purposes. The DWSP monitors for inorganic, organic and radiological parameters, including pesticides. Results for MCPA in raw (n = 273), treated (n = 218) and distribution system (n = 1) water samples during the 2010 to 2012 period of revealed no detections above the MDL of 0.05 µg/L.

In Quebec, MCPA was detected in 12.6% (average) of samples of surface water taken from four rivers in the corn and soybean growing areas of the province (Chibouet, des Hurons, Saint-Régis and Saint-Zéphirin rivers) between 2015 and 2017 (maximum concentration 0.97 µg/L) (Giroux, 2019). During the same period, Giroux (2019) reported MCPA detection frequencies (without concentrations) in other Quebec river systems ranging from 10% to 11.1% in two rivers of the Montérégie area; 36.4% to 54.5% in seven rivers of the Chaudières-Appalaches region; and 9.1% to 18.2% in six rivers of the Saguenay-Lac-Saint-Jean region.

In the Maritime provinces, MCPA was not detected in any of the 60 surface water samples taken in New Brunswick (2003 and 2004), Nova Scotia (2004) or Prince Edward Island (2003 and 2004) based on a detection limit of 1 µg/L (Murphy and Mutch, 2005). MCPA was not detected in the rivers of eight different municipalities with agricultural or urban activities in New Brunswick in 2004 (New Brunswick Department of Health, 2005).

In Saskatchewan, air samples collected in 2003 from May 12 to August 13 at Bratt's Lake, Hafford, and Waskesiu revealed a 94% detection frequency for MCPA (Yao et al., 2006). Average air concentrations (one metre above ground) for the three study areas were 513 pg/m^3 (Bratt's Lake), 82 pg/m^3 (Hafford) and 32.8 pg/m^3 (Waskesiu). Patterns of atmospheric occurrence reflected local pesticide application, volatilization from soil and atmospheric transport.

Messing et al. (2014) investigated air concentrations of pesticides during the summers of 2005 and 2007 for 90 days at four locations in the agricultural region of the Canadian Prairies (Lethbridge, Swift Current, Indian Head and Brandon) and at five locations in the Canadian Subarctic and Arctic (Nahanni National Park, Fort Simpson, Yellowknife, Arviat, Coral Harbour). MCPA was detected at all Prairie locations (ranging from 0.05 to 0.47 µg/sample) in both years, which is consistent with its use in agricultural production systems. MCPA was also detected at Arviat in 2007 at a concentration of 0.01 µg/sample.

Health Canada's Canadian Food Inspection Agency tested domestic and imported food products (i.e., fresh fruit and vegetables, and processed fruit and vegetable products) for MCPA between April 1, 2015 and March 31, 2016. MCPA was detected in 1/1 samples (0.0015 ppm) in domestic frozen Saskatoon berries and in 1/4 samples (0.0006 ppm) of imported blueberries (Health Canada, 2015/2016).

2.0 HEALTH CONSIDERATIONS

All pesticides, including MCPA, are regulated by the Pest Management Regulatory Agency (PMRA). PMRA conducts extensive evaluations and cyclical reviews of pesticides, including unpublished and proprietary information, as well as foreign reviews by other regulatory agencies such as the United States Environmental Protection Agency (US EPA). As such, this health assessment is primarily based on PMRA evaluations (Health Canada, 2006, 2007) and supporting documentation. Additionally, any reviews and relevant literature available since the completion of the PMRA evaluations were also considered.

2.1 Kinetics

Chronic and subchronic studies have shown that dogs are more sensitive than rats or mice to the effects of MCPA and related compounds, with effects seen at levels at least 10 times lower in dogs than in rats or mice. Allometric scaling of data from rats, dogs and humans indicates that the renal clearance of MCPA in dogs is approximately 10 times slower than in humans (Timchalk, 2004). The unique sensitivity of dogs to MCPA-mediated effects reported in the literature, therefore, may be attributed to the reduced renal clearance of organic acids (e.g., MCPA) leading to higher concentrations in blood compared in dogs relative to humans and other species. This evidence suggests that the dog is not an appropriate indicator species for MCPA-mediated toxicity in humans (Timchalk, 2004).

Absorption: MCPA is readily absorbed from the gastrointestinal tract (via gavage and following direct gastric intubation) in rats, dogs and humans (Elo, 1976; Fjeldstad and Wannag, 1977; Kolmodin-Hedman et al., 1983a, 1983b; Jahanshahi and Stow, 1995; Hardwick, 1999, 2000; Lappin et al., 2002). Oral administration of single MCPA doses of 5 or 100 mg/kg body weight (bw) resulted in peak plasma concentrations within 2 to 4 hours of dosing in rats and 4.5 to 7 hours in beagles (Lappin et al., 2002). Similar peak plasma concentrations were attained within 2 to 3 hours of dosing in rats with a single dose of 5 mg/kg bw of either MCPA-DMAS or MCPA-EHE (van Ravenzwaay et al., 2004). Human volunteers given an MCPA dose of 0.015 mg/kg bw per day exhibited a peak plasma concentration after 1 hour (Kolmodin-Hedman et al., 1983a). A comparison review of these metabolism studies showed that the plasma half-life in dogs (63 hours) was considerably longer than in rats (6 hours) and humans (11 hours) following a dose of 5 mg/kg bw of MCPA (Timchalk, 2004).

Distribution: In rats, MCPA is widely distributed to various tissues and organs (Elo, 1976); however, no significant accumulation of MCPA is observed in tissues (Jahanshahi and Stow, 1995; van Ravenzwaay et al., 2004).

Rats administered a single dose of 11.5 mg/kg bw [^{14}C]MCPA by direct gastric intubation showed peak tissue concentrations between 2 and 8 hours after dosing, followed by a rapid decline (Elo, 1976). Blood, kidney, suprarenal gland, lung, heart, liver, thyroid gland and bone marrow showed the highest concentrations.

In rats administered [^{14}C]MCPA in a single oral dose of 5 or 100 mg/kg bw, the radioactivity in the tissues and carcasses accounted for ≤ 2.3% of the dose at sacrifice (Jahanshahi and Stow, 1995; van Ravenzwaay et al., 2004). In the low dose rats (sacrificed on day 4), MCPA was non-detectable in most tissues, except in fat, skin and kidneys. In the high dose rats, (sacrificed on day 7), radioactivity was highest in fat, skin and kidneys, and higher concentrations were detected in more organs than for the lower dose. Additionally, females had higher levels of radioactivity than males, and small amounts of residual radioactivity in the kidneys were consistent with continued excretion of the compound (van Ravenzwaay et al., 2004).

Metabolism: In a fatal intoxication case involving a 23-year-old male, the forensic autopsy found 4-chloro-2-methylphenol in the body fluids and organ tissues, suggesting a metabolite of MCPA (Takayasu et al., 2008). In a study where five healthy human volunteers were given 15 µg/kg bw of MCPA, further investigation of free or conjugated MCPA in one individual showed that conjugation varied between 56% and 73% within the 72-hour collection period (Kolmodin-Hedman et al., 1983b).

The toxicokinetics and metabolism of MCPA-DMAS and MCPA-EHE are indistinguishable from those of MCPA (van Ravenzwaay et al., 2004). MCPA, MCPA-DMAS and MCPA-EHE were not extensively metabolized in rats after oral administration. Lappin et al. (2002) reported that rats orally exposed to 5 and 10 mg/kg/day of MCPA excreted the parent compound mainly in their urine (approximately 65% of the dose). The only significant metabolite in rat urine was hydroxymethylphenoxyacetic acid (HMCPA). A trace of the glycine conjugate of MCPA was also detected, albeit in an amount too small to reliably quantify. Dog urine contained a smaller proportion of MCPA (2%–29%) than rat urine; however, the glycine conjugate of MCPA was detected at much higher levels (up to 37% of the dose) and the taurine conjugate, which was not detected in rat urine, was also detected (up to 7% of the dose).

Elimination: In human volunteers given MCPA at 0.015 mg/kg bw, an average of 40% of the given dose was excreted in the urine during the first 24 hours (Kolmodin-Hedman et al., 1983b). In another study, volunteers given 5 mg of MCPA excreted 50% in the urine within 48 hours and 55% within 96 hours; by the fifth day, the concentration in the urine was below the detection limit (Fjeldstad and Wannag, 1977). No attempt to analyze metabolites was made in either human study, nor were the doses radiolabelled using [^{14}C]MCPA. Renal excretion is reported to be the major route of MCPA elimination in rats and dogs dosed orally with MCPA (Elo, 1976; Jahanshahi and Stow, 1995; Lappin et al., 2002; van Ravenzwaay et al., 2004). Dogs and rats have different recovery patterns. Studies showed that renal clearance in dogs was slower and less extensive than in rats (Lappin et al., 2002) or humans (Fjeldstad and Wannag, 1977; Kolmodin-Hedman et al., 1983a, 1983b). In rats, 75%–80% of the administered dose was excreted in the urine over 24 hours, irrespective of the dose. In dogs, after oral administration of MCPA in single doses of 5 or 100 mg/kg bw, elimination was 44%–73% and 26%–38%, respectively, over 120 hours (Lappin et al., 2002). These data suggest that humans fall between dogs and rats with respect to the ability to eliminate MCPA.

In rats, the parent compound (MCPA) was the major compound excreted in the urine, along with lower levels of an oxidation product (HMCPA) and trace amounts of the glycine conjugate (Lappin et al., 2002). In dogs, the parent compound was also present in the urine, but at lower levels than in the rat. In addition, three metabolites were recovered in the urine: the glycine conjugate, at higher levels than in the rat, and, to a lesser extent, HMCPA and a taurine conjugate (Lappin et al., 2002).

Fecal elimination was a minor route for both species; however, dogs had a higher proportion of MCPA in the feces compared with rats. Radioactivity was not detected in the expired air of rats orally dosed with [^{14}C]MCPA at 5 mg/kg bw (van Ravenzwaay et al., 2004).

In a comparison review of metabolism studies in rats, dogs and humans, Timchalk (2004) showed that dogs had a longer plasma half-life and slower elimination than rats and humans, which resulted in a substantially higher body burden of MCPA, at comparable doses, than in other species. In previous studies with organic acids that had similar pharmacokinetic properties, the dog had a more limited capacity than other species to excrete organic acids via the kidney. The authors suggested that saturation of renal secretion and increased renal tubule reabsorption may be responsible for this lower renal clearance. These differences in the pharmacokinetics of MCPA and other related organic acids between dogs and other species suggest that the use of dog toxicity data for determining human health risks from MCPA exposure may not be appropriate and thus the rat represents the best animal model for the human health risk assessment. For this reason, the PMRA chose not to use any of the dog data in their health risk assessment.

2.2 Health effects

The few existing epidemiological studies on the effects of MCPA indicate that the evidence for carcinogenicity and reproductive effects remains inconclusive. In animals, sub-chronic and chronic studies report systemic, kidney, liver, testicular, reproductive/developmental and nervous system effects, with the kidney identified as the most sensitive target organ. There was no evidence of carcinogenicity in the long-term studies and tests for genotoxicity and mutagenicity were largely negative.

2.3 Effects in humans

Acute effects: Symptoms of acute exposure to large doses of MCPA from poisoning include fatigue, weakness, anoxia, nausea, vomiting, diarrhea, lowering of blood pressure, body temperature disturbance, progressive hypotension, ataxia, neuromuscular irritability and convulsion (Popham and Davis, 1964; Johnson and Koumides, 1965; Jones et al., 1967; Palva et al., 1975; Bovey, 1980; Timonen and Palva, 1980; US EPA, 1984; Bradberry et al., 2000; Roberts et al., 2005). Poisoning events involving co-exposure to MCPA/bromoxynil have also been reported in the literature. Ingestion of large doses of MCPA (200 g/L) and bromoxynil (200 g/L) together has resulted in hyperthermia, tachycardia, tachypnea and metabolic acidosis (Chiew et al., 2018). Ingestion of a MCPA/bromoxynil co-formulation herbicide resulted in increased CO2 production, hyperthermia and metabolic disruption 18 hours after ingestion with death occurring at 20 hours post ingestion. Blood levels of MCPA and bromoxynil were reported as 83.9 µg/L and 137 µg/L, respectively (Berling et al., 2015).

Cancer: Epidemiological studies on phenoxy herbicides in Canada, the United States, Australia, New Zealand and several European countries largely involve multiple exposures to various chlorophenoxy herbicides (including 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-T), as well as other pesticides, raw materials, intermediates and processing chemicals. Cases of soft tissue sarcoma, non-Hodgkin's lymphoma (NHL) and Hodgkin's disease have been associated with phenoxy herbicides, including those contaminated with dioxin (which may increase the risk of certain cancers). However, the results have not been consistent (Mannetje et al., 2005). Some epidemiological studies have included MCPA among the herbicides examined (Hardell and Sandström, 1979; Eriksson et al., 1981; Hardell et al., 1981; Coggon et al., 1986; Vineis et al., 1986, 1991; Wiklund et al., 1987, 1988, 1989; Eriksson et al., 1990; Saracci et al., 1991; Bueno de Mesquito et al., 1993; Lynge, 1993; Kogevinas et al., 1995; Becher et al., 1996; Lynge, 1998; Hardell and Eriksson, 1999; Eriksson et al., 2008); however, very few of these studies reported outcomes specific to MCPA.

The Cross-Canada Study of Pesticides and Health (CCSPH), a population-based, case-control study of Canadian men aged 19 years and older in six provinces (Quebec, Ontario, Manitoba, Saskatchewan, Alberta, and British Columbia), evaluated cancer risks from exposure to several different pesticides with the potential for effect modification from asthma, allergies, hay fever, or asthma with allergies and hay fever combined (Pahwa et al., 2012). The CCSPH included 2,019 subjects with 513 incident NHL cases diagnosed between September 1, 1991 and December 31, 1994 and 1,506 population controls randomly selected from these same provinces. The risk of NHL associated with MCPA exposure was found to be elevated for individuals with asthma, allergies or hay fever compared to those without any of these conditions. An increase in risk was also observed for the use of MCPA by asthmatics compared to non-asthmatics; and those with allergies had higher risks of NHL for use of MCPA than those without allergies. The authors stated that the results could represent new leads or chance occurrences and recommended that further research be undertaken.

The limitations of the above studies, including small sample size, co-exposure to other pesticides, possible recall bias, measurement error, and a lack of detailed information on dose used and amount of time spent applying pesticides, make it difficult to interpret the results from these studies. More definitive studies with accurate assessment of MCPA-specific exposures are needed.

In a comprehensive review of 43 scientific publications (from 13 cohort and 27 case-control studies) exploring the association between exposure to phenoxy herbicides and soft tissue sarcoma (STS) and NHL, Jayakody et al. (2015) concluded that overall, the epidemiological evidence does not clearly support that phenoxy herbicides (including MCPA) cause STS or NHL. The authors focused mainly on associations with exposures to phenoxy herbicides as a group since the available studies involved different combinations/ exposure levels of phenoxy herbicides (without risk adjustments for co-exposures) and assumed that risks between phenoxy compounds were similar. Their analysis indicates that study findings are not entirely consistent, and if a hazard exists, the absolute increased risk of STS/NHL is likely small.

Developmental and reproductive toxicity: Few epidemiological studies have explored the reproductive and developmental outcomes of MCPA exposure. Arbuckle et al. (1999) explored the effect of phenoxy herbicide exposure on the risk of spontaneous abortion in an Ontario farm population involving 2,110 women and over 3,936 pregnancies, including 395 spontaneous abortions. Exposure during pre-conception and post-conception exposure windows was examined separately, with differentiation between early (< 12 weeks) and late (12–19 weeks) spontaneous abortions for each exposure window. Restricting the analysis to

MCPA exposure showed no elevated risk of spontaneous abortion from pre-conception exposure for all gestational ages; however, when looking at early and late spontaneous abortions separately, the risk for early abortions increased, whereas the risk for late abortions decreased. Additional analysis suggested that if pre-conception exposure to MCPA occurred for one month or more, the risk for early abortions further increased but the risk for late abortions was low. According to the authors, the results suggested a possible pre-conceptual effect with MCPA (and phenoxy herbicides) in adverse pregnancy outcomes, particularly for early spontaneous abortions (which may suggest a male-mediated effect due to the higher risk seen from not using protective equipment or clothing). Study limitations include small sample size, use of memory recall and the lack of detailed information on dose used and amount of time spent applying pesticides. Therefore, further study is needed to confirm these findings.

Haraux et al. (2018) explored the association between prenatal exposure to herbicides (assessed via meconium analysis) and the occurrence of isolated hypospadias (penile malformation). MCPA was detected in meconium samples from 28% of newborns in the isolated hypospadias (IH) group compared to 10.3% in the control group, and was significantly associated with IH ($p = 0.04$). The maximum detected level of MCPA was 284.4 ng/g of meconium. This case-control study (conducted between 2011 and 2014) suggests that prenatal exposure to MCPA may interfere with the development of the male genitalia. The authors indicate that the results should be interpreted with caution, given the relatively small number of study participants ($n = 25$), the use of a new technique for pesticide exposure analysis (meconium) and the potential for pesticide occurrence in diapers which could influence meconium pesticide levels.

