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Diquat in Drinking Water

Guideline Technical Document
for Public Consultation

Consultation period ends
October 2nd, 2020

Canada

Purpose of consultation

This guideline technical document outlines the evaluation of the available information on diquat with the intent of updating the guideline value for diquat in drinking water. The purpose of this consultation is to solicit comments on the proposed guideline, on the approach used for its development, and on the potential impacts of implementing it.

The existing guideline technical document on diquat, developed in 1986, recommended a health-based value of 0.07 mg/L (70 µg/L), based on eye damage observed in rats.

This document proposes a maximum acceptable concentration (MAC) of 0.05 mg/L (50 µg/L) for diquat in drinking water, based on eye damage observed in beagle dogs.

This document is available for a 90-day public consultation period. Please send comments (with rationale, where required) to Health Canada:

HC.water-eau.SC@canada.ca
or
Water and Air Quality Bureau, Health Canada
269 Laurier Avenue West, A.L. 4903D
Ottawa, ON K1A 0K9

All comments must be received before October 2nd, 2020. Comments received as part of this consultation will be shared with members of the Federal-Provincial-Territorial Committee on Drinking Water (CDW), along with the name and affiliation of their author. Authors who do not want their name and affiliation shared with CDW members should provide a statement to this effect along with their comments.

It should be noted that this guideline technical document may be revised following the evaluation of comments received, and a drinking water guideline will be established, if required. This document should be considered as a draft for comment only.

Proposed guideline

A maximum acceptable concentration (MAC) of 0.05 mg/L mg/L (50 µg/L) is proposed for diquat (measured as the cation) 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 diquat completed by Health Canada's Pest Management Regulatory Agency (PMRA) and supporting documents.

Exposure

In Canada, diquat is an herbicide that is deliberately applied to food crops and to water sources for weed control. As such, the general Canadian population is potentially exposed to diquat through food, and to a lesser extent, drinking water. In 2016 (the most recent year for which data are available), more than 500,000 kg of diquat (as active ingredient) was sold in Canada. Very low levels of diquat have been detected in foods. Data provided by provinces and territories that monitor for diquat in source and drinking water, indicate that levels of diquat are below the detection limit.

Health effects

In repeat-dose animal studies, diquat primarily targeted the eyes, causing cataracts, and also affected the kidneys and liver. The proposed MAC of 0.05 mg/L (50 µg/L) is based on cataract formation.

Analytical and treatment considerations

Currently, there is one method available for the analysis of diquat in drinking water. The method detection limit is about one to two orders of magnitude below the proposed MAC.

Treatment methods may remove diquat from drinking water supplies, although studies at all scales (bench, pilot or full) are limited or nonexistent. Granular activated carbon (GAC) adsorption is likely the most effective treatment option and membrane filtration techniques (nanofiltration and reverse osmosis) and oxidation may also be effective. Due to lack of performance studies, it is recommended that pilot- and/or bench-scale testing be conducted prior to full-scale implementation.

In cases where diquat removal is desired at a small system or household level, for example, when the drinking water supply is from a private well, a residential drinking water treatment unit may be an option. Adsorption (activated carbon) and reverse osmosis represent the best potential technologies for diquat removal. When using a residential drinking water treatment unit, it is important to take samples of water entering and leaving the treatment unit and send them to an accredited laboratory for analysis to ensure that adequate diquat removal is occurring.

Application of the guideline

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

The proposed guideline is protective against health effects from exposure to diquat in drinking water over a lifetime. Any exceedance of the proposed 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 diquat concentrations are above the proposed MAC then an investigation to determine the most appropriate way to reduce exposure to diquat 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 proposed MAC.

International considerations

Other national and international organizations have drinking water guidelines, standards and/or guidance values. 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. The three international values established for diquat are based on cataract formation in rats and differ in their interpretation of this study, and in the selection of both body weights and source allocation factors.

The United States Environmental Protection Agency (U.S. EPA) has established a maximum contaminant level (MCL) of 0.02 mg/L while the Australian National Health and Medical Research Council (NHMRC) has established a guideline value of 0.007 mg/L, for diquat in drinking water. The World Health Organization (WHO) has calculated a non-regulatory health-based value of 0.03 mg/L (30 µg/L) for diquat.

The European Union (EU) does not have a specific parametric value for individual pesticides. Instead, the EU has 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, such as health effects. Instead, the values are based on a policy decision to keep pesticides out of drinking water.

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1.0 Exposure Considerations

1.1 Sources and uses

Diquat, also called 1,1'-ethylene-2,2'-dipyridinium ion or 6,7-dihydrodipyrido[1,2-*a*:2',1'-*c*] pyrazinediium ion, is a non-selective contact herbicide and algaecide used as a pre-harvest desiccant for various terrestrial food and feed crops, for industrial oilseed and fibre crops, as a defoliant (e.g., potato haulm destruction), for non-cropland chemical mowing, and for the control of aquatic weeds and algae (Health Canada, 2008). Diquat acts by generating superoxides during photosynthesis which damage cell membranes and cytoplasm (Health Canada, 2008; WHO, 2014). In Canada, diquat is sold as diquat dibromide, a highly hygroscopic salt supplied in an aqueous solution (Health Canada, 2008; Health Canada, 2010). More than 500,000 kilograms active ingredient of diquat was sold in Canada in 2016 (Health Canada, 2016).

Diquat and diquat dibromide have the potential to reach surface water via runoff or spray drift (Health Canada, 2008). Once in the environment, diquat dibromide completely dissociates to diquat in water (WHO, 2014). Diquat will then rapidly dissipate by adsorption to particles (e.g., suspended matter, montmorillonite clay, and phytoplankton) and sediments on which it can be retained for very long periods of time, with half-lives of about a few days (e.g., less than 48 hours in surface waters) to more than a year reported for unsorbed and sorbed states, respectively (Emmett, 2002; WHO, 2016; Magalhães et al., 2018). Diquat is not expected to leach into groundwater as it strongly adsorbs to soil particles, rendering it immobile and persistent in soils (U.S. EPA, 1995a). Diquat does not hydrolyze and is resistant to microbial degradation under aerobic and anaerobic conditions (typically 5-7% removal from soil per year through microbial degredation) (U.S. EPA, 1995a; Emmett, 2002; Magalhães et al., 2018). It does undergo photodegradation, mainly to 1,2,3,4-tetrahydro-1-oxopyrido[1,2-a]-5-pyrazinium salt (TOPPS), but slowly with reported rates of 10-20% per year (depending on the experimental conditions) (EFSA, 2015; Magalhães et al., 2018). Diquat is removed from the water column by adsorption to soil sediments, aquatic vegetation, and organic matter (U.S. EPA, 1995a).

Owing to its very low volatility, diquat dibromide residue will occur in the air most likely as aerosols. However, its presence in the atmosphere over time is unlikely as it would normally be removed by gravitational settling, ending up in surface waters and/or in the soil where it will dissociate to the diquat ion (HSDB, 2010).

Numerous factors can affect the fate and persistence of diquat in the environment such as: water temperature, soil moisture, and the rate of microbial metabolism. Furthermore, the intensive use of fertilizers containing other cations (e.g., Ca^{2+}, Mg^{2+}, $NH_4^{(2+)}$, K^+) may lead to a greater desorption of diquat from sediment (Emmett, 2002).

1.2 Substance identity

Diquat (Chemical Abstracts Service registry number (CAS#) 2764-72-9; $C_{12}H_{12}N_2$) is a quaternary ammonium (divalent) cation from the bipyridylium chemical class; it has a molecular weight of 184.2 g/mol (U.S. EPA, 1995a; Health Canada, 2008; Health Canada, 2010).

Diquat is sold as diquat dibromide (or 6,7-dihydrodipyrido(1,2-a:2',1' -c) pyrazinediium dibromide or 1,1'-ethylene-2,2'-dipyridylium dibromide), an odourless, pale yellow crystalline solid (U.S. EPA, 1995a; Emmett, 2002). Diquat dibromide is highly soluble in water and readily dissociates to the diquat ion (U.S. EPA, 1995a; Emmett, 2002). Some of the physico-chemical properties of diquat dibromide are summarized in Table 1.

Table 1. Properties of diquat dibromide relevant to its presence in drinking water (Health Canada, 2008).

