

September 23, 2020

Environmental Quality Board
P.O. Box 8477
Harrisburg, PA 17105-8477
Submitted by [eComment](#)

**Re: Water Quality Standard for Manganese and Implementation (25 Pa. Code Chapters 93 and 96); Notice of Proposed Rulemaking
50 Pa.B. 3724, July 25, 2020**

I. Introduction/Executive Summary

Pursuant to the public notice published in the Pennsylvania Bulletin on July 25, 2020, the Pennsylvania Coal Alliance (“PCA”) offers the following comments on the proposed Water Quality Standard for Manganese and Implementation; Notice of Proposed Rulemaking [50 Pa.B. 3724]. For the reasons set forth below, PCA believes the adoption of a numeric water quality criterion for manganese of 0.3 mg/L in Table 5 at § 93.8c (relating to human health and aquatic life criteria for toxic substances) contradicts Act 40, is unnecessary to protect human health and fish and aquatic life, will significantly increase treatment costs for the coal mining industry and other vital Pennsylvania industries, and will not simplify treatment processes or dramatically decrease treatment costs for public water supply operators. The proposed rule is not in the best interest of the Environmental Quality Board, Pennsylvania’s businesses, and the citizens of Pennsylvania. PCA urges EQB to retain the current manganese water quality criterion of 1 mg/L and to establish the point of compliance with this criterion at the point of an existing or planned downstream potable water supply withdrawal as directed by Act 40.¹

II. Background

PCA is the principal trade organization representing underground and surface bituminous coal operators in Pennsylvania, as well as other associated companies whose businesses rely on coal mining and a strong coal economy. PCA member companies produce nearly 90 percent of the bituminous coal mined annually in Pennsylvania, making Pennsylvania the third largest coal producing state in the nation. Coal operators’ manganese discharges are regulated at the state level pursuant to 25 Pa. Code Chapters 87, 88, 89, and 90 and at the federal level pursuant to 40 C.F.R. Part 434.

¹ PCA incorporates by reference all of the comments it submitted to the Pennsylvania Department of Environmental Protection on February 26, 2018 concerning the Advance Notice of Rulemaking in this matter, a copy of which is attached as Exhibit A.

This proposed rulemaking will not solely, or even primarily, affect the coal industry. It will also significantly and unnecessarily increase treatment costs for other industry sectors. For example, as of February 2020, the Pennsylvania Department of Environmental Protection issued NPDES permits containing manganese limits or monitoring requirements to at least 99 non-coal mines, 174 industrial facilities, 196 public water suppliers, and 243 publicly-owned or other sewage treatment works in Pennsylvania. Adopting a more stringent manganese criterion and imposing it at the discharge point contrary to Act 40 will increase costs to individual homeowner customers of public water and public sewage facilities. Other activities could be newly subject to the proposed stringent manganese limits, including MS4 permits, construction stormwater permits, and industrial stormwater permits, many of which do not have available manganese treatment technologies.

III. The Proposed Rulemaking is Contrary to Law

a. The proposed rulemaking does not comply with Act 40

The proposed rulemaking does not comply with Section 1920-A of the Administrative Code of 1929 ("Act 40"), which the Governor signed on October 30, 2017,² for at least three reasons.

First, Act 40 required EQB to promulgate regulations by January 29, 2018 that move the point of compliance for manganese from the point of discharge to the point of an existing or planned downstream potable water supply ("PWS") withdrawal:

"The board shall promulgate regulations under the act of June 22, 1937 (P.L. 1987, No. 394), known as "The Clean Streams Law," or other laws of this Commonwealth that require that the water quality criteria for manganese established under 25 Pa. Code Ch. 93 (relating to water quality standards) shall be met, consistent with the exception in 25 Pa. Code § 96.3(d) (relating to water quality protection requirements). Within ninety days of the effective date of this subsection, the board shall promulgate proposed regulations."

EQB did not promulgate proposed regulations for 998 days after Act 40 became law.

Second, the General Assembly unmistakably intended to place the PWS compliance point for manganese at the same location as that for total dissolved solids, nitrite-nitrogen, phenolics, chloride, sulfate, and fluoride under 25 Pa. Code § 96.3(d). Thus, the proposed rulemaking disregards the plain language and does not conform to the intent of Act 40.

