Fond du Lac Band of Lake Superior Chippewa

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Nancy Schuldt Attachment

Denise Collins
Office of Administrative Hearings
Submitted via email to:
https://minnesotaoah.granicusideas.com/

Catherine O'Dell
Minnesota Pollution Control Agency
Submitted via email to:
Catherine.odell@state.mn.us

Feb. 11, 2022

Re: REQUEST FOR COMMENTS on Amendments being Considered to Rules Governing Water Quality Standards – Use Classification 1, Minnesota Rules chapters 7050, 7052, 7053, and 7060, Revisor's ID Number R-04727; OAH Discussion 37887

Dear Ms. Collins, Ms. O'Dell:

The Fond du Lac Band of Lake Superior Chippewa submits the following comments in response to the Minnesota Pollution Control Agency (MPCA) request for comments on amendments being considered to rules governing Water Quality Standards – Use Classification 1, Minnesota Rules chapters 7050, 7052, 7053, and 7060, Revisor's ID Number R-04727. These revisions to the rule language in chapters 7050 and 7060 under consideration are intended to address gaps and inconsistencies in their application to surface and groundwater.

Class 1 rule changes being considered include:

- Clarify and revise where the Class 1 water quality standards (WQS) apply; ensure the rule language clearly conveys that the standards apply to all groundwater. MPCA is also considering whether and how to expand the Class 1 designation to surface waters that: 1) are strongly connected to and impacting the quality of underlying/nearby groundwater, and 2) flow into and impact the quality of a designated Class 1 surface water. These additions are being considered to better protect sources of drinking water.
- Revise the numeric and narrative WQS. This includes updating existing values to be more health protective and adding WQS for some emerging pollutants of concern, including per- and polyfluoroalkyl substances (PFAS), and potentially pesticides, pharmaceuticals, algal toxins, disinfection by-products, and/or additional industrial chemicals.
- Consider whether to add the concept of Groundwater Contaminant Management Zones (GWCMZs) – a mechanism to identify contaminated groundwater and inform decision makers and the public of contamination.

The Band offers our feedback to several of these potential rule changes and questions posed by MPCA in this request for public comment

Clarify inclusion of groundwater as a Class 1 water in Minn. R. ch. 7050.

The Band strongly supports the objective of rule language that clarifies, unequivocally, that Class 1 WQS apply to all groundwater. However, we call attention to the recent Minnesota Supreme Court ruling on litigation brought by the Band and WaterLegacy, along with counsel for MPCA, in the case of *In re Reissuance of an NPDES/SDS Permit to United States Steel Corp. ("U.S. Steel"*), which offered a unified interpretation of Minnesota chapters 7050 and 7060 rules that applied all Class 1 WQS to protect groundwater. It could actually be problematic, rather than beneficial, to alter chapter 7050 or chapter 7060 rule language on which the Court's opinion is based.

In that ruling, the Court stated:

> [C]hapter 7060 makes clear that the highest priority use for groundwater is "as a source of drinking, culinary, or food processing water." Minn. R. 7060.0400 (2019). And in light of "the policy of the agency to consider the actual or potential use of the underground waters for potable water supply as constituting the highest priority use and as such to provide maximum protection to all underground waters," Minn. R. 7060.0200, the agency classified all groundwater "for use as potable water supply," Minn. R. 7060.0400. "Potable water" means "water which is or may be used as a source of supply for human consumption including drinking, culinary use, food processing, and other similar purposes, and which is suitable for such uses in its untreated state or when treated using generally recognized treatment methods." Minn. Stat. § 115.01, subd. 14.

Subsequently, the Court united and affirmed the definitions in Minn. R. chs. 7050 (Class 1) and 7060 (groundwater), noting:

> These standards for groundwater in chapter 7060 look strikingly similar to the definition of Class 1 waters in chapter 7050: "[A]ll waters of the state that are or may be used as a source of supply for drinking, culinary or food processing use, or other domestic purposes and for which quality control is or may be necessary to protect the public health, safety, or welfare." Minn. R. 7050.0140, subp. 2. The overlap between the classification of groundwater in chapter 7060 as potable water with a highest priority use as a source of drinking, culinary, or food processing water and the definition of Class 1 waters as a source of supply for drinking, culinary, or food processing use is another strong signal that groundwater is reasonably classified as a Class 1 water.

The MPCA's policy and practice, as evidenced in numerous agency Statements of Need and Reasonableness (SONARs) over many decades has sustained the agency's consistent interpretation that all groundwater is Class 1 water. In *U.S. Steel*, the Court recognized that MPCA's regulatory authority to protect groundwater applied to Class 1 WQS adopting national secondary as well as primary drinking water standards:

> Because the MPCA's interpretation of the ambiguous regulations contained within chapters 7050 and 7060 as classifying all groundwater as a Class 1 water is reasonable, longstanding, and supported by the evolution of the regulatory scheme, we hold that groundwater is a Class 1 water and that the MPCA properly

> exercised its authority in applying the Class 1 secondary drinking water standards to the 2018 Permit.

Thus, it seems apparent that any perceived lack of clarity in this issue has already been sufficiently addressed through the Minnesota Supreme Court ruling, and supporting MPCA's longstanding policy.

Add rule language specifying that MDH is the state agency that oversees drinking water treatment under the federal SDWA.

While Minn. R. ch. 7060 specifically cites the role of MDH as "setting treatment and other requirements to ensure the potability of underground water", this proposed rulemaking is an opportunity to also clarify MPCA's role and authorities for protecting the state's groundwater resources, which should be integrative and supportive of the roles of other state agencies (i.e., MN Department of Natural Resources (MN DNR) and the Minnesota Department of Health (MDH)). For this specific authority under the Safe Drinking Water Act, MDH is the responsible state agency and it would serve the public well to expressly state that in Minn. R. ch. 7050.

This rulemaking is also a time when the MN DNR's roles and responsibilities related to groundwater protection could be highlighted. The various state agencies each have distinct responsibilities but the overarching objective is for all agencies to work together for the protection of Minnesota's groundwater resources. From the 2010 statutorily-required report to the Minnesota Legislature, *Long-term Protection of the State's Surface Water and Groundwater Resources,*

> Over the last decade, the DNR has been heavily engaged in the development of our own reports, and reports of other agencies and institutions on water sustainability, water availability, groundwater protection and management, and surface water protection and management.
>
> In summary the Department recommends the following strategies:
>
> - Encourage and influence local engagement in management, prevention and demonstration efforts.
> - Deliver up-to-date protection tools and recommended best management practices.
> - Adopt a long-term focus for monitoring and prevention activities.
> - Enhance data collection and sharing and simplify public access to data.
> - Answer key questions and meet key information needs.
> - *Approach groundwater and surface water management and protection in a watershed context as a comprehensive hydrologic-ecologic system.* (emphasis added)
> - Provide adequate financial and technical resources at appropriate levels to maximize the effective management and protection of water resources. Well-conceived and competently administered programs will not provide long-term protection if inadequately funded.[1]

[1] https://files.dnr.state.mn.us/publications/waters/long-term_protection_surface_ground_water_201001.pdf , last visited 2-11-2022

Improve or remove Class 1 subclasses.

Existing subclasses have not to date been implemented in groundwater, nor have they offered any meaningful or additional protection to surface water. The Band strongly recommends that Class 1 subclasses be removed, for clarity and simplicity of enforcement. No such tiering of groundwater is necessary to enforce all Class 1 WQS, nor any rule amendment. Existing rules state, "Class 1 waters, domestic consumption…*includes all waters* of the state that are or may be used as a source of supply for drinking, culinary or food processing use, or other domestic purposes and for which quality control is or may be necessary to protect the public health, safety, or welfare." Minn. R. 7050.0140, subp. 2 (emphasis added).

Review and update surface waters that have Class 1 designations.

The Band supports MPCA maintaining all current Class 1 designations. We will be interested to review any forthcoming protocols and rationale developed for adding the Class 1 use designation to additional state waters.

Specify application to surface waters that are impacting the quality of Class 1 surface waters, groundwater.

This scenario should be a clear indication of the need to add Class 1 protections to new surface waters. Applicable and enforceable WQS must be sufficiently protective of downstream uses. MPCA raises the concern that nitrate pollution of surface waters that are not currently identified as Class 1 waters is affecting both downstream surface waters and ground waters used for drinking water. However, MPCA already has the authority to address nitrate pollution of surface waters that would not require new classifications or extensive monitoring that could result in years, if not decades, of delay. CWA §§ 402 and 303(d) and the state's implementing rules already provide the regulatory framework to protect downstream Class 1 surface waters from upstream point- and nonpoint source discharges of nitrates, as well as other pollutants, and should be fully enforced to protect downstream drinking water (surface or ground water).

Consider removal of designations where drinking water use is not occurring (e.g., Class 2A: cold-water, aquatic communities).

The Band emphatically opposes any removal of beneficial use designations simply because that use is not currently occurring. We support MPCA's preliminary decision to not move forward with a categorical disassociation of the Class 1 domestic consumption use and associated protections from Class 2A waters.

There is no compelling reason or justification for the removal of secondary drinking water MCLs. National Primary Drinking Water MCLs are mandatory for state public water systems, 40 C.F.R. § 141.3. National Secondary Drinking Water MCLs are recommended standards intended to serve "as guidelines for the States." 40 C.F.R. § 143.1. EPA has determined that secondary MCLs "are requisite to protect the public welfare" and "are "reasonable goals for drinking water quality." 40 C.F.R. § 143.3. Ever since 1983 when Minnesota's chapter 7050 rules were first enacted, MPCA has

applied both federal mandatory and federal recommended drinking water standards to protect Class 1 waters, including groundwater.

Revise numeric standards (update and add pollutants)

MPCA is considering a rule amendment to add emerging pollutants of concern to Class 1 WQS. In concept, such a rule amendment would be a positive step to ensure that drinking water standards in Minnesota adequately protect public health. Health Risk Limits (HRLs) are developed using risk assessment methods and toxicologic data from the US EPA. MPCA does not need to assess contaminant risks to accomplish this protection; the MDH has already done the scientific and epidemiologic research needed to protect human health from contaminants in drinking water:

> The 1989 Groundwater Protection Act authorizes MDH to review, develop and adopt health-protective guidance known as Health Risk Limits (HRLs) when groundwater quality monitoring results show the presence of contaminants. The safe drinking water standards specified in the 2001 Health Standards Statute require that the standards be based on scientific methods and be protective of vulnerable subpopulations such as infants and children.
>
> HRLs are used by partner state agencies for water monitoring and risk management purposes. HRL values are formally adopted through rulemaking. MDH plans to propose additional amendments to the HRL Rules in 2021 or 2022.[2]

The best and most straightforward way to ensure that MPCA numeric criteria are up-to-date with current science is to simply adopt all of the MDH HRLs for drinking water, plus continue to assess groundwater quality compliance using US EPAs Safe Drinking Water Act (SDWA) criteria including those limits set for secondary drinking water contaminants, without exemptions.

Enforcing both SDWA and MDH HRLs for secondary drinking water contaminants is critically important because many criteria that do not have maximum contaminant levels listed in the SDWA, or are only listed under Secondary Drinking Water Regulations can have serious human health impacts; manganese is a prime example. In the SDWA, manganese is considered a secondary contaminant with a limit of 50 micrograms per liter (µg/L). However, drinking water above a concentration of 100 µg/L manganese can cause Parkinson's Disease-like nervous system symptoms. Chloride is another example of a pollutant that at low concentrations is not considered generally harmful to human health. But at concentrations above the 250 milligrams per liter (mg/L) SDWA Secondary Criteria, chloride can adversely impact people suffering from heart and kidney disease. Sulfate is also considered a secondary contaminant with a SDWA limit of 250 mg/L to protect people from its "laxative effect". Limits set by HRLs *and* SDWA secondary contaminant criteria for sulfate and manganese are particularly important limits that protect the health of formula-fed infants.

[2] https://www.health.state.mn.us/communities/environment/risk/rules/water/index.html. Last visited 2/11/22

Adding new Class 1 WQS for emerging pollutants of concern, including per-and polyfluoroalkyl substances (PFAS), can and should be part of the planned amendments. The SDWA also includes maximum contaminant levels listed for Cryptosporidium, *Giardia lamblia*, Legionella, Heterotrophic Plate Count, Total Coliforms, and viruses, as well as requiring specific treatment techniques to reduce these contaminants in the public water supply. Disinfection by-products, especially Trihalomethanes, are also limited under the SDWA. Again, MPCA should rely on MDH and US EPA to determine what the maximum drinking water contaminant levels for those pharmaceuticals and industrial chemicals that do not yet have drinking water limits should be to protect human health.

Consider adding Groundwater Contaminant Management Zones (GWCMZs) to Minn. R. ch. 7060

The Band is uncertain as to the utility of this proposed action, and supports MPCA's decision not to pursue this in rulemaking. It is far more important for environmental integrity and protecting human health to clean up areas of groundwater contamination, including Superfund sites. Data in the MPCA's Groundwater Contamination Atlas provides a public transparency benefit; however, by designating GWCMZs it is reasonably foreseeable in a time when environmental cleanup dollars are difficult to secure, that it would instead have the effect of codifying areas of contamination rather than prioritizing their remediation.

The objective of the federal and state Superfund programs, as well as the EPA Brownfields program and the state's Voluntary Investigation and Cleanup or VIC program, is to remediate contamination so that contaminated groundwater will meet groundwater standards, not to preserve in perpetuity the contaminated status of sites. Designation of GWCMZs could very well undermine this objective.

Summary

The Band appreciates this opportunity to provide input early in the rulemaking process for MPCA's Class 1 WQS updates. We look forward to reviewing and commenting upon draft rule language and SONAR documentation in the future.

Sincerely,

Nancy Schuldt, Water Projects Coordinator
Fond du Lac Environmental Program

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Paula Maccabee Attachment 1

February 14 , 2022

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Comments also filed with OAH
(https://minnesotaoah.granicusideas.com/)

RE: Possible Class 1 Rule Amendments (Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

Dear Ms. O'Dell, Minnesota Pollution Control Agency Staff,

The following comments are submitted on behalf of WaterLegacy, a Minnesota non-profit organization founded to protect water from industrial pollution and to ensure that water quality standards are preserved, strengthened, and enforced to protect Minnesota's freshwater resources and human health. Additional conservation organizations joining these comments include: Friends of the Boundary Waters Wilderness, Honor the Earth, Minnesota Division of the Izaak Walton League of America, North American Water Office, Northeastern Minnesotans for Wilderness, Northern Lakes Scientific Advisory Panel, and W.J. McCabe (Duluth) Chapter of the Izaak Walton League of America.

The Minnesota Pollution Control Agency's ("MPCA") Rule Concepts Narrative states that the "main purpose" of the proposed Class 1 waters rulemaking "is to improve protection of Minnesota waters used for domestic consumption, which are all groundwater and Class 1 surface waters that are specifically identified in rule."[1] WaterLegacy and other conservation organizations support this stated objective.

However, we are concerned that several of the potential changes proposed by the MPCA would reduce rather than increase the protection for groundwater now applicable under Minnesota law.

Our comments are summarized as follows:

1) MPCA should not use new definitions that would disrupt the unity of groundwater protection and Class 1 water quality standards ("WQS") recently achieved by the 2021 Minnesota Supreme Court decision in the U.S. Steel Minntac case. The regulatory framework is sound, and "fixing" the definitions will do harm.

2) MPCA should treat all groundwater as Class 1 water, without subclasses, and should state clearly that all Minnesota groundwater is protected by Class 1 WQS.

[1] MPCA, Concepts for Amendments to Water Quality Standards Rules, Class 1 Waters, Dec.13, 2021 (Class 1 Concepts), available at https://www.pca.state.mn.us/sites/default/files/wq-rule4-24b.pdf

3) MPCA should not change current chapter 7050 rules that require compliance with both National Primary and Secondary Drinking Water Maximum Contaminant Levels ("MCLS") other than to remove exceptions for copper and lead. Class 1 WQS should be at least as restrictive as the lowest applicable drinking water standard.

4) MPCA should adopt Minnesota Health Department Health Risk Limits ("HRLs") as Class 1 WQS to prevent toxic contamination of Minnesota drinking water.

5) MPCA should address concerns about nitrate pollution of surface waters by prioritizing adoption of Class 2 nitrate WQS.

6) MPCA should not remove drinking water protections from Class 2A surface waters.

7) MPCA should clean up rather than codify areas where groundwater is contaminated and strengthen enforcement to remove and prevent groundwater pollution.

1. MPCA should not "fix" the unity of groundwater protection provided by the Minnesota Supreme Court's recent U.S. Steel Minntac decision.

WaterLegacy and the Fond du Lac Band of Lake Superior Chippewa litigated the U.S. Steel Corp. Minntac tailings basin case alongside counsel for the MPCA. The Minnesota Supreme Court ruling in this case, *In re Reissuance of an NPDES/SDS Permit to United States Steel Corp. ("U.S. Steel"*), 954 N.W.2d 572 (Minn. 2021), resulted in a unified interpretation of Minnesota chapters 7050 and 7060 rules that apply all Class 1 water quality standards to protect groundwater. The *U.S. Steel* decision was a major victory for protection of groundwater. It would be harmful, rather than beneficial, to alter chapter 7050 or chapter 7060 rule language on which the Court's opinion rests.

Excerpts from the Minnesota Supreme Court ruling in the *U.S. Steel* case quoted below demonstrate that the strongest and most coherent protection for Minnesota groundwater is to enforce, rather than deconstruct, existing rules protecting groundwater as potable water. The Court stated the policy of protecting groundwater for drinking, culinary, or food processing use:

> [C]hapter 7060 makes clear that the highest priority use for groundwater is "as a source of drinking, culinary, or food processing water." Minn. R. 7060.0400 (2019). And in light of "the policy of the agency to consider the actual or potential use of the underground waters for potable water supply as constituting the highest priority use and as such to provide maximum protection to all underground waters," Minn. R. 7060.0200, the agency classified all groundwater "for use as potable water supply," Minn. R. 7060.0400. "Potable water" means "water which is or may be used as a source of supply for human consumption including drinking, culinary use, food processing, and other similar purposes, and which is suitable for such uses in its untreated state or when treated using generally recognized treatment methods." Minn. Stat. § 115.01, subd. 14.

U.S. Steel, 954 N.W.2d at 579-80. The Court then unified the definitions of Class 1 waters in chapter 7050 and groundwater in chapter 7060:

> These standards for groundwater in chapter 7060 look strikingly similar to the definition of Class 1 waters in chapter 7050: "[A]ll waters of the state that are or may be used as a source of supply for drinking, culinary or food processing use, or other domestic purposes and for which quality control is or may be necessary to protect the public health, safety, or welfare." These standards for groundwater in chapter 7060 look strikingly similar to the definition of Class 1 waters in chapter 7050: "[A]ll waters of the state that are or may be used as a source of supply for drinking, culinary or food processing use, or other domestic purposes and for which quality control is or may be necessary to protect the public health, safety, or welfare." Minn. R. 7050.0140, subp. 2. The overlap between the classification of groundwater in chapter 7060 as potable water with a highest priority use as a source of drinking, culinary, or food processing water and the definition of Class 1 waters as a source of supply for drinking, culinary, or food processing use is another strong signal that groundwater is reasonably classified as a Class 1 water.

Id. at 580. The Court also explained MPCA's consistent state policy protecting all groundwater for Class 1 drinking water use.

> Since at least 1993, the MPCA (under a variety of administrations) has unequivocally and consistently stated in Statements of Need and Reasonableness (SONARs) that groundwater is a Class 1 water. *See Citizens Advocating Responsible Dev. v. Kandiyohi Cnty. Bd. of Comm'rs (CARD)*, 713 N.W.2d 817, 830 (Minn. 2006) (relying on agency SONARs as evidence of regulatory intent).
>
> In a 1993 SONAR, the MPCA stated that "[g]round waters (Class 1) are protected for just one beneficial use, drinking water, and only the drinking water standards apply to ground waters." 1993 SONAR 49 (Apr. 1993). A 2006 SONAR highlighted that chapter 7050 "contains statewide provisions that protect Minnesota's surface and ground water resources from pollution" before going on to state that "all ground water is protected for just one use, as an actual or potential source of drinking water (Class 1)." 2006 SONAR 1, 3 (May 2006). More recently, in a 2014 SONAR, the MPCA stated that "Minn. R. ch. 7050 addresses drinking water use through the Class 1 Domestic Consumption (DC) designation. Class 1 applies to all groundwater and specified surface waters." 2014 SONAR 5 (June 2014); *see also* 2007 SONAR 6 (July 2007) ("In Minnesota all ground water is protected as an actual or potential source of drinking water (Class 1)."); 2013 SONAR 8 (Nov. 2013) ("In Minnesota all ground water is protected as an actual or potential source of drinking water (Class 1 Domestic Consumption).").
>
> This history makes clear that the MPCA interprets the rules to mean that all groundwater is Class 1 water.

Id. at 581-82 (footnotes omitted). Finally, the Court recognized that MPCA has regulatory authority to protect groundwater by applying Class 1 WQS that have adopted national secondary as well as primary drinking water standards.

> Because the MPCA's interpretation of the ambiguous regulations contained within chapters 7050 and 7060 as classifying all groundwater as a Class 1 water is reasonable, longstanding, and supported by the evolution of the regulatory scheme, we hold that groundwater is a Class 1 water and that the MPCA properly exercised its authority in applying the Class 1 secondary drinking water standards to the 2018 Permit.

Id. at 583.

In colloquial terms, now that the Minnesota Supreme Court has ensured that there is *nothing* broken in Minnesota's groundwater protection rules or MPCA's authority to enforce them, the MPCA must not "fix" its definitions of Class 1 beneficial use.

Such rulemaking would destroy the coherent and protective ruling made by the Minnesota Supreme Court and reduce protection of groundwater for drinking water as a matter of public welfare and public health. Industrial polluters of groundwater would benefit at the expense of all other Minnesotans, including residents who drink well water and residents whose taxes pay the costs of wastewater treatment to make polluted water drinkable.

2. MPCA should protect all groundwater, without division, as Class 1 water.

The MPCA should protect all groundwater, without division, as Class 1 water. Minnesota Rule 7060.0400 already reflects the concept of unified groundwater classification:

> The waters of the state are classified according to their highest priority use, which for underground waters of suitable natural quality is their use now or in the future as a source of drinking, culinary, or food processing water . . . In making this classification, the agency recognizes that the underground waters of the state are contained in a series of related and often interconnected aquifers, such that if sewage, industrial waste, other waste, or other pollutants enter the underground water system, they may spread both vertically and horizontally. Thus, all underground waters are best classified for use as potable water supply in order to preserve high quality waters by minimizing spreading of pollutants, by prohibiting further discharges of wastes thereto, and to maximize the possibility of rehabilitating degraded waters for their priority use.

Although Minn. R. 7050.0221 suggests that groundwater might be divided into classes 1A, 1B, and 1C, creating tiers of groundwater is scientifically problematic and less protective than applying Class 1 WQS uniformly to groundwater. No such tiering of groundwater is necessary to enforce all Class 1 WQS. *U.S. Steel*, 954 N.W.2d at 583.

There is no need for any rule amendment to protect *all* groundwater from pollution exceeding Class 1 WQS. Existing rules state, "Class 1 waters, domestic consumption. . *includes all waters* of the state that are or may be used as a source of supply for drinking, culinary or food processing use, or other domestic purposes and for which quality control is or may be necessary to protect the public health, safety, or welfare." Minn. R. 7050.0140, subp. 2 (emphasis added).

Existing rules are consistent with state statutes. Minnesota Statutes state that *all* groundwater is protected from pollution irrespective of the use or ownership of surface lands. "Groundwater" means "water contained below the surface of the earth in the saturated zone *including, without limitation, all waters*" in various subsurface conditions. Minn. Stat. § 115.01, subd. 6 (emphasis added). "Pollution of water," similarly, means "the discharge of any pollutant into *any waters of the state* or the contamination of *any waters of the state*" so as to be actually or potentially harmful to public health, safety or welfare or uses of water, including domestic drinking water uses. *Id*., subd. 13. Finally, "waters of the state" means "*all* . . . wells, springs, reservoirs, aquifers, irrigation systems, drainage systems and *all other bodies or accumulations of water, surface or underground, natural or artificial, public or private*" in or bordering any part of Minnesota. *Id.*, subd. 22 (emphasis added).

Tiers of groundwater must be rejected, and existing rules protecting all groundwater for current drinking use or that of future generations must be retained and strictly applied.

3. MPCA should retain all federal MCLs as Class 1 WQS to protect groundwater, without exemptions.

Although most of the intended objectives in MPCA's Class 1 Concepts are opaque, MPCA has stated one definite intention. MPCA is conducting the Class 1 rulemaking in order to *remove* National Secondary Drinking Water Maximum Contaminant Levels ("MCLs") from Class 1 water quality standards and reduce protection of groundwater from these contaminants. This proposal is contrary to public health and public welfare.

In proposing deregulation of secondary MCL contaminants, MPCA misrepresents federal drinking water laws and policies, asserting that "[u]nder the federal Clean Water Act (CWA), WQS for the protection of domestic consumption should be solely based on human health considerations." (Class 1 Concepts, p. 2).

In fact, the Clean Water Act requires that state water quality standards shall protect both public "health" and public "welfare." 33 U.S.C. § 1313(c)(2)(A). More pointedly, MCLs to protect drinking water quality are established by the EPA under the 1974 Safe Drinking Water Act (Public Law 93-522), not under the Clean Water Act.

National Primary Drinking Water MCLs are mandatory for state public water systems, 40 C.F.R. § 141.3. National Secondary Drinking Water MCLs are recommended standards intended to serve "as guidelines for the States." 40 C.F.R. § 143.1. Regarding secondary MCLs, although federal law does not require that all water systems must meet these standards, the "EPA recommends them

to the States as reasonable goals."[2] Federal regulations also state that EPA has determined that secondary MCLs "are requisite to protect the public welfare" and "are "reasonable goals for drinking water quality." 40 C.F.R. §§ 143.2(f), 143.3. Since 1983 when Minnesota's chapter 7050 rules were first codified, these Minnesota state rules have applied both federal mandatory and federal recommended drinking water standards to protect Class 1 waters, including groundwater.[3]

There are strong policy reasons to apply secondary MCLs to protect drinking water. Secondary contaminants both harm public health and impair public welfare by making water undrinkable and increasing treatment costs for public water systems. Minnesota legislative policy to protect potable water finds that the waters of the state "constitute a unique natural resource of immeasurable value which must be protected and conserved for the benefit of the *health, safety, welfare, and economic well-being of present and future generations* of the people of the state." Minn. Stat. § 115.063(a)(1) (emphasis added). As a result, "the actual or potential use of the waters of the state for potable water supply is the highest use of that water and deserves maximum protection by the state." *Id*.(a)(2). The definition of Class 1 waters for drinking water and other domestic uses similarly requires protection of waters as necessary to "protect the public health, safety, or welfare." Minn. R. 7050.0140. There is no basis in federal law, state policy, or existing Class 1 rules to assert that Class 1 WQS should be solely based on health considerations.

However, many contaminants listed as secondary MCLs have adverse health effects. Copper now has a primary MCL of 1.3 mg/L due to effects on the gastrointestinal tract, the liver, and the kidneys. Fluoride has a primary MCL of 4.0 mg/L due to effects on teeth and bone disease.[4] Other secondary MCLs also have proven adverse public health effects, although primary MCLs have not yet been promulgated. Manganese harms the developing brains of children and infants. The Minnesota Health Department ("MDH") has set a Health Risk Limit ("HRL") of 100 µg/L (0.1 mg/L) for manganese,[5] while the World Health Organization ("WHO") recently recommended an even more stringent health-based limit of 80 µg/L (0.08 mg/L) to protect bottle-fed infants.[6] The WHO has also recommended a health-based guideline of 0.1 mg/L for silver, identical to EPA's secondary MCL, in order to prevent toxicity in long-term exposure.[7]

A 15-year follow up study in a prominent peer reviewed journal found that cognitive decline from dementia and Alzheimer's disease was greater in subjects with daily aluminum intake from drinking water higher than 0.1 mg/day ($p = 0.005$).[8] In addition to data from thousands of randomly selected cohorts, Rondeau cited animal studies, the presence of aluminum in senile plaques and neurofibrillary degeneration, and ecological studies suggesting that concentrations of aluminum in

[2] EPA, National Secondary Drinking Water Standards, online page, Exhibit 1.
[3] Minnesota Rules1983 Chapter 7050.0220 Rules excerpt, Exhibit 2, pdf 2-3.
[4] EPA, National Primary and National Secondary Drinking Water Regulations ("EPA MCLs"), Exhibit 3, pdf 2, 4.
[5] MDH, Human Health-Based Water Guidance Table ("MDH HRLs"), Exhibit 4, pdf 21.
[6] WHO, Manganese in Drinking-water, WHO/HEP/ECH/WSH/2021.5, 2021, Exhibit 5, p. 37.
[7] WHO, Silver in Drinking-water, WHO/SDE/WSH/03.04/14, 2014, Exhibit 6, p. 3; compare Exhibit 3.
[8] Rondeau *et al*., Aluminum and silica in drinking water and the risk of Alzheimer's disease or cognitive decline: findings from 15-year follow-up of the PAQUID cohort, *Am. J. Epidemiol*. 2009 Feb:169(4): 489-496 (Rondeau 2009), Exhibit 7.

drinking water of 0.1-0.2 mg/L may increase relative risk of Alzheimer's disease by ratios of 1.35 to 2.67. (Rondeau 2009). The secondary MCL currently in Class 1 WQS proscribes more than 0.2 mg/L of aluminum. (Exhibit 3).

The impetus for MPCA's attempt to remove secondary MCLs may be related to conditions in Minntac tailings basin NPDES/SDS permit that required U.S. Steel's eventual compliance with Minnesota's Class 1 groundwater sulfate standard. Enforcement of this groundwater sulfate standard was specifically affirmed by the Minnesota Supreme Court, which ruled, "we hold that groundwater is a Class 1 water and that the MPCA properly exercised its authority in applying the Class 1 secondary drinking water standards to the 2018 Permit, including the 250 mg/L sulfate standard. *U.S. Steel*, 954 N.W.2d at 583.

In a normal regulatory system, the Court's decision supporting agency authority would provide finality. In Minnesota's paradoxical regulatory system, the MPCA's authority to control sulfate pollution seems to be the very problem its Class 1 rulemaking seeks to address.

The EPA has recommended a secondary MCL of 250 mg/L for sulfate based on the fact that people will reject water as undrinkable due to taste considerations at that level. (Exhibit 3). The MDH explains that sulfate levels above 250 mg/L "may make the water taste bitter or like medicine" as well as corroding plumbing, particularly copper pipes.[9] The MDH advises that sulfate can cause diarrhea and dehydration, particularly in bottle-fed infants. (*Id.*)

There is little recent United States research on health effects of sulfate in drinking water. But a 2012 study in Pakistan referenced the EPA's 250 mg/L sulfate guideline and found that individuals moving into areas with sulfate concentrations higher than the sulfate guideline complained of health effects such as gastroenteritis.[10] The author emphasized that "existing data do not identify the level of sulfate in drinking water that would be unlikely to cause adverse human health effects." (Bashir 2012). In particular, the safe dose of sulfate below which infants would be protected has not been determined "partly because of the difficulty in locating a population of women feeding their infants formula mixed with unfiltered tap water containing high levels of sulfate." (*Id*.)

The U.S.D.A. National Institute of Food and Agriculture Extension also explains that elevated sulfate and the presence of sulfur-reducing bacteria in drinking water can result in the formation of hydrogen sulfide.[11] However, a "concentration high enough to be a drinking water health hazard also makes the water unpalatable," since less than 1 mg/L of hydrogen sulfide in water results in a "swampy" odor, and 1-2 mg/L of hydrogen sulfide "gives water a 'rotten egg' odor and makes the water very offensive." (USDA Drinking Water). If excessive sulfate or hydrogen sulfide is present in private drinking water, consumers would need to obtain an alternative water supply, try some type of treatment, or buy bottled water. (*Id*.).

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[9] MDH, Sulfate in Well Water Fact Sheet, Aug. 2, 2019, Exhibit 8.
[10] Bashir *et al*, Health Effects from Exposure to Sulphates and Chlorides in Drinking Water, *Pakistan J. of Med. & Health Sciences*, Vol. 6, No. 3, Jul-Sep 2012, Exhibit 9.
[11] U.S.D.A. National Institute of Food and Agriculture Extension, Drinking Water Contaminant – Sulfur, hydrogen sulfide, Aug. 23, 2019 ("USDA Drinking Water"), Exhibit 10.

Removing the existing Class 1 groundwater sulfate standard would impair both public health and public welfare. This WQS must be preserved to protect the health of infants and other vulnerable populations, preserve potable drinking water, and avoid a cost shift to private well owners and public water treatment systems so that polluters can avoid costs to control their pollution.

The MPCA's Class 1 Concepts did not discuss exemptions in existing Class 1 groundwater WQS from primary MCLs for copper and lead. Minn. R. 7050.0221, subp. 1(B). Due to these exemptions, Minnesota has no numeric WQS for copper or lead in groundwater, though both metals cause adverse health effects at or below the primary MCLs set by EPA.[12] As more industrial sources of groundwater copper and lead pollution seek permits, exemptions for these pollutants from Class 1 standards must be removed to protect drinking water from toxic levels of copper and lead.

In summary, Minnesota Class 1 WQS should, without exception, be at least as restrictive as the lowest applicable national drinking water standard.

4. MPCA should adopt Minnesota HRLs as Class 1 WQS.

MPCA's Class 1 Concepts propose a rule amendment to add emerging pollutants of concern to Class 1 WQS. Such a rule amendment would be an important positive step to ensure that drinking water standards in Minnesota adequately protect public health, including the health of fetuses, infants, the elderly, and other vulnerable populations. It is not necessary for the MPCA to assess contaminant risks to accomplish this protection; the MDH has already done the scientific and epidemiologic research needed to protect human health from contaminants in drinking water.

It is strongly recommended that where HRLs developed by the MDH are more protective than primary federal MCLs the MPCA's chapter 7050 Class 1 WQS should adopt HRLs to protect drinking water, including both groundwater and surface water.

MDH has already conducted the research needed to fill in gaps or strengthen protections required by federal MCLs in order to protect the health of Minnesotans. For several toxic contaminants potentially released to Minnesota drinking water, such as benzene (cancer), beryllium (intestinal lesions), and cadmium (kidney damage), HRLs are more stringent than primary MCLs.[13] These HRLs should be adopted as Class 1 WQS.

For many contaminants in addition to the toxic metals discussed previously, MDH has developed HRLs where EPA research has yet to develop an MCL. For example, MDH has developed HRLs for toxic pesticides, including DDD, DDE, and DDT (Exhibit 11), which may be outside EPA's jurisdiction due to the structure of federal law. MDH has also developed an acute HRL for PFOA

[12] The MCL for copper and the Maximum Contaminant Level Goal (level of a contaminant at which no known adverse effect would occur) for copper are both 1.3 mg/L; the MCL for lead is 0.015 mg/L, but the MCLG at which no known harm would occur is 0.000 mg/L. *See* 40 C.F.R. §§ 141.2 (for definitions), 141.51(b) (for MCLGs), Appx. A to Subpart Q of Part 141(I)(C) (for lead and copper); *see also* Exhibit 3.
[13] MDH, Comparison of State Water Guidance and Federal Drinking Water Standards, Dec. 2021, Exhibit 11; *see also* Exhibit 3, EPA MCLs, for summaries of adverse health effects.

(perfluorooctanoic acid) and a chronic HRL for PFOS (perfluorooctane sulfonate), where EPA has yet to develop an MCL despite multiple adverse health effects found in studies, including developmental effects to fetuses, immune effects, neurotoxicity, and liver tissue damage.[14]

Rather than determining retrospectively after harm has occurred which contaminants are emerging pollutants of concern, MPCA should adopt HRLs by incorporating them by reference as Class 1 WQS. Only HRLs that apply solely to wastewater treatment chemicals and have no potential to be discharged by any source to Minnesota Class 1 groundwater or surface water should be considered for exclusion from Class 1 WQS.

5. MPCA should prioritize adoption of a nitrate WQS for Class 2 surface waters and should enforce existing rules protecting downstream uses.

WaterLegacy and other conservation groups share the MPCA's concern that nitrate pollution of surface waters not currently identified as Class 1 waters is affecting both downstream surface waters and groundwaters used for drinking water. But there are direct and effective ways to address nitrate pollution of surface waters that neither require classifications that may devalue certain watersheds nor extensive monitoring that could result in years, if not decades, of delay.

First, the MPCA already has statutory and rule authority under Sections 402 and 303(d) of the Clean Water Act and state implementing rules to protect downstream Class 1 surface waters from upstream point source and non-point source discharge of nitrates. This authority should be rigorously enforced to limit nitrate pollution and protect downstream drinking water.

Second, the MPCA has already conducted scientific analysis needed to adopt a Class 2 standard for nitrates of 3.1 mg/L for Class 2A waters and 4.9 mg/L for other Class 2 waters to protect aquatic life, amphibians, endangered species, and species of state concern.[15] Adoption of a Class 2 nitrate standard would directly and effectively protect surface water quality for multiple uses. Hundreds of Minnesota residents petitioned the MPCA in April 2021 to prioritize Class 2 rulemaking to adopt nitrate standards that protect sensitive aquatic life.[16]

WaterLegacy and other signatories to this letter request that the MPCA enforce existing law and adopt a Class 2 WQS for nitrate, as the most effective and reliable way to address concerns about surface water nitrates affecting ecosystems and human health.

6. MPCA should not remove drinking water protection from Class 2A surface waters.

WaterLegacy and other conservation organizations signing this letter have consistently opposed removing drinking water standards from Class 2A surface waters. Adding threats to cold-water aquatic communities already under stress from pollution and climate change provides no benefit

[14] MDH, Toxicological Summary for: Perfluorooctane sulfonate, Aug. 2020, Exhibit 12, p. PFOS-9; *see also* Exhibit 3.
[15] MPCA, Aquatic Life WQS Technical Support Document for Nitrate, 2010, Exhibit 13, p. 8.
[16] Petition for Rulemaking to Protect Aquatic Life, Wild Rice, Wildlife and Human Health (2021), Exhibit 14.

to public health or public welfare. It would merely benefit industrial polluters at a cost of irrevocable ecosystem destruction and drinking water impairment.

7. MPCA should clean up rather than codify areas where groundwater is contaminated and strengthen enforcement to remove and prevent groundwater pollution.

The MPCA's Class 1 Concepts (p. 9) reflect the agency's decision not to pursue the addition of Groundwater Contaminant Management Zones ("GWCMZs") in Class 1 rulemaking. WaterLegacy supports this decision. Although data in the MPCA's Groundwater Contamination Atlas provides a public transparency benefit, taking any action to designate GWCMZs would have the effect of codifying areas of contamination rather than prioritizing their remediation.

There is a significant difference between providing information on locations where groundwater contaminant plumes require remediation and codifying their contaminated status. The objective of the federal and state Superfund programs is to remediate contamination so that contaminated groundwater will meet groundwater standards, not to preserve in perpetuity the contaminated status of sites. Designation of GWCMZs would undermine this objective.

WaterLegacy and other signatories to this letter support continued and accelerated implementation of both voluntary and legally compelled remediation to remove and mitigate groundwater contamination exceeding Class 1 WQS or Minnesota HRLs. In addition, the MPCA should review the potential for setting permitting requirements and taking enforcement actions under Minn. R. 7060.0600, subd. 2. These existing rules already prohibit discharge or deposit of pollutants to the unsaturated zone (surface land) that "may actually or potentially preclude or limit the use of the underground waters as a potable water supply" or "may pollute the underground waters."

This rule was enforced in *Kasal v. Minnesota Pollution Control Agency*, C1-93-2016, 1994 Minn. App. LEXIS 421, 1994 WL 175011 (Minn. Ct. App. May 10, 1994) (penalty approved for storing petroleum-contaminated soil). Minnesota's $850 million settlement with 3M for disposal of perfluorochemicals ("PFCs") that contaminated drinking water[17] was based on an amended complaint enforcing Minn. R. 7060.0600, subd. 2 among other legal authorities.[18] Rather than adopting a classification system that may serve to excuse or perpetuate the status of groundwater contamination, WaterLegacy and other conservation groups request that the MPCA review existing statutes and rules that can provide effective tools not just to remediate, but to prevent groundwater contamination.

Conclusion

WaterLegacy and other conservation organizations oppose the following: 1) changes to the structure of chapter 7050 or chapter 7060 definitions of drinking water, 2) development of tiers of Class 1 uses (such as class 1A, 1B, and 1C), 3) removal of secondary MCLs from Class 1 WQS, 4) removal of drinking water protections from Class 2A waters, and 5) designation of GWCMZs.

[17] Agreement and Order for Settlement, State of Minnesota v. 3M Company (2018), Exhibit 15.
[18] Amended Complaint, State of Minnesota v. 3M Company (2011), Exhibit 16.

The only changes in Minnesota Rules that WaterLegacy and other undersigned groups support at this time are: 1) changes to Minn. R. 7050.0221 to incorporate HRLs by reference as Class 1 WQS, and 2) removal from Minn. R. 7050.0221 of exceptions for copper and lead for primary MCLs otherwise incorporated as Class 1 WQS. Both of these changes are consistent with existing state policy and would increase protection of human health and public welfare.

In addition, WaterLegacy and other signatories requests MPCA action: 1) to implement its regulatory authority affirmed in the Minnesota Supreme Court's *U.S. Steel* case, 2) to enforce existing statutes and rules to protect downstream drinking water from upstream surface water pollution and to protect groundwater from pollution of the unsaturated zone, and 3) to prioritize adoption of a Class 2 nitrate WQS. Thank you for your consideration.

Sincerely yours,

Paula G. Maccabee
WaterLegacy Advocacy Director and Counsel

Friends of the Boundary Waters Wilderness
Honor the Earth
Minnesota Division of the Izaak Walton League of America
North American Water Office
Northeastern Minnesotans for Wilderness
Northern Lakes Scientific Advisory Panel
W.J. McCabe (Duluth) Chapter of the Izaak Walton League of America.

Exhibits 1-16 Attached

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By: OAH on 2/14/2022 @ 3:16 pm

Paula Maccabee Attachment 2

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT LIST & EXHIBITS

Exhibit 1	EPA, National Secondary Drinking Water Standards, p. F-76, available at https://www.gvsu.edu/cms4/asset/E1327343-09F0-03FF-AA9032F47AD1EB9C/standards.pdf
Exhibit 2	Minnesota Rules1983, Chapter 7050.0220 Rules, codification excerpt, available at https://www.revisor.mn.gov/rules/7050/date/1983
Exhibit 3	EPA, National Primary and National Secondary Drinking Water Regulations, available at https://www.epa.gov/sites/default/files/2016-06/documents/npwdr_complete_table.pdf
Exhibit 4	MDH, Human Health-Based Water Guidance Table, available at https://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html
Exhibit 5	WHO, Manganese in Drinking-water, WHO/HEP/ECH/WSH/2021.5, 2021, available at https://www.who.int/publications/i/item/WHO-HEP-ECH-WSH-2021.5
Exhibit 6	WHO, Silver in Drinking-water, WHO/SDE/WSH/03.04/14, 2014, available at https://www.who.int/water_sanitation_health/dwq/chemicals/silver.pdf
Exhibit 7	Rondeau *et al*., Aluminum and silica in drinking water and the risk of Alzheimer's disease or cognitive decline: findings from 15-year follow-up of the PAQUID cohort, *Am. J. Epidemiol*. 2009 Feb:169(4): 489-496, available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2809081/?report=printable
Exhibit 8	MDH, Sulfate in Well Water Fact Sheet, Aug. 2, 2019, available at https://www.health.state.mn.us/communities/environment/water/docs/wells/waterquality/sulfate.pdf
Exhibit 9	Bashir *et al*, Health Effects from Exposure to Sulphates and Chlorides in Drinking Water, *Pakistan J. of Med. & Health Sciences*, Vol. 6, No. 3, Jul-Sep 2012, available at https://www.researchgate.net/publication/242344864_Health_Effects_from_Exposure_to_Sulphates_and_Chlorides_in_Drinking_Water

Exhibit 10 U.S.D.A. National Institute of Food and Agriculture Extension, Drinking Water Contaminant – Sulfur, hydrogen sulfide, Aug. 23, 2019, available at https://drinking-water.extension.org/drinking-water-contaminant-sulfur-hydrogen-sulfide/

Exhibit 11 MDH, Comparison of State Water Guidance and Federal Drinking Water Standards, Dec. 2021, available at https://www.health.state.mn.us/communities/environment/risk/guidance/waterguidance.html

Exhibit 12 MDH, Toxicological Summary for: Perfluorooctane sulfonate, Aug. 2020, available at https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf

Exhibit 13 MPCA, Aquatic Life WQS Technical Support Document for Nitrate, 2010, available at https://www.pca.state.mn.us/sites/default/files/wq-s6-13.pdf

Exhibit 14 Petition for Rulemaking to Protect Aquatic Life, Wild Rice, Wildlife and Human Health (2021).

Exhibit 15 Agreement and Order for Settlement, State of Minnesota v. 3M Company (2018), available at https://www.ag.state.mn.us/Office/Cases/3M/docs/Agreement.pdf

Exhibit 16 Amended Complaint, State of Minnesota v. 3M Company (2011), available at https://www.mncourts.gov/mncourtsgov/media/High-Profile-Cases/27-CV-10-28862/Complaint-011811.pdf

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(Minnesota Rules chapters 7050, 7052, 7053, 7060)
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EXHIBIT 1

U.S. EPA National Secondary Drinking Water Standards

Secondary Drinking Water Standards are not MCLs, but unenforceable federal guidelines regarding taste, odor, color and certain other non-aesthetic effects of drinking water. EPA recommends them to the States as reasonable goals, but federal law does not require water systems to comply with them. States may, however, adopt their own enforceable regulations governing these contaminants. To be safe, check your State's drinking water rules.

Contaminants	Suggested Level
Aluminum	0.05 - 0.2 mg/l
Chloride	250 mg/l
Color	15 color units
Copper	1 mg/l
Corrosivity	Non-corrosive
Fluoride	2.0 mg/l
Foaming agents	0.5 mg/l
Iron	0.3 mg/l
Manganese	0.05 mg/l
Odor	3 threshold odor number
pH	6.5 - 8.5
Silver	0.1 mg/l
Sulfate	250 mg/l
Total dissolved solids (TDS)	500 mg/l
Zinc	5 mg/l

https://www.gvsu.edu/cms4/asset/E1327343-09F0-03FF-AA9032F47AD1EB9C/standards.pdf

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EXHIBIT 2

Subp. 16. **Limited resource value waters.** Limited resource value waters:

A. For point source discharges to surface waters classified as limited resource value waters pursuant to parts 7050.0200, number 7 and 7050.0300 to 7050.0380, the agency shall require treatment facilities which will provide effluents conforming to the following limitations:*

Substance or Characteristic	*Limiting Concentration*
5-Day Biochemical oxygen demand	15 milligrams per liter**

*All effluent limitations specified in subpart 6 shall also be applicable to dischargers to Class 7 waters, provided that unspecified toxic or corrosive substances shall be limited to the extent necessary to protect the designated uses of the receiving water or affected downstream waters.

**As measured by the arithmetic mean of all samples taken during any calendar month.

B. The agency shall allow treatment works to be constructed and/or operated to produce effluents to limited resource value waters at levels up to those stated in subpart 6 provided that it is demonstrated that the water quality standards for limited resource value waters will be maintained during all periods of discharge from the treatment facilities.

C. Notwithstanding the effluent limitations established by this section the quality of limited resource value waters shall not be such as to allow a violation of applicable water quality standards in waters of the state which are connected to or affected by water classified as limited resource value waters.

D. The classification of surface waters as limited resource value waters pursuant to parts 7050.0200, number 7 and 7050.0300 to 7050.0380 shall not supersede, alter, or replace the classification and designation of such waters as public waters pursuant to applicable provisions and requirements of Minnesota Statutes, chapter 105.

Subp. 17. **Compliance with terms and water quality standards.** No person who is in compliance with the terms and conditions of its permit issued pursuant to chapter 7070 shall be deemed in violation of any water quality standard in this rule for which a corresponding effluent limitation is established in the permit. However, exceedances of the water quality standards in a receiving water shall constitute grounds for modification of a permit(s) for any discharger(s) to the receiving water who is (are) causing or contributing to the exceedances. Chapter 7070 shall govern the modification of any such permit.

Subp. 18. **Ammonia water quality standard.** For the purpose of establishing limitations to meet the ammonia water quality standard, a statistic which estimates the central value (such as the mean or median) for ambient pH and temperature of the receiving water for the critical months shall be used.

Statutory Authority: *MS s 115.03 subd 1*

7050.0220 SPECIFIC STANDARDS OF QUALITY AND PURITY FOR DESIGNATED CLASSES OF INTRASTATE WATERS OF THE STATE.

The following standards shall prescribe the qualities or properties of the intrastate waters of the state which are necessary for the designated public use or benefit and which, if the limiting conditions given are exceeded, shall be considered indicative of a polluted condition which is actually or potentially deleterious, harmful, detrimental, or injurious with respect to such designated uses or established classes of the intrastate waters.

1. Domestic consumption.

Class A. The quality of this class of the intrastate waters of the state shall be such that without treatment of any kind the raw waters will meet in all respects both the mandatory and recommended requirements of the Public Health Service Drinking Water Standards-1962 for drinking water as specified in

Publication No. 956 published by the Public Health Service of the United States Department of Health, Education and Welfare, and any revisions, amendments, or supplements thereto. This standard will ordinarily be restricted to underground waters with a high degree of natural protection. The basic requirements are given below:

Substance or Characteristic	Limit or Range
Total coliform organisms	1 most probable number per 100 milliliters
Turbidity value	5
Color value	15
Threshold odor number	3
Methylene blue active substance (MBAS)	0.5 milligram per liter
Arsenic (As)	0.01 milligram per liter
Chlorides (Cl)	250 milligrams per liter
Copper (Cu)	1 milligram per liter
Carbon chloroform extract	0.2 milligram per liter
Cyanides (CN)	0.01 milligram per liter
Fluorides (F)	1.5 milligrams per liter
Iron (Fe)	0.3 milligram per liter
Manganese (Mn)	0.05 milligram per liter
Nitrates (NO_3)	45 milligrams per liter
Phenol	0.001 milligram per liter
Sulfates (SO_4)	250 milligrams per liter
Total dissolved solids	500 milligrams per liter
Zinc (Zn)	5 milligrams per liter
Barium (Ba)	1 milligram per liter
Cadmium (Cd)	0.01 milligram per liter
Chromium (Hexavalent, Cr)	0.05 milligram per liter
Lead (Pb)	0.05 milligram per liter
Selenium (Se)	0.01 milligram per liter
Silver (Ag)	0.05 milligram per liter
Radioactive material	Not to exceed the lowest concentrations permitted to be discharged to an uncontrolled environment as prescribed by the appropriate authority having control over their use.

Class B. The quality of this class of the intrastate waters of the state shall be such that with approved disinfection, such as simple chlorination or its equivalent, the treated water will meet in all respects both the mandatory and recommended requirements of the Public Health Service Drinking Water Standards -- 1962 for drinking water as specified in Publication No. 956 published by the Public Health Service of the United States Department of Health, Education and Welfare, and any revisions, amendments, or supplements thereto. This standard will ordinarily be restricted to surface and underground waters with a moderately high degree of natural protection. The physical and chemical standards quoted above for Class A intrastate waters shall also supply to these intrastate waters in the untreated state.

Class C. The quality of this class of the intrastate waters of the state shall be such that with treatment consisting of coagulation, sedimentation, filtration, storage, and chlorination, or other equivalent treatment processes, the treated water will meet in all respects both the mandatory and recommended requirements of the Public Health Service Drinking Water Standards -- 1962 for drinking water as specified in Publication No. 956 published by the Public

Health Service of the United States Department of Health, Education and Welfare, and any revisions, amendments, or supplements thereto. This standard will ordinarily be restricted to surface waters, and ground waters in aquifers not considered to afford adequate protection against contamination from surface or other sources of pollution. Such aquifers normally would include fractured and channeled limestone, unprotected impervious hard rock where intrastate water is obtained from mechanical fractures, joints, etc., with surface connections, and coarse gravels subjected to surface water infiltration. The physical and chemical standards quoted above for Class A intrastate waters shall also apply to these intrastate waters in the untreated state, except as listed below:

Substance of Characteristic	Limit or Range
Turbidity value	25

Class D. The quality of this class of the intrastate waters of the state shall be such that after treatment consisting of coagulation, sedimentation, filtration, storage, and chlorination, plus additional pre, post, or intermediate stages of treatment, or other equivalent treatment processes, the treated water will meet in all respects the recommended requirements of the Public Health Service Drinking Water Standards -- 1962 for drinking water as specified in Publication No. 956 published by the Public Health Service of the United States Department of Health, Education and Welfare, and any revisions, amendments, or supplements thereto. This standard will ordinarily be restricted to surface waters, and ground waters in aquifers not considered to afford adequate protection against contamination from surface or other sources of pollution. Such aquifers normally would include fractured and channeled limestone, unprotected impervious hard rock where water is obtained from mechanical fractures, joints, etc., with surface connections, and coarse gravels subjected to surface water infiltration. The concentrations or ranges given below shall not be exceeded in the raw waters before treatment:

Substance or Characteristic	Limit or Range
Arsenic (As)	0.05 milligram per liter
Barium (Ba)	1 milligram per liter
Cadmium (Cd)	0.01 milligram per liter
Chromium (Cr + 6)	0.05 milligram per liter
Cyanide (CN)	0.2 milligram per liter
Fluoride (F)	1.5 milligrams per liter
Lead (Pb)	0.05 milligram per liter
Selenium (Se)	0.01 milligram per liter
Silver (Ag)	0.05 milligram per liter
Radioactive material	Not to exceed the lowest concentrations permitted to be discharged to an uncontrolled environment as prescribed by the appropriate authority having control over their use.

In addition to the above listed standards, no sewage, industrial waste, or other wastes, treated or untreated, shall be discharged into or permitted by any person to gain access to any intrastate waters classified for domestic consumption so as to cause any material undesirable increase in the taste, hardness, temperature, toxicity, corrosiveness, or nutrient content, or in any other manner to impair the natural quality or value of the intrastate waters for use as a source of drinking water.

2. Fisheries and recreation.

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EXHIBIT 3

epa.gov/sites/default/files/2016-06/documents/npwdr_complete_table.pdf

National Primary Drinking Water Regulations

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
Acrylamide	TT[4]	Nervous system or blood problems; increased risk of cancer	Added to water during sewage/ wastewater treatment	zero
Alachlor	0.002	Eye, liver, kidney, or spleen problems; anemia; increased risk of cancer	Runoff from herbicide used on row crops	zero
Alpha/photon emitters	15 picocuries per Liter (pCi/L)	Increased risk of cancer	Erosion of natural deposits of certain minerals that are radioactive and may emit a form of radiation known as alpha radiation	zero
Antimony	0.006	Increase in blood cholesterol; decrease in blood sugar	Discharge from petroleum refineries; fire retardants; ceramics; electronics; solder	0.006
Arsenic	0.010	Skin damage or problems with circulatory systems, and may have increased risk of getting cancer	Erosion of natural deposits; runoff from orchards; runoff from glass & electronics production wastes	0
Asbestos (fibers >10 micrometers)	7 million fibers per Liter (MFL)	Increased risk of developing benign intestinal polyps	Decay of asbestos cement in water mains; erosion of natural deposits	7 MFL
Atrazine	0.003	Cardiovascular system or reproductive problems	Runoff from herbicide used on row crops	0.003
Barium	2	Increase in blood pressure	Discharge of drilling wastes; discharge from metal refineries; erosion of natural deposits	2
Benzene	0.005	Anemia; decrease in blood platelets; increased risk of cancer	Discharge from factories; leaching from gas storage tanks and landfills	zero
Benzo(a)pyrene (PAHs)	0.0002	Reproductive difficulties; increased risk of cancer	Leaching from linings of water storage tanks and distribution lines	zero
Beryllium	0.004	Intestinal lesions	Discharge from metal refineries and coal-burning factories; discharge from electrical, aerospace, and defense industries	0.004
Beta photon emitters	4 millirems per year	Increased risk of cancer	Decay of natural and man-made deposits of certain minerals that are radioactive and may emit forms of radiation known as photons and beta radiation	zero
Bromate	0.010	Increased risk of cancer	Byproduct of drinking water disinfection	zero
Cadmium	0.005	Kidney damage	Corrosion of galvanized pipes; erosion of natural deposits; discharge from metal refineries; runoff from waste batteries and paints	0.005
Carbofuran	0.04	Problems with blood, nervous system, or reproductive system	Leaching of soil fumigant used on rice and alfalfa	0.04

LEGEND

DISINFECTANT | DISINFECTION BYPRODUCT | INORGANIC CHEMICAL | MICROORGANISM | ORGANIC CHEMICAL | RADIONUCLIDES

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
Carbon tetrachloride	0.005	Liver problems; increased risk of cancer	Discharge from chemical plants and other industrial activities	**zero**
Chloramines (as Cl_2)	MRDL=4.0[1]	Eye/nose irritation; stomach discomfort; anemia	Water additive used to control microbes	**MRDLG=4[1]**
Chlordane	0.002	Liver or nervous system problems; increased risk of cancer	Residue of banned termiticide	**zero**
Chlorine (as Cl_2)	MRDL=4.0[1]	Eye/nose irritation; stomach discomfort	Water additive used to control microbes	**MRDLG=4[1]**
Chlorine dioxide (as ClO_2)	MRDL=0.8[1]	Anemia; infants, young children, and fetuses of pregnant women: nervous system effects	Water additive used to control microbes	**MRDLG=0.8[1]**
Chlorite	1.0	Anemia; infants, young children, and fetuses of pregnant women: nervous system effects	Byproduct of drinking water disinfection	**0.8**
Chlorobenzene	0.1	Liver or kidney problems	Discharge from chemical and agricultural chemical factories	**0.1**
Chromium (total)	0.1	Allergic dermatitis	Discharge from steel and pulp mills; erosion of natural deposits	**0.1**
Copper	TT[5]; Action Level=1.3	Short-term exposure: Gastrointestinal distress. Long-term exposure: Liver or kidney damage. People with Wilson's Disease should consult their personal doctor if the amount of copper in their water exceeds the action level	Corrosion of household plumbing systems; erosion of natural deposits	**1.3**
Cryptosporidium	TT[7]	Short-term exposure: Gastrointestinal illness (e.g., diarrhea, vomiting, cramps)	Human and animal fecal waste	**zero**
Cyanide (as free cyanide)	0.2	Nerve damage or thyroid problems	Discharge from steel/metal factories; discharge from plastic and fertilizer factories	**0.2**
2,4-D	0.07	Kidney, liver, or adrenal gland problems	Runoff from herbicide used on row crops	**0.07**
Dalapon	0.2	Minor kidney changes	Runoff from herbicide used on rights of way	**0.2**
1,2-Dibromo-3-chloropropane (DBCP)	0.0002	Reproductive difficulties; increased risk of cancer	Runoff/leaching from soil fumigant used on soybeans, cotton, pineapples, and orchards	**zero**
o-Dichlorobenzene	0.6	Liver, kidney, or circulatory system problems	Discharge from industrial chemical factories	**0.6**
p-Dichlorobenzene	0.075	Anemia; liver, kidney, or spleen damage; changes in blood	Discharge from industrial chemical factories	**0.075**
1,2-Dichloroethane	0.005	Increased risk of cancer	Discharge from industrial chemical factories	**zero**

LEGEND


DISINFECTANT


DISINFECTION BYPRODUCT


INORGANIC CHEMICAL


MICROORGANISM


ORGANIC CHEMICAL


RADIONUCLIDES

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
1,1-Dichloroethylene	0.007	Liver problems	Discharge from industrial chemical factories	**0.007**
cis-1,2-Dichloroethylene	0.07	Liver problems	Discharge from industrial chemical factories	**0.07**
trans-1,2, Dichloroethylene	0.1	Liver problems	Discharge from industrial chemical factories	**0.1**
Dichloromethane	0.005	Liver problems; increased risk of cancer	Discharge from industrial chemical factories	**zero**
1,2-Dichloropropane	0.005	Increased risk of cancer	Discharge from industrial chemical factories	**zero**
Di(2-ethylhexyl) adipate	0.4	Weight loss, liver problems, or possible reproductive difficulties	Discharge from chemical factories	**0.4**
Di(2-ethylhexyl) phthalate	0.006	Reproductive difficulties; liver problems; increased risk of cancer	Discharge from rubber and chemical factories	**zero**
Dinoseb	0.007	Reproductive difficulties	Runoff from herbicide used on soybeans and vegetables	**0.007**
Dioxin (2,3,7,8-TCDD)	0.00000003	Reproductive difficulties; increased risk of cancer	Emissions from waste incineration and other combustion; discharge from chemical factories	**zero**
Diquat	0.02	Cataracts	Runoff from herbicide use	**0.02**
Endothall	0.1	Stomach and intestinal problems	Runoff from herbicide use	**0.1**
Endrin	0.002	Liver problems	Residue of banned insecticide	**0.002**
Epichlorohydrin	TT[4]	Increased cancer risk; stomach problems	Discharge from industrial chemical factories; an impurity of some water treatment chemicals	**zero**
Ethylbenzene	0.7	Liver or kidney problems	Discharge from petroleum refineries	**0.7**
Ethylene dibromide	0.00005	Problems with liver, stomach, reproductive system, or kidneys; increased risk of cancer	Discharge from petroleum refineries	**zero**
Fecal coliform and *E. coli*	MCL[6]	Fecal coliforms and *E. coli* are bacteria whose presence indicates that the water may be contaminated with human or animal wastes. Microbes in these wastes may cause short term effects, such as diarrhea, cramps, nausea, headaches, or other symptoms. They may pose a special health risk for infants, young children, and people with severely compromised immune systems.	Human and animal fecal waste	**zero[6]**

LEGEND

DISINFECTANT | DISINFECTION BYPRODUCT | INORGANIC CHEMICAL | MICROORGANISM | ORGANIC CHEMICAL | RADIONUCLIDES

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
Fluoride	4.0	Bone disease (pain and tenderness of the bones); children may get mottled teeth	Water additive which promotes strong teeth; erosion of natural deposits; discharge from fertilizer and aluminum factories	**4.0**
Giardia lamblia	TT[7]	Short-term exposure: Gastrointestinal illness (e.g., diarrhea, vomiting, cramps)	Human and animal fecal waste	**zero**
Glyphosate	0.7	Kidney problems; reproductive difficulties	Runoff from herbicide use	**0.7**
Haloacetic acids (HAA5)	0.060	Increased risk of cancer	Byproduct of drinking water disinfection	**n/a[9]**
Heptachlor	0.0004	Liver damage; increased risk of cancer	Residue of banned termiticide	**zero**
Heptachlor epoxide	0.0002	Liver damage; increased risk of cancer	Breakdown of heptachlor	**zero**
Heterotrophic plate count (HPC)	TT[7]	HPC has no health effects; it is an analytic method used to measure the variety of bacteria that are common in water. The lower the concentration of bacteria in drinking water, the better maintained the water system is.	HPC measures a range of bacteria that are naturally present in the environment	**n/a**
Hexachlorobenzene	0.001	Liver or kidney problems; reproductive difficulties; increased risk of cancer	Discharge from metal refineries and agricultural chemical factories	**zero**
Hexachloro-cyclopentadiene	0.05	Kidney or stomach problems	Discharge from chemical factories	**0.05**
Lead	TT[5]; Action Level=0.015	Infants and children: Delays in physical or mental development; children could show slight deficits in attention span and learning abilities; Adults: Kidney problems; high blood pressure	Corrosion of household plumbing systems; erosion of natural deposits	**zero**
Legionella	TT[7]	Legionnaire's Disease, a type of pneumonia	Found naturally in water; multiplies in heating systems	**zero**
Lindane	0.0002	Liver or kidney problems	Runoff/leaching from insecticide used on cattle, lumber, and gardens	**0.0002**
Mercury (inorganic)	0.002	Kidney damage	Erosion of natural deposits; discharge from refineries and factories; runoff from landfills and croplands	**0.002**
Methoxychlor	0.04	Reproductive difficulties	Runoff/leaching from insecticide used on fruits, vegetables, alfalfa, and livestock	**0.04**
Nitrate (measured as Nitrogen)	10	Infants below the age of six months who drink water containing nitrate in excess of the MCL could become seriously ill and, if untreated, may die. Symptoms include shortness of breath and blue-baby syndrome.	Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits	**10**

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RADIONUCLIDES

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
Nitrite (measured as Nitrogen)	1	Infants below the age of six months who drink water containing nitrite in excess of the MCL could become seriously ill and, if untreated, may die. Symptoms include shortness of breath and blue-baby syndrome.	Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits	1
Oxamyl (Vydate)	0.2	Slight nervous system effects	Runoff/leaching from insecticide used on apples, potatoes, and tomatoes	0.2
Pentachlorophenol	0.001	Liver or kidney problems; increased cancer risk	Discharge from wood-preserving factories	zero
Picloram	0.5	Liver problems	Herbicide runoff	0.5
Polychlorinated biphenyls (PCBs)	0.0005	Skin changes; thymus gland problems; immune deficiencies; reproductive or nervous system difficulties; increased risk of cancer	Runoff from landfills; discharge of waste chemicals	zero
Radium 226 and Radium 228 (combined)	5 pCi/L	Increased risk of cancer	Erosion of natural deposits	zero
Selenium	0.05	Hair or fingernail loss; numbness in fingers or toes; circulatory problems	Discharge from petroleum and metal refineries; erosion of natural deposits; discharge from mines	0.05
Simazine	0.004	Problems with blood	Herbicide runoff	0.004
Styrene	0.1	Liver, kidney, or circulatory system problems	Discharge from rubber and plastic factories; leaching from landfills	0.1
Tetrachloroethylene	0.005	Liver problems; increased risk of cancer	Discharge from factories and dry cleaners	zero
Thallium	0.002	Hair loss; changes in blood; kidney, intestine, or liver problems	Leaching from ore-processing sites; discharge from electronics, glass, and drug factories	0.0005
Toluene	1	Nervous system, kidney, or liver problems	Discharge from petroleum factories	1
Total Coliforms	5.0 percent[8]	Coliforms are bacteria that indicate that other, potentially harmful bacteria may be present. See fecal coliforms and *E. coli*	Naturally present in the environment	zero
Total Trihalomethanes (TTHMs)	0.080	Liver, kidney, or central nervous system problems; increased risk of cancer	Byproduct of drinking water disinfection	n/a[9]
Toxaphene	0.003	Kidney, liver, or thyroid problems; increased risk of cancer	Runoff/leaching from insecticide used on cotton and cattle	zero
2,4,5-TP (Silvex)	0.05	Liver problems	Residue of banned herbicide	0.05
1,2,4-Trichlorobenzene	0.07	Changes in adrenal glands	Discharge from textile finishing factories	0.07

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RADIONUCLIDES

Contaminant	MCL or TT[1] (mg/L)[2]	Potential health effects from long-term[3] exposure above the MCL	Common sources of contaminant in drinking water	Public Health Goal (mg/L)[2]
1,1,1-Trichloroethane	0.2	Liver, nervous system, or circulatory problems	Discharge from metal degreasing sites and other factories	**0.2**
1,1,2-Trichloroethane	0.005	Liver, kidney, or immune system problems	Discharge from industrial chemical factories	**0.003**
Trichloroethylene	0.005	Liver problems; increased risk of cancer	Discharge from metal degreasing sites and other factories	**zero**
Turbidity	TT[7]	Turbidity is a measure of the cloudiness of water. It is used to indicate water quality and filtration effectiveness (e.g., whether disease-causing organisms are present). Higher turbidity levels are often associated with higher levels of disease-causing microorganisms such as viruses, parasites, and some bacteria. These organisms can cause short term symptoms such as nausea, cramps, diarrhea, and associated headaches.	Soil runoff	**n/a**
Uranium	30µg/L	Increased risk of cancer, kidney toxicity	Erosion of natural deposits	**zero**
Vinyl chloride	0.002	Increased risk of cancer	Leaching from PVC pipes; discharge from plastic factories	**zero**
Viruses (enteric)	TT[7]	Short-term exposure: Gastrointestinal illness (e.g., diarrhea, vomiting, cramps)	Human and animal fecal waste	**zero**
Xylenes (total)	10	Nervous system damage	Discharge from petroleum factories; discharge from chemical factories	**10**

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RADIONUCLIDES

NOTES

1 Definitions
- **Maximum Contaminant Level Goal (MCLG):** The level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs allow for a margin of safety and are non-enforceable public health goals.
- **Maximum Contaminant Level (MCL):** The highest level of a contaminant that is allowed in drinking water. MCLs are set as close to MCLGs as feasible using the best available treatment technology and taking cost into consideration. MCLs are enforceable standards.
- **Maximum Residual Disinfectant Level Goal (MRDLG):** The level of a drinking water disinfectant below which there is no known or expected risk to health. MRDLGs do not reflect the benefits of the use of disinfectants to control microbial contaminants.
- **Maximum Residual Disinfectant Level (MRDL):** The highest level of a disinfectant allowed in drinking water. There is convincing evidence that addition of a disinfectant is necessary for control of microbial contaminants.
- **Treatment Technique (TT):** A required process intended to reduce the level of a contaminant in drinking water.