Property	Diquat Dibromide	Interpretation
CAS#	85-00-7	
Molecular formula	$C_{12}H_{12}Br_2N_2$	
Molecular weight g/mol)	344.0	
Water solubility (g/L)	700 at 25°C	Highly soluble in water
vapour pressure (volatility)	<0.01 mPa at 20°C (for monohydrate)	Low volatility, unlikely to be present in air
Henry's law constant	5×10^{-9} Pa.m^3.mol	Low volatility, unlikely to be present in air
Dissociation constant	Not applicable	Complete dissociation
n-Octanol:water partition coefficient (Log K_{ow})	-4.60 at 20 °C	hydrophilic

The synthesis of diquat dibromide may result in the formation of ethylene dibromide (EDB) as a process impurity. However, test results have shown that EDB, which is not used as a pesticide, does not persist as an impurity in diquat products as it dissipates with time (U.S. EPA, 1995a). Furthermore, since the highly charged diquat is identified as the most toxicologically significant species in mammals (Health Canada, 2008; FAO and WHO, 2014), in the current assessment, dose levels and water concentrations are defined, whenever possible, in terms of diquat cation, herein referred to as diquat.

1.3 Exposure

As an herbicide, diquat is deliberately applied to food crops and to water sources for weed control. As such, the general Canadian population is exposed to diquat through food, and to a lesser extent, drinking water (Health Canada, 2008; Health Canada, 2010). Based on its environmental fate (see Section 1.1), significant residues of the herbicide are not expected in water sources, making drinking water a minor source of exposure (U.S. EPA, 2002; Health Canada, 2008; NHMRC and NRMMC, 2011; EFSA, 2015; OEHHA, 2016; WHO, 2016).

Data provided by the provinces and territories indicate that diquat levels are below the method reporting limit (MRL) or method detection limit (MDL) in all samples collected 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; Government of Ontario, 2019; Indigenous Services Canada, 2019; Ministère de l'Environnement et de la Lutte contre les changements climatiques du Québec, 2019; Nova Scotia Environment, 2019; P.E.I. Department of Communities, Land and Environment, 2019) (See Table 2).

Monitoring for diquat is not currently conducted in Manitoba, New Brunswick, Newfoundland and Labrador, Saskatchewan and Yukon (Manitoba Sustainable Development 2019; New Brunswick Department of Health, 2019; Newfoundland and Labrador Municipal Affairs and Environment, 2019; Saskatchewan Water Security Agency, 2019; Yukon Environmental Health Services, 2019).

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

Jurisdiction (MDL µg/L)	**Monitoring Period**	**Municipal/Non-municipal**	**Water Type** (Municipal: ground/surface – raw, treated, distributed)	**# Detects/ samples**
British Columbia (7)	2013-2018	Municipal	Surface – raw	0/18
FNIHB[a] Ontario Region (1 – 50)	2014-2018	Public Water Systems	Ground – raw	0/13
			Ground – treated	0/190
			Ground – distribution	0/16
			Surface – raw	0/33
			Surface – treated	0/308
			Surface – distribution	0/23
		Semi Public Water Systems	Ground – raw	0/3
			Ground – treated	0/16
			Ground – distribution	0/68
			Surface – raw	0/1
			Surface – treated	0/9
			Surface – distribution	0/2
		Private Water Systems	Ground – treated	0/3
			Ground – distribution	0/50
			Surface – treated	0/5
FNIHB[a] Atlantic Region (7 – 70)	2014-2018	Public Water Systems	Ground – treated	0/4
			Ground – distribution	0/4
			Surface – treated	0/1
FNIHB[a] Québec Region (0.1 – 0.4)	2014-2018		Drinking water system	0/4
Nova Scotia (1 – 7)	2007-2018	Municipal	Ground – raw	0/71
			Ground – treated	0/35
			Surface – raw	0/35
			Surface – treated	0/39
			Distributed	0/1
Ontario (0.1)	2008-2012	Municipal	Ground – raw	0/91
			Ground – treated	0/25
			Unknown – raw	0/213
			Unknown – treated	0/223
			Unknown – distribution	0/1
Prince Edward Island (10)	2007-2016	Municipal	Ground – raw	0/103
		Non-municipal	Ground – raw	0/137
Québec (0.1 –15)	2013-2018	Municipal	Ground – distribution	0/574
			Surface – distribution	0/1726
		Municipal (Special Projects) Potatoes project[b] [2017-2018]	Ground – raw	0/46
			Ground – treated	0/17
			Ground – distribution	0/5
		Small systems[c] [2012-2018]	Ground – raw	0/63
			Non-municipal	
			Ground – raw	0/43

[a]FNIHB – First Nations and Inuit Health Branch

[b]Potato Project 2017-2018: During the period covered, analysis results of diquat pesticide found in raw, treated or distributed ground water were obtained by the Ministry from 9 drinking water production stations.

[c]Small Systems Project 2012-2018: During the period covered, analysis results of diquat found in raw ground water were obtained by the Ministry from 25 water supply stations.

In foods, diquat residues are only expected when the herbicide is applied directly to food crops (e.g., when used as a desiccant on potato plants), although residue levels are likely to be low (U.S. EPA, 1995a; Health Canada, 2010; NHMRC and NRMMC, 2011). Results from residue trials conducted in Canada in 2015 were between 0.01-0.35 mg/kg (n = 24) for dry beans (including white and red kidney beans, soya beans, adzuki beans and fava beans); 0.07-0.58 mg/kg (n = 9) for chickpeas; 0.052-0.57 mg/kg (n = 8) for lentils; 0.15-2.1 mg/kg (n = 6) for barley (FAO, 2019). For animal commodities, testing in dairy cows fed diets containing 18, 50 and 84 ppm diquat for 30 days showed diquat levels were below the limit of quantification (LOQ) for all milk samples (LOQ= 0.001 mg/kg), and for samples of liver, kidney, fat and muscle (LOQ = 0.01 mg/kg), for all the dose groups. In eggs, levels were also less than the LOQ of 0.01 mg/kg (FAO, 2019).

2.0 Health Considerations

All pesticides, including diquat, are regulated by Health Canada's 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 (U.S. EPA). As such, this health assessment is primarily based on PMRA's evaluations (Health Canada, 2008; Health Canada, 2010) and supporting documentation. Additionally, any reviews and relevant literature available since PMRA's evaluations were completed were also considered.

2.1 Kinetics

Absorption: Ingested diquat is poorly absorbed (<10%) from the gastrointestinal tract of animals, including humans. Although bioavailability values of less than 10% are usually reported, a European Food Safety Authority's (EFSA) expert committee concluded that the available data support values of 3% to 4% instead (U.S. EPA, 1995a; Emmett, 2002; FAO and WHO, 2014; EFSA, 2015; Magalhães et al., 2018). Furthermore, there is a species variability regarding diquat absorption, with the dog exhibiting the highest absorption (Gupta and Crissman, 2013). Both the presence of food in the digestive tract and the presence of intestinal microflora that degrade diquat, significantly decrease absorption (Magalhães et al., 2018).

Distribution: Once in the aqueous phase of the blood, the small amount of absorbed diquat is rapidly (i.e., within 6-18 hours) and widely distributed to several organs and tissues (e.g., liver, kidney, adrenal glands), except the brain and spinal cord (Gupta and Crissman, 2013; Magalhães et al., 2018). Despite its wide distribution, the hydrophilic and highly polar diquat does not covalently bind to macromolecules nor does it accumulate in most of the tissues, except the eye lens. Despite high diquat levels being found in the liver, the kidney, the gastrointestinal tract, and the lungs just after dosing, the eye lens remained the primary site of significant deposit up to 96 hours post-exposure (U.S. EPA, 1995a; Emmett, 2002; FAO and WHO, 2014; EFSA, 2015; Magalhães et al., 2018).

Metabolism: Both experimental animal and human data indicate that the intracellular catabolism of free diquat is minimal and that it is predominately metabolized in the liver. As such, diquat metabolism proceeds by cytochrome P450 enzymes with the formation of diquat monopyridone and diquat dipyridone, as major and minor metabolites, respectively (Fuke et al., 1996; Emmett, 2002; FAO and WHO, 2014; WHO, 2014). Although not clearly identified in mammals, some data indicate that diquat biotransformation may also result in the formation of

picolinic acid (or pyridine-2-carboxylic acid), presumably via picolinamide (or pyridine-2-carboxamide) as an intermediate. In addition to all the above-mentioned metabolites, the production of volatile compounds has also been hypothesized. There is also some (*in vitro*) evidence of an alternative bacterial biotransformation of diquat occurring to a minor degree in the gastrointestinal tract with the monopyridone derivative as the major metabolite (Emmett, 2002; FAO and WHO, 2014; WHO, 2014; Magalhães et al., 2018).