2.4 Effects in animals

Acute toxicity: Acute oral toxicity data for MCPA in animals is summarized in Table 4.

Table 4. Summary of available animal acute oral toxicity data for MCPA

Forms of MCPA	Oral median lethal dose (LD50) (mg/kg bw)		
	Rats	Mice	Guinea pigs
MCPA acid	700–1,383[a,b,c,d]	439–800[e,f]	700[e]

References: a) Ben-Dyke et al. (1979); b) Rowe and Hymas (1954); c) US EPA (2003); d) US EPA (1990); e) RTECS (2005); f) Weed Science Society of America (1989)

Short- and long-term exposure studies conducted on the effects of MCPA, MCPA-DMAS and MCPA-EHE in mice and rats indicate health effects largely in the kidney and liver. Effects in other organs, hematological and neurological effects, and effects on reproductive organs have also been reported. In rats, developmental effects have been reported in the presence of maternal toxicity. However, no developmental effects have been observed in rabbits, although maternal toxicity was noted. Finally, no reproductive effects were observed in a two-generation rat study. Table 5 provides a summary of the relevant animal toxicity studies available for MCPA.

Table 5. Summary of relevant animal toxicity data for MCPA, MCPA-DMAS and MCPA-EHE

Species, number	Exposure duration	Compound and dose(s)	Critical and other effects	Ref.
Charles River rats (10/sex/dose)	90 days	**MCPA in the diet:** 0, 4, 8 or 16 mg/kg bw per day	**Kidney effects:** moderate to significant increased relative/absolute kidney weights (males). NOEL = 8 mg/kg bw per day.	Holsing and Kundzin (1970)
Rats (15/sex/dose)	90 days	**MCPA in the diet:** 0, 50, 150 or 450 ppm (equivalent to doses of 0, 3.6, 10.9 and 32.6 mg/kg bw per day for males and 0, 4.0, 12.1 and 35.8 mg/kg bw per day for females	**Kidney effects:** renal impairment (related to calcium and kidney weight changes). NOAEL = 3.6 mg/kg bw per day.	Kirsch (1985b)

Species, number	Exposure duration	Compound and dose(s)	Critical and other effects	Ref.
B6C3F1 mice (50/sex/ dose); + one satellite group (10/ sex/ dose)	2 years	**MCPA in the diet:** 0, 20, 100 or 500 ppm (equivalent to 0, 3.2, 15.7 or 79.5 mg/kg bw per day in males and 0, 3.9, 19.5 or 97.2 mg/kg bw per day in females)	**Kidney effects:** significantly increased kidney weights; kidney lesions (high-dose); non-dose-related increased renal hyperplasia (females); increased renal tubular epithelial focal hyperplasia (males). **Other effects:** systemic toxicity. NOEL = 15.7/19.5 mg/kg bw per day (males/females).	Kuhborth et al. (1988)
Mice (4–6/ sex/dose)	28 days	**MCPA in the diet:** 0, 100, 300, 900 or 2,700 ppm (equivalent to doses of 0, 19.1–22.0, 56.3–67.7, 173.4–184.8 and 453.7–820.1 mg/kg bw per day for males and 0, 20.7–26.2, 69.2–73.9, 193.4–223.9 and 442.3–956.3 mg/kg bw per day for females)	**Liver effects:** cloudy swelling in the liver of one mid-dose female. **Other effects:** motor disturbances, significant weight loss, spleen effects, decreased kidney weights, and reproductive effects in males. NOAEL = 173.4–184.8 mg/kg bw per day in males.	Kirsch (1985a)
Wistar rats (15/sex/dose)	3 months	**MCPA in the diet:** 0, 50, 500 or 2,500 ppm (equivalent to doses of 0, 3, 34 or 177 mg/kg bw per day for males and 0, 4, 42 or 188 mg/kg bw per day for females)	**Liver effects:** liver pathology **Other effects:** decreased body weight and weight gain; changes in clinical chemistry and hematological parameters, testicular atrophy and changes in motor activity. NOAEL[1] = 34 and 42 mg/kg bw per day (males/females)	Mellert et al. (1994b)
Wistar rats (50/sex/ dose); + 2 satellite groups (10–15/sex/ dose)	2 years	**MCPA in the diet:** 0, 20, 80 or 320 ppm (equivalent to 0, 1.1, 4.4 or 17.6 mg/kg bw per day in males and 0, 1.4, 5.7 or 23 mg/kg bw per day in females)	**Liver and kidney effects:** hepatotoxicity (increased alanine aminotransferase (ALT) levels) in females; kidney effects (pathological changes) in males. **Other effects:** systemic toxicity; changes in clinical chemistry; small decrease in male body weight and small/sporadic increase in female body weight; maximum tolerated dose (MTD) not achieved. NOEL = 1.1/1.4 mg/kg bw per day (males/females); NOAEL[1] = 4.4/5.7 mg/kg bw per day (males/females)	Kirsch (1988)

Species, number	Exposure duration	Compound and dose(s)	Critical and other effects	Ref.
Wistar rats: interim study (10/sex/dose); main study (50/sex/dose)	2 years	**MCPA-2-EHE in the diet:** interim study: 0, 11, 35, 106 mg/kg bw per day for males and 0, 13, 40, 128 mg/kg bw/day for females; main study: 0, 10, 29, 91 mg/kg bw per day for males and 0, 12, 38, 125 mg/kg bw/day for females	**Liver and kidney effects:** increased creatinine and urea; decreased protein levels and platelet count (males only); increased hyperplasia in bile duct (males only); increased prothrombin time (females only). **Other effects:** decreased body weight and body weight gain NOAEL = 29/38 mg/kg bw per day (males/females)	Buesen et al. (2012)
Albino rats (25/sex/dose/generation)	2 generations	**MCPA in the diet:** 0, 50, 150 or 450 ppm (corresponding to 0, 2.5, 7.5 or 22.5 mg/kg bw per day)	**Developmental effects:** statistically significant differences in body weight gain in male pups (F1a) and female pups (F1a and F1b) and in body weight/weight gain in F2a and F2b males and females. NOAEL = 22.5 mg/kg bw per day **Other:** Health Canada (2006) determined that the MTD was not achieved in this study. The MCPA Task Force Three submitted two additional one-generation range-finding reproduction studies, using either the acid or the 2-EHE form of MCPA at substantially higher dose levels which showed no adverse effects on pup body weight, indicating no increased sensitivity of the young relative to maternal animals (Health Canada, 2007).	MacKenzie (1986)
Pregnant female Wistar rats (number not specified)	Days 6–15 of gestation	**MCPA by gavage:** 0, 15, 60 or 120 mg/kg bw per day	**Reproductive effects:** maternal toxicity (treatment-related decreases in body weight, weight gain and food consumption). **Developmental effects:** decreased placental and fetal body weights, increased incidence of fetal skeletal retardation. NOAEL = 60 mg/kg bw per day	Hellwig and Hildebrand (1993a)

Species, number	Exposure duration	Compound and dose(s)	Critical and other effects	Ref.
Pregnant Himalayan rabbits (number not specified)	Days 7–19 of gestation	**MCPA by gavage:** 0, 15, 30 or 60 mg/kg bw per day	**Reproductive effects:** maternal toxicity (decreased body weight, weight gain and food consumption). NOAEL = 30 mg/kg bw per day	Hellwig and Hildebrand (1993b)
Pregnant CD rats (25/dose)	Days 6–19 of gestation	**MCPA-EHE by gavage:** 0, 23.5, 62.7 or 188 mg/kg bw per day (equivalent to 0, 15, 40 and 120 mg/kg bw per day as MCPA acid)	**Reproductive effects:** maternal toxicity (reduced body weight gains/food consumption). **Developmental effects:** litter resorption, decreased fetal weight and altered growth. NOAEL[1] = 40 mg of MCPA free acid/kg bw per day	Cappon (1999a)
Pregnant CD rats (17–25/dose)	Days 6–19 of gestation	**MCPA-DMAS by gavage:** 0, 18.5, 62 or 185 mg/kg bw per day (equivalent to 0, 15, 50 and 150 mg of MCPA free acid/kg bw per day)	**Reproductive effects:** maternal toxicity (clinical signs and mortality). **Developmental effects:** reduced mean fetal body weights; external and/or skeletal fetal malformations; fetal skeletal variations. NOAEL = 50 mg of MCPA free acid/kg bw per day	Cappon (1999b)

[1] As identified by the US EPA (2003)

MCPA-DMAS—dimethylamine salt of MCPA; MCPA-EHE or MCPA-2-EHE—MCPA 2-ethylhexyl ester; MTD—maximum tolerated dose; NOAEL—no observed adverse effect level; NOEL—no observed effect level.

2.5 Genotoxicity and carcinogenicity

The International Agency for Research on Cancer (IARC, 1983) evaluated MCPA and concluded that "no adequate data were available to evaluate the carcinogenicity of MCPA to experimental animals." The US EPA (2003, 2004c, 2004e) has classified MCPA as "not likely to be carcinogenic to humans," based on the lack of evidence of carcinogenicity in mice and rats.

Based on the weight of evidence, MCPA acid and its other forms are not considered to be of genotoxic concern in vivo. Genotoxicity tests provided equivocal results for sister chromatid exchange induction, and positive results were obtained for all three forms of MCPA in mammalian in vitro lymphocyte assays (Health Canada, 2006). This overall lack of genotoxicity following MCPA exposure is consistent with the lack of carcinogenicity in animals (Elliott, 2005). MCPA was not mutagenic in the majority of bacterial and mammalian cell gene mutation assays reported in the literature and did not induce DNA damage in the SOS chromotest. In addition, no in vivo evidence was found to suggest clastogenicity in bone marrow, and sister chromatid exchange tests gave negative or weakly positive results (Räsänen et al., 1977; Zetterberg, 1978, 1979; Nishimura et al., 1982; Moriya et al., 1983; Linnainmaa, 1984; Gelbke and Engelhardt, 1985a, 1985b, 1985c, 1985d; Kappas, 1988; Mersch-Sundermann et al., 1989; Mustonen et al., 1989; Jones et al., 1992; Adams et al., 1993a, 1993b, 1993c; Akhurst et al., 1993a, 1993b, 1993c; Jones et al., 1993a, 1993b; Proudlock et al., 1993a,1993b, 1993c; Elliott, 2005).

Long-term exposure and/or carcinogenicity studies with MCPA were conducted in rats (up to 23 mg/kg bw per day for 2 years; Kirsch, 1988) and mice (up to 97.2 mg/kg bw per day for 2 years; Kuhborth et al., 1988). No evidence of carcinogenicity was observed in either species.

2.6 Mode of action

Mode of action data on MCPA-mediated kidney effects are limited. Mechanisms of kidney toxicity have been suggested from data generated from studies of nephrotoxic acute kidney injury following MCPA mediated self-poisoning events (Flanagan et al., 1990; Bradberry et al., 2000; Roberts et al., 2005, 2011). Mohamed et al. (2015) suggested that MCPA may induce kidney toxicity through both the epithelial and vascular injury pathways, leading to cellular dysfunction, necrosis and apoptosis and causing reduced glomerular filtration rate and acute kidney injury. With evidence from Zychlinski and Zolnierowicz (1990) and Bradberry et al. (2000), Mohamed et al. (2015) proposed that epithelial injury may be initiated through adenosine triphosphate depletion (via uncoupled phosphorylation) leading to caspase pathway activation and cytoskeletal damage; vascular injury is proposed to occur via increased thromboxane A2 activity leading to vascular congestion through microvascular sludging and leukocyte aggregation.

Evidence for epithelial injury following MCPA exposure is supported by the findings of Wunnapuk et al. (2014), who measured various urinary biomarkers of renal injury in rats exposed to 40, 80, 200 and 400 mg/kg of MCPA (dimethylamine salt) in water. The biomarkers measured included albumin, β2-microglobulin and cystatin C, which indicate proximal tubular and glomerular injury; kidney injury molecule (KIM-1), a biomarker specific to proximal tubular injury; and neutrophil gelatinase associated lipocalin, which indicates proximal and distal tubular injury.

2.7 Selected key study

The PMRA (Health Canada, 2006; 2007; 2008) considers the kidney as the most sensitive target organ in the database. The 90-day study in rats by Kirsch (1985b) was identified as the key study for the human health risk assessment of MCPA in drinking water.

Kirsch (1985b) administered technical-grade MCPA (94.8%) to rats (15/sex/dose) at dietary concentrations of 0, 50, 150 or 450 ppm (equivalent to doses of 0, 3.6, 10.9 and 32.6 mg/kg bw per day for males and 0, 4.0, 12.1 and 35.8 mg/kg bw per day for females). At 450 ppm, an increase in creatinine values in the plasma of females was observed along with decreased cholesterol and calcium values in the males (the author indicated that these effects were difficult to evaluate given they occurred only in males). An increase in absolute and relative kidney weights in males was also observed. At 150 ppm, increased absolute kidney weights (108% of controls) were noted ($p < 0.05$). No changes were observed at the lowest level (50 ppm). Based on renal impairment related to calcium and kidney weight changes, it was concluded that the no-observed adverse effect level (NOAEL) lies between 50 and 150 ppm; however, no histopathological alterations were detected that correlated with this increase in kidney weight (Kirsch, 1985b). Based on this same study, Health Canada (2007) established a NOAEL of 3.6 mg/kg bw per day and a lowest-observed adverse effect level of 10.9 mg/kg bw per day based on kidney effects (increased absolute and relative weights, urinary bilirubin, crystals and pH).

3.0 DERIVATION OF THE HEALTH-BASED VALUE

A non-cancer health endpoint is most appropriate for the basis of a health-based value (HBV) for MCPA in drinking water since the available scientific evidence for carcinogenicity is largely negative in animals and inconclusive in humans. Health Canada (2006, 2007, 2008) has derived a chronic (lifetime) acceptable daily intake (ADI) for MCPA of 0.04 mg/kg bw per day using the NOAEL of 3.6 mg/kg bw per day from the 90-day study in rats by Kirsch (1985b). The NOAEL was based on kidney effects (increased absolute and relative weights, urinary bilirubin, crystals and pH). The ADI was calculated as follows:

$$\text{ADI} = \frac{\text{3.6 mg/kg bw per day}}{100}$$

$$= \text{0.036 mg/kg bw per day}$$

where:

- 3.6 mg/kg bw per day is the NOAEL in the Kirsch (1985b) 90-day study in rats; and
- 100 is the uncertainty factor which accounts for interspecies (×10) and intraspecies (×10) variability; PMRA recently concluded that an uncertainty factor for database deficiencies is no longer required since the 2-year combined chronic/carcinogenicity study by Buesen et al. (2012) completes the scientific database for MCPA, which is now considered adequate (Health Canada, 2020).

Based on the ADI of 0.036 mg/kg bw per day, an HBV for MCPA in drinking water was derived as follows:

$$\text{HBV} = \frac{0.036 \text{ mg/kg bw per day} \times 74 \text{ kg} \times 0.20}{1.53 \text{ L/day}}$$

$$\approx 0.35 \text{ mg/L (350 µg/L)}$$

where:

- 0.036 mg/kg bw per day is the ADI as calculated by Health Canada (2006);
- 74 kg is the adult body weight (Health Canada, 2021);
- 1.53 L per day is the daily volume of tap water consumed by an adult (Health Canada, 2021); and
- 0.20 is the default allocation factor since drinking water is not a major source of exposure to MCPA and there is evidence of MCPA in other exposure sources (i.e., food) (Krishnan and Carrier, 2013).

4.0 ANALYTICAL AND TREATMENT CONSIDERATIONS

4.1 Analytical methods to detect MCPA

Standardized methods available for the analysis of MCPA in source and drinking water and their respective MDLs are summarized in Table 6. MDLs are dependent on the sample matrix, instrumentation, and selected operating conditions and will vary between individual laboratories. These methods are subject to a variety of interferences (i.e., general and/or specific) which are outlined in the respective references.

Drinking water utilities should discuss sampling requirements with the accredited laboratory conducting the analysis to ensure that quality control procedures are met and that MRLs are low enough to ensure accurate monitoring at concentrations below the MAC. Sample processing considerations and method interferences for the analysis of MCPA in drinking water (e.g., sample preservation, storage) can be found in the references listed in Table 6. It is important to note that quenching is critical if an oxidant is present in samples, in order to prevent additional degradation of MCPA prior to analysis.