Act 40 does not authorize or direct EQB to propose a "Second Alternative Point of Compliance" for the manganese criterion at the point of discharge. Act 40 requires the point of compliance be at the PWS withdrawal. Pennsylvania law makes clear that agencies do not have the authority to disregard an unambiguous statutory directive. *See A.S. v. Pennsylvania State Police*, 143 A.3d 896, 903 (Pa. 2016) ("The statute's plain language generally provides the best indication of legislative intent."); *Lancaster Cnty. v. Pa. Labor Relations Bd.*, 94 A.3d 979, 986

² 71 P.S. § 510-20(j) (2017). EQB's failure to meet the statutory deadline is itself a violation of Act 40.

(Pa. 2014) (“[W]hen an administrative agency’s interpretation is inconsistent with the statute itself, or when the statute is unambiguous, such administrative interpretation carries little weight.”). Because there is no authority to propose a regulation that contradicts the plain language of Act 40, EQB should strike the “Second Alternative Point of Compliance” from the proposed rulemaking and consider only those comments in response to the proposed “First Alternative Point of Compliance.” Furthermore, proposing the Second Alternative Point of Compliance does not comply with the Regulatory Review Act because the alternative is not reflected in Annex A of the proposed rulemaking, as discussed in the immediate section below.

Third, Act 40 does not direct or authorize EQB to re-evaluate the current manganese criterion or propose a new criterion of 0.3 mg/L for manganese as a toxic substance. The statute simply directs EQB to change the point of compliance for the existing PWS manganese criterion of 1 mg/L. EQB “cannot look beyond the language of an unambiguous statute” and propose a regulation that changes the manganese criterion itself. *First Union Nat’l Bank v. Commonwealth*, 867 A.2d 711, 715 (Pa. Commw. Ct. 2005); *see also* Section 1921 of the Statutory Construction Act of 1972, 1 Pa. C.S. § 1921(a) (“The object of all interpretation and construction of statutes is to ascertain and effectuate the intention of the General Assembly.”). By proposing to remove the PWS 1 mg/L criterion and create a new toxic substance criterion, EQB disregards legislative intent of Act 40. Any review of the manganese water quality criterion itself should be done as part of DEP’s regular triennial review, not as part of implementing a simple, direct legislative requirement.

b. The proposed rulemaking does not comply with the Regulatory Review Act

The proposed regulation does not comply with the Regulatory Review Act, 71 P.S. §745.1 et seq. (“RRA”), for several reasons.

First, EQB must present a “proposed regulation” to IRRC for comment under Section 5 of the RRA, which means “a document intended for promulgation as a regulation.” 71 P.S. §745.3. Annex A, which embodies the First Alternative Point of Compliance, is the proposed regulation being presented to IRRC for consideration. The Second Alternative Point of Compliance is not a “proposed regulation.” It is not being presented to IRRC for comment as a proposal intended for promulgation as a regulation, it is merely a proposal on which EQB is seeking public comment. IRRC is not in a position to review the Second Alternative Point of Compliance under the criteria in Section 5.2 of the RRA. Therefore, the proposed rulemaking as it relates to this second alternative does not comply with the RRA.

Second, the RRA requires the promulgating agency to include “estimates of the direct and indirect costs to the Commonwealth, to its political subdivisions, and to the private sector.” 71 P.S. §745.5(a)(4). The Regulatory Analysis Form does not provide a complete economic analysis that considers the full impact of the proposed rule to these interests in Pennsylvania.

- Section 19 of the RAF addressing costs and savings to the regulated community simply states that “specific estimates of treatment cost and savings cannot be estimated at this time.” Estimating these costs is not difficult from readily available information known to

DEP. As discussed in Section VI below, the rule imposes very significant costs on the coal industry alone, an estimated \$44 to \$98 million annual increase in treatment costs.

- Section 20 of the RAF dealing with costs and savings to local governments states that “no costs will be imposed directly upon local governments.” This ignores the fact that many or most of the approximately 196 public water suppliers and 243 publicly-owned or other sewage treatment works in Pennsylvania which have NPDES permits containing manganese effluent limits or monitoring requirements will have to incur costs to treat manganese to a more stringent criterion if EQB adopts the proposal as a final rule.
- Section 21 of the RAF dealing with costs and savings to the state government states that “no costs will be imposed directly on state governments by this regulation.” This statement ignores the fact that DEP’s Bureau of Abandoned Mine Reclamation will be faced with significant cost to treat abandoned mine discharges for manganese to meet the proposed criterion.