2 Units are in milligrams per liter (mg/L) unless otherwise noted. Milligrams per liter are equivalent to parts per million (ppm).

3 Health effects are from long-term exposure unless specified as short-term exposure.

4 Each water system must certify annually, in writing, to the state (using third-party or manufacturers certification) that when it uses acrylamide and/or epichlorohydrin to treat water, the combination (or product) of dose and monomer level does not exceed the levels specified, as follows: Acrylamide = 0.05 percent dosed at 1 mg/L (or equivalent); Epichlorohydrin = 0.01 percent dosed at 20 mg/L (or equivalent).

5 Lead and copper are regulated by a Treatment Technique that requires systems to control the corrosiveness of their water. If more than 10 percent of tap water samples exceed the action level, water systems must take additional steps. For copper, the action level is 1.3 mg/L, and for lead is 0.015 mg/L.

6 A routine sample that is fecal coliform-positive or E. coli-positive triggers repeat samples--if any repeat sample is total coliform-positive, the system has an acute MCL violation. A routine sample that is total coliform-positive and fecal coliform-negative or E. coli-negative triggers repeat samples--if any repeat sample is fecal coliform-positive or E. coli-positive, the system has an acute MCL violation. See also Total Coliforms.

7 EPA's surface water treatment rules require systems using surface water or ground water under the direct influence of surface water to (1) disinfect their water, and (2) filter their water or meet criteria for avoiding filtration so that the following contaminants are controlled at the following levels:
- ***Cryptosporidium*:** 99 percent removal for systems that filter. Unfiltered systems are required to include Cryptosporidium in their existing watershed control provisions.
- **Giardia lamblia:** 99.9 percent removal/inactivation
- **Viruses:** 99.9 percent removal/inactivation
- ***Legionella*:** No limit, but EPA believes that if *Giardia* and viruses are removed/inactivated, according to the treatment techniques in the surface water treatment rule, *Legionella* will also be controlled.
- **Turbidity:** For systems that use conventional or direct filtration, at no time can turbidity (cloudiness of water) go higher than 1 nephelometric turbidity unit (NTU), and samples for turbidity must be less than or equal to 0.3 NTU in at least 95 percent of the samples in any month. Systems that use filtration other than the conventional or direct filtration must follow state limits, which must include turbidity at no time exceeding 5 NTU.
- **HPC:** No more than 500 bacterial colonies per milliliter
- **Long Term 1 Enhanced Surface Water Treatment:** Surface water systems or ground water systems under the direct influence of surface water serving fewer than 10,000 people must comply with the applicable Long Term 1 Enhanced Surface Water Treatment Rule provisions (e.g. turbidity standards, individual filter monitoring, *Cryptosporidium* removal requirements, updated watershed control requirements for unfiltered systems).
- **Long Term 2 Enhanced Surface Water Treatment:** This rule applies to all surface water systems or ground water systems under the direct influence of surface water. The rule targets additional *Cryptosporidium* treatment requirements for higher risk systems and includes provisions to reduce risks from uncovered finished water storages facilities and to ensure that the systems maintain microbial protection as they take steps to reduce the formation of disinfection byproducts. (Monitoring start dates are staggered by system size. The largest systems (serving at least 100,000 people) will begin monitoring in October 2006 and the smallest systems (serving fewer than 10,000 people) will not begin monitoring until October 2008. After completing monitoring and determining their treatment bin, systems generally have three years to comply with any additional treatment requirements.)
- **Filter Backwash Recycling:** The Filter Backwash Recycling Rule requires systems that recycle to return specific recycle flows through all processes of the system's existing conventional or direct filtration system or at an alternate location approved by the state.

8 No more than 5.0 percent samples total coliform-positive in a month. (For water systems that collect fewer than 40 routine samples per month, no more than one sample can be total coliform-positive per month.) Every sample that has total coliform must be analyzed for either fecal coliforms or E. coli. If two consecutive TC-positive samples, and one is also positive for E. coli or fecal coliforms, system has an acute MCL violation.

9 Although there is no collective MCLG for this contaminant group, there are individual MCLGs for some of the individual contaminants:
- **Haloacetic acids:** dichloroacetic acid (zero); trichloroacetic acid (0.3 mg/L)
- **Trihalomethanes:** bromodichloromethane (zero); bromoform (zero); dibromochloromethane (0.06 mg/L)

EPA 816-F-09-004 | MAY 2009

NATIONAL SECONDARY DRINKING WATER REGULATION

National Secondary Drinking Water Regulations are non-enforceable guidelines regarding contaminants that may cause cosmetic effects (such as skin or tooth discoloration) or aesthetic effects (such as taste, odor, or color) in drinking water. EPA recommends secondary standards to water systems but does not require systems to comply. However, some states may choose to adopt them as enforceable standards.

Contaminant	Secondary Maximum Contaminant Level
Aluminum	0.05 to 0.2 mg/L
Chloride	250 mg/L
Color	15 (color units)
Copper	1.0 mg/L
Corrosivity	Noncorrosive
Fluoride	2.0 mg/L
Foaming Agents	0.5 mg/L
Iron	0.3 mg/L
Manganese	0.05 mg/L
Odor	3 threshold odor number
pH	6.5-8.5
Silver	0.10 mg/L
Sulfate	250 mg/L
Total Dissolved Solids	500 mg/L
Zinc	5 mg/L

FOR MORE INFORMATION ON EPA'S SAFE DRINKING WATER:

visit: **epa.gov/safewater**

call: **(800) 426-4791**

ADDITIONAL INFORMATION:

To order additional posters or other ground water and drinking water publications, please contact the National Service Center for Environmental Publications at: **(800) 490-9198**, or email: **nscep@bps-lmit.com**.

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 4

https://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html

Human Health-Based Water Guidance Table

The Minnesota Department of Health (MDH) develops health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater.

When multiple substances are present MDH risk assessment methods require evaluation of the potential risk from the combined exposure. Information from the water guidance table below and a calculator for exposures to multiple chemicals is available for download:
MDH Water Guidance and Additivity Calculator (Excel).

Guidance Table

CAS Number	Chemical	Value Type	Exposure Duration	Value (µg/L)	Health Endpoint(s)
Find chemicals beginning with: A - C D - E F - M N - S T - Z					
83-32-9	Acenaphthene Toxicological Summary for Acenaphthene (PDF) Information Sheet: Acenaphthene in Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	ND	--
			Subchronic	200	Adrenal; Liver system
			Chronic	100	Adrenal; Liver system
			Cancer	NA	--
103-90-2	Acetaminophen	HRL_{15}	Acute	200	Liver system
			Short-term	200	Liver system
			Subchronic	200*	Liver system
			Chronic	200*	Liver system
			Cancer	NA	--

	Toxicological Summary for Acetaminophen (PDF) Information Sheet: Acetaminophen in Drinking Water (PDF)				
34256-82-1	Acetochlor Toxicological Summary for Acetochlor (PDF) *See also* degradates: Acetochlor ESA Acetochlor OXA Information Sheet: Acetochlor and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	30	Developmental; Liver system; Thyroid (E)
			Subchronic	30	Liver system; Male reproductive system; Nervous system; Kidney system
			Chronic	20	Liver system; Male reproductive system; Nervous system; Kidney system; Respiratory system
			Cancer	NA	--
187022-11-3	Acetochlor ESA (degradate of Acetochlor) Toxicological Summary for Acetochlor ESA (PDF) Information Sheet: Acetochlor ESA and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	500	Thyroid (E)
			Subchronic	500	Male reproductive system; Thyroid (E)
			Chronic	300	Male reproductive system; Thyroid (E)
			Cancer	NA	--
184992-44-4	Acetochlor OXA	HRL_{18}	Acute	ND	--
			Short-term	100	Thyroid (E)
			Subchronic	100*	Thyroid (E)
			Chronic	90	Thyroid (E)

	(degradate of Acetochlor) Toxicological Summary for Acetochlor OXA (PDF) Information Sheet: Acetochlor OXA and Drinking Water		Cancer	NA	--
67-64-1	Acetone Toxicological Summary for Acetone (HRL 2011) (PDF)	HRL_{11***}	Acute	ND	--
			Short-term	9,000	Kidney system
			Subchronic	8,000	Kidney system; Blood system
			Chronic	4,000	Kidney system; Blood system
			Cancer	NA	--
	Acetone Toxicological Summary for Acetone (HBV 2020) (PDF)	HBV_{20}	Acute	ND	--
			Short-term	5,000	Kidney system
			Subchronic	5,000*	Kidney system
			Chronic	3,000	Blood system; Liver system; Kidney system
			Cancer	NA	--
21145-77-7 or 1506-02-1	6-Acetyl-1,1,2,4,4,7-hexamethyltetraline (AHTN or Tonalide) Toxicological Summary for 6-Acetyl-1,12,4,4,7-hexamethyltetraline (PDF) Information Sheet: AHTN in Drinking Water (PDF)	HRL_{13}	Acute	ND	--
			Short-term	100	Liver system
			Subchronic	30	Liver system
			Chronic	20	Liver system
			Cancer	NA	--
79-06-1	Acrylamide Toxicological Summary for Acrylamide (PDF)	HRL_{15}	Acute	ND	--
			Short-term	7	Developmental; Male reproductive system; Nervous system
			Subchronic	7*	Developmental; Male reproductive system; Nervous system

	Information Sheet: Acrylamide in Drinking Water (PDF)		Chronic	7*	Developmental; Male reproductive system; Nervous system
			Cancer	0.2	Cancer
15972-60-8	Alachlor Toxicological Summary for Alachlor (PDF) *See also* degradates Alachlor ESA Alachlor OXA Information Sheet: Alachlor and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	100	Developmental; Kidney system
			Subchronic	60	Blood system; Liver system; Kidney system
			Chronic	9	Blood system; Liver system; Kidney system
			Cancer	NA	--
142363-53-9	Alachlor ESA Toxicological Summary for Alachlor ESA (PDF) (degradate of Alachlor)	RAA_{16}	Acute	ND	--
			Short-term	ND	--
			Subchronic	100	Blood system
			Chronic	50	Blood system
			Cancer	NA	--
171262-17-2	Alachlor OXA Toxicological Summary for Alachlor OXA (PDF) (degradate of Alachlor)	RAA_{16}	Acute	ND	--
			Short-term	ND	--
			Subchronic	100	Blood system
			Chronic	50	Blood system
			Cancer	NA	--
116-06-3	Aldicarb	HRL_{93}	Chronic	1	Nervous system
107-05-1	Allyl chloride (3 chloropropene)	HRL_{94}	Chronic	30	Nervous system
1066-51-9	Aminomethylphosphonic acid (AMPA)	HBV_{17}	Acute	ND	--
			Short-term	ND	--
			Subchronic	3000	Liver system; Kidney system

	Toxicological Summary for Aminomethylphosphonic acid (PDF) Information Sheet: AMPA and Drinking Water (PDF)		Chronic	1000	Liver system; Kidney system
			Cancer	NA	--
64285-06-9	Anatoxin-a Toxicological Summary for Anatoxin-a (PDF) Information Sheet: Anatoxin-a in Drinking Water (PDF)	RAA_{16}	Acute	ND	--
			Short-term	0.1	Nervous system
			Subchronic	ND	--
			Chronic	ND	--
			Cancer	ND	--
120-12-7	Anthracene Toxicological Summary for Anthracene (PDF) Information Sheet: Anthracene and Groundwater (PDF)	HRL_{93}***	Chronic	2,000	None
		RAA_{19}	Acute	ND	--
			Short-term	ND	--
			Subchronic	1,000	NA
			Chronic	600	NA
			Cancer	NA	--
7440-36-0	Antimony	HRL_{93}	Chronic	6	--
1912-24-9	Atrazine *See also degradates Deethylatrazine, Deethyldeisopropylatrazine, and Deisopropylatrazine* as described in the Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF)	HRL_{MCL}	Chronic	3	see USEPA Organic Chemicals table
7440-39-3	Barium	HRL_{93}	Chronic	2,000	Cardiovascular system
25057-89-0		HRL_{15}	Acute	400	Developmental; Female reproductive system

	Bentazon Toxicological Summary for Bentazon (PDF) Information Sheet: Bentazon in Drinking Water (PDF)		Short-term	60	Developmental
			Subchronic	50	Blood system
			Chronic	30	Thyroid
			Cancer	NA	--
71-43-2	Benzene Toxicological Summary for Benzene (PDF)	HRL_{09}	Acute	10	Developmental
			Short-term	10	Blood system; Immune system
			Subchronic	3	Blood system; Immune system
			Chronic	3**	Blood system; Immune system
			Cancer	2	Cancer
50-32-8	Benzo[a]pyrene Toxicological Summary for Benzo(a)pyrene (PDF) Information Sheet: Benzo(a)pyrene and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	0.5	Developmental; Nervous system
			Subchronic	0.5*	Developmental; Nervous system
			Chronic	0.5*	Developmental; Nervous system
			Cancer	0.1	Cancer
Go to > top.					
65-85-0	Benzoic acid	HRL_{93}	Chronic	30,000	None
119-61-9	Benzophenone Toxicological Summary for Benzophenone (PDF)	HBV_{20}	Acute	ND	--
			Short-term	900	Developmental
			Subchronic	100	Liver System; Kidney System
			Chronic	100**	Liver System; Kidney System
			Cancer	NA	--

	Information Sheet: Benzophenone and Groundwater (PDF)				
95-14-7	1H-Benzotriazole Toxicological Summary for 1H-Benzotriazole (PDF) Information Sheet: 1H-Benzotriazole, Tolyltriazole, 5-Methyl-1H-Benzotriazole and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	20	Developmental
			Subchronic	20*	Developmental
			Chronic	20*	Developmental
			Cancer	NA	--
29385-43-1	methyl-1H-Benzotriazole (Tolyltriazole) Toxicological Summary for Tolyltriazole and 5-methyl-1H-Benzotriazole (PDF) Information Sheet: 1H-Benzotriazole, Tolyltriazole, 5-Methyl-1H-Benzotriazole and Groundwater (PDF)	RAA_{19}	Acute	ND	--
			Short-term	20	Developmental
			Subchronic	20*	Developmental
			Chronic	20*	Developmental
			Cancer	NA	--
136-85-6	5-methyl-1H-Benzotriazole Toxicological Summary for Tolyltriazole and 5-methyl-1H-Benzotriazole (PDF) Information Sheet: 1H-Benzotriazole, Tolytriazole, 5-Methyl-1H-Benzotriazole and Groundwater (PDF)	RAA_{19}	Acute	ND	--
			Short-term	20	Developmental
			Subchronic	20*	Developmental
			Chronic	20*	Developmental
			Cancer	NA	--
7440-41-7	Beryllium	HRL_{93}	Cancer	0.08	Cancer
92-52-4		HRL_{93}	Chronic	300	Kidney system

	1,1'-Biphenyl (Diphenyl) Toxicological Summary for 1,1'-Biphenyl (PDF)	HBV_{21}	Acute	400	Kidney system
			Short-term	100	Kidney system
			Subchronic	100*	Kidney system
			Chronic	100*	Kidney system
			Cancer	10	Cancer
111-44-4	Bis(chloroethyl) ether (BCEE)	HRL_{93}	Cancer	0.3	Cancer
542-88-1	Bis(chloromethyl) ether (BCME)	HRL_{93}	Cancer	0.002	Cancer
80-05-7	Bisphenol A (BPA) Toxicological Summary for Bisphenol A (PDF) Information Sheet: Bisphenol A in Drinking Water (PDF)	HRL_{15}	Acute	ND	--
			Short-term	100	Developmental; Female reproductive system (E); Liver system; Male reproductive system (E); Kidney system; Thyroid (E)
			Subchronic	20	Liver system; Kidney system
			Chronic	20**	Liver system; Kidney system
			Cancer	NA	--
7440-42-8	Boron Toxicological Summary for Boron (PDF) Information Sheet: Boron and Drinking Water (PDF)	RAA_{17}	Acute	ND	--
			Short-term	500	Developmental
			Subchronic	500	Developmental
			Chronic	500	Developmental
			Cancer	NA	--
75-27-4	Bromodichloromethane Toxicological Summary for Bromodichloromethane (PDF) Information Sheet: Bromodichloromethane and Groundwater (PDF)	HRL_{93}	Cancer	6	Cancer
		HBV_{20}	Acute	400	Female reproductive system (E)
			Short-term	30	Immune system; Spleen
			Subchronic	30*	Immune system; Spleen
			Chronic	30	Liver system
			Cancer	3	Cancer
75-25-2	Bromoform	HRL_{93}	Cancer	40	Cancer
74-83-9	Bromomethane (Methyl bromide)	HRL_{93}	Chronic	10	Gastrointestinal system
71-36-3	n-Butanol	HRL_{93}	Chronic	700	Nervous system

85-68-7	Butyl benzyl phthalate (BBP) Toxicological Summary for Butyl benzyl phthalate (PDF) Information Sheet: Phthalates and Drinking Water (PDF)	HRL_{15}	Acute	100	Developmental (E)
			Short-term	100	Developmental (E)
			Subchronic	100*	Developmental (E)
			Chronic	100*	Developmental (E)
			Cancer	NA	--
85-70-1	Butylphthalyl butylglycolate (BPBG)	HRL_{93}	Chronic	7,000	None
7440-43-9	Cadmium Toxicological Summary for Cadmium (PDF) Information sheet: Cadmium and Drinking Water (PDF)	HRL_{15}	Acute	5	Developmental
			Short-term	1	Developmental; Nervous system; Kidney system
			Subchronic	1	Developmental; Skeletal
			Chronic	0.5	Kidney system; Skeletal
			Cancer	NA	--
298-46-4	Carbamazepine Toxicological Summary for Carbamazepine (PDF) Information Sheet: Carbamazepine in Drinking Water (PDF)	HRL_{13}	Acute	40	Developmental; Nervous system
			Short-term	40	Developmental; Blood system; Liver system; Immune system; Nervous system; Male reproductive system (E); Female reproductive system (E); Thyroid (E)
			Subchronic	40*	Developmental; Blood system; Liver system; Immune system; Nervous system; Male reproductive system (E); Female reproductive system (E);Thyroid (E)
			Chronic	40*	Developmental; Blood system; Liver system; Immune system; Nervous system; Male reproductive system (E); Female reproductive system (E); Thyroid (E)
			Cancer	NA	--
75-15-0	Carbon disulfide	HRL_{93}	Chronic	700	Developmental

56-23-5	Carbon tetrachloride Toxicological Summary for Carbon tetrachloride (PDF)	HRL_{13}	Acute	100	Developmental; Liver system
			Short-term	3	Liver system
			Subchronic	3*	Liver system
			Chronic	3*	Liver system
			Cancer	1	Cancer
133-90-4	Chloramben	HRL_{94}	Chronic	100	Liver system
108-90-7	Chlorobenzene	HRL_{93}	Chronic	100	Liver system
75-00-3	Chloroethane Toxicological Summary for Chloroethane (PDF)	RAA_{16}	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	ND	--
			Cancer	NA	--
67-66-3	Chloroform Toxicological Summary for Chloroform (PDF) Information Sheet: Chloroform and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	20	Developmental; Liver system; Immune system
			Subchronic	20*	Developmental; Liver system; Immune system
			Chronic	20*	Developmental; Liver system; Immune system
			Cancer	NA	--
95-57-8	2-Chlorophenol	HRL_{93}	Chronic	30	Developmental
1897-45-6	Chlorothalonil	HRL_{94}	Cancer	30	Cancer
2921-88-2	Chlorpyrifos Toxicological Summary for Chlorpyrifos (PDF) Information Sheet: Chlorpyrifos and Drinking Water (PDF)	HBV_{13}	Acute	2	Nervous system
			Short-term	0.6	Nervous system
			Subchronic	0.6*	Nervous system
			Chronic	0.6*	Nervous system
			Cancer	NA	--
5598-15-2	Chlorpyrifos Oxon (degradate of Chlorpyrifos)	RAA_{13}	Acute	0.9	Nervous system
			Short-term	0.4	Nervous system
			Subchronic	0.4*	Nervous system
			Chronic	0.4*	Nervous system

	Toxicological Summary for Chlorpyrifos Oxon (PDF) Information Sheet: Chlorpyrifos and Drinking Water (PDF)		Cancer	NA	--
16065-83-1	Chromium III	HRL_{94}	Chronic	20,000	None
18540-29-9	Chromium VI	HRL_{93}	Chronic	100	None
210880-92-5; 205510-53-8	Clothianidin Toxicological Summary for Clothianidin (PDF) Information Sheet: Clothianidin and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	200	Developmental
			Subchronic	200*	Developmental
			Chronic	200*	Developmental
			Cancer	NA	--
98-82-8	Cumene (Isopropyl benzene)	HRL_{93}	Chronic	300	None
21725-46-2	Cyanazine Toxicological Summary for Cyanazine (PDF) *See also degradates Cyanazine acid, Cyanazine amide, Deethylcyanazine, Deethylcyanazine acid, Deethylcyanazine amide, Deethyldeisopropylatrazine, and Deisopropylatrazine* as described in the Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF).	HRL_{18}	Acute	3	Developmental; Female reproductive system
			Short-term	3	Developmental; Female reproductive system
			Subchronic	3	Developmental; Female reproductive system; Liver system; Kidney system
			Chronic	1	None
			Cancer	NA	--
36576-43-9; 36576-42-8	Cyanazine acid (CAC) and Cyanazine amide (CAM) (degradates of Cyanazine)	RAA_{20}	Acute	3	Developmental; Female reproductive system
			Short-term	3	Developmental; Female reproductive system
			Subchronic	3	Developmental; Female reproductive

	Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF)				system; Liver system; Kidney system
			Chronic	1	None
			Cancer	NA	--
57-12-5	Cyanide, free	HRL_{93}	Chronic	100	Nervous system; Thyroid (E)
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6190-65-4	Deethylatrazine (DEA) (degradate of Atrazine) Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF)	RAA_{20}	Chronic	3	None
21725-40-6; 36749-35-6; 36556-77-1	Deethylcyanazine (DEC), Deethylcyanazine acid (DCAC), and Deethylcyanazine amide (DCAM) (degradates of Cyanazine) Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF)	RAA_{20}	Acute	3	Developmental; Female reproductive system
			Short-term	3	Developmental; Female reproductive system
			Subchronic	3	Developmental; Female reproductive system; Liver system; Kidney system
			Chronic	1	None
			Cancer	NA	--
3397-62-4; 1007-28-9	Deethyldeisopropylatrazine (DACT, DEDI, DDA) and Deisopropylatrazine (DIA) (degradates of Atrazine and Cyanazine) Toxicological Summary for Cyanazine and Atrazine Chlorinated Degradates (PDF)	RAA_{20}	Use Cyanazine HRL values if Cyanazine, CAC, CAM, DEC, DCAC or DCAM are present. Use Atrazine HRL value if Cyanazine, CAC, CAM, DEC, DCAC or DCAM are not present.		
93413-62-8; 386750-22-7; 300827-87-6; 93414-04-1	Desvenlafaxine Toxicological Summary for Desvenlafaxine (PDF)	HBV_{15}	Acute	ND	--
			Short-term	20	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)
			Subchronic	20*	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)

	Information Sheet: Venlafaxine and Desvenlafaxine and Drinking Water (PDF)		Chronic	20*	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)
			Cancer	NA	--
124-48-1	Dibromochloromethane	HRL_{93}	Chronic	10	Liver system
106-93-4	1,2-Dibromoethane (ethylene dibromide, EDB)	HRL_{93}	Cancer	0.004	Cancer
84-74-2	Dibutyl phthalate (DBP) Toxicological Summary for Dibutyl phthalate (PDF) Information Sheet: Phthalates and Drinking Water (PDF)	HRL_{15}	Acute	20	Developmental (E)
			Short-term	20	Developmental (E)
			Subchronic	20*	Developmental (E)
			Chronic	20*	Developmental (E)
			Cancer	NA	--
1918-00-9	Dicamba	HRL_{93}***	Chronic	200	Developmental
95-50-1	1,2-Dichlorobenzene	HRL_{93}	Chronic	600	Liver system
106-46-7	1,4-Dichlorobenzene (para)	HRL_{94}***	Cancer	10	Cancer
	1,4-Dichlorobenzene Toxicological Summary for 1,4-Dichlorobenzene (PDF) Information Sheet: 1,4-Dichlorobenzene and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	50	Developmental; Liver system; Nervous system
			Subchronic	50*	Developmental; Liver system; Nervous system
			Chronic	50*	Developmental; Liver system; Nervous system
			Cancer	NA	--
91-94-1	3,3'-Dichlorobenzidine	HRL_{93}	Cancer	0.8	Cancer
75-71-8	Dichlorodifluoromethane Toxicological Summary for Dichlorodifluoromethane (PDFs)	HRL_{11}***	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	700	None
			Cancer	NA	--
		RAA_{17}	Acute	ND	--
			Short-term	ND	--

	Dichlorodifluoromethane Toxicological Summary for Dichlorodifluoromethane (PDF) Information Sheet: Dichlorodifluoromethane and Drinking Water (PDF)		Subchronic	ND	--
			Chronic	500	None
			Cancer	NA	--
72-54-8	p,p'-Dichlorodiphenyldichloroethane (DDD)	HRL_{93}	Cancer	1	Cancer
72-55-9	p,p'-Dichlorodiphenyldichloroethylene (DDE)	HRL_{93}	Cancer	1	Cancer
50-29-3	p,p'-Dichlorodiphenyltrichloroethane (DDT)	HRL_{93}	Cancer	1	Cancer
75-34-3	1,1-Dichloroethane Toxicological Summary for 1,1-Dichloroethane (PDF)	RAA_{16}	Acute	ND	--
			Short-term	400	Nervous system
			Subchronic	400*	Nervous system
			Chronic	80	Nervous system
			Cancer	NA	--
107-06-2	1,2-Dichloroethane Toxicological Summary for 1,2-Dichloroethane (PDF)	HRL_{13}	Acute	ND	--
			Short-term	200	Liver system
			Subchronic	200*	Liver system
			Chronic	60	Kidney system; Liver system
			Cancer	1	Cancer
156-59-2	cis-1,2-Dichloroethene Toxicological Summary for cis-1,2-Dichloroethene (PDF) Information sheet: cis-1,2-Dichloroethene and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	20	Liver system
			Subchronic	10	Kidney system
			Chronic	6	Kidney system
			Cancer	NA	--
156-60-5	trans-1,2-Dichloroethene	HRL_{13}	Acute	ND	--
			Short-term	ND	--
			Subchronic	200	Immune system

	Toxicological Summary for trans-1,2-Dichloroethene (PDF)		Chronic	40	Immune system
			Cancer	NA	--
	trans-1,2-Dichloroethene Toxicological Summary for trans-1,2-Dichloroethene (HBV 2020) (PDF)	HBV_{20}	Acute	ND	--
			Short-term	ND	--
			Subchronic	50	Immune system
			Chronic	9	Immune system
			Cancer	NA	--
75-35-4	1,1-Dichloroethylene (Vinylidene chloride) Toxicological Summary for 1,1-Dichloroethylene (Vinylidene chloride) (PDF)	HRL_{11}***	Acute	ND	--
			Short-term	ND	--
			Subchronic	200	Liver system
			Chronic	200	Liver system
			Cancer	NA	--
	1,1-Dichloroethylene (Vinylidene chloride) Toxicological Summary for 1,1-Dichloroethylene (Vinylidene chloride) (2020) (PDF) Information Sheet: 1,1-Dichloroethylene and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	ND	--
			Subchronic	200	Liver system
			Chronic	200	Liver system
			Cancer	NA	--
75-43-4	Dichlorofluoromethane (DCFM) Toxicological Summary for Dichlorofluoromethane (PDF) Information sheet: Dichlorofluoromethane and Drinking Water (PDF)	RAA_{17}	Acute	ND	--
			Short-term	20	Developmental; Liver system; Immune system
			Subchronic	20*	Developmental; Liver system; Immune system;
			Chronic	20*	Developmental; Liver system; Immune system;
			Cancer	NA	--
75-09-2	Dichloromethane (Methylene chloride)	HRL_{MCL}	Chronic	5	see USEPA Organic Chemicals table
120-83-2	2,4-Dichlorophenol	HRL_{93}	Chronic	20	Immune system
94-75-7		HRL_{18}	Acute	ND	--

	2,4-Dichlorophenoxyacetic acid (2,4-D) Toxicological Summary for 2,4-Dichlorophenoxyacetic acid (PDF) Information Sheet: 2.4-D and Drinking Water (PDF)		Short-term	30	Adrenal; Developmental; Thyroid (E)
			Subchronic	30*	Adrenal; Developmental; Thyroid (E)
			Chronic	30*	Adrenal; Developmental; Thyroid (E)
			Cancer	ND	--
78-87-5	1,2-Dichloropropane Toxiocological Summary for 1,2-Dichloropropane (HBV 2021) (PDF)	HRL_{94}	Cancer	5	Cancer
		HBV_{21}	Acute	ND	--
			Short-term	20	Developmental
			Subchronic	20*	Developmental
			Chronic	20*	Developmental
			Cancer	3	Cancer
542-75-6	1,3-Dichloropropene	HRL_{94}	Cancer	2	Cancer
60-57-1	Dieldrin Toxicological Summary for Dieldrin (PDF) Information Sheet: Dieldrin and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	0.2	Developmental; Immune system; Nervous system
			Subchronic	0.2*	Developmental; Immune system; Nervous system
			Chronic	0.2	Developmental; Liver system; Immune system; Nervous system
			Cancer	0.006	Cancer
134-62-3	N,N-Diethyl-meta-toluamide (DEET) Toxicological Summary for N,N-Diethyl-meta-toluamide (DEET) (PDF) Information Sheet: DEET in Drinking Water (PDF)	HRL_{13}	Acute	ND	--
			Short-term	200	Developmental; Nervous system
			Subchronic	200*	Developmental; Nervous system
			Chronic	200*	Developmental; Nervous system
			Cancer	NA	--

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117-81-7	Di(2-ethylhexyl)phthalate (DEHP) Toxicological Summary for Di(2-ethylhexyl) phthalate (PDF) Information Sheet: Phthalates and Drinking Water (PDF)	HRL_{15}	Acute	20	Developmental (E); Male reproductive system (E)
			Short-term	20	Developmental (E); Male reproductive system (E)
			Subchronic	20*	Developmental (E); Male reproductive system (E)
			Chronic	20*	Developmental (E); Male reproductive system (E)
			Cancer	7	Cancer
84-66-2	Diethyl phthalate	HRL_{93}	Chronic	6,000	None
87674-68-8 163515-14-8	Dimethenamid & Dimethenamid-P Toxicological Summary for Dimethenamid & Dimethenamid-P (PDF) Information Sheet: Dimethenamid and Drinking Water (PDF) See also degradates: Dimethenamid ESA & Dimethenamid OXA (PDF)	HRL_{15}	Acute	ND	--
			Short-term	600	Developmental; Liver system; Nervous system; Female reproductive system
			Subchronic	600*	Developmental; Liver system; Nervous system; Female reproductive system
			Chronic	300	Liver system
			Cancer	NA	--
205939-58-8, 380412-59-9	Dimethenamid ESA Dimethenamid OXA Toxicological Summary for Dimethenamid Degradates: ESA and OXA (PDF) Information Sheet: Dimethenamid and Drinking Water (PDF)	RAA_{13}	Acute	ND	--
			Short-term	600	Developmental; Liver system; Nervous system; Female reproductive system
			Subchronic	600	Developmental; Liver system; Nervous system; Female reproductive system
			Chronic	300	Liver system
			Cancer	NA	--
105-67-9	2,4-Dimethylphenol	HRL_{93}	Chronic	100	Blood system; Nervous system
131-11-3	Dimethylphthalate	HRL_{94}	Chronic	70,000	Kidney system
51-28-5	2,4-Dinitrophenol	HRL_{94}	Chronic	10	Eyes
88-85-7		HRL_{18}	Acute	ND	--

	Dinoseb Toxicological Summary for Dinoseb (PDF) Information Sheet: Dinoseb and Drinking Water (PDF)		Short-term	8	Developmental
			Subchronic	8*	Developmental
			Chronic	8*	Developmental
			Cancer	NA	--
123-91-1	1,4-Dioxane Toxicological Summary for 1,4-Dioxane (PDF) Information Sheet: 1,4-Dioxane in Drinking Water (PDF)	HRL_{13}	Acute	ND	--
			Short-term	ND	--
			Subchronic	300	Liver system; Kidney system; Respiratory system
			Chronic	100	Liver system; Kidney system; Respiratory system
			Cancer	1	Cancer
298-04-4	Disulfoton	HRL_{94}	Chronic	0.3	Nervous system
57-63-6	17α-Ethinylestradiol Toxicological Summary for 17α-Ethinylestradiol (PDF) Information Sheet: 17α-Ethinylestradiol and Mestranol and Drinking Water (PDF)	HBV_{20}	Acute	ND	--
			Short-term	0.0005	Developmental (E); Female Reproductive system (E); Male Reproductive system (E)
			Subchronic	0.0002	Developmental
			Chronic	0.0002	Developmental
			Cancer	ND	--
100-41-4	Ethylbenzene Toxicological Summary for Ethylbenzene (2011) (PDF)	HRL_{11}***	Acute	ND	--
			Short-term	50	Liver system; Kidney system
			Subchronic	50*	Liver system; Kidney system
			Chronic	50*	Liver system; Kidney system
			Cancer	NA	--
	Ethylbenzene	HBV_{20}	Acute	ND	--
			Short-term	40	Liver system; Kidney system
			Subchronic	40*	Liver system; Kidney system

	Toxicological Summary for Ethylbenzene (2020) (PDF) Information Sheet: Ethylbenzene and Groundwater (PDF)		Chronic	40*	Liver system; Kidney system
			Cancer	NA	--
759-94-4	*S*-Ethyl-*N,N*-dipropylthiocarbamate (EPTC) Toxicological Summary for S-Ethyl-*N,N*-dipropylthiocarbamate (PDF) Information Sheet: S-Ethyl-*N,N*-dipropylthiocarbamate (EPTC) and Drinking Water (PDF)	HRL_{18}	Acute	300	Nervous system
			Short-term	300	Developmental; Female reproductive system; Nervous system
			Subchronic	90	Cardiovascular system
			Chronic	40	Cardiovascular system
			Cancer	NA	--
60-29-7	Ethyl ether Toxicological Summary for Ethyl ether (PDF)	RAA_{16}	Acute	ND	--
			Short-term	ND	--
			Subchronic	1000	Liver system; Kidney system
			Chronic	200	Liver system; Kidney system
			Cancer	NA	--
107-21-1	Ethylene glycol Toxicological Summary for Ethylene glycol (HRL 2011) (PDF)	HRL_{11}***	Acute	4,000	Developmental
			Short-term	4,000	Developmental
			Subchronic	2,000	Developmental; Kidney system
			Chronic	2,000	Developmental; Kidney system
			Cancer	NA	--
	Ethylene glycol Toxicological Summary for Ethylene glycol (HBV 2020) (PDF)	HBV_{20}	Acute	ND	--
			Short-term	2,000	Developmental
			Subchronic	2,000	Developmental; Kidney system
			Chronic	2,000	Developmental; Male reproductive system; Kidney system
			Cancer	NA	--
206-44-0		HRL_{18}	Acute	ND	--

	Fluoranthene Toxicological Summary for Fluoranthene (PDF) Information Sheet: Fluoranthene in Drinking Water (PDF)		Short-term	ND	--
			Subchronic	200	Liver system; Kidney system
			Chronic	70	Liver system; Kidney system
			Cancer	NA	see Guidance for Evaluating the Cancer Potency of PAH Mixtures (PDF)
86-73-7	Fluorene (9H-Fluorene) Toxicological Summary for Fluorene (PDF) Information Sheet: Fluorene and Groundwater (PDF)	HRL_{93}***	Chronic	300	Blood system
		HBV_{20}	Acute	ND	--
			Short-term	ND	--
			Subchronic	200	Blood system; Spleen
			Chronic	80	Blood system; Spleen
			Cancer	NA	--
72178-02-0	Fomesafen Toxicological Summary for Fomesafen (PDF)	HBV_{20}	Acute	ND	--
			Short-term	200	Developmental; Liver system; Immune system
			Subchronic	200*	Developmental; Liver system; Immune system
			Chronic	20	Liver system
			Cancer	NA	--
50-00-0	Formaldehyde	HRL_{94}	Chronic	1,000	Gastrointestinal system
1071-83-6; 38641-94-0; 40465-76-7; 34494-04-7; 114370-14-8; 39600-42-5	Glyphosate Toxicological Summary for Glyphosate (PDF) Information Sheet: Glyphosate and Drinking Water (PDF)	HBV_{17}	Acute	ND	--
			Short-term	1,000	Developmental
			Subchronic	1,000	Gastrointestinal system
			Chronic	500	Gastrointestinal system
			Cancer	NA	--
76-44-8	Heptachlor	HRL_{93}	Cancer	0.08	Cancer
1024-57-3	Heptachlor epoxide	HRL_{93}	Cancer	0.04	Cancer
118-74-1	Hexachlorobenzene	HRL_{93}	Cancer	0.2	Cancer
87-68-3	Hexachlorobutadiene	HRL_{93}	Chronic	1	Kidney system

110-54-3	Hexane (n-hexane)	HRL_{94}	Chronic	400	Nervous system
138261-41-3	Imidacloprid Toxicological Summary for Imidacloprid (PDF) Information Sheet: Imidacloprid and Groundwater (PDF)	HBV_{20}	Acute	100	Nervous system
			Short-term	2	Immune system
			Subchronic	2*	Immune system
			Chronic	2*	Immune system
			Cancer	NA	--
78-83-1	Isobutanol Toxicological Summary for Isobutanol (PDF) Information Sheet: Isobutanol and Drinking Water (PDF)	HBV_{16}	Acute	ND	--
			Short-term	ND	--
			Subchronic	700	Male reproductive system
			Chronic	300	Male reproductive system
			Cancer	NA	--
78-59-1	Isophorone	HRL_{93}	Chronic	100	Kidney system
330-55-2	Linuron	HRL_{93}	Chronic	1	Blood system
7439-96-5	Manganese Toxicological Summary for Manganese (PDF) Manganese in Drinking Water	HRL_{93***}	Chronic	100	Nervous system
		HBV_{20}	Acute	ND	--
			Short-term	100	Developmental; Nervous system
			Subchronic	ND	--
			Chronic	ND	--
			Cancer	NA	--
72-33-3	Mestranol Toxicological Summary for Mestranol (PDF) Information Sheet: 17α-Ethinylestradiol and Mestranol and Drinking Water (PDF)	RAA_{16}	Acute	ND	--
			Short-term	0.0007	Developmental (E); Female Reproductive system (E); Male Reproductive system (E)
			Subchronic	0.0002	Developmental
			Chronic	0.0002**	Developmental
			Cancer	ND	--
67-56-1	Methanol	HRL_{94}	Chronic	3,000	Liver system; Nervous system

94-74-6	2-Methyl-4-chlorophenoxyacetic acid (MCPA)	HRL_{93}	Chronic	3	Liver system; Kidney system
78-93-3	Methyl ethyl ketone (MEK, 2-butanone)	HRL_{94}	Chronic	4,000	Developmental
108-10-1	Methyl isobutyl ketone (MIBK)	HRL_{94}	Chronic	300	Liver system; Kidney system
1634-04-4	Methyl tert-butyl ether (MTBE) Toxicological Summary for Methyl tert-butyl ether (MTBE) (PDF) Information Sheet for Methyl tert-butyl ether (MTBE) and Drinking Water (PDF)	RAA_{13}	Acute	ND	--
			Short-term	700	Liver system; Nervous system; Kidney system
			Subchronic	700*	Liver system; Nervous system; Kidney system
			Chronic	700*	Liver system; Nervous system; Kidney system
			Cancer	60	Cancer
91-57-6	2-Methylnaphthalene Toxicological Summary for 2-Methylnaphthalene (PDF) Information Sheet: 2-Methylnaphthalene and Drinking Water (PDF)	RAA_{13}	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	8	Respiratory system
			Cancer	ND	--
95-48-7	2-Methylphenol (o-cresol)	HRL_{93}	Chronic	30	Nervous system
108-39-4	3-Methylphenol (m-cresol)	HRL_{93}	Chronic	30	Nervous system
106-44-5	4-Methylphenol (p-cresol)	HRL_{94}	Chronic	3	None
51218-45-2; 87392-12-9	Metolachlor and s-Metolachlor Toxicological Summary for Metolachlor and s-Metolachlor (HRL 2011) (PDF) *See also* degradates Metolachlor ESA Metolachlor OXA	HRL_{11***}	Acute	400	Developmental
			Short-term	400	Developmental
			Subchronic	300	None
			Chronic	300**	None
			Cancer	NA	--
	Metolachlor and s-Metolachlor	HBV_{20}	Acute	ND	--
			Short-term	300	Developmental
			Subchronic	300*	Developmental
			Chronic	300*	Developmental

	Toxicological Summary for Metolachlor and s-Metolachlor (HBV 2020) (PDF) Information Sheet: Metolachlor and Drinking Water (PDF)		Cancer	NA	--
171118-09-5	Metolachlor ESA Toxicological Summary for Metolachlor ESA (HRL 2011) (PDF) (degradate of Metolachlor)	HRL_{11***}	Acute	ND	--
			Short-term	ND	--
			Subchronic	4,000	Liver system
			Chronic	800	Liver system
			Cancer	NA	--
	Metolachlor ESA (degradate of Metolachlor) Toxicological Summary for Metolachlor ESA (HBV 2020) (PDF) Information Sheet: Metolachlor ESA/OXA and Drinking Water (PDF)	HBV_{20}	Acute	ND	--
			Short-term	ND	--
			Subchronic	7,000	Liver system
			Chronic	1,000	Liver system
			Cancer	NA	--
152019-73-3	Metolachlor OXA Toxicological Summary for Metolachlor OXA (HRL 2011) (PDF) (degradate of Metolachlor)	HRL_{11***}	Acute	ND	--
			Short-term	3,000	None
			Subchronic	3,000*	None
			Chronic	800	None
			Cancer	NA	--
	Metolachlor OXA	HBV_{20}	Acute	ND	--
			Short-term	5,000	None
			Subchronic	5,000*	None
			Chronic	1,000	None

	(degradate of Metolachlor) Toxicological Summary for Metolachlor OXA (HBV 2020) (PDF) Information Sheet: Metolachlor ESA/OXA and Drinking Water (PDF)		Cancer	NA	--
21087-64-9	Metribuzin Toxicological Summary for Metribuzin (PDF) See also degradates: Metribuzin DA, DK, and DADK (PDF)	HRL_{13}	Acute	30	Developmental; Nervous system
			Short-term	10	Thyroid (E)
			Subchronic	10*	Thyroid (E)
			Chronic	10*	Thyroid (E)
			Cancer	NA	--
35045-02-4; 56507-37-0; 52236-30-3	Metribuzin DA, DK, and DADK Toxicological Summary for Metribuzin Degradates DA, DK, and DADK (PDF) Information Sheet: Metribuzin Degradates in Drinking Water (PDF)	RAA_{12}	Acute	30	Developmental; Nervous system
			Short-term	10	Thyroid (E)
			Subchronic	10*	Thyroid (E)
			Chronic	10*	Thyroid (E)
			Cancer	NA	--
101043-37-2	Microcystin-LR Toxicological Summary for Microcystin-LR (PDF) Information Sheet: Microcystin-LR in Drinking Water (PDF)	HBV_{15}	Acute	NA	--
			Short-term	0.1	Liver System
			Subchronic	0.1*	Liver System
			Chronic	0.1	Liver System
			Cancer	NA	--
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91-20-3		HRL_{13}	Acute	70	Nervous system

	Naphthalene Toxicological Summary for Naphthalene (PDF)		Short-term	70	Nervous system
			Subchronic	70*	Nervous system
			Chronic	70	Nervous system; Spleen
			Cancer	NA	--
7440-02-0	Nickel, soluble salts	HRL_{93}	Chronic	100	None
14797-55-8	Nitrate (as nitrogen)	HRL_{MCL}	Acute	10,000	see USEPA Inorganic Chemicals table
62-75-9	N-Nitrosodimethylamine (NDMA) Toxicological Summary for N-Nitrosodimethylamine (NDMA) (PDF) Information Sheet: N- Nitrosodimethylamine and Water (PDF)	HBV_{17}	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	ND	--
			Cancer	0.005	Cancer
86-30-6	N-Nitrosodiphenylamine	HRL_{93}	Cancer	70	Cancer
84852-15-3	Nonylphenol Toxicological Summary for p-Nonylphenol, branched isomers (PDF) Information Sheet: Nonylphenols and Drinking Water (PDF)	HBV_{20}	Acute	ND	--
			Short-term	100	Developmental; Female reproductive system
			Subchronic	40	Kidney system
			Chronic	20	Kidney system
			Cancer	NA	--
140-66-9	4-*tert*-Octylphenol Toxicological Summary for 4-*tert*-Octylphenol (PDF) Information Sheet: Octylphenol and Drinking Water (PDF)	HBV_{20}	Acute	ND	--
			Short-term	100	Developmental
			Subchronic	100*	Developmental
			Chronic	100*	Developmental
			Cancer	NA	--
87-86-5		HRL_{15}	Acute	7	Developmental; Thyroid (E)

	Pentachlorophenol (PCP) Toxicological Summary for Pentachlorophenol (PCP) (PDF) Information Sheet: Pentachlorophenol and Drinking Water (PDF)		Short-term	7	Developmental (E); Thyroid (E)
			Subchronic	7*	Developmental (E); Liver system; Immune system; Male Reproductive system; Thyroid (E)
			Chronic	7*	Developmental (E); Liver system; Immune system; Male Reproductive system; Thyroid (E)
			Cancer	0.3	Cancer
45187-15-3; 375-73-5	Perfluorobutane sulfonate (PFBS) Toxicological Summary for Perfluorobutane sulfonate (PFBS) (HRL 2011) (PDF)	HRL_{11***}	Acute	ND	--
			Short-term	ND	--
			Subchronic	9	Blood system; Liver system; Kidney system
			Chronic	7	Blood system; Liver system; Kidney system
			Cancer	NA	--
	Perfluorobutane sulfonate (PFBS) Toxicological Summary for Perfluorobutane sulfonate (PFBS) (HBV 2020) (PDF) Information Sheet: PFBS and Drinking Water (PDF)	HBV_{20}	Acute	ND	--
			Short-term	3	Developmental; Female reproductive system (E); Thyroid (E)
			Subchronic	3*	Developmental; Female reproductive system (E); Thyroid (E)
			Chronic	2	Kidney system
			Cancer	NA	--
45048-62-2; 375-22-4	Perfluorobutanoate (PFBA) Toxicological Summary for Perfluorobutanoate (PFBA) (PDF)	HRL_{18}	Acute	ND	--
			Short-term	7	Liver system; Thyroid (E)
			Subchronic	7*	Liver system; Thyroid (E)
			Chronic	7*	Liver system; Thyroid (E)
			Cancer	NA	--

	Information Sheet: PFBA and Drinking Water (PDF)				
108427-53-8; 355-46-4; 3871-99-6	Perfluorohexane sulfonate (PFHxS) Toxicological Summary for Perfluorohexane sulfonate (PFHxS) (PDF) Information Sheet: PFHxS and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	0.047	Liver system; Thyroid (E)
			Subchronic	0.047	Liver system; Thyroid (E)
			Chronic	0.047	Liver system; Thyroid (E)
			Cancer	NA	--
92612-52-7; 307-24-4; 21615-47-4; 2923-26-4	Perfluorohexanoate (PFHxA) Toxicological Summary for Perfluorohexanoate (PFHxA) (PDF)	HBV_{21}	Acute	ND	--
			Short-term	0.2	Developmental; Thyroid (E)
			Subchronic	0.2*	Developmental; Thyroid (E)
			Chronic	0.2*	Developmental; Thyroid (E)
			Cancer	NA	--
45285-51-6; 335-67-1; 335-66-0; 3825-26-1; 2395-00-8; 335-93-3; 335-95-5	Perfluorooctanoate (PFOA) Toxicological Summary for Perfluorooctanoate (PFOA) (PDF) Information Sheet: PFOA and Drinking Water (PDF) Toxicokinetic Model Description	HRL_{18}	Acute	NA	--
			Short-term	0.035	Developmental; Liver system; Immune system; Kidney system
			Subchronic	0.035	Developmental; Liver system; Immune system; Kidney system
			Chronic	0.035	Developmental; Liver system; Immune system; Kidney system
			Cancer	NA	--
45298-90-6; 1763-23-1;		HRL_{09}***	Acute	ND	--
			Short-term	ND	--

29081-56-9; 70225-14-8; 2795-39-3; 29457-72-5	Perfluorooctane Sulfonate (PFOS) and Salts Toxicological Summary for Perfluorooctane Sulfonate (PFOS) and Salts (PDF)		Subchronic	ND	--
			Chronic	0.3	Developmental; Liver system; Thyroid (E)
			Cancer	NA	--
	Perfluorooctane Sulfonate (PFOS) Toxicological Summary for Perfluorooctane Sulfonate (PFOS) (2020) (PDF) Information Sheet: PFOS and Drinking Water (PDF)	HBV_{20}	Acute	NA	--
			Short-term	0.015	Adrenal (E); Developmental; Liver system; Immune system; Thyroid (E)
			Subchronic	0.015	Adrenal (E); Developmental; Liver system; Immune system; Thyroid (E)
			Chronic	0.015	Adrenal (E); Developmental; Liver system; Immune system; Thyroid (E)
			Cancer	NA	--
108-95-2	Phenol	HRL_{93}	Chronic	4,000	Developmental
1918-02-1	Picloram	HRL_{93}	Chronic	500	Liver system
1336-36-3	Polychlorinated biphenyls (PCBs)	HRL_{94}	Cancer	0.04	Cancer
1610-18-0	Prometon	HRL_{93}	Chronic	100	None
1918-16-7	Propachlor	HRL_{93}	Chronic	90	None
175013-18-0	Pyraclostrobin Toxicological Summary for Pyraclostrobin (PDF) Information Sheet: Pyraclostrobin in Drinking Water (PDF)	HBV_{16}	Acute	300	Developmental; Female Reproductive system
			Short-term	100	Developmental; Female Reproductive system; Gastrointestinal system; Spleen
			Subchronic	100	Developmental; Female Reproductive system; Gastrointestinal system; Blood system; Liver system; Immune system; Spleen
			Chronic	100**	Developmental; Female Reproductive system; Gastrointestinal system; Blood system; Liver system; Immune system; Spleen

			Cancer	NA	--
129-00-0	Pyrene Toxicological Summary for Pyrene (PDF) Information Sheet: Pyrene in Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	ND	--
			Subchronic	90	Kidney system
			Chronic	50	Kidney system
			Cancer	NA	--
91-22-5	Quinoline Toxicological Summary for Quinoline (PDF) Information Sheet: Quinoline and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	4	Blood system; Liver system; Kidney system; Respiratory system; Spleen
			Cancer	0.03	Cancer
7782-49-2	Selenium	HRL_{93}	Chronic	30	None
7440-22-4	Silver	HRL_{93}	Chronic	30	None
122-34-9	Simazine	HRL_{MCL}	Chronic	4	see USEPA Organic Chemicals table
7440-24-6	Strontium Toxicological Summary for Strontium (PDF) Information Sheet: Strontium and Groundwater (PDF)	RAA_{19}	Acute	ND	--
			Short-term	3000	Developmental; Skeletal
			Subchronic	3000*	Developmental; Skeletal
			Chronic	3000*	Developmental; Skeletal
			Cancer	NA	--
57-68-1; 1981-58-4	Sulfamethazine Toxicological Summary for Sulfamethazine (PDF) Information Sheet: Sulfonamide Antibiotics and Drinking Water (PDF)	HRL_{15}	Acute	ND	--
			Short-term	100	Thyroid
			Subchronic	100*	Thyroid
			Chronic	100*	Thyroid
			Cancer	NA	--
723-46-6		RAA_{13}	Acute	ND	--

	Sulfamethoxazole Toxicological Summary for Sulfamethoxazole (PDF) Information Sheet: Sulfonamide Antibiotics and Drinking Water (PDF)		Short-term	100	Thyroid
			Subchronic	100*	Thyroid
			Chronic	100*	Thyroid
			Cancer	NA	--
630-20-6	1,1,1,2-Tetrachloroethane	HRL_{93}	Chronic	70	Kidney system; Liver system
79-34-5	1,1,2,2-Tetrachloroethane	HRL_{94}	Cancer	2	Cancer
127-18-4	Tetrachloroethylene (PERC or PCE) Toxicological Summary for Tetrachloroethylene (PDF) Information Sheet: Tetrachloroethylene (PCE) and Drinking Water (PDF)	HRL_{MCL***}	Chronic	5	see USEPA Organic Chemicals table
		HBV_{21}	Acute	ND	--
			Short-term	ND	--
			Subchronic	7	Nervous system
			Chronic	7**	Nervous system
			Cancer	4	Cancer
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109-99-9	Tetrahydrofuran Toxicological Summary for Tetrahydrofuran (PDF) Information Sheet: Tetrahydrofuran and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	600	Developmental
			Subchronic	600*	Developmental
			Chronic	600*	Developmental
			Cancer	ND	--
7440-28-0	Thallium salts	HRL_{94}	Chronic	0.6	Liver system
153719-23-4	Thiamethoxam	HRL_{18}	Acute	ND	--
			Short-term	400	Developmental; Female reproductive system; Liver system

	Toxicological Summary for Thiamethoxam (PDF) Information Sheet: Thiamethoxam and Drinking Water (PDF)		Subchronic	200	Male reproductive system
			Chronic	200**	Male reproductive system
			Cancer	NA	--
7440-31-5	Tin	HRL_{94}	Chronic	4,000	Liver system; Kidney system
108-88-3	Toluene Toxicological Summary for Toluene 2011 (PDF)	HRL_{11***}	Acute	ND	--
			Short-term	200	Immune system; Nervous system
			Subchronic	200*	Immune system; Nervous system
			Chronic	200*	Immune system; Nervous system
			Cancer	NA	--
	Toluene Toxicological Summary for Toluene (2020) (PDF) Information Sheet: Toluene and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	70	Immune system; Nervous system
			Subchronic	70*	Immune system; Nervous system
			Chronic	70*	Immune system; Nervous system
			Cancer	NA	--
Not available	Total Petroleum Hydrocarbon. See Other Guidance: Chemical Specific				
8001-35-2	Toxaphene	HRL_{93}	Cancer	0.3	Cancer
120-82-1	1,2,4-Trichlorobenzene Toxicological Summary for 1,2,4-Trichlorobenzene (PDF)	HRL_{13}	Acute	ND	--
			Short-term	100	Liver system; Adrenal (E); Blood system
			Subchronic	100*	Liver system; Adrenal (E); Blood system
			Chronic	100	Liver system; Adrenal (E); Kidney system
			Cancer	4	Cancer
108-70-3	1,3,5-Trichlorobenzene	RAA_{12}	Acute	ND	--
			Short-term	100	Liver system; Adrenal (E); Blood System
			Subchronic	100*	Liver system; Adrenal (E); Blood system

	Toxicological Summary for 1,3,5-Trichlorobenzene (PDF)		Chronic	100	Liver system; Adrenal (E); Kidney system
			Cancer	4	Cancer
71-55-6	1,1,1-Trichloroethane Toxicological Summary for 1,1,1-Trichloroethane (PDF) Information Sheet: 1,1,1-Trichloroethane and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	ND	--
			Subchronic	9,000	Liver system; Male reproductive system
			Chronic	5,000	Liver system; Male reproductive system
			Cancer	NA	--
79-00-5	1,1,2-Trichloroethane	HRL_{93}	Chronic	3	Immune system
79-01-6	1,1,2-Trichloroethylene (TCE) Toxicological Summary for 1,1,2-Trichloroethylene (TCE) (PDF) Technical and Application Information Information Sheet: Trichloroethylene (TCE) and Water (PDF)	HRL_{15}	Acute	ND	--
			Short-term	0.4	Developmental; Immune
			Subchronic	0.4	Developmental; Immune
			Chronic	0.4**	Developmental; Immune
			Cancer	2	Cancer
75-69-4	Trichlorofluoromethane	HRL_{93}	Chronic	2,000	None
88-06-2	2,4,6-Trichlorophenol	HRL_{93}	Cancer	30	Cancer
93-76-5	2,4,5-Trichlorophenoxyacetic acid	HRL_{93}	Chronic	70	Developmental; Blood system
93-72-1	2-(2,4,5-Trichlorophenoxy) propionic acid	HRL_{MCL}	Chronic	50	see USEPA Organic Chemicals table
96-18-4	1,2,3-Trichloropropane Toxicological Summary for 1,2,3-Trichloropropane (PDF)	HRL_{13}	Acute	7	Developmental
			Short-term	7	Developmental
			Subchronic	7*	Developmental
			Chronic	7*	Developmental
			Cancer	0.003	Cancer

	Information Sheet: 1,2,3-TCP in Drinking Water (PDF)				
76-13-1	1,1,2-Trichloro-1,2,2-trifluoroethane	HRL_{93}	Chronic	200,000	None
101-20-2	Triclocarban Toxicological Summary for Triclocarban (PDF) Information Sheet: Triclocarban and Drinking Water (PDF)	RAA_{13}	Acute	ND	--
			Short-term	ND	--
			Subchronic	ND	--
			Chronic	100	Blood system; Liver system; Male reproductive system; Kidney system
			Cancer	NA	--
3380-34-5	Triclosan Toxicological Summary for Triclosan (PDF) Information Sheet: Triclosan and Drinking Water (PDF)	HRL_{15}	Acute	ND	--
			Short-term	50	Developmental; Liver system; Thyroid (E); Female Reproductive system (E)
			Subchronic	50*	Developmental; Liver system; Thyroid (E); Female Reproductive system (E)
			Chronic	50*	Developmental; Liver system; Thyroid (E); Female Reproductive system (E)
			Cancer	NA	--
526-73-8	1,2,3-Trimethylbenzene Toxicological Summary for 1,2,4-Trimethylbenzene; 1,3,5-Trimethylbenzene; and 1,2,3-Trimethylbenzene (2020) (PDF) Information Sheet: Trimethylbenzenes and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	30	Nervous system
			Subchronic	30*	Nervous system
			Chronic	30*	Nervous system
			Cancer	NA	--
95-63-6	1,2,4-Trimethylbenzene	HBV_{20}	Acute	ND	--
			Short-term	30	Nervous system
			Subchronic	30*	Nervous system

	Toxicological Summary for 1,2,4-Trimethylbenzene; 1,3,5-Trimethylbenzene; and 1,2,3-Trimethylbenzene (2020) (PDF) Information Sheet: Trimethylbenzenes and Groundwater (PDF)		Chronic	30*	Nervous system
			Cancer	NA	--
108-67-8	1,3,5-Trimethylbenzene Toxicological Summary for 1,3,5-Trimethylbenzene (2009) (PDF)	HRL_{09}***	Acute	ND	--
			Short-term	100	Liver system
			Subchronic	100*	Liver system
			Chronic	100*	Liver system
			Cancer	NA	--
	1,3,5-Trimethylbenzene Toxicological Summary for 1,2,4-Trimethylbenzene; 1,3,5-Trimethylbenzene; and 1,2,3-Trimethylbenzene (2020) (PDF) Information Sheet: Trimethylbenzenes and Groundwater (PDF)	HBV_{20}	Acute	ND	--
			Short-term	30	Nervous system
			Subchronic	30*	Nervous system
			Chronic	30*	Nervous system
			Cancer	NA	--
99-35-4	1,3,5-Trinitrobenzene	HRL_{93}	Chronic	0.3	None
78-51-3	Tris(2-butoxyethyl) phosphate (TBEP) Toxicological Summary for Tris(2-butoxyethyl) phosphate (PDF)	HBV_{20}	Acute	ND	--
			Short-term	30	Liver system
			Subchronic	30*	Liver system
			Chronic	30	Liver system
			Cancer	NA	--
115-96-8	Tris(2-chloroethyl) phosphate (TCEP) Toxicological Summary for Tris(2-chloroethyl)phosphate (PDF)	HRL_{13}	Acute	ND	--
			Short-term	300	Developmental; Nervous system; Kidney system
			Subchronic	200	Kidney system
			Chronic	200**	Kidney system
			Cancer	5	Cancer

	Information Sheet: TCEP in Drinking Water (PDF)				
13674-87-8	Tris(1,3-dichloroisopropyl)phosphate (TDCPP) Toxicological Summary for Tris(1,3-dichloroisopropyl)phosphate (PDF) Information Sheet: TDCPP and Drinking Water (PDF)	HBV_{21}	Acute	ND	--
			Short-term	ND	--
			Subchronic	20	Liver system; Kidney system
			Chronic	8	Kidney system; Male reproductive system
			Cancer	0.8	Cancer
7440-62-2	Vanadium	HRL_{94}	Chronic	50	None
93413-69-5; 99300-78-4	Venlafaxine Toxicological Summary for Venlafaxine (PDF) Information Sheet: Venlafaxine and Desvenlafaxine and Drinking Water (PDF)	HBV_{15}	Acute	ND	--
			Short-term	10	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)
			Subchronic	10*	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)
			Chronic	10*	Developmental; Gastrointestinal system; Male reproductive system; Nervous system (E)
			Cancer	NA	--
75-01-4	Vinyl Chloride Toxicological Summary for Vinyl Chloride (PDF) Information Sheet: Vinyl Chloride and Drinking Water (PDF)	HRL_{18}	Acute	ND	--
			Short-term	ND	--
			Subchronic	90	Liver system
			Chronic	10	Liver system
			Cancer	0.2	Cancer
1330-20-7		HRL_{11}***	Acute	800	Nervous system

	Xylenes Toxicological Summary for Xylenes (mixture of isomers, o, m, p) (PDF)		Short-term	300	Nervous system
			Subchronic	300*	Kidney system; Nervous system
			Chronic	300*	Kidney system; Nervous system
			Cancer	NA	--
	Xylenes Toxicological Summary for Xylenes (2020) (PDF) Information Sheet: Xylenes and Groundwater (PDF)	HBV_{20}	Acute	700	Nervous system
			Short-term	300	Developmental; Nervous system
			Subchronic	300	Developmental; Nervous system; Kidney system
			Chronic	300**	Developmental; Nervous system; Kidney system
			Cancer	NA	--
7440-66-6	Zinc	HRL_{94}	Chronic	2,000	None
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NA - Not Applicable
ND - Not derived due to insufficient information
None - Nonspecific effects that could not be attributed to an organ system
(E) = Endocrine mediated effect on the specified target organ

*Set at short-term value
**Set at subchronic value
***Dual guidance applies: See Dual Guidance for Drinking WaterFor more information about this page, please contact the Environmental Health Division: health.risk@state.mn.us

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WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 5

WHO/HEP/ECH/WSH/2021.5

Manganese in drinking-water

Background document for development of WHO *Guidelines for drinking-water quality*

This document replaces document reference number WHO/SDE/WSH/03.04/02

https://www.who.int/publications/i/item/WHO-HEP-ECH-WSH-2021.5

WHO/HEP/ECH/WSH/2021.5

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Preface

Access to safe drinking-water is essential to health, a basic human right and a component of effective policy for health protection. A major World Health Organization (WHO) function to support access to safe drinking-water is the responsibility "to propose ... regulations, and to make recommendations with respect to international health matters ...", including those related to the safety and management of drinking-water.