Elimination: Due to its poor absorption, ingested diquat is mainly (about 90%) excreted unchanged via the feces within 24 hours with virtually no biliary excretion (< 0.7% of the administered dose). In addition to the parent chemical, two other metabolites, diquat monopyridone and diquat dipyridone, are excreted in the feces (Emmett, 2002; Magalhães et al., 2018). Regarding absorbed diquat, it is primarily (> 90%) excreted within 48 hours in urine, mostly as the parent compound, followed by its two main metabolites and to a lesser extent picolinic acid (U.S. EPA, 1995a; Fuke et al., 1996; Emmett, 2002; NHMRC and NRMMC, 2011; FAO and WHO, 2014; WHO, 2014; EFSA, 2015; Magalhães et al., 2018).

2.2 Health effects

The database for the toxicity of diquat is adequate covering several endpoints and various types of exposure (U.S. EPA, 1995a, 2001; FAO/WHO, 2014; WHO, 2014; EFSA, 2015 give more thorough reviews). In general, diquat has a low acute toxicity. Repeated dose studies in animals show that diquat may induce toxicity in multiple organs (e.g., gastrointestinal tract, kidneys and liver) with the eye being the most sensitive endpoint (FAO and WHO, 2014; WHO, 2014; EFSA, 2015).

2.3 Effects in humans

Intentional ingestion of diquat by humans may result in poisoning and even death (Magalhaes et al., 2018). In general, the clinical features from acute poisoning include neurological disorders, gastrointestinal tract disturbances, renal failure, hepatic injury, and hemodynamic and cardiocirculatory complications (Valiante et al., 1992; Schmidt et al., 1999; Tanen et al., 1999; Fuke et al., 1996; Hantson et al., 2000; Jones and Vale, 2000; Emmett, 2002; Jovic-Stosic et al., 2009; WHO, 2014).

Epidemiological data specific to diquat herbicide are scarce but include reports of adverse health effects in manufacturing plant personnel (WHO, 2014).

2.4 Effects in animals

Diquat has been shown to be toxic to experimental animals with oral median lethal dose (LD_{50}) values reported for some species as follows: 215-235 mg diquat/kg bw in the rat; 125 mg diquat/kg bw in the mouse; 100-200 mg diquat/kg bw in the dog;100 mg diquat/kg bw in the rabbit and 100-300 mg diquat/kg bw in the monkey (U.S. EPA, 1995a; FAO and WHO, 2014; WHO, 2014; WHO, 2016; Magalhaes et al., 2018). Diquat metabolites were found to be less toxic with rat oral $LD_{50} > 4000$ and ≥ 2449 mg/kg bw reported for diquat monopyridone and TOPPS, respectively (WHO, 2014; Magalhaes et al., 2018). Adverse effects were related to the gastrointestinal tract, kidneys, and potentially the liver (U.S. EPA, 1995a; Emmett, 2002; WHO, 2014).

Both subchronic and chronic exposure to diquat resulted in eye damage (e.g., cataracts, extralenticular lesions such as vitreous adhesions, retinal detachment and synechia) in the exposed experimental animals including rats and dogs. Damage to the kidney, liver, adrenals,

epididymis, and haematological parameters were also reported (U.S. EPA, 2001; FAO and WHO, 2014; EFSA, 2015; WHO, 2016).

Ocular lesions (cataracts and lens opacities): Both subchronic and chronic (dietary) exposures to diquat dibromide have consistently resulted in eye damage in mouse (chronic oral toxicity study), rat (2-generation reproductive studies, subchronic oral toxicity and subchronic neurotoxicity study, chronic oral toxicity study) and dog (chronic toxicity study) (U.S. EPA, 1995a; Emmett, 2002; WHO, 2014; EFSA, 2015). Eye damage was observed following chronic ingestion of diquat (up to 48.27, 19.44 and 12.5 mg diquat/kg per day for mouse, rat and dog, respectively) and generally progressed from opacities of the lens to total opacification (i.e., cataracts); the incidence and severity of the damage were dose-related (Colley et al., 1985; Hopkins, 1990; Hodge, 1992; Emmett, 2002; WHO, 2014; EFSA, 2015). In the rat, cataracts were first observed at week 13 in the subchronic neurotoxicity study (No-observed-adverse-effect level (NOAEL), 8 mg/kg per day) (Horner, 1992a) and at week 10 in the chronic toxicity study (NOAEL, 0.58 mg/kg per day) (Colley et al., 1985). The dog was the most sensitive species exhibiting total opacities of the eyes at week 8 in females exposed to 2.5 mg diquat/kg bw per day and at week 16 in males exposed to 12.5 mg diquat/kg bw per day. From the one-year dog study, the highest chronic NOAEL was 0.53 mg diquat/kg bw per day, based on cataracts observed in female dogs (Hopkins, 1990).

Nephrotoxicity: Diquat can induce nephrotoxicity which is generally characterized by renal tubular necrosis with subsequent decrease in the clearance of diquat and worsening of the damage (Gupta and Crissman, 2013). Diquat-induced kidney lesions were reported in mice (one chronic study), rats (two rat multigenerational and one lifetime studies), and dogs (two chronic studies) orally exposed up to 48, 19, and 12.5 mg diquat/kg bw per day, respectively (U.S. EPA, 1995a; WHO, 2014; EFSA, 2015; OEHHA, 2016).

After 104 weeks, mice receiving at least 12 to 16 mg/kg per day of diquat, for males and for female, respectively, presented with kidney tubule dilation associated with increased kidney tubule hyaline droplets in females, and slightly increased (but insignificant; 6%; $p < 0.01$) kidney weights in males (Hodge, 1992; Emmett, 2002; WHO, 2014). Both male and female rats exposed for a lifetime to dietary levels of at least 2.9 to 3.6 mg/kg-day, respectively, displayed decreased renal clearance (both sexes), while only the males from the same dose groups had histological damage in the kidney (Colley et al., 1985; U.S. EPA, 2001; Emmett, 2002; OEHHA, 2016). Additionally, the incidence of haemorrhagic kidneys was increased in the foetuses of pregnant rats exposed to 40 mg/kg per day in a 2-generation developmental study (Wickramaratne, 1989). Dogs fed 12.5 mg/kg bw per day for one year had increased kidney weights although no related histopathological changes were noted (Hopkins, 1990; U.S. EPA, 2001; Emmett, 2002).

Reproductive and developmental toxicities: Experimental animal studies do not support a clear association between oral exposure to diquat and adverse reproductive and developmental outcomes (EFSA, 2015; OEHHA, 2016; WHO, 2016). No effect was observed on the reproductive function of rats fed a diet containing at least 25 mg diquat/kg per day in 2 separate multigenerational reproductive toxicity studies (Fletcher, 1972; Hodge, 1990). Data from studies in which pregnant mice (Palmer et al., 1978), rats (Wickramaratne, 1989) and rabbits (Hodge, 1989) were exposed by gavage to at least 4, 40, and 10 mg diquat/kg per day, respectively, suggested some teratogenic effects of diquat; however, these adverse effects were generally observed at the highest dose tested and there was no indication of an increased sensitivity of the offspring to (in utero and/or postnatal) diquat exposure. Friable and/or mottled livers were

observed in the foetuses of pregnant New Zealand rabbits exposed to 10 mg diquat/kg bw per day by gavage (Hodge, 1989; U.S. EPA, 2001).

Neurotoxicity and other effects: There was no evidence of neurotoxicity of diquat following oral exposure. The results of both acute and subchronic neurotoxicity studies in which rats (Horner, 1992a; Horner, 1992b) or mice (Minnema et al., 2016) were fed diets containing up to 38.5 or 150 mg diquat/kg bw per day, respectively, did not resulted in neurologically adverse effects (e.g., neuropathies, neurodegenerative effects) as evaluated by functional observation battery tests, motor activity testing and neuro-histopathological examinations (U.S. EPA, 1995a; U.S. EPA, 2001; Emmett, 2002; EFSA, 2015). Increased incidences of arteritis/periarteritis in blood vessels and paracortical cell hyperplasia in the lymph nodes were observed in male rats dosed with 14.88 mg diquat/kg bw per day (Colley et al., 1985).

2.5 Genotoxicity and carcinogenicity

The current evidence indicates that diquat is neither genotoxic nor carcinogenic (U.S. EPA, 2002; NHMRC and NRMMC, 2011; FAO and WHO, 2014; EFSA, 2015; OEHHA, 2016; WHO, 2016).

Diquat dibromide was negative in four mutagenicity assays (i.e., Ames tests, mouse bone marrow micronucleus assay, mouse dominant lethal assay, and unscheduled DNA synthesis in rat hepatocytes) but it was positive in two other studies (i.e., mouse lymphoma cell assay, human blood lymphocytes – with or without metabolic activation) (U.S. EPA, 1995a; WHO, 2016). Furthermore, there was no evidence of the genotoxicity of diquat monopyridone and TOPPS (FAO and WHO, 2014).