This failure to estimate imposed costs is contrary to Sections 5(a)(4) and (10) of the RRA and impedes the ability of the regulated community, general public, and legislative oversight committees to offer meaningful and informed comments on the proposed regulation. It also hinders IRRC from being able to carry out its statutory duty to determine whether the rulemaking satisfies the RRA.

Third, the RRA requires a statement of need for the regulations. 71 P.S. §745.5(a)(3). In its statement of need, EQB states that DEP:

“reviewed current scientific and current toxicological information to comprehensively evaluate the manganese standard as it relates to the water uses identified in § 93.3 (related to protected water uses) and, in particular, to determine the need to develop manganese toxics criteria related to human health and aquatic life exposure...All of the residents and visitors of the Commonwealth will benefit from updating the Chapter 93 water quality criterion for manganese of 0.3 mg/L because it provides the appropriate protection for all water uses and users of the surface waters. Current scientific data demonstrates that manganese is a neurotoxin when levels to maintain adequate health are exceeded.”

(See Regulatory Analysis Form, section (10)).

In fact, there is no need for the proposed regulation because DEP has overestimated the toxic effects of manganese, the treatment costs for PWS operations, and the benefit to residents of Pennsylvania. As described in detail in paragraphs V and VIII below, based on the most current scientific data for manganese, which DEP failed to analyze, a manganese concentration of up to 2 mg/L in surface water does not pose a threat to human health or aquatic life. Further, revising the manganese criterion will adversely affect residents of the Commonwealth because it will likely force companies in the coal industry, and perhaps other vital industries as well, to shut down or dramatically curtail operations, resulting in more un-reclaimed abandoned mine lands.

Third, the RRA requires the RAF to provide a “detailed explanation of how the data was obtained and why the data is acceptable.” 71 P.S. §745.5(a)(14). EQB has not provided acceptable data because it is relying on scientific data that is over 17 years old to establish a proposed criterion of 0.3 mg/L, when current science suggests that manganese concentrations up to 2 mg/L are protective of human health. EQB has not provided the scientific support required for the implementation of the proposed rulemaking.

Lastly, the RRA requires “a description of any alternative regulatory provisions which have been considered and rejected and a statement that the least burdensome acceptable alternative has been selected.” 71 P.S. §745.5(a)(12). Based on this directive, EQB should clearly strike the “Second Alternative Point of Compliance” from the proposed rulemaking and consider only those comments in response to the proposed “First Alternative Point of Compliance.” The First Alternative is plainly the least burdensome alternative (and the only alternative that at least partly complies with Act 40), as it establishes the manganese criterion at the point of withdrawal. For these reasons, the proposed rulemaking is clearly contrary to the RRA.

c. The proposed manganese water quality criterion is not approvable by EPA because it is not based on sound science

The proposed manganese water quality criterion of 0.3 mg/L does not meet the requirements of the federal Clean Water Act and therefore is not approvable by EPA. EPA must approve a state’s proposed water quality standards under section 303(c) of the Clean Water Act before they may be used to establish water quality-based effluent limitations or TMDLs. EPA evaluates whether the state is adopting criteria that protect the designated water uses “based on sound scientific rationale consistent with 40 CFR §131.11.” 40 CFR 131.5(a)(2). EPA *must* disapprove the state’s water quality standards if they do not meet this standard. 40 CFR 131.5(b). Moreover, numeric criteria should be based on EPA’s section 304(a) guidance, section 304(a) guidance modified to reflect site-specific conditions, or “other scientifically defensible methods.” 40 CFR.131.11(b).