Table 6. Standardized methods for the analysis of MCPA in water

Method (Reference)	Methodology	Interferences/Comments	MDL (µg/L)
EPA 555 Rev 1.0 (US EPA, 1992)	High Performance Liquid Chromatography with Ultraviolet Detection (HPLC-UV)	Acid-rinse glassware and acidify sodium sulfate reagent to prevent loss of organic acids.	0.8
EPA-RCA:8151A (US EPA, 1996)	Gas Chromatography with Electron Capture Detection (GC/ECD)	Ensure complete esterification to ensure adequate recoveries	0.056[a]
USGS-NWQL: O-1131-95 (USGS, 1996)	High Performance Liquid Chromatography with Ultraviolet Detection (HPLC-UV)	None listed in method	0.050
USGS-NWQL: O-2060-01 (USGS., 2001)	High Performance Liquid Chromatography with Mass Spectrometry Detection (HPLC-MS)	None listed in method	0.0155
ISO 15913 (ISO 2000)	Gas Chromatography with Mass Spectrometry (GC-MS)	Ensure that samples are collected as per method to avoid interferences.	Not available

[a] estimated detection limit

MDL—method detection limit

4.2 Treatment considerations

Treatment technologies available to effectively decrease MCPA concentrations in drinking water include activated carbon, membrane filtration, oxidation and advanced oxidation processes (AOPs), as well as biological filtration. The reported removals varied depending on operating conditions and ranged from > 65% to 100% for nanofiltration (NF) and reverse osmosis (RO); from 60% to > 90% for oxidation and AOPs; and from 40% to 100% for biological filtration. Activated carbon produced variable results in terms of MCPA removal but may provide viable treatment options depending on operating conditions. The application of conventional filtration was ineffective at removing MCPA from water.

At the residential scale, certified treatment devices relying on reverse osmosis or activated carbon adsorption are expected to be effective for removal of MCPA.

4.2.1 Municipal scale

The selection of an appropriate treatment process will depend on many factors, including the raw water source and its characteristics, the operational conditions of the selected treatment method and the utility's treatment goals. Bench- or pilot-scale testing is recommended to ensure that the source water can be successfully treated and that optimal process design is established.

The surface water study by Donald et al. (2007) discussed in Section 1.3 investigated MCPA concentrations in treated water from 15 drinking water reservoirs. Each drinking water supply had various treatment processes, but all of them included chlorination and most included flocculation (alum) and settling, activated carbon and/or sand filtration. The study provided only overall statistics; raw data were not provided to determine the performance of individual treatment processes, only overall statistics. The mean MCPA concentrations in reservoir and treated water were 57 ng/L and 31 ng/L, respectively (n = 163), with a maximum MCPA concentration in the treated water of 865 ng/L. A mean reduction of 45% in MCPA concentration was calculated from 26 paired samples, with a range of 0% to 93%.

When using oxidation, AOPs and biological processes for pesticide removal in drinking water, it is important to be aware of the potential formation of by-products due to degradation of the target compound (Ikehata and Gamal El-Din, 2006; Beduk et al., 2012; Li et al., 2019). The primary objective should be removal of the pesticide with the secondary objective being the minimization of by-product formation if they are of health concern. In addition, water utilities should consider the potential for the formation of disinfection by-products depending on the oxidant selected and the source water quality.

4.2.1.1 Conventional treatment

While conventional filtration (chemical coagulation, clarification and rapid sand filtration) alone is not expected to be effective, the addition of chlorine during the disinfection step may reduce MCPA concentrations through oxidation (see Section 4.2.1.4). A small number of studies on the removal of MCPA through conventional filtration can be found in the literature. The concentrations of organic compounds, such as pesticides, may be reduced through coagulation/flocculation if they are hydrophobic or have high molecular weight and have acidic functional groups (Randtke, 1988). The chemical properties of MCPA (moderately lipophilic; substituted acetic acid) may result in limited removal by conventional water treatment.

Operational data collected from conventional water treatment plants in Lethbridge and Carmangay, Alberta showed reductions of 50% and 0% in MCPA in treated water, respectively (Byrtus et al., 2004).

4.2.1.2 Activated carbon adsorption

Activated carbon adsorption is a widely used technology to reduce the concentration of micropollutants, including pesticides, in drinking water (Petrie et al., 1993; Ignatowicz, 2009; Haist-Gulde and Happel, 2012; van der Aa et al., 2012; Abdel daiem et al., 2015). Activated carbon can be applied in two ways: slurry application using powdered activated carbon (PAC) and fixed-bed reactors with granular activated carbon (GAC) (Chowdhury et al., 2013).

Data generated through bench-scale testing to determine adsorption coefficients for pesticides is useful in predicting whether activated carbon adsorbs a particular pesticide (US EPA, 2011). In general, pesticides with an adsorption capacity constant (e.g., Freundlich coefficient [K]) greater than 200 µg/g $(L/\mu g)^{1/n}$ are considered to be amenable to removal by carbon adsorption (Speth and Miltner, 1998; Speth and Adams, 1993; US EPA, 2011). However, it is important to note that the presence of natural organic matter (NOM) adds complexity to activated carbon treatment because NOM competes directly for adsorption sites or fouls the carbon by blocking pores (Chowdhury et al., 2013). In the case of MCPA, the presence of sodium chloride may reduce adsorption through a screening effect (Abdel daiem et al., 2015). Furthermore, adsorption capacity is pH dependent such that the removal of MCPA increases with decreasing pH value (see Table 7) (Kim et al., 2008).

Adsorption isotherm tests show that activated carbon has a high adsorption capacity for MCPA as a result of attractive electrostatic interactions (see Table 7) and that it has a greater adsorption capacity compared to 2,4-D (Abdel daiem et al., 2015). Compared to atrazine, MCPA was found to be weakly adsorbed onto activated carbon bed in a mini-column and may substantially reduce the lifetime of the activated carbon bed (Gérard and Barthélemy, 2003).

The use of GAC is an effective approach for treating organic contaminants that are regularly found in source water at concentrations of concern (Chowdhury et al., 2013). The capacity of GAC to remove pesticides by adsorption depends on the filter velocity, empty bed contact time (EBCT), the GAC characteristics (type, particle size, reactivation method), the absorbability of the contaminant, and the filter run time (Haist-Gulde and Happel, 2012).

Operational data from a municipal-scale treatment plant in Atlanta, Georgia, using conventional pre-treatment with a GAC filter-adsorber showed that this type of treatment system can reduce low influent MCPA levels of 0.47 µg/L to below 0.02 µg/L (Frick and Dalton, 2005). No information was provided on the operational conditions of the GAC adsorber used in this study. A pilot study by Boucherie et al. (2010) (Table 8) was able to achieve a removal rate of greater than 86% using GAC.

The use of PAC offers the advantage of providing virgin carbon when required (e.g., during the pesticide application season) (Miltner et al., 1989). Removal efficiency depends on the PAC characteristics (type and particle size), dose, contact time, contaminant adsorbability and NOM presence (Gustafson et al., 2003; Summers et al., 2010; Haist-Gulde and Happel, 2012; Chowdhury et al., 2013).

Based on the limited available studies, MCPA is weakly adsorbed. Since the adsorption capacity of activated carbon can be affected by many factors, including the compound's ionic character and the solution pH, appropriate testing (e.g., jar tests and rapid small-scale column tests) should be conducted to confirm removal.

Table 7. Adsorption studies for MCPA

Initial Concentration (µg/L)	Activated Carbon	V spec[a] (m^3/kg)	Bv[b] (m^3/m^3)	Overall description	Reference
500	F400	51	23,460	Bench-scale. Reconstituted mineral water with added humic acids. AC: Mass: 200 ±5 mg; ρ: 425 kg/m^3 Micro-column: H: 25 cm, D: 0.5 cm, carbon bed height: ±2.4 cm; Flow rate: 3 mL/min; EBCT: 0.16 min.	Gérard and Barthélemy (2003)
100		54	15,610		
Initial Concentration	**Activated Carbon**	**pH**	**K (mol/kg)/ $(mol/m^3)^{-1/n}$**	**Overall description**	**Reference**
0.45 mol/m^3	F400	3.5	2.89	Bench-scale. Distilled water. Jar test AC mass: 0.001 to 0.25 g in 200 mL solution; particle ρ: 682 kg/m^3	Kim et al. (2008)
		7.0	0.61		
		10.0	0.62		
Initial Concentration (mg/L)	**Activated Carbon**	**KF (L/g)**		**Overall description**	**Reference**
50–500	Sorbo Norit	3.29		Bench-scale. Distilled water, 100 mg of AC in 100 mL of adsorbate aqueous solution of varying concentration. AC: particle size: 0.45–1.0 mm.	Abdel daiem et al. (2015)
	Ceca AC40	2.0			

[a] V spec—specific throughput (data from Haist-Gulde and Happel, 2012)

[b] Bv—breakthrough volume (data from Haist-Gulde and Happel, 2012)

AC—activated carbon; D—diameter; EBCT—empty bed contact time; H—height; K—Freundlich coefficient; KF—Langmuir constant.

Table 8. Removal of MCPA by activated carbon

Initial Concentration (µg/L)	Activated Carbon	Effluent Concentration (µg/L)	Overall description		Reference
0.42	No details	0.06 (86%)	EBCT = 5 min	**Pilot-scale** Column: D: 300 mm; H: 2 m Ozonated/ deozonated water Flow rate: 750 L/h; pH 7.2; 16.9 to 17.7°C Concentration remains the same over 3 days	Boucherie et al. (2010)
		> 0.05 (> 88%)	EBCT = 10 min		

EBCT—empty bed contact time

D—diameter; EBCT—empty bed contact time; H—height.

4.2.1.3 Membrane filtration

In general, NF and RO are effective pressure-driven membrane processes for the removal of pesticides from drinking water (Van der Bruggen and Vandecasteele, 2003; US EPA, 2011). The effectiveness of NF and RO for pesticide removal is dependent on the membrane characteristics, pesticide properties, feed-water composition, operating conditions and membrane fouling (Hofman et al., 1997; Taylor, 2000; Košutić and Kunst, 2002; Van der Bruggen and Vandecasteele, 2003; Schippers et al., 2004; Plakas and Karabelas, 2012; Fini et al., 2019).

Since size exclusion is the main mechanism for pesticide removal using NF and RO membranes, the molecular weight cut-off (MWCO) of the membrane is an important characteristic. When choosing a membrane, the molecular weight of MCPA (200.62 Da) should be considered. In addition to the sieving effect, retention of small pesticide molecules by larger pore-size membranes can be influenced by the physicochemical interactions between the pesticide and the membrane surface (Plakas and Karabelas, 2012). Bellona et al. (2004) present a flow chart that can be used to assess the potential for removal by membrane filtration by taking into account the characteristics of the pesticide in water (e.g., molecular weight, log Kow, molecular diameter) and those of the membrane (e.g., MWCO, pore size). MCPA is somewhat hydrophobic (log Kow > 2) and has a fairly low pKa, indicating potential for further removal through hydrophobic bonding to the membrane surface and electrostatic exclusion (Bellona et al., 2004; Plakas and Karabelas, 2012).

A pilot-scale study on the use of NF membranes with water pre-treated with conventional filtration reported average MCPA removals of 99% to effluent levels below 0.1 µg/L (see Table 9) (Schippers et al., 2004). A study on the use of ultra-low pressure (ULP)-RO membranes achieved a retention level > 97% of MCPA, and after 3 years, no decline in retention of pesticides, including MCPA, was observed (Bonné et al., 2000).

Bench and pilot-scale studies have shown that NF and RO are effective for the removal of MCPA from drinking water (Hofman et al., 1997; Bonné et al., 2000; Taylor 2000; Schippers et al., 2004; Fini et al., 2019). Studies using a variety of membrane types and operating conditions for MCPA removal are reported in Table 9. These data demonstrate that rejections of MCPA ranging from > 65% to 100% can be achieved.

Košutić and Kunst (2002) showed that rejection by NF and RO membranes is primarily governed by the sieving mechanism and by physiochemical effects (e.g., electrostatic repulsions).

A laboratory-scale study by Fini et al. (2019) found that NF membranes had lower rejection rates (MWCO > the molecular weight of MCPA) than RO and low-pressure (LP) RO membranes. The rejection rate for LP-RO membranes was found to be correlated with feed recovery where the rejection rate increased from 94.5% to 99% by increasing the recovery from 10% to 90%. The study also found that adsorption of the pesticide onto the surface of the membranes was greater for RO than NF. An increase in concentration in the feed water increased adsorption onto all the membranes and did not affect the rejection rate.

Table 9. MCPA removal via reverse osmosis and nanofiltration

Influent Concentration	Rejection rate	Membrane Type	Process Description	Reference
2 µg/L	99%	NF	Pilot-scale: 3-stage configuration with five, three, and two pressure vessels per stage. Each pressure vessel contains 3 elements with D: 4 in and H: 40 in. Dosed with 2 µg/L of pesticides; Average flux: 13.0 gsfd (L/m^{2}h); recovery: 80%	Schippers et al. (2004)
5 µg/L	97%–99%	ULP-RO	Pilot-scale: Feed flow rate: 9 m^3/h; Recovery: 85%.	Bonné et al. (2000)

Influent Concentration	Rejection rate	Membrane Type	Process Description		Reference
9.6 µg/L (ppb)	100%	Polyamide-urea RO MWCO: 200–300 Da	Flux: 7.65 gsfd; Recovery: 49.3%; Feed pressure: 117 psi	Pilot-scale: 2 stage system consisting of three elements per pressure vessel. Ground water from a 196-foot well. CaCO3: 120 mg; TDS: 250 mg/L; TOC: 3 mg/L.	Taylor (2000)
6.3 µg/L (ppb)	100%		Flux: 4.06 gsfd; Recovery: 75.7%; Feed pressure: 120 psi		
12.2 µg/L (ppb)	92.4%		Flux: 7.3 gsfd; Recovery: 72.0%; Feed pressure: 135 psi		
1 mg/L	> 65%[a]	Polyamide thin film composite (TFC) NF MWCO: 200–400 Da	**Bench-Scale:** ultra-pure Milli-Q water Dead-end filtration with 50 mm diameter membrane disc. Test conditions: P:10 bar; 22 °C; Terminated at 50% recovery		Fini et al. (2019)
	> 70%[a]	Polyamide TFC NF MWCO: >200 Da			
	> 90%[a]	LP-RO MWCO: >100 Da			
	> 95%[a]	RO MWCO: > 100 Da			
2–4 mg/L (ppm)	82.3%–93.6%	Thin layer composite RO	**Bench-scale:** Test conditions: P: 17 bar; Short run 3 h.		Košutić and Kunst (2002)
	91.2%	Polyamide NF			
4.5 µg/L	95%[b]	Cellulose-acetate RO	**Bench-scale:** surface water pre-treated with coagulation, sedimentation, filtration, and ultra-filtration. Test conditions: All membranes in 4 × 40 spiral-wound configuration, 9% recovery. At 80% recovery, initial concentration of 1.4 µg/L and composite polyamide RO: 93% rejection estimated from modelling data.		Hofman et al. (1997)
	97%[b]	Composite polyamide RO			
	97%[b]	ULP-RO			

[a] as read from graph

[b] as cited in Taylor, 2000

D—diameter; gsfd—gallons per square foot per day; H—height; MWCO—molecular weight cut-off; NF—nanofiltration; P—pressure; RO—reverse osmosis; TDS—total dissolved solids; TFC—thin film composite; TOC—total organic carbon; ULP—ultra low pressure.

4.2.1.4 Oxidation and hydrolysis

Hydrolysis is the main chemical reaction that initiates the degradation of the esters of phenoxy acids in aqueous systems. During hydrolysis, phenoxy acid is generated from the esters of MCPA. The rate of the reaction is determined by the water pH and temperature. MCPA-EHE does not hydrolyze within a pH range of 5 to 7, and its half-life (DT50) value at pH 9 is < 117 h (Muszynski et al., 2020).

Pilot- and bench-scale oxidation studies of MCPA using ozone (O3) and ultraviolet (UV) photolysis indicated that both processes can remove MCPA from water, with removal efficiency ranging from 83% to > 90%. The effectiveness of oxidation depends on a variety of factors including oxidant dose, disinfectant demand, temperature and pH (see Table 10) (Meijers et al., 1995; Bourgine et al., 1997; Benitez et al., 2004; Hollender et al., 2009; Boucherie et al., 2010). Hu et al. (2000) calculated the ozonation rate constant for MCPA and confirmed that ozone has a relatively high level of reactivity toward MCPA.

A pilot-scale study evaluated the efficiency of both ozonation and GAC adsorption for removal of 36 pesticides, including MCPA, in surface water. The treatment processes were evaluated separately and found to have similar efficiency for MCPA removal. The ozonation process reduced MCPA concentrations in the treated water in the range from below the detection level of 0.05 µg/L to 0.06 µg/L (86% to > 88% removal). The authors concluded that the synergy effect of the two processes was needed to remove other pesticides, which were partially (weakly) removed (defined as 10%–70%) by ozonation (Boucherie et al., 2010). Another pilot-scale study reported that oxidation of MCPA increased when the O3 dose increased (Halevy et al., 2013). Similar results were reported by Meijers et al. (1995), indicating that MCPA was easily degraded in river water at an ozone dose slightly higher than that required for drinking water disinfection (reported as the O3 to dissolved organic carbon [DOC] ratio). The results indicated that removal increased when the pH and temperature increased.