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as *International standards for drinking-water*. It was revised in 1963 and 1971 under the same title. In 1984–1985, the first edition of the WHO *Guidelines for drinking-water quality* (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects, reviewing selected microorganisms, was published in 2002. The third edition of the GDWQ was published in 2004, the first addendum to the third edition was published in 2006, and the second addendum to the third edition was published in 2008. The fourth edition was published in 2011, and the first addendum to the fourth edition was published in 2017.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation relating to aspects of protection and control of drinking-water quality is accordingly prepared and updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other information to support the GDWQ, describing the approaches used in deriving guideline values, and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants of potential health concern in drinking-water. In the first and second editions, these constituted Volume 2 of the GDWQ. Since publication of the third edition, they comprise a series of free-standing monographs, including this one.

For each chemical contaminant or substance considered, a background document evaluating the risks to human health from exposure to that chemical in drinking-water was prepared. The draft health criteria document was submitted to a number of scientific institutions and selected experts for peer review. The draft document was also released to the public domain for comment. Comments were carefully considered and addressed, as appropriate, taking into consideration the processes outlined in *Policies and procedures used in updating the WHO guidelines for drinking-water quality* and the WHO *Handbook for guideline development*.

The revised draft was submitted for final evaluation at expert consultations.

During preparation of background documents and at expert consultations, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health Criteria monographs and Concise International Chemical Assessment Documents; the International Agency for Research on Cancer; the Joint Food and Agriculture Organization of the United Nations (FAO)/WHO Meeting on Pesticide Residues; and the Joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite, in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO website and in the current edition of the GDWQ.

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The work of the following experts was crucial in the development of this document and others in the second addendum to the fourth edition:

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The draft text was discussed at the expert consultations for the second addendum to the fourth edition of the GDWQ, including on 28–30 March 2017, 13–14 July 2018 and 2 March 2021. The final version of the document takes into consideration comments from both peer reviewers and the public, including P Aggett, Emeritus University of Central Lancashire, United Kingdom; C Alzamora, International Manganese Institute, France; V Bhat, formerly NSF International, United States of America; P Brandhuber, Brandhuber Water Quality & Treatment, United States of America; H Costa, Departamento da Qualidade, Portugal; J Donohue, Environmental Protection Agency, United States of America; JO Falkinham, Virginia Tech, United States of America; J Hunt, Drinking Water Inspectorate, United Kingdom; WR Knocke, Virginia Tech, United States of America; B Lampe, NSF International, United States of America; D Lee, PUB, Singapore; L Lejon, Flemish Agency for Care and Health, Belgium; O Loebel, European Federation of National Association of Water Services, Belgium; C Nishida, WHO, Switzerland; S Robjohns, Public Health England, United Kingdom; L Rogers, WHO, Switzerland; J Tobiason, University of Massachusetts Amherst, United States of America; and M Valcke, Institut National de Santé Publique du Québec, Canada.

The coordinator was Ms J De France, WHO. Strategic direction was provided by Mr B Gordon, WHO. Dr E Petersen and Dr S Madsen provided liaisons with the Joint FAO/WHO Expert Committee on Food Additives and the Joint FAO/WHO Meeting on Pesticide Residues. Dr R Brown and Ms C Vickers, WHO, provided liaisons with the International Programme on Chemical Safety. Dr M Perez contributed on behalf of the WHO Radiation Programme. Dr A Faragher, Biotext, Australia, was responsible for the scientific editing of the document.

Many individuals from various countries contributed to the development of the GDWQ. The efforts of all who contributed to the preparation of this document are greatly appreciated.

Acronyms and abbreviations

bw	body weight
CI	confidence interval
CNS	central nervous system
EFSA	European Food Safety Authority
GABA	gamma-aminobutyric acid
GI	gastrointestinal
GV	guideline value
LD_{50}	The dose of a chemical that has been calculated to cause death in 50% of a defined experimental animal population
MMT	methylcyclopentadienyl manganese tricarbonyl
Mn	manganese
LOAEL	lowest-observed-adverse-effect level
NOAEL	no-observed-adverse-effect level
OR	odds ratio
$PM_{2.5}$	particulate matter less than or equal to 2.5 μm in diameter
PM_{10}	particulate matter less than or equal to 10 μm in diameter
PND	postnatal day
TDI	tolerable daily intake
UF	uncertainty factor
USA	United States of America
WHO	World Health Organization

Contents

Executive summary

Manganese is an essential element that originates in the environment as a result of natural and anthropogenic sources. Oral exposure of the general population to manganese occurs primarily through food; however, manganese may also be present in drinking-water in varying amounts. Higher levels can occur as a result of industrial discharges, and under acidic or reducing conditions that are found in groundwater and in some lakes and reservoirs.

A number of epidemiological studies have identified associations between neurotoxic effects in children and increased exposure to manganese in drinking-water. Although limitations in these studies preclude their use for quantitative risk assessment, the findings support the choice of neurotoxicity as a key end-point of concern. The health risk assessment for manganese in drinking-water is based on studies in rats orally exposed to manganese that report neurotoxic effects consistent with those observed in the epidemiological studies.

A provisional health-based guideline value (pGV) of 80 µg/L is established for total manganese, based on identified health considerations for bottle-fed infants. Although infants have been identified as the most susceptible subpopulation, the pGV is also applicable to the general population as a whole. The health-based GV is considered provisional because of the high level of uncertainty in the overall assessment (as reflected in a composite uncertainty factor of 1000). As part of the hazard assessment phase of water safety planning, water sources should be assessed to determine if manganese is present. Where manganese is present at concentrations close to the pGV or the water is treated to remove manganese, routine monitoring should be conducted post-treatment. Several methods for removing manganese are available, including oxidation/filtration, adsorption/oxidation, softening/ion exchange and biological filtration. Selection of the appropriate treatment system for manganese removal depends on the form of manganese (dissolved or particulate) present in the source water. In general, treatment methods used for manganese rely on a combination of processes (e.g. oxidation, adsorption, filtration) to remove both the dissolved and particulate forms.

In cases where meeting the pGV is technically or financially unfeasible, incremental improvement is encouraged. Risks to infants arising from exceedance of the pGV may be mitigated by following the World Health Organization recommendation for exclusive breastfeeding, or by using an alternative safe source of drinking-water to prepare formula.

1 General description

1.1 Identity

Manganese is a transition metal and one of the most abundant metals in Earth's crust, frequently co-occurring with iron. It is a component of more than 100 minerals but is not found naturally in its pure (elemental) form (ATSDR, 2012). Manganese can exist in 11 oxidation states; the most environmentally and biologically important manganese compounds are those that contain Mn(II), Mn(III), Mn(IV) or Mn(VII). Manganese can form a large variety of complexes by combining with other elements such as oxygen, sulfur and chlorine, and with carbonates and silicates (Stokes & NRCC, 1988; ATSDR, 2012). Some of these compounds are listed in Table 1.1.

Table 1.1. Some manganese compounds

Compound	Chemical Abstracts No.	Molecular formula
Manganese(II) chloride	7773-01-5	$MnCl_2$
Manganese(II, III) oxide (manganese tetroxide)	1317-35-7	Mn_3O_4 ($MnO.Mn_2O_3$)
Manganese dioxide	1313-13-9	MnO_2
Potassium permanganate	7722-64-7	$KMnO_4$
Sodium permanganate	10101-50-5	$NaMnO_4$
Manganese sulfate	7785-87-7	$MnSO_4$

Source: ATSDR (2012).

1.2 Physicochemical properties

The physical and chemical properties of manganese and manganese compounds vary substantially (Table 1.2). These characteristics determine environmental behaviour and fate, exposure potential, and the toxicological impact of each compound or dissolved ion.

Table 1.2. Physicochemical properties of manganese and manganese compounds

Property	Mn	$MnCl_2$	Mn_3O_4	MnO_2	$KMnO_4$	$MnSO_4$
Melting point (°C)[a]	1244	650	1564	Loses oxygen at 535 °C	Decomposes at <240 °C	700
Boiling point (°C)[a]	1962	1190	No data	No data	No data	Decomposes at 850 °C
Density (g/cm^3)[a]	7.21–7.44	2.98	4.86	5.03	2.70	3.25
Water solubility (g/L at 20°C)[b]	0.001	799	Virtually insoluble	Virtually insoluble	≥64	>10

Sources: [a] ATSDR (2012); [b] European Chemicals Agency (2021).

1.3 Organoleptic properties

The taste threshold for dissolved Mn(II) has been estimated as 75.4 mg/L (50% population threshold). Dissolved Mn(II) is colourless and is visually undetectable at concentrations as high as 506 mg/L (maximum tested). In contrast, particulate Mn(IV) can be visually detected at a

concentration of 0.005 mg/L. Estimates of the taste threshold of particulate Mn(IV) are confounded by discolouration of the water by these compounds but have been reported to be >0.05 mg/L (Sain, Griffin & Dietrich, 2014).

When manganese is not adequately removed during treatment, soluble Mn(II) compounds may undergo oxidation (e.g. as a result of disinfection or other treatment processes), forming manganese oxides, which can cause discoloured water and staining of laundry and fixtures. This is supported by numerous studies of drinking-water systems that have reported that manganese concentrations above 0.02 mg/L cause complaints about discoloured water, staining of plumbing fixtures and laundry, and general dissatisfaction with the water quality (Sly, Hodgkinson & Arunpairojana, 1990; Sommerfield, 1999; Casale, LeChevallier & Pontius, 2002; Kohl & Medlar, 2006; Tobiason et al., 2008). An extensive review of the literature conducted by Kohl & Medlar (2006) indicated that manganese can be deposited in distribution systems as manganese oxides even when the concentration leaving the treatment plant is as low as 0.02 mg/L.

1.4 Major uses and sources

Manganese is used principally in the manufacture of iron and steel alloys. Manganese compounds are also used in fertilizers, livestock feeding supplements, fungicides, varnishes and pottery glazes (IPCS, 1999; ATSDR, 2012; International Manganese Institute, 2014). Manganese dioxide and other manganese compounds are used in products such as dry-cell batteries, glass and fireworks.

Potassium and sodium permanganate are common oxidants used for cleaning, bleaching and disinfection. Permanganate can be added during water treatment to remove iron and manganese, and improve taste and odour (IOM & National Research Council, 1982; ATSDR, 2012; Health Canada, 2019). Manganese can also be present as an impurity in coagulants (principally ferric-based coagulants) used in drinking-water treatment. Water treatment media with manganese oxide surfaces are used in some locations for potable water treatment (ATSDR, 2012) to remove iron and manganese. Manganese that accumulates on these media during the treatment process can be released into treated water when filters are improperly operated.

An organomanganese compound, methylcyclopentadienyl manganese tricarbonyl (MMT), can be used at low concentrations[1] as an octane-enhancing agent in unleaded petrol in some countries; in Canada, its use declined sharply after 2004 following voluntary action by Canadian petroleum refiners (Lynam et al., 1999; Walsh, 2007; Health Canada, 2019).

Manganese occurs naturally in many surface water and groundwater sources. Manganese in surface water and groundwater can result from natural leaching (e.g. from rock and soil weathering) and anthropogenic activities (e.g. industrial discharges, mining, landfill leaching) (Stokes & NRCC, 1988; Kohl & Medlar, 2006; Ljung & Vahter, 2007; ATSDR, 2012). The species of manganese in soil are dependent on the pH of the soil and/or the water, the reduction potential of the water and, to a lesser extent, soil mineralogy, oxidative microbial activity and organic matter content.

[1] Equivalent to 8.3 mg manganese/L in USA.

The main sources of particulate manganese in ambient air are industrial activities, including iron and steel production, burning of MMT-containing petrol, operation of power plants and coke ovens, and mining operations (creating dust). Manganese can also be released into the atmosphere during volcanic eruptions and forest fires, and from ocean spray and soil erosion (Stokes & NRCC, 1988; IPCS, 1999; US EPA, 2004).

1.5 Environmental fate

In water, the transport and partitioning of manganese depend on the chemical form present and its solubility, which is determined by pH, oxidation–reduction potential and characteristics of the available anions. Manganese can occur in particulate, colloidal and dissolved forms in surface water. The dissolved form (Mn(II)) is most common in groundwater, given that low levels of dissolved oxygen favour reduction of Mn(IV) to dissolved Mn(II). Most manganese salts are soluble in water to some extent (ATSDR, 2012; Health Canada, 2019). Manganese levels and retention in soils depend on the organic content and cation exchange capacity of the soil (ATSDR, 2012).

In air, manganese can exist as suspended particulate matter, which is removed largely by gravitational settling.

2 Environmental levels and human exposure

2.1 Water

Manganese occurs naturally in many surface water and groundwater sources, from dissolution of manganese oxides, carbonates and silicates in soil and rock. Anthropogenic activities (industrial discharges, mining and landfill leaching) can also be a source of manganese contamination of water (Stokes & NRCC, 1988; Kohl & Medlar, 2006; Ljung & Vahter, 2007).

When reducing conditions are present in groundwater, higher concentrations of dissolved manganese are favoured; up to 1300 µg/L in neutral groundwater and 9600 µg/L in acidic groundwater have been reported (ATSDR, 2012). Manganese levels tend to be lower in flowing rivers and streams because of the presence of dissolved oxygen, which limits the amount of manganese that is dissolved. Surface water supplies such as lakes and reservoirs can, however, become seasonally stratified, which causes the lower sections of the water body to become anoxic. This allows release of dissolved Mn(II) into the water column from manganese oxides that are present in sediments at the bottom of the water body (Civardi & Tompeck, 2015). Less commonly, elevated manganese concentrations can also occur in stream sources. The concentration is dependent on stream-flow conditions and the water sources feeding the stream (Brandhuber et al., 2013; Health Canada, 2019). Higher manganese levels in water bodies with higher dissolved oxygen are usually associated with industrial pollution.

Manganese concentrations in seawater are reported to range from 0.4 to 10 µg/L (ATSDR, 2012), with an average of about 2 µg/L. Levels in fresh water typically range from 1 to 200 µg/L. Manganese has been detected in about 97% of surface water sites in the United States of America at a median concentration of 16 µg/L (US EPA, 2002; ATSDR, 2012). ATSDR (2012) reported that a river water survey in the USA found dissolved manganese levels ranging from <11 to >51 µg/L. Since 1991, the National Water-Quality Assessment Project of the United States Geological Survey has gathered limited data on manganese from representative study basins around the USA, starting in 1991. Combined, these data indicate

a median manganese level of 16 µg/L in surface waters, with 99th percentile concentrations of 400–800 µg/L (Leahy & Thompson, 1994; USGS, 2001; US EPA, 2003).

Overall, the detection frequency of manganese in groundwater in the USA is high (approximately 70% of sites), as a result of the ubiquity of manganese in soil and rock. Groundwater in the USA contains median manganese levels of 5–150 µg/L (ATSDR, 2012).

The National Water-Quality Assessment Project data indicate that the 99th percentile level of manganese is generally higher in groundwater (5600 µg/L) than in surface waters, but the median level is lower in groundwater (5 µg/L) than in surface waters (16 µg/L) (USGS, 2001; US EPA, 2003). In contrast, maximum average annual concentrations were reported to be 3000 µg/L for groundwater and 500 µg/L for surface water for 179 treatment plants located across North America (Kohl & Medlar, 2006). However, the median values for groundwater and surface waters were similar and below 100 µg/L.

In the USA, the National Inorganic and Radionuclide Survey collected data from 989 community public water systems served by groundwater in 49 states between 1984 and 1986. Manganese was detected in 68% of systems, with a median concentration of 10 µg/L. Supplementary survey data from public water systems supplied by surface water in five states reported concentration ranges similar to those of groundwater (US EPA, 2002). The United States Environmental Protection Agency is currently monitoring manganese at more locations than were studied for the National Inorganic and Radionuclide Survey in 1989. As of July 2020, 1.9% of systems detected manganese at levels higher than 300 µg/L, and 88.5% of these systems detected manganese at levels higher than 0.4 µg/L (US EPA, 2020).

In Germany, the manganese concentration in drinking-water supplied to more than 98% of all households was less than 20 µg/L in 1991 (Bundesgesundheitsamt, 1991). More recently in Germany, it was reported that less than 1% of approximately 52 000 drinking-water samples taken post-treatment from water works supplying more than 1000 m^3 during 2017–2019 contained manganese at levels exceeding 50 µg/L (Federal Ministry of Health & Federal Environment Agency, 2021).

In the United Kingdom, four seasonal monitoring surveys were conducted on final drinking-water for up to 20 sites in England and Wales identified as being at potential risk of high manganese concentrations; 18 of these were public supplies, and two were private supplies. In general, low levels of total manganese, ranging from <0.1 to 11 µg/L, were reported (WRC, 2014). Among more than 44 000 drinking-water compliance samples taken in England and Wales in 2016, only 16 exceeded 50 µg/L; the maximum value reported was 706 µg/L, and the 95th percentile was 3.4 µg/L (PK Marsden, Drinking Water Inspectorate, personal communication, April 2017).

Low levels of manganese in source or treated water (current or historical) may accumulate in the distribution system and periodically lead to high levels of manganese at the tap due to physical disturbances or water quality changes (e.g. chemical release). In addition, other contaminants (such as heavy metals) that deposit with manganese oxides in the distribution system may also be released into the water and reach consumers' taps (Friedman et al., 2010; Brandhuber et al., 2015).

Exposure to high levels (400–1700 µg/L) of manganese in drinking-water have been reported in some regions, including low- or middle-income countries such as Bangladesh, Burma, China

and India (He, Liu & Zhang, 1994; Wasserman et al., 2006; Bacquart et al., 2012, 2015). Recent survey data from Costa Rica showed manganese levels up to 980 µg/L (Darner Mora Alvarado, Laboratorio National de Aguas, personal communication, June 2018).

2.1.1 Speciation of manganese in water

The concentration of manganese in groundwater and surface waters is influenced by the local chemical environment (e.g. organic carbon content, cation exchange capacity, pH, Eh – a measure of the redox state of a solution, and mineral and particulate content). These factors determine manganese speciation and, in turn, solubility (Stokes & NRCC, 1988; Kohl & Medlar, 2006). The most common oxidation states for manganese in natural water are Mn(II) and Mn(IV) (Stokes & NRCC, 1988; ATSDR, 2012; Rumsby et al., 2014)). The Mn(III), Mn(V) and Mn(VI) oxidation states are not stable in neutral (pH ≈7) solutions. In reducing environments and acidic media, and in the presence of nitrates, sulfates or chlorides, Mn(III) and Mn(IV) are reduced to Mn(II) (Stokes & NRCC, 1988; Kohl & Medlar, 2006; ATSDR, 2012). At alkaline pH (pH >8–9) and under oxidizing conditions (such as those found during water treatment where chlorine, chlorine dioxide, ozone or permanganate are used), Mn(II) is converted to Mn(IV), resulting in precipitation of manganese as Mn(IV) compounds, which are found as particulates in water (Kohl & Medlar, 2006). Mn(VII) can also be present within drinking-water treatment plants due its common use as a water treatment chemical.

A United Kingdom study (WRC, 2014) measured total and soluble manganese in 18 public supplies. The soluble manganese was reported as Mn(II), and the difference between the total and soluble was reported as Mn(IV). The Mn(II):Mn(IV) ratio varied from 8.3 to 0.04 but was typically about 1.4, indicating that slightly more Mn(II) than Mn(IV) was present in drinking-water. The survey also included analysis of two private borehole water supplies, in which high concentrations of manganese were detected, nearly all of which was in the Mn(II) form.

2.2 Food

Food is the most important source of manganese exposure for the general population. Since manganese is essential for photosynthesis and energy metabolism in plants, it is ubiquitous in vegetable-based foods, particularly whole grains, nuts and rice. Leafy vegetables, tea, seeds and legumes are also good sources (IOM, 2001; ATSDR, 2012; Freeland-Graves, Mousa & Kim, 2016). Co-exposure to dietary fibre, oxalic acids, tannins and phytic acids reduces manganese absorption (Gibson, 1994; Freeland-Graves, Mousa & Kim, 2016), whereas a low iron status (as reflected in low serum ferritin concentrations, which are possibly sex specific) can result in increased manganese absorption (Finley, 1999). For infants, both breast milk and breast milk substitutes may be sources of exposure, although constituents in both may affect bioavailability.

In the European Union, dietary manganese intakes in adolescents and adults were estimated to range between 2 and 6 mg/day, with most values being around 3 mg/day. Estimated manganese intake in children is lower: 1.5–3.5 mg/day (EFSA, 2013). The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment reviewed the results of the 2000 Total Diet Study, which measured exposure of the United Kingdom population to manganese. For adults, the mean and 97.5th percentile dietary intake rates of manganese were reported to be 5.2 mg/day and 9.2 mg/day, respectively. It was concluded that the estimated total dietary intake of manganese was unlikely to pose a risk to healthy adults (Committee on Toxicity, 2020). Dietary intakes in the Canadian population (all age groups) during the period 1993–

2007 were between 3.1 and 4.3 mg/day (Health Canada, 2019). These levels are comparable to the estimated dietary intakes of 3.8 mg/day in the USA (ATSDR, 2012) and 4.2 mg/day in Sweden (VKM, 2018). People eating vegetarian diets and diets typical of more developed countries may have manganese intakes as high as 10.9 mg/day (IOM, 2001).

Based on the results of the United States Food and Drug Administration's Total Diet Study (conducted from 1991 to 1997), among adults 19 years of age and older, the median and 95th percentile intakes of manganese from food were 2.1–2.3 mg/day and 5.2–6.3 mg/day, respectively, for men, and 1.6–1.8 mg/day and 4.3–4.6 mg/day, respectively, for women (IOM, 2001). Additionally, mean and 95th percentile manganese intakes from food among pregnant and lactating women were 2.1–2.6 mg/day and 5.8–5.9 mg/day, respectively (IOM, 2001).

Manganese concentrations reported in breast milk vary widely. A study of 70 human milk samples collected from breastfeeding women in Argentina (n = 21), Namibia (n = 6), Poland (n = 23) and the USA (n = 20) reported three- to four-fold differences in manganese concentrations in breast milk between the populations studied, with mean concentrations ranging between 1.6 and 11.6 µg/L (Klein et al., 2017). In this study, the average concentration of manganese from breast milk of US mothers was 2.71 µg/L (range 1.5–5.9 µg/L; n = 20) at approximately 7 months postpartum. In an earlier study of American mothers (Casey, Hambidge & Neville, 1985), the average concentration of manganese from breast milk was estimated at 3.7 µg/L (range 2.7–5.4 µg/L; n = 11) from days 6 to 31 postpartum, and the highest levels were measured at day 1 postpartum. Levels decreased to an average of 1.9 µg/L at 3 months postpartum (Casey, Hambidge & Neville, 1985; IOM, 2001).

In an analysis of seven studies of mothers residing in the European Union, the European Food Safety Authority (EFSA, 2013) reported mean manganese concentrations of 3–30 µg/L in breast milk. In an analysis conducted by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (Committee on Toxicity, 2020), the average exposure from breast milk for the 0–4-month age group was estimated to be 5.4 µg/kg/day, and 8.1 µg/kg/day for high-level exposure, in the United Kingdom, assuming a maximum concentration of manganese in breast milk of 40 µg/L. Based on preliminary data, Health Canada estimated a median manganese content of 2.2 ng/g (2.2 µg/L) in breast milk using data from the Total Diet Study (Health Canada, 2019).

Manganese concentrations reported in breast milk substitutes also vary widely and have been reported to be higher than in breast milk. In a study of manganese in infant breast milk substitutes and nutritional beverages for young children in the USA and France, the measured concentrations of manganese ranged from 230 to 830 µg/L in formulas labelled for infant use (Frisbie et al., 2019). Levels in soy-based human breast milk substitutes were particularly high. Mitchell et al. (2020) estimated mean daily manganese intakes in breast-fed infants and children in four age ranges (from 3 weeks to 18 months), based on estimated breast milk consumption rates among German breast-fed infants and published data on manganese concentrations in breast milk among populations in several countries in North America, Europe and Asia. The weighted mean of means for manganese concentrations in breast milk based on these data was 7.7 µg/L; the mean manganese intake from breast milk is estimated to be 1.2 µg/kg/day for a 3-week-old infant (maximum of 4.67 µg/kg/day) and 1.8 µg/kg/day for an 18-month old child (maximum of 6.97 µg/kg/day).

In the United Kingdom, average exposure to manganese for infants aged 0–6 months feeding exclusively on ready-to-feed formula was estimated to be 6.5–8.5 µg/kg/day and 10–

13 µg/kg/day in those who consumed high levels. Exposure to manganese where tap water with manganese concentrations of 1.4–15 µg/L was used to reconstitute formula was estimated at 9–14 µg/kg/day in average consumers and 14–21 µg/kg/day in high-level consumers (Committee on Toxicity, 2020). Average exposures based on concentrations detected in infant formulas in the USA and France (as reported by Frisbie et al., 2019, and assessed by Mitchell et al., 2020) would be higher. Once solid foods are introduced, the contribution of manganese intake from milk becomes less significant.

The World Health Organization (WHO)/Food and Agriculture Organization of the United Nations Codex Committee and the Expert Panel of the Life Science Research Office have set guidance levels of manganese for infant formula intended to be marketed as breast milk substitute to meet nutritional requirements. The minimum guidance level is 1 µg/100 kcal, and the upper level[2] is 100 µg/100 kcal (67 µg/100 mL) (Raiten, Talbot & Waters, 1998; WHO & FAO, 2016).

2.3 Air

Levels of manganese compounds in air vary widely, depending on the proximity of point sources, such as ferroalloy production facilities, coke ovens and power plants. Average manganese levels in ambient air near industrial sources have been reported to range from 220 to 300 ng/m^3, whereas ambient manganese levels in urban and rural areas without point sources have been reported to range from 10 to 70 ng/m^3. Over the past 30 years, levels of manganese emitted from the metals industry have decreased substantially because of the installation of emission controls (ATSDR, 2012).

The United States Environmental Protection Agency (US EPA, 2007) estimated the geometric mean annual background concentration of manganese in particulate matter less than or equal to 10 µm in diameter (PM_{10}) in urban areas to be 6.68 ng/m^3 (range 0.85–614 ng/m^3), based on 114 measurements in 20 urban locations across the USA. Existing data show little difference in manganese levels in ambient air between areas where MMT is used in petrol and areas where MMT is not used (Lynam et al., 1999).

Low manganese levels have been reported in atmospheric particulate matter in Canada, with a mean concentration of manganese in ambient air from 2009 to 2013 of 1.25×10^{-3} µg/m^3 (ranging from below the limit of detection to 6.2×10^{-2} µg/m^3), as averaged over 24 hours by the National Air Pollution Surveillance Program (Galarneau et al., 2016). Levels of manganese ($PM_{2.5}$ and PM_{10}) dropped between the late 1980s and early 2000s by 13–77% (Health Canada, 2010).

More recently, the United Kingdom reported average manganese concentrations in ambient air across rural and urban locations to be generally in the range 1–18 ng/m^3, although levels up to 76 ng/m^3 were also measured, associated with steel-making industries (DEFRA, 2019).

Loranger, Zayed & Forget (1994) found ambient air manganese concentrations to be significantly correlated with traffic density. Areas of intermediate and high traffic densities in

[2] Upper guidance levels are for nutrients without sufficient information for a science-based risk assessment. These levels are derived on the basis of meeting nutritional requirements of infants and an established history of apparent safe use.

Montreal had ambient air manganese concentrations above the natural background level of 40 ng/m^3 (Loranger & Zayed, 1994; Loranger, Zayed & Forget, 1994).

2.4 Bioaccumulation

Manganese can bioaccumulate in lower organisms (e.g. phytoplankton, algae, molluscs, some fish) but not in higher organisms; biomagnification in food chains is not expected to be significant (ATSDR, 2012).

2.5 Biomarkers of exposure

Manganese levels in urine (see section 3.4) can be used as a biomarker of exposure. However, urinary manganese reflects short-term exposure of only a few hours (Andersen, Gearhart & Clewell, 1999; Signes Pastor et al., 2019). Other biomarkers of manganese exposure include blood concentration, with background levels ranging from 6.7 to 7.6 µg/ml (Roels et al., 1992; Mergler et al., 1994; Loranger & Zayed, 1995), hair concentration (Fergusson, Holzbecher & Ryan, 1983; Chutsch & Krause, 1987; Eastman et al., 2013), and toenail concentration (Signes Pastor et al., 2019).

Although manganese levels in blood do not reflect long-term exposure, the blood platelet level of monoamine oxidase is an early biochemical indicator of adverse oxidative effects of manganese (Benedetti & Dostert, 1989; Humfrey et al., 1990; Abdelouahab et al., 2010).

Hair has been used as a longer-term biomarker of exposure in epidemiological studies. Proper treatment is required to ensure that any potential external manganese contamination is removed (Eastman et al., 2013).

More recently, toenail samples have been reported to be reliable biomarkers of environmental manganese exposure, including exposure to manganese from drinking-water (Signes Pastor et al., 2019). In theory, the slow growth of nails could provide an indication of exposure over several months (Signes Pastor et al., 2019).

2.6 Estimated total exposure and relative contribution of drinking-water

Manganese is essential to proper physiological function in both humans and other animals. It is required as a component or cofactor for many cellular enzymes (e.g. manganese superoxide dismutase, pyruvate carboxylase) and can activate many others (e.g. kinases, decarboxylases, transferases, hydrolases) that are also activated by similar divalent cations (IPCS, 2002).

The highest exposure to manganese is usually from food and is estimated to range from 2 to 6 mg/day in adults, although higher values have been reported (see section 2.2). Manganese intake from drinking-water is normally substantially lower than intake from food. At the drinking-water concentrations described above (section 2.1), the intake of manganese from drinking-water could be one or more orders of magnitude less than intake from food, assuming a daily water intake of 2 L.

There is potential for increased exposure to manganese in bottle-fed infants compared with breastfed infants – from the concentrated or powdered formula itself as well as the tap water used to prepare the formula. As noted in sections 2.1 and 2.2, there is high variability in manganese levels in drinking-water and in breast milk substitutes. However, in 4–18-month-old children in the United Kingdom, the exposure to manganese from tap water (2–15 µg/L) was found to make a negligible contribution to total exposure (Committee on Toxicity, 2020).

Further, once solid foods are introduced, the contribution from formula becomes less significant. The relative immaturity of the hepatobiliary excretion of manganese in infants can increase the internal dose or body burden in this age group (see sections 3.1 and 3.4).

Exposure to manganese from air is generally several orders of magnitude less than exposure from the diet, typically around 0.04 ng/day, on average (US EPA, 1990), although this can vary substantially depending on proximity to a manganese source.

Care should be taken when extrapolating estimated intakes from different sources to the relative uptake from each source, as factors such as bioavailability and manganese speciation play key roles in uptake and potential toxicity (Health Canada, 2019).

3 Toxicokinetics and metabolism in humans and animals

3.1 Absorption

Following ingestion, manganese is subject to homeostatic control through both regulation of its absorption from the gastrointestinal (GI) tract and its hepatobiliary excretion. Following inhalation exposure, manganese may bypass this homeostatic control and be transported to the brain via the olfactory system (Aschner, Erikson & Dorman, 2005; Roth, 2006). Therefore, absorption of manganese via inhalation differs significantly from absorption through ingestion of drinking-water. The metabolism of manganese, including its absorption, has been reviewed by Chen, Bornhorst & Aschner (2018).

Absorption of manganese from the GI tract is regulated by normal physiological processes. Absorption has been suggested to take place through both an active transport mechanism (Garcia-Aranda, Lifshitz & Wapnir, 1984) and passive diffusion (Bell, Keen & Lönnerdal, 1989). In humans, GI absorption of manganese appears to be influenced by sex, with higher levels of absorption reported in females than in males. This may be related to the lower iron status of women and their higher iron requirement (Finley, Johnson & Johnson, 1994).

A 7-week study in which seven adult male volunteers ingested high-fibre diets that naturally contained 12.0–17.7 mg of manganese per day (0.17–0.25 mg/kg body weight [bw] per day) found that an average of 7.6% ± 6.3% of the manganese was absorbed during weeks 5–7, with no measurable net retention of manganese (Schwartz, Apgar & Wein, 1986). Similarly, an average absorption of 8.4% ± 4.7% was observed in seven adults ingesting infant formula containing manganese (Sandström et al., 1986). Johnson, Lykken & Korynta (1991) studied the absorption of radiolabelled manganese from various plant foods in adult men and women, and reported absorption rates of 1.4–5.5%, which were significantly lower than the mean values of 7.8–10.2% from controls receiving Mn(II) chloride dissolved in water. A mean manganese absorption of 6.0–6.2% was observed from chard (Davidsson et al., 1991). Oral studies in animals generally yield similar absorption results (Pollack et al., 1965; Davis, Zech & Greger, 1993; Finley et al., 1997; Zheng, Kim & Zhao, 2000). EFSA noted that the absorption of manganese across the GI tract in adults is below 10% (EFSA, 2013).

Several factors can influence the degree to which manganese in foods is absorbed following ingestion. These include intake of dietary fibre, oxalic acids and phytic acids, which tend to decrease manganese absorption, in some cases substantially (Chen, Bornhorst & Aschner, 2018; Gibson, 1994; IOM, 2001; US EPA, 2002; Aschner, Erikson & Dorman, 2005; ATSDR, 2012). Iron and manganese are substrates of the same transport system for absorption (Davis,

Malecki & Greger, 1992), and thus manganese absorption is closely linked to iron absorption; iron-deficient diets lead to increased absorption of both iron and manganese (Thomson, Olatunbosun & Valverg, 1971; Sandström et al., 1986; Finley, 1999), independent of manganese body stores (Mena et al., 1969; Chandra & Shukla, 1976; Shukla, Chandra & Seth, 1976; Finley & Davis, 1999; Arnich et al., 2004). The absorption of manganese is also related inversely to the level of calcium in the diet (Schroeder, Balassa & Tipton, 1966; McDermott & Kies, 1987; Lutz, Schroff & Scharrer, 1993). Certain constituents of tea, such as tannins, can result in reduced manganese absorption (Freeland-Graves & Llanes, 1994).

Some studies have reported no difference in tissue manganese concentrations or bioavailability following equivalent exposures to manganese from dietary and drinking-water sources (without consideration of fasted state) (Ruoff 1995; Foster et al., 2015). Conversely, other reports and risk assessments (Ruoff, 1995; US EPA, 2002; Bouchard et al., 2011; Health Canada, 2019) suggest that absorption and bioavailability of manganese are greater from drinking-water (in a fasted state) than from food. However, reliable quantitative data comparing the bioavailability and absorption of different chemical forms of manganese from drinking-water were not found.

Manganese absorption from the GI tract may be higher in infants than in adults (Keen, Bell & Lönnerdal, 1986; Davidsson et al., 1989; Johnson, Lykken & Korynta, 1991; Finley, Johnson & Johnson, 1994; IOM, 2001; Health Canada, 2010), with up to 40% absorption reported (Neal & Guilarte, 2013; Dörner et al., 1989). This may be attributable to a compensatory mechanism related to greater metabolic needs of infants compared with adults (Santamaria, 2008). The increased absorption may place neonates and infants at greater risk of exposure to high levels of manganese than older children and adults (Neal & Guilarte, 2013).

Studies in rats have demonstrated that young animals absorb significantly more manganese from the gut than do mature animals (Lönnerdal et al., 1987). Experimental animal studies have also shown that manganese crosses the blood–brain barrier at a rate four times higher in neonates than in adults (Mena, 1974).

Some constituents of both infant formula and breast milk may affect manganese bioavailability. Breast milk substitutes made from soy protein contain high levels of phytic acids and vegetable proteins, which probably decrease manganese bioavailability (Keen, Bell & Lönnerdal, 1986). If the formula is also iron fortified, manganese bioavailability may be reduced (as indicated above), since the use of the same transport system to cross the gut mucosa results in competition between non-haem iron and manganese (Davis, Malecki & Greger, 1992). However, studies on the inhibitory influences of iron have produced conflicting results (Freeland-Graves, 1994).

Soluble forms of manganese, such as manganese chloride, have been reported to be more readily absorbed than the complex-associated trivalent oxidation state of manganese found in breast milk (Roels et al., 1997). This complex can bind to lactoferrin; lactoferrin receptors in the brush border membranes of epithelial cells throughout the length of the small intestine subsequently regulate its uptake from the GI tract. In contrast, infant formula contains manganese in the divalent oxidation state. This divalent state does not bind to lactoferrin, and therefore lactoferrin receptors cannot regulate intestinal uptake (Erikson et al., 2007; Health Canada, 2019).

Absorption of manganese from breast milk (8.2% ± 2.9%) consumed by adults has been reported to be higher than from cow's milk (2.4% ± 1.7%) or soy formula (0.7% ± 0.2%), as

measured using extrinsic labelling and whole-body retention measurements (Davidsson et al., 1989). The difference in absorption may be due to manganese speciation differences, as well as levels of lactoferrin (Davidsson et al., 1989; US EPA, 1997; Health Canada, 2019).

3.2 Distribution

Following GI tract absorption, manganese is distributed via the systemic circulation to all tissues. The highest levels are usually found in the liver, kidney, pancreas and adrenal glands (Tipton & Cook, 1963; Sumino et al., 1975; Aschner & Aschner, 2005; ATSDR, 2012). Manganese accumulates preferentially in certain regions of the brain in infants and young animals (Zlotkin & Buchanan, 1986; Kontur & Fechter, 1988; Chan et al., 1992; Lai et al., 1999). Within blood, manganese may be present in plasma (bound to albumin), red blood cells (bound to haemoblobin) and white blood cells. The chemical form and solubility of manganese can influence its distribution (Health Canada, 2019).

In both rats and mice, exposure of fetuses, neonates and pups resulting from maternal exposure is reported to be possible, given that manganese crosses the placental barrier and may be found in milk (Health Canada, 2019).

3.3 Metabolism

Mn(II) is the predominant form of manganese in biological systems; however, in many enzymes, manganese is present as Mn(III) (Utter, 1976; Leach & Lilburn, 1978; Aschner, Erikson & Dorman, 2005). This suggests that, over time, Mn(II) in plasma is oxidized to Mn(III) (ATSDR, 2012), although the mechanisms involved in this conversion are not fully elucidated (Roth, 2006). The valence state of manganese is reported to influence manganese retention and toxicity (Yokel, Lasley & Dorman, 2006; Health Canada, 2019).

3.4 Elimination

The main route of elimination of manganese from the body is faecal elimination via hepatobiliary excretion (ATSDR, 2012). Only a small proportion (0.1–2%) is eliminated in the urine (Davis & Greger, 1992; Park et al., 2003). Small amounts of manganese are also excreted in sweat, hair, nails, and the milk of lactating mothers (Roels et al., 1992; Merian et al., 2004; Health Canada, 2010).

Possibly because of the incomplete development of the biliary excretion system in human infants, which is the primary route of manganese elimination (Cotzias et al., 1976; Lönnerdal, 1994), infants retain higher levels of manganese than adults during the early neonatal period, with up to 20% retention reported in formula-fed infants (Aschner & Aschner, 2005). Dörner et al. (1989) reported high retention of manganese in infants ingesting both human milk and cow's milk formulas; absolute retention was highest in formula-fed infants. In addition, the manganese contents of erythrocytes in infants up to the age of 6 weeks are 7–9% higher than those in adults (Hatano et al., 1985). Collipp, Chen & Maitinsky (1983) reported manganese levels in hair that increased significantly from birth (0.19 µg/g) to 6 weeks of age (0.865 µg/g), and remained elevated at 4 months (0.685 µg/g) in infants given breast milk substitutes, whereas infants given breast milk exhibited no significant increase.

The reduced capacity of infants for biliary excretion compared with adults implies that neonates and young children will acquire a higher body burden of manganese from a given exposure. Along with the important neurodevelopmental processes occurring in neonates, this may render

them particularly susceptible to toxicity from exposure to manganese by exceeding the homeostatic concentration (Neal & Guilarte, 2013; Health Canada, 2019).

3.5 Physiologically based pharmacokinetic models

Physiologically based pharmacokinetic models have been developed for manganese in several species, including rats, monkeys and humans (reviewed by Health Canada, 2019). Models for monkeys and humans (Schroeter et al., 2011, 2012) allow estimation of manganese concentrations following exposure by multiple routes (ingestion, inhalation and injection) in numerous tissues (including, liver, lung, nasal cavity, bone, blood, olfactory bulb, cerebellum, globus pallidus and pituitary gland). The models also account for differences in manganese tissue-binding capacities, preferential fluxes of manganese in specific (brain) tissues and homeostatic control processes (i.e. reduced intestinal absorption and induced biliary secretion at elevated levels of exposure). The models could be useful for estimating manganese exposure levels that would cause an increase in tissue concentrations (Shroeter et al., 2011; Gentry et al., 2017). However, as the human model has not been validated against actual measurements in brain tissue, simulations for brain tissue using the model would need to be treated with caution (Health Canada, 2019).

A human model recently developed to predict brain manganese levels based on blood manganese levels from occupational epidemiological data showed consistency between model predictions and measurements (Ramoju et al., 2017). Further, Yoon et al. (2019) have updated a previously published model that includes drinking-water as an exposure source for manganese and predicts bioavailability of manganese from drinking-water in children. Based on model simulations, children did not appear to be at a greater risk from manganese in drinking-water than adults; however, more data and validation are needed.

4 Effects on humans

4.1 Essentiality

Manganese is an essential element for many living organisms, including humans. Some enzymes (e.g. manganese superoxide dismutase) structurally require manganese, and some (e.g. kinases, decarboxylases) are activated by manganese. These enzymes can play a role in several biological processes such as bone formation, free radical defence, neurotransmitter synthesis and ammonia clearance in the brain (Erikson & Aschner, 2019). Manganese plays a physiological role for a number of organ systems in the body, and is required for growth and development (including development of the nervous system and brain), especially in early life (Aschner & Aschner, 2005).

Adverse health effects can be caused by inadequate intake or overexposure. Manganese deficiency in humans appears to be rare because manganese is present in many common foods. A specific deficiency syndrome has not been clinically described in humans (IOM, 2001). In male subjects fed a conventional diet providing manganese at 2.59 mg/day for 3 weeks (baseline), followed by a purified diet containing manganese at 0.11 mg/day for 39 days, adverse effects were described. These included dermatitis and miliaria crystalline (prickly heat/heat rash) in five of the seven subjects at the end of the depletion period; the symptoms disappeared as repletion began (Friedman et al., 1987).

Requirements for manganese have not been established because of inadequate data (WHO, 1996; IOM, 2001; EFSA, 2013). Accordingly, some institutions have established adequate

intake levels based primarily on studies of reported intakes, such as in the USA (IOM, 2001) and the European Union (EFSA, 2013). The Food and Nutrition Board of the Institute of Medicine (IOM, 2001) has set adequate intake levels for manganese at 2.3 mg/day for men and 1.8 mg/day for women. Adequate intake levels for manganese for other age groups were set at 0.003 mg/day for infants from birth to 6 months, 0.6 mg/day for infants from 7 months to 1 year, 1.2 mg/day for children aged 1–3 years, 1.5–1.9 mg/day for children aged 4–13 years and 1.6–2.3 mg/day for adolescents (IOM, 2001). EFSA (2013) also applied an adequate intake approach, proposing 3 mg/day for all adults, including pregnant and lactating women. An adequate intake of 0.02–0.5 mg/day was proposed for infants aged 7–11 months, reflecting the wide range of intakes in this age group that appear adequate. Adequate intake levels for manganese for other age groups were established at 0.5 mg/day in children aged 1–3 years and 3.0 mg/day for adolescents.

The Institute of Medicine (IOM, 2001) set a tolerable upper intake level for manganese of 11 mg/day for adults, based on a review of manganese intake (0.7–10.9 mg/day) for adults eating diets typical of developed countries, and vegetarian diets (Greger, 1999; IOM, 2001). This was supported by evidence reported by Davis & Greger (1992) that women given daily supplements of 15 mg of manganese (as an amino acid–chelated manganese supplement) for 90 days experienced no adverse effects other than a significant increase in lymphocyte manganese-dependent superoxide dismutase, a biomarker that increases in direct relation to manganese exposure (Greger, 1998, 1999).

The Expert Group on Vitamins and Minerals (EVM) conducted an evaluation of data to establish a safe upper limit for manganese in the diet (EVM, 2003). Although it was concluded that no safe upper limit could be derived for manganese, an acceptable total dietary intake of 12.2 mg/day for the general population and 8.7 mg/day for older adults was thought appropriate. The EVM considered two large cohort studies in its evaluation (Kondakis et al., 1989; Vieregge et al., 1995), both of which assessed neurotoxicity as an end-point following drinking-water exposure to manganese. Of these two studies, the EVM considered that reported by Vieregge et al. (1995) to be the most robust. This assessed manganese burden in a cross-sectional study of adults (mean age 57 years; range 41–86 years) in rural Germany with 10–40 years exposure to drinking-water supplied from well water. Two groups homogeneous with regard to age, sex, nutritional habits and drug intake were established, based on manganese levels in well water: Group A was exposed to levels >0.3 mg/L (range 0.3–2.16 mg/L) and Group B to levels <0.05 mg/L. Neurological assessment of parkinsonian symptoms (Columbia University Rating Scale) was carried out by clinicians blinded to the exposure status. The authors reported no significant difference in neurological outcomes between the two groups.

4.2 Acute exposure

No studies to assess potential adverse effects following acute exposure to manganese in humans were identified.

4.3 Short-term exposure

Accidental ingestion of low doses of potassium permanganate (containing manganese at about 1.8 mg/kg bw/day) for 4 weeks in a 66-year-old man was associated with muscle weakness and neurological disturbances, including impaired mental capacity (Holzgraefe et al., 1986; Bleich et al., 1999). However, the quantitative and qualitative details of exposure necessary to establish manganese as the direct cause are lacking. Consumption of hydrated manganese sulfate (three tablespoons daily, total duration unknown) was associated with lethargy,

vomiting, abdominal pain, profuse diarrhoea, liver failure, acute renal injury, acute respiratory distress, myocardial dysfunction, shock with lactic acidosis and death within 72 hours in a 50-year old man undertaking a protein-free diet and consuming several herbal teas during a liver-cleansing diet (Sánchez et al., 2012).

4.4 Long-term exposure

4.4.1 Systemic effects

Data are lacking on systemic toxic effects in humans following ingestion of manganese. This may be due to the homeostatic mechanisms that strictly control levels of manganese absorbed following oral exposure and protect the body from the toxic effects of excess manganese. A possible association between manganese exposure and infant mortality was reported by Hafeman et al. (2007). In Bangladesh, infants (<1 year of age) exposed to manganese in water at levels ≥0.4 mg/L experienced elevated mortality during the first year of life compared with unexposed infants (odds ratio [OR] = 1.8; 95% confidence interval [CI] = 1.2 to 2.6]. The data were adjusted for water arsenic, indicators of social class and other variables without an appreciable impact on the results.

In a pilot study carried out in North Carolina, USA, Spangler & Spangler (2009) reported that, for every log increase in groundwater manganese concentration, there was an increase in the number of county-level infant deaths of 2.074 per 1000 live births, after adjustments were made for low birth weight, economic status, education and ethnicity.

The utility of these studies in the current assessment is limited because other confounding exposures, in addition to manganese exposure, could have been responsible for the deaths reported.

Organ-specific adverse effects are reported in the relevant sections below.

4.4.2 Neurologic effects

Evidence of adverse effects resulting from chronic exposure to high levels of manganese in humans is mainly derived from occupational inhalation exposures. The central nervous system (CNS) is the chief target of manganese toxicity. Neurotoxic effects resulting from exposure to manganese can be categorized as those affecting behavioural end-points (e.g. reflexes, motor activity, learning, memory, sensory ability), structural end-points (e.g. gliosis, neuroinflammation, neurostructural alterations) and neurochemical end-points (altered neurotransmitter systems) (Health Canada, 2019). The neurological impacts of inhaled manganese have been well documented in workplace studies of humans chronically exposed to elevated levels (Canavan, Cobb & Srinker, 1934; Cook, Fahn & Brait, 1974; Roels et al., 1999; ATSDR, 2012). The syndrome known as “manganism” is caused by inhalation exposure to very high levels of manganese dusts or fumes. It is characterized by weakness, anorexia, muscle pain, apathy, slow speech, a monotonous tone of voice, an emotionless “mask-like” facial expression and slow, clumsy movement of the limbs. These severe clinical effects that occur as the disease progresses are generally thought to be irreversible; however, reversibility of some early symptoms and clinical effects has been reported (ATSDR, 2012). Some motor functions may be affected following chronic exposure to levels of manganese of ≤1 mg/m^3 (if the inhaled manganese is respirable). For example, overt clinical symptoms of manganism have been reported following chronic exposure to manganese at concentrations of 0.73 mg/m^3 in

respirable dust (Roels et al., 1992; Mergler et al., 1994). Also, subclinical neurological effects have been described in workers exposed to air manganese concentrations in the range 0.07–0.97 mg/m^3. These effects include decreased performance in neurobehavioural tests; significantly poorer eye–hand coordination, hand steadiness and reaction time; poorer postural stability; and lower levels of cognitive flexibility (ATSDR, 2012).

By the oral exposure route, manganese is regarded as one of the least toxic essential elements. However, as a result of toxicokinetic differences between inhalation and oral intakes, there is some controversy about whether the neurological effects observed with inhalation exposure also occur following chronic oral exposure. Accidental ingestion of 125 mL of an 8% solution of potassium permanganate for 4 weeks was associated with impaired mental capacity and muscle weakness after several weeks. After 9 months, a Parkinson-like syndrome was noted (Holzgraefe et al., 1986).

A number of epidemiological studies have reported neurological effects in adult populations exposed to high environmental manganese concentrations (e.g. Kawamura et al., 1941 – in drinking-water at a concentration possibly up to 28 mg/L; Florence & Stauber, 1989 – in soil; Kondakis et al., 1989 – in drinking-water up to 2.3 mg/L; Iwami et al., 1994 – in food and water, with higher concentrations in food). However, no neurological effects were found in another epidemiological study of the adult population exposed to manganese in drinking-water at a level of up to 2.2 mg/L (Vieregge et al., 1995). Due to limitations in the exposure assessment methods and related uncertainty in the oral exposure concentrations in the study populations, the epidemiological data are insufficient to evaluate the causal relationship between manganese exposure and neurological effects.

As noted in section 3.4, infants and children are potentially a sensitive group with regard to exposure to high levels of manganese. Case studies report potential neurological effects and/or behavioural problems in children following oral exposure to high levels of manganese (Woolf et al., 2002; Sahni et al., 2007).

A large number of epidemiological studies have been carried out to assess potential adverse neurological outcomes (e.g. behavioural disinhibition; lower scores in tests of executive function, reading and digit agility) in children and infants following environmental exposure to elevated levels of manganese in drinking-water and/or food (e.g. He, Liu & Zhang, 1994; Zhang, Liu & He, 1995; Wasserman et al., 2006, 2011; Wright et al., 2006; Bouchard et al., 2007, 2011; Kim et al., 2009; Claus Henn et al., 2010, 2012; Farias et al., 2010; Riojas-Rodríguez et al., 2010; Khan et al., 2011, 2012; Menezes-Filho et al., 2011; Oulhote et al., 2014; Yu et al., 2014; Haynes et al., 2015; and reviews of studies by Bjørklund, Chartrand & Aaseth, 2017; Iyare, 2019; Kullar et al., 2019; Schullehner et al., 2020). Seven of these studies, which investigated the association between early-life manganese exposure (based on measured blood, hair or dentin manganese concentrations) and performance on tests of executive function, were reviewed by Leonhard et al. (2019), who reported that these associations were generally non-statistically significant but in the negative direction, although there were some positive (favourable) associations between dentin manganese and test performance. Although specific limitations are discussed below, some general limitations include the lack of establishment of causality due to cross-sectional design, potential limitations in exposure estimates from drinking-water and/or dietary intakes, and a need for enhanced validation of the biomarkers of exposure used. Therefore, none of these studies are sufficiently robust to be a key study on their own, because of limitations often related to the design of the epidemiological study or to the exposure assessment. However, together, they provide evidence to support

neurotoxicity as the key end-point in humans. Given that many of the earlier (pre-2011) epidemiology studies were reviewed by ATSDR (2012), only a limited number of early studies and those published after 2012 are discussed below.

Canadian children aged 6–13 years exposed to well drinking-water with high (0.61 mg/L) or low (0.16 mg/L) manganese concentrations were estimated to have daily manganese exposures of 0.02 mg/kg bw/day and 0.007 mg/kg bw/day, respectively. Manganese levels in hair were significantly higher in those exposed to high concentrations of manganese in drinking-water. In this pilot study, a statistically significant relationship was established between increased levels of oppositional behaviours (breaking rules, getting annoyed or angered, and hyperactivity) and increased levels of manganese in drinking-water (Bouchard et al., 2007). No manganese-related differences were observed for tests related to cognitive problems (disorganization, slow learning, lack of concentration). In a follow-up study, the authors assessed intellectual function in Canadian children aged 6–13 years in relation to manganese intake from water and food (estimated as 0–0.03 mg/kg bw/day and 0.01–0.44 mg/kg bw/day, respectively). Findings demonstrated associations between increased estimated manganese intakes from water and intellectual impairment in children, as reflected in full scale and performance IQ scores. Higher concentrations of manganese measured in hair were also associated with a lower full scale IQ score, and manganese levels in hair increased with increased consumption of manganese from drinking-water, but not from food (Bouchard et al., 2011).

An analysis of the Canadian school-aged cohort by Oulhote et al. (2014) described associations of exposure to manganese, determined from measurements in water and hair, with adverse effects on memory, attention, motor function, and parent- and teacher-reported hyperactive behaviours. The authors concluded that exposure to manganese in water was associated with poorer neurobehavioural performance in children, even at low levels (a steeper decrease in memory and motor function was reported at drinking-water concentrations of >100 µg/L and >180 µg/L, respectively). There was no significant association between manganese exposure and hyperactivity.

A follow-up assessment of this cohort at age 10.5–18 years (n = 287) has recently been reported, using the same methodology (Dion et al., 2018). Manganese concentrations in tap water ranged from 0.2 to 90 µg/L (geometric mean 14.4 µg/L), with 40% of the cohort being exposed to levels >50 µg/L. Higher levels of manganese in tap water were associated with lower performance IQ scores in girls and higher performance IQ in boys. The authors proposed that this finding may indicate a sex-related difference in manganese toxicity. In addition, a significant decrease in performance IQ scores was reported for children who had been exposed to higher concentrations of manganese between the earlier study and the follow-up assessment. However, this only related to a small number of households. Thus, the finding should be interpreted carefully. The hair manganese exposure biomarker was not significantly associated with IQ score in this follow-up study.

These studies considered several covariates (e.g. lead and arsenic in the drinking-water, socioeconomic status and maternal factors) that may confound the association between manganese and cognitive abilities. These studies also have limitations that need careful consideration when interpreting findings, including the following.

- The cross-sectional design of the studies does not allow causality to be established (Bouchard et al., 2007, 2011; Oulhote et al., 2014).

- The studies did not account for potential prenatal manganese exposure.
- Metabolic or genetic disorders that could alter manganese absorption and excretion were not considered.
- The number of participants was small – 46 children in the Bouchard et al. (2007) pilot study, 362 children in the Bouchard et al. (2011) baseline study and 287 children in the Dion et al. (2018) follow-up study.
- Selection bias cannot be discounted as few details were provided on the eligibility criteria of subjects and characteristics of those lost for follow-up.
- Although some important covariates were considered (e.g. lead, arsenic), there remains a possibility for unmeasured confounders.
- Potential confounding factors, including consumption of water from other sources and smoking in the household, were not evaluated in the Bouchard et al. (2007) pilot study but were evaluated in the Bouchard et al. (2011) and Dion et al. (2018) studies.

In an additional publication, Bouchard et al. (2018) assessed the IQ scores of 259 children aged 5.9–13.7 years from 189 households in New Brunswick, Canada, against additional indicators of manganese exposure from drinking-water: concentration in tap water; intake from the consumption of water divided by the child's weight; and manganese concentration in children's hair, toenail clippings and saliva. These biomarkers are considered by the authors to represent accumulation of manganese following long-term, low-level exposure (see also Ntihabose et al., 2018). Exposure levels from drinking-water were generally lower (geometric mean 5.96 µg/L; range <0.03–1046 µg/L) than those reported in the authors' previous studies with a different cohort (Bouchard et al., 2011). Exposure levels were <5 µg/L in 48% of children and >400 µg/L in 4% of children. There was no clear evidence of an association between exposure to manganese and cognitive development in the cohort, although the authors suggested possible sex-specific associations between measured manganese concentrations and performance IQ scores. In boys, performance IQ scores were higher with higher manganese concentrations, whereas, in girls, higher manganese concentrations were associated with poorer performance IQ scores. It should be noted, however, that significance of this observation was not established for all parameters measured.

A pooled and sex-stratified analysis of cross-sectional study data from two Canadian populations suggests that boys are less sensitive to manganese exposure–related decrements in performance IQ than girls; benchmark concentration levels (BMCLs) of 75, 153 and 386 µg/L corresponded to decrements in performance IQ of 1%, 2% and 5%, respectively, in boys, whereas BMCLs of only 9, 21 and 74 µg/L corresponded to similar decrements in performance IQ in girls. Limitations described above preclude the use of this work for quantitative risk analysis, but the study's findings nonetheless support neurotoxicity as a key end-point of concern following exposure to manganese in drinking-water (Kullar et al., 2019).

In a prospective cohort study, Rahman et al. (2017) evaluated the effects of exposure to manganese in drinking-water on cognitive and behavioural characteristics of schoolchildren in Bangladesh (n = 1265), from conception to 10 years of age. Exposure levels were in the range 0.001–6.6 mg/L (median 0.2 mg/L) during pregnancy and <0.001–8.7 mg/L (median 0.34 mg/L) at 10 years. As arsenic was also present in the drinking-water, the manganese statistical analysis was restricted to the children with low arsenic exposure. The authors reported that prenatal exposure to manganese (<3 mg/L) in drinking-water was positively

associated with cognitive function in girls, whereas boys appeared to be unaffected. In boys, early life exposure to manganese in drinking-water was associated with a decreased risk of emotional problems (OR = 0.39; 95% CI = 0.19 to 0.82). In girls, there was an association between prenatal exposures and low prosocial scores (OR = 1.48; 95% CI = 1.06 to 1.88).