As discussed in more detail in paragraph V, the proposed 0.3 mg/L criterion is not based on sound scientific rationale or other scientifically defensible methods and therefore EPA cannot approve it. First, the rationale for proposing a 0.3 mg/L water quality criterion for manganese omits acknowledgment and discussion of the most current manganese data, which confirms that a modifying factor of 3 is unnecessary and that there is no conclusive evidence to suggest that exposure to manganese in drinking water at 2 mg/L will lead to adverse health effects in humans. (See Gradient Report attached as Exhibit B; Song *et al.* (2018);³ Yoon *et al.* (2019)⁴). There is

³ Song, G; Van Landingham, CB; Gentry, PR; Taylor, MD; Keene, AM; Andersen, ME; Clewell, HJ; Yoon, M. 2018. "Physiologically-based pharmacokinetic modeling suggests similar bioavailability of Mn from diet and drinking water." *Toxicol. Appl. Pharmacol.* 359:70-81. doi: 10.1016/j.taap.2018.09.023.

⁴ Yoon, M; Ring, C; Van Landingham, CB; Suh, M; Song, G; Antonijevic, T; Gentry, PR; Taylor, MD; Keene, AM; Andersen, ME; Clewell, HJ. 2019. "Assessing children's exposure to manganese in drinking water using a PBPK model." *Toxicol. Appl. Pharmacol.* 380:114695. doi: 10.1016/j.taap.2019.114695.

no indication that DEP was aware of or considered the most current data when it developed the proposed manganese water quality standard of 0.3 mg/L.

In addition, the proposed manganese criterion of 0.3 mg/L is not based on EPA's section 304(a) guidance, as required by 40 C.F.R.131.11(b). EPA chose not to regulate manganese with a National Primary Drinking Water Regulation (NPDWR) because manganese is generally not considered to be toxic. 68 Fed. Reg. 42898, 42903-04 (July 18, 2003) ("After reviewing the best available public health and occurrence information, EPA has made the determination not to regulate manganese with a NPDWR at this time, because it would not present meaningful opportunity for health risk reductions for persons served by PWS."). Instead, the federal criterion for manganese is a secondary maximum contaminant level, which is not based on toxic effects, but rather is intended to minimize laundry stains and objectionable tastes in beverages. Nevertheless, EQB rejected EPA guidance and proposed to regulate manganese as a toxic substance. Because the proposed criterion of 0.3 mg/L is not based on sound science and the rationale used to justify it is not scientifically defensible, EPA cannot approve it.

IV. The proposed rulemaking would regulate manganese in a manner inconsistent with the surrounding coal mining states

Under either Alternative, Pennsylvania would be regulating manganese in a manner inconsistent with the surrounding coal mining states who have all adopted less restrictive manganese standards.

In West Virginia, "*the manganese human health criterion shall only apply within the five-mile zone immediately upstream above a known public or private water supply used for human consumption.*" W. Va. Code § 47-2-6.2.d.

In Kentucky, all streams are designated for warm water aquatic habitat and primary and secondary contact recreation. "*The designation for domestic water supply is applicable only at points of intake.*" Section 401 K.A.R. 5:026. Kentucky does not have a PWS standard, an aquatic life standard, or a human health standard for manganese. Rather, it regulates mine discharges consistent with 40 C.F.R. §434.

In Indiana, "*all waters that are used for public or industrial water supply must meet the standards for those uses at the points where the water is withdrawn.*" 327 IAC 2-1-3(3). Indiana does not have a PWS standard or an aquatic life standard for manganese. Rather, it regulates mine discharges consistent with 40 C.F.R. §434.

In Illinois, "*...waters of the State shall meet the public and food processing water supply standards . . . at any point at which water is withdrawn for treatment and distribution as a potable supply or for food processing.*" 35 ILCS §303.202.

In Ohio, "*all surface waters within five hundred yards of an existing public water supply surface water shall be classified as 'Public Water Supply.'*" OAC 3745-1-07(B)(3)(a). Ohio does not have a PWS standard, an aquatic life standard, or a human health standard for manganese. Rather, it regulates mine discharges consistent with 40 C.F.R. §434.

If EQB adopts the proposed rulemaking, Pennsylvania's regulation of manganese will be at odds with and more stringent than every neighboring coal mining state.