Bourgine et al. (1997) reported limited data from a pilot-scale test using UV photolysis to reduce the concentrations of several pesticides, including MCPA, in groundwater. MCPA was easily degraded by UV photolysis, with > 85% removal. A bench-scale study reported effective degradation of MCPA using a low-pressure UV lamp. Approximately 90% degradation of MCPA was reported at pH levels in the range of 5.0 to 9.0, compared to only 6 % degradation at pH 3.0. The authors attributed the higher degradation to the higher reactivity of the dissociated form of MCPA at pH > 5.0 [pKa 3.7 (for this study)]. However, it should be noted that the initial MCPA concentration was relatively high (Benitez et al., 2004).

Processes like oxidation, hydrolysis and biodegradation of MCPA may result in the formation of degradation by-products such as phenoxyacetic acid and 2-methyl-4-chlorophenol (2-M4CP) (Zertal et al., 2001; McManus et al., 2017; Kelly et al., 2019; Muszynski et al., 2019; Morton et al., 2019). The formation of oxalic and glycolic acids was reported after oxidation of 18.3 µg/L of MCPA with an ozone dose of 4.3 mg/L and pH 8.0 in aqueous solution (Struif et al., 1978). Camel and Bermond (1998) reported that mineralization of pesticides using ozonation or advanced oxidation techniques is generally incomplete. As a result, water treatment utilities using these methods should include sand or GAC filtration following oxidation. It has been observed that the use of ozonation prior to GAC adsorption or sand filtration increases the biological activity of the filter bed and may extend bed life for pesticide removal (Lambert and Graham, 1995; Hollender et al., 2009).

Table 10. Removal of MCPA via oxidation

Oxidant	Influent Concentration	Oxidant dose (mg/L), O3/DOC ratio (g/g) or Energy (Wh/m³)	Removal	Process Description	References
O3	0.42 µg/L	1.2–2.3 mg/L	86% to > 88%	Pilot scale: Sand filtered water spiked with MCPA; CT[a] = 1.1–5.4 mg min/L; dissolved O3 residual: 0.13–0.68 mg/L; pH 7.3; water flow rate: 12 m³/h.	Boucherie et al., (2010)
	10 µg/L	0.75 mg/L	14%	Pilot scale: Pre-treated surface water (PAC adsorption, coagulation, flocculation/ sedimentation/ filtration) spiked with MCPA; 3 ozone contactors in series with total contact time 30 min; water flow rate 7.89 L/ min; pH 7.3–7.6; turbidity 0.11–0.15 NTU; 13–19°C; DO 9.0–11.2 mg/L.	Halevy et al., (2013)
		1.5 mg/L	74%		
		2.25 mg/L	86%		
	0.9–6.4 µg/L	1.0 g/g	83%	CT[a] = 7.3; pH 7.2; 5⁰ C — Bench-scale: Pre-treated river water (coagulation and flotation); DOC 2.2 mg C/L; Br^- 100 µg/L, $HCO3^-$ 1.6 mM; 23 pesticides.	Meijers et al., (1995)
		1.0 g/g	87%	CT[a] = 3.3; pH 7.2; 20⁰ C	
		1.0 g/g	90%	CT[a] = 1.1; pH 8.3; 20⁰C	
UV	NA	Energy 100–500 Wh/m³	62.5%–100 Wh/m³ 86%–200 Wh/m³ 94%–300 Wh/m³	Pilot scale: groundwater; pH 7.1–7.2; turbidity 0.1–0.2 NTU; TOC 0.5–1.54 mg/L; nitrate 35–40 mg/L; sulphate 50–60 mg/L; medium pressure (MP) mercury lamp.	Bourgine et al., (1997)
	50 mg/L	NA	90% (in 20 min)	Bench scale: ultra-pure water; pH levels 3, 5, 7 and 9; LP lamp with a radiation intensity 2.03 × 10^{-6} Eins/s	Benitez et al., (2004)

CT[a]—disinfection criterion (mg*min/L). Contact time (T) calculated using a T10 value

DO—dissolved oxygen; DOC—dissolved organic carbon; LP—low pressure; NA—not available; PAC—powdered activated carbon; TOC—total organic carbon; UV—ultraviolet.

4.2.1.5 Advanced oxidation processes

In general, the applied UV dose for degradation of micro-pollutants in water is dependent on the UV power (kW), the UV transmittance of the water and the flow through the UV reactor. A pilot-scale UV/hydrogen peroxide (H2O2) advanced oxidation system compared the performance of medium-pressure and low-pressure UV lamps in terms of degradation of organic micropollutants, including MCPA. Table 11 presents removal efficiencies for both MP and LP lamps under the same experimental conditions. The MP lamp used in the study was not equipped with a screen to filter lower UV wavelengths that can induce nitrite formation in the water; therefore, an increase of nitrite concentration was measured in the treated water (approximately 0.5 mg N/L). This increase was not observed in the water treated with the LP lamp (< 0.05 mg N/L) (Lekkerkerker-Teunissen et al., 2013).

Benitez et al. (2004) reported an increase in the efficiency of the UV/H2O2 process for the degradation of MCPA as compared to photodegradation alone. A higher reaction rate constant was observed for the UV/H2O2 process when compared to the reaction rate of the photodegradation process alone: 0.154 (min^{-1}) with H2O2 dose of 1×10^3 M; 0.212 (min^{-1}) with H2O2 dose of 2×10^3 M; and 0.128 (min^{-1}) for the single UV radiation process.

Table 11. Removal of MCPA via advanced oxidation processes (Lekkerkerker-Teunissen et al., 2013)

Process	Influent Concentration (µg/L)	H2O2 dose (mg/L)	UV dose (mJ/cm^2)	Removal	Process Description
UV/ H2O2	5–10	5	875	> 90% (MP)	Pilot-scale: 2 reactors –MP (power-4.4 kW), LP (power-1.32 kW); Experimental conditions: 100% UV ballast (output), UV-transmittance 72.5%, design flow 5 m^3/h per reactor. Feed water: treated water from a conventional DWTP spiked with MCPA: avg. DOC 4.7 mg/L; avg. nitrate 3.64 mg N/L; avg. bicarbonate 170 mg/L; UV transmission 73–83% UV-T.
			741	> 60% (LP)	

DOC—dissolved organic carbon; DWTP—drinking water treatment plant; LP—low pressure; MP—medium pressure; UV—ultraviolet.

4.2.1.6 Biological filtration

Biological filtration processes include slow sand filtration, engineered biological filtration (biofiltration), and riverbank filtration (RBF). For slow sand filtration, biodegradation is the significant removal mechanism. Engineered biofiltration involves the use of rapid granular media filters (i.e., anthracite/sand or GAC) without the maintenance of a disinfectant residual across the filter bed (Symons et al., 2000). RBF involves locating vertical or horizontal water supply wells near a river in order to use the riverbank and adjacent aquifer as a natural filter to remove contaminants. As water proceeds to the groundwater table, contaminant concentrations are reduced through adsorption, biodegradation, and dilution with groundwater (Piet and Zoeteman, 1980; Bize et al., 1981; Kuehn and Mueller, 2000; Ray et al., 2002). Natural attenuation through RBF is one of the most basic methods of water treatment (Verstraeten and Heberer, 2002; Sørensen et al., 2006). However, adsorption plays only a minor role in the case of polar organic compounds such as MCPA (Huntscha et al., 2013).

Full-, pilot- and bench- scale studies have reported that biodegradation of MCPA can be an effective treatment method, achieving 40% to 100% removal of MCPA in water (Woudneh et al., 1996; Gonzalez et al., 2006, 2017; Huntscha et al., 2013; Halevy, 2013; Kruger et al., 2015; Samuelsen et al., 2017; Matamoros and Franco, 2018; Vandermaesen et al., 2019; Oberleitner et al., 2020).

In general, phenoxyacid pesticides may degrade under aerobic and anaerobic conditions (Muszynski et al., 2020). However, published data indicate that a high level of degradation of MCPA is achieved under aerobic conditions (see Tables 12 to 16) but that degradation is negligible in anaerobic environments (Harrison et al., 1998; Albrechtsen et al., 2001; Gonzalez et al., 2017; Matamoros and Franco, 2018; Morton et al., 2019). A field study observed greater than 99% biodegradation of MCPA after 14 days under aerobic conditions. No biodegradation was observed in anaerobic tests (Harrison et al., 1998).

Slow sand filtration

A full-scale study examined the capacity of biologically active sand filters to mineralize MCPA in eleven drinking water treatment plants (DWTPs) (see Table 12). However, all treatment plants, except one, included rapid sand filtration in different treatment trains. The authors observed that MCPA was mineralized in all sand filters. In general, the observed MCPA mineralization across the sand filters was consistent in time and across the samples taken from different filters at the same treatment plant. In addition, the mineralization of MCPA was found to be positively correlated with the pH of the water, the availability of ammonia, carbon sources, iron and oxygen concentrations, and negatively correlated with the sulphate concentration (Vandermaesen et al., 2019).

A pilot-scale study found that two slow sand filters had a high capacity to remove MCPA to a level below the detection limit of 0.1 µg/L (see Table 13). Consistent removal of MCPA was observed during a period of 18 days. The authors concluded that the removal of MCPA involved a continuous biodegradation process, and that its efficiency was not affected by the variations in flow rate and bed depths under the tested conditions (Woudneh et al., 1996).

Another study investigated the capacity of three different biofilter materials, including sand, to remove several phenoxyacid herbicides from agricultural runoff waters (see Table 12). The average degradation of all tested phenoxyacid pesticides in the sand column was 77%. The removal of MCPA declined with an increase in the hydraulic loading rate (HLR) to the column. The HLR was fairly low, indicating that the results would be representative of a slow sand filter or riverbank filter (Matamoros and Franco, 2018).

Samuelsen et al. (2017) reported that a selected non-motile, hydrophobic microbiological strain (originating from a groundwater aquifer) with a strong degree of adhesion to sand, showed a high mineralization rate (> 50%) at all tested MCPA concentrations (0.016–25.0 mg/L) (see Table 12). These results demonstrate that the surface hydrophobicity and adherence abilities of the biological activity are the parameters controlling sustained biodegradation in flow-through sand columns and they must be considered when selecting biological filtration.

Table 12. Slow sand filtration

Influent Concentration (µg/L)	Removal	EBCT (min) or HLR (m/day)	Process Description	References
0.008–0.18	37.0–55.5%	NA	Full scale: 11 DWTPs; intake water: surface water, groundwater and blended water; pH 6.3–8.4; $NH4^{+}$ 0.2–3.1 mg/L; $NO3^{-}$ 0.0–10 mg/L; $PO4^{3-}$ 0.01–1.6 mg/L; $SO4^{2-}$ 2.0–180.3 mg/L; non-purgeable organic carbon 0.7–12.8 mg/L; 11.2–13.9°C; (% removal is a range for 8 sand filter samples).	Vandermaesen et al., (2019)
10	93%	0.3 m/day	Bench scale: 30-day acclimation; agricultural runoff water spiked with MCPA; column: 100 cm sand; 10 µg/L pesticide mixture; Test period of 20 days; pH 6.8; DO 3.4±0.9 mg/L; TSS 76 mg/L; ammonia 0.79±0.91 mg/L; nitrates 0.9±0.1 mg/L.	Matamoros and Franco, (2018)
	57%	1.4 m/day		
1,000	~100 %	NA	Bench scale: inoculated sand columns with four microbiological strains; quality of the water not provided; flow rate 0.08 mL/min (the results provided for a non-motile, hydrophobic strain)	Samuelsen et al., (2017)

DO—dissolved oxygen; DWTP—drinking water treatment plant; EBCT—empty bed contact time; HLR—hydraulic loading rate; NA—not available; TSS—total suspended solids.

Table 13. Pilot-scale slow sand filtration (Woudneh et al., 1996)

Influent Concentration (µg/L)	Removal	Bed depth (mm)	Flow rate (m/h)	Contact time (h)	Process description
6–10	100%	300	0.06	4.3[a]	Pilot-scale: 3 gravel filters in series and 2 biological filters in parallel. The results are presented only for biological filters.
			0.12	2.15[b]	
		500	0.06	7.16[a]	
			0.12	3.58[b]	

[a] Average of three independent measurements conducted October–December 1994

[b] Average of five independent measurements conducted October–December 1994

Engineered biological filtration

González et al. (2006) studied the degradation of MCPA in a bench-scale fixed-bed bioreactor (FBBR) by microorganisms isolated from a freshwater stream. The degradation of MCPA started after 10 days and the concentration reached half of the influent concentration after 13 days (see Table 14). The system was capable of degrading MCPA; however, a lag phase was needed to start the degradation. The authors indicated that the FBBR was considered a good model system since it was able to simulate the biological degradation in different water qualities. Although 2-M4CP was identified as a main degradation by-product of MCPA under aerobic conditions (Juhler et al., 2001; Muszynski et al., 2020), it was not found in the treated water (González et al. 2006).

Table 14. Engineered biological filtration

Influent Concentration (µg/L)	Removal		EBCT (min) or Flow rate (mL/min)	Process description	References
10	~100% (in 24 days)	FBBR	15 mL/min	Bench-scale FBBR: Column: porous sintered glass in which a biofilm has been grown (90 days); Feed water: primary treated wastewater spiked with MCPA.	Gonzales et al., (2006)

EBCT—empty bed contact time; FBBR—fixed-bed bioreactor.

Riverbank filtration

Four RBF sites, two anoxic (silty sand) and two oxic (gravel), with different travel distances (42–633 m) were studied for the removal of 194 micropollutants, including MCPA. MCPA was detected only two times in the surface water at concentrations greater than the limit of quantitation (LOQ 16.2 ng/L) (see Table 15). Detected MCPA concentrations corresponded to seasonal application of pesticides from March to May. Overall, the authors observed that the RBF efficiency to remove micropollutants, including MCPA, depended on travel distance. In addition, the total concentration of all micropollutants decreased by approximately 50% from the river to the first well (B1) at all RBF sites, demonstrating the general operating principle of RBF as a water purification method (Oberleitner et al., 2020).

Huntscha et al. (2013) conducted field tests to quantify the MCPA concentrations for RBF sites with short travel times (hours to a few days). Spatiotemporal sampling was performed during a high-discharge event during the pesticides application season. Although the MCPA concentration in the river water increased up to 10-fold after the discharge peak, it was quantifiable only at two piezometers with the shortest travel time (see Table 16). The authors concluded that the MCPA was degraded, with estimated half-lives in the range of a few hours (0.2–38 hours).

Table 15. Riverbank filtration (Oberleitner et al., 2020)

Influent Concentration (ng/L)	Removal	Wells	Process Description
23	100% (Spring 2018)	Ea-B1	Four RBF sites: River Ems (Ea and Eb) and River Ruhr (Ra and Rb). At each site, 3 wells (B1, B2 and B3) and an abstraction well (W) were sampled. Travel distance (time) to abstraction wells: 633 m (Ea) and 89 m (Eb) (50–60 days); 72 m (Ra), 42 m (Rb) (2 days). Three sampling campaigns: summer 2017, fall 2017 and spring 2018. High discharge rates: December 2017 to February 2018; low discharge rates: April 2017 to July 2017. (Results are presented for wells B1 and W for both sites, Ea and Eb, for which MCPA was detected in the river water).
		Ea-W	
19		Eb-B1	
		Eb-W	

RBF—riverbank filtration.

Table 16. Riverbank filtration (Huntscha et al., 2013)

Influent Concentration (ng/L)	RBF (ng/L)	Travel time (h)	Transect/ piezometer	Date	Process Description
70–410	50	6.5–28	B/R042	May 3, 2010	Field test: Short groundwater (oxic conditions) travel times from surface water infiltration to groundwater extraction (a few days). High discharge event, on May 3, 2010 (159 m^3/s). Sampling: transects A (2 piezometers) and transect B (3 piezometers). (Results are presented only for 2 piezometers (transect B) with detectable concentration).
410–651	20	6.5–28	B/R042	May 4, 2010	
130–245	27	0.5–6.5	B/R050	May 4, 2010	

RBF—riverbank filtration.

4.2.2 Residential scale

In cases where MCPA removal is desired at the household level, for example, when a household obtains its drinking water from a private well, a residential drinking water treatment unit may be an option for decreasing MCPA concentrations in drinking water. Before a treatment unit is installed, the water should be tested to determine the general water chemistry and MCPA concentration in the source water.

To verify that a treatment unit is effective, water entering and leaving the treatment unit should be sampled periodically and submitted to an accredited laboratory for analysis. Units can lose removal capacity through use and time and need to be maintained and/or replaced. Consumers should verify the expected longevity of the components in the treatment unit according to the manufacturer's recommendations and arrange for servicing as required. Residential-scale systems may have a rated capacity that exceeds the volumes needed for a single residence, and thus may also be used in small systems.