The results of lifetime studies in mice (Hodge, 1992) and rats (Colley et al., 1985) found no evidence of carcinogenicity for diquat (U.S. EPA, 1995a; NHMRC and NRMMC, 2011; FAO and WHO, 2014; EFSA, 2015; WHO, 2016). The U.S. EPA has classified diquat dibromide as a Group E carcinogen (i.e., evidence of non-carcinogenicity for humans) (U.S. EPA, 1995a) while the International Agency for Research on Cancer (IARC) has not reviewed the carcinogenicity of either diquat or diquat dibromide.

2.6 Mode of action

In mammals, the cytotoxicity of diquat was found to be associated with the parent compound. As such, the mode of action of the cytotoxicity of the highly charged cation is generally similar to that of the other bipyridyl herbicides such as paraquat. It involves oxidation-reduction (redox) cycling that generate reactive oxygen species (ROS) and/or reactive nitrogen species (RNS), and depletes the cellular pyridine nucleotides, subsequently leading to oxidative stress, cellular dysfunction, and potentially cellular necrosis (Gallagher et al., 1995; Jones and Vale, 2000; Emmett, 2002; Fussell et al., 2011; Gupta and Crissman, 2013; Gupta, 2014; Magalhaes et al., 2018). *In vivo* redox cycling as well as *in vitro* lipid peroxidation of diquat have been demonstrated (Sandy et al., 1986; Circu et al., 2017).

Overall, even though it is not conclusively defined, it is anticipated that diquat redox cycling may be responsible for the specific toxicity findings, i.e., eye damage (or cataract), observed in the toxicological studies since the eyes accumulate diquat more than other tissues (Emmett, 2002; OEHHA, 2016).

2.7 Selected key study

Health Canada's PMRA (2008, 2010, and 2019) considered the eye as the most sensitive target organ across the database. The one-year investigation in dogs conducted by Hopkins

(1990) was identified as the key study for the human health risk assessment of diquat in drinking water.

Groups of beagle dogs (four/sex/dose) were fed diets containing 0, 0.5, 2.5, and 12.5 mg diquat/kg bw per day in the form of diquat dibromide (equivalent 0, 0.46, 2.42 and 11.48 mg diquat/kg per day for males; 0, 0.46, 2.42, and 13.21 mg diquat/kg per day for females) for 52 weeks (Hopkins, 1990; U.S. EPA, 1995a; U.S. EPA, 2001; Health Canada, 2008; WHO, 2014; WHO, 2016). There were no treatment-related adverse effects on survival, clinical signs, haematology, clinical chemistry, urinalysis, and gross pathology (except the eye) at any dose level (U.S. EPA, 2001; WHO, 2014). The major treatment-related finding from this study, which was observed starting at the 2.5 mg/kg dose level, was eye damage. Its incidence and severity increased with increasing dose. In the 2.5 mg/kg dose group, two out of four females exhibited unilateral cataracts (i.e., lens opacity), which first occurred during the 8th week for one female and 40th week for the other. In the highest dose group, three of four females and all males presented with bilateral lens opacity that first occurred during the 16th week for males and 24th week for females. In addition to lens opacities, the other treatment-related alterations in the high-dose group included inflammatory changes in the gastrointestinal tract in both sexes, reproductive effects in males as well as statistically significant increased kidney weights in all dogs. Furthermore, the males from the two top dose groups had decreased weights of the adrenals and epididymis although these changes did not corroborate with any histopathological changes in the corresponding organs (except in the gastrointestinal tract) (U.S. EPA, 1995a; U.S. EPA, 2001; WHO, 2014; WHO, 2016). An oral NOAEL of 0.5 mg diquat/kg bw per day was identified in this study, based on unilateral cataracts in females and decreased weights of the adrenals and epididymis in males at the lowest LOAEL of 2.5 mg diquat/kg bw per day (U.S. EPA, 1995a; WHO, 2014).

The findings in Hopkins (1990) are also supported by a 2-year study in rats (Colley et al., 1985). In Colley et al. (1985), a combined twoyear chronic/carcinogenicity study, male and female Sprague-Dawley rats (50/sex/dose) were fed diets containing 0, 5, 15, 75 or 375 ppm diquat dibromide (equivalent to (male-female): 0-0, 0.19-0.24, 0.58-0.72, 2.91-3.64, and 14.88-19.44 mg diquat/kg bw per day) for 104 weeks. There was also an interim sacrifice (10/sex/dose) that took place at 52 weeks (Colley et al., 1985; U.S. EPA, 1995a; U.S. EPA, 2001; Health Canada, 2008; WHO, 2014; WHO, 2016). There were no treatment-related adverse effects on organ weights, urinalysis and blood biochemistry parameters (WHO, 2014). The major significant finding was the formation of cataracts. Gradual lenticular opacities occurred throughout the study, followed by total opacity (i.e., cataract) which first occurred in few animals from the two-highest dose groups by week 11. Based on ophthalmologic and histopathological examinations of the eyes, the incidence of total lens opacity increased with increasing dose and time. At interim sacrifice, a few males in the 75 ppm group and up to 95% of both sexes in the 375 ppm group had cataracts. At study termination, these incidences increased to 15% in the 75 ppm group and 100% for both sexes in the 375 ppm group. Additionally, rats with severe cataracts exhibited extralenticular eye lesions (e.g., vitreous adhesions, retinal detachment, iritis, and intraocular haemorrhage) (U.S. EPA, 2001). Even though cataracts were identified in the three highest-dose groups at study termination, only one rat per sex in the 15 ppm dose group had total cataract. Based on these findings and the poor survival rate in the highest-dose groups, the data were subsequently re-evaluated and it was concluded that the incidence and severity of cataracts in the 15 ppm dose group were comparable to that of controls (Harling et al., 1997; WHO, 2014; WHO, 2016). Therefore, a systemic NOAEL of 0.58 mg diquat/kg bw per day is

identified in this study, based on eye effects observed at the lowest LOAEL of 2.91 mg diquat/kg bw per day (U.S. EPA, 1995a; WHO, 2014).

3.0 Derivation of the health-based value

As noted above, the NOAEL of 0.5 mg/kg bw per day from the dog study by Hopkins (1990) which showed cataract formation was selected as the basis for the current risk assessment. The NOAEL of 0.5 mg/kg bw per day is based on unilateral cataracts in females and decreased adrenal and epididymides weights in males. Also considered in the derivation of the acceptable daily intake (ADI) is the NOAEL of 0.58 mg/kg per day in females based on eye lesions from the study by Colley et al. (1985).

Using the NOAEL of 0.5 mg diquat/kg per day, an ADI for diquat (i.e., the bipyridyl divalent cation) (Health Canada, 2008, 2019) was calculated as follows:

$$\text{ADI} = \frac{0.5 \text{ mg/kg bw per day}}{100}$$

$$= 0.005 \text{ mg/kg bw per day}$$

Where:

- 0.5 mg/kg bw per day is the NOAEL, based on cataracts formation in dogs; and
- 100 is the uncertainty factor, selected to account for interspecies variation (×10) and intraspecies variation (×10).

Based on the ADI of 0.005 mg/kg bw per day, a health-based value (HBV) for diquat in drinking water was derived as follows:

$$\text{HBV} = \frac{0.005 \text{ mg/kg bw per day} \times 74 \text{ kg} \times 0.2}{1.53 \text{ L/day}}$$

$$= 0.05 \text{ mg/L (50 µg/L)}$$

Where:

- 0.005 mg/kg bw per day is the ADI derived above;
- 74 kg is the adult body weight (Health Canada, in preparation);
- 0.20 is the default allocation factor for drinking water (Krishnan and Carrier, 2013);
- 1.53 L per day is the drinking water intake rate estimated for an adult (Health Canada, in preparation).

4.0 Analytical and Treatment Considerations

4.1 Analytical methods to detect diquat

One standardized analytical method is available for the analysis of diquat in drinking water and the MDL is summarized in Table 3. MDLs are dependent on the sample matrix, instrumentation, and selected operating conditions and will vary between individual laboratories. A number of accredited laboratories in Canada were contacted, and it was indicated that MDLs were in the same order of magnitude as that reported in Table 3. The reported MRLs ranged

between 1 and 7 µg/L (ALS Environmental, 2019; Bureau Veritas Laboratories, 2019; SGS Environmental Services, 2019).

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 maximum acceptable concentration (MAC). Sample processing considerations for the analysis of diquat in drinking water (e.g., sample preservation, storage) can be found in the reference listed in Table 3. It is important to note that quenching is critical if an oxidant is present in samples in order to prevent additional degradation of diquat prior to analysis.