Henn et al. (2017) reported on a prospective birth cohort study that assessed associations between prenatal manganese exposure and placental transfer, and neurodevelopment in 2-year old children (*n* = 224) living near a former mining area in rural Oklahoma, USA. Increased concentrations of manganese in maternal blood at or near the time of delivery were associated with lower neurodevelopment scores at 2 years of age. When adjusted for potential confounders, including arsenic and lead, the interquartile range for maternal blood manganese level was associated with a reduction in mental and psychomotor indices of –3.0 (95% CI = –5.3 to –0.7) and –2.3 (95% CI = –4.1 to –0.4) points, respectively. Cord manganese concentration was not associated with the neurodevelopment scores. The authors highlighted several limitations of the study, including the potential influence of timing of sample collection on manganese levels (given that little is known about how levels of manganese in maternal blood vary during labour and delivery), the small sample size and potential sampling bias due to loss at follow-up.

The potential joint action of manganese and lead on full scale and verbal IQs was assessed in a study of Korean children (average age 9.6 years). Participants were separated into two groups, based on blood manganese levels of <14 µg/L (*n* = 131) and >14 µg/L (*n* = 130); blood lead levels showed no difference between the two exposure groups. A significant inverse association was found between blood manganese and blood lead (combined group) and full scale and verbal IQ scores when the group was considered as a whole. Blood lead levels were shown to be a significant predictive variable for full scale and verbal IQ scores in the high manganese group, but not in the low manganese group. The authors concluded that the results are consistent with a joint toxic action of lead and manganese on full scale and verbal IQ scores (Kim et al., 2009).

A longitudinal study of 448 children born in Mexico investigated the neurotoxic effects of early-life exposure to manganese. Blood samples from children at ages 12 and 24 months were measured, and mental and psychomotor development was scored at 6-month intervals between 12 and 36 months. The study reported a possible biphasic dose–response relationship for manganese exposure and neurodevelopment, which would be consistent with the fact that manganese is both an essential element and toxic (Claus Henn et al., 2010). The same authors published a second study that evaluated manganese–lead interactions in the cohort and suggested a possible synergism between lead and excessive manganese in the impairment of mental and psychomotor skill development (Claus Henn et al., 2012).

In a study of school-aged children in Bangladesh, Khan et al. (2011) reported an association between increasing manganese concentration in drinking-water and negative behaviour in the classroom. The authors adjusted for arsenic exposure, sex, body mass index, maternal education and arm circumference as confounders. A follow-up study addressed a potential association between combined exposure to manganese and arsenic in drinking-water and academic achievement in school-aged children (*n* = 840). Exposure to drinking-water containing manganese levels >400 µg/L was significantly associated with decreased mathematics test scores after adjustment for confounders (arsenic exposure, school grade, maternal education, paternal education, head circumference, and within-teacher correlations in rating the children) (Khan et al., 2012). These findings should be interpreted with caution

because the possibility of co-exposure to other neurotoxic substances such as lead could not be eliminated, and total manganese exposures were not well characterized.

A meta-analysis that included articles published between January 2000 and March 2012 assessed the potential for an association between manganese, arsenic and cadmium exposure and neurodevelopment and behavioural disorders in children (Rodríguez-Barranco et al., 2013). Of the 17 articles relating to manganese exposure, 14 reported a significant negative effect on neurodevelopment and behavioural disorders. Of these, four studies used measurements of manganese in hair as a biomarker of exposure (Wright et al., 2006; Riojas-Rodríguez et al., 2010; Bouchard et al., 2011; Menezes-Filho et al., 2011). Rodríguez-Barranco et al. (2013) suggested that a 50% increase in manganese levels in hair was associated with a decrease of 0.7 points in the IQ (performance and verbal) of children aged 6–13 years. However, the meta-analysis was limited by the low number of subjects (n = 556).

A longitudinal mutlicentre cohort study in China reported an association between high prenatal exposure to manganese (based on umbilical cord serum concentrations) and lower scores in Neonatal Behavioural Neurological Assessments in mother–newborn pairs (n = 933) (adjusted ß = –1.1; 95% CI = –1.4 to 0.7; $p < 0.01$), after adjustment for confounders, including parents' age, education, incomes, occupation and smoking status. Other variables evaluated included neonate gestational age, sex, birth weight, and lead and mercury exposures (Yu et al., 2014). Limitations to the assessment included lack of long-term follow-up and no consideration of socioeconomic impacts on prenatal development.

Haynes et al. (2015) described a significant association between high blood (>11.2 µg/L) and high hair (>747 ng/g) manganese concentrations and lower full scale IQ scores in US children aged 7–9 years (n = 404), compared with control groups (blood: 8.2–11.2 µg/L; hair: 207–747 ng/g). The authors reported an inverted U-shaped association between the biomarkers of blood and hair manganese and cognition: both low and high blood and hair manganese concentrations were associated with lower full scale IQ and subscale IQ scores. Significant negative associations were observed between full scale IQ and the highest and middle two quartiles of blood manganese (β = –3.51; 95% CI = –6.64 to –0.38) and hair manganese (β = –3.66; 95% CI = –6.9 to –0.43). Confounders including creatinine, blood lead, community, sex, and parents' IQ and education were considered and adjusted for by the authors. However, a degree of bias may have been introduced to the analysis through exclusion of some participants as a result of missing data on one or more model covariates, as well as exclusion of participants with high manganese levels.

4.4.3 Reproductive and developmental effects

No studies to assess the potential reproductive toxicity of manganese following oral exposure in humans were identified.

A potential association between prenatal exposure to manganese and reduced birth weight was investigated in a number of studies (Zota et al., 2009; Yu, Cao & Yu, 2013; Chen et al., 2014; Eum et al., 2014; Guan et al., 2014). However, none of these studies established a statistical link. In addition, elevated maternal blood manganese was associated with depressed neurodevelopmental scores in children (Chung et al., 2015; Henn et al., 2017) and reduced intrinsic functional connectivity of emotional brain areas in children (de Water et al., 2017).

As discussed in section 4.4.2, there is some evidence of an adverse effect on neurodevelopment in infants and children exposed to elevated manganese levels, including through drinking-water (He, Liu & Zhang, 1994; Zhang, Liu & He, 1995; Wasserman et al., 2006, 2011; Bouchard et al., 2007, 2011; Kim et al., 2009; Claus Henn et al., 2010, 2012; Farias et al., 2010; Khan et al., 2011, 2012; Oulhote et al., 2014; Yu et al., 2014; Haynes et al., 2015; Henn et al., 2017; Rahman et al., 2017). Although, individually, these studies have limitations that prevent the establishment of causality, when evaluated collectively, the weight of evidence suggests an association between exposure to manganese and developmental neurotoxicity.

4.4.4 Immunological effects

No studies to assess potential adverse effects on the immune system following long-term exposure to manganese in humans were identified.

4.4.5 Genotoxicity and carcinogenicity

The genotoxic potential of manganese in humans has not been defined (IPCS, 1999; ATSDR, 2012). No monograph on manganese is available from the International Agency for Research on Cancer, and manganese is not listed in the United States National Toxicology Program's *14th report on carcinogens* (NTP, 2016).

5 Effects on animals and in vitro test systems

5.1 Essentiality

In animals experimentally maintained on manganese-deficient diets, effects include impaired growth, skeletal abnormalities, reproductive deficits, ataxia of the newborn, and defects in lipid and carbohydrate metabolism (Hurley & Keen, 1987).

5.2 Acute exposure

ATSDR (2012) noted that the acute lethality of manganese in animals appears to vary depending on the animal species and whether exposure is via gavage or dietary ingestion. The acute toxicity of manganese compounds is relatively low. The oral LD_{50} of manganese chloride in adult rats is reported to range between 331 and 642 mg/kg bw. Manganese acetate has an oral LD_{50} in rats of 1082 mg/kg bw, and manganese sulfate an oral LD_{50} of 782 mg/kg bw.

Following a single exposure of rats to aqueous manganese chloride (50 mg/kg) by gavage, neurological effects were reported. These included a significant and reversible decrease in total activity, delayed acquisition of an avoidance reaction in response to unconditioned and conditioned stimuli, an increased latent period of conditioned reflex activity, and a temporary worsening of the learning process (Shukakidze, Lazriev & Mitagvariya, 2003).

5.3 Short-term exposure

5.3.1 Systemic effects

A 14-day exposure of rats to a manganese dose of 1300 mg/kg bw/day (as manganese sulfate) in feed resulted in no deaths. Hepatic changes appeared to vary depending on the chemical species and whether exposure was via gavage or dietary ingestion. Reductions in liver weight

were reported in male rats but not in mice given manganese at 3900 mg/kg bw/day (as manganese sulfate) in feed for 14 days (NTP, 1993). Exposure of male rats to manganese at 271 mg/kg bw/day (as manganese chloride) in drinking-water for 2 or 4 weeks did not result in changes in liver weight, histology or function (Rivera-Mancía et al., 2009). However, in a 13-week study in which rats were fed manganese at up to 618 mg/kg bw/day (as manganese sulfate), liver weights were decreased in males (at ≥33 mg/kg bw/day) and females (at 618 mg/kg bw/day) (NTP, 1993). Similarly, male mice administered dietary manganese at concentrations of 1950 mg/kg bw/day (as manganese sulfate) for 13 weeks showed reduced relative and absolute liver weights, whereas similarly exposed female mice showed no hepatic effects (NTP, 1993).

Gastric irritation in the form of patchy necrosis of the stomach epithelium was observed in guinea-pigs administered manganese at 10 mg/kg bw/day via gavage for 30 days (Chandra & Imam, 1973); the method of administration might have contributed to the observed effects. Male mice fed high doses of manganese in food for 13 weeks showed mild hyperplasia and hyperkeratosis of the forestomach; no effects were seen in female mice, or male and female rats (NTP, 1993).

Decreased body weight gain was observed in rats and mice following oral exposure to manganese. In the 14-day NTP study, rats were administered dietary concentrations of 0–50 000 ppm Mn(II) sulfate monohydrate (equivalent to 25–370 mg/kg bw, according to the authors of the NTP report), and decreases in body weight gain of 57% in male rats and 20% in female rats were reported. Similar decreases of 50% were described by Ávila et al. (2008) in Wistar rats receiving manganese at 760 mg/kg bw/day (as manganese chloride) in drinking-water. No changes in eating habits in the lowest dose group were observed. Rats in the highest dose group showed decreased weight gain, which could in part be attributed to a decrease in feed consumption because the manganese presumably rendered it unpalatable. The authors noted signs of starvation in rats of this high-exposure group. No histopathological changes were reported in the exposed animals. The authors suggested that the decrease in weight gain might have been compounded by manganese interference in metabolism of calcium, phosphorus and iron.

5.3.2 Neurological effects

In infant monkeys exposed to manganese chloride in milk feed at a manganese level of 328 mg/kg bw/day for 4 months, there were no marked differences in gross motor maturation, growth, cerebrospinal fluid levels of dopamine or serotonin metabolites, or performance on tests of cognitive end-points in the exposed animals compared with controls. Decreased activity during sleep at 4 months of age and decreased play activity at 1–1.5 months of age were noted (Golub et al., 2005). The authors proposed that the behavioural effects were indicative of subtle neurobehavioural changes.

Neurobehavioural effects have also been observed in adult rats orally exposed to inorganic manganese for periods of 30 days to 22 weeks (Calabresi et al., 2001; Centonze et al., 2001; Shukakidze, Lazriev & Mitagvariya, 2003; Torrente, Colomina & Domingo, 2005; Vezér et al., 2005, 2007). The lowest daily dose of manganese reported to be associated with neurobehavioural effects in adult rats was 5.6 mg/kg bw/day (as manganese chloride in the diet for 30 days). The 5.6 mg/kg/day dose was identified as a lowest-observed-adverse-effect level (LOAEL), based on severely impaired cognitive performance in a maze test (Shukakidze, Lazriev & Mitagvariya, 2003). In adult mice exposed to 10 or 30 mg/kg bw/day (as manganese

chloride) via gavage for 8 weeks, no changes in open-field activity were reported (Moreno et al., 2009a). Conversely, in mice exposed during postnatal days (PNDs) 20–34, subsequent exposure to manganese in adulthood at 10 or 30 mg/kg bw/day for 8 weeks was associated with a decrease in open-field novelty-seeking behaviour and total overall movement in the open field in males, but not in females (Moreno et al., 2009a).

Other studies have also reported subtle neurobehavioural effects in animals following oral exposure to manganese at 8–20 mg/kg bw/day during neonatal periods (Kristensson et al. 1986; Pappas et al., 1997; Brenneman et al., 1999; Dorman et al., 2000; Tran et al., 2002a, b; Garcia et al., 2006; Reichel et al., 2006; Moreno et al., 2009a; Kern, Stanwood & Smith, 2010; Kern & Smith, 2011; Beaudin, Nisam & Smith, 2013). In general, evidence from these studies supports subtle neurobehavioural effects following short-term neonatal exposures at manganese doses of ≥10–20 mg/kg bw/day. Kern, Stanwood & Smith (2010) reported a comprehensive evaluation of the neurodevelopmental effects of manganese exposure in Sprague–Dawley rats exposed via the oral route to manganese at 25 or 50 mg/kg bw/day from birth to PND 21, corresponding to the period of development of dopaminergic pathways in regions of the brain that are important in the regulation of executive function behaviours (involving attention, learning and memory). Behavioural tests (open arena, elevated plus maze and 8-arm radial maze) were performed, and levels of dopamine receptor and transporter proteins were measured in the brain. At the higher tested dose (50 mg/kg bw/day), altered locomotor activity and behavioural disinhibition in the open area test on PND 23, altered learning and increased number of errors in the radial maze on PND 23, and impaired learning/memory (delay/failure to reach the learning criterion and increased number of learning errors in the 8-arm radial test) over PNDs 33–46 were observed. In addition, at the lower dose (25 mg/kg bw/day), increased stereotypic behaviour on a greater number of session days during the 8-arm radial maze test (shift in goal-oriented behaviour, indicating impaired spatial memory) and a reduced level of D1-like receptors in the dorsal striatum were reported. Manganese exposure (up to 50 mg /kg bw/day) did not affect fear and anxiety (as measured by elevated plus maze performance). A LOAEL of 25 mg/kg bw/day can be identified from this study.

In a follow-up study, the authors reported that, without exposure beyond PND 21, the observed neurochemical effects lasted into adulthood, with altered dopamine receptor levels and astrogliosis (as measured by glial fibrillary acidic protein) being observed. Behavioural changes were not observed in animals exposed as adults; however, enhanced locomotor response to a D-amphetamine challenge was seen in adults exposed during the neonatal period (Kern & Smith, 2011).

Histopathological changes in the rat brain following short-term neonatal oral exposure to manganese are not consistently reported. Although several in vivo exposure studies reported an association between increased manganese exposure in rats and histopathological changes in the rat brain (Chandra & Shukla, 1978; Pappas et al., 1997; Bikashvili, Shukakidze & Kiknadze, 2001; Shukakidze et al., 2002; Lazrishvili et al., 2009; Moreno et al., 2009b; Wang et al., 2012; Krishna et al., 2014), other in vivo studies have reported no evidence of such a histopathological association, despite changes in brain biochemistry (Kristensson et al., 1986; Dorman et al., 2000).

Oral doses ranging from 1 to 150 mg/kg bw/day produced neurological effects in rats and mice, mainly involving alterations in neurotransmitter and enzyme levels in the brain. These changes were sometimes accompanied by clinical signs, such as incoordination and changes

in activity level (ATSDR, 2012). Deskin, Bursian & Edens (1980) reported an increase in monoamine oxidase activity in the hypothalamus in rats intubated with a daily dose of manganese at 20 mg/kg bw/day from birth to 24 days of age. In rats administered manganese at 150 mg/kg bw/day (as manganese chloride), a rigid and unsteady gait was observed after 2–3 weeks, which was no longer apparent after 7 weeks of exposure (Kristensson et al., 1986).

More recent studies have continued investigations of brain chemistry alterations in animals following acute to intermediate-duration oral exposure to manganese (Desole et al., 1997; Lipe et al., 1999; Ranasinghe et al., 2000; Calabresi et al., 2001; Liu et al., 2006; Morello et al., 2007; Ávila et al., 2008; Moreno et al., 2009a). Neuropathology was reported following manganese exposure, as evidenced by neuronal damage and/or increased oxidative stress (Spadoni et al., 2000; Liu et al., 2006; Ávila et al., 2008). Behavioural assessments in rats have found changes in measures related to fear, locomotor activity and cognitive performance (Calabresi et al., 2001; Shukakidze, Lazriev & Mitagvariya, 2003; Torrente, Colomina & Domingo, 2005; Vezér et al., 2005, 2007). In some of these studies, electrophysiological changes in the brain were associated with behavioural changes (Calabresi et al., 2001; Vezér et al., 2005, 2007).

5.3.3 Immunological effects

Alterations in white blood cell counts were reported in rats and mice following oral exposure to manganese in a 13-week study (NTP, 1993). Male rats were administered manganese at 33–520 mg/kg bw/day and female rats 40–618 mg/kg bw/day. Increased neutrophil counts were seen in the males at levels of manganese ≥33 mg/kg bw/day. There was a decrease in lymphocyte count in males at ≥130 mg/kg bw/day, and in total leukocytes in females at ≥155 mg/kg bw/day (NTP, 1993). Komura & Sakamoto (1991) reported decreased white blood cell counts in mice following exposure to manganese at 284 mg/kg bw/day (as manganese acetate, manganese chloride or manganese dioxide) for 100 days. It is not known if any of these changes are associated with significant impairment of immune system function.

5.4 Long-term exposure

5.4.1 Systemic effects

Limited animal data are available on the effects on systemic target tissues of exposure to manganese by ingestion.

Chronic ingestion of manganese at 1–2 mg/kg bw/day produced changes in appetite and a reduction in haemoglobin synthesis in rabbits, pigs and cattle (Hurley & Keen, 1987). Two-year oral exposures to extremely high doses (1800–2250 mg/kg bw/day as Mn(II) sulfate) in male and female mice resulted in hyperplasia, erosion and inflammation of the forestomach. The authors concluded that this was due to direct contact irritation of the GI epithelium and was of minor consequence; no effects were seen in rats (NTP, 1993). When rats were fed manganese at up to 232 mg/kg bw/day (as manganese sulfate) and mice up to 731 mg/kg bw/day (as manganese sulfate) for 2 years, no significant hepatic histological changes were observed in either species (NTP, 1993).

In a 2-year study, male rats exposed to manganese at 200 mg/kg bw/day (as manganese sulfate in food) showed a significant fall in body weight (10% lower than controls); however, in

females, body weights were unaffected. This was unrelated to food intakes, which were similar for males and females in all groups (NTP, 1993).

5.4.2 Neurological effects

Neurotoxicity is a known effect of long-term exposure to inhaled manganese in humans and animals. However, the potential for neurotoxicity in animals resulting from chronic oral exposure is less well characterized.

A limited number of animal studies have observed manganism-type effects in animals similar to those seen in humans. Muscular weakness and lower limb rigidity were observed in four male rhesus monkeys given oral doses of manganese at 6.9 mg/kg bw/day (as manganese chloride) for 18 months. Degenerated neurons in the substantia nigra were observed at autopsy (Gupta, Murthy & Chandra, 1980). A staggered gait and histochemical changes were also reported in two third-generation mice (total number not stated) treated with manganese at 10.6 mg/kg bw/day (as manganese chloride) in drinking-water (Ishizuka, Nishida & Kawada, 1991). Fine sensorimotor function, learning and attention tasks were affected in adult male Long Evans rats orally exposed to manganese at ≥25 mg/kg bw/day during PNDs 1–21 or throughout life (beginning at PND 1) (Beaudin, Nisam & Smith, 2013; Beaudin et al., 2017). The presence and severity of effects were dependent on the dose and duration of exposure. Many studies report altered behaviours following developmental manganese exposure, including hyperactivity, altered social interactions, transient ataxia, altered acoustic startle, impaired learning and increased stereotypic behaviours (Kristensson et al. 1986; Dorman et al., 2000; Tran et al., 2002a, b; Golub et al., 2005; Moreno et al. 2009b; Kern, Stanwood & Smith, 2010; Kern & Smith, 2011).

Many of the animal studies address changes in brain chemical end-points following exposure to manganese, particularly during the early postnatal and juvenile periods. Alterations in the dopaminergic, noradrenergic, serotonergic or gabaergic systems; increased monoamine oxidase; and decreased iron levels have been reported (Chandra & Shukla, 1978; Deskin, Bursian & Edens, 1981; Kristensson et al., 1986; Dorman et al., 2000; Tran et al., 2002a, b; Reichel et al., 2006; Anderson, Cooney & Erikson, 2007; Anderson et al., 2009; Moreno et al., 2009a; Kern, Stanwood & Smith, 2010; Kern & Smith, 2011). Transient effects on biogenic amine levels, and activities of dopamine β-hydroxylase and monoamine oxidase in rat brain were noted with long-term exposures to manganese at oral exposure levels ranging from around 1 to >2000 mg/kg bw/day (as manganese chloride, manganese acetate, or Mn(II, III) oxide) (Lai, Leung & Lim, 1984; Eriksson, Lenngren & Heilbronn, 1987; Subhash & Padmashree, 1990; Desole et al., 1997; Ranasinghe et al., 2000; Calabresi et al., 2001). An increase in physical activity level and a transient increase in dopaminergic function were observed in rats given manganese at 40 mg/kg bw/day for 65 weeks (Nachtman, Tubben & Commissaris, 1986).

5.4.3 Reproductive and developmental effects

The results of several studies in rats and mice indicate that ingestion of manganese can delay reproductive maturation in male animals (ATSDR, 2012). Testosterone levels were reduced in male rats given an oral manganese dose of 13 mg/kg bw/day for 100–224 days (Laskey et al., 1982), and delayed growth of the testes was observed in young rats ingesting manganese at 140 mg/kg bw/day for 90 days (Gray & Laskey, 1980). These effects do not appear to have been severe enough to affect male reproductive function (ATSDR, 2012). Sperm abnormalities

were reported in several studies in mice following oral exposure to manganese (Joardar & Sharma, 1990; Elbetieha et al., 2001; Ponnapakkam, Sam & Izard, 2003; Ponnapakkam et al., 2003). Male reproductive performance was lowered at manganese levels as low as 23 mg/kg bw/day in mice exposed over a 21-day period (Joardar & Sharma, 1990).

The results of most studies indicate that oral exposure to manganese does not result in reproductive toxicity in female rodents (e.g. rats, mice) or rabbits (ATSDR, 2012), although increased post-implantation loss was observed in female rats in at least one study (Szakmáry et al., 1995).

Results from several developmental studies in rodents and rabbits are equivocal. Data from the majority of these studies indicate that manganese exposure during part or all of gestation results in increased manganese levels in the pups (Järvinen & Ahlström, 1975; Kontur & Fechter, 1988) but generally caused either no measurable effect (Grant, Blazak & Brown, 1997), transient effects such as weight decreases and hyperactivity (Pappas et al., 1997), or self-correcting effects on skeletal and organ development (Szakmáry et al., 1995).

Studies involving oral exposures to manganese in drinking-water or by gavage in neonatal pups reported changes in brain neurochemistry (ATSDR, 2012). The data from one recent study indicate that rodent pups administered manganese at 22 mg/kg bw/day in drinking-water from birth to weaning (21 days) had changes in brain neurochemistry and evoked sensory response (Dorman et al., 2000).

Although results are varied and inconsistent, taken together, the weight of evidence suggests that excess manganese exposure during development can lead to alterations in brain chemistry and behavioural development (ATSDR, 2012).

Several animal studies of the effects of manganese on reproductive development report developmental effects (Gray & Laskey 1980; Laskey et al., 1982, 1985). In pre-weanling mice exposed to manganese at 1050 mg/kg bw/day (as Mn(II, III) oxide) from PND 15 (to a maximum of 90 days), decreased growth of reproductive organs (preputial gland, seminal vesicle and testes) was reported. Laskey et al. (1982) showed a significant decrease in the number of pregnancies in rats following dietary manganese exposure at feed concentrations ranging from 0 to 3500 ppm during gestation, continuing during nursing and after weaning. No other adverse effects were noted. In a further study, Laskey et al. (1985) showed decreased serum testosterone levels in pre-weanling rats administered manganese at levels between 0 and 214 µg/kg bw/day (as Mn(II, III) oxide) from birth to 21 days of age.

5.4.4 Immunological effects

Alterations in white blood cell counts were reported in rats and mice following oral exposure to manganese. Rats fed manganese at up to 232 mg/kg bw/day (as manganese sulfate) and mice fed up to 731 mg/kg bw/day (as manganese sulfate) for 2 years showed no gross or histopathological changes, or organ weight changes in the lymph nodes, pancreas, thymus or spleen (NTP, 1993).

5.4.5 Genotoxicity and carcinogenicity

5.4.5.1 Genotoxicity

Results of genotoxicity testing are equivocal and do not allow for a clear understanding of the genotoxic potential of manganese. In vitro studies, including tests for mutagenicity, chromosomal aberrations, sister chromatid exchanges and cell transformations, have reported mutagenic or clastogenic potential associated with manganese; however, results vary depending on the form of manganese and test system used. Results of in vivo studies in mammals are inconsistent and do not allow for an overall conclusion about the genotoxic potential of manganese. This information has been summarized in detail in a number of published reviews (European Commission, 2000; Health Canada, 2010, 2019; Assem, Holmes & Levy, 2011).

In vitro bacterial gene mutation tests have yielded both positive and negative results, whereas in vitro tests with fungi and mammalian cells have been predominantly positive. Manganese chloride produced an increased frequency of mutations in *Salmonella* Typhimurium strain TA1537, but negative results in other strains, whereas manganese sulfate was reported to produce both positive and negative results in separate studies in *Salmonella* strain TA97, but negative results in other strains (ATSDR, 2012). Several positive results were obtained with various manganese compounds (including manganese sulfate and manganese chloride) in *Photobacterium fischeri* and *Escherichia coli*, as well as in *Saccharomyces cerevisiae*, mouse lymphoma cells and hamster embryo cells (NTP, 1993; ATSDR, 2012). It has been suggested that the absence of mutagenicity of manganese in some of the Ames assays could be due to lack of bioavailability of the metal ion, which may result from chelation of the metal ions by components of the culture media, or from competition for active transport sites (NTP, 1993).

Oberly, Piper & McDonald (1982) reported positive results for manganese chloride in the mouse lymphoma assay, without metabolic activation, at doses of 80, 60 and 40 µg/mL. Manganese chloride was also positive in the Comet assay (single cell gel assay) with cultured human lymphocytes (De Méo et al., 1991). Induction of cell transformations in Syrian hamster embryo cells has also been shown at a manganese chloride concentration of 0.13 mM (16.4 µg/mL) (Casto, Meyers & DiPaolo, 1979).

NTP (1993) reported that manganese sulfate (12 500 ppm, or 12 500 µg/mL assuming the density of the culture media is 1 g/mL) induced sister chromatid exchanges without metabolic activation in mouse fibroblasts (Andersen, 1983), Chinese hamster ovary (CHO) cells (Galloway et al., 1987) and human lymphocytes (Andersen, 1983). With metabolic activation, manganese sulfate was also positive for sister chromatid exchanges in CHO cells (NTP, 1993). Potassium permanganate did not induce chromosomal aberrations in Syrian hamster embryo cells when tested without metabolic activation (Tsuda & Kato, 1977).

In vivo tests in *Drosophila melanogaster* did not report an association between exposure to manganese sulfate or manganese chloride and induction of sex-linked recessive lethal mutations or somatic mutations, respectively (Rasmuson, 1985; Valencia et al., 1985; NTP, 1993). No heritable translocations in mice were detected following administration of manganese sulfate in the diet for 7 weeks, and no dominant lethal mutations in rats were found following administration of manganese sulfate by gavage once a day for 1–5 days (Newell, Jorgenson & Simmon, 1974, as cited in NTP, 1993).

Administration of manganese sulfate and potassium permanganate increased the frequency of sperm head abnormalities, chromosomal aberrations and micronuclei in rat bone marrow (ATSDR, 2012). In Swiss albino mice exposed to manganese sulfate by the oral route at manganese doses of 33–132 mg/kg bw/day for 3 weeks, there was also an increase in the frequency of sperm head abnormalities, chromosomal aberrations and micronuclei in bone marrow cells (Joardar & Sharma, 1990). Similar findings were reported for oral exposure to potassium permanganate at manganese doses of 22.6, 45.2 and 132.1 mg/kg bw/day for 3 weeks, with an increase in the frequency of sperm head abnormalities and chromosomal aberrations in bone marrow cells (Joardar & Sharma, 1990). Significant chromosomal damage did not occur in bone marrow or in spermatogonial cells of male rats orally exposed to manganese at 0.014 mg/kg bw/day (as manganese chloride) for 180 days (Dikshith & Chandra, 1978).

5.4.5.2 Carcinogenicity

Clear evidence for the carcinogenicity of manganese from an oral route of exposure has not been established. A 2-year oral study of manganese sulfate in rats and mice produced equivocal evidence of carcinogenicity (NTP, 1993). In rats fed manganese sulfate (manganese at 30–331 mg/kg bw/day in males and 26–270 mg/kg bw/day in females), no treatment-related increases in tumour incidence were reported. In mice fed manganese sulfate (manganese at 63–722 mg/kg bw/day in males and 77–905 mg/kg bw/day in females), the incidence of follicular cell adenoma of the thyroid was increased slightly in high-dose animals compared with controls. These increases were not statistically significant, and the tumours were observed at the end of the study only. As well, follicular cell adenoma of the thyroid appears with low frequency in historical control male mice of this strain. Thus, the significance of these results and their relevance to normal human exposure to manganese are questionable.

5.6 Mode of action

Although there is clear evidence that the primary target of manganese toxicity is the CNS – where it impairs cellular transport systems, enzyme activities and receptor functions – the principal mode of action of manganese neurotoxicity has not been clearly established (Aschner & Aschner, 1991; Aschner et al., 2007). Occupational studies reporting severe neurotoxic effects have focused research into potential modes of action on areas of the brain concerned with movement, principally the organs of the basal ganglia, the globus pallidus, the putamen and caudate nucleus, the substantia nigra and the dopaminergic system (WRC, 2014). Many studies investigating effects of manganese on these areas of the brain have been published, but interpretation is difficult because of differences in the experimental methodologies used.

Manganese is selectively taken up by the globus pallidus and the substantia nigra, accumulating in neurons, astrocytes and oligodendrocytes. This is mediated by transferrin receptors. Once inside the cell, manganese is transported through a calcium one-way transporter into mitochondria, where it accumulates. It is hypothesized that accumulation of manganese results in several interrelated processes, ultimately leading to neurotoxicity. These processes include free radical formation (Desole et al., 1994, 1995; Hussain et al., 1997; Taylor et al., 2006), neurotransmitter impairment (Chandra, Srivastava & Shukla, 1979; Deskin, Bursian & Edens, 1980; Chandra & Shukla, 1981; Lai et al., 1982; Lai, Leung & Lim, 1984; Subhash & Padmashree, 1991; Komura & Sakamoto, 1994; Ranasinghe et al., 2000; Calabresi et al., 2001; Montes et al., 2001; Tran et al., 2002a, b; Fitsanakis et al., 2006; McDougall et al., 2008; Peneder et al., 2011) and mitochondrial dysfunction (Gavin, Gunter & Gunter, 1992; Zheng, Ren &

Graziano, 1998). The generation of free radicals can disrupt the processes of oxidative phosphorylation and ATP synthesis, and lead to cellular dysfunction, apoptosis/necrosis and cell death.

Elevated intracellular manganese levels are linked with the pharmacologic disruption of iron regulation, a process that appears to play a role in neurotoxicity (Kwik-Uribe et al., 2003; Kwik-Uribe & Smith, 2006; Reaney, Bench & Smith, 2006; Crooks et al., 2007). A further consequence of elevated intracellular manganese levels is disruption of the regulation and interaction of neurotransmitters, including dopamine, glutamate and gamma-aminobutyric acid (GABA) in the basal ganglia (Chandra, Srivastava & Shukla, 1979; Deskin, Bursian & Edens, 1980; Chandra & Shukla, 1981; Lai et al., 1982; Lai, Leung & Lim, 1984; Subhash & Padmashree, 1991; Komura & Sakamoto, 1994; Ranasinghe et al., 2000; Calabresi et al., 2001; Montes et al., 2001; Tran et al., 2002a, b; Fitsanakis et al., 2006; McDougall et al., 2008; Burton & Guilarte, 2009; Peneder et al., 2011).

Dopamine plays a role in regulating cognition, behaviour, locomotor activity and neuroendocrine secretion (Fitsanakis et al., 2006; Farina et al., 2013; Guilarte, 2013). In addition, executive function behaviours (e.g. memory, learning, attention) are regulated by dopaminergic pathways (Kern, Stanwood & Smith, 2010). Neurological deficits in animal studies were reported to be accompanied by altered dopamine transporter and dopamine receptor levels, in addition to altered response to dopamine antagonists. Given that dopamine transporter levels are known to increase throughout development, it is possible that cognitive and neurobehavioural effects reported in children following manganese exposure are related to its effects on the dopaminergic system during development (Neal & Guilarte, 2013).

Glutamate is the most prevalent excitatory neurotransmitter in the brain and appears to play a role in various CNS functions, including cognition, learning and memory, as well as in CNS development (Fitsanakis et al., 2006). Mechanistic studies demonstrate that elevated levels of manganese in astrocytes can impair the glycine/glutamate–GABA cycle, which is essential for optimal CNS function because it produces excitatory (glutamate) and inhibitory (GABA) neurotransmitters (Erikson & Aschner, 2003; Aschner et al., 2009; Sidoryk-Wegrzynowicz et al., 2009; Farina et al., 2013; Karki, Lee & Aschner, 2013; Sidoryk-Wegrzynowicz & Aschner, 2013a, b).

6 Overall database and quality of evidence

6.1 Summary of health effects

Manganese is an essential element, and trace levels are necessary for human health. The acute toxicity of manganese compounds may vary depending on route of administration; however, in general, inorganic manganese compounds have low acute oral toxicity.

Manganese is able to cross the blood–brain barrier through capillary endothelial cells (ATSDR, 2012), and the weight of evidence from animal and human studies suggests that the CNS is the primary concern for manganese toxicity in mammals, with effects reported at low doses. Exposure to high levels of manganese is associated primarily with neurological and cognitive effects, including reduced intellectual function, hyperactive behaviours and neurodevelopmental effects. A number of epidemiological studies have reported neurological effects in adult populations exposed to high levels in drinking-water, as well as in children following ingestion of manganese-contaminated water (He, Liu & Zhang, 1994; Zhang, Liu &

He, 1995; Wasserman et al., 2006, 2011; Kim et al., 2009; Claus Henn et al., 2010, 2011; Farias et al., 2010; Bouchard et al., 2011; Khan et al., 2011, 2012; Oulhote et al., 2014; Yu et al., 2014; Haynes et al., 2015; Henn et al., 2017; Rahman et al., 2017; Iyare, 2019). Another study (described in section 4.4.2) did not find any association (Vieregge et al., 1995).

However, the quality of the epidemiological studies is variable, particularly with respect to the reliability of the exposure estimates. No single study shows a clear causal relationship between manganese dose and neurotoxicity. Although limitations in these studies prevent their use in quantitative risk assessment, collectively they provide qualitative support that the critical effect in animal studies – neurotoxicity – is also relevant in humans.

Animal studies identified neurotoxicity as an end-point of concern following oral exposure to manganese. Some of these studies assessed neurodevelopmental end-points in early life that were supported by corresponding neurochemical findings (Kern, Stanwood & Smith, 2010; Kern & Smith, 2011; Beaudin, Nisam & Smith, 2013).

Infants and children are considered to have a greater sensitivity to manganese toxicity than adults. Infants are particularly vulnerable because of greater GI absorption and immaturity of their homeostatic control of bile excretion, meaning that they excrete less manganese (Valcke et al., 2018).

Existing studies and reports do not provide adequate evidence to assess potential carcinogenicity from oral exposure to manganese in humans. Equivocal evidence of the carcinogenicity of manganese sulfate in a 2-year oral toxicity study in rats and mice was reported (NTP, 1993). Further, no manganese compounds have been reviewed by the International Agency for Research on Cancer with respect to their carcinogenic potential or are included in the National Toxicology Program's report on substances that are known, or may be reasonably anticipated, to cause cancer in humans (NTP, 2016).

6.2 Adequacy of the database

Cross-sectional and prospective cohort epidemiology studies have investigated the potential adverse neurological effects in humans following chronic exposure to manganese though drinking-water. However, the ability to quantify the findings is limited by numerous uncertainties, particularly with regard to assessing manganese exposure levels. Longitudinal epidemiology studies with robust exposure measurements and valid established or novel biomarkers of effect would inform and refine the dose–response relationship for the spectrum of end-points observed.

Other data gaps in humans include the limited information on reproductive or immunological effects following oral exposures, effects of chronic exposure, and information on the mode of action associated with neurological effects.

Laboratory animal studies report subtle neurobehavioural effects following manganese exposure during the neonatal period (Kristensson et al., 1986; Pappas et al., 1997; Brenneman et al., 1999; Dorman et al., 2000; Tran et al., 2002a, b; Reichel et al., 2006; Moreno et al., 2009a; Kern, Stanwood & Smith, 2010; Kern & Smith, 2011; Beaudin, Nisam & Smith, 2013; Beaudin et al., 2017). Although a number of LOAELs have been identified in rodents, the suitability of rodent models to assess potential neurotoxicity in humans has been debated, because of differences in the neurological effects seen in humans and rodents. The human

tremor and gait disorders that are preceded by psychological symptoms, including irritability and emotional lability, are not seen in rodents. Although there may be differences in species' nutritional requirements for dietary manganese (US EPA, 2004), only an adequate intake level and tolerable upper intake level for manganese in humans have been reported to date (IOM, 2001), and a level representing essentiality has not been established. Effects seen in children following exposure to manganese involve the dopaminergic system, and mechanistic data indicate that there are commonalities between rodents and non-human primates with respect to the involvement of this system in manganese-induced neurotoxicity (Neal & Guilarte, 2013).

Results from the most robust animal dose–response studies that assessed and quantified neurological effects are consistent with the epidemiological studies. They identified a neurodevelopmental LOAEL for manganese of 25 mg/kg bw/day in rats following oral exposure in early life (Kern, Stanwood & Smith, 2010; Kern & Smith, 2011; Beaudin, Nisam & Smith, 2013; Beaudin et al., 2017). These studies characterized executive function parameters that reflect effects reported in epidemiological studies, such as behavioural hyperactivity (as measured using the open arena assessment) and learning deficits (measured using the 8-arm radial maze) following early-life exposures. They demonstrated that the behavioural and sensorimotor effects observed are accompanied by corresponding neurostructural and neurochemical changes. Long-term follow-up demonstrated the ability of manganese exposure in early life to result in effects that persist into adulthood, after levels in the brain have returned to normal (Beaudin, Nisam & Smith, 2013).

7 Practical considerations

7.1 Monitoring

As part of the hazard assessment phase of water safety planning, water sources should be assessed to determine if manganese is present. In general, manganese concentrations are stable between seasons in groundwater but may vary between wells in close proximity to each other. Manganese concentrations in lakes and reservoirs where there is sufficient depth for the development of thermoclines and layers of low oxygen can vary substantially seasonally, and more frequent and targeted monitoring may be needed. (Health Canada, 2019). Management of these source waters is important, where possible; otherwise water should be treated to remove manganese.

Where manganese is present at concentrations close to the guideline value (GV) or the water is treated to remove manganese, routine monitoring should be conducted post-treatment. In many small rural supplies, if resources are limited, monitoring may be minimal. Whenever possible, sampling should be designed to determine whether manganese is at concentrations in excess of the GV. If manganese deposits or precipitation of insoluble manganese result in lack of acceptability of drinking-water because of its organoleptic properties, this indicates that treatment for manganese removal is not optimized or that the distribution system is not appropriately managed.

7.2 Analytical methods and achievability

Total manganese (dissolved and particulate fractions) should be monitored. Quantifying the individual fractions is also important for determining the appropriate manganese treatment method and for monitoring treatment performance. In general, membrane filters with pore diameters between 0.22 µm and 0.45 µm are recommended for fractionating dissolved and particulate manganese (Carlson, Knocke & Gertig, 1997; Kohl & Medlar, 2006; Brandhuber

et al., 2013). Guidance is available on filtration and preservation procedures for measuring dissolved or particulate metal concentrations (APHA, AWWA & WEF, 2012). Water systems that are experiencing difficulties controlling manganese in treated water, and that are directly oxidizing manganese using potassium permanganate, chlorine dioxide or ozone, may also consider quantifying the colloidal fraction of selected samples within the treatment plant.

Sensitive methods are available for measuring manganese in biological and environmental samples. Colorimetric methods are suited to monitoring source waters and water within treatment plants to assess treatment effectiveness; they have detection limits of 10–70 µg/L (ISO, 1986; Brandhuber et al., 2013). The United States Environmental Protection Agency has four recommended analytical methods for analysing total manganese in drinking-water: Method 200.5 revision 4.2, Method 200.7. revision 4.4, Method 200.8 revision 5.4 and Method 200.9 revision 2.2 (US EPA, 2014). These use inductively coupled plasma atomic emission spectrometry (ICP-AES), inductively coupled plasma mass spectrometry (ICP-MS) and graphite furnace atomic absorption (GFAA) spectrometry, and have detection limits of 0.005–50 µg/L (ATSDR, 2012). In addition, one standardized analytical method is available (SM3125), which uses ICP-MS and has a detection limit of 0.002 µg/L (APHA, AWWA & WEF, 1992, 1995, 1998, 2005, 2012). Atomic absorption spectroscopy is also used for determining manganese concentrations in biological samples (e.g. urine, faeces, hair) at a detection limit as low as 1 µg/L for urine and 0.2 µg/g for hair (ATSDR, 2012). None of the methods described above distinguish between the different oxidation states of manganese (ATSDR, 2012).

7.3 Source control

Manganese contamination of drinking-water sources is generally due to natural occurrence in the underlying rocks and soil; as a result, source control may be limited. Options for controlling levels in groundwater include drilling a new well or blending water from different wells, where possible. For lake and reservoir sources, management of the sources to prevent release of manganese from sediment, particularly when there is a thermocline and the lower water levels become anoxic, is important. Aeration and variable depth intakes are control options for lowering manganese levels in water entering the treatment plant. Hypolimnetic aeration and oxygenation can be used to add dissolved oxygen to reservoirs to minimize manganese release from sediments while maintaining stratification (Gantzer, Bryant & Little, 2009; Bryant et al., 2011; Munger et al., 2016). Variable depth intake is an option for treatment plants that have deep reservoirs and a multilevel intake system. These systems can select the level in the reservoir from which water is drawn into a plant, based on the water quality at different depths (Brandhuber et al., 2013).

7.4 Treatment methods and performance

Manganese concentrations in drinking-water are easily lowered to less than 0.05 mg/L using common treatment methods, including oxidation/filtration, adsorption/oxidation, softening/ion exchange, and biological filtration. In well-operated and optimized systems, manganese concentrations can be reduced to less than 0.02 mg/L (Kohl & Medlar, 2006; Tobiason et al., 2008, 2016; Knocke et al., 2010; Brandhuber et al., 2013). Selection of the appropriate treatment system for manganese removal depends on the form of manganese (dissolved or particulate) present in the source water. Dissolved Mn(II) is most often the predominant form present in anoxic and acidic groundwater or lakes. However, depending on the pH and the dissolved oxygen content of the water, a combination of dissolved and particulate manganese can be present. In general, treatment methods used for manganese rely on a combination of

processes (e.g. oxidation, adsorption, filtration) to remove both the dissolved and particulate forms (Health Canada, 2019).

A commonly used technology for decreasing manganese concentrations in drinking-water is based on directly oxidizing dissolved Mn(II) to form MnO_x(s) particulates, which are then physically removed – for example, by clarification and granular media filtration or low-pressure membrane filtration. The chemical oxidants typically used include permanganate (MnO_4^-), chlorine dioxide (ClO_2) and ozone. Under high pH conditions, chlorine and oxygen may also be effective (Knocke, Hoehn & Sinsabaugh, 1987; Knocke et al., 1990). Effective oxidation of manganese depends on several factors, including pH and Eh, temperature, reaction time, alkalinity, and the total oxidant demand in the water (e.g. presence of iron, sulfide, nitrate, ammonia and organic compounds) (Casale, LeChevallier & Pontius, 2002; Brandhuber et al., 2013). The use of oxidation for the removal of manganese may form disinfection by-products, which should be considered when selecting and optimizing treatment processes. In addition, treatment plants using ozone should be aware that, depending on the water quality, ozone can oxidize Mn(II) into soluble MnO_4^-, and effective removal will not occur (Gregory & Carlson, 2001; Reisz et al., 2008). The effectiveness of physical removal processes depends on manganese entering the filter being in the particulate form (Tobiason et al., 2008). These processes typically remove 80–99% of manganese and, depending on the oxidant, can easily achieve treated water concentrations below 0.04 mg/L (Health Canada, 2019).

Another treatment technique for manganese removal is the use of MnO_x(s)-coated filter media that adsorb dissolved Mn(II) and catalyse oxidation at the surface in the presence of free chlorine. These coatings develop on anthracite coal or silica sand filter media as a result of the presence of dissolved Mn(II) and free chlorine across the filter bed (Knocke, Hamon & Thompson, 1988; Knocke, Occiano & Hungate, 1990; Tobiason et al., 2008; Islam et al., 2010; Knocke et al., 2010; Bazilio et al., 2016). The adsorbed Mn(II) is subsequently oxidized by the presence of free chlorine across the filter to create new MnO_x(s) adsorption sites (i.e. continuously regenerated). Only partial removal of the MnO_x(s) coating occurs during backwashing, resulting in a net increase in MnO_x(s) adsorption sites over the time of operation (Hargette & Knocke, 2001). In many treatment plants, the MnO_x(s)-coated media process initiates and sustains itself without operators being aware that it is occurring (Kohl & Medlar, 2006; Brandhuber et al., 2013). This process can routinely achieve very low treated water manganese concentrations (<0.015 mg/L), even when pre-filter manganese concentrations are as high as 0.5 mg/L. The location of this process within a treatment plant can vary. For surface water treatment plants that chlorinate before filtration, it is often part of the existing particle removal filtration process. When pre-filter chlorination is not practised, an adsorptive contactor unit can be placed following filtration (Knocke et al., 2010; Brandhuber et al., 2013; Tobiason et al., 2016).

Traditional manganese greensand is another adsorption/oxidation process using a granular filter media processed from glauconite sand. Glauconite is synthetically coated with a thin layer of manganese base material (manganous ions), which is then converted to a MnOx(s) coating by conditioning the greensand in a $KMnO_4$ or chlorine solution (Knocke, Occiano & Hungate, 1990; Sommerfield, 1999). This medium has a large adsorptive capacity for removing dissolved Mn(II) (1.5 kg/m^3). Greensand is typically smaller (effective size 0.30–0.35 mm) than silica sand, so it is good at capturing small particles. Since the head loss generated is higher than an equivalent bed depth of silica sand, most applications of greensand use pressure filtration (Brandhuber et al., 2013). Kohl & Medlar (2006) reported that groundwater treatment

plants using manganese greensand filtration achieved manganese removals of 86–100%: from average influent concentrations of 0.35–0.52 mg/L to average treated water concentrations of below 0.020 mg/L. Greensand filters are best applied in groundwater systems with iron and manganese concentrations <5 mg/L (Kohl & Medlar, 2006).

Biofiltration can successfully remove manganese from groundwater (Mouchet, 1992; Li et al., 2005; Burger et al., 2008; Kohl & Dixon, 2012) and to a lesser extent from surface water (Kohl & Dixon, 2012; Granger, Stoddart & Gagnon, 2014; Hoyland et al., 2014). Removal of manganese using biofiltration relies on the ability of naturally occurring manganese-oxidizing bacteria present in biofilms on filter media to adsorb and oxidize dissolved Mn(II) and form particulate Mn(IV), which can then be removed by backwashing. Kohl & Dixon (2012) reported data from eight treatment plants in Canada, Europe and China that used downflow mono-medium sand biofilters. These treatment plants were capable of >93% removal of manganese to achieve treated water concentrations below the method detection limit of 0.03 mg/L. An important consideration for utilities considering a transition from $MnO_x(s)$-coated media filtration to biofiltration is the potential for release of previously accumulated manganese on the filter media once the free chlorine residual across the filters is terminated (Gabelich et al., 2006; Kohl & Dixon, 2012).

Treatment plants that use lime or soda ash softening can also remove manganese by raising the pH of the water (e.g. >9.5–10) above the solubility of various manganese hydroxide and carbonate solid phases. The elevated pH in lime or lime–soda ash softening will also greatly increase the rate at which dissolved Mn(II) is oxidized in the presence of dissolved oxygen. Where dissolved oxygen is present, oxidized $MnO_x(s)$ solids will be formed. Raising the pH of the source water to remove dissolved Mn(II) is not a cost-effective treatment approach by itself; rather, this treatment method is typically used only if softening of the source water is also required. A lime softening treatment plant reported a reduction in the average manganese concentration from 0.520 mg/L in the source water to an average of 0.001 mg/L in the treated water (Kohl & Medlar, 2006). Dissolved Mn(II) can also be removed through cation exchange in zeolite softening processes. As with other cation exchange processes, backwashing the zeolite, typically with a brine solution, removes the manganese (as well as iron, calcium and magnesium) accumulated on the resin.

In addition to manganese in source water, chemical addition and treatment plant processes can contribute to the total amount of manganese that must be managed in drinking-water systems. The three main sources of manganese from treatment plant operations are (Tobiason et al., 2008):

- the presence of manganese impurities in coagulants (principally ferric-based coagulants);
- resolubilization of Mn(II) from the reduction of $MnO_x(s)$ solids stored in sedimentation basins as a result of anoxic conditions in the basin; and
- the presence of dissolved manganese in recycle streams from solid-processing operations.

Where a community water supply is not available, manganese removal on a small scale or at the household level is an option. Ion exchange (i.e. water softener) and greensand filtration can be used at the point of entry to a home to reduce the likelihood of discoloured water, and staining of laundry and fixtures. However, deficient operation or maintenance of greensand filters and softeners has been associated with increased manganese concentrations in homes treating well water (Barbeau, Carriere & Bouchard, 2011). To remove manganese for drinking-

water at a specific tap in a home, point-of-use units based on reverse osmosis are the most effective and reliable treatment technology. Point-of-use units using cation exchange media, such as pour-through filters, are also moderately effective in reducing manganese concentrations (Health Canada, 2019).

7.5 Distribution system

Low levels of manganese in source or treated water (current or historical) can accumulate in the distribution system and periodically release manganese to result in high levels at the tap. Notably, Brandhuber et al. (2015) estimated manganese stored on distribution system pipes based on data collected in Friedman et al. (2010). The mass of deposited manganese ranged from 0.1 mg/ft^2 to 10 000 mg/ft^2, with an estimated median of 210 mg/ft^2, equivalent to approximately 3.8 lbs manganese/mile (based on a 6-inch-diameter pipe) or 7.7 lbs manganese/mile (based on a 12-inch-diameter pipe). Brandhuber et al. (2015) noted that only 1.5% of the deposit would need to be released to exceed a concentration of 1 mg/L in water.

Releases of manganese can occur periodically due to physical or hydraulic disturbances to the system (e.g. mains breaks or hydrant flushing) or changes in water chemistry (e.g. changes in pH, temperature, chlorine residual, and source water type/blending). Physical and hydraulic disturbances most often release particulate manganese and can cause discoloured water and consumer complaints. Chemical releases can go unnoticed if manganese occurs predominantly in the dissolved form. Both types of releases can result in manganese exposure from drinking-water at the tap. Other contaminants (e.g. arsenic, barium, chromium, lead, uranium) that deposit with manganese oxides in the distribution system may also be released into the water and reach consumers' taps (Schock, 2005; Friedman et al., 2016).

Another detrimental influence of manganese in distribution systems is its impact on the stability of lead scales in lead pipes, lead service lines, lead solders and lead-containing fixtures, which can increase the risk of lead release (Del Toral, Porter & Schock, 2013; Schock et al., 2014). The presence of manganese in distribution systems can also interfere with the effectiveness of corrosion control chemicals (Wasserstrom et al., 2017; Trueman et al., 2019).

It is therefore appropriate to implement a range of controls within the distribution system to minimize the likelihood of manganese release events. These typically involve maintaining stable water chemistry and minimizing several factors: the manganese levels entering the distribution system, the amount of manganese oxide deposits in the distribution system (through best practices for water mains cleaning), and physical or hydraulic disturbances (US EPA, 2006; Friedman et al., 2010; Ginige, Wylie & Plumb, 2011; Brandhuber et al., 2015; Health Canada, 2019).

8 Conclusions

Manganese is an essential nutrient that acts as a component of several enzymes and participates in a number of important physiological processes. Although manganese is essential, deficiencies are unlikely because levels in the diet are generally ample to provide adequate amounts for human health. However, elevated levels of manganese in drinking-water have been associated with toxicity. Recognizing data gaps and uncertainty, a number of authoritative bodies have established health-based values for manganese, including lifetime drinking-water levels and dietary upper levels. Differences and limitations in terms of the data considered at

the time of assessment and their interpretation result in a wide range of proposed values (Health Canada, 2019).

8.1 Derivation of the provisional guideline value

The reassessment of the risk posed by manganese identified emerging evidence supporting the oral route as a potentially important route of exposure for manganese toxicity. For drinking-water, a health-based GV is therefore warranted. In 2004, WHO derived a health-based value based on average daily intakes reported in dietary studies in healthy adult women (Greger, 1999; IOM, 2001; WHO, 2004). However, the current reassessment also considers more recent epidemiological data that indicate potential for adverse effects in populations exposed to lower concentrations of manganese in drinking-water.

Despite the data from more recent epidemiological studies (Bouchard et al., 2011, 2018; Khan et al., 2011, 2012; Rodríguez-Barranco et al., 2013; Oulhote et al., 2014; Yu et al., 2014; Haynes et al., 2015; Henn et al., 2017; Rahman et al., 2017; Dion et al., 2018; Ntihabose et al., 2018), uncertainties regarding manganese dose–response properties in the susceptible population remain. Further, there are questions about the bioavailability of the different chemical forms of manganese in drinking-water, including in comparison with food. The limitations in the human epidemiological oral studies, such as lack of an accurate assessment of manganese exposure levels, absence of determination of temporality of effects, and potential confounding factors, preclude their use in GV derivation. Further, no studies are available that specifically address the potential for increased susceptibility to manganese of infants (0–4 months of age), especially bottle-fed infants. Although these studies cannot be used in a quantitative manner to establish a health-based value, they qualitatively support the use of the identified critical end-point of developmental neurotoxicity in animal studies.

The most robust animal toxicity data are from studies conducted in rats. These include exposure during the neonatal period, a life stage with increased susceptibility. From multiple well-conducted studies in rats, a LOAEL for manganese of 25 mg/kg bw/day can be identified based on adverse neurological indices, such as behavioural and sensorimotor effects, and corresponding neurostructural and neurochemical changes in exposed offspring, some of which persisted into adulthood after levels of manganese in the brain had returned to normal (Kern, Stanwood & Smith, 2010; Kern & Smith, 2011; Beaudin, Nisam & Smith, 2013; Beaudin et al., 2017). As noted by Health Canada (2019), several other studies reported neurotoxicity resulting from oral exposure to manganese in rats, mice or monkeys at lower doses (Chandra & Shukla, 1978; Chandra, Shukla & Saxena, 1979; Chandra, Srivastava & Shukla, 1979; Deskin, Bursian & Edens, 1980; Gupta, Murthy & Chandra, 1980; Öner & Sentürk, 1995; Sentürk & Öner, 1996; Shukakidze et al., 2002; Tran et al., 2002b; Shukakidze, Lazriev & Mitagvariya, 2003; Golub et al., 2005; Vezér et al., 2005, 2007; Lazrishvili et al., 2009; Moreno et al., 2009a, b). However, study limitations, such as the lack of a clear account of animal dosing and lack of information concerning long-term effects, confound the interpretation of these studies. Nonetheless, these studies support neurotoxicity as a key end-point of concern for risk assessment.

To calculate the tolerable daily intake (TDI) based on exposure through drinking-water, the 25 mg/kg/day LOAEL is divided by an uncertainty factor (UF) of 1000, comprising:

- 10 for interspecies uncertainty due to the noted interspecies differences between rodents and humans;

- 10 for intraspecies differences due to uncertainties in the level of variation within the human population; and
- 10 for database uncertainties, including the use of a LOAEL rather than a NOAEL.

$$\text{TDI} = \frac{25 \text{ mg/kg bw/day}}{1000}$$

$$= 0.025 \text{ mg total manganese/kg bw/day}$$

Numerous factors might influence the extent of toxicity specific to drinking-water exposure, such as differing chemical forms and valence states in drinking-water, and the higher absorption and increased retention of manganese in infants compared with adults (Health Canada, 2019). Milk or soy-based formula comprises the total diet in non-breast-fed infants for the first few months of life. As noted in section 2.6, there is potential for increased exposure to manganese in this group compared with breast-fed infants because of manganese in both the tap water used to prepare formula and the concentrated or powdered formula itself. The source allocation from drinking-water is assumed to be half of the total potential exposure, with the balance from the formula. Accordingly, an allocation factor of 50% for drinking-water is applied for this assessment. As noted in sections 2.1 and 2.3, there is high variability in manganese concentrations in both drinking-water and formula. Contributions from other sources are not expected to be significant for this age group.

Using the above TDI, allowing for a 50% allocation and a 5 kg body weight for a bottle-fed infant consuming 0.75 L water per day, yields a **health-based GV for manganese of 0.08 mg/L for bottle-fed infants**. This is the subpopulation most susceptible to manganese exposure; therefore, this health-based GV is applicable for the general population as a whole.

$$\text{Health-based guideline value} = \frac{0.025 \text{ mg/kg bw/day} \times 5 \text{ kg} \times 0.5}{0.75 \text{ L/day}}$$

$$= 0.08 \text{ mg/L}$$

This GV is provisional (pGV) because of the high level of uncertainty (as reflected in the composite UF of 1000). It is important to note that levels below this health-based value may result in significant organoleptic acceptability problems – for example, at concentrations as low as 0.02 mg/L. Manganese can deposit on the surface of pipes, causing discolouration of the water and affecting consumer acceptability when it is disturbed.

8.2 Considerations in applying the guideline value

The pGV is for total manganese. The presence of particulate manganese in drinking-water systems can also cause acceptability problems; therefore, aesthetic as well as health aspects should be considered when setting regulations and standards for drinking-water quality.

Manganese levels in drinking-water can be an issue in both high- and low-income countries, and should be considered in establishing national standards and local guidance. Resource-limited suppliers, in particular, may have difficulty in achieving the pGV; in such cases, incremental improvements towards meeting the pGV are encouraged. This is a particular problem for groundwater, for which treatment may be minimal and prohibitively expensive. In such instances, benefits from a reliable, microbiologically safe groundwater source should be assessed against the risks posed by an alternative source that may be subject to faecal

contamination. Issues of acceptability of the drinking-water should also be taken into account, since reduced acceptability may lead consumers to turn to more aesthetically acceptable but less microbiologically safe water supplies. However, what is acceptable varies, and it is vital that a sufficient supply of microbiologically safe water that is acceptable is always available, even if some guidelines or standards for chemicals such as manganese cannot be immediately met.

It should be remembered that the GV is provisional, having been derived with an uncertainty factor of 1000 applied; the previous health-based value was 400 µg/L. As well, the end-point for the pGV, which is cognitive effects, is affected by many other factors. Understanding that the pGV was derived considering the most susceptible subpopulation (bottle-fed infants), risks to infants arising from exceedance of the pGV may be mitigated by following WHO's recommendation for exclusive breastfeeding (WHO, 2014). If this is not possible or supplementary feeding is required, an alternative safe drinking-water source (e.g. bottled water that is certified by the responsible authorities), if available, may be used to prepare infant formula.

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EXHIBIT 6

WHO/SDE/WSH/03.04/14

English only

Silver in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

Originally published in **Guidelines for drinking-water quality,** 2nd ed. Vol. 2. *Health criteria and other supporting information.* World Health Organization, Geneva, 1996.

https://www.who.int/water_sanitation_health/dwq/chemicals/silver.pdf

Preface

One of the primary goals of WHO and its member states is that "all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water." A major WHO function to achieve such goals is the responsibility "to propose regulations, and to make recommendations with respect to international health matters ...."

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as International Standards for Drinking-Water. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO Guidelines for drinking-water quality (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared/updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A "final task force" meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO internet site and in the current edition of the GDWQ.

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J.K. Fawell, Water Research Centre, United Kingdom
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U. Lund, Water Quality Institute, Denmark
(organic constituents and pesticides)
B. Mintz, Environmental Protection Agency, USA
(disinfectants and disinfectant by-products)

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Regional Office for Europe:
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O. Espinoza, Environment and Health

Ms Marla Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

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The convening of the experts meetings was made possible by the financial support afforded to WHO by the Danish International Development Agency (DANIDA), Norwegian Agency for Development Cooperation (NORAD), the United Kingdom Overseas Development Administration (ODA) and the Water Services Association in the United Kingdom, the Swedish International Development Authority (SIDA), and the following sponsoring countries: Belgium, Canada, France, Italy, Japan, Netherlands, United Kingdom of Great Britain and Northern Ireland and United States of America.

GENERAL DESCRIPTION

Identity

Silver (CAS no. 7440-22-4) is present in silver compounds primarily in the oxidation state +1 and less frequently in the oxidation state +2. A higher degree of oxidation is very rare. The most important silver compounds from the point of view of drinking-water are silver nitrate ($AgNO_3$, CAS no. 7761-88-8) and silver chloride (AgCl, CAS no. 7783-90-6).

Physicochemical properties (1)

Property	*$AgNO_3$*	*AgCl*
Colour	White	White, darkens when exposed to light
Melting point (°C)	212	455
Water solubility at 25 °C (g/litre)	2150	0.00186

Major uses

The electrical and thermal conductivity of silver are higher than those of other metals. Important alloys are formed with copper, mercury, and other metals. Silver is used in the form of its salts, oxides, and halides in photographic materials and alkaline batteries, or as the element in electrical equipment, hard alloys, mirrors, chemical catalysts, coins, table silver, and jewellery. Soluble silver compounds may be used as external antiseptic agents (15–50 µg/litre), as bacteriostatic agents (up to 100 µg/litre), and as disinfectants (>150 µg/litre) (*2*).

Environmental fate

Silver occurs in soil mainly in the form of its insoluble and therefore immobile chloride or sulfide. As long as the sulfide is not oxidized to the sulfate, its mobility and ability to contaminate the aquatic environment are negligible. Silver in river water is "dissolved" by complexation with chloride and humic matter (*3*).

ANALYTICAL METHODS

The detection limit of the spectrographic and colorimetric method with dithizone is 10 µg of silver per litre for a 20-ml sample. The detection limit of atomic absorption spectroscopy (graphite furnace) is 2 µg of silver per litre, and of neutron activation analysis, 2 ng of silver per litre (*4*).

ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Air

Ambient air concentrations of silver are in the low nanogram per cubic metre range (*5*).

Water

Average silver concentrations in natural waters are 0.2–0.3 µg/litre. Silver levels in drinking-water in the USA that had not been treated with silver for disinfection purposes varied between “non-detectable” and 5 µg/litre. In a survey of Canadian tapwater, only 0.1% of the samples contained more than 1–5 ng of silver per litre (*5*). Water treated with silver may have levels of 50 µg/litre or higher (*4*); most of the silver will be present as nondissociated silver chloride.

Food

Most foods contain traces of silver in the 10–100 µg/kg range (*6*).

Estimated total exposure and relative contribution of drinking-water

The median daily intake of silver from 84 self-selected diets, including drinking-water, was 7.1 µg (*6*). Higher figures have been reported in the past, ranging from 20 to 80 µg of silver per day (*7*). The relative contribution of drinking-water is usually very low. Where silver salts are used as bacteriostatic agents, however, the daily intake of silver from drinking-water can constitute the major route of oral exposure.

KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

Silver may be absorbed via the gastrointestinal tract, lungs, mucous membranes, and skin lesions (*5*). The absorption rate of colloidal silver after oral application can be as high as 5% (*8*). Most of the silver transported in blood is bound to globulins (*5*). In tissues, it is present in the cytosolic fraction, bound to metallothionein (*9*). Silver is stored mainly in liver and skin and in smaller amounts in other organs (*5,10*). The biological half-life in humans (liver) ranges from several to 50 days (*9*).

The liver plays a decisive role in silver excretion, most of what is absorbed being excreted with the bile in the faeces. In mice, rats, monkeys, and dogs, cumulative excretion was in the range 90–99%. Silver retention was about 10% in the dog, <5% in the monkey, and <1% in rodents (*10*). In humans, under normal conditions of daily silver exposure, retention rates between 0 and 10% have been observed (*5*).

EFFECTS ON LABORATORY ANIMALS AND *IN VITRO* TEST SYSTEMS

Acute exposure

Oral LD_{50} values between 50 and 100 mg/kg of body weight have been observed for different silver salts in mice (*11*).

Short-term exposure

Hypoactive behaviour was observed in mice that had received 4.5 mg of silver per kg of body weight per day for 125 days (*12*).

Long-term exposure

After 218 days of exposure, albino rats receiving approximately 60 mg of silver per kg of body weight per day via their drinking-water exhibited a slight greyish pigmentation of the eyes, which later intensified (*13*). Increased pigmentation of different organs, including the eye, was also observed in Osborne-Mendel rats after lifetime exposure to the same dose (*14*). Antagonistic effects between silver and selenium, involving the selenium-containing enzyme glutathione peroxidase, were observed in Holtzman rats (*15*).

Mutagenicity and related end-points

In the *rec*-assay with *Bacillus subtilis*, there were no indications that silver chloride was mutagenic (*16*). Reverse mutations in *Escherichia coli* were not induced by silver nitrate (*17*). In the DNA repair test with cultivated rat hepatocytes, silver nitrate solution was positive only at a moderately toxic concentration (*18*). Silver nitrate increased the transformation rate of SA7-infected embryonic cells of Syrian hamsters (*19*).

Carcinogenicity

Silver dust suspended in trioctanoin injected intramuscularly in Fischer 344 rats of both sexes was not carcinogenic (*20*).

EFFECTS ON HUMANS

The estimated acute lethal dose of silver nitrate is at least 10 g (*21*).

The only known clinical picture of chronic silver intoxication is that of argyria, a condition in which silver is deposed on skin and hair, and in various organs following occupational or iatrogenic exposure to metallic silver and its compounds, or the misuse of silver preparations. Pigmentation of the eye is considered the first sign of generalized argyria (*21*). Striking discoloration, which occurs particularly in areas of the skin exposed to light, is attributed to the photochemical reduction of silver in the accumulated silver compounds, mainly silver sulfide. Melanin production has also been stimulated in some cases (*22,23*).

It is difficult to determine the lowest dose that may lead to the development of argyria. A patient who developed a grey pigmentation in the face and on the neck after taking an unknown number of anti-smoking pills containing silver ethanoate was found to have a total body silver content of 6.4 ± 2 g (*22*). It has been reported that intravenous administration of only 4.1 g of silver arsphenamine (about 0.6 g of silver) can lead to argyria (*24*). Other investigators concluded that the lowest intravenous dose of silver arsphenamine causing argyria in syphilis patients was 6.3 g (about 0.9 g of silver) (*21*). It should be noted that syphilis patients suffering from argyria were often already in a bad state of health and had been treated with bismuth, mercury, or arsphenamine in addition to silver.

CONCLUSIONS

Argyria has been described in syphilitic patients in poor health who were therapeutically dosed with a total of about 1 g of silver in the form of silver arsphenamine together with other toxic metals. There have been no reports of argyria or other toxic effects resulting from the exposure of healthy persons to silver.

On the basis of present epidemiological and pharmacokinetic knowledge, a total lifetime oral intake of about 10 g of silver can be considered as the human NOAEL. As the contribution of drinking-water to this NOAEL will normally be negligible, the establishment of a health-based guideline value is not deemed necessary. On the other hand, special situations may exist where silver salts are used to maintain the bacteriological quality of drinking-water. Higher levels of silver, up to 0.1 mg/litre (a concentration that gives a total dose over 70 years of half the human NOAEL of 10 g), could then be tolerated without risk to health.

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Aluminum and silica in drinking water and the risk of Alzheimer's disease or cognitive decline: findings from 15-year follow-up of the PAQUID cohort

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Abstract

The authors examined associations between exposure to aluminum or silica from drinking water and risk of cognitive decline, dementia and Alzheimer's disease. Subjects were followed-up for 15 years with an active search for incident cases of dementia, aged 65 years and over living in 91 civil drinking water areas in Southern France. Two measures of exposure to aluminum were assessed: a geographical exposure and an individual exposure taking into account the daily consumption of tap and bottled water. A total of 1,925 subjects free from dementia at baseline and with reliable water assessment were analyzed.

Using random effects models, cognitive decline with time was greater in subjects with a higher daily aluminum intake from drinking water (≥ 0.1 mg/day, $p = 0.005$) or a higher geographical exposure to aluminum. Using a Cox model, a high daily intake of aluminum was significantly associated with increased risk of dementia. Conversely, an increase of 10 mg/day in silica intake was associated with a reduced risk of dementia (adjusted RR = 0.89, $p = 0.036$). However, the geographical exposure to aluminum or silica from tap water was not associated with dementia. High consumption of aluminum from drinking water may be a risk factor for Alzheimer's disease.

Keywords: Aged, Aged, 80 and over, Aluminum, adverse effects, analysis, Alzheimer Disease, chemically induced, diagnosis, epidemiology, Cognition Disorders, chemically induced, diagnosis, epidemiology, Drinking, Environmental Exposure, Female, Follow-Up Studies, France, epidemiology, Humans, Incidence, Male, Proportional Hazards Models, Psychiatric Status Rating Scales, Questionnaires, Rural Population, Silicon Dioxide, adverse effects, analysis, Urban Population, Water Pollutants, Chemical, Water Supply, analysis
Keywords: Aluminum, Alzheimer's disease, cognitive functions, dementia, drinking water, silica

Alzheimer disease (AD) is a neurodegenerative cerebral disorder defined as a progressive deterioration of cognitive function and loss of autonomy. Although knowledge of the pathophysiology of AD has greatly progressed over the past decades, its causal mechanisms are far from clear.

The hypothesis that aluminum (Al) exposure is aetiologically related to Alzheimer's disease has led to much debate. The possibility of such a relation was suggested by the presence of aluminum in senile plaques and neurofibrillary degeneration, two histological lesions that are characteristic of the disease (1). Several studies report that intake of aluminum (2, 3) increases expression of amyloid protein in rodent tissues, a step that may be critical to the development of Alzheimer's disease. Ecological studies have suggested that concentrations of aluminum in drinking water of 0.1–0.2 mg/l may increase the risk of Alzheimer's disease with relative risk or odds ratio ranging from 1.35 to 2.67 (4–8). All the epidemiological studies thus far, except one (9), however, have ignored the individual daily intake of drinking water.

Some, but not all, epidemiological and experimental studies suggest silica species can reduce aluminum oral absorption and/or enhance aluminum excretion and protect against aluminum-induced adverse effects (5, 9, 10). The silica (Si) content of tap water can vary according to the geographical region, with typically high Si levels in hard water areas and low levels in soft water areas. In two studies carried out in Egypt (11) or UK (12), bottled water of all brands (spring or mineral waters) contained higher levels of Si than tap water. This may well be because tap water treatment (i.e. by Al flocculation) decreases the Si content. We previously reported a geographical association between aluminum and silica and the cognitive decline or dementia on the data of the PAQUID (Personnes âgées Quid) cohort (4, 5) for subjects followed during 8 years and with a low number of exposed subject. Our aim in the present work was to analyse the associations with more precise daily Al or silica intake on a larger cohort followed-up to 15 years, with additional exposed subjects and with a majority of new events occurring after the 8-year of follow-up.

MATERIALS AND METHODS

Participants/recruitment

Figure 1 illustrates the study flow chart. Briefly, PAQUID is an ongoing prospective-cohort population-based study of the epidemiology of dementia and Alzheimer's disease in the elderly population in France (13). The study beginning in 1988, initially included a community-based cohort of 3,777 elderly people, aged 65 and older, and living at home in one of 75 randomized rural or urban drinking water areas of the administrative areas of Gironde or Dordogne in southwestern France. Subjects were randomly selected from electoral rolls and were followed-up regularly between 1988 and 2004. The PAQUID study was approved by an ethical review committee.

To increase the number of exposed subjects we added the data of the ALMA+ cohort (for aluminum – maladie d'Alzheimer). This cohort of 400 subjects was randomly selected from electoral rolls at the same time as the 10-year follow-up of the PAQUID cohort. These subjects aged 75 years and over at entry lived at home in one of the 14 drinking water areas of the administrative area of Dordogne in south-western France with five drinking water areas with mean levels of Al between 0.050 and 0.100 mg/l and nine areas with Al $\geq$ 0.100 mg/l. These subjects, first seen in 1999 then in 2003, were expected to be comparable with the subjects seen at the 10-year follow-up of the PAQUID cohort. The cognitive decline was analyzed on the PAQUID cohort and the ALMA+ cohort. Dementia and AD were investigated only on the PAQUID cohort because of the non-symmetrical screening process in the two cohorts and because of the two different follow-up.

Assessment of cognitive functions, dementia and AD

At baseline, a psychologist who gathered sociodemographic data, medical antecedents, and functional disability saw subjects at home. Intellectual functioning assessment included an evaluation of global mental status (Mini-Mental State Examination, MMSE) (14) and a battery of other tests. At the end of the visit, the psychologists systematically completed a standardized questionnaire designed to obtain the criteria for dementia according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III R) (15). A senior neurologist subsequently saw subjects who met these criteria at home to confirm and complete the DSM-III R criteria for dementia and to apply the National Institute of Neurological and Communication Disorders and Stroke/Alzheimer's Disease and Related Disorders Association criteria for AD (16) and the Hachinski score (17) for vascular dementia.

Measure of exposure and water consumption

On the basis of information given by the sanitary administration, we respectively divided the PAQUID sample and the ALMA+ sample into 77 and 14 drinking water areas. For each area, we computed a weighted mean of all measures of aluminum and silica by using the results of chemical analyses of drinking water carried out by the sanitary administration between 1991 and 1994. In order to evaluate the past exposure of subjects, the history of the water distribution network over the previous ten years (1981–1991) was evaluated into the PAQUID cohort.

The 8-year follow-up questionnaire in the PAQUID cohort and the three following ones as well as the first and second in the ALMA+ cohort included a dietary investigation that contained specific questions relating to the daily consumption of tap water (including water used in making tea, coffee, soup or alcoholic drink) and bottled water (spring or mineral) and their brand most frequently consumed. The first non-missing information collected was used for each individual exposure, assuming a stable daily water consumption over the period of observation. The composition of the various bottled waters was provided by the respective distributing companies. On the contrary to the mineral water, the composition of bottled spring water may change over time; even so we used an average over several measurements across time (mean number of values 1.9). For each subject, a daily mean intake of aluminum or silica from tap water and/or bottled water was computed. The statistical analyses are then based on two kinds of drinking water indicators for aluminum or silica: a geographical exposure (in mg/liter) previously used in the PAQUID cohort (5) and an individual indicator, more precise (in mg/day) taking daily bottled and tap water consumption into account.

Statistical analysis

Analyses of cognitive decline were performed using a random effects linear regression model, including subject-specific random intercept and slope to take into account the intra-subject correlation. A random intercept specific for each geographical area controlled for the potential intra-area correlation. Since the distribution of the MMSE scores was not normal, we analyzed the square root of the number of errors according to time (5). Besides the variable *time* representing the number of years after the initial visit, a binary indicator for the initial visit was introduced to account for the first-passing effect, possibly due to stress. Aluminum was considered as a quantitative variable, or as a binary variable with the threshold of 0.1 mg/liter already used in previous ecological studies (6, 7), or 0.1 mg/day for individual exposure, or in four classes according to the three terciles (on subjects) under 0.1 mg/day and the category above 0.1 mg/day. Silica was considered as a quantitative variable or as a binary variable with 11.25 mg/liter for the geographical exposure (the median in our sample) or with 10.55 mg/day as the cut-off for the individual exposure (the median of daily intake in our sample) or into 4 classes according to the four quartiles. We adjusted for potential confounders: educational level (18), wine consumption (19), place of residence (rural versus urban) and the cohort (PAQUID or ALMA+).

To examine the robustness of the results in the main analysis on cognitive decline we assessed influence diagnostics, using the Cook's D statistic (20) in the final adjusted model. The 20 most globally influential subjects were removed and updated estimates of model parameters were computed.

Analyses of the risk of dementia or AD were performed using a Cox proportional hazard model with delayed entry (21) to estimate relative risks (RR) and to adjust for covariates. Age was taken as the basic time scale in the analysis, so that the risks of dementia or AD were adjusted non-parametrically for age. A stratified analysis for gender was performed (21).

All analyses were conducted using the MIXED and PHREG in the SAS software, version 9.1 (SAS Institute, INC., Cary, North Carolina).

RESULTS

Among the 4,177 subjects (3,777 from PAQUID and 400 from ALMA+) who initially agreed to participate, 207 with prevalent dementia were excluded. The current study is restricted to the 1,925 subjects (among the 3,970 non-demented at their first visit) in 91 geographical areas, who have non-missing values for daily consumption of Al or Silica from drinking water and for adjustment covariates. Subjects from PAQUID lost to follow-up or died before the 8-year of follow-up, had no measure of water consumption and were excluded from the study. Baseline characteristics of the study sample are shown in table 1.

The PAQUID sample at the 10-year follow-up and the ALMA+ sample at entrance were as expected very similar (mean age = 82.52 and 82.31, p = 0.51; MMSE scores = 24.91 and 25.93, p<0.0001; percentage of women = 61.66% and 59.27%, p = 0.47; percentage of high educated patients = 70.66 and 66.53, p = 0.18). The ALMA+ patients had a higher consumption of Al from drinking water (mean = 0.136 mg/day) than in the PAQUID cohort (mean = 0.009 mg/day), p<0.0001.

The mean consumption of drinking water was 0.94 (SD = 0.49) liters/day. Tap water was the sole source of water intake for 43.7 percent of the subjects; 40.3 percent drank only bottled water. The compositions of Al in tap water varied greatly from one parish to another from 0.001 to 0.514 mg/liter, with a mean value of 0.043 mg/liter (median = 0.009 mg/liter) depending largely on the method of water treatment used (i.e. by Al flocculation or not). In bottled water, when available or detectable, the concentrations of Al are very small with a maximum value of 0.032 mg/liter and with a mean value of 0.002 mg/liter (median = 0). Silica levels in tap water ranged from 4.2 to 22.4 mg/liter and were inversely related to aluminum concentrations, but this negative correlation was weak in our study (Pearson correlation coefficient = −0.18, $p = 0.13$). In bottled water, the concentrations of Si ranged from 2 mg/liter to 77.6 mg/liter. The daily mean intake of Al and Si from drinking water is described in Table 2. The correlation between geographical exposures and individual exposure was 0.71 (p<0.001) for aluminum and 0.13 (p<0.001) for silica. Among subjects studied, 112 were exposed to more than 0.1mg/day of aluminum essentially due to a high consumption of tap water with high levels of Al.

Relation between cognitive functions and water composition into the PAQUID and ALMA+ cohort

Aluminum intake interacted significantly with time (Table 3). Cognitive decline was greater in subjects with a high daily Al intake (greater than 0.1 mg/day or an increase of 0.1 mg/day). However, Al had no significant association with the values of the MMSE scores at inception in the cohort. As an example, a woman without a diploma aged 75 years at inception, with a low daily silica intake (<10.55mg/day) and a low daily Al intake (<0.1 mg/day) would in average lose 1.5 points on the MMSE score between the first follow-up and the 15-year follow-up; but with a high daily Al intake (≥ 0.1 mg/day), she would lose 5.0

points. In these models, even after adjustment for different factors, significant but very low intra-parish correlation was obtained (in model 1 from Table 3, the variance of the intra-parish random effect = 0.008, p = 0.019). This may mean that other geographical factors may also influence cognitive decline.

The same tendencies were obtained using the geographical tap water exposure: cognitive decline with time was greater in subjects exposed to high levels of aluminum (models 3 and 4, Table 3). Neither individual intake of silica nor geographical exposure was significantly associated with cognitive functions.

The interaction between Al and time was no longer significant (p = 0.78) when excluding the demented subjects. This suggests that cognitive decline with time is related to daily Al intake only when associated with a dementia process.

Among the 20 most influential subjects (about 1% of the sample) 7 had a high consumption of aluminum (> 0.100 mg/day). The parameter estimate for aluminum by time after deleting the 20 most influential patients was unchanged but had a larger p-value (β = 0.045, p = 0.01) than on the full dataset.

When repeating the cognitive decline analysis using only the PAQUID sample we observed very similar interactions aluminum or silica with time (model 2 in Table 3, β = 0.020, p = 0.004 for Al; β = −0.003, p = 0.10 for silica).

The principal lifetime occupation with an eight-class variable was also added. The effects of aluminium by time and silica by time (not shown in the tables) were unchanged, respectively β = 0.046 (p = 0.009) and β = −0.004 (p = 0.35) in model 1.

Relation between dementia or Alzheimer's disease and water composition into the PAQUID cohort

Over the 15-years of follow-up of the PAQUID cohort 1,677 subjects were analyzed and 461 subjects were diagnosed with dementia; the mean follow-up duration was 11.3 years. Only 13 subjects had high daily consumption of Al from drinking water (≥ 0.1 mg/day), among them 6 (46.2%) were demented. There were 364 subjects (78.9 percent) classified as having Alzheimer's disease (probable or possible). The incidence rates for all causes of dementia and for Alzheimer's disease were estimated as 2.44 per 100 person-years and 1.92 per 100 person-years, respectively. The risk of dementia was higher for subjects with a high daily Al intake (adjusted relative risk (RR) = 2.26 for Al≥0.1 mg/day, *p = 0.049,* model 5, Table 4). Conversely, an increase of 10 mg/day in silica intake was associated with a reduced risk of dementia (adjusted RR = 0.89, *p* = 0.036, model 5). No tendency for a dose-response effect for aluminum was apparent (likelihood ratio statistic = 3.52, 3 df, *p* = 0.32, model 7, table 4) even though a significant linear relation between aluminum and dementia was obtained in model 6 (adjusted RR for aluminum = 1.28 for an increase of 0.1 mg/day, *p* = 0.017). The model 6 with aluminum as a continuous variable was slightly better than that (model 5) in which aluminum was in two classes (Akaike difference = 1.1). There was no significant interaction between aluminum and silica concentrations.

Analyses restricted to cases classified as Alzheimer's disease (364 cases) also suggested a deleterious effect of high aluminum intakes and a protective effect of high silica intake. These effects were not significant for other types of dementia (97 cases, data not shown).

Using the geographical tap water exposure, the concentrations of Al or Silica were no more associated with the risk of dementia or AD, although the tendencies were similar (results not shown here).

DISCUSSION

We found that the cognitive decline and the risk of dementia were higher for high consumption of Al from drinking water. Even if almost the same tendencies as previously published on Paquid (5) were obtained on the effect of geographical exposure to aluminum, this exposure was no more significantly associated with dementia. This result being based on a small number of exposed subjects in this sample (n = 46 with Al ≥0.100mg/l), it may be explained by a lack of power in the analysis. This strengthens the importance of using an individual rather than a geographical exposure. The analysis did not show any evidence for silica intake to be associated with the evolution of cognitive functions; however it showed an inverse association between silica intake from drinking water and the risk of dementia, or more specifically of AD.

Biases and limitations

The findings of our study warrant some caution in interpretation, owing to some limitations.

Although we adjusted for several potential confounding factors, the possibility of residual confounding cannot be completely excluded. We thus adjusted for several individual factors such as age, sex, wine consumption, educational level, place of residence potentially associated with the bottled water consumption.

Subjects drinking only bottled water may have a particular exposure since they are not-exposed to aluminum from drinking water and can be more exposed to silica (if the bottled water contains high levels of silica). We repeated the main analyses excluding those persons. In the dementia analysis on the Paquid sample (749 subjects excluded over 1,677), the effect of aluminum remained equivalent (for instance the model 5 in Table 4 became, RR = 2.31, p = 0.045), but silica was no more significant (RR = 1.04, p = 0.13).

The bottled water consumption may also change with time and may be different for demented patients compared to non-demented patients. We studied this evolution on the subsample of 476 subjects from the PAQUID cohort seen at each follow-up time since the assessment of daily water consumption (T8, T10, T13, T15). The intraclass correlation coefficient based on a random effect linear regression for the daily intake of bottled water was equal to 0.54. This indicates that the daily bottled intake was rather stable between T8 and T15. The same tendencies were observed for the 402 non-demented patients (ρ = 0.55), and for the 74 demented patients (ρ = 0.47). It seems that the disease does not change that much the consumption habits of bottled drinking water. Furthermore the water consumption information was mainly collected on non-demented patients (1406/1677 = 83.8%). All these comments strengthen the validity of our results even if the information for the bottled water consumption was only available after the 8-year follow-up.

We may think that the social or educational level may influence the bottled water consumption and so the daily intake of Al or Si. A high consumption of bottled water leads to a lower Al intake and most of the time to a greater silica intake. The mean daily bottled consumption was not significantly different in our sample for high educated patients (0.48 liter/day) compared to low educated patients (0.47 liter/day). In the analyses of dementia in the PAQUID cohort, only 13 subjects were exposed to more than 0.1 mg/day of aluminum, essentially due to a high consumption of tap water with high levels of Al. These subjects were distributed in 5 drinking water areas with more than 0.05 mg/liter. Even though the number of subjects with a high daily Al intake was low, almost half of them (6/13) developed a dementia over the 15-years of follow-up.

Food contribute ~95 % and drinking water 1 to 2% of the typical human's daily Al intake. However, the very limited available data suggest oral aluminum bioavailability, namely the fraction that is actually taken up into the blood stream) from food (~ 0.1%) is less than from water (~ 0.3%). Yokel et al. (22) recently suggested that food provides ~25-fold more Al to systemic circulation, and potential Al body burden, than

does drinking water. Evidence surrounding the relationship between aluminum in food and the risk of AD is very minimal (23), probably due to the difficulty in obtaining accurate exposure information in dietary studies.

Strengths

A great advantage of our study was that we had an estimate of the daily individual intakes of Al and silica supplied by the drinking water, and not merely the geographical concentrations of these elements, as in most epidemiologic studies previously published (4, 5, 7, 24). This individual intake of drinking water is more precise and leads to more accurate findings.

Only one recent French cohort (EPIDOS) analyzed also the individual daily consumption of aluminum or silica from drinking water (9). At baseline, low silica concentration was associated with low cognitive performance and with more AD patients. No significant changes were observed with aluminum intakes. These results corroborate our results for silica only. The EPIDOS study was however a selected population of volunteers not representative of the general population and with much lower levels of aluminum (maximum = 0,063 mg/liter).

The study of cognitive functions in addition to the risk of dementia has two main methodological interests. First, the evolution of the MMSE score is not sensitive to diagnostic errors that may be present in the detection of AD cases. Secondly, cognitive decline precedes by three to five years the occurrence of dementia and is less subject to competitive morbidity or mortality.

The survey design incorporates a grouping of the participants into drinking water areas, this has the advantage to give heterogeneity in the drinking water exposures or other environmental factors but this may induce a correlation of the observations. In a random effect survival model (5, 25) no significant intra-group correlation was observed ($p = 0.31$). The effects of aluminum (RR = 2.22, SE = 0.43 for models in Table 4) and silica (RR = 0.90, SE = 0.05) were unchanged. It is thus unlikely that some unmeasured environmental factor shared by the members of the same parish could play a confounding role on dementia.

Further studies are needed to settle the debate over the link between aluminum or silica in drinking water and neurological disorders and cognitive impairment. Ideally, in such studies individual data on drinking water exposure as well as other relevant risk factors is needed to assess this potential risk.

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Abbreviations

Al	Aluminum
AD	Alzheimer's Disease
MMSE	Mini Mental State Examination
Si	Silica

Footnotes

Conflict of interest: none

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Figures and Tables

Figure 1

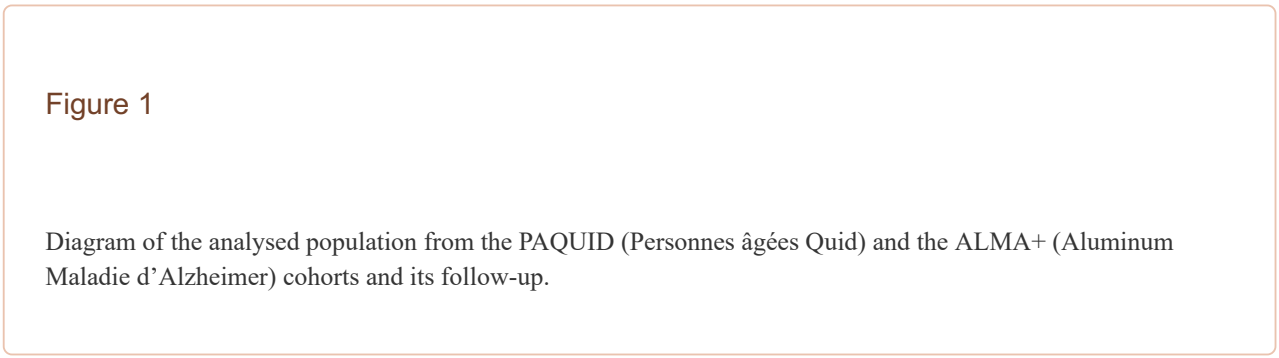


Diagram of the analysed population from the PAQUID (Personnes âgées Quid) and the ALMA+ (Aluminum Maladie d'Alzheimer) cohorts and its follow-up.

Table 1

Distribution of potential confounding variables across levels of aluminum concentrations, the PAQUID and ALMA+ cohorts, France, 1988–2003.

	Aluminum from tap water (n = 1,883*)		**Daily consumption of aluminum (from tap water and/or bottled water) (n = 1,925)**		
	Geographical exposure		**Individual exposure**		
Characteristics at baseline	**≥ 0.100 mg/liter (n=216)**	**< 0.100 mg/liter (n = 1,667)**	**≥ 0.100 mg/day (n = 112)**	**< 0.100 mg/day (n = 1,813)**	**Total (n = 1,925)**
Silica from tap water (geographical exposure)					
≥ 11.25 mg/liter	131 (60.7%)	1,033 (62.2%)	73 (65.2%)	1,091 (61.8%)	1,164 (62.1%)
< 11.25 mg/liter	85 (39.3%)	627 (37.8%)	39 (34.8%)	673 (38.2%)	712 (37.9%)
Daily intake of silica (from tap water and/or bottled water)					
≥ 10.55 mg/day	141 (65.3%)	860 (51.6%)	87 (77.7%)	935 (51.6%)	1,022 (53.1%)
< 10.55 mg/day	75 (34.7%)	807 (48.4%)	25 (22.3%)	878 (48.4%)	903 (4.9%))
Gender					
Male	89 (41.2%)	640 (38.4%)	48 (42.9%)	696 (38.4%)	744 (38.6%)
Female	127 (58.8%)	1,027 (61.6%)	64 (57.1%)	1,117 (61.6%)	1,181 (62.4%)
Education					
No education or primary school (ages 6 through 12 years) without diploma	77 (35.7%)	481 (28.9%)	36 (32.1%)	539 (29.7%)	575 (29.9%)
At least primary school with diploma	139 (64.3%)	1,186 (71.1%)	76 (67.9%)	1,274 (70.3%)	1,350 (70.1%)
Place of residence					
Rural	182 (84.3%)	604 (36.2%)	100 (89.3%)	721 (39.8%)	821 (42.7%)
Urban	34 (15.7%)	1,063	12 (10.7%)	1,092 (60.2%)	1,104

*Tap water aluminum concentrations were not available for each geographical area, thus among the 1,925 subjects analyzed, only 1,883 had no missing values for tap water aluminum concentration

Table 2

Daily intakes of aluminum and silica supplied by drinking water (n = 1925)

Element	Intake in mg /day mean ± SD* (min-max)	Amount supplied by tap water	Amount supplied by bottled water	Pearson correlati coefficient
Aluminum	0.025 ± 0.08 (0–1.03)	95.9 %	4.1 %	P = 0.17 (*p<0.0001*)
Silica	13.37 ± 10.76 (0–108)	41.0 %	59.0 %	

*SD, standard deviation

Table 3

Daily consumption of aluminum and silica (mg/day) or geographical exposure to aluminum and silica from drinking water and cognitive decline for the square root of the number of errors in the Mini-Mental State Examination, the PAQUID and ALMA+ cohorts, France, 1988–2003.

	Cognitive decline*	
Daily consumption (mg/day)	**β (SD†)**	***p-values***
Model 1		
Aluminum (≥ 0.1 vs < 0.1)	−0.15 (0.098)	0.08
Time (years) by aluminum	0.049 (0.018)	**0.005**
Silica (≥10.55 vs < 10.55)	−0.022 (0.029)	0.46
Time (years) by silica	−0.005 (0.004)	0.24
Model 2		
Aluminum (continuous‡)	−0.031 (0.023)	0.19
Time (years) by aluminum	0.017 (0.005)	**0.001**
Silica (continuous§)	−0.020 (0.014)	0.15
Time (years) by silica	−0.003 (0.002)	0.11
Geographical exposure (mg/liter)		
Model 3		
Aluminum (≥ 0.1 vs < 0.1)	−0.12 (0.070)	0.09
Time (years) by aluminum	0.038 (0.011)	**<0.001**
Silica (≥11.25 vs < 11.25)	−0.018 (0.034)	0.60
Time (years) by silica	−0.003 (0.004)	0.45
Model 4		
Aluminum (continuous‡)	−0.023 (0.024)	0.35
Time (years) by aluminum	0.014 (0.004)	**<0.001**
Silica (continuous§)	−0.032 (0.053)	0.55
Time (years) by silica	−0.0004 (0.007)	0.99

*adjusted for *time,* an indicator for the first follow-up *(indicT0), age, time* by *age, gender, time* by *gender, indicT0* by *gender, educational level, time* by *educational level, indicT0* by *educational level, cohort.*
†SD, standard deviation
‡aluminum given for an increase of 0.1 mg/day
§silica given for an increase of 10 mg/day

Table 4

Daily aluminum or silica consumption from drinking water and risk of dementia or Alzheimer's disease, the PAQUID cohort, France, 1988–2003.

	Dementia (461 cases)			Alzheimer (364 cases)		
Variable in mg/day	**RR***	**95% CI***	***p-value***	**RR***	**95% CI***	***p-value***
Model 1†						
Al* ≥0.1 vs <0.1	2.59	1.15, 5.80	**0.021**	3.35	1.49, 7.52	**0.003**
Model 2†						
Al (continuous)§	1.29	1.05, 1.58	**0.014**	1.36	1.11, 1.67	**<0.001**
Model 3†						
Si* ≥10.55 vs <10.55	0.91	0.76, 1.10	0.330	0.91	0.74, 1.12	0.360
Model 4†						
Si (continuous)#	0.89	0.80, 0.98	**0.002**	0.88	0.79, 0.99	**0.030**
Model 5‡						
Al ≥0.1 vs <0.1	2.26	1.00, 5.07	**0.049**	2.80	1.24, 6.32	**0.013**
Si (continuous) #	0.89	0.81, 0.99	**0.036**	0.89	0.79, 1.00	**0.045**
Model 6‡						
Al (continuous) §	1.28	1.05, 1.58	**0.017**	1.34	1.09, 1.65	**<0.006**
Si (continuous) #	0.89	0.81, 0.99	**0.028**	0.88	0.79, 0.99	**0.035**
Model 7‡						
Al						
<0.0012	1			1		
[0.0012–0.0045[	0.96	0.76, 1.21	0.727	0.99	0.76, 1.28	0.910
[0.0045–0.1000[	0.98	0.78, 1.24	0.860	1.05	0.81, 1.37	0.698
≥0.1000	2.34	1.03, 5.32	**0.044**	3.04	1.32, 6.97	**0.009**
Si (quartiles)						
> 15.45	1			1		
]10.55–15.45]	1.14	0.87, 1.49	0.354	1.14	0.84, 1.55	0.403
]5.86–10.55]	1.34	1.03, 1.75	**0.029**	1.38	1.03, 1.86	**0.034**
≤5.86	1.33	1.01, 1.74	**0.041**	1.33	0.98, 1.80	0.071

*CI, confidence interval; RR, relative risk; Al, aluminum; Si, silica

†Nonparametrically adjusted for age and gender

‡Nonparametrically adjusted for age and gender and parametrically adjusted for educational level, wine consumption and place of residence

§RR given for an increase of 0.1 mg/day of aluminum

#RR given for an increase of 10 mg/day of silica

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

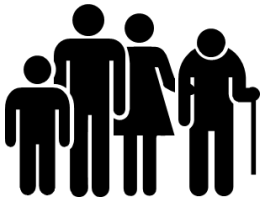
EXHIBIT 8

Sulfate

Sulfate occurs naturally in most of Minnesota's groundwater. Higher levels of sulfate are common in the western part of the state. At high levels, sulfate can give water a bitter or medicinal taste and can have laxative effects.

You can find out the level of sulfate in your water by having the water tested at a laboratory.

Health Risks for Humans

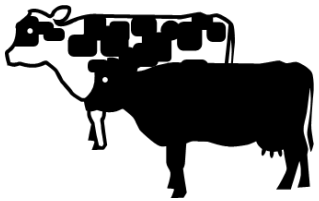


People who are not used to water with high sulfate can get diarrhea and dehydration from drinking the water. Infants are often more sensitive to sulfate than adults. To be safe, **only use water with a sulfate level lower than 500 milligrams per liter (mg/L) to make infant formula.** Older children and adults may get used to high sulfate levels after a few days.

Other Problems Sulfate Can Cause

Sulfate levels above 250 mg/L may make the water taste bitter or like medicine. High sulfate levels may also corrode plumbing, particularly copper piping. In areas with high sulfate levels, plumbing materials more resistant to corrosion, such as plastic pipe, are commonly used.

Health Risks for Animals



Animals are also sensitive to high levels of sulfate. In young animals, high levels may be associated with severe, chronic diarrhea and even death. Animals tend to get used to sulfate over time. Diluting water high in sulfate with water low in sulfate can help avoid problems of diarrhea and dehydration in young animals and animals not used to drinking high sulfate water. Contact a veterinarian or your county office of the Minnesota Extension Service for more information.

Ways to Treat Sulfate

Four types of treatment systems will remove sulfate from drinking water:

- **Reverse osmosis** pushes water through a membrane with tiny pores. The membrane stops many contaminants, including sulfate, while allowing water to pass through. Reverse osmosis usually removes between 93 and 99 percent of the sulfate in drinking water, depending on the type of treatment unit.
- **Distillation** is a process that boils water, making steam. The steam rises and leaves contaminants, such as sulfate behind. With proper operation, distillation units can remove nearly 100 percent of sulfate.
- **Anion exchange** is the most common method of removing large quantities of sulfate from water for commercial, livestock, and public supplies. It is not commonly used for individual household water treatment. It is a process that replaces negatively charged ions (such as sulfate) with sodium chloride or potassium chloride (salts).
- **Adsorptive media filtration** has a charged media bed that can force ions of the opposite charge (such as sulfate) to be pulled out of the water and attach to the media.

Learn more about these treatment options at the "Home Water Treatment" webpage.

Note that water softeners, carbon filters, and sediment filters **do not remove sulfate**.

How Sulfate Gets Into Groundwater

As water moves through soil and rock formations that contain sulfate minerals, some of the sulfate dissolves into the groundwater. Minerals that contain sulfate include magnesium sulfate (Epsom salt), sodium sulfate (Glauber's salt), and calcium sulfate (gypsum).

Sulfate in Minnesota Groundwater

The level of sulfate in most groundwater in Minnesota is low, less than 250 milligrams per liter (mg/L). High levels of sulfate (sometimes above 1000 mg/L) are more common in the southwestern areas of Minnesota and along the western boundary of the state. High levels of sulfate also occur, though less commonly, in some wells in the northeastern and southeastern parts of the state.

Resources

Home Water Treatment
(www.health.state.mn.us/communities/environment/water/factsheet/hometreatment.html).

Licensed Well and Boring Contractor Directory
(www.health.state.mn.us/lwcsearch).

Search for Accredited Laboratories
(www.health.state.mn.us/labsearch).

Water Quality, Well Testing, Well Disinfection
(www.health.state.mn.us/wellwater).

MDH District Offices

625 North Robert Street
P.O. Box 64975
St. Paul, Minnesota 55164-0975
651-201-4600 or 800-383-9808
health.wells@state.mn.us
www.health.state.mn.us/wells

705 Fifth Street Northwest
Bemidji, Minnesota 56601
218-308-2100

11 East Superior Street
Duluth, Minnesota 55802
218-302-6166

1505 Pebble Lake Road
Fergus Falls, Minnesota 56537
218-332-5150

3333 West Division Street
St. Cloud, Minnesota 56301
320-223-7300

1400 East Lyon Street
Marshall, Minnesota 56258
507-476-4220

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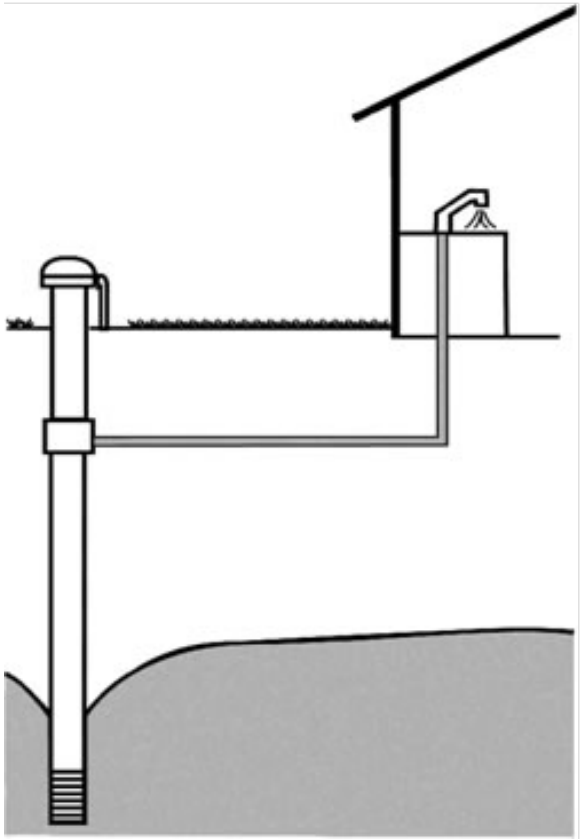
Sulfate in Well Water



DEPARTMENT OF HEALTH

Well Management Section
Environmental Health Division

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 9

ORIGINAL ARTICLE

Health Effects from Exposure to Sulphates and Chlorides in Drinking Water

MUHAMMAD TARIQ BASHIR, SALMIATON ALI, *ADNAN BASHIR

ABSTRACT

This study was designed keeping in view the negative and harmful effects of high levels of Sulphates and Chlorides present in drinking water sources after investigating Sulphate and Chloride contents. Sadiqabad, Rahim Yar Khan, Khanpur and Liaqatpur cities of district RYK, Punjab, Pakistan were investigated for the Sulphate and Chloride levels in different drinking water sources.53 and 23 percent of Sulphate and Chloride samples respectively were found having values greater than the guideline value obtained from the whole district of Rahim Yar Khan. Health Survey was conducted in the areas with higher contents of Sulphates and Chlorides .Almost 55 percent of population confirmed laxative effect and taste problem. Suggestions to prevent health effects were given.
Key words: Health effects, sulphates, chloride, drinking water

INTRODUCTION

In Pakistan, most of the population relies on shared water sources. Whether it is ground water, nearby river, ponds or even harvested rainwater, these water sources are usually shared by both humans and animals. Human uses include purposes such as bathing, washing, laundering, cooking and drinking. These uncontrolled varieties of human and animal use potentially alter the quality of natural source waters significantly. This calls for the need for effective management that warrants the maintenance of the fitness for use of water resources on a sustained basis, achieving a balance between usage and environmental protection.

Globally the subject of contaminant levels in drinking water has been a long contentious issue. However, in Pakistan and other developing nations where relevant institutional capacities are either non-existent or fragile, robust surveillance and early warning systems for chemical contaminants rarely exist. In cases where they do, the focus is on water access and not water quality bearing in mind the peculiarity of the location. Whereas water supply is seen as a national issue, pollution is mainly felt at, and dealt with, at the local level. National governments, with few exceptions, have little information on the relative importance of various types of pollution (agriculture, municipal, industrial, animal husbandry, aquaculture, etc.) and therefore have no notion of which is of greatest economic or public health significance (Abbaspour, 2007). Consequently, it is difficult to develop a strategic water quality management plan or to efficiently focus domestic and donor funds on priority issues as quality surveillance. Our study is one of the few independent reports that attempt to evaluate the concentrations of chlorides and sulphates in drinking water sources in Pakistan with an attempt to provide by surveys, epidemiological linkages to suggest potential health effects from exposure to elevated levels of the chemicals in drinking water.

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MATERIALS AND METHODS

Rahim Yar Khan District has an area of 11,880 square kilometers and comprises four Tehsils, which are Liaqatpur, Khanpur, Rahim Yar Khan, Sadiqabad with a total population of more than 4.73 million in 2011. The district Rahimyarkhan lies between 27.40' - 29.16' N latitudes and 60.45' - 70.01' E longitudes. The climate of the district is hot and dry in the summer and cold and dry in the winter.

Water samples were collected from different water sources (hand pumps, tube wells, canals and public water supply systems) from cities of Sadiqabad, Rahimyarkhan, Khanpur and Liaqatpur during the period of 2010-11. Water quality determinations of sulphate and chloride contents were carried out in chemistry laboratories of Sadiqabad College of Technology Sadiqabad, and Agriculture Department, Punjab Pakistan. Chloride was measured by silver nitrate titration using a chromate indicator, and a chloride ion-selective electrode. Sulphate ion was precipitated in a hydrochloric acid medium with barium chloride to form $BaSO_4$ crystals of uniform size. Light absorbance of the $BaSO_4$ suspension was then measured by nephelometery using a turbidimeter.

Sulphate concentration was extrapolated with the help of a prepared standard curve (15).

With collaborative assistance received from a local non-governmental organization (SAWACO), a health survey was conducted in areas with high values of chlorides and sulfates. Volunteers assisted in the administration of questionnaires among population in polluted areas in 1st quarter of 2012. Results were analyzed to identify any health concerns related to the elevated levels of chloride and sulfates in source waters available for residents each considered community.

RESULTS

A total of one hundred and fifty one samples were analyzed during the study. This consisted of hand pumps (n=88), tube wells (n=54), surface water (Canals) (n=06) and public water supply system (n=03). Out of the 151 samples analysed, 47% has sulphate levels within guideline limits while 53 percent of the samples had values above the limits. The number of samples with sulphate levels within and above guideline values is presented in Fig. 1. Curiously, as in Table 1, sulphate concentrations of a sample was as high as 7760 mg/L for samples collected from hand pumps. Altogether, 6.7%, 18.5% and 25.2% respectively had sulphate values within the range 250-300mg/L, 300-500mg/L and > 500 mg/L respectively. Out of 151 samples analysed, 77 percent had chloride levels within guideline value (Fig 2). For samples that exceeded the guideline values, chloride concentration was relatively low (23%) (Table 2). However, high chloride levels of up to 3190 mg/L were detected in samples from hand pumps. On the whole, 4.0%, 9.3% and 9.9% respectively had sulphate values within the range 250-300mg/L, 300-500mg/L and > 500 mg/L respectively.

Results from the health survey revealed that prolonged exposure to excessive levels of chlorides and sulphates may be attributable to health effects in the sampled population. In areas where consistently higher than guideline values were observed, residents complained of gastrointestinal tract problems such as diarrhea, nausea, inflammatory bowel disease. Almost fifty five percent among survey reported diarrheal symptoms and consequent dehydration. From an analysis of our survey questionnaires, chloride concentrations in excess of about 250 mg/Litre was associated with detectable taste in water. Consumers can, however, become accustomed to concentrations in excess of 250 mg/Litre. Individuals moving into areas with high Sulphate concentrations from areas with low Sulphate concentrations in drinking water complained about health effects such as gastroenteritis. Although it was not possible to screen out the possibility of gastroenteritis resulting from other sources, for example bacterial infection; tourists, hunters and students not normally resident in Rahimyarkhan were generally more affected. Questionnaire response also revealed that water distribution system in the urban area is either un-adequate or has reached its full development. Physical observation revealed that there is no public water supply system in rural area considered in the study neither was there any water treatment plant. Consequently, most of population resolve to the use of groundwater through electric pumps or hand pumps.

Fig.1: Samples (%) with sulphate levels within (-ve) and above (+ve) guideline values

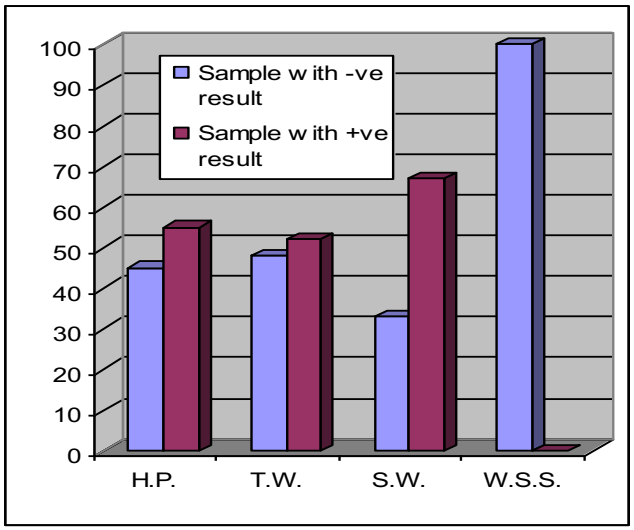

Fig. 2: Samples (%) with chloride levels within (-ve) and above (+ve) guideline values

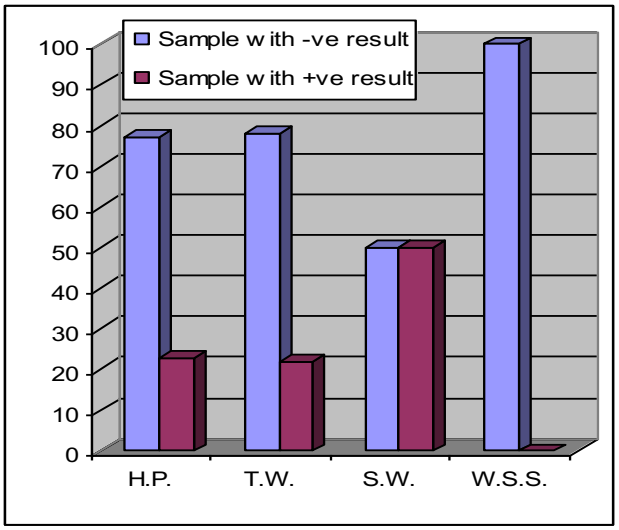

Table 1: Observed sulphate levels of different water samples

Sample source	Sulphate levels within guideline values	Sulphate levels higher than guideline values	Range (Mg/L)
HP	40(45.5%)	48(54.44%)	31.2-7760
TW	26(48.5%)	28(51.85%)	0-1990
SW	02 (33.33%)	04(66.67%)	180-413
WSS	03 (100%)	0	82.3-99.2

Table 2: Observed chloride levels of different water samples

Sample source	Sulphate levels within guideline values	Sulphate levels higher than guideline values	Range (Mg/L)
HP	68(77.27%)	20(22.73%)	14-3190
TW	42(77.78%)	12(22.22%)	18-780
SW	02 (50.00%)	03(50.00%)	35-56
WSS	03(100%)	0	148-405

DISCUSSION

Sulfates occur naturally in drinking water, usually as a combination of sulfur and oxygen. Some minerals present in soil also get dissolved and are ultimately released to groundwater as Sulfates. A number of health concerns regarding sulfate in drinking water have been raised because of reports that diarrhea may be associated with the ingestion of water containing high levels of sulfate. In the current study, high sulfate levels were observed especially in hand-pumps. In most developing countries, major settlements enjoy pipe borne water supply albeit erratic. In the rural communities, bore holes fitted with hand pumps serve as the main source of alternative 'potable' water. In an age where more and more emphases is suggested to be placed on the provision of hand pumps and wells for rural settlements, the results of this study thus gives cause for concern. High sulphate levels in drinking water as observed in the current study may be attributable to relatively shallow depth of wells attached to these pumps and the proximity to resources of pollution from human dwelling and animal yards. One striking observation in support of this assumption was the high sulphate levels from hand pumps on lands close to cattle feed lots and intensive agricultural sites in Punjab where chemical fertilizers were regularly applied.

There may be up to one percent sulfate present in gastric fluids. Normally, the body maintains a homeostasis between absorbed inorganic Sulphate, Sulphate compounds, and renal excretion; membrane transport and regulation contribute to this homeostasis. There have been a number of studies conducted to determine the toxicity of sulphate in humans. Chien et al. presented case reports of diarrhea in three infants exposed to water containing Sulphate (ranging from 630-1,150mg/L)[10]. However, there were other potential causes of the diarrhea in these infants like consuming infant formula with high osmolarity or the presence of microbial pathogens that were not thoroughly addressed by the investigators. Almost fifty five percent among survey reported diarrheal symptoms and consequent dehydration. These are mainly related to sulphate toxicity and due to these above mentioned effects patients having dehydration. Sulfates have a laxative effect that leads to dehydration especially infants are more prone to its effects. But with passage of time, people and young live stocks become acclimated to the sulfate and the symptoms disappear[9].

A survey conducted in North Dakota found a slight increase in the percentage of people (28%) who reported that their drinking water had a laxative effect when the drinking water contained 500 to 1,000 mg/L Sulphate compared to the percentage of people (21%) who reported a laxative effect from drinking water that contained <500 mg/L. Fifty one percent of people who consumed water with 1,000 to 1,500 mg/L reported a laxative effect. Arguably, the generally accepted concern is that which relates to greater risk from the laxative effects of sulfate when vulnerable populations experience an abrupt change from drinking water with low sulfate concentrations to drinking water with high sulfate concentrations. One such potentially sensitive population is infants receiving their first bottles containing tap water, either as water alone or as formula mixed with water. Another group of people who could potentially be adversely affected by water with high Sulphate concentrations are transient populations like tourists, hunters, and other temporary visitors who moves into areas with high Sulphate concentrations in the drinking water from areas with low Sulphate concentrations in drinking water[12].

It is suggested that most people may experience laxative effect when they drank water containing >1000 mg of Sulphate per litre[13,14]. However, like other ones, the current study may not be assertive about a statistically significant association between consumption of water with excessive sulphate levels and clinical syndromes experienced by the surveyed population. The science of sulphate levels in drinking water is itself rocked with inherent questions which still remain answered. Where reported studies suggest that a certain sulfate level would not be likely to cause adverse effects, existing data do not identify the level of sulfate in drinking water that would be unlikely to cause adverse human health effects.

Again with the assumption of acclimatization or adaptation to certain levels of sulphates in drinking water, findings on how long this takes is still yet to be published. Furthermore, in referring to the potential health effects of elevated sulfate levels in drinking water, one is quick to refer to vulnerable populations as being at risk, particularly infants. However, there are no dose-response studies to substantiate this partly because of the difficulty of locating a population of women feeding their infants formula mixed with unfiltered tap water containing high levels of sulfate. Consequently, it appears that there is not enough scientific evidence on which to base a regulation but a mere health advisory in places where drinking water has sulfate levels of >500mg/L, based solely on precautionary principle

Chlorides occur in surface and groundwater as a result of intrusion from both natural and anthropogenic sources, such as run-off containing road de-icing salts, the use of inorganic fertilizers, landfill leachates, septic tank effluents, animal feeds, industrial effluents, irrigation drainage, and seawater intrusion in coastal areas (DNHW, 1978). Available data reveal that the mean chloride concentration in several rivers in the United Kingdom is in the range 11–42mg/litre during 1974–81 (Brooker and Johnson, 1984). Also evidence of a general increase in chloride concentrations in groundwater and drinking-water has been found (WHO, 1978). In developed nations, aquifers prone to seawater intrusion have been found to contain chloride at concentrations ranging from 5 to 460 mg/litre (Phelan, 1987), whereas contaminated wells in developing nations such as the Philippines have been reported to have an average chloride concentration of 141 mg/litre (Morales, 1987). Chloride levels in unpolluted waters are often below 10 mg/litre and sometimes below 1mg/litre (WHO, 1996)

However, high chloride levels of up to 3190 mg/L were detected in samples from hand pumps in our current study. Chloride in surface water and groundwater from both natural and anthropogenic sources, such as extensive use of Potassium fertilizer in which Potassium Chloride is used during production, landfill leachates, septic tank effluent, animal feeds, industrial effluents, and irrigation drainage. High values of Chlorides may also be due to extensive use of Sodium Chloride in production of industrial chemicals such as Caustic Soda, Chlorine, Sodium Chlorite and Sodium hypochlorite. The chloride ion is highly mobile and is transported to nearby watershed and river basins.

Usually, chloride concentrations in excess of about 250 mg/Litre can give rise to detectable taste in water, but the threshold depends upon the associated cations, a typical example being Sodium. The presence of sodium in drinking water is of significant health concerns. Therefore, the US Environmental Protection Agency (EPA) now requires drinking water to be monitored for sodium and public water suppliers are directed to report local health authorities any concentration above 250 mg/L. Chlorides in drinking water usually create taste and odor problems at concentrations exceeding 250 mg/L. In New Hampshire from 1983 to 2003 the NHDOT replaced more than 424 private wells contaminated by road salt at a cost of $3.2 million. Several public water supply wells have also been abandoned due to contamination[1,2]. Although excessive intake of drinking-water containing sodium chloride at concentration above 250mg/L has been reported to produce hypertension[3], this effect is believed to be related to the sodium ion concentration. Consumers may become accustomed to concentrations in excess of 250mg/L.

In humans, 88% of chloride is extracellular and contributes to the osmotic activity of body fluids. A normal adult human body contains approximately 81.7g chloride. On the basis of a total obligatory loss of chloride of approximately 530mg/day, a dietary intake for adults of 9mg of chloride per kg of body weight has been recommended for children up to 18 years of age, a daily dietary intake of 45 mg of chloride should be sufficient[4]. A dose of 1 g of sodium chloride per kg of bodyweight was reported to have been lethal in a 9-week-old child[7]. Chloride toxicity has not been observed in humans except in the special case of impaired sodium chloride metabolism, e.g. in congestive heart failure[8]. Healthy individuals can tolerate the intake of large quantities of chloride provided that associated intake of fresh water. Little is known about the effect of prolonged intake of large amounts of chloride in the diet. As in experimental animals, hypertension associated with sodium chloride intake appears to be related to the sodium rather than the chloride ion[4]. However, adverse effects related to high chloride concentration are increased number of polymorhonuclear leukocyte and disturbed blood cell counts in full blood count analysis.

CONCLUSION

On a conclusive note, the current study revealed that higher than guideline levels of consumers of sulfates and chlorides in available drinking water in Rahimyarkhan. Consumers can however, become accustomed to concentrations in excess of 250mg/Litre. Individuals moving into areas with high Sulphate concentrations from areas with low Sulphate concentrations in drinking water complained about health effects such as gastroenteritis. Although

it was not possible to screen out the possibility of gastroenteritis resulting from other sources, for example bacterial infection; tourists, hunters and students not normally resident in Rahim Yar Khan were generally more affected. It is thus suggested that efforts be made to provide at least one laboratory in each city working in collaboration with health officials in district hospitals.

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WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 10

DRINKINGWATER AND HUMAN HEALTH

POSTED ONAUGUST 23, 2019 BY DRINKING-WATER

Drinking Water Contaminant – Sulfur, hydrogen sulfide

Contents

Sources of sulfate and hydrogen sulfide in drinking water

Sulfates are a combination of sulfur and oxygen and are a part of naturally occurring minerals in some soil and rock formations that contain groundwater. The mineral dissolves over time and is released into groundwater. Sulfate minerals can cause scale buildup in water pipes similar to other minerals and may be associated with a bitter taste in water that can have a laxative effect on humans and young livestock. Sulfate can make cleaning clothes difficult.

Hydrogen sulfide gas also occurs naturally in some groundwater. It is formed from decomposing underground deposits of organic matter, such as decaying plant material. It is found in deep or shallow wells and also can enter surface water through springs, although it quickly escapes to the atmosphere. Hydrogen sulfide often is present in wells drilled in shale or sandstone, or near coal or peat deposits or

oil fields. Sulfur-reducing bacteria, which use sulfur as an energy source, are the primary producers of large quantities of hydrogen sulfide. These bacteria chemically change natural sulfates in water to hydrogen sulfide. Sulfur-reducing bacteria live in oxygen-deficient environments such as deep wells, plumbing systems, water softeners, and water heaters. These bacteria can flourish on the hot water side of a water distribution system. Hydrogen sulfide gas produces an offensive “rotten egg” or “sulfur water” odor and taste in the water. In some cases, the odor may be noticeable only when the water is initially turned on or when hot water is run. Heat forces the gas into the air, which may cause the odor to be especially offensive in a shower. A nuisance associated with hydrogen sulfide includes its corrosiveness to metals such as iron, steel, copper and brass. It can tarnish silverware and discolor copper and brass utensils. Hydrogen sulfide also can cause yellow or black stains on kitchen and bathroom fixtures. Coffee, tea and other beverages made with water containing hydrogen sulfide may be discolored, and the appearance and taste of cooked foods can be affected.

Occasionally, a hot water heater is a source of hydrogen sulfide odor. The magnesium corrosion control rod present in many hot water heaters can chemically reduce naturally occurring sulfates to hydrogen sulfide.

A problem that can result from sulfate in water is sulfur-oxidizing bacteria. These nonpathogenic (not health-threatening) bacteria convert sulfide into sulfate, producing a dark slime that can clog plumbing and/or stain clothing. Blackening of water or dark slime coating the inside of toilet tanks may indicate a sulfur-oxidizing bacteria problem. Sulfur-oxidizing bacteria are less common than sulfur-reducing bacteria.

Potential health effects of sulfate and hydrogen sulfide in drinking water

Sulfate may have a laxative effect that can lead to dehydration and is of special concern for infants. With time, most individuals will become acclimated to the sulfate and the symptoms disappear.

Hydrogen sulfide is flammable and poisonous. Usually it is not a health risk at concentrations present in household water. Atmospheric hydrogen sulfide concentrations can be elevated when water with hydrogen sulfide is released into confined areas.

Sulfur-oxidizing bacteria pose no known human health risk. Sulfur-reducing bacteria pose no known health risk.

Testing for sulfate and hydrogen sulfide in drinking water

The quality of water supplied by public water systems is regulated by the U.S. Environmental Protection Agency (EPA). Sulfate is classified under the Secondary Maximum Contaminant Level standards, which are based on aesthetic factors such as taste, odor, and staining properties of water, rather than health effects. The standard in drinking water for sulfate is 250 milligrams per liter (mg/l), sometimes expressed as 250 parts per million (ppm). Secondary standards and guidelines and are not enforced.

Hydrogen sulfide is not regulated by the EPA. A concentration high enough to be a drinking water health hazard also makes the water unpalatable. The odor of water with as little as 0.5 ppm of hydrogen sulfide concentration is detectable by most people. Concentrations less than 1 ppm give the water a “musty” or “swampy” odor. A 1-2 ppm hydrogen sulfide concentration gives water a “rotten egg” odor and makes the water very offensive.

Consumers of private drinking water can have water tested for sulfate through laboratory analysis. The rotten-egg odor of hydrogen sulfide gas generally makes testing unnecessary. In addition, the gas readily dissipates when water is exposed to the atmosphere.

Options for sulfate and hydrogen sulfide in drinking water

Secondary standards for drinking water contaminants are established as guides to manage aesthetic properties of water. Drinking water suppliers are not required by federal law to meet these secondary standards. If sulfate levels in drinking water approach or exceed the standard, some public water suppliers voluntarily reduce or remove sulfate from the water.

If excessive sulfate or hydrogen sulfide is present in private drinking water, consumers can obtain an alternate water supply or use some type of treatment to remove the impurity.

It may be possible to obtain a satisfactory alternate water supply by drilling a new well in a different location or a deeper well in a different aquifer. Another alternate source is bottled water that can be purchased in stores or direct from bottling companies. This alternative might be considered when the primary concern is water for food preparation and drinking. Several methods of removing sulfate from water are available. The treatment method selected depends on many factors including the level of sulfate in the water, the amount of iron and manganese in the water, and if bacterial contamination also must be treated. The best option also depends on how much treated water is needed.

Options for treating small quantities of water with sulfate include distillation and reverse osmosis. The most common method of treating large quantities of water is ion exchange.

Hydrogen sulfide formation may be reduced in some instances. Performing a shock chlorination procedure may reduce, but does not eliminate, the sulfur reducing bacteria.

If hydrogen sulfide odor is associated primarily with the hot water system, a hot water heater modification may reduce the odor. Replacing the water heater's magnesium corrosion control rod with one made of aluminum or another metal may improve the situation.

Depending on the concentration, hydrogen sulfide may be removed with activated carbon filters, oxidizing filters, or chemical oxidation and filtration.

EXTENSION FOUNDATION

COOPERATIVE EXTENSION

USDA National Institute of Food and Agriculture
U.S. DEPARTMENT OF AGRICULTURE

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 11

Comparison of State Water Guidance and Federal Drinking Water Standards

December 2021

The information below is intended to assist with evaluation of levels of drinking water contaminants.

An Excel table providing the Comparison of State Water Guidance and Federal Drinking Water Standards (Excel) (http://www.health.state.mn.us/communities/environment/risk/docs/guidance/waterguidance.xlsx) is also available for download.

For questions or more information, please contact Health Risk Assessment Unit (http://www.health.state.mn.us/communities/environment/risk/contact.html) .