V. Manganese does not pose a human health threat

The proposed manganese criterion of 0.3 mg/L is overly conservative and inconsistent with current science. The Human Health Rationale for the proposed manganese criterion of 0.3 mg/L was based on outdated science that has several limitations. First, DEP derived the proposed criterion of 0.3 mg/L by relying on EPA's 2002 IRIS profile which, based on scientific studies preceding 2002, recommended applying a modifying factor ("MF") of 3 to the oral reference dose ("RfD") for exposure to manganese in drinking water. EPA recommended an MF of 3 in 2002 because there was concern about possible increased uptake of manganese from drinking water, possible adverse health effects following lifetime human consumption of 2 mg/L manganese, and possible increased risk of manganese bioavailability in infants, especially those fed formula made with water containing manganese. However, scientific studies evaluating manganese as recently as 2019 indicate that an MF of 3 is no longer needed to derive a health-protective drinking water concentration for manganese. The updated science suggests not only that the amount of manganese absorbed from food or water does not differ, but also that children are no more at risk from manganese exposure in water than adults. (*See* Gradient Report, Section 3; Song *et al.* (2018); Yoon *et al.* (2019)). The results from these scientific studies was not available when EPA released its 2002 IRIS profile, which is now based on outdated science. Moreover, as referenced in Section III above, one year after the IRIS profile was released, EPA chose not to regulate manganese with an NPDWR because manganese is generally not considered to be toxic. 68 Fed. Reg. 42898, 42903-04 (July 18, 2003) ("After reviewing the best available public health and occurrence information, EPA has made the determination not to regulate manganese with a NPDWR at this time, because it would not present meaningful opportunity for health risk reductions for persons served by PWS.").

Application of an MF of 3 to the manganese oral RfD is no longer consistent with the best available science; therefore, DEP should not have applied it to the derivation of a manganese drinking water value. Removing the MF of 3 to be consistent with current science results in a manganese criterion of approximately 1 mg/L, which is equivalent to the current PWS manganese criterion. (*See* Gradient Report, Section 4.1). Therefore, the current manganese criterion of 1 mg/L is protective for human consumption and should not be revised. Further, because manganese is a naturally occurring element that is an essential nutrient, sustaining deficient levels of manganese in the human body (typically below 1 mg/day) is associated with adverse health effects, such as impaired bone development. (*See* Gradient Report, Section 2.1; IOM, (2001)). Furthermore, as DEP has adopted EPA's secondary maximum contaminant level of 0.05 mg/L for manganese in finished drinking water, the likelihood that any consumer would regularly drink water exceeding 1 mg/L is extremely low.

The Human Health Rationale on which EQB's rulemaking is based, in part, also relied on several community studies with serious limitations that make it impossible to attribute the reported effects to manganese. (*See* Agency for Toxic Substances and Disease Registry (2012)

and Health Canada (2019) (discussing limitations of Bouchard et al. (2011), Khan et al. (2011, 2012), Oulhote et al. (2014))). Although these limitations preclude deriving conclusions from these studies regarding manganese in water and potential health effects in humans, they were used in developing the proposed rulemaking. When considering the most current science, maintaining a manganese criterion of 1 mg/L at the point of intake for an existing or planned potable water supply is protective for human consumption.

VI. The proposed rulemaking would impose significant, unnecessary costs to the coal mining industry

The proposed manganese criterion of 0.3 mg/L would pose significant, unnecessary costs on the coal mining industry, especially if it is imposed at the discharge location under the second alternative of the proposed rule. Specifically, a 0.3 mg/L criterion would result in additional costs associated with the doubling to tripling of chemical use, handling and disposing of increased sludge volumes, additional or new treatment technologies to comply with pH limits, and additional or new treatment technologies to meet aluminum effluent limits. (*See Tetra Tech Report, attached as Exhibit C, Section 5*). If the proposed criterion is adopted, annual treatment costs for the coal mining industry are estimated to increase by \$44 to \$98 million, with increased alkaline chemical addition expected to emit approximately 45,000 additional tons of carbon dioxide each year. The treatment improvements that may be necessary to control pH and address the conflicting effluent limits for manganese and aluminum are estimated to result in additional costs upwards of \$200 million. (*See Tetra Tech Report, Section 5*).