Health Canada does not recommend specific brands of drinking water treatment units, but it strongly recommends that consumers use units that have been certified by an accredited certification body as meeting the appropriate NSF International Standard/American National Standard (NSF/ANSI) for drinking water treatment units. The purpose of these standards is to establish minimum requirements for the materials, design and construction of drinking water treatment units that can be tested by a third party. This ensures that materials in the unit do not leach contaminants into the drinking water (i.e., material safety). In addition, the standards include performance requirements that specify the removal that must be achieved for specific contaminants (e.g., reduction claim) that may be present in water supplies. Certification organizations (i.e., third party) provide assurance that a product conforms to applicable standards and must be accredited by the Standards Council of Canada (SCC). Accredited organizations in Canada include:

- CSA group;
- NSF International;
- Water Quality Association;
- UL LLC;
- Bureau de normalisation du Québec (available in French only);
- International Association of Plumbing and Mechanical Officials; and
- Truesdail Laboratories, Inc.

An up-to-date list of accredited certification organizations can be obtained from the SCC. The drinking water treatment technologies that are expected to be effective for MCPA removal at the residential scale include adsorption and RO. Currently, MCPA is not included in the performance requirements of NSF/ANSI standards. However, consumers can use a treatment unit that is certified to the NSF standards for adsorption and RO to ensure that the material safety has been tested. These standards are NSF/ANSI Standard 53—Drinking Water Treatment Units—Health Effects and NSF/ANSI Standard 58—Reverse Osmosis Drinking Water Treatment Systems (NSF/ANSI, 2020a, 2020b). In addition, units that have been certified for the removal of other chlorophenoxy herbicides such as 2,4-D are likely to be effective for the removal of MCPA.

Water that has been treated using RO may be corrosive to internal plumbing components. Therefore, these units should be installed only at the point of use. Also, as large quantities of influent water are needed to obtain the required volume of treated water, these units are generally not practical for point-of-entry installation.

5.0 MANAGEMENT STRATEGIES

All water utilities should implement a risk management approach, such as the source-to-tap or water safety plan approach, to ensure water safety (CCME, 2004; WHO, 2011, 2012). These approaches require a system assessment to characterize the source water, describe the treatment barriers that prevent or reduce contamination, to identify the conditions that can result in contamination, and to implement control measures. Operational monitoring is then established, and operational/management protocols are instituted (e.g., standard operating procedures, corrective actions and incident responses). Compliance monitoring is determined and other protocols to validate the water safety plan are implemented (e.g., record keeping and consumer satisfaction). Operator training is also required to ensure the effectiveness of the water safety plan at all times (Smeets et al., 2009).

5.1 Monitoring

MCPA can be present in groundwater and surface water in areas where it is being used depending on the type and extent of its application, environmental factors (e.g. amount of precipitation, soil type, hydrogeological setting, etc.) and environmental fate (e.g., mobility, leaching potential, degradation etc.) in the surrounding area. Water utilities should consider the potential for MCPA to enter source water (e.g., raw water supply to the drinking water system) based on site-specific considerations.

When it is determined that MCPA may be present, and monitoring is necessary, then surface and groundwater sources should be characterized to determine the concentration of MCPA. This should include monitoring of surface water sources during periods of peak use and rainfall events and/or monitoring of groundwater annually. Where baseline data indicate that MCPA is not present in source water, monitoring may be reduced.

Where treatment is required to remove MCPA, operational monitoring should be implemented to confirm whether the treatment process is functioning as required. The frequency of operational monitoring will depend on the water quality, the fluctuations of the raw water concentrations and the treatment process. Responsible authorities should be aware of the impact of NOM on activated carbon and oxidation systems, as it may impact water quality objectives for MCPA removal.

Where treatment is in place for MCPA removal, compliance monitoring (i.e., paired samples of source and treated water to confirm the efficacy of treatment) should be conducted at least annually and during periods of peak use. When routine operational monitoring indicates the potential for contaminant breakthrough, such as with GAC, monitoring should be conducted at least quarterly to plan for the regeneration or replacement of the media. When a degradation process is utilized, like oxidation, by-product formation should also be considered.

6.0 INTERNATIONAL CONSIDERATIONS

Other national and international organizations have drinking water guidelines, standards and/or guidance values for MCPA in drinking water. Variations in these values can be attributed to the age of the assessments or to differing policies and approaches, including the choice of key study and the use of different consumption rates, body weights and source allocation factors (Table 17).

Table 17. Comparison of international drinking water values for MCPA

Agency (Year)	Value (mg/L)	Key Endpoint (Reference)	NO(A)EL (mg/kg bw per day)	UF	ADI (mg/kg bw per day)	BW (kg)	DW Intake (L/d)	AF (%)	Comments
Health Canada —MAC (2020)	0.35	Kidney effects (increased absolute and relative weights, urinary bilirubin, crystals and pH) (Kirsch (1985b)	3.6 (NOAEL)	100	0.036	74	1.53	20	
US EPA (2004d; 2018)	0.03 (non-regulatory lifetime HA)	Hepato-toxicity and nephro-toxicity (Kirsch, 1986)	4.4 (NOAEL)	1000	0.004 (RfD)	70	2	20	Health advisories are informal technical guidance for unregulated drinking water contaminants.

Agency (Year)	Value (mg/L)	Key Endpoint (Reference)	NO(A)EL (mg/kg bw per day)	UF	ADI (mg/kg bw per day)	BW (kg)	DW Intake (L/d)	AF (%)	Comments
WHO (2016)	0.7 mg/L (non-regulatory HBV)	Changes in clinical chemistry parameters indicative of kidney effects (Kirsch et al., 1985; Mellert et al., 1994a,b,c)	12 (NOAEL)	100	0.12	60	2	20	WHO deemed a guideline unnecessary as MCPA usually occurs in drinking water or sources at concentrations well below those of health concern. The HBV can be used to help interpret monitoring data.
NHMRC and NRMMC (2011) (Australia)	0.04 mg/L	Decreased body weight gain, spleen hemosiderosis, increased absolute kidney weight (males), and evidence of chronic nephropathy (Kirsch, 1988).	1.1 (NOEL)	100	0.01	70	2	10	

Agency (Year)	Value (mg/L)	Key Endpoint (Reference)	NO(A)EL (mg/kg bw per day)	UF	ADI (mg/kg bw per day)	BW (kg)	DW Intake (L/d)	AF (%)	Comments
EU (1998)	0.1 µg/L	The EU uses a value of 0.1 µg/L for any individual (single) pesticide, and a value of 0.5 µg/L for total pesticides found in drinking water. In establishing these values, the EU did not consider the science related to each pesticide, including health effects. Instead, the values are based on a policy decision aimed at keeping pesticides out of drinking water.							

ADI—acceptable daily intake; AF—allocation factor; BW—body weight; DW—drinking water; EU—European Union; HA—Health Advisory; HBV—health based value; MAC—maximum acceptable concentration; MCL—maximum contaminant level; NHMRC and NRMMC—National Health and Medical Research Council and National Resource Management Ministerial Council; NOEL—no observed effect level; NOAEL—no observed adverse effect level; RfD—reference dose; UF—uncertainty factor; US EPA—United States Environmental Protection Agency; WHO—World Health Organisation.

7.0 RATIONALE

MCPA is a commonly used herbicide in Canada. It is registered for use in Canada for agricultural sites, for fine turf, lawns and sod, in forestry and at industrial sites. MCPA is used extensively in Canada, particularly in the Prairies, and is among the top 10 pesticides sold in Canada. Although MCPA is used widely in Canada, exposure data do not indicate significant levels in drinking water.

MCPA is considered unclassifiable with respect to carcinogenicity in humans, based on inadequate data from epidemiological studies and a lack of adequate animal studies. The MAC for MCPA in drinking water has been established based on kidney effects in rats.

Health Canada, in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water, has established a MAC of 0.35 mg/L (350 µg/L) based on the following considerations:

- An HBV of 0.35 mg/L (350 µg/L) based on kidney effects in rats;
- MCPA can be accurately measured at concentrations well below the MAC; and
- Drinking water treatment technologies are available to remove MCPA to below the MAC.

The MAC protects against potential health effects from MCPA exposure. As part of its ongoing guideline review process, Health Canada will continue to monitor new research in this area, including the outcomes of PMRA's evaluations, and recommend any changes to this guideline technical document that it deems necessary.

8.0 REFERENCES

Abdel daiem, M.M., Rivera-Utrilla, J., Sánchez-Polo, M., and Ocampo-Pérez, R. (2015). Single, competitive, and dynamic adsorption on activated carbon of compounds used as plasticizers and herbicides. Science of the Total Environment, 537, 335–342.

Adams, K., Kirkpatrick, D., Godfrey, A., Anderson, A. and Dawe, I.S. (1993a). Chinese hamster ovary/HGPRT locus assay: MCPA acid: Final report: Lab Project No.: JEL 29/921115. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK. 42 pp. (as cited in Cal-EPA, 2000; US EPA, 2003).

Adams, K., Ransome, S., Anderson, A. and Dawe, I.S. (1993b). Chinese hamster ovary/HGPRT locus assay: MCPA DMAS: Final report: Lab Project No.: JEL 27/921113. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK. 42 pp. (as cited in Cal-EPA, 2000; US EPA, 2003).

Adams, K., Henly, S., Anderson, A. et al. (1993c). Chinese hamster ovary/HGPRT locus assay: MCPA 2-EHE: Lab Project No.: JEL 28/921114. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK (as cited in US EPA, 2003).

Akhurst, L.C., King, J., Anderson, A. and Dawe, I.S. (1993a). MCPA DMAS: Metaphase chromosome analysis of human lymphocytes cultured in vitro: Lab Project No.: JEL 30/921176. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK (as cited in Cal-EPA, 2000; US EPA, 2003).

Akhurst, L.C., King, J., Anderson, A. and Dawe, I.S. (1993b). MCPA acid: Metaphase chromosome analysis of human lymphocytes cultured in vitro: Lab Project No.: JEL 32/921190. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK (as cited in Cal-EPA, 2000; US EPA, 2003).

Akhurst, L.C., King, J., Anderson, A., Dawe, I.S. et al. (1993c). MCPA 2-EHE: Metaphase chromosome analysis of human lymphocytes cultured in vitro: Lab Project No.: JEL 31/921188. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK (as cited in US EPA, 2003).

Albrechtsen, H., Mills, M.S., Aamand, J. and Bjerg, P.L. (2001). Degradation of herbicides in shallow Danish aquifers: An integrated laboratory and field study. Pest Manag. Sci., 57(4): 341–350.

Arbuckle, T.E., Savitz, D.A., Mery, L.S. and Curtis, K.M. (1999). Exposure to phenoxy herbicides and the risk of spontaneous abortion. Epidemiology, 10(6): 752–760.

Becher, H., Flesch-Janys, D., Kauppinen, T., Steindorf, K., Manz, A. and Wahrendorf, J. (1996). Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins. Cancer Causes Control, 7: 312–321.

Beduk, F., Aydin, M. E. and Ozcan, S. (2012). Degradation of malathion and parathion by ozonation, photolytic ozonation, and heterogeneous catalytic ozonation processes. Clean—Soil, Air, Water 40 (2): 179–87.

Bellet, E.M., van Ravenzwaay, B., Pigott, G. and Leemings, N. (1999). Chronic dietary toxicity and oncogenicity evaluations of MCPA (4-chloro-2-methylphenoxyacetic acid) in rodents. Regul. Toxicol. Pharmacol., 30: 223–232.

Bellet, E.M., van Ravenzwaay, B., Hellwig, J., and Pigott, G. (2001). Reproductive toxicity of MCPA (4-chloro-2-methylphenoxyacetic acid) in the rat. Int. J. Toxicol., 20: 29–38.

Bellona, C., Drewes, J.E., Xu, P. and Amy, G. (2004). Factors affecting the rejection of organic solutes during NF/RO treatment—a literature review. Wat. Res., 38(12): 2795–2809.

Ben-Dyke, R., Sanderson, D.M. and Noakes, D.N. (1979). Acute toxicity data for pesticides. World Rev. Pestic. Control, 9: 119–127.

Benitez, F.J., Acero, J.L., Real, F.J. and Roman, S. (2004). Oxidation of MCPA and 2,4-D by UV radiation, ozone, and the combinations UV/H2O2 and O3/H2O2. J. Environ. Sci. Health, B39(3): 393–409.

Berling, I., Buckley, N.A., Mostafa, A., Downes, M.A., Grice, J., Medley, G., Roberts, M.S. and Isbister, G.K. (2015). 2-Methyl-4-chlorophenoxyacetic acid and bromoxynil herbicide death. Clin Toxicol (Phila). 53(5): 486–488.

Bize, J., Grenet, B. and Maneglier, H. (1981). Purifying capacity of the alluvial complex bordering a river. Tech. Sci. Munic., 76(7): 393–401.

Bonné, P.A.C., Beerendonk, E.F., van der Hoek, J.O. and Hofman, J.A.M.H. (2000). Retention of herbicides and pesticides in relation to aging of RO membranes. Desalination, 132: 189–193.

Boucherie, C., Lecarpentier, C., Fauchon, N., Djafer, M. and Heim, V. (2010). "Ozone" and "GAC filtration" synergy for removal of emerging micropollutants in a drinking water treatment plant? Water Sci. Tech-W Sup, 10(5): 860–868.

Bourgine, F., Chapman, J. and Martin, S. (1997). Traitement des pesticides par photolyse UV: les aspects scientifiques du traitement de l'eau en vue de sa potabilisation. TSM. Techniques sciences méthodes, génie urbain génie rural, (7–8), 23–29.

Bovey, R.W. (1980). Toxicology of phenoxy herbicides in animals and man—General considerations. In: The science of 2,4,5-T and associated herbicides. John Wiley and Sons, New York, NY. pp. 71–132.

Bradberry, S.M., Watt, B.E., Proudfoot, A.T. and Vale, J.A. (2000) Mechanisms of toxicity, clinical features, and management of acute chlorophenoxy herbicide poisoning: a review. J. Toxicol. Clin. Toxicol. 38: 111–22.

British Columbia Ministry of Health (2019). Personal communication with D. Fishwick.

Bueno de Mesquito, H.B., Doornbos, G., van der Kuip, D.A.M., Kogevinas, M. and Winkelmann, R. (1993). Occupational exposure to phenoxy herbicides and chlorophenols and cancer mortality in The Netherlands. Am. J. Ind. Med., 23: 289–300.

Buesen, R., Strauss, V., Küttler, K., Becker, M. and van Ravenzwaay, B. (2012). MCPA-2-EHE Combined Chronic Toxicity/Carcinogenicity Study in Wistar Rats Administration via the diet up to 24 Months. Unpublished report. BASF, Ludwigshafen, Germany (Project No. 80S0351/07128; PMRA #2224379).

Byrtus, G., Pongar, K., Browning, C., Burland, R., McGuinness, E. and Humphries, D. (2004). A summary of pesticide residue data from Alberta Treated Water Survey, 1995–2003. Environmental Assurance Service, Alberta Environment, Edmonton, Alberta. 57 pp.

Camel, V. and Bermond, A. (1998). The use of ozone and associated oxidation processes in drinking water treatment. Water Res., 32(11): 3208–3222.

Cappon, G.D. (1999a). A prenatal developmental toxicity study of MCPA-EHE in rats. Unpublished report, WIL Research Laboratories, Ashland, OH, October 12 (Laboratory Study No. WIL-325004; MRID 44954101) (as cited in US EPA, 2003.

Cappon, G.D. (1999b). A prenatal developmental toxicity study of MCPA-DMA in rats. Unpublished report, WIL Research Laboratories, Ashland, OH, October 12 (Laboratory Study No. WIL-325003; MRID 44954102) (as cited in US EPA, 2003.

Caux, P.-Y., Kent, R.A., Bergeron, V., Fan, G.T. and Macdonald, D.D. (1995). Environmental fate and effects of MCPA: a Canadian perspective. Crit. Rev. Environ. Sci. Technol., 25(4): 313–376.

CCME (2004) From source to tap: Guidance on the multi-barrier approach to safe drinking water. Canadian Council of Ministers of the Environment, Winnipeg, Manitoba. Available at www.ccme.ca/assets/pdf/mba_guidance_doc_e.pdfCCME (2004).

Chiew, A.L., Page, C.B., Clancy, D., Mostafa, A., Roberts, M.S. and Isbister, G.K. (2018). 2-Methyl-4-chlorophenoxyacetic acid (MCPA) and bromoxynil herbicide ingestion. Clin Toxicol (Phila). 56(5): 377–380.

Chowdhury, Z.K., Summers, R.S., Westerhoff, G.P., Leto, B.J., Nowack, K.O. and Corwin, C.J. (2013). Activated carbon: Solutions for improving water quality. Passantino, L.B. (ed.). American Water Works Association. Denver, Colorado.

Coggon, D., Pannett, B., Winter, P., Acheson, E.D. and Bonsall, J. (1986). Mortality of workers exposed to 2-methyl-4-chlorophenoxyacetic acid. Scand. J. Work Environ. Health, 12: 448–454.