A | B | C | D | E | F | G | H | I | J | K | L
M | N | O | P | Q | R | S | T | U | V | W | X | Y | Z

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Acenaphthene 83-32-9			2000	DWEL	100	HRL18	Chronic
Acetaminophen 103-90-2					200	HRL15	Acute
Acetochlor 34256-82-1					20	HRL18	Chronic
Acetochlor ESA 187022-11-3					300	HRL18	Chronic
Acetochlor OXA 184992-44-4					90	HRL18	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Acetone 67-64-1					4000	HRL11	Chronic
Acetone 67-64-1					3000	HBV20	Chronic
Acetyl-1,1,2,4,4,7 hexamethyltetraline (AHTN), 6- 21145-77-7 or 1506-02-1					20	HRL13	Chronic
Acifluorfen (sodium) 62476-59-9			100	cancer			
Acrylamide 79-06-1	TT	zero	70	DWEL	0.2	HRL15	Cancer
Acrylonitrile 107-13-1			6	cancer			
Alachlor 15972-60-8	2	zero	40	cancer	9	HRL18	Chronic
Alachlor ESA 142363-53-9					50	RAA16	Chronic
Alachlor OXA 171262-17-2					50	RAA16	Chronic
Aldicarb 116-06-3	3	1	7	life-time	1	HRL93	Chronic
Aldicarb sulfone 1646-88-4	2	1	7	life-time			
Aldicarb sulfoxide 1646-87-3	4	1	7	life-time			
Aldrin 309-00-2			0.2	cancer			
Allyl Chloride 107-05-1					30	HRL94	Chronic
Ametryn 834-12-8			60	life-time			
Aminomethylphosphonic acid (AMPA) 1066-51-9					1000	HBV17	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Ammonia 7664-41-7			30000	life-time			
Ammonium sulfamate 7773-06-0			2000	life-time			
Anatoxin-a 64285-06-9					0.1	RAA16	Short Term
Anthracene 120-12-7			10000	DWEL	2000	HRL93	Chronic
Anthracene 120-12-7			10000	DWEL	600	RAA19	Chronic
Antimony 7440-36-0	6	6	6	life-time	6	HRL93	Chronic
Arsenic 7440-38-2	10	zero	2	cancer			
Asbestos (fibers/l >10Fm length) 1332-21-4	7 MFL	7 MFL	700 MFL	cancer			
Atrazine 1912-24-9	3	3	700	DWEL	3	HRLMCL	Chronic
Barium 7440-39-3	2000	2000	700	1 day	2000	HRL93	Chronic
Baygon 114-26-1			3	life-time			
Bentazon 25057-89-0			200	life-time	30	HRL15	Chronic
Benz[a]anthracene (PAH) 56-55-3							
Benzene 71-43-2	5	zero	3	life-time	2	HRL09	Cancer
Benzo[a]pyrene 50-32-8	0.2	zero	0.5	cancer	0.1	HBV20	Cancer
Benzo[b]fluoranthene (PAH) 205-99-2							

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Benzo[g,h,i]perylene (PAH) 191-24-2							
Benzo[k]fluoranthene (PAH) 207-08-9							
Benzoic Acid 65-85-0					30000	HRL93	Chronic
Benzophenone 119-61-9					100	HBV20	Chronic
Benzotriazole, -1H 95-14-7					20	HBV20	Short Term
Benzotriazole, methyl-1H 29385-43-1					20	RAA19	Short Term
Benzotriazole, 5-methyl-1H 136-85-6					20	RAA19	Short Term
Beryllium 7440-41-7	4	4	70	DWEL	0.08	HRL93	Cancer
Beta particle and photon activity (formerly man-made radionuclides)	4 mrem/yr	zero	4 mrem/yr	cancer			
Biphenyl, 1,1'- (Diphenyl) 92-52-4					300	HRL93	Chronic
Biphenyl, 1,1'- (Diphenyl) 92-52-4					10	HBV21	Cancer
Bis(2-chloro-1-methylethyl) ether 108-60-1			300	life-time			
Bis(2-chloroethyl) ether 111-44-4					0.3	HRL93	Cancer
Bis(2-chloromethyl) ether 542-88-1					0.002	HRL93	Cancer
Bisphenol A 80-05-7					20	HRL15	Subchronic
Boron 7440-42-8			3000	1 day	500	RAA17	Short Term

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Bromacil 314-40-9			70	life-time			
Bromate 7789-38-0	10	zero	5	cancer			
Bromobenzene 108-86-1			60	life-time			
Bromochloromethane 74-97-5			90	life-time			
Bromodichloromethane 75-27-4	80	zero	100	cancer	6	HRL93	Cancer
Bromodichloromethane 75-27-4	80	zero	100	cancer	3	HBV20	Cancer
Bromoform 75-25-2	80	zero	200	10 day	40	HRL93	Cancer
Bromomethane 74-83-9			10	life-time	10	HRL93	Chronic
Butanol, 1- 71-36-3					700	HRL93	Chronic
Butyl benzyl phthalate 85-68-7			7000	DWEL	100	HRL15	Acute
Butylate 2008-41-5			400	life-time			
Butylphthalyl butylglycolate 85-70-1					7000	HRL93	Chronic
Cadmium 7440-43-9	5	5	5	life-time	0.5	HRL15	Chronic
Carbamazepine 298-46-4					40	HRL13	Acute
Carbaryl 63-25-2			400	DWEL			
Carbofuran 1563-66-2	40	40					

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Carbon Disulfide 75-15-0					700	HRL93	Chronic
Carbon tetrachloride 56-23-5	5	zero	30	life-time	1	HRL13	Cancer
Carboxin 5234-68-4			700	life-time			
Chloramben 133-90-4			100	life-time	100	HRL94	Chronic
Chloramine 10599-90-3	4000	4000	3000	life-time			
Chlordane 12798-03-6	2	zero	4	life-time			
Chlorine 7782-50-5	4000	4000	3000	1 day			
Chlorine dioxide 10049-04-4	800	800	800	1 day			
Chlorite 7758-19-2	1000	800	800	1 day			
Chlorobenzene 108-90-7	100	100	100	life-time	100	HRL93	Chronic
Chloroethane 75-00-3						RAA16	
Chloroform 67-66-3	80	70	70	life-time	20	HRL18	Short Term
Chloromethane 74-87-3			400	10 day			
Chlorophenol, 2- 95-57-8			40	life-time	30	HRL93	Chronic
Chlorothalonil 1897-45-6			150	cancer	30	HRL94	Cancer
Chlorotoluene o- 95-49-8			100	life-time			

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Chlorotoluene p- 106-43-4			100	life-time			
Chlorpyrifos 2921-88-2			2	life-time	0.6	HBV13	Short Term
Chlorpyrifos oxon 5598-15-2					0.4	RAA13	Short Term
Chromium (total) 7440-47-3	100	100	100	DWEL			
Chromium III 16065-83-1					20000	HRL94	Chronic
Chromium VI 18540-29-9					100	HRL93	Chronic
Chrysene (PAH) 218-01-9							
Clothianidin 210880-92-5 or 205510-53-8					200	HRL18	Short Term
Copper (at tap) 7440-50-8	TT - action level of 1.3 mg/L	1300					
Cumene (Isopropyl benzene) 98-82-8					300	HRL93	Chronic
Cyanazine 21725-46-2			1	life-time	1	HRL18	Chronic
Cyanazine acid (CAC) (degradate of Cyanazine) 36576-43-9					1	RAA20	Chronic
Cyanazine amide (CAM) (degradate of Cyanazine) 36576-42-8					1	RAA20	Chronic
Cyanide 143-33-9	200	200	200	1 day			
Cyanide, free 57-12-5					100	HRL93	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Cyanogen chloride 506-77-4			50	1 day			
Cylindrospermopsin 143545-90-8			0.7	10 day			
Dimethyl tetrachloroterephthalate (DCPA or Dacthal) 1861-32-1			70	life-time			
Dalapon (sodium salt) 75-99-0	200	200	200	life-time			
Deethylatrazine (DEA) (degradate of Atrazine) 6190-65-4					3	RAA20	Chronic
Deethylcyanazine (DEC) (degradate of Cyanazine) 21725-40-6					1	RAA20	Chronic
Deethylcyanazine acid (DCAC) (degradate of Cyanazine) 36749-35-6					1	RAA20	Chronic
Deethylcyanazine amide (DCAM) (degradate of Cyanazine) 36556-77-1					1	RAA20	Chronic
Deethyldeisopropylatrazine (DACT, DEDI, DDA) (degradate of Atrazine and Cyanazine) 3397-62-4					1	RAA20	Chronic
Deisopropylatrazine (DIA) (degradate of Atrazine and Cyanazine) 1007-28-9					1	RAA20	Chronic
Desvenlafaxine 93413-62-8; 300827-87-6; 386750-22-7; 93414-04-1					20	HBV15	Short Term
Desvenlafaxine - succinate salt 386750-22-7					20	HBV15	Short Term

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Di(2-ethylhexyl)adipate 103-23-1	400	400	400	life-time			
Di(2-ethylhexyl)phthalate 117-81-7	6	zero	300	cancer	7	HRL15	Cancer
Diazinon 333-41-5			1	life-time			
Dibromochloromethane 124-48-1	80	60	60	life-time	10	HRL93	Chronic
Dibromochloropropane (DBCP) 96-12-8	0.2	zero	3	cancer			
Dibromoethane, 1,2- (Ethylene dibromide (EDB)) 106-93-4	0.05	zero	2	cancer	0.004	HRL93	Cancer
Dibutyl phthalate 84-74-2			4000	DWEL	20	HRL15	Acute
Dicamba 1918-00-9			4000	life-time	200	HRL93	Chronic
Dichloroacetic acid 79-43-6	60	zero	30	life-time			
Dichlorobenzene 541-73-1			600	life-time			
Dichlorobenzene, 1,2- 95-50-1	600	600	600	life-time	600	HRL93	Chronic
Dichlorobenzene, 1,4- 106-46-7	75	75	75	life-time	10	HRL94	Cancer
Dichlorobenzene, 1,4- 106-46-7	75	75	75	life-time	50	HBV20	Short Term
Dichlorobenzidine, 3,3'- 91-94-1					0.8	HRL93	Cancer
Dichlorodifluoromethane 75-71-8			1000	life-time	700	HRL11	Chronic
Dichlorodifluoromethane 75-71-8			1000	life-time	500	RAA17	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Dichlorodiphenyldichloroethane, p,p'- (DDD) 72-54-8					1	HRL93	Cancer
p,p'-Dichlorodiphenyldichloroethylene, (DDE) 72-55-9					1	HRL93	Cancer
Dichlorodiphenyltrichloroethane, p,p'-(DDT) 50-29-3					1	HRL93	Cancer
Dichloroethane, 1,1- 75-34-3					80	RAA16	Chronic
Dichloroethane (EDC), 1,2- 107-06-2	5	zero	40	cancer	1	HRL13	Cancer
Dichloroethylene, 1,1- 75-35-4	7	7	6	cancer	200	HRL11	Subchronic
Dichloroethylene, 1,1- 75-35-4	7	7	6	cancer	200	HBV20	Subchronic
Dichloroethylene (cis-1,2-) 156-59-2	70	70	10	life-time	6	HRL18	Chronic
Dichloroethylene (trans-1,2-) 156-60-5	100	100	100	life-time	40	HRL13	Chronic
Dichloroethylene (trans-1,2-) 156-60-5	100	100	100	life-time	9	HBV20	Chronic
Dichlorofluoromethane 75-43-4					20	RAA17	Short Term
Dichloromethane 75-09-2	5	zero	200	life-time	5	HRLMCL	Chronic
Dichlorophenol, 2,4- 120-83-2			20	life-time	20	HRL93	Chronic
Dichlorophenoxyacetic acid, 2,4-(2,4-D) 94-75-7	70	70	200	DWEL	30	HRL18	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Dichloropropane, 1,2- 78-87-5	5	zero	60	cancer	5	HRL94	Cancer
Dichloropropane, 1,2- 78-87-5	5	zero	60	cancer	3	HBV21	Cancer
Dichloropropene, 1,3- 542-75-6			30	1 day	2	HRL94	Cancer
Dieldrin 60-57-1			0.2	cancer	0.006	HRL18	Cancer
Diethyl phthalate 84-66-2			30000	DWEL	6000	HRL93	Chronic
Diethyl-meta-toluamide (DEET), N,N- 134-62-3					200	HRL13	Short Term
Diisopropylmethylphosphonate 1445-75-6			600	life-time			
Dimethenamid 87674-68-8					300	HRL15	Chronic
Dimethenamid Ethanesulfonic acid (ESA) 205939-58-8					300	RAA13	Chronic
Dimethenamid Oxanilic acid (OXA) 380412-59-9					300	RAA13	Chronic
Dimethenamid-p 163515-14-8					300	HRL15	Chronic
Dimethrin 70-38-2			2000	life-time			
Dimethyl methylphosphonate 756-79-6			100	life-time			
Dimethyl phthalate 131-11-3					70000	HRL94	Chronic
Dimethyl tetrachloroterephthalate (DCPA or Dacthal) 1861-32-1			70	life-time			

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Dimethylphenol, 2,4- 105-67-9					100	HRL93	Chronic
Dinitrobenzene,1,3- 99-65-0			1	life-time			
Dinitrophenol, 2,4- 51-28-5					10	HRL93	Chronic
Dinitrotoluene, 2,4- 121-14-2			5	cancer			
Dinitrotoluene, 2,6- 606-20-2			5	cancer			
Dinoseb 88-85-7	7	7	7	life-time	8	HRL18	Short Term
Dioxane, 1,4- 123-91-1			35	cancer	1	HRL13	Cancer
Diphenamid 957-51-7			200	life-time			
Diquat 85-00-7	20	20	20	DWEL			
Disulfoton 298-04-4			0.7	life-time	0.3	HRL94	Chronic
Dithiane (1,4-) 505-29-3			80	life-time			
Diuron 330-54-1			100	DWEL			
Endothall 145-73-3	100	100	50	life-time			
Endrin 72-20-8	2	2	2	life-time			
Epichlorohydrin 106-89-8	TT2	zero	70	DWEL			
17α-Ethinylestradiol 57-63-6					0.0002	HBV20	Subchronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Ethyl dipropylthiocarbamate, S-(EPTC) 759-94-4					40	HRL18	Chronic
Ethyl ether 60-29-7					200	RAA16	Chronic
Ethylbenzene 100-41-4	700	700	700	life-time	50	HRL11	Short Term
Ethylbenzene 100-41-4	700	700	700	life-time	40	HBV20	Short Term
Ethylene glycol 107-21-1			6000	10 day	2000	HRL11	Subchronic
Ethylene glycol 107-21-1			6000	10 day	2000	HBV20	Subchronic
Ethylene Thiourea (ETU) 96-45-7			7	DWEL			
Fenamiphos 22224-92-6			0.7	life-time			
Fluometuron 2164-17-2			90	life-time			
Fluoranthene 206-44-0					70	HRL18	Chronic
Fluorene 86-73-7			1000	DWEL	300	HRL93	Chronic
Fluorene 86-73-7			1000	DWEL	80	HBV20	Chronic
Fluoride 7681-49-4	4000	4000					
Fomesafen 72178-02-0					20	HBV20	Chronic
Fonofos 944-22-9			10	life-time			
Formaldehyde 50-00-0			1000	life-time	1000	HRL94	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Glyphosate 1071-83-6	700	700	20000	1 day	500	HBV17	Chronic
Gross alpha particle activity	15 pCi/L	zero	15 pCi/L	cancer			
Heptachlor 76-44-8	0.4	zero	0.8	cancer	0.08	HRL93	Cancer
Heptachlor epoxide 1024-57-3	0.2	zero	0.4	cancer	0.04	HRL93	Cancer
Hexachlorobenzene 118-74-1	1	zero	2	cancer	0.2	HRL93	Cancer
Hexachlorobutadiene 87-68-3			10	DWEL	1	HRL93	Chronic
Hexachlorocyclopentadiene 77-47-4	50	50	200	DWEL			
Hexachloroethane 67-72-1			1	life-time			
Hexane, n- 110-54-3			4000	10 day	400	HRL94	Chronic
Hexazinone 51235-04-2			400	life-time			
HMX 2691-41-0			400	life-time			
Imidacloprid 138261-41-3					2	HBV20	Short Term
Indeno[1,2,3,-c,d]pyrene (PAH) 193-39-5							
Isobutanol 78-83-1					300	HBV16	Chronic
Isophorone 78-59-1			100	life-time	100	HRL93	Chronic
Isopropyl methylphosphonate 1832-54-8			700	life-time			
Isopropylbenzene (cumene) 98-82-8			4000	DWEL	300	HRL93	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Lead (at tap) 7439-92-1	TT action level is 0.015 mg/L	zero					
Lead (at tap) 7439-92-1	0.1 for allergic dermatitis	100					
Lindane 58-89-9	0.2	0.2	200	DWEL			
Linuron 330-55-2					1	HRL93	Chronic
Malathion 121-75-5			200	1 day			
Maleic hydrazide 123-33-1			4000	life-time			
Manganese 7439-96-5			300	life-time	100	HRL93	Chronic
Manganese 7439-96-5			300	life-time	100	HBV20	Short Term
Mercury (inorganic) 7487-94-7	2	2	2	1 day			
Mestranol 72-33-3					0.0002	RAA16	Subchronic
MCPA (2-Methyl-4-chlorophenoxyacetic acid) 94-74-6			30	life-time	3	HRL93	Chronic
Methanol 67-56-1					3000	HRL94	Chronic
Methomyl 16752-77-5			200	life-time			
Methoxychlor 72-43-5	40	40	40	life-time			
Methyl ethyl ketone 78-93-3			4000	life-time	4000	HRL94	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Methylene chloride (see Dichloromethane) 75-09-2							
Methyl isobutyl ketone 108-10-1					300	HRL94	Chronic
Methyl parathion 298-00-0			1	life-time			
Methyl tertiary butyl ether (MTBE) 1634-04-4					60	RAA13	Cancer
Methylnaphthalene, 2- 91-57-6					8	RAA13	Chronic
Methylphenol, 2- 95-48-7					30	HRL93	Chronic
Methylphenol, 3- 108-39-4					30	HRL93	Chronic
Methylphenol, 4- 106-44-5					3	HRL94	Chronic
Metolachlor 51218-45-2			700	life-time	300	HRL11	Subchronic
Metolachlor 51218-45-2			700	life-time	300	HBV20	Short Term
s-Metolachlor 87392-12-9					300	HRL11	Subchronic
s-Metolachlor 87392-12-9					300	HBV20	Short Term
Metolachlor ESA 171118-09-5					800	HRL11	Chronic
Metolachlor ESA 171118-09-5					1,000	HBV20	Chronic
Metolachlor OXA 152019-73-3					800	HRL11	Chronic
Metolachlor OXA 152019-73-3					1,000	HBV20	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Metribuzin 21087-64-9			70	life-time	10	HRL13	Short Term
Metribuzin DA 35045-02-4					10	RAA12	Short Term
Metribuzin DADK 52236-30-3					10	RAA12	Short Term
Metribuzin DK 56507-37-0					10	RAA12	Short Term
Microcystin-LR 101043-37-2			0.3	10 day	0.1	HBV15	Short Term
Molybdenum 7439-98-7			40	life-time			
Monochloroacetic acid 79-11-8	60	70	70	life-time			
Naphthalene 91-20-3			100	life-time	70	HRL13	Acute
Nickel 7440-02-0			100	life-time	100	HRL93	Chronic
Nitrate (as N) 14797-55-8	10000	10000	10000	1 day	10000	HRLMCL	Acute
Nitrite (as N) 14797-65-0	1000	1000	1000	1 day			
Nitroguanidine 556-88-7			700	life-time			
Nitrophenol p- 100-02-7			60	life-time			
N-Nitrosodimethylamine (NDMA) 62-75-9					0.005	HBV17	Cancer
Nitrosodiphenylamine, N- 86-30-6					70	HRL93	Cancer
Nonylphenol, p- 84852-15-3					20	HBV20	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Octylphenol, 4-tert 140-66-9					100	HBV20	Short Term
Oxamyl (Vydate) 23135-22-0	200	200	10	1 day			
Paraquat 1910-42-5			30	life-time			
Pentachlorophenol 87-86-5	1	zero	9	cancer	0.3	HRL15	Cancer
Perchlorate 14797-73-0			15	life-time			
Perfluorobutane sulfonate (PFBS) 45187-15-3; 375-73-5					7	HRL11	Chronic
Perfluorobutane sulfonate (PFBS) 45187-15-3; 375-73-5					2	HBV20	Chronic
Perfluorobutyrate (PFBA) 45048-62-2; 375-22-4					7	HRL18	Short Term
Perfluorohexane sulfonate (PFHxS) 108427-53-8; 355-46-4; 3871-99-6					0.047	HBV20	Short Term
Perfluorohexanoate (PFHxA) 92612-52-7; 307-24-4; 21615-47-4; 2923-26-4					0.2	HBV21	Short Term
Perfluorooctanoic Acid (PFOA) and salts 45285-51-6; 335-67-1; 335-66-0; 3825-26-1; 2395-00-8; 335-93-3; 335-95-5			0.07	life-time	0.035	HRL18	Short Term
Perfluorooctane Sulfonate (PFOS) and salts 45298-90-6; 1763-23-1; 29081-56-9; 70225-14-8; 2795-39-3; 29457-72-5			0.07	life-time	0.3	HRL09	Chronic

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Perfluorooctane Sulfonate (PFOS) and salts 45298-90-6; 1763-23-1; 29081-56-9; 70225-14-8; 2795-39-3; 29457-72-5			0.07	life-time	0.015	HBV20	Short Term
Phenanthrene (PAH) 85-01-8							
Phenol 108-95-2			2000	life-time	4000	HRL93	Chronic
Picloram 1918-02-1	500	500	700	DWEL	500	HRL93	Chronic
Polychlorinated biphenyls (PCBs) 1336-36-3	0.5	zero	10	cancer	0.04	HRL94	Cancer
Prometon 1610-18-0			200	1 day	100	HRL93	Chronic
Pronamide 23950-58-5			100	cancer			
Propachlor 1918-16-7			100	cancer	90	HRL93	Chronic
Propazine 139-40-2			10	life-time			
Propham 122-42-9			100	life-time			
Pyraclostrobin 175013-18-0					100	HBV16	Short Term
Pyrene 129-00-0					50	HRL18	Chronic
Quinoline 91-22-5					0.03	HBV20	Cancer
Radium 226 & 228 (combined) 7440-14-4	5 pCi/L	zero					
RDX 21-82-4			2	life-time			

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Selenium 7782-49-2	50	50	50	life-time	30	HRL93	Chronic
Silver 7440-22-4			100	life-time	30	HRL93	Chronic
Simazine 122-34-9	4	4	700	DWEL	4	HRLMCL	Chronic
Strontium 7440-24-6			4000	life-time	3000	RAA19	Short Term
Styrene 100-42-5	100	100	100	life-time			
Sulfamethazine 57-68-1					100	HRL15	Short Term
Sulfamethoxazole 723-46-6					100	RAA13	Short Term
Sulfamethazine, sodium salt 1981-58-4					100	HBV13	Short Term
TCDD, 2,3,7,8- (Dioxin) 1746-01-6	0.00003	zero	0.00002	cancer			
Tebuthiuron 34014-18-1			500	life-time			
Terbacil 5902-51-2			90	life-time			
Terbufos 13071-79-9			0.4	life-time			
Tetrachloroethane, 1,1,1,2- 630-20-6			70	life-time	70	HRL93	Chronic
Tetrachloroethane, 1,1,2,2- 79-34-5			40	cancer	2	HRL94	Cancer
Tetrachloroethylene 127-18-4	5	zero	10	life-time	5	HRLMCL	Chronic
Tetrachloroethylene 127-18-4	5	zero	10	life-time	4	HBV21	Cancer

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Tetrachloroterephthalic acid 2136-79-0			100000	1 day			
Tetrahydrofuran 109-99-9					600	HRL18	Short Term
Thallium 7440-28-0	2	0.5	7	1 day	0.6	HRL94	Chronic
Thiamethoxam 153719-23-4					200	HRL18	Subchronic
Tin 7440-31-5					4000	HRL94	Chronic
Toluene 108-88-3	1000	1000	2000	10 day	200	HRL11	Short Term
Toluene 108-88-3	1000	1000	2000	10 day	70	HBV20	Short Team
Toxaphene 8001-35-2	3	zero	3	cancer	0.3	HRL93	Cancer
Trichloroacetic acid 76-03-9	60	20	20	life-time			
Trichlorobenzene, 1,2,4- 120-82-1	70	70	70	life-time	4	HRL13	Cancer
Trichlorobenzene, 1,3,5- 108-70-3			40	life-time	4	RAA12	Cancer
Trichloroethane, 1,1,1- 71-55-6	200	200	40000	10 day	5000	HRL18	Chronic
Trichloroethane, 1,1,2- 79-00-5	5	3	3	life-time	3	HRL93	Chronic
Trichloroethylene 79-01-6	5	zero	200	DWEL	0.4	HRL15	Short Term
Trichlorofluoromethane 75-69-4			2000	life-time	2000	HRL93	Chronic
Trichlorophenol, 2,4,6- 88-06-2			10	DWEL	30	HRL93	Cancer

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Trichlorophenoxyacetic acid, 2,4,5- 93-76-5			70	life-time	70	HRL93	Chronic
2(2,4,5-trichlorophenoxy) propionic acid , (2,4,5-TP or Silvex) 93-72-1	50	50	50	life-time	50	HRLMCL	Chronic
Trichloropropane,1,2,3- 96-18-4			100	DWEL	0.003	HRL13	Cancer
Trichloro-1,2,2-trifluoroethane, 1,1,2- 76-13-1					200000	HRL93	Chronic
Triclocarban 101-20-2					100	RAA13	Chronic
Triclosan 3380-34-5					50	HRL15	Short Term
Trifluralin 1582-09-8			10	life-time			
Trimethylbenzene, 1,2,3- 526-73-8					30	HBV20	Short Term
Trimethylbenzene, 1,2,4- 95-63-6					30	HBV20	Short Term
Trimethylbenzene, 1,3,5- 108-67-8			10000	1 day	100	HRL09	Short Term
Trimethylbenzene, 1,3,5- 108-67-8			10000	1 day	30	HBV20	Short Term
Trinitrobenzene, 1,3,5- 99-35-4					0.3	HRL93	Chronic
Trinitroglycerol 55-63-0			5	1 day			
Trinitrotoluene, 2,4,6- 118-96-7			2	life-time			
Tris(2-butoxyethyl) phosphate (TBEP) 78-51-3					30	HBV20	Short Term

Chemical Name CAS Number (if available)	EPA MCL (µg/L)	EPA MCLG (µg/L)	Lowest EPA Health Advisory (µg/L)	Type of HA value	Lowest MDH Value (µg/L)	Type and date of MDH value	Duration of Exposure
Tris(2-chloroethyl) phosphate (TCEP) 115-96-8					5	HRL13	Cancer
Tris(1,3-dichloroisopropyl) phosphate (TDCPP) 13674-87-8					0.8	HBV21	Cancer
Uranium 7440-61-1	30	zero	20	DWEL			
Vanadium 7440-62-2					50	HRL94	Chronic
Venlafaxine - free base 93413-69-5					10	HBV15	Short Term
Venlafaxine - HCl salt 99300-78-4					10	HBV15	Short Term
Vinyl chloride 75-01-4	2	zero	2	cancer	0.2	HRL18	Cancer
White phosphorous 7723-14-0			0.1	life-time			
Xylenes 1330-20-7	10000	10000	7000	DWEL	300	HRL11	Short Term
Xylenes 1330-20-7	10000	10000	7000	DWEL	300	HBV20	Short Term
Zinc 7440-66-6			2000	life-time	2000	HRL94	Chronic

* The 30-day (Short-term duration) exceedance for the following contaminants are a special concern. Click on the links for more detail: Acrylamide (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#acryla) ; Benzene (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#benze) ; Benzo[a]pyrene (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#benzo) ; Cadmium (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#cd) ; Carbon tetrachloride

(http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#carbontet) ; cis-1,2-Dichloroethylene (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#dichlorohbv) ; Di(2-ethylhexyl) phthalate (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#dieh) ; Pentachlorophenol (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html#pcp) .

[1]EPA's incremental cancer risk of 10^{-4} has been converted to MDH's risk of 10^{-5}

[2]Treatment technology

[3]Total for trihalomethanes is 0.08 mg/L

[4]EPA MCL is for total chromium, using an RfD for Cr VI, MDH guidance is for chromium VI alone

[5]Secondary drinking water regulations

[6] See guidance for Chloroethane

Abbreviations

EPA - Environmental Protection Agency

DWEL - EPA Drinking Water Equivalent Level

HBV- MDH Health-Based Value

HRL - MDH Health Risk Limit

MCL - Maximum Contaminant Level

MCL HRL - EPA's MCL adopted into MDH HRL rule

RAA - MDH Risk Assessment Advice

Links

2018 Edition of the Drinking Water Standards and Health Advisories (PDF) (https://semspub.epa.gov/work/HQ/100002014.pdf)
Environmental Protection Agency
Health-Based Guidance for Water (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/index.html)
Human Health-Based Water Guidance Table (http://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html)

Note: *The table above and the accompanying spreadsheet are provided for convenience to compare Minnesota guidance with available federal standards or guidance. Though we update the table regularly, MDH does not guarantee that EPA values are current at any given time. Please reference National Primary Drinking Water Regulations (https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations) for current EPA standards and guidance.*

Updated Monday, 24-Jan-2022 04:56:55 CST

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 12

Health Based Guidance for Water
Health Risk Assessment Unit, Environmental Health Division
651-201-4899

Web Publication Date: August 2020

Toxicological Summary for: Perfluorooctane sulfonate

CAS: 45298-90-6 (anion)
1763-23-1 (acid)
29081-56-9 (ammonium salt)
70225-14-8 (diethanolamine salt)
2795-39-3 (potassium salt)
29457-72-5 (lithium salt)

Synonyms: PFOS, Perfluorooctane sulfonic acid

MDH conducted a focused re-evaluation that used three recent state and federal comprehensive reviews (ATSDR 2018, New Jersey DWQI 2017, and USEPA 2016b) as a starting point. MDH identified additional studies and conducted supplemental analysis to comply with MDH's methodology.

Short-term, Subchronic and Chronic* Non-Cancer Health Based Value (nHBV) = 0.015 µg/L**

*Due to the highly bioaccumulative nature of PFOS within the human body, serum concentrations are the most appropriate dose metric and the standard equation to derive the HBV is not appropriate. Short-term exposures have the potential to stay in the body for an extended period of time. In addition, accumulated maternal PFOS is transferred to offspring (i.e., placental and breastmilk transfer). A single HBV has therefore been recommended for short-term, subchronic, and chronic durations. The HBV was derived using a toxicokinetic (TK) model previously developed by MDH (Goeden et al. 2019). Model details and results are presented below.

**Relative Source Contribution (RSC): Using the most recent publications regarding PFOS serum levels in infants and young children as well as the National Report on Human Exposure to Environmental Chemicals (CDC, 2017) for older children and adults, RSCs of 0.5 (50%) and 0.2 (20%) were selected for infants/young children and chronic steady-state conditions, respectively.

Intake Rate: In keeping with MDH's peer-reviewed and promulgated methodology, 95th percentile water intake rates (Table 3-1, 3-3 and 3-5, USEPA 2019) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2011) were used. Breastmilk concentrations were calculated by multiplying the maternal serum concentration by a PFOS breastmilk transfer factor of 1.7%. For the breast-fed infant exposure scenario, a period of exclusive breastfeeding for one year was used as representative of a reasonable maximum exposure scenario. [Note: "exclusively breast-fed" intake rates refers to infants whose sole source of milk comes from human breastmilk, with no other milk substitutes (USEPA 2011, page 15-2).]

A simple equation is typically used to calculate HBVs at the part per billion level with results rounded to one significant digit. However, the toxicokinetic model used to derive the HBV for PFOS showed that serum concentrations are impacted by changes in water concentrations at the part per trillion level. As a result, the HBV contains two digits.

Reference Dose/Concentration:	HED/Total UF = 0.000307/100 = 0.0000031 mg/kg-d (or 3.1 ng/kg-d) (adult C57BL/6 male Mice). *[The corresponding serum concentration is 2.36/100 = 0.024 mg/L. Note: this serum concentration is inappropriate to use for individual assessment.***]*
Source of toxicity value:	Determined by MDH in 2018
Point of Departure (POD):	2.36 µg/mL (or mg/L) serum concentration (Dong et al 2011, NOAEL)
Dose Adjustment Factor (DAF):	Toxicokinetic Adjustment based on Chemical-Specific Clearance Rate = Volume of Distribution (L/kg) x (Ln2/Half-life, days) = 0.23 L/kg x (0.693/1241 days) = 0.00013 L/kg-day. (Half-life from Li et al 2018.)
Human Equivalent Dose (HED):	POD x DAF = 2.36 mg/L x 0.00013 L/kg-d = 0.000307 mg/kg-d
Total uncertainty factor (UF):	100
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics), 10 for intraspecies variability, and 3 for database uncertainty (impacts on serum thyroxine (T4) in developing animals have been reported at serum concentrations ~3-fold lower than the POD. Additional studies regarding thyroid effects and a more complete assessment of developmental immune effects are warranted.)
Critical effect(s):	increased IL-4 and decreased SRBC specific IgM levels
Co-critical effect(s):	decreased pup body weight; increased fasting serum insulin and glucose in pups; suppressed SRBC response, increased NK cell activity and decreased IgM; decreased total and free T4 (maternal and pups); decreased adrenal weight, decreased serum corticosterone and adrenocorticotropic hormone levels in serum, and corticotropin-releasing hormone concentration in hypothalamus; and changes in cholesterol and histological changes in the liver (adults)
Additivity endpoint(s):	Adrenal (E), Developmental, Hepatic (liver) system, Immune, and Thyroid (E)

***Serum concentration is useful for informing public health policy and interpreting population-based exposure potential. This value is based on population-based parameters and should not be used for clinical assessment or for interpreting serum levels in individuals.

Toxicokinetic Model Description (Goeden 2019):

PFOS is well absorbed and is not metabolized. Serum concentrations can be calculated from the dose and clearance rate using the following equation.

$$Serum\ Concentration\ \left(\frac{mg}{L}\right) = \frac{Dose\left(\frac{mg}{kg \cdot day}\right)}{Clearance\ Rate\left(\frac{L}{kg \cdot day}\right)}$$

Where:
Dose (mg/kg-day) = Water or Breastmilk Intake (L/kg-day) x Level in Water or Breastmilk (mg/L) and
Clearance (L/kg-d) = Volume of distribution (L/kg) x (Ln 2/half-life (days))

Two exposure scenarios were examined: 1) an infant fed formula reconstituted with contaminated water starting at birth and continuing ingestion of contaminated water through life; and 2) an infant exclusively breast-fed for 12 months, followed by drinking contaminated water. In both scenarios the simulated individuals began life with a pre-existing body burden through placental transfer of PFOS (maternal serum concentration x 40%) based on average cord to maternal serum concentration ratios reported in the literature. The serum concentration of the mother at delivery was assumed to be at steady-state and was calculated by using the equation above with a time-weighted 95th percentile intake from birth to 30 years of age (0.048 L/kg-d). During lactation a 95th percentile water intake rate of 47 mL/kg-d and a body weight of 65.1 kg ((USEPA 2019), Table 3-3) was used to calculate daily maternal serum concentrations.

Consistent with MDH methodology, 95th percentile water intake and upper percentile breastmilk intake rates were used to simulate a reasonable maximum exposed individual. A PFOS breastmilk transfer factor of 1.7%, based on average breastmilk to maternal serum concentration ratios reported in the literature, was used to calculate breastmilk concentration. According to the 2016 Breastfeeding Report Card (CDC, 2016), nearly 66 percent of mothers in Minnesota report breastfeeding at six months, dropping to 41% at twelve months. MDH chose to use the breastmilk intake rates for exclusively breastfed infants, as reported in USEPA 2011, for one year for the breast-fed infant scenario.

Daily post-elimination serum concentration was calculated as:

$$Serum\ Conc.\left(\frac{mg}{L}\right) = \left[Prev.\ day\ Serum\ Conc.\left(\frac{mg}{L}\right) + \frac{Today's\ Intake(mg)}{V_d\left(\frac{L}{kg}\right) \times BW(kg)}\right] \times e^{-k}$$

To maintain mass balance, daily maternal serum concentrations and loss-of-chemical via transfer to the infant as well as excretion represented by the clearance rate, were calculated.

Summary of Reasonable Maximum Exposure (RME) Scenario Model Parameters

Model Parameter	Value Used
Half-life	1241 days (mean value for all ages, Li et al 2018) (5th to 95th percentile range: 803 – 2263 days)
Volume of distribution (Vd)	0.23 L/kg (US EPA 2016c)
Vd Age Adjustment Factor	2.1 age 1-30 days decreasing to 1.2 age 5-10 years and 1.0 after age 10 years (Friis-Hansen 1961)
Clearance Rate (CR)	0.00013 L/kg-d, calculated from Vd x (Ln 2/half-life)
Placental transfer factor *(% of maternal serum level)*	40% (mean of mean paired maternal:cord blood ratios reported in the literature. Range of mean values 30 – 60%.) (Mean 95th percentile value 81%, range 70 – 106%.)
Breastmilk transfer factor *(% of maternal serum level)*	1.7% (mean of mean paired maternal serum:breastmilk ratios reported in the literature. Range of mean values 1 – 3%.) (No 95th percentile values reported in literature.)
Water Intake Rate (L/kg-d)	95th percentile consumers only (default values, MDH 2008) (Table 3-1 (for ages ≥ 2 yrs), 3-3 (for lactating women), and 3-5 (for ages < 2yr)) (USEPA 2019)
Breastmilk Intake Rate (L-kg-d)	Upper percentile exclusively breast-fed infants (Table 15-1, US EPA 2011)
Body weight (kg)	Calculated from water intake and breastmilk intake rate tables

A relative source contribution factor (RSC) is incorporated into the derivation of a health-based water guidance value to account for non-water exposures. MDH utilizes the Exposure Decision Tree process presented in US EPA 2000 to derive appropriate RSCs. MDH relied upon the percentage method to reflect relative portions of water and non-water routes of exposure. The values of the duration specific default RSCs (0.5, 0.2, and 0.2 for short-term, subchronic, and chronic, respectively) are based on the magnitude of contribution of these other exposures that occur during the relevant exposure duration (MDH 2008). **In the case of PFOS, the RSC concept must be applied in a framework recognizing the long elimination half-life of PFOS, such that a person's serum concentration at any given age is not only the result of his or her current or recent exposures within the duration of concern, but also from exposure from years past.**

Serum concentrations are the best measure of cumulative exposure and can be used in place of the RfD in the Decision Tree process. Biomonitoring results (serum concentrations) from the general population (National Report on Human Exposure to Environmental Chemicals (CDC 2017) and new residents who were not historically exposed to contaminated water in the East Metro (Nelson, 2016) can be used to represent non-water or background exposures for older children and adults. For infants and young children, MDH conducted a review of the literature to identify appropriate background serum concentrations.

Serum concentrations in the general population have decreased over time, but appear to increase with age, with older children and adults exhibiting higher serum levels. This trend direction is in the opposite direction than MDH's RME model serum predictions. However, it is

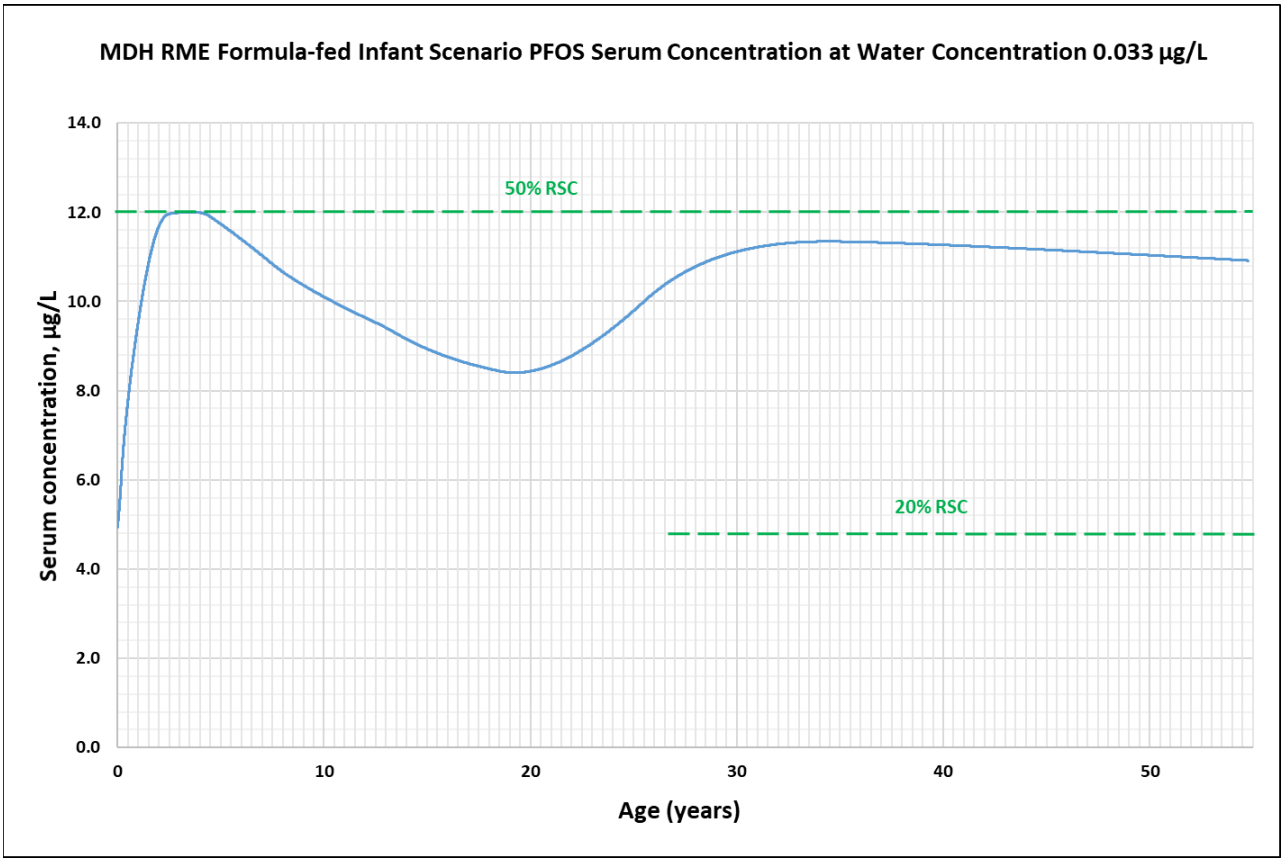
critical to note that background exposure levels are the result of decreasing historical exposure while MDH's model predicts serum concentrations resulting from a constant contaminated water source over time.

The apportionment to water ingestion can be calculated by taking a ceiling of 80% and subtracting a conservative (high-end) serum value from the most recent biomonitoring data. Eighty percent of the serum concentration associated with the RfD would be 19.2 µg/L (24 µg/L x 0.8). Subtracting the 95th percentile serum level (8.82 µg/L) for three to five year olds (Ye et al 2018) as non-water background exposure for infants and young children from the 80% ceiling leaves a residual serum concentration of 10.4 µg/L (19.2 – 8.82) for ingestion of contaminated water. This residual concentration is approximately 43% of the serum concentration at the RfD (24 µg/L) and approximately 54% of the 80% ceiling value (19.2 µg/L), supporting the use of an RSC of 50% for infants and young children.

Since exposures take years to eliminate it is also important to consider the higher-background steady-state serum levels in older age groups. To determine the appropriate RSC for steady-state conditions the 95th percentile (18.26 µg/L) from the most recent NHANES data (2015-2016, (Nelson 2018)) was used to determine that the floor value of 20% is the appropriate RSC for steady-state conditions.

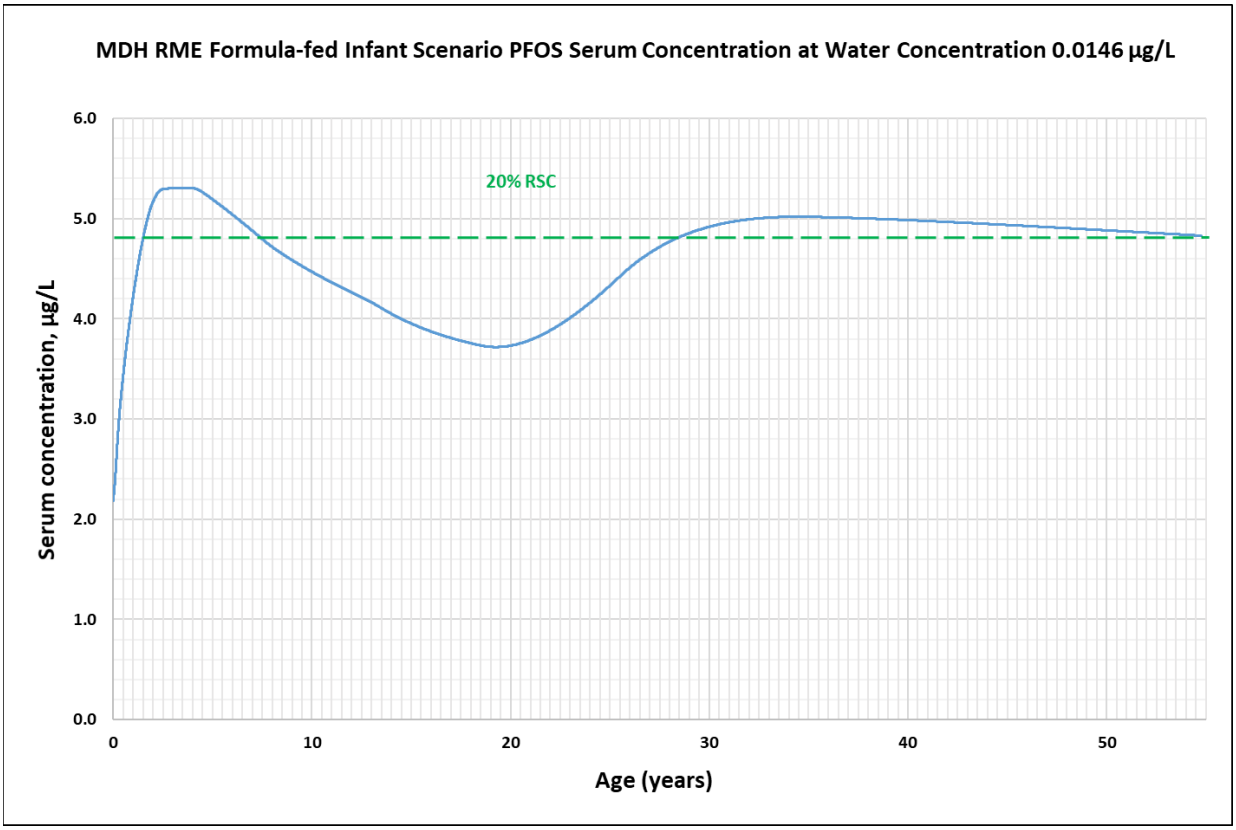
As mentioned above, two exposure scenarios were examined: 1) an infant fed formula reconstituted with contaminated water starting at birth and continuing ingestion of contaminated water through life; and 2) an infant exclusively breast-fed for 12 months, followed by drinking contaminated water through life. For the first scenario, the formula-fed infant, the water concentration that maintains a serum concentration attributable to drinking water at or below an RSC of 50% in infants and young children is 0.033 µg/L (Figure 1).

Figure 1. Formula-fed infant scenario serum concentrations over a lifetime, based on MDH's RME and an RSC of 50% for infants and young children.



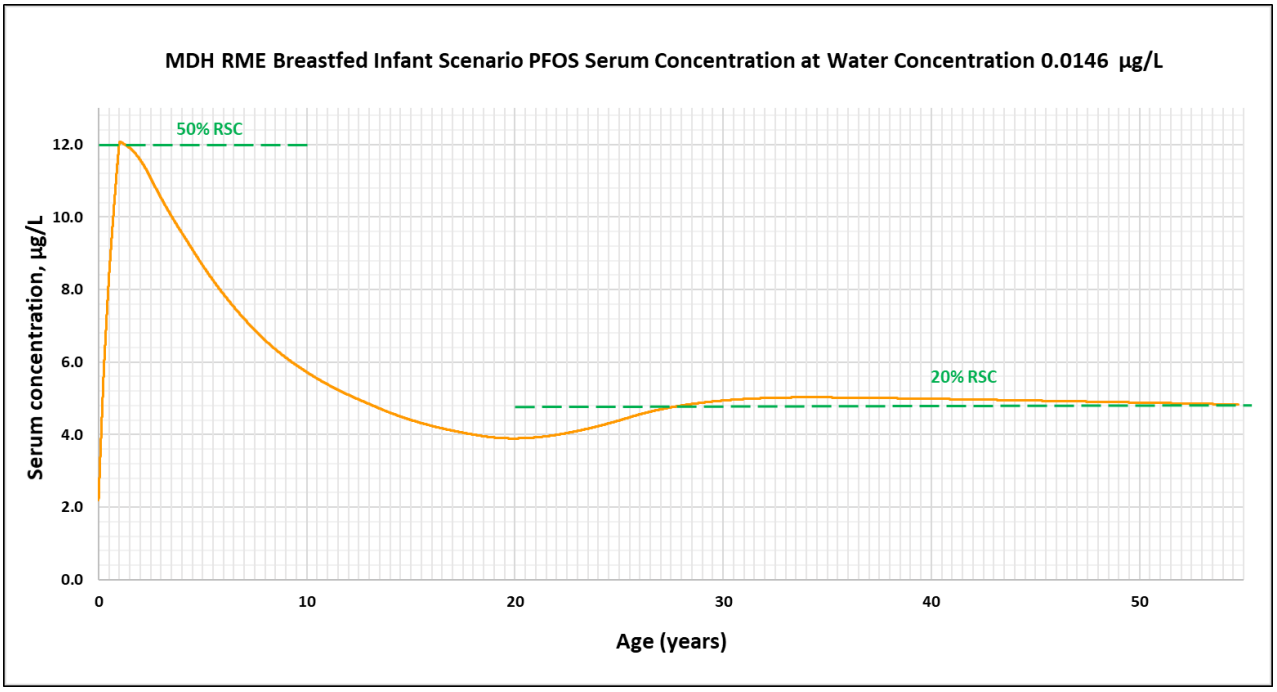
However, because of the long half-life the serum concentration curve is very flat, and serum levels in older children and adults exceeds the steady state RSC of 20%. In order to keep serum concentrations at steady state at or below 20% the water concentration had to be lowered to 0.0146 µg PFOS/L water (Figure 2).

Figure 2. Formula-fed infant scenario serum concentrations over a lifetime, based on MDH's RME and an RSC of 20% for steady-state.



For the second scenario, the breast-fed infant, the water concentration that maintains a serum concentration attributable to drinking water at or below an RSC of 50% in infants and young children is 0.0146 µg/L (Figure 3).

Figure 3. Breast-fed infant scenario serum concentrations over a lifetime, based on MDH's RME and an RSC of 50% for infants and young children.



The water concentration of 0.0146 µg/L also maintains serum concentrations at steady state at or below 20%.

To ensure protection of all segments of the population, the final health-based value for PFOS is set at 0.0146, rounded to 0.015 µg/L.

Cancer Health Based Value (cHBV) = Not Applicable

Cancer classification:	Suggestive Evidence of Carcinogenic Potential (USEPA 2016b,d)
Slope factor (SF):	Not Applicable
Source of cancer slope factor (SF):	Not Applicable
Tumor site(s):	Liver and thyroid tumors were identified in both control and exposed animals at levels that did not show direct relationship to dose.

Volatile: No

Summary of Guidance Value History:

A chronic nHBV of 1 µg/L was first derived in 2002. A revised chronic nHBV of 0.3 µg/L was derived in 2007 and promulgated as an nHRL in 2009. In 2017, MDH derived a revised nHBV (applicable to all durations) of 0.027 µg/L. In 2018, MDH revised the nHBV (applicable to all

durations) to 0.015 µg/L. The 2018 value is lower than the previous value as the result of: 1) incorporating a more recent, community-based shorter half-life value and 2) additional toxicological information. In 2020 MDH incorporated updated water intake rates (US EPA 2019). Using the updated intake rates did not change the HBV value.

Summary of toxicity testing for health effects identified in the Health Standards Statute (144.0751):

Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. MDH has considered the following information in developing health protective guidance.

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested for specific effect?	Yes	Yes	Yes	Yes	Yes
Effects observed?	Yes[1]	Yes[2]	Yes[3]	Yes[4]	Yes[5]

Comments on extent of testing or effects:

[1] Human epidemiological studies have examined a number of endocrine targets, including thyroid hormone levels and/or thyroid disease, reproductive hormones and insulin levels. Results from these studies have provided limited support for an association between PFOS and thyroid endpoints. Stronger associations were found in populations at risk for iodine deficiency or positive anti-TPO antibodies (a marker for autoimmune thyroid disease).

Investigators from one laboratory have reported increased FSH and decreased LH and testosterone at doses similar in magnitude to the critical study LOAEL. However, there are concerns regarding the study design and these effects are not listed as co-critical at this time. Decreases in adrenal gland weight as well as serum corticosterone and adrenocorticotropic hormone levels have been observed at doses similar in magnitude to the critical study LOAEL. Changes in expression of POMC (proopiomelanocortin), ACTHr (adrenocorticotropic hormone receptor) and CRH (corticotropin-releasing hormone) genes were also observed. These effects have been included as co-critical effects. Multiple studies in laboratory animals have reported decreased serum thyroid levels, in particular, thyroxin (T4) in offspring and adult animals at exposure levels similar in magnitude to the critical effect. Transcriptional changes of genes, in part regulated by thyroid hormones, involved in neurodevelopment have also been reported. However, the biological or functional significance of these changes are not clear. A NOAEL for thyroid hormone impacts in offspring has not been identified. As a result, a database uncertainty factor has been incorporated into the RfD calculation. Changes in total and free T4 have been identified as co-critical effects and Thyroid (E) has been identified as an Additivity Endpoint.

[2] Human epidemiology studies have evaluated associations for three categories of altered immune response: immunosuppression (altered antibody response, infectious disease resistance), hypersensitivity (asthma, eczema, allergies), and autoimmunity. The strongest evidence comes from fairly consistent associations with antibody response to vaccines.

However, consistent associations between serum PFOS and rates of infectious disease have not been reported.

Studies in laboratory animals have shown that PFOS exposure alters several immunologic measures (e.g., suppression of SRBC response and/or natural killer cell activity) in adult animals. A single developmental immune study evaluating effects resulting from *in utero* exposure only has been conducted. A database uncertainty factor was incorporated into the RfD calculation, in part, due to the need for a more comprehensive assessment of potential developmental immune effects. Immune suppression was identified as the critical effect and forms the basis of the RfD. Immune System has been identified as an Additivity Health Endpoint.

[3] Human epidemiology studies have suggested an association between prenatal PFOS serum levels and lower birth weight, however, this association has not been consistent.

Studies conducted in laboratory animals have identified several sensitive developmental effects, including decreased pup body weight, changes in energy metabolism (e.g., glucose levels, lipid metabolism) and decreased thyroid hormone levels. Some of these developmental effects were identified as co-critical effects and are included as an Additivity Health Endpoint. Additional effects, including increased pup death, were observed at higher exposure levels.

[4] Human epidemiology studies have evaluated alterations in reproductive hormones, menstrual cycle length, onset of menopause, endometriosis, breastfeeding duration, effects on sperm, and fertility. Findings have not been consistent across studies or there are too few studies to interpret the results. Since menstruation, parturition and breastfeeding are elimination routes the possibility of reverse causation has been raised for several of the endpoints evaluated in females. An association between preconception serum PFOS, gestational diabetes, and pregnancy induced hypertension has been reported in populations with serum PFOS concentrations of 0.012-0.017 μg/mL (or 12 – 17 μg/L).

Studies in laboratory animals indicate that fertility is not a sensitive endpoint, with post-implantation loss, decreases in male reproductive organ weights, decreased epididymal sperm count, and evidence of blood-testes-barrier disruption at exposure levels higher than those causing developmental or immune toxicity.

[5] There have been limited evaluations of neurotoxicity in humans. Human epidemiological studies have not provided consistent associations between exposure to PFOS and neurobehavioral, neuropsychiatric or cognitive outcomes in childhood or adulthood.

A limited number of developmental neurotoxicity and adult neurotoxicity studies have been conducted in laboratory animals. Increased motor activity and decreased habituation of male offspring was reported following gestational and lactational exposure at levels higher than those causing the critical effect. Results from studies using water maze tests for learning and

memory in animals exposed during development or as adults have yielded inconsistent results or effects only at higher dose levels.

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WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 13

https://www.pca.state.mn.us/sites/default/files/wq-s6-13.pdf

Aquatic Life Water Quality Standards Technical Support Document for Nitrate

Triennial Water Quality Standard Amendments to Minn. R. chs. 7050 and 7052

DRAFT For External Review, November 12, 2010

Minnesota Pollution Control Agency

TBD 2010

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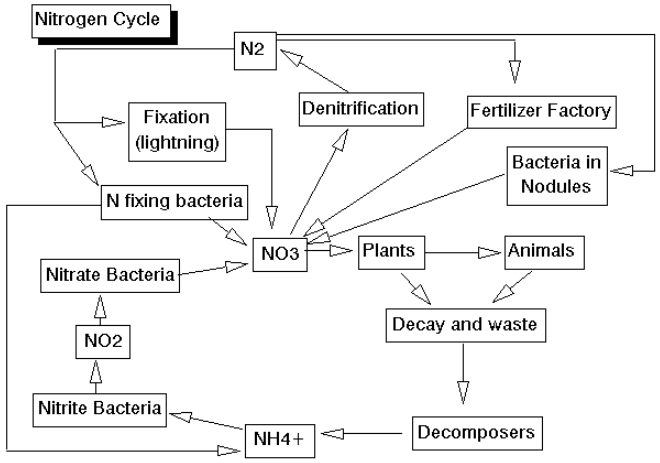
Contents

Introduction

Nitrate is formed as part of the breakdown of organic wastes, production by nitrogen-fixing plants, and through industrial production. Sources of excess nitrate in the environment can be linked to human activities on the landscape that result in the release of nitrogen to surface and ground waters. Nitrogen cycling in the environment results in nitrogenous compounds such as ammonia denitrifying into the more stable and conservative nitrate ion (NO_3).



Obtained from: www.marietta.edu/~biol/102/ecosystem.html

Concern regarding the toxicity of nitrate to aquatic organisms was brought to the attention of the MPCA from comments made during the preceding 2005-2008 rules revision by the Minnesota Center for Environmental Advocacy and concerns raised by the Minnesota Department of Natural Resources. The scientific literature has documented nitrate toxicity at concentrations that are environmentally relevant (Camargo and Alonso, 2006) to concentrations reported from Minnesota surface waters. In addition, the Minnesota State Legislature in 2010 approved funding for the MPCA to develop aquatic life standards for nitrogen and nitrate. Development of a nitrate standard is part of the effort to address these concerns. The MPCA is also engaged in developing a nitrogen budget for the state that focuses on total nitrogen in surface waters.

Natural sources of nitrate to surface waters in the state vary; however, when nitrate concentrations in surface water samples from “reference” areas (i.e., areas with relatively little human impact) are compared to samples from areas of greater human impact, the reference areas exhibit much lower nitrate concentrations. Nitrate concentrations in these reference areas are typically below 1 mg/L (Heiskary and Wilson, 2005). Still, elevated concentrations of nitrate have been documented in surface waters throughout the state. A comprehensive assessment of these data is beyond the scope of this document, but current trends in the data suggest that increased nitrate concentrations are associated with areas of higher human activity on the landscape.

In the surface water quality standards for Minnesota's Class 1 waters, protected as drinking water sources, human exposure to nitrates is regulated through the Federal Safe Drinking Water Act, with the Maximum Contaminant Level set at 10 milligrams/liter (mg/L), and a nitrite standard set at 1 mg/L. However, there is little guidance for protection of United States waters from the impacts of nitrate toxicity to aquatic organisms. The importance of nitrate toxicity to aquatic organisms has been a concern to aquaculture management for many years. In the environment, nitrate toxicity has not been a subject of scrutiny compared to the more toxic ammonia and nitrite. This document will present the technical discussion of surface water exposures and resulting toxicity of nitrate to aquatic organisms, and will propose a draft water quality standard necessary for the protection of aquatic life.

Why is nitrate not in a nutrient standard?

Nitrate is the form of nitrogen most available for use by plants. In freshwater systems, nitrogen is not a limiting nutrient for aquatic plant growth and excess nitrogen, primarily in the nitrate form, may accumulate in these systems. In contrast, growth of saltwater plants typically is limited by available nitrogen in the ecosystem. As such, the transport of excess nitrogen, predominantly as nitrate from freshwater systems, has been implicated – along with phosphorus – in the formation of oxygen-depleted areas in many marine sites including the Gulf of Mexico. The cause of these oxygen-depleted areas is largely the result of nutrient enrichment or eutrophication (excess algal growth and decay).

In Minnesota, water quality standards have been adopted to protect lakes from conditions of eutrophication, and the current rule revision includes draft standards to protect against eutrophication in rivers. Nutrient standards are based on phosphorus concentration as the primary cause of eutrophication, and efforts to develop these standards considered the roles of both phosphorus and nitrogen. In developing the eutrophication standards, monitoring data was examined and compared to a number of responses measured in the biological community like fish assemblages and abundances. No clear trend was established for the role of nitrogen in the response of these organisms or any direct contribution to eutrophication. Efforts to develop a total nitrogen budget center on addressing contributions of nitrogen in state surface water to protect downstream effects in the Mississippi River basin; however, this effort differs from the need to develop a nitrate toxicity standard in that it does not address the immediate or short-term effects of nitrate in any given lake or stream. In surface water, nitrate is the predominant form of total nitrogen, reported as nitrate-N, in concentrations above about 4 mg/L. (See the River Nutrient technical support document for further discussion). This concentration of nitrate is within the range of concentrations reported for effects to aquatic organisms.

How and why water quality standards are developed

Minnesota's Water Quality Standards (WQSs) are designed to be protective of the beneficial uses of groundwater and surface waters. In surface waters, protection encompasses normal growth and reproduction of aquatic animal and plant populations,

human recreational uses, consumption of aquatic biota, and sources of drinking water in some waters. WQSs consist of three parts: 1) the classification of designated, beneficial uses of water bodies 2) narrative protection goals and numeric criteria that are concentrations of contaminants considered protective of aquatic life or the other designated beneficial uses, and 3) mechanisms designed to avoid degradation [or "promote nondegradation"] (federal anti-degradation) of water quality. This document focuses on the draft water quality standard for protection of the aquatic life community for nitrate.

Development of the draft nitrate standard relies on sound scientific studies that provide the data needed to characterize and quantify how pollutants affect aquatic organisms. Toxicity data used to develop numeric criteria were evaluated based on national EPA guidance (USEPA, 1985), requirements in Minn. R. chs. 7050 and 7052, methods outlined by the American Society for Testing and Materials(ASTM, 2009), and a number of EPA testing methods. The key steps in developing new numeric water quality criteria involved:

1) A thorough search of the scientific literature by using electronic and printed databases. This search was performed for literature published through May 2010.
2) Compiling articles, reports and similar documentation based on their relevance to the issue. In this case, the search terms "nitrate", "toxicity" and "freshwater" served to provide the bulk of literature considered for review.
3) Reviewing these articles to screen out those that were outside of the scope of interest and to determine the usefulness of reported endpoints. For example, articles were found that reported toxicity of silver nitrate or used terrestrial organisms. Neither of these fit the scope of assessing the toxicity of the nitrate ion in freshwater aquatic systems.
4) Tabulating pertinent toxicity endpoints to be used in the calculation of draft acute and chronic standards (see Table 1a).

Articles were reviewed and critiqued based on the information reported. Occasionally, correspondence with the author was needed to clarify issues or obtain additional information. Information from the literature was retrieved from a search of academic databases. Primary literature search databases were MPCA library resources, University of Minnesota library, Scirus (www.scirus.com), Google Scholar (scholar.google.com), U.S. EPA ECOTOX and other sources. Scientific studies were assessed for quality based on guidance provided by the EPA and published ASTM methods of testing protocol (ASTM). Additional information for assessing studies has been summarized in guidance from the MPCA (MPCA 2010). Because WQSs are set to be protective for a specific beneficial use, rounding based on the correct significant figures was done to the preceding digit to maintain a concentration that is below the calculated values.

Aquatic Life Criteria Development

Numeric water quality criteria consist of a Final Acute Value (FAV), a Maximum Standard (MS) and a Final Chronic Value (FCV) (see U.S. EPA (1985) for more details).

These values are interrelated and are calculated on an assumption that allows for protection of 95% of aquatic communities. Much of this assumption is based on the fact that not all aquatic organisms present in the environment can be feasibly tested for their sensitivity to environmental contaminants. Therefore, calculation of numeric water quality criteria relies on toxicity endpoints provided through laboratory tests using organisms that are either cultured for this purpose or collected from the field and tested. These organisms, then, are surrogates or representatives of a variety of different families of organisms, such that they represent an approximation of the assemblage of North American aquatic organisms dependent on adequate water quality for their survival and reproduction. The use of either cultured or field collected organisms must follow consistent methodology that assures for the soundness of outcomes in the tests performed.