In contrast, PCA understands that manganese can already be easily treated by public water supply operators. Fate and transport evaluations indicate that manganese in treated mine water effluent is likely to be oxidized to insoluble forms and precipitated in a stream within a short distance (less than one-half to one mile) from a typical coal mining discharge point. (*See Tetra Tech Report, Section 6*). It is unlikely that manganese from a treated discharge could reach a withdrawal point in a dissolved form that would require additional treatment by water supply operators. In other words, once treated effluent from a coal mine facility reaches a PWS withdrawal, manganese is more likely to be found in a particulate form that would not require additional treatment by the water supplier. In explaining the history of the current 1 mg/L PWS criterion, the DEP Bureau of Clean Water Rationale document states that, in 1979, an “average up-to-date water plant can probably handle soluble manganese concentrations without too much difficulty. A well-designed plant can handle 1.5 to 2 parts per million.” (*See DEP Rationale document, page 2*). Further, public water suppliers’ current conventional treatment systems already have the chemicals, feed systems (e.g., pre-chlorination), and treatment processes (sedimentation and filtration) that can remove manganese from source waters and are unlikely to require process modifications or new equipment.

VII. The proposed rulemaking would impose significant, unnecessary costs to other vital industries

As of February 2020, DEP issued NPDES permits containing manganese limits or monitoring requirements to at least 99 non-coal mines, 174 industrial facilities, 196 public water suppliers, and 243 publicly-owned or other sewage treatment works in Pennsylvania. Treating manganese to the proposed criterion of 0.3 mg/L would impose additional unnecessary costs to these public and private permittees, especially if the criterion is set at the discharge point, with no measurable benefit to the environment. For example, the Vice President of Production and Operations of New Enterprise Stone & Lime Company testified at the Senate Environmental Resources and Energy Committee Manganese Rulemaking Hearing on September 9, 2020 that complying with the proposed new limits would increase capital costs from \$150,000 to \$320,000 for its facilities and add \$450,000 a year in operating costs. Further, testimony indicated that some of the company's facilities cannot meet the proposed 0.3 mg/L and would be forced to shut down because of treatment space limitations, an inadequate number of employees, and/or inability to pass along increased treatment costs to customers. The proposed criterion of 0.3 mg/L could severely strain, or worse, shut down basic materials companies, without benefiting the public or environment.

VIII. The current manganese BAT limits adequately protect aquatic life

The proposed manganese criterion of 0.3 mg/L is unnecessary because manganese is not toxic to aquatic life at concentrations expected to be encountered in Pennsylvania. Specifically, EPA has not published recommended maximum or continuous concentration criteria for manganese to prevent acute or chronic toxicity impacts to aquatic life in surface water. Toxicological information indicates that the federal BAT limits of 2 mg/L (monthly average) and 4 mg/L (daily maximum) for the coal industry protect fish and aquatic life, including the most sensitive aquatic species which has an acute manganese toxicity concentration of 8.6 mg/L, which is much higher than current BAT limits. (*See Tetra Tech Report, Section 7*).

It is also important to remember that the acute toxicity of manganese to aquatic life depends on water hardness. Therefore, because mine drainage has a higher hardness concentration than laboratory test conditions reported in these studies, the concentration at which manganese is acutely toxic to aquatic species in surface waters will be greater than what is reported in laboratory studies (i.e. greater than 8.6 mg/L for the most sensitive species). Therefore, the manganese BAT effluent limits of 2 mg/L and 4 mg/L provide adequate protection for even the most sensitive freshwater fish and aquatic life whether in surface water or laboratory conditions.

IX. Incidental consequences

The proposed rulemaking would also result in significant negative unintended consequences. It is very likely to:

- Substantially curtail if not eliminate Subchapter-F and Subchapter G remaining incentives under Pennsylvania's surface mining program (25 Pa. Code Chapter

87, Subchapter F and 25 Pa. Code Chapter 88, Subchapter G) because it will disincentivize operators from re-mining and reclaiming abandoned mines if they must treat non-Subchapter F and non-Subchapter-G discharges to 0.3 mg/L due to the treatment costs being too high.