Table 3. Standardized analytical methods for the analysis of diquat in drinking water.

Method (Reference)	Methodology	MDL (µg/L)	Interferences/Comments (operational considerations)
U.S. EPA Methods			
EPA-NERL[1]: 549.2 (U.S. EPA, 1997)	Liquid-Solid Extraction and High-Performance Liquid Chromatography (HPLC) with UV Detection	0.72	Matrix; Ca^{2+}, Mg^{2+} can cause low recovery Diquat adsorbs to surfaces, especially glass which needs to be accounted for during sampling and analysis

[1]NERL - National Exposure Research Laboratory

4.2 Treatment considerations

Studies of treatment technologies covering the removal efficiency of diquat from drinking water are not available. It is stated that granulated activated carbon (GAC) should remove diquat from water (U.S. EPA, 1995b; WHO, 2017). Reverse osmosis (RO), nanofiltration (NF) and oxidation are possible technologies for diquat removal.

4.2.1 Municipal-scale treatment

Typical water treatment that includes conventional filtration (chemical coagulation, clarification, and rapid sand filtration) is not effective for diquat removal from drinking water (WHO, 2017). Diquat is highly adsorptive to soil, has a medium molecular weight and slowly degrades naturally in water through photolysis; these properties influence how diquat is removed from drinking water. The selection of an appropriate treatment process for a specific water supply 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. Appropriate pilot- or bench-scale testing is recommended to ensure the source water can be successfully treated.

When using oxidation or advanced oxidation processes (AOP) for pesticide removal in drinking water, it is important to be aware of the potential for 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). Removal of the target pesticide alone does not ensure that the treatment is efficient and that full mineralization (to carbon dioxide, inorganic ions and water) has been achieved. 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. Pilot-scale testing is an important step for water utilities considering oxidation and AOP treatment processes for pesticide removal in drinking water.

4.2.1.1 Activated carbon adsorption

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

GAC has been recommended as a possible treatment technology for diquat removal from drinking water (U.S. EPA, 1995b; WHO, 2017), however references to support this recommendation were not provided in these reports. U.S. EPA (2016) indicates that small system compliance technologies for diquat removal are GAC, point-of-use GAC and PAC, although no references to full-, pilot- or bench-scale studies indicating performance were given.

There is very limited published literature of activated carbon for diquat adsorption and none on adsorption capacity or performance. As such, prior to full-scale implementation it is essential to conduct appropriate pilot- or bench-scale testing. Diquat removal from natural water using activated carbon can be negatively affected by competition from other contaminants or natural organic matter (NOM), biofilm development, temperature, influent concentration, carbon size and hydraulic loading rate (Speth and Miltner, 1998; Haist-Gulde and Happel, 2012).

Data generated through bench-scale testing to determine adsorption coefficients for pesticides are useful in predicting whether activated carbon adsorbs a particular pesticide (U.S. EPA, 2011). In general, pesticides with an adsorption capacity constant (e.g., Freundlich coefficient) greater than 200 $\mu g/g(L/\mu g)^{1/n}$ are considered to be amenable to removal by carbon adsorption (Speth and Adams, 1993; Speth and Miltner, 1998, U.S. EPA, 2011). The authors noted however, that the capacity of activated carbon is affected by many factors, including the compound's ionic character and the solution pH. The Freundlich coefficients in organic-free water for diquat ranged from 103 $\mu g/g(L/\mu g)^{1/n}$ at a pH of 3.0, to 2,910 $\mu g/g(L/\mu g)^{1/n}$ at a pH of 6.1 and increased to 122,000 $\mu g/g(L/\mu g)^{1/n}$ at a pH of 10.1 (Speth and Miltner, 1998). These results illustrate the large effect of pH on adsorption, with very little at the low pH of 3.0, to reasonable at near-neutral pH and quite high at higher pH.

The use of PAC offers the advantage of providing virgin carbon when required (e.g., during the pesticide application season) (Miltner et al., 1989). The removal efficiency of PAC depends on the PAC type and dose, the contact time, the PAC characteristics (type, particle size), the adsorbability of the contaminant and the presence of NOM (Gustafson et al., 2003; Summers et al., 2010; Haist-Gulde and Happel, 2012; 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 adsorbability of the contaminant, the filter run time (Haist-Gulde and Happel, 2012). In addition, because GAC fixed bed adsorbers are typically operated on a continuous basis, the GAC can become fouled (or preloaded) with NOM and it may be completely or partially ineffective for pesticide removal (Knappe et al., 1999; Summers et al., 2010; Haist-Gulde and Happel, 2012; Chowdhury et al., 2013).

Several other studies investigated diquat removal from wastewater through batch experiments using GAC or various adsorbents not typically used in drinking water treatment (Dichiara et al., 2015; Hao et al., 2015; Li et al. 2017; Duman et al., 2019). The initial concentrations for these studies were much higher than typically found in drinking water ranging from 5.43 and 80 mg/L. The removal efficiencies varied between 72.9 and 89.3%, the adsorption

capacities between 36.45 and 197.53 mg/g and the adsorption rate from 0.612 to 29.1 mg/m·min, depending on the adsorbent and the experimental conditions.

4.2.1.2 Membrane filtration

In general, NF and RO are effective pressure-driven membrane processes for the removal of pesticides from drinking water. The effectiveness of NF and RO on pesticide removal is dependent on the membrane characteristics, pesticide properties, feed water composition, operating conditions and membrane fouling (Van der Bruggen and Vandecasteele, 2003; Plakas and Karabelas, 2012; Bellona et al., 2004).

Since the main mechanism for pesticide removal using NF and RO membranes is size exclusion, the molecular weight cut-off (MWCO) of the membrane is an important characteristic. There are no studies evaluating membrane filtration for the removal of diquat, however, based on the molecular weight (184.2 g/mol), membranes with a lower MWCO than this value may be effective. Diquat is highly polar and hydrophilic which are physical and chemical properties that decrease the effectiveness of membrane rejection (Plakas and Karabelas, 2012), and therefore the removal of diquat will most likely be due to size exclusion alone.

Bellona et al. (2004) present a flow-chart using the characteristics of the pesticide in water (e.g., molecular weight, log K_{ow}, molecular diameter) and those of the membrane (e.g., MWCO, pore size) which could be used to determine the potential for removal of diquat by membrane filtration. It is important to perform appropriate testing prior to full-scale implementation with membrane and source water under the proposed operating conditions to ensure that adequate diquat removal is occurring.

4.2.1.3 Oxidation

Chemical oxidation using chlorine dioxide can be effective treatment methods for removing diquat from water depending on a variety of factors including oxidant dose, contact time, disinfectant demand, temperature and pH.

Bench-scale testing conducted with oxidants including chlorine dioxide, permanganate and chlorine was conducted to determine diquat degradation in distilled water (Gomaa and Faust, 1971). The initial concentrations used in this study are orders of magnitude higher than what would typically be found in source water (15 – 30 mg/L) and the oxidant doses are high (chlorine dioxide dose of 6.75 mg/L). It was shown that chlorine dioxide was the oxidant of choice for diquat removal, with good removal for pH greater than 8.0 and with complete reaction in less than one minute. The use of chlorine dioxide would require pre-adjustment of pH to slightly alkaline levels. Chlorine was found to be less effective than chlorine dioxide but was also more effective at higher pH. Potassium permanganate was also investigated and did not exhibit adequate diquat removal. Formation of by-products was not discussed in this study.

Relatively slow reaction rates for diquat have been reported for ozonation (Yao and Haag, 1991; Hu et al., 2000). Hu et al. (2000) conducted a bench-scale study evaluating oxidation rate constants of twenty-four pesticides using ozone. The tests were carried out using synthetic raw water at a pH of 7.5, ionic strength of 10^{-3} M and 100 µM $NaHCO_3$. Using an ozone dose of 1.3 mg/L, a rate constant of 67.9 $M^{-1}s^{-1}$ was obtained, which was the second lowest of all pesticides examined. As a comparison, 2,4-dichlorophenoxyacetic acid (2,4-D) had a reaction rate of 298 $M^{-1}s^{-1}$ and Warfarin had a reaction rate greater than 21,000 $M^{-1}s^{-1}$.

Yao and Haag (1991) investigated the oxidation reaction rate constants for forty-five organic compounds using ozone (Yao and Haag, 1991). A reaction rate of 0.6 $M^{-1}s^{-1}$ was obtained at a pH of 3.1 and a half-life of 15 hours was determined for pH of 7.