Donald, D.B., Cessna, A.J., Sverko, E. and Glozier, N.E. (2007). Pesticides in surface drinking-water supplies of the northern great plains. Environ. Health Perspect., 115(8): 1183–1191.

Donald, D.B., Cessna, A.J. and Farenhorst, A. (2018). Concentrations of Herbicides in Wetlands on Organic and Minimum-Tillage Farms. J. Environ. Qual. 47:1554–1563.

Elliott, B. (2005). Review of the genotoxicity of 4-chloro-2-methylphenoxyacetic acid. Mutagenesis, 20(1): 3–13.

Elo, H. (1976). Distribution and elimination of 2-methyl-4-chlorophenoxyacetic acid (MCPA) in male rats. Acta Pharmacol. Toxicol., 39(1): 58–64.

Environment and Climate Change Canada. (2015). Pesticides in the Nelson River watershed, 2006 to 2011. Environ. Clim. Change Canada. Available at https://www.canada.ca/en/environment-climatechange/services/freshwater-quality-monitoring/pesticides-research/nelson-river-watershed-2006-2011.html

Environment Canada (2011). Presence and Levels of Priority Pesticides in Selected Canadian Aquatic Ecosystems. Water Science and Technology Directorate. Cat. No.: En14–40/2011E-PDF.

Eriksson, M., Hardell, L., Berg, N.O., Möller, T. and Axelson, O. (1981). Soft-tissue sarcomas and exposure to chemical substances: a case-referent study. Br. J. Ind. Med., 38: 27–33.

Eriksson, M., Hardell, L. and Adami, H.-O. (1990). Exposure to dioxins as a risk factor for soft tissue sarcoma: a population-based case-control study. J. Natl. Cancer Inst., 82(6): 486–490.

Eriksson, M., Hardell, L., Carlberg, M. and Akerman, M. (2008). Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis. Int. J. Cancer: 123, 1657–1663.

Fini, M.N., Madsen, H. T. and Muff, J. (2019). The effect of water matrix, feed concentration and recovery on the rejection of pesticides using NF/RO membranes in water treatment. Sep. Purif. Technol., 215:521–527. DOI. 10.1016/j.seppur.2019.01.047.

Fjeldstad, P. and Wannag, A. (1977). Human urinary excretion of the herbicide 2-methyl-4-chlorophenoxyacetic acid. Scand. J. Work Environ. Health, 3: 100–103.

Flanagan, R.J., Meredith, T.J., Ruprah, M., Onyon, L.J. and Liddle, A. (1990). Alkaline diuresis for acute poisoning with chlorophenoxy herbicides and ioxynil. Lancet 335: 454–8.

Frick, E.A. and Dalton, M.S. (2005). Characterization of anthropogenic organic compounds in the source water and finished water for the City of Atlanta, October 2002–September 2004. Proceedings of the 2005 Georgia Water Resources Conference, April 25–27, 2005, University of Georgia, Athens, GA.

Gelbke, H.P. and Engelhardt, G. (1985a). Cytogenetic investigations in Chinese hamsters after a single oral administration of MCPA: bone marrow chromosome analysis. BASF Aktiengesellschaft, Ludwigshafen, Germany (Report No. 10M0046/8304) as cited in Elliott, 2005).

Gelbke, H.P. and Engelhardt, G. (1985b). Cytogenetic investigations in Chinese hamsters after a single oral administration of MCPA: sister chromatid exchange (SCE). BASF Aktiengesellschaft, Ludwigshafen, Germany (Report No. 16M0046/8305) as cited in Elliott, 2005).

Gelbke, H.P. and Engelhardt, G. (1985c). Cytogenetic investigations in Chinese hamsters after a single oral administration of MCPA: sister chromatid exchange (SCE). BASF Aktiengesellschaft, Ludwigshafen, Germany (Report No. 16M0046/8356) (as cited in Elliott, 2005).

Gelbke, H.P. and Engelhardt, G. (1985d). Cytogenetic investigations in Chinese hamsters after a single oral administration of MCPA: bone marrow chromosome analysis. BASF Aktiengesellschaft, Ludwigshafen, Germany (Report No. 10M0046/8367) (as cited in Elliott, 2005).

Gérard, M.-C. and Barthélemy, J. P. (2003). An assessment methodology for determining pesticides adsorption on granulated activated carbon. Biotechnol. Agron. Soc. Environ., 7(2): 79–85

Giroux, I. (2019). Présence de pesticides dans l'eau au Québec : Portrait et tendances dans les zones de maïs et de soya—2015 à 2017, Québec. Ministère de l'Environnement et de la Lutte contre les changements climatiques, Direction générale du suivi de l'état de l'environnement, 64 p. + 6 ann. Available at www.environnement.gouv.qc.ca/pesticides/mais_soya/portrait2015-2017/rapport-2015-2017.pdf

Gonzalez, S., Müller, J., Petrovic, M., Barceló, D. and Knepper, T.P. (2006). Biodegradation studies of selected priority acidic pesticides and diclofenac in different bioreactors. Environ. Pollut., 144(3): 926–932.

Gonzalez, A.J., Fortunato, M.S., Gallego, A. and Korol, S.E. (2017). Simultaneous biodegradation and detoxification of the herbicides 2,4-dichlorophenoxyacetic acid and 4-chloro-2-methylphenoxyacetic acid in a continuous biofilm reactor. Water Air Soil Pollut., 228(8).

Gustafson, D.K., Carr, K.H., Carson, D.B., Fuhrman, J.D., Hackett, A.G., Hoogheem, T.J., Snoeyink, V.L., Curry, M., Heijman, B., Chen, S., Herti, P. and van Wesenbeeck, I. (2003). Activated carbon adsorption of chloroacetanilide herbicides and their degradation products from surface water supplies. J. Water Supply Res. Technol. AQUA, 52(6): 443–454.

Haist-Gulde, B. and Happel, O. (2012). Removal of pesticides and their ionic degradates by adsorptive processes. Report no. 4022. Water Research Foundation, Denver, Colorado.

Halevy, P. anderson, W.B. and Huck, P.M (2013). Pilot-scale investigation of advanced water treatment technologies in a eutrophic river in Southern Ontario. In: Proceedings of the American Water Works Association Water Quality Technology Conference, Long Beach, California. American Water Works Association, Denver, Colorado.

Haraux, E., Tourneux, P., Kouakam, C., Stephan-Blanchard, E., Boudailliez, B., Leke, A., Klein, C. and Chardon, K. (2018). Isolated hypospadias: The impact of prenatal exposure to pesticides, as determined by meconium analysis. Env. Int. 119: 20–25. Available at: https://reader.elsevier.com/reader/sd/pii/S0160412018302022?token=B660FFC6A31830155B80A0421D74E03905D96A045F4135B5ADCAE53C1050A7203AEC286EEB8B9D47E82F3BAD45A4CBF2&originRegion=us-east-1&originCreation=20210603135336

Hardell, L. and Eriksson, M. (1999). A case-control study of non-Hodgkin lymphoma and exposure to pesticides. Cancer, 85(6): 1353–1360.

Hardell, L. and Sandström, A. (1979). Case-control study: soft tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. Br. J. Cancer, 39: 711–717.

Hardell, L., Eriksson, M., Lenner, P. and Lundgren, E. (1981). Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case-control study. Br. J. Cancer, 43: 169–176.

Hardwick, T. (1999). 14C-MCPA: Absorption and excretion in the beagle dog. Unpublished report, Covenance Laboratories Ltd., North Yorkshire, UK, December (Final Report No. 729/D1141; MRID 4559301) (as cited in US EPA, 2003).

Hardwick, T. (2000). 14C-MCPA: Metabolite profiles in the beagle dog. Unpublished report, Covenance Laboratories Ltd., North Yorkshire, UK, June (Amended Final Report No. 729/201-D1141; MRID 4559302) (as cited in US EPA, 2003).

Harrison, I., Leader, R.U., Higgo, J.J.W. and Williams, G.M. (1998). A study of the degradation of phenoxyacid herbicides at different sites in a limestone aquifer. Chemosphere, 36(6): 1211–1232.

Health Canada. (2005). Internal report. MCPA use profile for Water Quality and Health Bureau, Health Canada. Pest Management Regulatory Agency, Health Canada, Ottawa, Ontario, September 7.

Health Canada (2006). Re-evaluation of the lawn and turf uses of the herbicide 4-chloro-2-methylphenoxy acetic acid (MCPA). Pest Management Regulatory Agency, Health Canada, Ottawa, Ontario, April 28 (PACR2006-05). Available at www.pmra-arla.gc.ca/english/pubs/pacr-e.html

Health Canada (2007). Proposed re-evaluation decision. The agricultural, forestry and industrial site uses of the herbicide (4-chloro-2-methylphenoxy)acetic acid (MCPA). Pest Management Regulatory Agency, Health Canada, Ottawa, Ontario. Consultation document of July 2007 (PRVD2007-01). Available at www.pmra-arla.gc.ca/english/pubs/prvd-e.html

Health Canada (2008). Re-evaluation decision. (4-chloro-2-methylphenoxy)acetic acid (MCPA). Pest Management Regulatory Agency, Health Canada, Ottawa, Ontario. (RVD2008-20). Available at https://www.canada.ca/en/health-canada/services/consumer-product-safety/reports-publications/pesticides-pest-management/decisions-updates.html#rvd-drv

Health Canada (2015/2016). Annual report: National Chemical Residue Monitoring Program and Chemistry Food Safety Oversight Program. Canadian Food Inspection Agency, Health Canada, Ottawa, Ontario.

Health Canada (2018). Pest control products sales report for 2018. Pest Management Regulatory Agency, Health Canada, Ottawa, Ontario. Available at https://www.canada.ca/en/health-canada/services/consumer-product-safety/reports-publications/pesticides-pest-management/corporate-plans-reports/pest-control-products-sales-report.html

Health Canada (2020). Personal Communication with the Health Evaluation Directorate, Pest Management Regulatory Agency (PMRA).

Heath Canada (2021). Canadian exposure factors used in human health risk assessments. Fact Sheet. Health Canada, Ottawa, Ontario. Available at: https://www.canada.ca/en/health-canada/services/chemical-substances/fact-sheets/canadian-exposure-factors-human-health-risk-assessments.html

Hellwig, J. and Hildebrand, B. (1993a). Study of the prenatal toxicity of MCPA-acid in rats after oral administration (gavage): Lab Project No. 30R0374/91096. Unpublished study prepared by BASF Aktiengesellshaft, Ludwigshafen, Germany. 302 pp. (as cited in US EPA, 2003).

Hellwig, J. and Hildebrand, B. (1993b). Study of the prenatal toxicity of MCPA-acid in rabbits after oral administration (gavage): Lab Project No. 40R0374/91095. Unpublished study prepared by BASF Aktiengesellshaft, Ludwigshafen, Germany. 230 pp. (as cited in US EPA, 2003).

Hofman, J.A.M.H., Beerendonk, E.F., Folmer, H.C. and Kruithof, J.C. (1997). Removal of pesticides and other micropollutants with cellulose-acetate, polyamide and ultra-low pressure reverse osmosis membranes. Desalination, 113: 209–214.

Hollender, J., Zimmermann, S.G., Koepke, S., Krauss, M., McArdell, C.S., Ort, C., Singer, H., von Gunten, U. and Siegrist, H. (2009). Elimination of organic micropollutants in a municipal wastewater treatment plant upgraded with a full-scale post-ozonation followed by sand filtration. Environ. Sci. Technol. 43, 7862–7869.

Holsing, G.C. and Kundzin, M. (1970). Final report: three-months dietary administration—rats. BASF Aktiengesellschaft, Ludwigshafen, FRG (as cited in US EPA, 1984; WHO, 2003).

HSDB (2010). Hazardous Substances Data Bank Number 1127. MCPA CAS # 94-74-6. US National Library of Medicine, Bethesda, MD. Last revision date: 2003-02-14. Available at https://pubchem.ncbi.nlm.nih.gov/source/hsdb/1127

Huntscha, S., Rodriguez Velosa, D.M., Schroth, M.H. and Hollender, J. (2013). Degradation of polar organic micropollutants during riverbank filtration: Complementary results from spatiotemporal sampling and push-pull tests. Environ. Sci. Technol., 47(20): 11512–11521.

Hu, J., Morita, T., Magara, Y. and Aizawa, T. (2000). Evaluation of reactivity of pesticides with ozone in water using the energies of frontier molecular orbitals. Wat. Res., 34(8): 2215–2222.

IARC. (1983). (4-Chloro-2-methylphenoxy)acetic acid (MCPA). In: Miscellaneous pesticides. International Agency for Research on Cancer, Lyon, France. IARC Monogr. Eval. Carcinog. Risks Hum., 30: 255–269.

Ignatowicz, K. (2009). Selection of sorbent for removing pesticides during water treatment. J. Hazard. Mater, 169(1–3), 953–957. doi:10.1016/j.jhazmat.2009.04.061

Ikehata, K. and El-Din, M.G. (2006). Aqueous pesticide degradation by hydrogen peroxide/ultraviolet irradiation and Fenton-type oxidation processes: A review. J. Environ. Eng. Sci. 5(2): 81–135.

Indigenous Services Canada. (2019). Personal communication with X. Redhead.

ISO (2000). Water quality—Determination of selected phenoxyalkanoic herbicides, including bentazones and hydroxybenzonitriles by gas chromatography and mass spectrometry after solid phase extraction and derivatization. ISO 15913:2000(E). International Organization for Standardization. Geneva, Switzerland.

Jahanshahi, M. and Stow, R. (1995). ^{14}C-MCPA: absorption, distribution, metabolism and excretion in the rats. Unpublished report, Corning Hazelton (Europe), North Yorkshire, UK, June 20 (Laboratory Report No. 1149/5-1011; MRID 43755202) (as cited in US EPA, 2003).

Jayakody, N., Harris, E.C. and Coggan, D. (2015). Phenoxy herbicides, soft tissue sarcoma and non-Hodgkins lymphoma: a systematic review of evidence from cohort and case-control studies. Br. Med. Bull. 114(1): 75–94. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4548713/pdf/emss-64740.pdf

Johnson, J.R.M. and Koumides, O. (1965). A further case of MCPA poisoning. Br. Med. J., 2: 629–630.

Jones, D.J.R., Knight, A.G. and Smith, A.J. (1967). Attempted suicide with herbicide containing MCPA. Arch. Environ. Health, 14: 363–367.

Jones, E., Kitching, J. anderson, A. and Dawe, I.S. (1992). Ames Salmonella typhimurium bacterial reverse mutation assay on MCPA-DMAS. Huntingdon Research Centre Ltd., Huntingdon, UK (HRC Study No. JEL/26/921053) (as cited in Cal-EPA, 2000).

Jones, E., Kitching, J., Anderson, A. and Dawe, I.S. (1993a). Ames Salmonella typhimurium bacterial reverse mutation assay on MCPA acid. Huntingdon Research Centre Ltd., Huntingdon, UK (HRC Study No. JEL 26/920957) (as cited in Cal-EPA, 2000).

Jones, E., Kitching, J., Anderson, A., and Dawe, I.S. (1993b). Ames Salmonella typhimurium bacterial reverse mutation assay on MCPA-2-EHE. Unpublished study prepared by Huntingdon Research Centre Ltd., Huntingdon, UK (Report No. JEL 25/921054) (as cited in US EPA, 2003).

Juhler, R.K., Sorensen, S.R. and Larsen, L. (2001). Analysing transformation products of herbicide residues in environmental samples. Water Res., 35(6): 1371–1378.

Kappas, A. (1988). On the mutagenic and recombinogenic activity of certain herbicides in *Salmonella typhimurium* and in *Aspergillus nidulans*. Mutat. Res., 204: 615–621 (as cited in Elliott, 2005).

Kelly, J., Morrison, G., Skillen, N., Manesiotis, P. and Robertson, P.K.J. (2019). An investigation of the role of pH in the rapid photocatalytic degradation of MCPA and its primary intermediate by low-power UV LED irradiation. Chem. Eng. J., 359: 112–118.

Kim, T.Y., Park, S.S., Kim, S.J. and Cho, S.Y. (2008). Separation characteristics of some phenoxy herbicides from aqueous solution. Adsorption, 14(4–5), 611.

Kirsch, P. (1985a). Study of the toxicity of MCPA in mice after 4 weeks administration in the diet. Unpublished study, Department of Toxicology, BASF Aktiengesellschaft, Ludwigshafen, FRG, April 4 (Laboratory Study No. 50S0046; MRID 00165470) (as cited in US EPA, 2003).

Kirsch, P. (1985b). Report on the study of the toxicity of MCPA in rats after 3-months administration in the diet. Department of Toxicology, BASF Aktiengesellschaft, Ludwigshafen, FRG, April 1 (Laboratory Study No. 3150046/8302; MRID 00165471; microfiche) (as cited in US EPA, 2003).