Acute effects of nitrate on aquatic organisms include survival endpoints from reported tests. These acute tests are typically of short duration (2 – 4 days). Acute toxicity is described primarily through calculated values of point estimates of lethal or effects concentration affecting 50% of the test population, referred to as LC50 or EC50, respectively. Chronic effects are measured primarily from reports of survival, reproduction, and growth of test organisms. These tests are performed over many days or weeks depending on the organism used and specific protocols for minimum test duration, and are typically referred to as full or partial life cycle tests. Further discussion of chronic endpoints is found in the MPCA guidance (MPCA, 2010).

Toxicity information used for development of the numeric criteria for nitrate was provided through reports from scientific studies published in the open literature. Most studies considered were from work published over the past ten years. Results of acceptable studies were reviewed from 89 references published in the scientific literature. Table 1b lists all the studies considered for use in water quality criteria development, with the acceptable acute studies used to develop the numeric criteria in Table 2. Studies considered for use in numeric criteria development were those performed using sodium nitrate as a toxicant. Other carrier salts reported for the nitrate ion are calcium and potassium. Few studies reported results using calcium nitrate, and based on the recent work by EPA assessing chloride toxicity, the potassium ion exerts its own level of toxicity that would confound effects of toxicity endpoints if used together with nitrate. The literature has much information about the toxicity of ammonium nitrate, which is a common agricultural fertilizer, but these too were not included, because ammonia is a much more toxic chemical. The Minnesota water quality chronic standard for ammonia has already been established at 40 ppb for class 2B surface waters.

Most of the studies reviewed were found to have no useful toxicity information for development of draft criteria because they used species that are not native to North America or the studies were otherwise unsuitable. Nine studies reported endpoints of acute toxicity for ten genera of freshwater animals that were used to calculate the final acute value. Procedures for calculating full (Tier I) aquatic life criteria require acceptable toxicity endpoints for 8 taxonomic family-level categories. This formality provides assurance of calculating a final acute value that is protective of aquatic communities.

During the initial phases of draft standard development, information provided in the published literature was not enough to fulfill this requirement. Discussions with the EPA Region 5 Water Quality Branch resulted in their offer to perform additional toxicity tests to fill this gap. These tests provided toxicity information for seven freshwater species, which served to fulfill the additional taxonomic categories. The endpoints of those tests were provided to the MPCA for use in developing the numeric criteria development. As these endpoints are preliminary, changes to the draft values for water quality standards may be possible. A final report of these tests performed by EPA is anticipated by the end of 2010.

Development of acute water quality criteria

Acute endpoints of nitrate toxicity to aquatic organism ranged from 100.1 milligrams/liter nitrate-N (mg/L) for the aquatic insect *Hydropsyche occidentalis* to 1903 mg/L nitrate-N for the lake whitefish. Overall, invertebrates appeared to be the most sensitive to nitrate toxicity, as this group is represented in the four lowest ranked values in the calculation of the Final Acute Value (FAV) as presented in Table 1a. Invertebrates represent most of the acute toxicity endpoints below the median LC50 of all reported values. Aquatic insects represent the group of invertebrates most commonly reported in the literature, and two caddisfly species were shown to have the lowest acute toxicity values for nitrate (Camargo and Ward, 1995). Study results from the 2010 EPA toxicity tests reported one stonefly (*Amphinemura*) and one midge (*Chironomus*) as being somewhat less sensitive, and mollusks also vary somewhat in their sensitivity to nitrate in tests reported by EPA. Two species of cladoceran, *Ceriodaphnia dubia* and *Daphnia magna* (Scott and Crunkilton, 2000) had the smallest difference in toxicity endpoints reported for any group of related organisms. Overall, invertebrates varied in their toxicity endpoints by just over an order of magnitude. Lowest and highest reported species acute values ranged a little more than two times. In contrast to invertebrates, fish were shown to be the least acutely sensitive of all organisms tested. Toxicity endpoints for amphibians were shown to be more acutely sensitive to nitrate than endpoints reported for fish, but not as sensitive as invertebrates. Supporting data from Smith (Smith et al., 2005), however, reported green frogs as being quite sensitive to nitrate exposure. Nevertheless, these data were not used in the calculation of the draft standard because the tests involved direct feeding of the test organisms during the exposures with nitrate, which is not recommended during acute exposures. Additional testing of amphibian exposures to nitrate is currently underway by EPA.

Development of chronic water quality criteria

The chronic criterion value can be determined either by developing a species sensitivity distribution and following the same methods used to calculate the FAV, or by using an acute to chronic ratio. Data sources provided eighteen acceptable chronic endpoints, but did not provide enough reported endpoints for different species to fulfill the necessary 8 taxonomic categories. Chronic toxicity endpoints (Table 3) for invertebrates were

reported only for two cladoceran species, *Ceriodaphnia dubia* and *Daphnia magna*. For vertebrates, lake trout has the lowest reported chronic endpoints (McGurk et al., 2006). Schuytema and Nebeker (Schuytema and Nebeker, 1999a, Schuytema and Nebeker, 1999c, Schuytema and Nebeker, 1999b) reported 10 day endpoints ([Lowest/No]-Observed-Adverse-Effect-Level, LOAEL/NOAEL respectively) for the amphibian life-stage of the pacific tree frog and the red-legged frog. These endpoints are reported in Table 3 only for purposes of comparison to other chronic data. These tests were of relatively short duration and methods for tests using amphibians vary.

Data were available to compute acute to chronic ratios (ACR) based on acute and chronic toxicity data for three species (Table 1a). An acute to chronic ratio of 17 was calculated for *Ceriodaphnia dubia*, but no ACR could be computed for *Daphnia magna* as its associated chronic test was of short duration. Selecting an appropriate ACR is achieved through examination of the acute toxicity data. EPA guidance recommends calculating the geometric mean ACR for each species for which data are available. The ACR are compared to their corresponding acute toxicity endpoint and examined for any increasing or decreasing trends among the ranks of all acute values. In this dataset, the observed trend was for the ACR values to decrease as the acute values increased. EPA guidance suggests that given this trend, an appropriate ACR can be selected from the species whose acute toxicity value is closest to the FAV, which for the dataset was calculated to be 83.4 mg/L. *Ceriodaphnia dubia*'s acute value of 374 mg/L resulted in an ACR of 17, which among the calculated ACRs is closest in value to 83.4. In lieu of a calculated ACR, Minnesota rules allows for the use of a default ACR of 18. Acute-to-chronic ratios calculated from test data are preferred over use of the default value. Selecting the ACR for an invertebrate is reasonable as invertebrate species account for a number of the most sensitive organisms used for calculating the FAV and invertebrates represent the six lowest acute endpoints used in the numeric criteria calculation. A final chronic value of 4.9 mg/L was calculated as the quotient of the FAV divided by the ACR (Table 1a). This value is considered protective as it falls below most chronic values found in the literature. The exception to this is the chronic toxicity of nitrate to Lake Trout reported by McGurk (McGurk et al., 2006). Effects on fry weight, a critical chronic endpoint, was reported as a NOEC = 1.6 mg/L and a LOEC = 6.25 mg/L Nitrate-N. An acceptable endpoint using the geometric mean of these chronic endpoints was calculated as the Maximum Acceptable Toxicant Concentration (MATC) = 3.16 mg/L nitrate-N. As provided in EPA guidance and in MN R. 7050, selecting a final chronic value for an economically and ecologically important species is appropriate. In Minnesota, cold-water fisheries, designated in MN R. 7050 as class 2A waters, have critical recreational and economic value. This designation provides for a means to protect for cold water species including lake trout. In consideration of this, and using the endpoints reported by McGurk, the draft chronic criterion value for these class 2A waters will be 3.1 mg/L nitrate-N. All other class 2 waters will have a draft chronic criterion value of 4.9 mg/L nitrate-N.

Additional considerations of nitrate toxicity to aquatic organisms

Toxicity testing performed by the EPA included a test using the amphipod *Hyalella azteca*. In March 2010, EPA began efforts to examine existing methods for culturing and testing of *H. azteca* to determine whether common laboratory practices to date may influence undue sensitivity in the organism. As part of this effort, EPA retested *Hyalella* with nitrate using preliminary outcomes from this examination. The retest reported a much less acutely sensitive endpoint for *Hyalella*, recorded as a preliminary value of >800 mg/L. A final report from EPA is anticipated in late 2010. As a result, the place held by *Hyalella* in the species sensitivity ranking was changed (Table 1a). The importance of this retesting is to assure proper assessment of nitrate toxicity to *Hyalella*, and to provide for the amphipod's representation as a key taxonomic position for criteria development.

Another goal in development of this draft standard was to attempt examining whether nitrate toxicity exhibits any trend with water hardness, similar to that shown for some metals. No relationship with hardness was evident based on the review of existing toxicity data.

Conclusion

Nitrate is both a naturally occurring substance and important nutrient in the life-cycle of plants in natural and cultivated settings. It can also be a common toxicant in Minnesota surface waters when present at concentrations exceeding those of reference areas where there is little human impact to the landscape. This document proposes a draft standard for the protection of aquatic life in lakes and streams designated as class 2 waters of the state. This use classification sets specific rules for protecting cold waters (class 2A) uses and cool/warm water (class 2B) uses. The draft water quality standards for nitrate were developed in efforts to protect these uses based on best available scientific information. EPA guidelines provide the means of examining data reported from toxicity tests using aquatic organisms in efforts to calculate concentrations of chemicals that are protective of aquatic life. The draft acute value (maximum standard) calculated is 41 mg/L nitrate-N for a 1-day duration, and the draft chronic value is 4.9 mg/L nitrate-N for a 4-day duration. In addition, a draft chronic value of 3.1 mg/L nitrate- N (4-day duration) was determined for protection of class 2A surface waters.

Table 1a. Ranked endpoints and calculation of draft criteria. Acute and chronic values are mg/L nitrate-N.

Genus/Species	Taxon		GMAV	Endpoint	Reference	Rank	P	Acute endpt./Chronic endpt. = ACR
Coregonus clupeaformis	Fish		1903.00	LC50	McGurk, et al. 20(	17	0.94	
Oncorynchus mykiss	Fish		1658.00	LC50	Buhl, K and J.J. H	16	0.89	
Pimephales promelas	Fish		1426.80	LC50	EPA	15	0.83	1815.9/339.29 = 5.35
Chironomus dilutus	Insecta		1230.50	LC50	EPA	14	0.78	
Salvelinus namaycush	Fish		1121.00	LC50	McGurk, et al. 20(	13	0.72	1121/131.9 (EC50/EC20) = 8.5
Megalonaias nervosa	mussel		937.00	LC50	EPA	12	0.67	
Hyalella			> 800	LC50	EPA	11	0.61	
Rana aurora	Amphibia		636.30	LC50	Schuytema, G.S.	10	0.56	
Pomacea paludosa	Molluska		516.20	LC50	Corrao, N.M. et al	9	0.50	
Amphinemura delosa	Insecta		476.00	LC50	EPA	8	0.44	
Pseudacris regilla	Amphibia		471.12	LC50	Schuytema, G.S.	7	0.39	
Daphnia magna	Crustacea		441.27	LC50	Scott, G., and R.I	6	0.33	
Sphaerium simile	mussel		376.00	LC50	EPA	5	0.28	
Ceriodaphnia dubia	Crustacea		374.00	LC50	Scott, G., and R.I	4	0.22	374/22 = 17
Lampsilis siliquiodea	Molluska		357.00	LC50	EPA	3	0.17	
Cheumatopsyche pettiti	Insecta		138.70	LC50	Carmargo, J. and	2	0.11	
Hydropsyche occidentalis	Insecta		100.10	LC50	Carmargo, J. and	1	0.06	

		GMAV	P	ln GMAV	(ln GMAV)	Sq Rt P
	Ceriodaphnia dubia	374.00	0.22	5.9243	35.0968	0.4714
	Lampsilis siliquiodea	357.00	0.17	5.8777	34.5478	0.4082
	Cheumatopsyche pettiti	138.70	0.11	4.9323	24.3277	0.3333
	Hydropsyche occidentalis	100.10	0.06	4.6062	21.2168	0.2357

s2 Num	1.3351				
s2 Denom	0.0309				
S2	43.2348				
Sqrt S2	6.5753	Chronic Criteria:	**83.43/17 =**		**4.91**
L	2.9537				
A	4.4240				
FAV	**83.4300**				

Table 1b. Summary of all nitrate toxicity data considered for use in standard development found in the open literature and provided in preliminary results from EPA toxicity tests.

Species Name/Common name	Native to N.A.?	Taxon	Effect Conc. (mg/L)	Reported Endpoint	Reference
Acipenser beari/ Siberian Sturgeon	No	Fish	1028	LC50	(Hamlin, 2006)
Acipenser beari/ Siberian Sturgeon	No	Fish	601	LC50	(Hamlin, 2006)
Acipenser beari/ Siberian Sturgeon	No	Fish	397	LC50	(Hamlin, 2006)
Amphinemura delosa/ Stonefly	Yes	Insecta	476	LC50	EPA
Catla catla/ Indian major carp	No	Fish	35	LC50	(Tilak, 2006)
Catla catla/ Indian major carp	No	Fish	33	LC50	(Tilak, 2006)
Catla catla/ Indian major carp	No	Fish	1401	LC50	(Tilak, 2006)
Catla catla/ Indian major carp	No	Fish	1251	LC50	(Tilak, 2006)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	374	LC50	(Schuytema and Nebeker, 1999b)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	374	LC50	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	14.1	LOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	113	LOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	14.1	LOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	35.9	LOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	35.9	LOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	7.1	NOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	56.5	NOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	7.1	NOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	17.9	NOEC	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia/ Water flea	Yes	Cladoceran	17.9	NOEC	(Scott and Crunkilton, 2000)
Cheumatopsyche pettiti/ caddisfly	Yes	Insecta	128	LC50	(Camargo and Ward, 1995)
Cheumatopsyche pettiti/ caddisfly	Yes	Insecta	154	LC50	(Camargo and Ward, 1995)
Cheumatopsyche pettiti/ caddisfly	Yes	Insecta	113.5	LC50	(Camargo and Ward, 1992)

Cheumatopsyche pettiti/ caddisfly	Yes	Insecta	165.5	LC50	(Camargo and Ward, 1992)
Chironomus dilutes/ midge	Yes	Insecta	1230.5	LC50	EPA
Cirrhinus mrigala/ Indian major carp	No	Fish	153	LC50	(Tilak, 2006)
Cirrhinus mrigala/ Indian major carp	No	Fish	163	LC50	(Tilak, 2006)
Cirrhinus mrigala/ Indian major carp	No	Fish	1055	LC50	(Tilak, 2006)
Cirrhinus mrigala/ Indian major carp	No	Fish	1023	LC50	(Tilak, 2006)
Coregonus clupeaformis/ Lake whitefish	Yes	Fish	1903	LC50	(Tilak, 2006)
Coregonus clupeaformis/ Lake whitefish	Yes	Fish	64.4	EC50	(Tilak, 2006)
Daphnia magna/ Water flea	Yes	Cladoceran	323	LC50	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	453	LC50	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	611	LC50	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	717	LOEC	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	717	LOEC	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	358	NOEC	(Scott and Crunkilton, 2000)
Daphnia magna/ Water flea	Yes	Cladoceran	358	NOEC	(Scott and Crunkilton, 2000)
Eulimnogammarus toletanus/ amphipod	No	Crustacea	85.0	LC50	(Camargo et al., 2005)
Eulimnogammarus toletanus/ amphipod	No	Crustacea	62.5	LC50	(Camargo et al., 2005)
Eulimnogammarus toletanus/ amphipod	No	Crustacea	22.2	LC10	(Camargo et al., 2005)
Eulimnogammarus toletanus/ amphipod	No	Crustacea	9.5	LC10	(Camargo et al., 2005)
Hyalella azteca/ scud	Yes	Crustacea	>800	LC50	EPA
Hydropsyche exacellata/ caddisfly	No	Insecta	269.5	LC50	(Camargo et al., 2005)
Hydropsyche exacellata/ caddisfly	No	Insecta	31.8	LC10	(Camargo et al., 2005)
Hydropsyche exacellata/ caddisfly	Yes	Insecta	90	LC50	(Camargo and Ward, 1995)
Hydropsyche exacellata/ caddisfly	Yes	Insecta	105	LC50	(Camargo and Ward, 1995)
Hydropsyche exacellata/ caddisfly	Yes	Insecta	97.3	LC50	(Camargo and Ward, 1992)
Hydropsyche occidentalis/ caddisfly	Yes	Insecta	109	LC50	(Camargo and Ward, 1992)

Labeo rohia/ Indian major carp	No	Fish	119	LC50	(Tilak, 2006)
Labeo rohia/ Indian major carp	No	Fish	123	LC50	(Tilak, 2006)
Labeo rohia/ Indian major carp	No	Fish	1434	LC50	(Tilak, 2006)
Labeo rohia/ Indian major carp	No	Fish	1351	LC50	(Tilak, 2006)
Lampsilis siliquoidea	Yes	Mussel	357	LC50	EPA
Oncorynchus mykiss/ Rainbow trout	Yes	Fish	1658	LC50	(Buhl and Hamilton, 2000)
Pimephales promelas/ fathead minnow	Yes	Fish	1815.9	LC50	EPA
Pimephales promelas/ fathead minnow	Yes	Fish	339.3		EPA
Pimephales promelas/ Fathead minnow	Yes	Fish	1010	LC50	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	1607	LC50	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	1406	LC50	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	717	NOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	1434	LOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	358	NOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	717	LOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	358	NOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	717	LOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	358	NOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	717	LOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	1435	LOEC	(Scott and Crunkilton, 2000)
Pimephales promelas/ Fathead minnow	Yes	Fish	1435	LOEC	(Scott and Crunkilton, 2000)
Pomacea paludosa/ Florida Apple Snail	Yes	Molluska	1001	LC50	(Corrao et al., 2006)
Pomacea paludosa/ Florida Apple Snail	Yes	Molluska	1001	LC50	(Corrao et al., 2006)
Pomacea paludosa/ Florida Apple Snail	Yes	Molluska	504	EC50	(Corrao et al., 2006)
Pomacea paludosa/ Florida Apple Snail	Yes	Molluska	622	EC50	(Corrao et al., 2006)
Potamopyrgus antipodarum/ aquatic snail	No (Exotic)	Molluska	1042	LC50	(Alonso and Camargo, 2003)

Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	643	LC50	(Schuytema and Nebeker, 1999a)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	578	LC50	(Schuytema and Nebeker, 1999a)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	56.7	NOAEL	(Schuytema and Nebeker, 1999a)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	111	LOAEL	(Schuytema and Nebeker, 1999a)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	1749.8	LC50	(Schuytema and Nebeker, 1999b)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	266.2	LC50	(Schuytema and Nebeker, 1999b)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	259.1	LOAEL	(Schuytema and Nebeker, 1999b)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	126.3	NOAEL	(Schuytema and Nebeker, 1999b)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	30.1	LOAEL	(Schuytema and Nebeker, 1999b)
Pseudacris regilla/ Pacific Treefrog	Yes	Amphibian	30.1	NOAEL	(Schuytema and Nebeker, 1999b)
Rana aurora/ red-legged frog	Yes	Amphibian	636.3	LC50	(Schuytema and Nebeker, 1999c)
Rana aurora/ red-legged frog	Yes	Amphibian	235	LOAEL	(Schuytema and Nebeker, 1999c)
Rana aurora/ red-legged frog	Yes	Amphibian	116.8	NOAEL	(Schuytema and Nebeker, 1999c)
Salvelinus namaycush/ Lake trout	Yes	Fish	1121	LC50	(McGurk et al., 2006)
Salvelinus namaycush/ Lake trout	Yes	Fish	189.6	EC50	(McGurk et al., 2006)
Sphaerium simile/ fingenail clam	Yes	Mussel	376	LC50	EPA
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	438.4	LC50	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	871.6	LC50	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	521.7	EC50	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	56.7	NOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	111	LOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	24.8	NOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	56.7	LOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	111	NOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	230.4	LOAEL	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	471	LC50	(Schuytema and Nebeker, 1999a)

Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	471	LC50	(Schuytema and Nebeker, 1999a)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	1,955.8	LC50	(Schuytema and Nebeker, 1999b)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	1236.2	LC50	(Schuytema and Nebeker, 1999b)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	494.4	LOAEL	(Schuytema and Nebeker, 1999b)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	259.1	NOAEL	(Schuytema and Nebeker, 1999b)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	126.3	LOAEL	(Schuytema and Nebeker, 1999b)
Xenopus laevis/ African Clawed Frog	No (Exotic)	Amphibian	65.6	NOAEL	(Schuytema and Nebeker, 1999b)

Table 2. Acute data of nitrate toxicity used for the development of draft water quality criteria.
All test endpoints are for test duration of 96 h unless otherwise noted (*) .

Species Name	Taxon	Effect Conc. (mg/L)	GMAV (mg/L)	Endpoint	Reference
Amphinemura delosa	Insecta	476.0		LC50	EPA
Ceriodaphnia dubia	Crustacea	374	374.0	LC50* (48h)	(Scott and Crunkilton, 2000)
Ceriodaphnia dubia	Crustacea	374		LC50* (48h)	(Schuytema and Nebeker, 1999b)
Cheumatopsyche pettiti	Insecta	154	138.7	LC50	(Camargo and Ward, 1995)
Cheumatopsyche pettiti	Insecta	113.5		LC50	(Camargo and Ward, 1992)
Cheumatopsyche pettiti	Insecta	165.5		LC50	(Camargo and Ward, 1992)
Cheumatopsyche pettiti	Insecta	128		LC50	(Camargo and Ward, 1995)
Chironomus dilutus	Insecta	1230.5		LC50	EPA
Coregonus clupeaformis	Fish	1903		LC50	(McGurk et al., 2006)
Daphnia magna	Crustacea	611.0	447.1	LC50* (48h)	(Scott and Crunkilton, 2000)
Daphnia magna	Crustacea	323		LC50* (48h)	(Scott and Crunkilton, 2000)
Daphnia magna	Crustacea	453.0		LC50* (48h)	(Scott and Crunkilton, 2000)
Hyalella azteca	Crustacea	72.6	72.6	LC50	EPA
Hydropsyche occidentalis	Insecta	105	100.1	LC50	(Camargo and Ward, 1995)
Hydropsyche occidentalis	Insecta	97.3		LC50	(Camargo and Ward, 1992)
Hydropsyche occidentalis	Insecta	109		LC50	(Camargo and Ward, 1992)
Hydropsyche occidentalis	Insecta	90		LC50	(Camargo and Ward, 1995)
Lampsilis siliquiodea	Molluska	357.0	357.0	LC50	EPA
Oncorynchus mykiss	Fish	1658	1658.0	LC50	(Buhl and Hamilton, 2000)
Pimephales promelas	Fish	1815.9	1426.8	LC50	
Pimephales promelas	Fish	1010		LC50	(Scott and Crunkilton, 2000)
Pimephales promelas	Fish	1607		LC50	(Scott and Crunkilton, 2000)

Pimephales promelas	Fish	1406		LC50	(Scott and Crunkilton, 2000)
Pomacea paludosa	Molluska	1001	1001.0	LC50	(Corrao et al., 2006)
Pomacea paludosa	Molluska	1001		LC50	(Corrao et al., 2006)
Pseudacris regilla	Amphibia	266.2	645.0	LC50* (10d)	(Schuytema and Nebeker, 1999b)
Pseudacris regilla	Amphibia	643		LC50	(Schuytema and Nebeker, 1999a)
Pseudacris regilla	Amphibia	578		LC50* (10d)	(Schuytema and Nebeker, 1999a)
Pseudacris regilla	Amphibia	1749.8		LC50	(Schuytema and Nebeker, 1999b)
Rana aurora	Amphibia	636.3	636.3	LC50* (16d)	(Schuytema and Nebeker, 1999c)
Salvelinus namaycush	Fish	1121	1121.0	LC50	(McGurk et al., 2006)
Sphaerium simile	Molluska	376.0	376.0	LC50	EPA

Table 3. Summary of chronic data from nitrate toxicity tests found acceptable in support of water quality standard development

Species Name	Taxon	Effect Conc. (mg/L)	End point	MATC (mg/L)	Test Dur. (d)	ACR Use?	Reference	Notes
Ceriodaphnia dubia	Crustacea	35.9	LOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	17.9	NOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	17.9	NOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	7.1	NOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	7.1	NOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	35.9	LOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	14.1	LOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	113	LOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	14.1	LOEC		7	Y	(Scott and Crunkilton, 2000)	
Ceriodaphnia dubia	Crustacea	56.5	NOEC	22	7	Y; MATC	(Scott and Crunkilton, 2000)	
Coregonus clupeaformis	Fish	64.4	EC50		120	N	(McGurk et al., 2006)	No weight endpoint
Daphnia magna	Crustacea	717	LOEC		7	N	(Scott and Crunkilton, 2000)	Test dur. Short
Daphnia magna	Crustacea	358	NOEC		7	N	(Scott and Crunkilton, 2000)	Test dur. Short
Daphnia magna	Crustacea	358	NOEC		7	N	(Scott and Crunkilton, 2000)	Test dur. Short
Daphnia magna	Crustacea	717	LOEC	507	7	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	358	NOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	1435	LOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	717	NOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	1434	LOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	358	NOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	717	LOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	358	NOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	1435	LOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short

Pimephales promelas	Fish	717	LOEC		18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	717	LOEC	717	18	N	(Scott and Crunkilton, 2000)	Test dur. Short
Pimephales promelas	Fish	339.3	EC50		28	Y	EPA	
Pomacea paludosa	Molluska	504	EC50		14	N	(Corrao et al., 2006)	Beyond Upper limit
Pomacea paludosa	Molluska	622	EC50		14	N	(Corrao et al., 2006)	Beyond Upper limit
Pseudacris regilla	Amphibia	259.1	LOAEL (L)		10	N	(Schuytema and Nebeker, 1999b)	acute test
Pseudacris regilla	Amphibia	126.3	NOAEL (L)	181	10	N	(Schuytema and Nebeker, 1999b)	acute test
Pseudacris regilla	Amphibia	30**	LOAEL (W)		10	N	(Schuytema and Nebeker, 1999b)	acute test
Pseudacris regilla	Amphibia	30**	NOAEL (W)	30	10	N	(Schuytema and Nebeker, 1999b)	acute test
Pseudacris regilla	Amphibia	111	LOAEL		10	N	(Schuytema and Nebeker, 1999a)	acute test
Pseudacris regilla	Amphibia	56.7	NOAEL	79.3	10	N	(Schuytema and Nebeker, 1999a)	Not ELS
Rana aurora	Amphibia	235	LOAEL (W)		16	N	(Schuytema and Nebeker, 1999c)	No acute
Rana aurora	Amphibia	116.8	NOAEL (W)	166	16	N	(Schuytema and Nebeker, 1999c)	No acute
Rana aurora	Amphibia	29**	LOAEL (L)		16	N	(Schuytema and Nebeker, 1999c)	No acute
Rana aurora	Amphibia	29**	NOAEL (L)		16	N	(Schuytema and Nebeker, 1999c)	No acute
Salvelinus namaycush	Fish	189.6	EC50		120	N	(McGurk et al., 2006)	
Salvelinus namaycush	Fish	1.6	NOEC		120	Y	(McGurk et al., 2006)	
Salvelinus namaycush	Fish	6.25	LOEC	3.16	120	Y; MATC	(McGurk et al., 2006)	

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WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 14

PETITION FOR RULEMAKING TO PROTECT AQUATIC LIFE, WILD RICE, WILDLIFE, AND HUMAN HEALTH

WHEREAS, protection of clean water sustains plant, animal, and human life in Minnesota, supports diverse communities, serves as a foundation for jobs and prosperity, and preserves Minnesota's most precious resource for generations to come; and

WHEREAS, the Minnesota Pollution Control Agency ("MPCA") has devoted excessive time and effort in rulemaking that seeks to deregulate pollution by removing, weakening, or restricting the application of numeric standards; or downgrading waters to give them less protection; and

WHEREAS, the MPCA's mission by statute is "to achieve a reasonable degree of purity of water, air and land resources of the state consistent with the maximum enjoyment and use thereof in furtherance of the welfare of the people of the state" in the "public interest;" and

WHEREAS, scientific research, including peer-reviewed literature and research which the MPCA has authored or co-authored, demonstrates that additional numeric criteria and beneficial use listings are required to protect aquatic life, wild rice, wildlife, and human health;

THEREFORE, under Minnesota Statutes 14.09 and Minnesota Rules 1400.2040, the undersigned Minnesota residents and organizations ("Petitioners") join in a Petition for Rulemaking as follows:

1. Petitioners request that the MPCA amend Minnesota Rule 7050.0222 to set enforceable class 2 numeric criteria for the following pollutants to protect aquatic life and the health of wildlife and humans who consume aquatic life:

   A. Sulfate – a new statewide criterion to prevent mercury and nutrient related harms.
   B. Nitrates – a new statewide criterion to protect sensitive aquatic life.
   C. Specific conductivity – new criteria applicable by Ecoregion to protect aquatic life.
   D. Chloride – a statewide criterion more stringent than 230 mg/L to protect aquatic life.

2. Petitioners request that the MPCA amend Minnesota Rules in chapter 7050 to state that all waters where wild rice is an "existing use" since November 28, 1975 are "waters used for the production of wild rice," including but not limited to wild rice waters identified in the Minnesota Department of Natural Resources ("DNR") 2008 report to the Legislature and since then by the MPCA, the DNR, and/or tribes.

FURTHER, Petitioners request that the MPCA cease or indefinitely defer the Agency's current rulemaking workplan priorities that undermine protection of water quality, including: 1) the repeal of class 1 drinking water numeric criteria or other rule changes that reduce protection of class 1 groundwater and surface water; and 2) the downgrading of class 2A or 2B waters, especially without a complete and public Clean Water Act use attainability analysis for each water body.

Rulemaking Petition Signatures (2021-04)

First and Last Name	Organization Name (if applicable)	Street Address	City	Zip Code	State
Tom Anderson	Aligning with Nature	8010 275th Ave NE	North Branch	55056	MN
Peg Furshong	CURE (Clean Up The River Environment	117 S 1st Street	Montevideo	56265	MN
Tim King	Dreams United/Sueños Unidos	15261 County 38	Long Prairie	56347	MN
Libby Bent	Duluth for Clean Water	2423 E 2nd Street	Duluth	55812	MN
Michael Koppy	Duluth Superior Friends Meeting	1802 1st st.	Duluth	55802	MN
Scott Beauchamp	Friends of the Boundary Waters	2550 University Ave	St. Paul	55114	MN
Maureen Hackett	Howling For Wolves	PO Box 4099	Hopkins	55343	MN
Jane Dow	Mankato Zero Waste	37 Capri Drive	Mankato	56001	MN
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Matthew Schaut	Minnesota River Valley Audubon Chapte	3720 27th Ave S	Minneapolis	55406	MN
Shodo Spring	Mountains and Waters Alliance	16922 Cabot Ave	Faribault	55021	MN
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Kaare Melby	Organic Consumers Association	6771 South Silver Hill Drive	Finland	55603	MN
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Terry Ford	Echo Spirits at Saint Joan of Arc Catholic Church	3549 Hennepin Avenue	Minneapolis	55408	MN
Tanya Beyer Hovi	Epiphanies Afield	7898 Hovi's Road	Virginia	55792	MN
John Beaton	Fairhaven Farm	5818 Munger Shaw Rd	Saginaw	55779	MN
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Deanne Roquet		315 West Oxford Street	Duluth	55803	MN

Rulemaking Petition Signatures (2021-04)

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Emrys Stramer		2416 17th Ave S	Minneapolis	55404	MN
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Marlys Sushoreba		8119 Mark Lake Road	Side Lake	55781	MN
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Candace Dow		1425 W 28th St, #315	Minneapolis	55408	MN
Lisa Ciorlieri		2332 Branch Street	Duluth	55812	MN
Paul Ryals		74 375th Avenue NW	Stanchfield	55080	MN
Connie Grundhofer		235 Linda Ave	Circle Pines	55014	MN
Peggy Roeske		1235 Gun Club Rd., Apt. 226	White Bear Lake	55110	MN
Lisa Ragsdale		2009 Bryant Ave. S., # 4	Minneapolis	55405-2828	MN
Jean O. Haslett		312 Linden St. N.	Northfield	55057	MN
Dr. Kenneth A. Harris		5099 157th St N	Hugo	55038	MN

Rulemaking Petition Signatures
(2021-04)

Scott Doblar		712 E. King	Winona	55987	MN
Harriet McCleary		2440 Stevens Ave. #2	Minneapolis	55404	MN
Nan Corliss		10300 Morris Rd	Bloomington	55437	MN
Randy Nies		3407 Harriet Ave. S. Apt. 2	Minneapolis	55408	MN
Shauna Armstrong		911 22nd ave s	Minneapolis	55404	MN
Richard Fish		5345 37th Ave So	Minneapolis	55417	MN
Verlaine Halvorsen		3510 The Mall	Minnetonka	55345	MN
Emma Needham		516 1/2 N 9th St	Brainerd	56401	MN
Raymond Bissonnette		143 Dahlia Street	Mahtomedi	55115	MN
Scott Russell		3124 44th Ave. So.	Minneapolis	55406	MN
Susan Brust		7700 N Field Ridge Road	Grant	55110	MN
William Cronin		36 Barton Ave SE	Minneapolis	55414	MN
Ken Engelhart		4724 E 45th Street	Minneapolis	55406	MN
Tina Krauz		701 W 5th St	Grand Marais	55604	MN
Kimberly Nieman		4550 Orchid Circle	Plymouth	55446	MN
Margi Preus		1747 Columbus Ave	Duluth	55803	MN
Sheila Schally		1104 Creekside Circle	Stillwater	55082	MN
Elizabeth Ulrich		10942 Rhode Island Ave S	Bloomington	55438	MN
Kimberly Lewis		1500 Lasalle Ave., #418	Minneapolis	55403	MN
Peggy Doerrie		3318 Grimes Avenue North	Robbinsdale	55422	MN
Cynthea Gillespie		556 Mariner Way	Woodbury	55129	MN
Lynn Bode		231 Hickory Street	Duluth	55811	MN
Meredith Myers-Petro		2826 39th Ave S	Minneapolis	55406	MN
Pamela Strom		1229 Hague Avenue	St. Paul	55104	MN
Eric Morrison		1202 Cherokee Ave.	West St. Paul	55118	MN
Edna Mullen		1272 Richland Avenue	St. Charles	55972	MN
Jess Koski		44 Reservation River Rd	Grand Portage	55606	MN
DyAnn Andybur		4119 McCulloch St	Duluth	55804	MN
Kevin Lanigan		3979 Trotters Court	Eagan	55123	MN
Michelle Strangis		1800 Irving Ave S	Minneapolis	55403	MN
Todd Wade		3443 Jasper Ct. NE	Rochester	55906	MN
Tania Aubid		46811 196th Pl	McGregor	55760	MN
Andrea West		4055 White Bear Avenue	White Bear Lake	55110	MN
Sid Pranke		84 Wabasha Street South, #	St. Paul	55107	MN
Sally Harris		1506 Laurel Av Lower	St. Paul	55104	MN
Brett Ostby		617 20th Street NE	Rochester	55906	MN
Cheryl Gonia		1330 Highland Dr	Winona	55987	MN
Michael Alexander		78 10th Street East	St. Paul	55101	MN
Kara Larson		733 Larch st.	Cloquet	55720	MN
Hannah Rovegno		125 5th St NW	East Grand Forks	56721	MN
Patrick Murphy		951 Iglehart Ave	St. Paul	55104	MN
Jalene Eden		17139 Groningen Rd	Sandstone	55072	MN
Jami Halder		12 N 64th Ave west	Duluth	55807	MN
Elizabeth & Andrew Urban		1347 Walsh Road	Ely	55731	MN
Dianne Hudson		701 West 5th Street	Grand Marais	55604	MN
Kevin Proescholdt		2833 43rd Ave S	Minneapolis	55406	MN
C. M. Smiley		10516 France Ave S #315	Bloomington	55431	MN
Kimberly Feilmeyer		935 Linwood	St. Paul	55105	MN
Denise Mack		470 Ely Street NE	Fridley	55432	MN
Barbara Jones		PO Box 94	Ely	55731	MN
Donna kamuth		328 Gunflint Trail	Grand Marais	55604	MN
Erin Enger		5941 Wisconsin Cir N	New Hope	55428	MN
Michael Pfeifer		11326 Rosemill lane north	Champlin	55316	MN
Richard Grant Hawthorne		4230 Abbott Av S	Minneapolis	55410	MN
Jean Haslett		312 Linden St. N.	Northfield	55057-1425	MN
Merikay Garrett		2107 Park Lake Ln	Mahtowa	55707	MN
Anne Hejny		1601 Dupont Avenue N	Minneapolis	55411	MN
Julia Bach		721 W	Minneapolis	55409	MN

Rulemaking Petition Signatures (2021-04)

Larry Bogolub		1424 Lincoln Ave	St. Paul	55105	MN
Eric Ristau		1844 Bayard Ave	St. Paul	55116	MN
MaryLou Wilm		2919 45th Av S	Mpls	55406	MN
Gwen and Mason Myers		12009 Hilloway Rd W	Minnetonka	55305	MN
Analiese Miller		1028 Carrie Street	West St. Paul	55118	MN
Mark Lauderbaugh		12501 Nicollet Ave Unit 414	Burnsville	55337	MN
Richard Newmark		810 Woodduck Drive	Woodbury	55125	MN
Faye Duvall		2550 Manitou Island	White Bear Lake	55110	MN
Lori Huska		211 N 24th Ave E	Duluth	55812	MN
Doug Herron		4300 West River Parkway, #2	Minneapolis	55406	MN
June Stuhr		3033 46th Ave S	Minneapolis	55406	MN
Lynne Markus		9175 Pinehurst Road	Woodbury	55125	MN
Chris Turnwall		1121 44th Avenue N.E.	Columbia Heights	55421	MN
Carol Bechtel		4300 West River Pkwy S. #40	Minneapolis	55406	MN
Rena Nordlund		2536 Providence Rd	Duluth	55811	MN
Stephanie Digby		1682 Taylor Avenue West	St. Paul	55104	MN
Donald Pederson		4325 Tioga Street	Duluth	55804	MN
James Conway		4620 Valley DR NW	Rochester	55901-6508	MN
Jackie Metelak		521 Robert Ct.	St. Paul	55115	MN
Amy Freeman		528 E Camp St	Ely	55731	MN
Joan Janezich		10217 10th Ave Circle S	Bloomington	55420	MN
Lisa Fitzpatrick		5229 Peabody St	Duluth	55804	MN
Betsey Porter		10040 Penn Ave S	Bloomington	55431	MN
Doretta (Dorie) Reisenweber		101 West Kent Road	Duluth	55812-1152	MN
Nancy Giguere		1471 Edmund Ave	St. Paul	55104	MN
Zabelle Stodola		131 N Hawthorne Road	Duluth	55812	MN
Candice Pierce		5192 LaVaque Junction Roa	Hermantown	55811	MN
Kelsey Murphy		4945 Countryside Drive	Shoreview	55126	MN
Maureen K. Johnson		6763 253rd Ave. NE	Stacy	55079	MN
Susan Darley-Hill		1710 E 7th St	Duluth	55812	MN
Brian Hill		1710 E 7th St	Duluth	55812	MN
Linda Herron		2617 E. Fifth St.	Duluth	55812	MN
Earle Tonra		3911 Girard Av N	Minneapolis	55412	MN
David Reisenweber		101 W Kent Rd	Duluth	55812	MN
Karen Reichensperger		1199 Minnesota Blvd	Ely	55731	MN
Kathryn Null		850 Egret Lane	Waconia	55387	MN
Lorna Landgren		1235 N Airport Rd	Cook	55723	MN
Kathleen Gates		1006 W Lyon Ave	Lake City	5504q	MN
Katren Garrett		2107 Park Lake Ln	Mahtowa	55707	MN
Jordan Langner		1028 Carrie St	West St. Paul	55118	MN
Nicole Everling		1639 Sherwood Way	Eagan	55122	MN
Jon Hayenga		421 2nd St NW	Stewartville	55976	MN
Steven George		5970 Blesner Lake Rd	Finland	55603	MN
Andrew St. Croix		5412 Avondale St.	Duluth	55804	MN
Elizabeth Songalia		649 Waseca St.	St. Paul	55107	MN
Samuel Engel		4424 30th ave s	Minneapolis	55406	MN
Peter Borden		896 Sherwood Avenue	St. Paul	55106	MN
Michael Overend		557 Scenic Drive	Two Harbors	55616	MN
Jennifer Schally		1104 Creekside Circle	Stillwater	55082	MN
Pete McDonnell		1111 Minnesota Ave NW	Bemidji	56601	MN
Penny Cragun		3780 London Rd	Duluth	55804	MN
Nancy Haarmeyer		19 Old Ski Hill Road	Grand Marais	55604	MN
Meg Kearnd		52t Sparkman Ave	Duluth	55803	MN
Mary Androff		2201 Jackson Circle	Marine on St. Croix	55047	MN
D. Jones-Williams		1743 #7 Gervais Ave.	Maplewood	55109	MN
Dave Crawford		1520 Lexington Parkway Nor	St. Paul	55117	MN
Cheryl LaPlante		9137 McCamus Rd	Brookston	55711	MN
Marie Nickell		10526 County 113	Mabel	55954	MN

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Gay Trachsel		420 W Faribault St	Duluth	55803	MN
Andrea Childs		2240 Devin Lane	Long Lake	55356	MN
Steve Jorgenson		36901 Xenon St NW	Princeton	55371	MN
Jackie Smolen		11309 Oakvale rd n	Minnetonka	55305	MN
Karen Anderson		5630 Mahoney Ave	Minnetonka	55345	MN
Jeri Thurber		2925 Monterey Ave	St. Louis Park	55416	MN
Lisa Hanes Goodlander		2323 Windsor Lane	Woodbury	55125	MN
Ruth Katz		3380 Highway 21	Babbitt	55706	MN
Rowan Glaser		1606 Breda Ave	St. Paul	55108	MN
Joe Chovan		2000 15th ave se	St. Cloud	56304	MN
Janice Johnson		3329 47Ave. S.	Minneapolis	55406	MN
Nellie Scheffler		5234 Howard Gnesen Rd	Duluth	55803	MN
Jean Elton Turbes		1004 Chester Park Drive	Duluth	55812	MN
Hilary Bown		5234 Howard Gnesen Rd.	Duluth	55803	MN
Jane Soltau		2002 East 4th Street	Duluth	55812	MN
Marjorie Pitz		182 Mounds Blvd.	St. Paul	55106	MN
Bruce Tyler		1471 Edmund Ave	St. Paul	55104	MN
Jess Cheney		5445 Portland	Minneapolis	55417	MN
Linda Vukson		5331 Juniata Street	Duluth	55804-1341	MN
Rebecca Cramer		3148 29th Ave S	Minneapolis	55406-1922	MN
Thomas Sullivan		4061 209th LN NW	Oak Grove	55303	MN
Theresa Koenig		4756 5th Ave s	Duluth	55803	MN
Steve Lelchuk		3943 Bryant Ave S Apt 9	Minneapolis	55409	MN
Tracy Kugler		1316 Seminary Ave.	St. Paul	55104	MN
GJean Bierly		505 E 17th St	Blue Earth	56013	MN
John Gaunt		4351 Aldrich Avenue South	Minneapolis	55409	MN
Kenneth Kaseforth		10724 Beard Ave. S.	Bloomington	55431	MN
Mary Arps Thompson		1370 White Lake Dr	Duluth	55803	MN
Laura Schauland		9609 Arrowhead	Isabella	55607	MN
Robert Scheierl		1109 NE 5th avenue	Grand Rapids	55744	MN
Jami Gaither		25288 County 2	Shevlin	56676	MN
Sharon Clark		735 Nelson Rd.	Maple Plain	55359	MN
Shannon Barber-Meyer		13463 Ironwood Rd	Ely	55731	MN
William Thomas		3415 Harriet Avenue	Minneapolis	55408	MN
Ann Miller		2921 E 1st St	Duluth	55812	MN
Carol Theobald		1237 E Madison St	Ely	55731	MN
Patricia Buck		82 Kelsey Whiteface Rd	Cotton	55724	MN
Joan Hughex		4088 Utica Ave S	Minneapolis	55416	MN
Joseph Wenzel		93 Midwest Ave. N	Lake Elmo	55042	MN
Waverly Reibel		701 N. 2nd Street, Apt. 517	Minneapolis	55401	MN
Annika Simon		269 Meadowood Lane	Vadnais Heights	55127	MN
Debra George		1345 Cohansey St	St. Paul	55117	MN
Cristina Czaia		4014 15th Ave S	Minneapolis	55407	MN
Mary Hoffman		12522 Parkwood Dr.	Burnsville	55337	MN
Sherry Rovig		1982 Lismore Rd	Duluth	55804	MN
Lisa Pugh		13990 Romberg Shores Rd	Ely	55731	MN
Kenneth Matysik		4819 Thomas Ave. S.	Minneapolis	55410	MN
Amelia Hummel		4368 France Ave N	Robbinsdale	55422-1565	MN
Laverne Capan		1522 No. 8th Ave East	Duluth	55805	MN
Julia Kloehn		5879 Nikolai Rd	Finland	55603	MN
Bonnie Elmquist		15790 25th Ct N	Plymouth	55447	MN
Lisa Pollei		9578 Thunderbluff Rd NW	Oronoco	55960	MN
Linville Doan		9900 Hudson Blvd. #304	Duluth	55808	MN
Rebecca Shedd		4554 Wentworth Ave	Minneapolis	55419	MN
Hilary Sandall		15573 River Rd	North Branch	55056	MN
Merikay Garrett		2107 Park Lake Lane	Mahtowa	55707	MN
Esther Ouray		3351 Columbus Ave S	Minneapolis	55407	MN
Chris Cowen		1373 Breda	St. Paul	55108	MN

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Dr. Tracy Sides		11423 Neal Ave N	Stillwater	55072	MN
Sue Menter		428 Bear Ave S	St. Paul	55127	MN
Susan Borden		896 Sherwood Ave	St. Paul	55106	MN
Glenn Witte		3804 Hayes St NE	Columbia Heights	55421	MN
Elizabeth Burr		2025 Fairmount Ave.	St. Paul	55105-1548	MN
Alva Pingel		13894 Birchwood Ave,	Rosemount	55068	MN
Jen Pearson		4532 London Rd.	Duluth	55804	MN
Zoe Bird		4918 37th Ave. So.	Minneapolis	55417	MN
Eric Hedeen		16759 French LN NE	Bemidji	56601	MN
Anne Reich		751 Pine Cone Trail	Marine on St. Croix	55047	MN
Annah Gardner		1906 1st Ave S	Minneapolis	55403	MN
Jane Thimke		1728 E. 1st St. #3	Duluth	55812	MN
Joe Dufficy		1919 Cleveland Street NE	Minneapolis	55418	MN
John Kantar		3426 Saint Paul Avenue Min	Minneapolis	55416	MN
Karen Graham		11600 37th Ave N	Plymouth	55441	MN
Clara Ueland		1902 Homestead Trail	Long Lake	55356	MN
Bryan Wyberg		2458 Farrington Cir	Roseville	55113	MN
C. John Hildebrand		1220 Powderhorn Terrace #2	Minneapolis	55407	MN
Nancois Congere		131 Monroe ST	Anoka	55303	MN
Joe May		10533 W River Rd	Brooklyn Park	55443-1231	MN
James Heutmaker		14813 Maple Trl SE	Prior Lake	55372	MN
Therese Zemlin		1461 Kent St	St. Paul	55117	MN
Signe Martell		2149 Goodrich Ave	St. Paul	55105	MN
Bruce Johnson		6763 253rd Ave NE	Stacy	55079	MN
Kristin Rolf		9619 Pine Ln	Britt	55710	MN
Joan Beaver		325 Edgewood Ave	Stillwater	55082	MN
Joyce Pfaff		1920 So 1st St #2205	Minneapolis	55454	MN
Paul Wotzks		13226 N Hwy 74	Altura	55910	MN
Pat Shea		5317 Blake Road	Minneapolis	55436	MN
Mark and Debra Thurlo		14601 Atrium Way, Unit 329	Minnetonka	55345-4767	MN
Kathleen Stuebner		17635 24th Ave N	Plymouth	55447	MN
April Narcisse		8140 Rhode Island Circle	Bloomington	55438-3400	MN
Joel Roberts		1882 Colvin Ave St	St. Paul	55116	MN
Carl Dawson		40 Judith Drv.	Chaska	55318	MN
Terry Richmond		2900 County Road 19	Maple Plain	55359	MN
Dennis Kaleta		181 Old Ski Hill Rd.	Grand Marais	55604	MN
Tim Wallace		8982 Norway Ridge Rd	Zim	55738	MN
Jon Damon		10932 Beard Ave S	Bloomington	55431	MN
Sundae Morse		603 3rd St W	Northfield	55057	MN
Lisa Bergerud		3024 36th Ave s	Minneapolis	55406	MN
Susan Knapp		360 Third St	Marine on St Croix	55047	MN
Jerilee Reilly		20300 Franconia Trail	Shafer	55074	MN
Nancy Sampson		1660 Lexington Pkwy. N.	St. Paul	55117	MN
Karen Reece		1420 Frankson Avenue	St. Paul	55108	MN
Sharon Kutter		10917 County 47	Grey Eagle	56336	MN
Ann Katherine		30242 Cababa First road	Grand Rapids	55744	MN
Jim Fournier		740 Mississippi River Blvd S	St. Paul	55116	MN
Wendy Mcculley		Montrose Place	St. Paul	55104	MN
Jean Larson		1885 Tatum St.	St. Paul	55113	MN
Richard Newmark		810 Woodduck Dr	Woodbury	55125	MN
Jeremy Olmscheid		901 1st Ave	Albany	56307-9485	MN
Linda Peterson		3001 Washburn Place	Bloomington	55431	MN
Jackie Holmbeck		17620 25th Ave. No.	Plymouth	55447	MN
Liz Welch		3447 10th Ave SO	Minneapolis	55407	MN
Pat Fillmore		16 Lindsay Court	St. Cloud	56301	MN
Nan Harmeyer		Old Ski Hill Road	Grand Marais	55604	MN
Denny Wagner		360 1st St N Apt 249	Minneapolis	55401	MN
Jean Bixley		32230 Roanoke Street NW	Cambridge	55008	MN

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Emily Levang		1346 W Arrowhead Rd, Apt A	Duluth	55812	MN
Valerie Myntti		1166 MN Blvd	Ely	55731	MN
Mary Creighton		501 6th St. S.	Virginia	55792	MN
Thomas Childs		8326 Robert St.	Babbitt	55706	MN
Erin Jordahl Redlin		3012 Armour Terrace	St. Anthony	55418	MN
Lloyd Hansen		3001 Washburn Pl	Bloomington	55431	MN
James Herther		1585 Cohansey St. Apt 201	St. Paul	55117	MN
Kathelen Weinberg		4640 Cascade Beach Road	Lutsen	55612	MN
Shannon Anderson		708 South Ave	North Mankato	56003	MN
Gretchen Bratvold		3444 Edmund Blvd	Minneapolis	55406	MN
Jeanne Fahlstrom		3111 Garfield St. NE	Minneapolis	55418	MN
Lee Waltz		3080 Rush Point Drive	Rush City	55069	MN
Richard Hawthorne		4230 Abbott Avenue South	Minneapolis	55410	MN
Barry Knapp		1165 Knoll Ct NW	Rochester	55901	MN
Nadja Reubenova		4537 29th Avenue South	Minneapolis	55406	MN
Dean Borgeson		36030 Bonnie Lakes Rd	Crosslake	56442	MN
Doretta Reisenweber		101 West Kent Road	Duluth	55812	MN
Anna Yliniemi		2103 W 11th St	Duluth	55806	MN
David Gagne		3517 E. 26th Street	Minneapolis	55406	MN
Patricia Moses		478 Bayview Dr.	Roseville	55113	MN
Heidi Sobanja		102 Sobanja Lane	Grand Marais	55604	MN
Gio Cerise		16421 Olivine St NW	Ramsey	55303	MN
Diane Tessari		5375 Eureka Road	Excelsior	55331	MN
Michael Reid		1251 Edmund Ave	St. Paul	55104	MN
Dennis Good		7140 N. Dark Lake Rd.	Britt	55710	MN
Barbara Evan		525 Burlington Rd	St. Paul	55119	MN
Ellen Hinchcliffe		3545 46th Ave S	Minneapolis	55406	MN
Robert Kosuth		1224 E. 11th Street	Duluth	55805	MN
Meg Kearns		525 Sparkman Ave.	Duluth	55803	MN
Thomas Matkovits		9612 Lonsdale Circle	Minnetonka	55305	MN
Kathleen Hutchins		537 17th Ave NW	St. Paul	55112	MN
Jamie Hoerter		2448 Hutchinson RD	Duluth	55811	MN
Joannne Englund		2650 University Ave. West #3	St. Paul	55114-1926	MN
Diana Cumming		3210 Cleveland St. NE	Minneapolis	55418	MN
Mark Fitzpatrick		5229 Peabody St.	Duluth	55804	MN
Catherine Reece		8155 Cameo Circle	Inver Grove Height	55076	MN
Alice Madden		31st and 16th Av S	Minneapolis	55407	MN
William K. Dustin		4654 Linden Trail N	Lake Elmo	55042	MN
Lauren Mitchell		5624 45th Ave N	Crystal	55422	MN
Dominic Cerise		16421 Olivine St NW	Ramsey	55303	MN
J.Isabelle Dyck		211 2nd St. NW #217	Rochester	55901	MN
Kate Dougherty		2117 Hillcrest Drive	Duluth	55811	MN
Julie Light		1492 Wedgewood Road	Albert Lea	56007	MN
Troy Rogers		2536 Jefferson Street	Duluth	55812	MN
Carole Rust		1826 N. Alameda St.	Roseville	55113	MN
Mary Dylkowski		23140 W Martin Lake Dr	Stacy	55079	MN
Jennifer Keck		703 Brian Lane	Brainerd	56401	MN
Leslie McDonald		15824 Park Terrace Drive	Eden Prairie	55346	MN
Robin Raplinger		916 17th St N	Virginia	55792	MN
John Rusterholz		2787 Marion St	Roseville	55113	MN
Candice Pierce		5192 LaVaque Junction Roa	Hermantown	55811	MN
Joe Foss		6030 6th St. NE	Fridley	55432	MN
Linda Rolf		1900 Ave S, 26	Minneapolis	55403	MN
Gail Frethem		5241 10th Ave. So.	Minneapolis	55417	MN
Lawrence Landherr		563 Co Rd B West	Roseville	55113	MN
Barbara Buehl		9965 Windsor Terrace	Eden Prairie	55347	MN
Norm Herron		2617 E. Fifth St.	Duluth	55812	MN
Karen Johnson		3813 Gauvitte St.	Columbia Heights	55421	MN

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Erich Wunderlich		413 5th St SE	Minneapolis	55414	MN
Julius Salinas		95 Stillmeadow Road	Esko	55733	MN
Carla Arneson		1177 Ring Rock Rd	Ely	55731	MN
Maureen Skelly		8050 Central Ave.	Spring Lake Park	55432	MN
Wesley Sisson		133 Summit St.	Duluth	55803	MN
Rob Bullis		19088 Dodge St Nw	Elk River	55330	MN
Michael Miles		7340 Kochia Lane	Victoria	55386	MN
Grant Thrall		4038 Blaisdell Avenue South	Minneapolis	55409	MN
Martha Baxter		3709 Grand Way	St. Louis Park	55416	MN
Steven Kingsbury		26415 Pigeon Loft Rd NE	Stacy	55079	MN
Thomas Swedberg		182 Scenic Drive	Knife River	55609	MN
Linda Mockler		3091 Evelyn St	Roseville	55113	MN
Jack S. Sneve		4484 Normanna Road	Duluth	55803	MN
Mary Leinfelder		2215 Minneapolis Ave.	Minneapolis	55406	MN
Karen Hulstrand		1204 Everett st. s.	Stillwater	55082	MN
Pat Becchetti		513 S. 5th St.	Stillwater	55082	MN
Sonja Miedtke		71977 200 Ave	Hayfield	55940	MN
Catherine Lundoff		3816 13th Ave. So.	Minneapolis	55407	MN
Sharon Bachman		13000 Sylvan Ave	Lindstrom	55045	MN
Elizabeth Choma		1929 Fremont Ave. S. #34	Minneapolis	55403	MN
Mary Thompson		1370 White Lake Dr	Duluth	55803	MN
Joanna Padden		209 Main St. S	St. Michael	55376	MN
Amy Grace		722 Everett St S	Stillwater	55082	MN
Sandy Loney		5730 Birchdale Road	Brainerd	56401	MN
Cecilia Dingledy		3443 Jasper Ct NE	Rochester	55906	MN
David Carlson		5818 CR2	Ft. Ripley	56449	MN
Kimberly Swenson-Zakula		4650 St James Gate	Excelsior	55331	MN
Elene Loecher		4300 W. River Pkwy. #205	Minneapolis	55406	MN
Tara Widner		4127 Irving Ave N	Minneapolis	55412	MN
Judy Enenstein		2856 Irving Ave. S.	Minneapolis	55408	MN
Susan Dragsten		221 1st Ave N.E. #32	Minneapolis	55413	MN
Anita Gille		4117 w 8th st	Duluth	55807	MN
Barb Powell		1081 Felty Ave SE #303	Rochester	55904	MN
Jo-Ann Sramek		4882 Woodridge Drive	Duluth	55811	MN
Michael Keepper		105 Market St W	Wabasha	55981	MN
Kristine Hites		3609 Bloomington Ave.	Minneapolis	55407	MN
Jan Karon		1112 South Lake Ave	Duluth	55802	MN
Liz Dailey		512 W 22nd St	Minneapolis	55405	MN
Sue Halligan		1190 Schooner Way	Woodbury	55125	MN
Kathie Cerra		4522 Arden Ave S	Edina	55424	MN
Lindsey Lang		2090 Passi Rd	Ely	55731	MN
Hannah Watson		920 21st St NW #9	Bemidji	56601	MN
Timothy Alvar		2849 Lakewood Junction Rd	Duluth	55804	MN
John Harrington		30726 Ivywood Trl	Stacy	55079-9283	MN
David Evans		5440 1st Ave S	Minneapolis	55419	MN
Mary Johannsen		2418 Aldrich Ave North	Minneapolis	55411	MN
Michael Steffes		3098 East Castle Danger Rd	Two Harbors	55616-2007	MN
Wendy Ward		901 Wilke Street	Marine on St. Croix	55047	MN
Ron Wetzell		4837 East Upland Crest	Columbia Heights	55421	MN
Elizabeth Dailey		512 W 22nd St	Minneapolis	55405-3201	MN
Eileen Anderson		5356 Holiday Road	Minnetonka	55345	MN
Michael Poisson		9273 Hamline Ave	Lexington	55014	MN
Lois Norrgard		10368 Colunbus Circle,	Bloomington	55420	MN
Duane Lee		15428 Panola Dr	Lindstrom	55045	MN
Chad Jones		1220 stanford ave	Duluth	55811	MN
Joanne Sieck		5877 River Ridge	Rochester	55906	MN
Karrie Vrabel		3844 Bloomington Ave	Minneapolis	55407	MN
Dylan Koltz-Hale		1310 E 10th St.	Duluth	55805	MN

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(2021-04)

Amy Fish		16550 Herbs Road	Detroit Lakes	56501	MN
Colleen Simmons		5917 Grass Lake Terrace	Minneapolis	55419	MN
Mary Miller		3804 Cedar Lake Place	Minneapolis	55416	MN
Scott Vizecky		3194 Co Hwy 4	Hendricks	56136	MN
Tony Griffin		18 Larch drive	Duluth	55810	MN
Kristin Daniels		1815 White Bear Ct	White Bear Lake	55110	MN
Anita Rauschenfels		721 W 5th St	Duluth	55806-2437	MN
Gary Payne		3947 River Ridge Drive	Brainerd	56401	MN
Donna Seabloom		18829 Waco St NW	Elk River	55330	MN
Karen Peters		3420 Cleveland St. NE	Minneapolis	55418	MN
Sally Fineday		24056 Cap Endres Road NE	Cass Lake	56633	MN
Shannon Hedren		6404 Warren Ave	Edina	55439	MN
Diane Bublitz		1514 Aspen Lane	St Cloud	56303	MN
Nancy Cosgriff		2115 Jackson Circle	Marine on St. Croix	55047	MN
Paul Steinhauser		600 Birchwood Ave	Birchwood	55110	MN
Carol Weber		5223 Silver Maple Circle	Minnetonka	55343	MN
Dodd Cosgrove		756 Widsten Circle	Wayzata	55391	MN
Lynn Levine		2301 Westridge Lane	Minneapolis	55416	MN
Paula Rusterholz		2787 Marion St	Roseville	55113	MN
Kaitlyn Featherstone		1008 Bush Street	Red Wing	55066	MN
Dawn Tuveson		1046 Wyncrest Ct.	Woodbury	55129	MN
Sierra Erickson		2105 Wisconsin ave.	Benson	56215	MN
Jaci Christenson		12309 Fiona Ave N	White Bear Lake	55110	MN
Nichole McDonald		16401 Irvine Ave NW	Bemidji	56601	MN
Renee Butters		3272 Greenbrier Street	Vadnais Heights	55127	MN
Diane Hiniker		147 Bloomquist Mtn. Rd.	Grand Marais	55604	MN
Bonita Schwartz		13376 Elaine court	Savage	55378	MN
David Washburn		225 9th St E, #601	St. Paul	55101	MN
Paula Tompkins		1220 13th St. N.	St. Cloud	56303	MN
Susan Spaeth		12 Bubalo Dr	Duluth	55808	MN
Deborah Fischer		825 Kenwood Ave	Duluth	55811	MN

STATE OF MINNESOTA
MINNESOTA POLLUTION CONTROL AGENCY

STATE OF MINNESOTA)
) ss.
COUNTY OF HENNEPIN)

AFFIDAVIT OF WAVERLY REIBEL
FOR RULEMAKING PETITION

Waverly Reibel being duly sworn, deposes and says:

1. My name is Waverly Reibel and I live at 701 N. 2nd St, Apt. 517 in Minneapolis.

2. I have a B.S. in Environmental Science and a Master's Degree of Environmental Management, and I serve as WaterLegacy's Community Engagement Coordinator. I am responsible for a variety of communications and administrative tasks and functions for WaterLegacy, including management of online communications and the database where electronic signatures are logged.

3. On Thursday, March 25, 2021, WaterLegacy began distributing online through Google Forms survey administration system a petition requesting that the Minnesota Pollution Control Agency amend Minnesota class 2 and chapter 7050 rules and change its priorities to protect people and clean water (the "Rulemaking Petition"). The Rulemaking Petition language in Attachment A is identical to the language that was provided in the online Petition. For an electronic signature to be submitted, WaterLegacy required a full name, address, city, state and zip code.

4. On Thursday, April 8, 2021, I exported the information provided by all electronic signers of the Rulemaking Petition between March 25, 2021 and April 8, 2021 into a Microsoft Excel document. These signatures are provided with the text of the Rulemaking Petition in Attachment A.

5. I have carefully reviewed the electronic record of signatures to the Rulemaking Petition to verify that Attachment A accurately reflects all persons who signed the Rulemaking Petition and that no organizations or individuals are identified in Attachment A who did not, in fact, sign the Rulemaking Petition. As of April 8, 2021, 33 organizations and 460 individuals signed the Rulemaking Petition.

FURTHER YOUR AFFIANT SAYETH NOT.

Waverly Reibel

WAVERLY REIBEL

Subscribed and sworn to before me this day April 9, 2021.