4.2.2 Residential-scale treatment

In cases where diquat 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 diquat concentrations in drinking water. If guidance is required, consumers should contact their responsible drinking water authority. Before a treatment unit is installed, the water should be tested to determine the general water chemistry and diquat concentration in the source water. There is a lack of performance testing of treatment technologies for diquat removal; however, adsorption (activated carbon) and RO are treatment technologies that may remove diquat at the residential-scale. 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 service it when required. Systems classified as residential scale may have a rated capacity to treat volumes greater than that 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 (NSF)/American National Standards Institute (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 ;
- 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.

There are no certified drinking water treatment units for the removal of diquat. However, adsorption (activated carbon) and RO treatment units may be effective in removing diquat. Consumers can use a treatment unit that is certified to the standards for reverse osmosis or adsorption to ensure that the material safety has been tested.

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 (Canadian Council of Ministers of the Environment (CCME), 2004; WHO, 2011, 2012). These approaches require a system assessment to characterize the source water, to 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, 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

Diquat 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 diquat to enter source water (e.g., raw water supply to the drinking water system) based on site-specific considerations.

When it is determined that diquat may be present and monitoring is necessary then surface and groundwater sources should be characterized to determine the concentration of diquat. 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 diquat is not present in source water, monitoring may be reduced.

Where treatment is required to remove diquat, 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, fluctuations of the raw water concentrations and the treatment process. Responsible authorities should be aware of the impact of NOM on activated carbon systems, as it may impact water quality objectives for diquat removal.

Where treatment is in place for diquat removal, compliance monitoring (i.e., paired samples of source and treated water to confirm the efficacy of treatment) should be conducted at a minimum on an annual basis. When routine operational monitoring indicates the potential for contaminant breakthrough, such as with GAC, monitoring should be conducted quarterly. When a degradation process, like oxidation, is utilized, by-product formation should also be considered.

6.0 International Considerations

This section presents drinking water guidelines, standards and/or guidance from other national and international organizations. 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.

The U.S. EPA has set a maximum contaminant level (MCL) of 0.02 mg/L while the Australian National Health and Medical Research Council has established a guideline value of 0.007 mg/L for diquat in drinking water (NHMRC and NRMMC, 2011). The World Health Organization (WHO) has calculated a non-regulatory health-based value of 0.03 mg/L (30 µg/L) (WHO, 2016). These three values are based on cataract formation observed in a 2-year rat study conducted in 1985, but differ in their interpretation (i.e., NOAEL) and in the selection of body weights and of source allocation factors (Table 4).

The European Union (EU) does not have a specific chemical parametric value for individual pesticides. Instead, the EU has 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, such as health effects. Instead, the values are based on a policy decision to keep pesticides out of drinking water.

Table 4. Comparison of international drinking water values for diquat.

Agency (Year)	**Value** (mg/L)	**Key Endpoint** (Reference)	**NOAEL** (mg/kg bw/d)	**UF**	**ADI** (mg/kg bw/d)	**BW** (kg)	**DW Intake** (L/d)	**AF** (%)	**Comments**
HC - proposed MAC (2019)	0.05	Cataracts in dogs (Hopkins, 1990)	0.5	100	0.005	74	1.53	20	
U.S. EPA (1992)	0.02	Cataracts in rats (Colley et al., 1985)	0.22	100	0.0022	70	2	20	
WHO (2016)	0.03	Cataracts in rats (Colley et a., 1985)	0.58	100	0.0058	60	2	20	ADI established by JMPR (FAO/WHO, 2014). JMPR states the 1-year dog study by Hopkins (1990) supports their ADI.
Australia (2011)	0.007	Cataracts in rats	0.2	100	0.002	70	2	10	No reference for the cataract study is provided in NHMRC and NRMMC, 2011 although description is consistent with Colley et al., 1985.
EU (1998)	0.1 µg/L	The EU has 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 to keep pesticides out of drinking water.							

ADI - Acceptable daily intake
AF - Allocation factor
BD - Body weight
DW - Drinking water
NOAEL - No adverse effect level
UF - Uncertainty factor

7.0 Rationale

Diquat is registered in Canada for use as a desiccant on crops and to control water-weeds and algae in still and slow-moving water. Despite its common use in Canada, data provided by provinces and territories that monitor for diquat in source and drinking water, indicate that levels of diquat are not significant. The eyes (cataract formation) are considered the target organ for diquat toxicity. Although no human studies have investigated the effects of diquat on eyes, animal studies conducted in several species (mice, rats, dogs) have consistently shown eye damage following repeated exposure to diquat.

Health Canada in collaboration with the Federal-Provincial-Territorial Committee on Drinking Water is proposing a MAC of 0.05 mg/L (50 µg/L) for diquat in drinking water based on the following considerations:

- An HBV of 0.05 mg/L (50 µg/L) based on cataract formation in dogs.
- Diquat can be accurately measured at concentrations well below the proposed MAC.
- Diquat can likely be removed at the municipal scale.

The proposed MAC is protective of potential health effects from diquat exposure. As part of its ongoing guideline review process, Health Canada will continue to monitor new research in this area and recommend any change to this guideline technical document that it deems necessary.

8.0 References

ALS Environmental (2019). Personal communication with A. Ganouri-Lumsden, Waterloo, ON.

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): 179-187.

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: 2795-2809.

British Columbia Ministry of Health (2019). Personal communication with D. Fishwick, Drinking Water Manager.

Bureau Veritas Laboratories (2019). Personal communication with C. MacDermid, Mississauga, ON.

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.pdf

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.

Circu, M.L., Maloney, R.E. and Aw, T.Y. (2017). Diquat-induced cellular pyridine nucleotide redox changes and alteration of metabolic enzyme activities in colonic carcinoma cells. Chem. Biol. Interact., 264: 43-51.

Colley, J., Warren, S. and Heywood, R. (1985). Diquat dibromide: Evaluation of potential carcinogenicity and chronic toxicity by prolonged dietary administration to rats. Final Report: HRC Report No. ICI 406-83763. Unpublished study prepared by Huntington Research Center; MRID 00145855 (as cited in U.S. EPA, 1995a).

Dichiara, A.B., Harlander, S.F., Rogers, R.E. (2015). Fixed bed adsorption of diquat dibromide from aqueous solution using carbon nanotubes. RSC Adv., 5(76): 61508-61512.

Duman, O., Özcan, C., Gürkan Polat, T. and Tunç, S. (2019). Carbon nanotube-based magnetic and non-magnetic adsorbents for the high-efficiency removal of diquat dibromide herbicide from water: OMWCNT, OMWCNT-Fe_3O_4 and OMWCNT-κ-carrageenan-Fe_3O_4 nanocomposites. Environ.Pollut., 244: 723-732.

EFSA (2015). Conclusion on the peer review of the pesticide risk assessment of the active substance diquat. European Food Safety Authority. EFSA Journal, 13(11): 4308.

Emmett, K. (2002). Risk assessments for diquat dibromide - appendix A (publication number 00-10-046). In: Final supplemental environmental impact statement for diquat dibromide. The Water Quality Program. Washington State Department of Ecology. Report Number 02-10-052.

FAO and WHO (2014). Pesticide residues in food - 2013. Report of the joint meeting of the FAO panel of experts on pesticide residues in food and the environment and the WHO core assessment group on pesticide residues. FAO plant production and protection paper 219. World Health Organization. Food and Agriculture Organization of the United Nations. Rome. Geneva, Switzerland.

FAO (2019). Pesticides residues in food - 2018: Residues and analytical aspects. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Core Assessment Group on Pesticide Residues. FAO Plant Production and Protection Paper 234. Food and Agriculture Organization of the United Nations. Rome.

Fletcher, K. (1972). Diquat dibromide: Three-generation reproduction study in rats. ICI study no. HO/IH/R/334A. DPR vol. 226-005 # 916116 (as cited in OEHHA, 2016).

Fuke, C., Ameno, K., Ameno, S., Kinoshita, H. and Ijiri, I. (1996). Detection of two metabolites of diquat in urine and serum of poisoned patients after ingestion of a combined herbicide of paraquat and diquat. Arch. Toxicol., 70(8): 504-507.

Fussell, K.C., Udasin, R.G., Gray, J.P., Mishin, V., Smith, P.J., Heck, D.E. and Laskin, J.D. (2011). Redox cycling and increased oxygen utilization contribute to diquat-induced oxidative stress and cytotoxicity in Chinese hamster ovary cells overexpressing NADPH-cytochrome P450 reductase. Free Radic. Biol. Med., 50(7): 874-882.

Gallagher, E.P., Buetler, T.M., Stapleton, P.L., Wang, C., Stahl, D.L. and Eaton, D.L. (1995). The effects of diquat and ciprofibrate on mRNA expression and catalytic activities of hepatic xenobiotic metabolizing and antioxidant enzymes in rat liver. Toxicol. Appl. Pharmacol., 134(1): 81-91.