Kirsch, P. (1986) Report: Study on the Chronic Toxicity and Oncogenic Potential of MCPA in Rats: Final Report: Project No. 71S0046/8345. Unpublished study prepared by BASF Ag. 2026 p (as cited in US EPA, 2004d).

Kirsch, P. (1988). Study on the chronic toxicity and oncogenic potential of MCPA in rats. Unpublished report, Department of Toxicology, BASF Aktiengesellschaft, Ludwigshafen/Rhein, FRG, May 18 (Study No. 71S0045/8345; MRID No. 40634101) (as cited in Bellet et al., 1999 and US EPA, 2003).

Kolmodin-Hedman, B., Höglund, S. and Åkerblom, M. (1983a). Studies on phenoxy acid herbicides. I. Field study. Occupational exposure to phenoxy acid herbicides (MCPA, dichlorprop, mecoprop and 2,4-D) in agriculture. Arch. Toxicol., 54(4): 257–265.

Kolmodin-Hedman, B., Hoglund, S., Swensson, A., Akerblom, M. (1983b), Studies on phenoxy acid herbicides. II. Oral and dermal uptake and elimination in urine of MCPA in humans, Arch. Toxicol., 54(4), 267–273.

Kogevinas, M., Kauppinen, T., Winkelmann, R., Becher, H., Bertazzi, P.A., Bueno-de-Mesquita, H.B., Coggon, D., Green, L., Johnson, E., Littorin, M., Lynge, E., Marlow, D.A., Mathews, J.D., Neuberger, M., Benn, T., Pannett, B., Pearce, N. and Saracci, R. (1995). Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: 2 nested case-control studies. Epidemiology, 6: 396–402.

Košutić, K. and Kunst, B. (2002). Removal of organics from aqueous solutions by commercial RO and NF membranes of characterized porosities. Desalination, 142(1), 47–56.

Krishnan, K. et Carrier, R.. (2013). The use of exposure source allocation factor in the risk assessment of drinking-water contaminants. J. Toxicol. Environ. Health B Crit. Rev., 16(1):39–51.

Kruger, U.S., Johnsen, A.R., Burmolle, M., Aamand, J. and Sorensen, S.R. (2015). The potential for bioaugmentation of sand filter materials from waterworks using bacterial cultures degrading 4-chloro-2-methylphenoxyacetic acid. Pest Manag. Sci., 71(2): 257–265.

Kuehn, W. and Mueller, U. (2000). Riverbank filtration: an overview. J. Am. Water Works Assoc., 92(12): 60–69.

Kuhborth, B., Deckaidt, K., Mirea, D., Schilling, K. and Hildebrand, B. (1988). Study on the oncogenic potential of MCPA in mice. Unpublished report, BASF Aktiengesellschaft, Ludwigshafen, FRG, July 26 (Project No. 80S0046/8358; MRID No. 40792301) (as cited in Bellet et al., 1999 and US EPA, 2003).

Lambert, S.D. and Graham, N.J. (1995). A comparative evaluation of the effectiveness of potable water filtration processes. Aqua London, 44(1): 38–51.

Lappin, G.J., Hardwick, T.D., Stow, R., Pigott, G.H. and van Ravenzwaay, B. (2002). Absorption, metabolism and excretion of 4-chloro-2-methylphenoxyacetic acid (MCPA) in rat and dog. Xenobiotica, 32(2): 153–163.

Lekkerkerker-Teunissen, K., Knol, A.H., Derks, J.G., Heringa, M.B., Houtman, C.J., Hofman-Caris, C.H.M., Beerendonk, E.F., Reus, A., Verberk, J.Q.J.C. and van Dijk, J.C. (2013). Pilot plant results with three different types of UV lamps for advanced oxidation. Ozone Sci. Eng., 35(1):38–48.

Li, W., Zhao, Y., Yan, X., Duan, J., Saint, C.P. and Beecham, S. (2019). Transformation pathway and toxicity assessment of glyphosate in aqueous solution during UV photolysis and photocatalysis. Chemosphere, 234: 204–214.

Linnainmaa, K. (1984). Induction of sister chromatid exchanges among workers occupationally exposed to phenoxy acid herbicides 2,4-D and MCPA. Teratog. Carcinog. Mutagen., 3: 269–279.

Lynge, E. (1993). Cancer in phenoxy herbicide manufacturing workers in Denmark, 1947-87—an update. Cancer Causes Control, 4: 261–272.

Lynge, E. (1998). Cancer incidence in Danish phenoxy herbicide workers, 1947–1993. Environ. Health Perspect., 106(2): 683–688.

MacKenzie, K.M. (1986). Two-generation reproductive study with MCPA in rats. Final report. Unpublished study, Hazelton Laboratories America Inc., Madison, WI, November 3 (Laboratory Study No. 6148-100; MRID 40041701) (as cited in Bellet et al., 2001 and US EPA, 2003).

Manitoba Sustainable Development. (2019). Personal communication with D. Coulibaly, Water Quality Management Section.

Mannetje, A.'t., McLean, D., Cheng, S., Boffetta, P., Colin, D. and Pearce, N. (2005). Mortality in New Zealand workers exposed to phenoxy herbicides and dioxins. Occup. Environ. Med., 62: 34–40.

Matamoros, V. and Franco, J. (2018). Assessing the use of sand, peat soil and pine bark for the attenuation of polar pesticides from agricultural run-off: A bench-scale column experiment. Environ. Sci. Pollut. Research, 25(21): 20640–20647.

McManus, S.L., Coxon, C.E., Mellander, P.E., Danaher, M. and Richards, K.G. (2017). Hydrogeological characteristics influencing the occurrence of pesticides and pesticide metabolites in groundwater across the republic of Ireland. Sci. Total Environ., (601–602): 594–602.

Meijers, R.T., Oderwald-Muller, E.J., Nuhn, P.A.N.M. and Kruithof, J.C. (1995). Degradation of pesticides by ozonation and advanced oxidation. Ozone Sci. Eng., 17(6): 673–686.

Mellert W. et al. (1994a). MCPA-DMA salt—Subchronic oral dietary toxicity and neurotoxicity study in Wistar rats. Unpublished report no. 50C0189/91140 by BASF Aktiengesellschaft, Ludwigshafen, Germany. Submitted to WHO by the MCPA Task Force Three (as cited in WHO, 2016.

Mellert W. et al. (1994b). MCPA-acid—Subchronic oral dietary toxicity and neurotoxicity study in Wistar rats. Unpublished report no. 50C0374/91133 by BASF Aktiengesellschaft, Ludwigshafen, Germany. Submitted to WHO by the MCPA Task Force Three (as cited in US EPA, 2003; WHO, 2016.

Mellert W. et al. (1994c). MCPA-2-EH-ester—Subchronic oral dietary toxicity and neurotoxicity study in Wistar rats. Unpublished report no. 50C0385/91141 by BASF Aktiengesellschaft, Ludwigshafen, Germany. Submitted to WHO by the MCPA Task Force Three (as cited in WHO, 2016.

Mersch-Sundermann, V., Hofmeister, A., Mueller, G. and Hof, H. (1989). [Examination of mutagenicity of organic microcontaminations of the environment. III. Communication: The mutagenicity of selected herbicides and insecticides with the SOS chromotest.] Zentrabl. Hyg., 189: 135–146 (in German).

Miltner, R.J., Baker, D.B., Speth, T.F. and Fronk, C.A. (1989). Treatment of Seasonal Pesticides in Surface Waters. J. Am. Water Works Assoc., 81(1): 43–52.

Ministère de l'Environnement et de la Lutte contre les changements climatiques (2019). Personal communication with P. Cantin.

Mohamed, F., Endre, Z.H. and Buckley, N.A. (2015). Role of biomarkers of nephrotoxic acute kidney injury in deliberate poisoning and envenomation in less developed countries. Br J Clin Pharmacol. 80(1): 3–19.

Moriya, M., Ohta, T., Watanabe, K., Miyazawa, T., Kato, K. and Shirasu, Y. (1983). Further mutagenicity studies on pesticides in bacterial reversion assay system. Mutat. Res., 116: 185–216.

Morton, P.A., Fenell, C., Cassidy, R., Doody, D., Fenton, O., Mellander, P-E. and Jordan, P. (2019). A review of the pesticide MCPA in the land-water environment and emerging research needs. WIREs Water. Available at https://onlinelibrary.wiley.com/doi/abs/10.1002/wat2.1402

Munira, S., Farenhorst, A., Sapkota, K., Nilsson, D. and Sheedy, C. (2018). Auxin Herbicides and Pesticide Mixtures in Groundwater of a Canadian Prairie Province. J. Environ. Qual. 47:1462–1467.

Murphy, C. and Mutch, J. (2005). Unpublished water monitoring data from the Environment Canada Atlantic Region. Collected as part of the Environment Canada Pesticide Science Fund (as cited in PMRA, 2005d).

Murray, J., Andersen, D. and Kent, R. (2004). Presence, levels and relative risks of priority pesticides in selected Canadian aquatic ecosystems. An Environment Canada Pesticides Science Fund Project. Year 1 (2003–2004) annual report. National Water Research Institute, Environment Canada, Burlington, Ontario.

Mustonen, R., Elovaara, E., Zitting, A., Linnainmaa, A. and Vainio, H. (1989). Effects of commercial chlorophenolate, 2,3,7,8-TCDD, and pure phenoxyacetic acids on hepatic peroxisome proliferation, xenobiotic metabolism and sister chromatid exchange in the rat. Arch. Toxicol., 63: 203–208.

Muszynski, P., Brodowska, M.S. and Paszko, T. (2020). Occurrence and transformation of phenoxy acids in aquatic environment and photochemical methods of their removal: A review. Environ. Sci. Pollut. R. 27:1276–1293.

New Brunswick Department of Health (2005). Personal communication from R. Albert.

New Brunswick Department of Environment and Local Government. (2019). Personal communication with K. Gould.

Newfoundland and Labrador Municipal Affairs and Environment. (2019). Personal communication with H. Khan.

NHMRC and NRMMC (2011). Australian Drinking Water Guidelines Paper 6 National Water Quality Management Strategy. National Health and Medical Research Council, National Resource Management Ministerial Council, Commonwealth of Australia, Canberra. Available at https://www.Nhmrc.Gov.Au/about-us/Publications/Australian-Drinking-Water-Guidelines.

Nishimura, N., Nishimura, H. and Oshima, H. (1982). Survey on mutagenicity of pesticides by the *Salmonella*-microsome test. J. Aichi Med. Univ. Assoc., 10: 305–312 (as cited in Elliott, 2005).

Nova Scotia Environment. (2019). Personal communication with A. Polegato.

NSF/ANSI (2020a). NSF International/American National Standards Institute Standard 53: Drinking water treatment units-health effects. NSF International, Ann Arbor, Michigan.

NSF/ANSI (2020b). NSF International/American National Standards Institute Standard 58: Reverse osmosis drinking water treatment systems. NSF International, Ann Arbor, Michigan.

Oberleitner, D., Schulz, W., Bergmann, A. and Achten, C. (2020). Impact of seasonality, redox conditions, travel distances and initial concentrations on micropollutant removal during riverbank filtration at four sites. Chemosphere, 250: 1–10.

Ontario Ministry of the Environment, Conservation and Parks .(2020). Personal communication with S. Deshpande.

Pahwa, M., Harris, S.A., Hohenadel, K., McLaughlin, J.R., Spinelli, J.J., Pahwa, P., Dosman, J.A. and Blair, A. (2012). Pesticide use, immunologic conditions, and risk of non-Hodgkin lymphoma in Canadian men in six provinces. Int. J. Cancer: 131, 2650–2659.

Palva, H.L.A., Koivisto, O. and Palva, I.P. (1975). Aplastic anaemia after exposure to a weed killer, 2-methyl-4-chlorophenoxyacetic acid. Acta Haematol., 53: 105–108.

PEI Department of Communities, Land and Environment (2019). Personal communication with G. Somers.

Petrie, A.J., Melvin, M.A., Plane, N.H. and Littlejohn, J.W. (1993). The effectiveness of water treatment processes for removal of herbicides. Sci. Total Environ., 135(1–3), 161–169.

Piet, G.J. and Zoeteman, B.C.J. (1980). Organic water quality changes during sand bank and dune filtration of surface waters in the Netherlands. J. Am. Water Works Assoc., 72(7): 400–404.

Plakas, K.V. and Karabelas, A.J. (2012). Removal of pesticides from water by NF and RO membranes—a review. Desalination, 287: 255–265.

Popham, R.D. and Davis, D.M. (1964). A case of MCPA poisoning. Br. Med. J., 1: 677–678.

Proudlock, R.J., Taylor, K., Anderson, A. and Dawe, I.S. (1993a). MCPA DMAS micronucleus test. Huntingdon Research Centre Ltd., Huntingdon, UK, April 2 (HRC Study No. JEL 33/921197) (as cited in Cal-EPA, 2000; Elliott, 2005).

Proudlock, R.J., Elmore, E.A., Anderson, A. and Dawe, I.S. (1993b). MCPA acid micronucleus test. Huntingdon Research Centre Ltd., Huntingdon, UK, April 23 (HRC Study No. JEL 35/921198) (as cited in Cal-EPA, 2000; Elliott, 2005).

Proudlock, R.J., Goodenough, A.J., Anderson, A. and Dawe, I.S. (1993c). MCPA 2-EHE micronucleus test. Huntingdon Research Centre Ltd., Huntingdon, UK, April 22 (HRC Study No. JEL 33/921197) (as cited in Elliott, 2005).

Randtke, S.J. (1988). Organic contaminant removal by coagulation and related process combinations. J. Am. Water Works Assoc., 80(5): 40–56.

Räsänen, L., Hattula, M.L. and Arstila, A.U. (1977). The mutagenicity of MCPA and it soil metabolites, chlorinated phenols, catechols and some widely used slimicides in Finland. Bull. Environ. Contam. Toxicol., 18(5): 565–571.

Ray, C., Grischek, T., Schubert, J., Wang, J.Z. and Speth, T.F. (2002). A perspective of riverbank filtration. J. Am. Water Works Assoc., 94(4): 149–160.

Reynolds, G., Graham, N. and Perry, R. (1989). Aqueous ozonation of pesticides: A review. Ozone: Sci. Eng., 11(4): 339–382.

Roberts, D.M., Seneviratne, R., Mohammed, F., Patel, R., Senarathna, L., Hittarage, A., Buckley, N.A., Dawson, A.H. and Eddleston, M. (2005). Intentional self-poisoning with the chlorophenoxy herbicide 4-chloro-2-methylphenoxyacetic acid (MCPA). Ann. Emerg. Med. 46: 275–84.

Roberts, D.M., Dawson, A.H., Senarathna, L., Mohamed, F., Cheng, R., Eaglesham, G. and Buckley, N.A. (2011). Toxicokinetics, including saturable protein binding, of 4-chloro-2-methyl phenoxyacetic acid (MCPA) in patients with acute poisoning. Toxicol. Lett. 2011; 201: 270–6.

Rowe, V.K. and Hymas, T.A. (1954). Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. Am. J. Vet. Res., 15: 622–629.

RTECS. (2005). Registry of Toxic Effects of Chemical Substances: MCPA. CAS # 94-74-6. RTECS No.: AG1575000. Chemical profiles created by Canadian Centre for Occupational Health and Safety, Hamilton, Ontario.

Samuelsen, E.D., Badawi, N., Nybroe, O., Sørensen, S.R. and Aamand, J. (2017). Adhesion to sand and ability to mineralise low pesticide concentrations are required for efficient bioaugmentation of flow-through sand filters. Appl. Microbiol. Biotechnol., 101(1): 411–421.

Saracci, R., Kogevinas, M., Bertazzi, P.-A., Bueno de Mesquita, B.H., Coggon, D., Green, L.M., Kauppinen, T., L'Abbé, K.A., Littorin, M., Lynge, E., Mathews, J.D., Neuberger, M., Osman, J., Pearce, N. and Winkelmann, R. (1991). Cancer mortality in workers exposed to chlorophenoxy herbicides and chlorophenols. Lancet, 338(8774): 1027–1032.

Saskatchewan Water Security Agency (2019). Personal communication with S. Ferris.

Sattar, M.A. and Paasivirta, J. (1980). Fate of chlorophenoxy acetic acids in acid soil. Chemosphere, 9: 745–752.

Schippers, J.C., Kruithof, J.C., Nederlof, M.M., Hofman, J.A.M.H. and Taylor, J.S. (2004). Integrated membrane systems. American Water Works Association Research Foundation and American Water Works Association, Denver, CO (Report No. 90899).

Smeets, P.W.M.H., Medema, G.J. and van Dijk, J.C. (2009). The Dutch secret: how to provide safe drinking water without chlorine in the Netherlands. Drinking Water Engineering and Science, 2: 1–14.

Smith, A.E. and Hayden, B.J. (1981). Relative persistence of MCPA, MCPB and mecoprop in Saskatchewan soils, and the identification of MCPA in MCPB-treated soils. Weed Sci., 21: 179–183.