Notary Public

KURTIS M KRAUSE
NOTARY PUBLIC - MINNESOTA
My Commission Expires Jan. 31, 2025

My Commission expires: 1.31.25

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 15

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2/20/2018 3:34 PM
Hennepin County, MN

Exhibit 15
WL Class 1 Rule Comments

STATE OF MINNESOTA
COUNTY OF HENNEPIN

DISTRICT COURT
FOURTH JUDICIAL DISTRICT

State of Minnesota, by its Attorney General, Lori Swanson, its Commissioner of Pollution Control, John Linc Stine, and its Commissioner of Natural Resources, Tom Landwehr,

Plaintiff,

vs.

3M Company,

Defendant.

Case Type: Other Civil
Judge Kevin S. Burke
Court File No. 27-CV-10-28862

AGREEMENT AND ORDER

The State of Minnesota, by its Attorney General and its Commissioners of Pollution Control and Natural Resources, and 3M Company voluntarily enter into this Agreement, which fully and finally resolves the above-entitled matter.

I. DEFINITIONS

Whenever the terms listed below are used in this Agreement, the following definitions shall apply:

1. "3M" shall mean 3M Company, a corporation incorporated in the State of Delaware with its principal place of business in Maplewood, Minnesota.

2. "3M Grant for Water Quality and Sustainability Fund" shall mean a separate account established in the State's Remediation Fund pursuant to Minn. Stat. §§ 115B.17 subd. 7 and 116.155, subd. 3(3).

3. "Attorney General" shall mean the Attorney General of the State of Minnesota (or her authorized designee) and her successors, and the Minnesota Attorney General's Office.

4. "DNR" shall mean the Minnesota Department of Natural Resources, a statutory agency of the State of Minnesota responsible for administering and enforcing Minnesota statutes

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and rules relating to the preservation, conservation, management and regulation of natural resources of the State. *See* Minn. Stat. Chs. 84, 85, 94 and 103G. Reference to the DNR shall include its Commissioner, Tom Landwehr (or his authorized designee(s)), and his successors.

5. "Effective Date" shall mean the date the Court issues its Order approving this Agreement.

6. "Grant" shall mean the grant described in paragraph 13.

7. "MPCA" shall mean the Minnesota Pollution Control Agency, a statutory agency of the State of Minnesota responsible for administering and enforcing Minnesota statutes and rules relating to water, land and air pollution. *See* Minn. Stat. Chs. 115, 115B and 116. Reference to the MPCA shall include its current Commissioner, John Linc Stine (or his authorized designee(s)), and his predecessor and successors.

8. "Parties" shall mean collectively 3M and the State.

9. "PFCs" shall mean per- and poly-fluorinated chemicals.

10. "SACO" shall mean the 2007 Settlement Agreement and Consent Order entered by and between the MPCA and 3M on May 22, 2007.

11. "State" shall mean the Attorney General, the MPCA, and the DNR.

12. "Working Group" shall mean a working group established by the MPCA and the DNR, consisting of representatives of the MPCA, the DNR, East Metropolitan Area municipalities, and 3M. The composition of the Working Group may vary depending on the project(s) at issue set forth in paragraphs 14.A.-.C.

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II. PAYMENT

13. 3M will make a Grant in the amount of $850 million to the State which shall be held in the 3M Grant for Water Quality and Sustainability Fund, within fifteen (15) days from the Effective Date of this Agreement.

14. The MPCA and/or the DNR shall use the Grant (net of costs, fees, and expenses), and any interest earned or any other appreciation in value, for projects that are reasonable and necessary to achieve the purposes of this Agreement:

A. As the first and highest priority, the MPCA and/or the DNR shall utilize the Grant referenced in paragraph 13 above to enhance the quality, quantity and sustainability of the drinking water in the East Metropolitan Area, which shall include, but is not necessarily limited to, the cities of Woodbury, Oakdale, Lake Elmo, Cottage Grove, St. Paul Park, Afton, and Newport and the townships of West Lakeland and Grey Cloud Island. The goal of this highest priority work is to ensure clean drinking water in sufficient supply to residents and businesses in the East Metropolitan Area to meet their current and future water needs. Examples of projects in this first priority may include, but are not limited to, the development of alternative drinking water sources for municipalities and individual households (including but not limited to creation or relocation of municipal wells), the treatment of existing water supplies, water conservation and efficiency, open space acquisition, and groundwater recharge (including projects that encourage, enhance, and assist groundwater recharge). For individual households, projects may include, but are not limited to, connecting those residences to municipal water supplies, providing individual treatment systems, or constructing new wells. The MPCA shall conduct a source assessment and feasibility study regarding the role of the Valley Branch Water District's project known as Project 1007 in the conveyance of PFCs in the environment. In

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selecting and performing activities pursuant to this paragraph, the State shall prioritize water supplies where health based values, health risk limits, and/or health risk indices for PFCs are exceeded.

B. As the second highest priority, and after the MPCA and/or the DNR have reasonably achieved the goal set forth above in paragraph 14.A., the MPCA and/or the DNR shall utilize the Grant on projects that restore and enhance aquatic resources, wildlife, habitat, fishing, resource improvement, and outdoor recreational opportunities in the East Metropolitan Area and in downstream areas of the Mississippi and St. Croix Rivers. These projects may include, but are not limited to: (i) aquatic habitat and water resource protection and restoration; (ii) terrestrial and water trails; (iii) boat ramps and/or fishing piers along the Mississippi River, Lake Elmo, or other waterbodies in or downstream of the East Metropolitan Area; (iv) the restoration of wildlife habitat; and (v) implementation of other terrestrial conservation and recreational improvements in the same geographic area. While implementing the goal set forth above in paragraph 14.A., the MPCA and/or the DNR shall have immediate access of up to $20 million of Grant funds to undertake the goals set forth in this paragraph 14.B.

C. As the third highest priority, and if any portion of the Grant remains (other than the amounts set forth in paragraphs 15-16 below) after the MPCA and/or the DNR have reasonably achieved the goals set forth above in paragraph 14.A.-.B., the MPCA and/or the DNR shall utilize the Grant to fund residual, statewide water resources, habitat restoration, open space preservation, recreation improvements, and other sustainability projects.

15. The Grant includes reimbursement to the Remediation Fund for all costs of the MPCA under the SACO, except as provided in paragraph 19. Notwithstanding paragraph 14.A.-

.C. above, the MPCA shall have immediate access to the amount referenced in this paragraph for any lawful purpose as set forth in Minn. Stat. §§ 115B.20, subd. 2 and 116.155, subd. 2.

16. The Grant also includes reimbursement to the Remediation Fund in the amount of $300,441.95 for the reasonable costs incurred by the MPCA and/or the DNR for assessing damages to natural resources, pursuant to Minn. Stat. § 115B.04, subd. 1(3). Notwithstanding paragraph 14.A.-.C. above, the MPCA and/or the DNR shall have immediate access to the amount referenced in this paragraph for any lawful purpose as set forth in Minn. Stat. §§ 115B.20, subd. 2 and 116.155, subd. 2.

17. The MPCA and/or the DNR shall form a Working Group to identify and recommend projects referenced in paragraphs 14A.-C. above. Pursuant to Minn. Stat. §§ 116.155 and 115B.20, the MPCA and/or the DNR shall have the ultimate responsibility, in their discretion, to determine the projects to be implemented under this Agreement (provided that the MPCA and/or the DNR will adhere to the spending prioritizations described above). The MPCA and/or the DNR will also consult with municipalities and the Metropolitan Council as necessary and appropriate on implementation of projects under paragraph 14.A. above and may use Grant monies to reimburse those entities for projects undertaken that meet the goals set forth in paragraph 14.A. above. The MPCA and/or the DNR may use Grant monies to retain technical experts to assist the Working Group.

III. RELEASE OF CLAIMS AND DISMISSAL

18. In consideration of the stipulated relief, the sufficiency of which is acknowledged, including 3M's payments specified herein, the State fully and completely releases and waives against 3M and its affiliates, subsidiaries, parent corporations and companies, predecessors, successors, and current or former employees, directors, attorneys, shareholders, agents,

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representatives, insurers, and the like ("Released Parties"), any and all claims or causes of action known or unknown through the Effective Date of this Agreement, related to claims alleged in the State's Amended Complaint or that could have been alleged by the State in its Amended Complaint for natural resource damages, including under the Minnesota Environmental Response and Liability Act, the Minnesota Water Pollution Control Act, any statute or common law theory, arising out of or relating to 3M's manufacture, distribution, disposal or other environmental management of PFCs or the release of 3M PFCs into the environment. The MPCA also fully and completely releases and waives any and all claims against 3M relating to the MPCA's costs incurred in 2017 under the SACO. 3M fully and completely releases and waives any and all claims against the State relating to the Amended Complaint, including any claim for contribution and/or indemnity, and attorney fees and costs and expenses. 3M further fully and completely releases and waives any and all claims against the State relating to reimbursement of MPCA costs incurred in 2017 under the SACO.

19. The SACO shall remain in place, and 3M shall continue to be bound by the terms of the SACO, including the continuation of reimbursement of the MPCA's costs and 3M's ongoing implementation of the remedy approved by the MPCA for 3M's Cottage Grove, Woodbury, and Oakdale Sites. In addition, for a period of five (5) years after the Effective Date of this Agreement, 3M agrees to pay up to $40 million to fund the projects and/or activities set forth in paragraph VIII.B. of the SACO for temporary purposes, which shall include but are not limited to individual home water treatment systems that can be cost effectively connected within such five (5) year period to municipal systems, provision of bottled water, temporary municipal water treatment systems and the operation and maintenance of the temporary safe drinking water projects and activities. Otherwise, except for temporary measures referenced in the preceding

sentence, the Grant shall fund future projects that would have been payable under the SACO. If the Grant is depleted, the provisions of the SACO shall once again become operative. The Parties will annually review the continuing need for the SACO in light of the implementation of the projects outlined above, including projects related to the Washington County Landfill.

20. Within five (5) days from the Effective Date of this Agreement, the State and 3M will file a Stipulation of Dismissal with Prejudice, dismissing the Amended Complaint with prejudice and without attorneys' fees, expenses, and costs to either Party.

IV. GENERAL TERMS

21. The Parties are executing this Agreement for the sole purpose of settling and fully resolving the State's claims against 3M, which are disputed. Nothing about the Agreement shall constitute any admission by either Party of fault, responsibility, wrongdoing, or liability on the part of the Released Parties, nor does it constitute evidence of liability or wrongful conduct on the part of either Party, or any admission by either Party regarding the validity of any statutory or regulatory action by the State. Nothing in this Agreement shall be construed as an admission that 3M has legal responsibility for any contamination or other injury associated with the Washington County Landfill. This Agreement shall not be admissible in any future administrative or judicial proceeding as evidence of fault or liability in any investigation, claim, action, suit, or proceeding, or federal or state court or arbitration proceeding. Nothing in this Agreement shall relieve either Party of its obligation to comply with all applicable Minnesota and federal laws and regulations.

22. Nothing in this Agreement shall limit the Attorney General, the MPCA, and/or the DNR's ability to bring claims against any person or entity not covered by this Agreement.

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23. This Agreement may be executed in counterparts, each of which constitutes an original, and all of which shall constitute one and the same Agreement. This Agreement may be executed by facsimile or electronic copy in any image format.

24. The person signing this Agreement for 3M warrants that he or she is authorized to execute this Agreement, that 3M has been fully advised by its counsel before entering into the Agreement, and that he or she executes this Agreement in an official capacity that binds 3M. The persons signing this Agreement for the Attorney General, the MPCA, and the DNR warrant that they have been authorized to do so by the Attorney General, the MPCA, and the DNR, respectively, and they do so in their official capacities. This Agreement constitutes the full and complete terms of the agreement entered into by the Parties.

25. The Parties agree that the Hennepin County District Court shall retain jurisdiction over this matter for purposes of enforcing the Agreement, including any dispute between the Parties regarding selection and/or implementation of the priority projects as described in paragraph 14.A.-.C. above. The Parties request that, upon his retirement, the Honorable Kevin Burke shall be appointed by the Court as a Consensual Special Magistrate, pursuant to Minn. R. Gen. Prac. 114.02, to carry out the duties in the preceding sentence, with his reasonable fees and expenses to be paid with Grant monies. The Court shall also retain jurisdiction of this matter for purposes of enforcing the Order for Judgment. 3M and the State may each retain one expert to provide technical assistance in evaluating any issues that arise under this paragraph. The Parties agree that, before filing any motion under this paragraph, they shall meet and confer in an attempt to resolve any dispute and shall further mediate such dispute with Judge Burke prior to filing any motion with the Court in a further attempt to resolve any outstanding issues. If Judge

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Burke is unavailable, or if the Parties otherwise mutually agree, the Parties will select a mutually agreeable substitute to serve as a Consensual Special Magistrate.

26. The failure of 3M, the Attorney General, the MPCA, and/or the DNR to exercise any rights under this Agreement shall not be deemed a waiver of any right or any future rights.

27. If any part of this Agreement shall be found or held to be invalid or unenforceable by any court of competent jurisdiction, such invalidity or unenforceability shall not affect the remainder of this Agreement.

28. The Agreement shall be binding and enforceable against 3M, including any acquirer of 3M or its business.

29. This Agreement may be amended only by written agreement between the Parties and subject to approval by the Court.

30. This Agreement, including any issues relating to interpretation or enforcement, shall be governed by the laws of the State of Minnesota.

31. Each of the Parties is represented by counsel, participated in the drafting of this Agreement, and agrees that the Agreement's terms may not be construed against or in favor of any of the Parties by virtue of draftsmanship. The Parties agree to perform such further acts and to execute and deliver such further documents as may reasonably be necessary to carry out this Agreement.

THE PARTIES ENTER INTO AND APPROVE THIS AGREEMENT AND SUBMIT IT TO THE COURT SO THAT IT MAY BE APPROVED AND ENTERED AS AN ORDER.

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FOR THE STATE OF MINNESOTA

LORI SWANSON

Attorney General
State of Minnesota

Date: 2/20/18

Alan I. Gilbert (No. 0034678)
Solicitor General

445 Minnesota Street, Suite 900
St. Paul, Minnesota 55101-2127
(651) 757-1426 (Voice)
(651) 296-1410 (TTY)

FOR THE MINNESOTA POLLUTION CONTROL AGENCY

Date: 2/20/2018

JOHN LINC STINE
Commissioner
Minnesota Pollution Control Agency
520 Lafayette Road
St. Paul, MN 55155

FOR THE MINNESOTA DEPARTMENT OF NATURAL RESOURCES

Date: Feb. 20, 2018

TOM LANDWEHR
Commissioner
Minnesota Department of Natural Resources
500 Lafayette Road
St. Paul, MN 55155

FOR 3M COMPANY

Date: 2/20/18

JOHN BANOVETZ
Senior Vice President, Research and Development and Chief Technology Officer
3M Company
3M Center
St. Paul, MN 55144

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Exhibit 15
WL Class 1 Rule Comments

ORDER

Based upon the foregoing Agreement, it is SO ORDERED.

Feb 20, 2018
Date

Kevin S Burke
THE HONORABLE KEVIN S. BURKE
JUDGE OF THE DISTRICT COURT

WaterLegacy Comments on Possible Class 1 Rule Amendments Feb. 14, 2022
(Minnesota Rules chapters 7050, 7052, 7053, 7060)
Revisor's ID Number R-04727, OAH Discussion 37887

EXHIBIT 16

STATE OF MINNESOTA DISTRICT COURT

COUNTY OF HENNEPIN FOURTH JUDICIAL DISTRICT

Case Type: Other Civil

Judge Joseph Klein

State of Minnesota, by its Attorney General, Lori Swanson, its Commissioner of Pollution Control, Paul Aasen, and its Commissioner of Natural Resources, Tom Landwehr,

Court File No. 27-CV-10-28862

Plaintiff,

AMENDED COMPLAINT

vs.

3M Company,

Defendant.

The State of Minnesota, by its Attorney General, Lori Swanson, its Commissioner of Pollution Control, Paul Aasen, and its Commissioner of Natural Resources, Tom Landwehr, for its Complaint against Defendant 3M Company, alleges as follows:

INTRODUCTION

1. For over 50 years, Defendant 3M Company produced at facilities in Minnesota chemicals known as perfluorochemicals, or PFCs. 3M used these chemicals in the production of a variety of consumer, commercial, and industrial products, including stain repellents like Scotchguard™, fire retardants, and chemical products. 3M disposed of waste and discharged wastewater containing PFCs in Minnesota, causing pollution of Minnesota ground and surface water and injury to the natural resources of the State of Minnesota. The State of Minnesota, through its Attorney General, Commissioner of Pollution Control, and Commissioner of Natural Resources, brings this action as trustee for the State's natural resources and to recover damages

for injury, loss, and destruction of Minnesota's natural resources caused by 3M's pollution of the environment.

PARTIES

2. The State of Minnesota (sometimes hereinafter referred to as the "State") is a sovereign state of the United States of America acting as trustee of the natural resources of the State of Minnesota, including all groundwater, surface water, wetlands, sediments, and aquatic life, including fish.

3. Lori Swanson, the Attorney General of the State of Minnesota, is authorized under Minn. Stat. §§ 115B.17, subd. 7 and 115.071, subd. 3, and has common law authority, including *parens patriae* authority, to bring this action on behalf of the State of Minnesota and its citizens to enforce Minnesota law and to recover the damages and other relief requested in this Complaint.

4. Paul Aasen is the Commissioner of the Minnesota Pollution Control Agency ("MPCA"). The MPCA is a statutory agency of the State of Minnesota responsible for administering and enforcing Minnesota statutes and rules relating to water, land and air pollution. *See* Minn. Stat. Chs. 115, 115B and 116 (2010).

5. The MPCA is authorized to adopt and enforce rules "in order to prevent, control or abate water pollution . . . ." Minn. Stat. § 115.03, subd. 1(e).

6. Tom Landwehr is the Commissioner of the Minnesota Department of Natural Resources ("MDNR"). The MDNR is a statutory agency of the State of Minnesota responsible for administering and enforcing Minnesota statutes and rules relating to the preservation, conservation, management and regulation of natural resources of the State. *See* Minn. Stat. Chs. 84, 85, 94 and 103 G (2010).

7. Money recovered by the State for natural resource damages is deposited into the State's remediation fund. Minn. Stat. § 116.155, subd. 3(1) (2010). The general portion of the remediation fund is appropriated to the Commissioners of MPCA and MDNR, among other things, "to take actions related to releases of hazardous substances, or pollutants or contaminants as provided in section 115B.20." Minn. Stat. § 116.155, subd. 2(1) (2010). Minn. Stat. § 115B.20 (2010) authorizes money appropriated from the remediation fund to be spent for the "assessment and recovery of natural resource damages by [the MPCA] and the commissioner of natural resources for administration, planning and implementation by the commissioner of natural resources of the rehabilitation, restoration, or acquisition of natural resources to remedy injuries or losses to natural resources resulting from the release of a hazardous substance."

8. Defendant 3M Company (sometimes hereinafter referred to as "3M") is a corporation incorporated in the State of Delaware and has its principal place of business in Maplewood, Minnesota. 3M Company's resident agent for service of process is CT Corporation System Inc., which is located at 100 South Fifth Street, Number 1075, Minneapolis, Minnesota 55402 in Hennepin County.

JURISDICTION AND VENUE

9. Jurisdiction exists in this Court under Minn. Stat. § 484.01 (2010), and venue exists in this Court under Minn. Stat. §§ 542.07 and 542.09 (2010). Among other things, 3M's registered agent is located in Hennepin County, and waters impaired by 3M's discharge of PFCs are located in Hennepin County.

FACTUAL BACKGROUND

I. 3M Produced PFCs in Minnesota for Over 50 Years.

10. 3M began research and development of a group of chemicals known as perfluorochemicals, or PFCs, in Minnesota in the late 1940s. The company began commercial production of PFCs in Minnesota in the early 1950s. 3M used PFCs to manufacture many consumer, commercial, and industrial products, including but not limited to stain repellents like Scotchguard™, fire retardants, stain removers, paints, hydraulic fluids, semi-conductors, and other chemical products.

11. PFCs are a class of chemicals--not natural to the environment--in which fluorine atoms replace the hydrogen atoms that are normally attached to the carbon "backbone" of hydrocarbon molecules. For purposes of this Complaint, "PFCs" include all perfluorochemicals manufactured by 3M, and all byproducts, compounds, and/or waste containing any perflourochemical associated with 3M's manufacture, treatment, disposal, discharge, or release of perfluorochemicals. PFCs include these chemicals and associated compounds:

- perfluorooctonoate (also known as "perfluorooctonoic acid" or "PFOA");
- perfluorooctane sulfonate (also known as "PFOS");
- perfluorobutanoate (also known as also known as "perfluorobutyrate," "perfluorobutyric acid" or "PFBA"); and
- perfluorobutane sulfonate" (also known as "nonafluorobutanesulphonic acid" or "PFBS").

12. For decades, 3M manufactured PFOS, PFOA and other PFCs at facilities in the Twin Cities metropolitan area in Minnesota. 3M was the sole manufacturer of PFOS in the United States and a major manufacturer of PFOA.

13. The chemical structure of PFCs make them resistant to breakdown or environmental degradation. As a result, they are persistent when released into the environment.

Some PFCs have been found to bioaccumulate in humans and animals. A 2005 report by the U.S. Department of Health and Human Services found that "human exposure to PFOS and PFOA lead to the buildup of these chemicals in the body."

14. Following negotiations with the United States Environmental Protection Agency ("EPA"), 3M announced in the year 2000 that it would stop producing PFOS in Minnesota. At the time of this announcement, the EPA wrote in a news release: "3M data supplied to EPA indicated that these chemicals are very persistent in the environment, have a strong tendency to accumulate in human and animal tissues and could potentially pose a risk to human health and the environment over the long term. EPA supports the company's plans to phase out and develop substitutes by year's end for the production of their involved products." 3M stopped producing PFOS in Minnesota in late 2002.

II. PFCs Have Adverse Health and Environmental Consequences.

15. Numerous studies have shown that PFCs pose serious risks to human health and the environment.

16. In a Public Health Assessment released for comment in August 2010, the Minnesota Department of Health described the results of some studies of the impact of PFCs on human health and the environment. For example, the Department cited studies indicating that it has been reported that:

a. Exposure to high levels of PFOA, PFOS and PFBA is acutely toxic to test animals;

b. Some long term animal studies suggest that exposure to PFOA could increase the risk of tumors of the liver, pancreas and testes;

c. Chronic or subchronic exposure to low doses of PFOA in rats typically results in reduction in body weight and weight gain;

d. Adverse immune system effects have been reported in mice exposed to high doses of PFOA; and

e. Adverse developmental effects have been observed in the offspring of pregnant rats and mice exposed to high doses of PFOA and PFOS.

17. The Department also noted that a recent study by 3M of its employees suggested a positive association "between PFOA exposure and prostate cancer, cerebrovascular disease, and diabetes." The Assessment further notes that: "PFCs disposed of [by 3M] have impacted soil, groundwater, surface water, sediments, biota, and nearby drinking water wells, both public and private." The Department also pointed out that residents of the eastern metropolitan area of the Twin Cities showed elevated PFC levels in blood tests when compared to the U.S. population as a whole.

18. In its 2007 notice placing special restrictions on the construction of wells in a 12 square mile "special well construction area" in Washington County, the Minnesota Department of Health pointed to various health concerns with PFCs, including that: "In animal studies, high concentrations of PFCs harm the liver and thyroid. Developmental problems have been seen in the offspring of rats and mice exposed to PFCs while pregnant."

19. In its 2007 notice placing special restrictions on the construction of wells in a 12 square mile "special well construction area" in Washington County, the Minnesota Department of Health pointed to various health concerns with PFCs, including that: "In animal studies, high concentrations of PFCs harm the liver and thyroid. Developmental problems have been seen in the offspring of rats and mice exposed to PFCs while pregnant."

20. In a 2005 Health Consultation report relating to PFCs at 3M's Cottage Grove facility, the Agency for Toxic Substances and Disease Registry of the U.S. Department of Health and Human Services states that, "Animal studies have shown that PFOA and APFO (its ammonium salt) are easily absorbed through ingestion, inhalation, and dermal contact." The report states that "[e]xposure to high levels of PFOA and PFOS is acutely toxic in test animals" and that "[c]hronic or sub-chronic exposure to lower doses of PFOA in rats typically results in reduction in body weight and weight gain, and in liver effects such as an increase in liver weight and alterations in lipid metabolism." The report further states that, "A 90-day study of relatively high-dose oral PFOA exposure in rhesus monkeys resulted in adverse effects on the adrenal glands, bone marrow, spleen, lymphatic system, and death in some animals."

21. In 2009, the EPA issued provisional health advisories, guidance on toxicity values, and soil screening levels relating to potential risk from exposure to PFOA and PFOS in the environment. The limits set on PFC exposure by the EPA are comparable to limits set by the Minnesota Department of Health. Environmental authorities in other states and in foreign countries also have set safe drinking water values for PFCs that are comparable to the limits established by the Minnesota Department of Health.

22. In 2009, EPA placed PFOA and PFOS on its Third Contaminant Candidate List for possible regulation under the Safe Drinking Water Act, 42 U.S.C. § 300g 1(b)(1)(B)(i). 74 Fed. Reg. 51850 (October 8, 2009).

23. During the time that it manufactured PFCs, 3M extensively studied the impact of PFCs on human health and the environment. 3M knew or should have known that as a result of its regular disposal of PFCs and PFC-containing wastes, it was reasonably likely that PFCs would be released from the disposal sites and would reach the groundwater, surface water and

sediments and to result in injury, destruction, and loss of natural resources of the State, including groundwater, surface water, sediments and aquatic life such as fish. 3M knew or should have known of the potentially harmful effects that PFCs have on human health and the environment. 3M knew or should have known that the discharge of PFCs would pollute groundwater and surface water of the State, making them unavailable to the citizens of the State for their normal and designated uses, including as sources of drinking water and habitat for fish which may be consumed as food.

III. 3M Discharged PFCs in Minnesota For Decades.

24. For decades, 3M disposed of waste and discharged wastewater containing PFCs in Minnesota. 3M is responsible for releasing PFCs into the Minnesota environment, causing pollution of groundwater, surface water, and sediments and resulting in injury, destruction and loss of natural resources of the State.

25. 3M disposed of wastes containing PFCs at several sites in the Twin Cities Minnesota metropolitan area, including at least the following:

a. its industrial facility located in the City of Cottage Grove, Minnesota ("the 3M Cottage Grove Site");

b. a disposal site located in the City of Oakdale, Minnesota ("the 3M Oakdale Disposal Site");

c. a disposal site located on the border of the cities of Cottage Grove and Woodbury, Minnesota in the area encompassed by Woodbury Drive (County Road 19) and Cottage Grove Drive ("the 3M Woodbury Disposal Site"); and

d. the Washington County Landfill, located in the City of Lake Elmo, Minnesota.

For years, 3M buried wastes containing PFCs in unlined dumps, thereby releasing PFCs into the groundwater beneath the sites and ultimately into other groundwater.

26. 3M also discharged wastewater containing PFCs from the 3M Cottage Grove Site into surface water of the State which flows into the Mississippi River. 3M did so both directly and indirectly. For years, 3M piped wastewater containing PFCs directly into a stream that flows directly into the Mississippi River. In addition, 3M disposed of waste containing PFCs on land in close proximity to the Mississippi River, allowing this waste to leach into the river.

27. Over 100 square miles of groundwater have been contaminated by 3M's disposal of PFCs, and the source of residential drinking water for tens of thousands of Minnesotans is potentially affected by the contamination caused by 3M's disposal of PFCs. The area of contamination includes four major aquifers; namely, the St. Peter, Prairie du Chien, Jordan, and Franconia aquifers. These four aquifers serve as the sole source of drinking water for approximately 125,000 or more Minnesotans who reside in the Twin Cities area.

28. PFCs have also polluted Lake Elmo and approximately 139 miles of the Mississippi River from St. Anthony Falls in Minneapolis (Hennepin County) downstream to the La Moille Dam (Lock and Dam No. 6), south of Winona.

29. 3M's release and discharge of PFCs into the groundwater and surface water are in violation of Minnesota water quality rules and were not authorized or permitted by the State. 3M was not authorized by the Minnesota Pollution Control Agency to discharge PFCs into waters of the State at any of the sites where 3M disposed of wastes containing PFCs or at other facilities 3M owned or operated.

30. Minn. Rule 7050.0210, subp. 2, provides: "No sewage, industrial waste, or other wastes shall be discharged from either point or nonpoint sources into any waters of the state so as

to cause any nuisance conditions, such as the presence of significant amounts of floating solids, scum, visible oil film, excessive suspended solids, material discoloration, obnoxious odors, gas ebullition, deleterious sludge deposits, undesirable slimes or fungus growths, aquatic habitat degradation, excessive growths of aquatic plants, or other offensive or harmful effects." Minn. Rule 7053.0205, subp. 2, sets forth the same prohibition as found in Minn. Rule 7050.0210, subp. 2. The discharge of PFCs from sites where 3M disposed of wastes and discharged wastewater have polluted waters of the State and precluded and adversely affected the use of underground waters for potable use, thereby causing nuisance conditions and other offensive and harmful effects on waters of the state within the meaning of Minn. Rules 7050.0210, subp. 2 and 7053.0205, subp. 2.

31. Minn. Rule 7050.0210, subp. 13, provides that "[n]o sewage, industrial waste, or other wastes shall be discharged from either a point or a nonpoint source into the waters of the state in such quantity or in such manner alone or in combination with other substances as to cause pollution as defined by law." Underground waters, including the primary drinking water aquifers in the eastern metropolitan area of the Twin Cities, have been polluted by the discharge of PFCs from sites where 3M disposed of wastes containing PFCs.

32. Minn. Rule 7060.0600, subp. 2, provides that "[n]o sewage, industrial waste, other waste, or other pollutants shall be allowed to be discharged to the unsaturated zone or deposited in such place, manner, or quantity that the effluent or residue therefrom, upon reaching the water table, may actually or potentially preclude or limit the use of the underground waters as a potable water supply, nor shall any such discharge or deposit be allowed which may pollute the underground waters." 3M allowed PFCs to be discharged at sites where 3M disposed of wastes containing PFCs in such a way as to pollute underground waters, including the primary drinking

water aquifers in the eastern metropolitan area of the Twin Cities, and to actually or potentially limit or preclude the use of the underground waters as a potable water supply.

IV. 3M's Discharge of PFCs Caused Damage To the State's Natural Resources.

33. The State and its citizens place a high value on the State's natural water resources.

34. 3M released PFCs into the Minnesota environment, thereby causing pollution of groundwater and surface water, and causing injury to and destruction and loss of natural resources of the State of Minnesota. PFCs from wastes disposed of by 3M are now widespread in the environment in the Twin Cities metropolitan area. PFCs released by 3M have been found in soil, groundwater, surface water, sediments and fish.

35. As a result of the injury caused by 3M to the State's natural resources by the discharge of PFCs into the environment, the State and its citizens face substantial costs to provide alternative sources of groundwater for domestic and other uses and to restore surface waters for the full use and enjoyment of the public.

1. Well and Water Usage Restrictions.

36. The Minnesota Department of Health has established health-based standards for human consumption of PFCs in drinking waters. Under Minnesota law, a health risk limit ("HRL") is "a concentration of a substance or chemical adopted by rule of the commissioner of health that is a potential drinking water contaminant because of a systemic or carcinogenic toxicological result from consumption." Minn. Stat. § 103H.005, subd. 4 (2010). Before the Department establishes an HRL through formal rulemaking, the Department develops interim guidelines known as Health Based Values ("HBVs"). HBVs are based on source studies that are of the same quality as those used to develop formal HRLs.

37. In 2002, the Minnesota Department of Health issued HBVs for PFOA and PFOS in drinking water.

38. In 2007, the Minnesota Legislature expressly authorized and directed the Minnesota Department of Health to adopt HRLs for PFOA and PFOS using a special provision in Minnesota's Administrative Procedures Act which is reserved for rules that "address a serious and immediate threat to the public health, safety or welfare." Laws of Minnesota 2007, chapter 37, sec. 1; Minn. Stat. § 143.388, subd. 1(1) (2010). The Department thereafter in 2007 adopted temporary rules setting HRLs for PFOA and PFOS in groundwater. In 2009, the Department adopted permanent Heath Risk Limits, or HRLs, for PFOA and PFOS. The Department has also issued HBVs for PFBA and PFBS.

39. In addition, the Minnesota Pollution Control Agency ("MPCA") issues generic health-based criteria for soil that are based on a standard exposure scenario for contaminated sites. MPCA has issued Soil Reference Values for evaluating the risks to public health from concentrations of PFOA and PFOS in residential (Tier I) and industrial (Tier II) soils.

40. PFCs have been found in groundwater and soils at the sites used by 3M to dispose of wastes containing PFCs at levels that exceed the health based standards (HRLs and HBVs) adopted by the Minnesota Department of Health and the Soil Reference Values issued by the MPCA. PFC concentrations in the groundwater at the 3M disposal sites at times have been more than 100 times higher than the health-based standards established by the Minnesota Department of Health.

41. In addition, as noted above, PFCs have been found in four major drinking water aquifers (St. Peter, Prairie du Chien, Jordan and Franconia) which lie below and down gradient from the sites where 3M disposed of PFC-containing wastes. These aquifers are the sole source

of drinking water for approximately 125,000 residents of the eastern metropolitan area of the Twin Cities, including residents of the cities of Oakdale, Lake Elmo, Woodbury, and Cottage Grove. As a result, the source of residential drinking water for tens of thousands of Minnesotans is potentially affected by PFC contamination caused by 3M.

42. In 2007, the Minnesota Department of Health issued a Special Well Construction Advisory, which remains in effect, for an area of more than twelve square miles in Washington County, due to the contamination of groundwater caused by 3M's disposal and releases of PFCs. Through the Advisory, the Department placed limits on the installation and operation of any new groundwater wells for drinking and other purposes within the well advisory area because of the PFC contamination. These restrictions, which result from 3M's disposal and discharge of PFCs, will result in substantial additional costs to public and private entities that use the affected groundwater to meet their present and future water supply needs.

2. Surface Water Impairment and Fish Consumption Advisories.

43. Because of high levels of PFOS in tissues of fish from certain parts of the Mississippi River and from Lake Elmo, the Minnesota Department of Health has recommended that citizens limit their fish consumption from these waters.

44. In 2008, as a result of these fish consumption advisories, the Minnesota Pollution Control Agency listed certain areas of the Mississippi River and Lake Elmo as "impaired" under Section 303(d) of the federal Clean Water Act, 33 U.S.C. § 1313(d). A surface water is "impaired" when it does not meet applicable water quality standards or fully support applicable beneficial uses (such as recreational fishing) due to pollution from point or nonpoint sources. Minn. Rule 7050.0150, subp. 4.H.

45. The PFC contamination which led to these fish advisories and impairment listings resulted from or was significantly contributed to by 3M's releases of PFCs into the environment,

including discharge of industrial wastewater containing PFCs from the 3M Cottage Grove Site, and discharge of extracted groundwater containing PFCs from the 3M Woodbury Disposal Site and the 3M Oakdale Disposal Site.

3. The Natural Resources Damages Caused by 3M Have Not Been Remedied and Are Ongoing.

46. In 2007 the Minnesota Pollution Control Agency and 3M entered into a Settlement Agreement and Consent Order ("Remediation Consent Order") requiring 3M to take certain steps to remediate its releases of PFCs at the 3M Cottage Grove Site, the 3M Oakdale Disposal Site and the 3M Woodbury Disposal Site. The Remediation Consent Order, however, did not address 3M's liability for the injuries that its release of PFCs have caused to the natural resources of the State; to the contrary, Section XXV(D) of the Remediation Consent Order specifically reserved the State's claims for natural resource damages associated with 3M's releases of PFCs. The Remediation Consent Order does not compensate the State for the injury to and destruction and loss of its natural resources caused by 3M's releases of PFCs, or restore the State's injured natural resources to their pre-release condition.

47. The State brings this action to compensate the State and its citizens for the current and ongoing injury to, destruction of, and loss of natural resources which have resulted from 3M's conduct.

48. The damages caused by 3M's disposal and discharge of PFCs, including the injuries to and destruction and loss of use of the State of Minnesota's natural resources, are continuing. The sources of PFC pollution at sites where 3M disposed of or discharged PFCs have not been controlled or abated, and PFCs continue to spread in the environment. This injury and damage will continue into the future, unless and until the resources are restored.

CLAIMS FOR RELIEF

COUNT ONE - DAMAGES UNDER MERLA

49. The State re-alleges all prior paragraphs of this Complaint.

50. Chapter 115B of the Minnesota Statutes is known as the Minnesota Environmental Response and Liability Act, or MERLA.

51. Under MERLA, the State of Minnesota is the trustee of the air, water, and wildlife of the State. Minn. Stat. § 115B.17, subd. 7.

52. Under Minn. Stat. § 115B.17, subd. 7, an action pursuant to § 115B.04 for damages with respect to air, water or wildlife may be brought by the Attorney General in the name of the State of Minnesota as trustee for the State's natural resources.

53. Under Minn. Stat. § 115B.04, subd. 1a, any person who is responsible for a release or threatened release of a hazardous substance from a facility is strictly liable, jointly and severally, for damages which result from the release or threatened release or to which the release or threatened release significantly contributes, including "(3) all damages for any injury to, destruction of, or loss of natural resources, including the reasonable costs of assessing such injury, destruction, or loss."

54. 3M is a "person who is responsible" for the release of PFCs into the environment under Minn. Stat. § 115B.03, subd. 1 and subd. 3. This includes for the following reasons:

a. With respect to the release of PFCs at the 3M Cottage Grove Site:

i. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(1) and subd. 3(1)-(3) because 3M owned and operated the site; engaged in the business of generating, storing, treating, and disposing of wastes and wastes containing PFCs at the site; and knowingly permitted

use of the site for the regular disposal of wastes and for the disposal of wastes containing PFCs;

ii. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(2) because it owned or possessed waste containing PFCs and arranged, by contract, agreement, or otherwise, for the disposal, or transport for disposal of the waste at the site; and

iii. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(1) because it owned and operated facilities including pipes, equipment and installations at the site from which PFCs were released into the environment.

b. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(2) for the release of PFCs from the 3M Oakdale Disposal Site because it owned or possessed waste containing PFCs and arranged, by contract, agreement, or otherwise, for the disposal, or transport for disposal of the waste at the site.

c. With respect to the release of PFCs at the 3M Woodbury Disposal Site:

i. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(1) and subd. 3(1)-(3) because 3M owned and operated the site and knowingly permitted use of the site for the regular disposal of wastes and for the disposal of wastes containing PFCs; and

ii. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(2) because it owned or possessed waste containing PFCs and arranged, by contract, agreement, or otherwise, for the disposal, or transport for disposal of the waste at the site.

d. 3M is a responsible person under MERLA, Minn. Stat. § 115B.03, subd. 1(2) for the release of PFCs from the Washington County Landfill because it owned or possessed waste containing PFCs and arranged, by contract, agreement, or otherwise, for the disposal, or transport for disposal of the waste at this landfill. The Washington County Landfill is a closed, mixed municipal waste landfill which was operated under an MPCA permit by the Counties of Ramsey and Washington from approximately 1969 to 1975. This landfill is a "qualified facility" as defined in the Landfill Cleanup Act ("LCA"). Under the LCA, the MPCA has assumed responsibility for long term environmental response actions to address releases from the Washington County Landfill, including releases of PFCs. The LCA does not absolve 3M of liability under MERLA for any injury to, destruction of, or loss of natural resources associated with releases of PFCs from the Washington County Landfill.

55. Under Minn. Stat. § 115B.02, subd. 15, "release" means any "spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment which occurred at a point in time or which continues to occur." The "release" of PFCs as defined in Minn. Stat. § 115B.02, subd. 15, has occurred and continues to occur at the sites where 3M disposed of wastes containing PFCs --- including at least the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site, and the Washington County Landfill --- including releases to groundwater, surface water and sediments.

56. Under Minn. Stat. § 115B.02, subd. 8(3), "hazardous substance" means "any hazardous waste." Under Minn. Stat. § 115B.02, subd. 9 (1), "hazardous waste" means "any hazardous waste as defined in section 116.06, subdivision 11, and any substance identified as a hazardous waste pursuant to rules adopted by the agency under section 116.07."

57. Under Minn. Stat. § 116.06, subd. 11, "hazardous waste" means "any refuse, sludge, or other waste material or combinations of refuse, sludge or other waste materials in solid, semisolid, liquid, or contained gaseous form which because of its quantity, concentration, or chemical, physical, or infectious characteristics may . . . (b) pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, or disposed of, or otherwise managed."

58. PFCs released into the environment by 3M, including at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill, including PFOA, PFOS, PFBA and PFBS, are hazardous substances as defined in MERLA, Minn. Stat. § 115B.02, subd. 8. PFCs are hazardous wastes, and therefore hazardous substances. PFCs pose a substantial present or potential hazard to human health and the environment in the way that 3M managed its wastes and wastewater containing PFCs. Many studies of PFCs have identified actual and potential hazards to human health from exposure to PFCs. The Minnesota Department of Health ("MDH"), and a number of other state and federal agencies and foreign countries, have used the available health information on PFCs to establish health-based drinking water standards for PFCs. MDH health-based standards are generally comparable to those set by other agencies that have set such standards. PFCs are found in soil and groundwater at four or more sites in Minnesota where 3M disposed of wastes containing PFCs. These wastes were disposed of in unlined disposal areas, allowing PFCs to spread to groundwater in a 100 square mile area. PFCs in groundwater at and down gradient from the 3M disposal sites have exceeded MDH's health-based standards, including exceedances in both municipal and private drinking water wells. PFCs at 3M disposal sites at times have been 100 times higher than the health-based standards that MDH established.

MPCA's Soil Reference Values for PFCs have also been exceeded at the 3M disposal sites. Additionally, 3M's disposal and discharge of PFCs has resulted in certain Minnesota surface waters being formally declared as impaired because of MDH advisories to limit human consumption of fish from those waters. PFCs are hazardous wastes and hazardous substances under MERLA.

59. Under Minn. Stat. § 115B.02, subd. 5, the term "facility" means:

a. any building, structure, installation, equipment, pipe or pipeline (including any pipe into a sewer or publicly owned treatment works), well, pit, pond, lagoon, impoundment, ditch, landfill, storage container, motor vehicle, rolling stock, or aircraft;

b. any watercraft of any description, or other artificial contrivance used or capable of being used as a means of transportation on water; or

c. any site or area where a hazardous substance, or a pollutant or contaminant, has been deposited, stored, disposed of, or placed, or otherwise come to be located.

The areas where 3M disposed of wastes containing PFCs, including the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, and the 3M Woodbury Disposal Site; pipes, equipment and installations from which PFCs have been discharged at the 3M Cottage Grove Site; and the Washington County Landfill, are "facilities" as defined in MERLA, Minn. Stat. § 115B.02, subd. 5.

60. Under Minn. Stat. § 115B.02, subd. 10, "natural resources" has the meaning set forth in Minn. Stat. § 116B.02, subd. 4. Under Minn. Stat. § 116B.02, subd. 4, "natural resources" "shall include, but not be limited to, all mineral, animal, botanical, air, water, land, timber, soil, quietude, recreational and historical resources. Scenic and esthetic resources shall also be considered natural resources when owned by any governmental unit or agency." Waters,

soil, animals, and recreational resources are "natural resources" within the foregoing definitions, and have been injured by 3M's release of PFCs.

61. Under Minn. Stat. § 115B.02, subd. 19, "water" has the meaning given to the term "waters of this state" in Minn. Stat. § 115.01, subd. 22. Minn. Stat. § 115.01, subd. 22 defines "waters of this state" as "all streams, lakes, ponds, marshes, watercourses, waterways, wells, springs, reservoirs, aquifers, irrigation systems, drainage systems and all other bodies or accumulations of water, surface or underground, natural or artificial, public or private, which are contained within, flow through, or border upon the state or any portion thereof." The waters of the State, including surface water and underground water, have been injured by 3M's release of PFCs within the meaning of the foregoing definitions.

62. 3M is: a) responsible for the release of a hazardous substance, namely PFCs, from facilities including at least the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill; and b) is strictly liable, jointly and severally, for the damages which resulted from the release or to which the release or threatened release significantly contributed, including all damages for any injury to, destruction of, or loss of natural resources, including the reasonable costs of assessing such injury, destruction, or loss. Releases of PFCs into the environment from these facilities have resulted in, or have significantly contributed to, injury to, destruction of, or loss of natural resources of the State, including groundwater, surface water, sediments and aquatic life including fish. 3M's liability under Minn. Stat. § 115B.04, subd. 1(3) also includes the State's reasonable costs of assessing such injury, destruction, or loss.

63. Releases of PFCs for which 3M is responsible have caused injury to, destruction of and loss of groundwater, surface water, sediments and aquatic life, including fish, and have

resulted in loss of use, value, benefits and enjoyment of these resources by the State and its citizens and in the imposition of substantial costs to the public to meet current and future water supply needs and restore impaired surface waters.

64. Pursuant to MERLA, Minn. Stat. § 115B.04, subd. 1(3), the State is entitled to recover from 3M all damages incurred through the time of trial, and for all future damages, for injury to, destruction of, or loss of natural resources which have resulted from 3M's releases of PFCs into the environment, including releases at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill, or to which those releases significantly contributed, including the State's reasonable costs of assessing such injury, destruction, or loss.

65. Under Minn. Stat. § 115B.14, 3M is responsible for the State's costs, disbursements, and attorneys fees in bringing this action.

COUNT TWO - DAMAGES UNDER THE MWPCA

66. The State re-alleges all prior paragraphs of this complaint.

67. Sections 115.01 - 115.09 of the Minnesota Statutes are known as the Minnesota Water Pollution Control Act, or MWPCA.

68. Under Minn. Stat. § 115.071, subd. 3(b), a person may be required to forfeit and pay to the state a sum which constitutes just compensation for any loss or destruction to wildlife, fish or other aquatic life and for other actual damages to the state caused by an unauthorized discharge of pollutants, where the discharge violates, among other things, provisions of the MWPCA or any rules promulgated by the Minnesota Pollution Control Agency.

69. Under Minn. Stat. § 115.071, subd. 3 (b), "Any person who violates any provision of this chapter or chapter 114C or 116, . . . or of (1) any effluent standards and limitations or

water quality standards, (2) any permit or term or condition thereof, (3) any national pollutant discharge elimination system filing requirements, (4) any duty to permit or carry out inspection, entry or monitoring activities, or (5) any rules, stipulation agreements, variances, schedules of compliance, or orders issued by the agency, . . ." may be required to "(b) forfeit and pay to the state an additional sum to constitute just compensation for any loss or destruction to wildlife, fish or other aquatic life and for other actual damages to the state caused by an unauthorized discharge of pollutants."

70. The damages provided for in section 115.071, subd. 3(b), may be recovered by a civil action brought by the Attorney General in the name of the State of Minnesota.

71. Under Minn. Stat. § 115.01, subd. 10, a "person" includes a private corporation. 3M is a "person" within the meaning of Minn. Stat. § 115.01, subd. 10.

72. Under Minn. Stat. § 115.01, subd. 4, "discharge" means "the addition of any pollutant to the waters of the state or to any disposal system." Under Minn. Stat. § 115.01, subd. 22, "waters of the state" means "all streams, lakes, ponds, marshes, watercourses, waterways, wells, springs, reservoirs, aquifers, irrigation systems, drainage systems and all other bodies or accumulations of water, surface or underground, natural or artificial, public or private, which are contained within, flow through, or border upon the state or any portion thereof." 3M's disposal and discharge of PFCs have caused discharge of pollutants into waters of the State.

73. Discharges of PFCs into waters of the State, as defined in the MWPCA, Minn. Stat. § 115.01, subd. 4, 5, 6 and 22, including discharges to groundwater and surface water, have occurred and continue to occur, including at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County

Landfill. These discharges have polluted and adversely impacted groundwater, surface water, and other natural resources in the Twin Cities area.

74. Under Minn. Stat. § 115.01, subd. 12, "pollutant" means "any sewage, industrial waste, or other wastes, as defined in this chapter, discharged into a disposal system or to waters of the state." Under Minn. Stat. § 115.01, subd. 13, "pollution of water," "water pollution," or "pollute the water" means: " (a) the discharge of any pollutant into any waters of the state or the contamination of any waters of the state so as to create a nuisance or render such waters unclean, or noxious, or impure so as to be actually or potentially harmful or detrimental or injurious to public health, safety or welfare, to domestic, agricultural, commercial, industrial, recreational or other legitimate uses, or to livestock, animals, birds, fish or other aquatic life; or (b) the alteration made or induced by human activity of the chemical, physical, biological, or radiological integrity of waters of the state." PFCs discharged into waters of the State, including at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill, including PFOA, PFOS, PFBA and PFBS, are pollutants as defined in the MWPCA, Minn. Stat § 115.01, subd. 12. The releases of PFCs resulted in water pollution.

75. 3M's discharges of PFCs to waters of the state have violated rules promulgated under the MWPCA, including:

a. Minn. R. 7050.0210, subp. 2. 3M's discharges of PFCs have resulted in listing of certain surface waters of the State as impaired and created offensive and harmful effects in groundwater;

b. Minn. R. 7050.0210, subp. 13. 3M's discharges of PFCs into groundwater, as defined in the MWPCA, Minn. Stat. § 115.01, subd. 6, were unauthorized and were in

such quantity and manner as to cause water pollution as defined in the MWPCA, Minn. Stat. § 115.01, subd. 13;

c Minn. R. 7053.0205, subp. 2. 3M's discharge of PFCs into waters of the State, as defined in the MWPCA, Minn. Stat. § 115.01, subd. 9 and 22, caused nuisance conditions and other offensive or harmful effects; and

d. Minn. R. 7060.0600, subp. 2. 3M discharged PFCs in such places and in such a manner that PFCs have polluted underground waters, including the primary drinking water aquifers in the eastern metropolitan area, and have actually or potentially limited or precluded the use of underground waters as a potable water supply.

76. 3M's discharges of PFCs into waters of the State, including at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill, were not authorized or permitted by the State. These discharges have resulted in, or have significantly contributed to, loss and destruction of fish or other aquatic life and other actual damages to the State.

77. 3M's unauthorized discharges of PFCs have polluted waters of the State within the meaning of Minn. Stat. § 115.01, subd. 13, by creating nuisance conditions; by rendering state waters unclean and impure so as to be actually or potentially harmful or detrimental to public health, safety, and welfare, or to domestic, commercial, recreational, or other legitimate uses; by altering the chemical integrity of the waters by human activity; and by rendering groundwater actually and potentially unavailable for drinking water purposes.

78. Pursuant to the MWPCA, Minn. Stat. § 115.071, subd. 3(b), 3M is liable to pay the State just compensation for all loss and destruction of fish or other aquatic life and for all other actual damages to the State caused by its unauthorized discharges of PFCs to waters of the

State. Pursuant to the MWPCA, Minn. Stat. § 115.071, subd. 3(b), 3M may be required to forfeit and pay to the State just compensation for all loss and destruction, through the time of trial, and in the future, of fish and other aquatic life and for all other actual damages to the State caused by 3M's unauthorized discharges of PFCs into waters of the State, including discharges at and from the 3M Cottage Grove Site, the 3M Oakdale Disposal Site, the 3M Woodbury Disposal Site and the Washington County Landfill, or to which such discharges significantly contributed, including the State's reasonable costs of assessing such loss, destruction, and other damages.

79. The damages caused by 3M's discharge of PFCs, including the injuries to and destruction and loss of use of the State of Minnesota's natural resources, are continuing. The sources of PFC pollution at sites where 3M disposed of or discharged PFCs have not been controlled or abated and PFCs continue to spread in the environment. This injury and damage will continue into the future, unless and until the resources are restored.

COUNT THREE - DAMAGES FOR TRESPASS

80. The State re-alleges all prior paragraphs of this Complaint.

81. Groundwater, surface water, sediments, and aquatic life including fish are natural resources of the State of Minnesota, and the State of Minnesota is the trustee of these resources on behalf of its citizens, now and in the future. These natural resources are property of the State.

82. For decades, 3M disposed of wastes containing PFCs at various sites in the Twin Cities metropolitan area, and discharged PFCs directly and indirectly into the Mississippi River. As a result of these actions, PFCs have entered and spread in and have contaminated groundwater, surface water and sediments in the Twin Cities metropolitan area of Minnesota and have contaminated aquatic life including fish in affected surface water.

83. The entry of 3M's PFCs into these natural resources, which constitute property of the State of Minnesota, was unlawful. The State never authorized or permitted 3M to release PFCs into groundwater, surface water or sediments. Furthermore, 3M knew or should have known that its disposal and discharge of PFCs was substantially certain to result in PFCs entering and polluting these natural resources of the State.

84. The entry of PFCs into the State's groundwater, surface water and sediments is continuing. The sources of PFC pollution at sites where 3M disposed of or discharged PFCs have not been controlled or abated and PFCs continue to spread in the environment.

85. The entry of 3M's PFCs into the State's groundwater, surface water, sediments and the contamination of fish has injured and destroyed these natural resources, and resulted in a loss of the public's ability to use them for their normal and designated uses. Such injury to, destruction of and loss of use of these resources will continue into the future, unless and until the resources are restored.

86. 3M is liable for damages to compensate the State for the injury to, destruction of and loss of use of the State's natural resources caused by 3M's continuing trespass in the form of PFC contamination of the State's property.

COUNT FOUR - DAMAGES FOR COMMON LAW NUISANCE

87. The State re-alleges all prior paragraphs of this Complaint.

88. The use, enjoyment and existence of the State's groundwater, surface water and sediments, free from interference, is a right common to the citizens of the State.

89. The contamination of groundwater, surface water and sediments with PFCs materially and substantially interferes with State citizens' free enjoyment of these natural resources, and constitutes a public nuisance.

90. 3M knew or should have known that its disposal of wastes containing PFCs would pollute groundwater and surface water of the State, making them unavailable to the citizens of the State for their normal and designated uses, including as sources of drinking water and habitat for fish which may be consumed as food.

91. The nuisance conditions described in this Complaint are continuing, and the sources of PFC pollution at sites where 3M disposed or discharged PFCs have not been controlled or abated and PFCs continue to spread in the environment.

92. The impairment of the State's groundwater and surface water by PFC contamination has injured and destroyed these natural resources, and resulted in a loss of the public's ability to use them. Such injury to and destruction and loss of use of the State's natural resources will continue into the future, unless and until the resources are restored.

93. 3M is liable for damages to compensate the State for the injury, destruction and loss of use of the State's natural resources caused by the continuing public nuisance created by PFC contamination of the State's property.

COUNT FIVE - DAMAGES FOR STATUTORY NUISANCE

94. The State re-alleges all prior paragraphs of this Complaint.

95. Minn. Stat. § 561.01 (2010) provides that: "[a]nything which is injurious to health, or indecent or offensive to the senses, or an obstruction to the free use of property, so as to interfere with the comfortable enjoyment of life or property, is a nuisance. An action may be brought by any person whose property is injuriously affected or whose personal enjoyment is lessened by the nuisance, and by the judgment the nuisance may be enjoined or abated, as well as damages recovered."

96. The use, enjoyment and existence of the State's groundwater, surface water and sediments, free from interference, is a right common to the people of the State.

97. The contamination of groundwater, surface water and sediments with PFCs materially and substantially interferes with citizens' free enjoyment of these natural resources, and constitutes a public nuisance.

98. 3M knew or should have known that its disposal of wastes containing PFCs would pollute groundwater and surface water of the State, making them unavailable to the people of the State for their normal and designated uses, including as sources of drinking water and habitat for fish which may be consumed as food.

99. The nuisance conditions described in this Count are continuing. The sources of PFC pollution at sites where 3M disposed or discharged PFCs have not been controlled or abated and PFCs continue to spread in the environment.

100. The pollution of the State's groundwater and surface water by PFC contamination has injured and destroyed these natural resources, and resulted in a loss of the public's ability to use and enjoy them. Such injury to, destruction of and loss of use of natural resources will continue into the future, unless and until the resources are restored.

101. 3M is liable for damages to compensate the State for the injury, destruction and loss of use and enjoyment of the State's natural resources caused by the continuing public nuisance created by PFC contamination of the State's property.

COUNT SIX - DAMAGES FOR NEGLIGENCE

102. The State re-alleges all prior paragraphs of this Complaint.

103. At all times material to this Complaint, 3M owed to the State and its citizens a duty to dispose of PFC-containing wastes in a manner that would protect the public from reasonably foreseeable harm.

104. 3M owed to the State and its citizens a duty to comply with Minnesota water protection rules and regulations, including but not limited to Minn. Rules 7050.0210, subp. 2 and 13; 7053.0205, subp. 2; and 7060.0600, subp. 2.

105. 3M breached its duties set forth in this Complaint, by (1) disposing of PFC-containing wastes at various sites in the Twin Cities metropolitan area in Minnesota in circumstances in which it knew or should have known that PFCs were reasonably likely to be released from the disposal sites and reach the groundwater, surface water and sediments, and (2) discharging PFC-containing wastes directly or indirectly into the Mississippi River.

106. 3M's conduct in breach of its duties has resulted in contamination of groundwater, surface water, sediments and aquatic life, including fish, with PFCs. This damage to the natural resources of the State was reasonably foreseeable to 3M. 3M breached its duties and knew or should have known of the potentially harmful effects of PFCs on human health and the environment.

107. The injuries to natural resources described in this Complaint are continuing because the sources of PFC pollution at sites where 3M disposed of or discharged PFCs have not been controlled or abated and PFCs continue to spread in the environment.

108. The contamination of the State's groundwater, surface water, sediments and aquatic life, including fish, by 3M's releases of PFCs has injured and destroyed natural resources of the State, and resulted in a loss of the public's ability to use those natural resources. Such

injury to and destruction and loss of use is continuing and will continue into the future, unless and until the resources are restored.

109. 3M is liable for damages to compensate the State and its citizens for the injury to, destruction of, and loss of use of the State's natural resources caused by 3M's negligence.

PRAYER FOR RELIEF

WHEREFORE, the State of Minnesota respectfully asks this Court to award judgment against Defendant 3M Company as follows:

1. Awarding judgment against Defendant under MERLA for damages for injury to, destruction of, or loss of natural resources which resulted through the time of trial, and for all future damages, from releases of PFCs by 3M, or to which those releases significantly contributed, including the State's reasonable costs of assessing such injury, destruction, or loss;

2. Declaring that Defendant is responsible under MERLA for all damages that the State may suffer in the future for injury to, destruction of, or loss of natural resources which result from releases of PFCs into the environment, or to which those releases significantly contribute, including the State's reasonable costs of assessing such injury, destruction, or loss;

3. Awarding judgment against Defendant under the MWPCA for just compensation for all loss to or destruction of fish or other aquatic life and for all other actual damages to the State caused through the time of trial, and in the future, by 3M's unauthorized discharge of pollutants into waters of the State, or to which such discharges significantly contributed, including the State's reasonable costs of assessing such loss or destruction;

4. Declaring that Defendant is responsible under the MWPCA to pay just compensation for all loss to or destruction of fish or other aquatic life and for all other actual damages to the State caused in the future by 3M's unauthorized discharge of pollutants into

waters of the State, or to which these discharges significantly contributed, including the State's reasonable costs of assessing such loss or destruction;

5. Awarding judgment against Defendant for damages for all injury to, destruction of and loss of use of the State's natural resources caused by 3M's continuing trespass in the form of contamination of the State's property with PFCs, under the *parens patriae* doctrine, the general equitable powers of the court, and any other authority;

6. Awarding judgment against Defendant for damages for all injury to, destruction of and loss of use of the State's natural resources caused by the continuing common law and statutory nuisance created by 3M's contamination of the State's property with PFCs, under the *parens patriae* doctrine, the general equitable powers of the court, and any other authority;

7. Awarding judgment against Defendant for damages to compensate the State for the injury to, destruction of and loss of use of the State's natural resources caused by 3M's negligent release of PFCs into the environment, under the *parens patriae* doctrine, the general equitable powers of the court, and any other authority;

8. Awarding the State its attorneys' fees, witness fees and costs and disbursements to bring this action as provided in MERLA, Minn. Stat. § 115B.14 and other applicable law; and

9. Granting such other and further relief as provided by law and/or as the Court deems just and proper.

Dated: 1-18-11

Respectfully submitted,

LORI SWANSON
Attorney General
State of Minnesota

ALAN C. WILLIAMS
Assistant Attorney General
Atty. Reg. No. 0117328

ROBERT B. ROCHE
Assistant Attorney General
Atty. Reg. No. 289589

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(651) 757-1390 (Voice)
(651) 296-1410 (TTY)

ATTORNEYS FOR PLAINTIFF
STATE OF MINNESOTA

MINN. STAT. § 549.211
ACKNOWLEDGMENT

The party or parties on whose behalf the attached document is served acknowledges through their undersigned counsel that sanctions may be imposed pursuant to Minn. Stat. § 549.211 (2010).

Dated: 1-18-11

ROBERT B. ROCHE
Assistant Attorney General
Atty. Reg. No. 0289589

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(651) 757-1372 (Voice)
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ATTORNEYS FOR PLAINTIFF
STATE OF MINNESOTA

AG: #2755923-v1

AFFIDAVIT OF SERVICE BY FACSIMILE AND U.S. MAIL

Re: ***State of Minnesota, by its Attorney General, Lori Swanson, its Commissioner of Pollution Control, Paul Aasen, and its Commissioner of Natrual Resources, Tom Landwehr vs. 3M Company***
Court File No. 27-CV-10-28862

STATE OF MINNESOTA)
) ss.
COUNTY OF RAMSEY)

Mary L. Moldestad, being first duly sworn, deposes and says:

That at the City of St. Paul, County of Ramsey and State of Minnesota, on January 18, 2010, she caused to be served the *Amended Complaint*, by facsimile to the numbers indicated below and by depositing the same in the United States mail at said city and state, true and correct copy(ies) thereof, properly enveloped with prepaid first class postage, and addressed to:

Mark W. Lee
Maslon Edelman Borman & Brand, LLP
3300 Wells Fargo Center
90 South 7th Street
Minneapolis, MN 55402-4140
Fax: (612) 642-8355

David K. Snyder
Eckberg, Lammers, Briggs, Wolff & Vierling, PLLP
1809 Northwestern Avenue
Stillwater, MN 55082
Fax: (651) 439-2923

Mary L. Moldestad
MARY L. MOLDESTAD

Subscribed and sworn to before me
this 18th day of January, 2011.

Judy M. Fusco
NOTARY PUBLIC

AG: #2755974-v1

JUDY M FUSCO
NOTARY PUBLIC - MINNESOTA
MY COMMISSION
EXPIRES JAN

STATE OF MINNESOTA

OFFICE OF THE ATTORNEY GENERAL

LORI SWANSON
ATTORNEY GENERAL

SUITE 900
445 MINNESOTA STREET
ST. PAUL, MN 55101-2127
TELEPHONE: (651) 297-1075

January 18, 2010

VIA FACSIMILE & U.S. MAIL

Mark Thompson
District Administrator
Hennepin County Government Center
1251 Court Tower
300 South Sixth Street
Minneapolis, MN 55487

Re: ***State of Minnesota, by its Attorney General, Lori Swanson, its Commissioner of Pollution Control, Paul Aasen, and its Commissioner of Natural Resources, Tom Landwehr vs. 3M Company***
Court File No. 27-CV-10-28862

Dear Mr. Thompson:

Enclosed for filing with your office, please find the original of Plaintiff's Amended Complaint and Affidavit of Service regarding the above-entitled matter.

Thank you for your assistance.

Sincerely,

ROBERT B. ROCHE
Assistant Attorney General

(651) 757-1372 (Voice)
(651) 297-4139 (Fax)

Enclosures

cc: Mark W. Lee, Maslon Edelman Borman & Brand
David K. Snyder, Eckberg, Lammers

AG: #2755949-v1