Gomaa, H.M. and Faust, S.D. (1971). Kinetics of Chemical Oxidation of Dipyridylium Quaternary Salts. J. Agric. Food Chem., 19(2): 302-307.

Government of Ontario (2019). Drinking water surveillance program, available at: https://www.ontario.ca/data/drinking-water-surveillance-program

Gupta, P.K. (2014). Chapter 24 - Herbicides and fungicides. In: Biomarkers in toxicology. Gupta, R.C. (ed.). pp. 409-431.

Gupta, R.C. and Crissman, J.W. (2013). Chapter 42- Agricultural chemicals. In: Haschek and Rousseaux's Handbook of Toxicologic Pathology. Haschek, W.M., Rousseaux, C.G. and Wallig, M.A. (eds.). 3rd. pp. 1349-1372.

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.

Hantson, P., Wallemacq, P. and Mahieu, P. (2000). A case of fatal diquat poisoning: Toxicokinetic data and autopsy findings. J. Toxicol. Clin. Toxicol., 38(2): 149-152.

Hao, Y., Wang, Z., Gou, J. Wang, Z. (2015). Kinetics and thermodynamics of diquat removal from water using magnetic graphene oxide nanocomposite. Can.J.Chem.Eng., 93(10): 1713-1720.

Harling, R.J., Buist, D. and Gopinath, C. (1997). Diquat dibromide - Evaluation of potential carcinogenicity and chronic toxicity by prolonged dietary administration to rats: Addendum report 2: 2 year data. Report no. ICI 406/83763.Unpublished study prepared by Huntingdon Life Sciences, Huntingdon, Cambridgeshire, England (as cited in WHO, 2016).

Health Canada (2008). Proposed re-evaluation decision. PRVD2008-12. Diquat dibromide. Pest Management Regulatory Agency (PMRA); Health Canada, Ottawa, ON.

Health Canada (2010). Re-evaluation decision. PRVD2010-03. Diquat dibromide Pest Management Regulatory Agency (PMRA); Health Canada, Ottawa, ON.

Health Canada (2016). Pest control products sales report for 2016. PMRA, Health Canada, Ottawa, Ontario.

Health Canada (2019). Personal communication from the Health Evaluation Directorate, Pest Management Regulatory Agency (PMRA).

Hodge, M.C.E. (1989). Diquat: Teratogenicity study in the rabbit. ICI Study no. CTL/P/2379; MRID 41198901 (as cited in U.S. EPA, 1995a and OEHHA, 2016).

Hodge, M.C.E. (1990). Diquat: Multigeneration study in the rat. ICI Study no. CTL/P/2462. DPR vol. 226-090; MRID 41531301 (as cited in U.S. EPA, 1995a OEHHA, 2016).

Hodge, M.C.E. (1992). Diquat: Two year feeding study in mice. ICI Study no. CTL/P/3409; MRID 42219801 (as cited in U.S. EPA, 1995a OEHHA, 2016).

Hopkins, M.N. (1990). Diquat: 1 year feeding study in dogs. ICI Study no. CTL/P/2596. DPR vol. 226-094. Unpublished study prepared by ICI Central Toxicology Laboratory; MRID 41730301 (as cited in U.S. EPA, 1995a and WHO, 2016).

Horner, J.M. (1992a). Diquat: Subchronic neurotoxicity study in rats. ICI study no. CTL/P/3751.Unpublished study prepated by ICI Central Toxicology Laboratory; MRID 42616101 (as cited in U.S. EPA, 1995a).

Horner, J.M. (1992b). Diquat: Acute neurotoxicity study in rats. ICI Study no. CTL/P/3789. Unpublished study prepated by ICI Central Toxicology Laboratory; MRID 42666801 (as cited in U.S. EPA, 1995a and WHO, 2016).

HSDB (2010). Diquat dibromide. U.S. National Library of Medicine. National Institutes of Health, Health and Human Services, https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~9Eswj1:1:cpp.

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

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

Jones, G.M. and Vale, J.A. (2000). Mechanisms of toxicity, clinical features, and management of diquat poisoning: A review. Clin. Toxicol., 38(2): 123-128.

Jovic-Stosic, J., Babic, G. and Todorovic, V. (2009). Fatal diquat intoxication. Vojnosanit. Pregl., 66(6): 477-481.

Knappe, D.R.U., Snoeyink, V.L., Roche, P., Prados, M.J. and Bourbigot, M-M. (1999). Atrazine removal by preloaded GAC. J. Am. Water Works Assoc., 91(10): 97-109.

Krishnan, K. and 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.

Li, W., Zhao, Y., Yan, X., Duan, J., Saint, C.P. and Beecham, S. (2019). Transformation Pathway and Toxicity Assessment of Malathion in Aqueous Solution during UV Photolysis and Photocatalysis. Chemosphere 234: 204-214.

Li, Y., Zhao, R., Chao, S., Sun, B., Zhang, N., Qiu, J., Wang, C. Li, X. (2017). A flexible magnesium silicate coated electrospun fiber adsorbent for high-efficiency removal of a toxic cationic herbicide. New J. Chem., 41(24): 15601-15611.

Magalhães, N., Carvalho, F. and Dinis-Oliveira, R.J. (2018). Human and experimental toxicology of diquat poisoning: Toxicokinetics, mechanisms of toxicity, clinical features, and treatment. Hum. Exp. Toxicol., 37(11): 1131-1160.

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

Miltner, R.J., Baker, D.B., Speth, T.F., Fronk, C.A. (1989). Treatment of seasonal pesticides in surface waters. J. Am. Water Works Assoc., 81: 43–52. Available at: https://doi.org/10.1002/j.1551-8833.1989.tb03321.x

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

Minnema, D.J., Travis, K.Z., Breckenridge, C.B., Sturgess, N.C., Butt, M., Wolf, J.C., Zadory, D., Herberth, M.T., Watson, S.L., Cook, A.R. and Botham, P.A. (2016). Dietary administration of diquat for 13 weeks does not result in a loss of dopaminergic neurons in the substantia nigra of C57BL/6J mice. Regul. Toxicol. Pharmacol., 75: 81-88.

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://Nhmrc.Gov.Au/about-Us/Publications/Australian-Drinking-Water-Guidelines.

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

OEHHA (2016). Public health goal for carbofuran, diquat, endrin, picloram and thiobencarb in drinking water. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, U.S.

Palmer, A.K.; Edwards, J.A. and Woodhouse, R.N. (1978). Effect of diquat on pregnancy of the mouse. ICI Study no. 167/77642. Unpublished study prepared by Huntingdon Research Centre and submitted by Chevron Chemical Co.; MRID 00061637 (as cited in U.S. EPA, 1995a).

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

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

Sandy, M.S., Moldeus, P., Ross, D. and Smith, M.T. (1986). Role of redox cycling and lipid peroxidation in bipyridyl herbicide cytotoxicity: Studies with a compromised isolated hepatocyte model system. Biochem. Pharmacol., 35(18): 3095-3101.

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

Schmidt, D.M., Neale, J. and Olson, K.R. (1999). Clinical course of a fatal ingestion of diquat. J. Toxicol. Clin. Toxicol., 37(7): 881-884.

SGS Environmental Services (2019). Personal communication with D. Griffin. Lakefield, ON.

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. Drink. Water Eng. Sci., 2: 1-14.

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. AWWA Journal, 90(4): 171.

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.

Tanen, D.A., Curry, S.C. and Laney, R.F. (1999). Renal failure and corrosive airway and gastrointestinal injury after ingestion of diluted diquat solution. Ann. Emerg. Med., 34(4 Pt 1): 542-545.

U.S. EPA (1995a). Reregistration Eligibility Decision (RED). Diquat dibromide. List A. Case 0288. Office of Prevention, Pesticides and Toxic Substances. Special review and reregistration division. United States Environmental Protection Agency, EPA 738-R-95-016, Washington, D.C.

U.S. EPA (1995b). National Primary Drinking Water Regulations - Diquat. U.S. Environmental Protection Agency, EPA 811-F-95-003 m-T.

U.S. EPA (1997). Method 549.2 - Determination of Diquat and Paraquat in drinking water by liquid-solid extraction and high performance liquid chromatography with ultraviolet detection. Rev. 1.0.

U.S. EPA (2001). Diquat dibromide: Third HIARC report: Revisit/Re-evaluation of database by the HED hazard identification assessment review committee. U.S. Environmental Protection Agency, HED Doc No. 0050369, Washington, D.C.

U.S. EPA (2002). Diquat dibromide TRED facts. Office of Prevention, Pesticides and Toxic Substances. United States Environmental Protection Agency, Washington, D,C.