Soderquist, C.J. and Crosby, D.G. (1975). Dissipation of 4-chloro-2-methylphenoxyacetic acid (MCPA) in a rice field. Pestic. Sci., 6: 17–33.

Speth, T.F. and Adams, J.Q. (1993). GAC and air stripping design support for the Safe Drinking Water Act, strategies and technologies for meeting SDWA requirements. Clark, R. and S. Summers, Eds., Lewis Publishers, Ann Arbor, MI, pp. 47–89.

Speth, T.F. and Miltner, R.J. (1998). Technical note: adsorption capacity of GAC for synthetic organics. Jour. AWWA. 90(4): 171–174.

Sørensen, S.R., Schultz, A., Jacogsen, O.S. and Aamand, J. (2006). Sorption, desorption and mineralisation of the herbicides glyphosate and MCPA in samples from two Danish soil and subsurface profiles. Environ. Pollut. 141, 184–194.

Struger, J., Fletcher, T., Martos, P., Ripley, B. and Gris, G. (2002). Pesticide concentrations in the Don and Humber river watersheds (1998–2000). Interim report. Environment Canada and the Ontario Ministry of the Environment, December (as cited in PMRA, 2005c).

Struger, J., L'Italien, S. and Sverko, E. (2004). In-use pesticide concentrations in surface waters of the Laurentian Great Lakes, 1994–2000. J. Great Lakes Res., 30(3): 435–450.

Struger, J., Marvin, C., Sveko, E., Dove, A., Palace, V., Todd, A. and Fletcher, T. (2005). Unpublished water monitoring data from the Environment Canada Ontario Region. Collected as part of the Environment Canada Pesticide Science Fund (as cited in PMRA, 2005d).

Struif, B., Weil, L. and Quentin, K. (1978). Verhalten herbizider Phenoxy-alkan-carbonsäuren bei der Wasseraufbereitung mit Ozon. Z.f. Wasser-und Abwasserforschung 11. Jahrgang Nr.3/4/78 (as cited in Reynold et al., 1989)

Summers, R.S., Knappe, D.R.U. and Snoeyink, V.L. (2010). Chapter 14: Adsorption of organic compounds by activated carbon. In: Edzwald, J.K. (ed.), Water quality and treatment: A handbook on drinking water. 6th edition. McGraw-Hill, New York, NY.

Symons, J.M., Bradley Jr., L.C. and Cleveland, T.C. (2000). The drinking water dictionary. American Water Works Association, Denver, Colorado.

Takayasu, T., Hayashi, T., Ishida, Y., Nosaka, M., Mizunuma, S., Miyashita, T., Kawaguchi, M., Kimura, A. and Kondo, T. (2008). A fatal intoxication from ingestion of 2-methyl-4-chlorophenoxyacetic acid (MCPA). J Anal Toxicol., 32(2):187–91.

Taylor, J.S. (Ed.). (2000). Flat sheet, bench and pilot testing for pesticide removal using reverse osmosis. American Water Works Association, Denver, CO.

Timchalk, C. (2004). Comparative inter-species pharmacokinetics of phenoxyacetic acid herbicides and related organic acids. Evidence that the dog is not a relevant species for evaluation of human health risk. Toxicology, 200(1): 1–19.

Timonen, T.T. and Palva, I.P. (1980). Acute leukemia after exposure to a weed killer, 2-methyl-4-chlorophenoxyacetic acid. Acta Haematol., 63: 170–171.

Tuominen, T., Pasternak, J., Sekela, M., Ryan, A., Hii, B., Strub, R., McPherson, B., Wan, M., Kuo, J. and Hamilton, C. (2005). Unpublished water monitoring data from the Environment Canada Pacific and Yukon Region. Collected as part of the Environment Canada Pesticide Science Fund (as cited in PMRA, 2005d).

University of Hertfordshire (2019). MCPA. Pesticide Properties Database (PPDB). Available at http://sitem.herts.ac.uk/aeru/ppdb/en/Reports/427.htm

USDA (2001) Agricultural Research Service (ARS) Pesticide Properties Database on MCPA (94-74-6). US Department of Agriculture. MCPA last updated May 1999.

US EPA (1984). Health and environmental effects profile for MCPA and MCPB. Environmental Criteria and Assessment Office, Office of Research and Development, US Environmental Protection Agency (NTIS/PB88-162391; microfiche).

US EPA (1990). Registration on standard for pesticide products containing MCPA as the active ingredient. Government Reports Announcement and Index (GRA & I), Issue 07. US Environmental Protection Agency (NTIS/PB90-146556).

US EPA (1992). Method 555: Determination of chlorinated acids in water by high performance liquid chromatography with a photodiode array ultraviolet detector, revision 1.0. Environmental Monitoring Systems Laboratory, Office of Research and Development, United States Environmental Protection Agency, Cincinnati, OH.

US EPA (2003). MCPA: Toxicology chapter for RED (Reregistration Eligibility Decision). Office of Prevention, Pesticides, and Toxic Substances, US Environmental Protection Agency, Washington, DC, July 1 (Document ID: EPA-HQ-OPP-2004-0239-0019).

US EPA (2004a). Environmental Fate and Effects Division's risk assessment for the reregistration eligibility document for 2-methyl-4-chlorophenoxyacetic acid (MCPA). US Environmental Protection Agency (Document ID: EPA-HQ-OPP-2004-0239-0006).

US EPA (2004b). R.E.D. Facts: MCPA. Office of Prevention, Pesticides and Toxic Substances, US Environmental Protection Agency, September (7508C; Document ID: EPA-HQ-OPP-2004-0239-0002).

US EPA (2004c). MCPA summary. US Environmental Protection Agency (Document ID: EPA-HQ-OPP-2004-0156-0003).

US EPA (2004d). Reregistration Eligibility Decision for MCPA (2-methyl-4-chlorophenoxyacetic acid). List A Case 0017. Office of Prevention, Pesticides, and Toxic Substances, US Environmental Protection Agency, Washington, DC, September 30 (Document ID: EPA-HQ-OPP-2004-0239-0003).

US EPA (2004e). MCPA—Corrected first report of the Hazard Identification Assessment Review Committee. Office of Prevention, Pesticides, and Toxic Substances, US Environmental Protection Agency, Washington, DC (Document ID: EPA-HQ-OPP-2004-0239-0018).

US EPA (2011). Finalization of guidance on incorporation of water treatment effects on pesticide removal and transformation in drinking water exposure assessments.

US EPA (2018). 2018 Edition of the Drinking Water Standards and Health Advisories. Office of Water US Environmental Protection Agency Washington, DC. Available at https://www.epa.gov/sites/production/files/2018-03/documents/dwtable2018.pdf

USGS (1996). Method O-1131-95—Determination of pesticides in water by CarboPak-B solid-phase extraction and HPLC. United States Geological Survey, National Water Quality Laboratory.

USGS (2001). Method O-2060-01—Determination of pesticides in water by graphitized carbon-based solid-phase extraction and HPLC-MS. United States Geological Survey, National Water Quality Laboratory.

Van der Aa, L.T.J., Kolpa, R.J., Rietveld, L.C. and van Dijk, J.C. (2012). Improved removal of pesticides in biological granular activated carbon filters by pre-oxidation of natural organic matter. J. Water Supply Res. Technol. AQUA, 61(3): 153–163.

Van der Bruggen, B. and Vandecasteele, C. (2003). Removal of pollutants from surface water and groundwater by nanofiltration: overview of possible applications in drinking water industry. Environ. Pollut., 122(3): 435–445.

Vandermaesen, J., Horemans, B., Degryse, J., Boonen, J., Walravens, E. and Springael, D. (2019). The pesticide mineralization capacity in sand filter units of drinking water treatment plants (DWTP): Consistency in time and relationship with intake water and sand filter characteristics. Chemosphere, 228: 427–436.

Van Ravenzwaay, B., Pigott, G. and Leibold, E. (2004). Absorption, distribution, metabolism and excretion of 4-chloro-2-methylphenoxyacetic acid (MCPA) in rats. Food Chem. Toxicol., 42(1): 115–125.

Verstraeten, I.M. and Heberer, T. (2002). Organic chemical removal issues. Chapter 17 in: Riverbank filtration: Improving source-water quality. Ray, C., Melin, G. and Linsky, R.B. (eds.). Springer, Kluwer Academic Publishers. Netherlands. pp. 321–330.

Vineis, P., Terracini, B., Ciccone, G., Cignetti, A., Colombo, E., Donna, A., Maffi, D.A., Pisa, R., Ricci, P., Zanini, E. and Comba, P. (1986). Phenoxy herbicides and soft-tissue sarcomas in female rice-weeders: a population-based case control study. Scand. J. Work Environ. Health, 13(1): 9–17.

Vineis, P., Faggiano, F., Tedeschi, M. and Ciccone, G. (1991). Incidence rates of lymphomas and soft-tissue sarcomas and environmental measurements of phenoxy herbicides. J. Natl. Cancer Inst., 83(5): 362–363.

Waite, D.T., Bailey, P., Sproull, J.F., Quiring, D.V., Chau, D.F., Bailey, J. and Cessna, A.J. (2005). Atmospheric concentrations and dry and wet deposits of some herbicides currently used on the Canadian Prairies. Chemosphere, 58: 693–703.

Weed Science Society of America. (1989). Herbicide handbook. 6th edition. Weed Science Society of America, Champaign, IL. pp. 171–173.

WHO. (2011). Guidelines for drinking-water quality. Fourth edition. World Health Organization, Geneva, Switzerland. Available at www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/

WHO (2012). Water safety planning for small community water supplies. World Health Organization, Geneva, Switzerland. Available at www.who.int/water_sanitation_health/publications/small-commwater_supplies/en/

WHO (2016). MCPA in drinking-water. Background document for development of WHO Guidelines for drinking-water quality. World Health Organization, Geneva, Switzerland. Available at https://www.who.int/water_sanitation_health/water-quality/guidelines/chemicals/mcpa-background-jan17.pdf

Wiklund, K., Dich, J. and Holm, L.-E. (1987). Risk of malignant lymphoma in Swedish pesticide appliers. Br. J. Cancer, 56: 505–508.

Wiklund, K., Dich, J. and Holm, L.-E. (1988). Soft tissue sarcoma risk in Swedish licensed pesticide applicators. J. Occup. Med., 30(10): 801–804.

Wiklund, K., Dich, J. and Holm, L.-E. (1989). Risk of soft tissue sarcoma, Hodgkin's disease and non-Hodgkin lymphoma among Swedish licensed pesticide applicators. Chemosphere, 18(1–6): 395–400.

Worthing, C.R. and Hance, R.J. (eds.). (1991). The pesticide manual. 9th edition. British Crop Protection Council, Farnham, UK.

Woudneh, M.B., Lloyd, B.J. and Stevenson, D. (1996). Removal of herbicides by biological filters. In: Advances in slow sand/alternative biofiltration. Graham, N. and Collins, M.R. (eds.). John Wiley & Sons. Chichester, United Kingdom. pp. 211–221.

Wunnapuk, K., Liu, X., Gobe, G.C., Endre, Z.H., Peake, P.W., Grice, J.E., Roberts, M.S. and Buckley, N.A. (2014). Kidney biomarkers in MCPA-induced acute kidney injury in rats: Reduced clearance enhances early biomarker performance. Toxicol Lett. 225: 467–78.

Yao, Y., Tuduri, L., Harner, T., Blanchard, P., Waite, D., Poissant, L., Murphy, C., Belzer, W., Aulagnier, F., Li, Y.-F. and Sverko, E. (2006). Spatial and temporal distribution of pesticide air concentrations in Canadian agricultural regions. Atmos Env 40: 4339–4351.

Yukon Environmental Health Services (2019). Personal communication with P. Brooks

Zertal, A., Sehili, T. and Boule, P. (2001). Photochemical behaviour of 4-chloro-2-methylphenoxyacetic acid: Influence of pH and irradiation wavelength. J. Photochem. Photobiol. A., 146(1–2): 37–48.

Zetterberg, G. (1978). Genetic effects of phenoxy acids on microorganisms. Ecol. Bull., 27: 193–204.

Zetterberg, G. (1979). Mechanism of the lethal and mutagenic effects of phenoxyacetic acids in Saccharomyces cerevisiae. Mutat. Res., 60: 291–300.

Zychlinski, L. and Zolnierowicz, S (1990). Comparison of uncoupling activities of chlorophenoxy herbicides in rat liver mitochondria. Toxicol. Lett. 52: 25–34.

APPENDIX A: LIST OF ABBREVIATIONS

2,4-D	2,4-dichlorophenoxyacetic acid
2-M4CP	2-methyl-4-chlorophenol
2,4,5-T	2,4,5-trichlorophenoxyacetic acid
ADI	Acceptable daily intake
ALT	Alanine aminotransferase
ANSI	American National Standards Institute
AOP	Advanced oxidation process
BW	Body weight
CAS RN	Chemical Abstracts Service registry number
DNA	Deoxyribonucleic acid
DOC	Dissolved organic carbon
DWSP	Drinking Water Surveillance Program
DWTP	Drinking water treatment plant
EBCT	Empty bed contact time
ECD	Electron capture detection
EPA	Environmental Protection Agency (United States)
EU	European Union
FBBR	Fixed-bed bioreactor
FNIHB	First Nations and Inuit Health Branch
GAC	Granulated activated carbon
HBV	Health-based value
HLR	Hydraulic loading rate

HMCPA	4-chloro-2-hydroxymethyl-phenoxyacetic acid
HPLC	High performance liquid chromatography
IARC	International Agency for Research on Cancer
K	Freundlich coefficient
KIM-1	Kidney injury molecule
Log Kow	Octanol:water partition coefficient
LOQ	Limit of quantitation
LP	Low pressure
MAC	Maximum acceptable concentration
MCL	Maximum contaminant level
MCPA	2-methyl-4-chlorophenoxyacetic acid
MCPA-DMAS	MCPA dimethylamine salt
MCPA-EHE	MCPA 2-ethylhexyl ester
MDL	Method detection limit
MP	Medium pressure
MRL	Method reporting limit
MTD	Maximum tolerated dose
MWCO	Molecular weight cut-off
NF	Nanofiltration
NHL	Non-Hodgkin's lymphoma
NOAEL	No-observed-adverse-effect level
NOEL	No-observed-effect level
NOM	Natural organic matter
NSF	NSF International

NTU	Nephelometric turbidity unit
PAC	Powdered activated carbon
PEI	Prince Edward Island
PMRA	Pest Management Regulatory Agency
RBF	Riverbank filtration
RfD	Reference dose
RO	Reverse osmosis
SSC	Standards Council of Canada
STS	Soft tissue sarcoma
TOC	Total organic carbon
TFC	Thin film composite
ULP	Ultra-low pressure
US EPA	United States Environmental Protection Agency
UV	Ultraviolet
V spec	Specific throughput
WHO	World Health Organization

APPENDIX B: CANADIAN WATER QUALITY DATA

Table 18. Levels of MCPA in Canadian sources from the Environment Canada National Water Quality Surveillance Program (2003–2005)

Jurisdiction (year sampled)	No. detects/ samples	MDL (ng/L)	Range (ng/L)		25th percentile (ng/L)	Mean (ng/L)	Median (ng/L)	75th percentile (ng/L)
			Min	Max				
Tap Water								
AB, SK, MB—rural communities (2004–2005)	NA/28	-	0.58	865.00	-	96.50	-	-
Surface Water								
BC—Lower Fraser Valley and Okanagan Basin (2003–2005)	83/92	0.05	< 0.05	789	0.520	-	1.290	12.7
BC—Lower Fraser Valley (2003–2005)	-	-	0.08	789	-	-	-	-
ON (2003)	116/162	0.58	0.66	1230	0.29	-	1.94	4.69
ON (2004)	128/228	0.58	0.58	350	0.29	-	1.09	6.51
ON (2005)	71/183	0.58	1.04	69.1	-	-	-	-
QC (2003)	6/51	10	< 10	120	-	-	-	-
QC (2004)	11/70	6–10	< 6	110	-	-	-	-
QC (2005)	12/59	10	< 10	1200	-	-	-	-
NB (2003–2005)	1/57	100	-	-	-	-	-	-
PEI (2003–2005)	0/82	100	-	-	-	-	-	-
NS (2003–2005)	1/48	100–200	-	-	-	-	-	-

Jurisdiction (year sampled)	No. detects/ samples	MDL (ng/L)	Range (ng/L)		25th percentile (ng/L)	Mean (ng/L)	Median (ng/L)	75th percentile (ng/L)
			Min	Max				
Rivers								
AB, SK, MB—8 sites (2003)	59/64	0.58	< 0.58	176	5.74	-	11.95	23.85
Reservoir Water								
AB, SK, MB—15 sites (2003–2004)	204/206	0.58	< 0.58	374	13.80	-	27.45	68.10
Groundwater								
BC—Okanagan Basin (2003–2005)	-	-	0.11	0.31	-	-	-	-

MDL—method detection limit; NA—not available.

Note: Adapted from Environment Canada (2011)