U.S. EPA (2011). Finalization of Guidance on Incorporation of Water Treatment Effects on Pesticide Removal and Transformation in Drinking Water Exposure Assessments.

U.S. EPA (2016). Occurrence Analysis for Potential Source Waters for the third Six-Year Review of National Primary Drinking Water Regulations. 810-R-16-008.

Valiante, F., Farinati, F., Dal Santo, P., Germana, B., Di Mario, F. and Naccarato, R. (1992). Upper gastrointestinal injury caused by diquat. Gastrointest. Endosc., 38(2): 204.

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: 435-445.

WHO (2004). Diquat 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/dwq/chemicals/diquat.pdf

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

WHO (2014). Pesticide residues in food - 2013: Toxicological evaluations. Joint meeting of the FOA panel of experts on pesticide residues in food and the environment and the WHO core assessment group on pesticide residues. Sponsored jointly by the Food and Agriculture Organization of the United Nations and World Health Organization. World Health Organization, Geneva, Switzerland.

WHO (2016). Diquat in drinking-water. Background document for development of WHO guidelines for drinking-water quality. World Health Organization, WHO/FWC/WSH/16.50, Geneva, Switzerland.

WHO (2017). Guidelines for Drinking-Water Quality. Fourth Edition. Incorporating the First Addendum. https://apps.who.int/iris/bitstream/handle/10665/254637/9789241549950-eng.pdf;jsessionid=D3717A19B57F35A4E2AF8E2D7CC11C83?sequence=1

Wickramaratne, G. (1989). Diquat: Teratogenicity study in the rat. ICI Study no. CTL/P/2331; Study No. RR0399. Unpublished study prepared by ICI Central Toxicology Laboratory; MRID 41198902 (as cited in U.S. EPA, 1995a).

Yao, C.C. and W.R. Haag (1991). Rate constant for direct reaction of ozone with several drinking water contaminants. Wat. Res., 25(7): 761-773.

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

Appendix A: List of acronyms

ADI	Acceptable daily intake
ANSI	American National Standards Institute
AOP	Advanced oxidation process
CAS#	Chemical Abstracts Service registry number
CCME	Canadian Council of Ministers of the Environment
EBCT	Empty bed contact time
EFSA	European Food Safety Authority
EU	European Union
FAO	Food and Agriculture Organization of the United Nations
FNIHB	First Nations and Inuit Health Branch
GAC	Granulated activated carbon
HBV	Health-based value
HPLC	High-performance liquid chromatography
IARC	International Agency for Research on Cancer
LD_{50}	Median lethal dose
LOQ	Limit of quantification
MAC	Maximum acceptable concentration
MCL	Maximum contaminant level
MDL	Method detection limit
MRL	Method reporting limit
MWCO	Molecular weight cut-off
NERL	National Exposure Research Laboratory
NF	Nanofiltration
NHMRC	National Health and Medical Research Council (Australia)
NOAEL	No-observed-adverse-effect level
NOM	Natural organic matter
NRMMC	Natural Resources Management Ministerial Council (Australia)
NSF	NSF International
OEHHA	Office of Environmental Health Hazard Assessment
PAC	Powdered activated carbon
P.E.I.	Prince Edward Island
PMRA	Pest Management Regulatory Agency
RNS	Reactive nitrogen species
RO	Reverse osmosis
ROS	Reactive oxygen species
SCC	Standards Council of Canada
TOPPS	1,2,3,4-tetrahydro-1-oxopyrido[1,2-a]-5-pyrazinium salt
U.S.	United States
U.S. EPA	United States Environmental Protection Agency
UV	Ultraviolet
WHO	World Health Organization

Appendix B: Provincial and Territorial anticipated impacts

Please note that this information is not available in both official languages because the source of the information is not subject to the Official Languages Act.

Alberta

No information submitted

British Columbia

No significant impact is expected by this change.

Manitoba

Manitoba has no data on diquat in drinking water. However, diquat is used in the province as a desiccant on seed and table potatoes. Although potatoes are typically grown on sandier soils, this is not expected to be a source of concern as diquat tends to be fixed in the soil and not leach. Therefore, the impact of the proposed lowering of the diquat in drinking water guideline to 0.05 mg/L is expected to be minimal in Manitoba.

New Brunswick

At this time, it is not anticipated that the lowering of the MAC for Diquat in drinking water (from .07 mg/L to 0.05 mg/L) will have any impacts for New Brunswick.

Newfoundland and Labrador

Newfoundland and Labrador has not conducted monitoring for diquat.

Of the 300 surface water sources in the province, 261 (87%) are protected under the Water Resources Act. Of the 179 groundwater sources in the province, 61 (34%) are protected under the Water Resources Act. This provides for an extensive source water protection program that reduces the risk of contamination for drinking water sources.

The existence of diquat within public water supplies is not common due to the 85% protection rate for drinking water supplies and the fact that herbicide spraying is not permitted in protected water supply areas.

The majority of Newfoundland and Labrador's drinking water systems do not have any treatment other than disinfection as 64% of the systems service very small populations (fewer than 500 people). Due to this fact, the ability for the majority of communities to treat for diquat contamination would be limited. The cost would have to be assessed on a case by case basis but the risk of diquat would be low due to the 87% protection rate of public water supplies.

Action items: Consider monitoring for diquat in public drinking water supply areas that have communities within the protected water supply areas and agriculture areas which rely on groundwater public water supplies.

Northwest Territories

No information submitted

Nova Scotia

No information submitted

Nunavut

No information submitted

Ontario

Ontario is supportive of the consultation document proposing to lower the Canadian Drinking Water Quality Guideline for diquat from 0.07 mg/L to 0.05 mg/L. Existing data indicates that the few detections of diquat in drinking water in Ontario were well below the proposed guideline value. This change will have no impact in Ontario.

Prince Edward Island

We do not expect any impact in consideration of lowering the diquat drinking water guideline from 0.07 to 0.05 mg/L within the Province of Prince Edward Island. As you will see from the exposure data, it is simply a compound we have not detected in drinking water samples, and given the detection / reporting limit for samples analyzed to date it would not appear likely that the lower value of 0.05 mg/L will change this.

Quebec

Au Québec, d'avril 2013 à mars 2018, 2319 analyses du diquat ont été réalisées dans le cadre de l'application du Règlement sur la qualité de l'eau potable, pour un total de 137 réseaux de distribution. Seuls les responsables de réseaux de distribution desservant plus de 5 000 personnes ont l'obligation de réaliser des analyses chaque trimestre pour vérifier le respect des normes pour les pesticides. Les responsables de réseaux, non visés par cette obligation, peuvent également juger pertinent de réaliser ces analyses.

Sur la période d'avril 2013 à mars 2018, aucune des analyses réalisées n'a présenté un résultat de diquat supérieur à la norme québécoise de 50 µg/L et la concentration maximale mesurée durant cette période est de 15 µg/L. Ainsi, aucune de ces analyses n'a dépassé la recommandation canadienne proposée de 50 µg/L pour le diquat dans l'eau potable. Par ailleurs, depuis 2012, dans le cadre de son Programme de surveillance de la qualité de l'eau potable, le Ministère réalise un projet de suivi des pesticides dans des installations de production d'eau potable alimentant 5 000 personnes et moins. Dans le cadre de ce projet, 157 analyses du diquat ont été réalisées pour 40 installations de production d'eau potable et ce pesticide n'a été détecté dans

aucune de ces analyses. Sur la base de ces résultats, les impacts attendus de la révision de la recommandation proposée sont faibles.

Saskatchewan

The Water Security Agency (WSA) has reviewed the proposed guideline document for Diquat in drinking water and supports the proposed MAC of 0.05 mg/L (50 µg/L).

A review of the provincial water quality database revealed that there is no monitoring data for Diquat levels in raw or treated drinking water or surface water in Saskatchewan. There are no studies (bench, pilot or full-scale) available for removing diquat from drinking water, however, researchers opinioned that membrane and adsorption (activated carbon) systems represent the best potential treatment systems for diquat removal from water. Water treatment plants (WTPs) in Saskatchewan regulated by the WSA continue to adopt these and other forms of systems for treating and supplying drinking water to consumers. Given the limited use of Diquat in the province especially for weed control activities and the current/evolving water treatment processes in use in the province, WSA believes that the new MAC for diquat if eventually adopted as a drinking water standard in the province may not pose a compliance challenge. WSA may monitor or request waterworks permittees to monitor diquat levels in treated drinking water once the national guideline is adopted, particularly for systems reliant on surface water for the raw water supply.

Yukon

It is not anticipated that the draft guideline technical document for diquat in drinking water will have a significant impact on large public drinking water systems in the Yukon.