- Result in more abandoned mine sites, as current operators may simply forfeit bonds for post-mining discharges because they cannot afford the cost increase necessary to cover the higher treatment costs. As a result, water treatment now being performed by operators at no cost to the state will be discontinued.
- Cause fewer watershed organizations to volunteer to reclaim abandoned mines because of the significant additional costs to comply with the more stringent proposed criterion of 0.3 mg/L. Therefore, these valuable environmental protection projects will likely stop.
- Impose significant costs on the Bureau of Abandoned Mine Reclamation program to treat or fund treatment of abandoned mine land discharges.
- Impose significant costs on DEP to treat the sites permitted under the Commonwealth's now defunct Alternative Bond System, pursuant to the *Pennsylvania Federation of Sportsmen's Clubs, Inc. v. Quigley*, Civil No. 1:99-CV-1791 (M.D. Pa.) (Settlement Agreement, 11/2/2016).
- Lead to the recalculation of post-mining discharge treatment trust funds to address increased manganese treatment costs where, in many cases, the trusts are underfunded and the responsible companies no longer exist.
- Dramatically reduce the use of general permits for stormwater, such as MS4 or industrial stormwater permits, because regulating manganese as a toxic substance will likely disqualify the use of general permits where manganese is present in the discharge. Most facilities that currently use general stormwater permits do not treat for manganese. They now would need to obtain an individual NPDES permit, which will increase cost and delay projects, some undoubtedly to the point of not proceeding at all.

X. Conclusion

The proposed rulemaking is contrary to law, is unnecessary to protect human health and fish and aquatic life, will significantly increase treatment costs for several vital Pennsylvania industries, will not simplify treatment processes or dramatically decrease treatment costs for public water supply operators, and will result in significant unintended negative consequences to the Commonwealth. For these reasons, PCA requests that EQB withdraw the rule as proposed

and promulgate the rule as directed by Act 40. EQB should apply the current manganese water quality criterion of 1 mg/L at the point of intake of existing and planned PWS.

For these reasons, PCA also requests that EQB maintain the current 1 mg/L PWS standard; remove the proposed water quality criteria for toxic substances, as the criterion derived is based on outdated science; and lastly, comply with Act 40 and promulgate the exception in 96.3(d) as provided in the proposed rulemaking.

Sincerely,

A handwritten signature in black ink that reads "Rachel Gleason". The script is cursive and fluid, with the first name "Rachel" and last name "Gleason" clearly distinguishable.

Rachel Gleason
Executive Director
Pennsylvania Coal Alliance

Exhibit A

**Pennsylvania Coal Alliance Comments on
Advance Notice of Rulemaking**

February 26, 2018

February 26, 2018

PA Department of Environmental Protection
Policy Office
400 Market Street
P.O. Box 2063
Harrisburg, PA 17105-2063

Re: Water Quality Standard for Manganese; Advance Notice of Proposed Rulemaking
[48 Pa.B 605]
[Saturday, January 27, 2018]

Dear Secretary McDonnell:

Pursuant to the public notice published by the Pennsylvania Bulletin on January 27, 2018, the Pennsylvania Coal Alliance (PCA) offers the following comments on the Pennsylvania Department of Environmental Protection's Water Quality Standard for Manganese; Advance Notice of Proposed Rulemaking (ANPR) [48 Pa.B 605].

The PCA is the principal trade organization representing underground and surface bituminous coal operators in Pennsylvania, as well as other associated companies whose businesses rely on coal mining and a strong coal economy. PCA member companies produce nearly 90 percent of the bituminous coal mined annually in Pennsylvania, making our Commonwealth the third largest coal producing state in the nation.

As indicated in the ANPR, on October 30, 2017, subsection (j) was added to §1920-A of the Administrative Code of 1929 (Act 40 of 2017). Act 40 directed the Environmental Quality Board to promulgate proposed regulations within 90 days requiring that the water quality criteria for manganese established under 25 Pa Code Chapter 93 (relating to water quality standards) shall be met consistent with the exception in 25 Pa Code §96.3(d) (relating to water quality protection requirements).

Initially, in the development of current regulations, manganese was chosen because it was seen as a surrogate for many metals. Hence, the control of manganese was thought to control other metals. This was based on the notion that manganese requires high pH and/or circumneutral pH with a strongly oxidizing environment in order to precipitate. Thus, theoretically, all transition metals should precipitate in conditions that would precipitate manganese. We have since learned that this theory does not work, especially for aluminum or oxyanions such as selenite, and as such the U.S. Environmental Protection Agency (EPA) has taken the position

that all water quality constituents should be addressed on an individual basis. Accordingly, metals such as aluminum and iron are individually regulated.

At most, adding manganese to the group of constituents for which the water quality criterion must be met at the point of all existing or planned surface potable water withdrawals found in 25 Pa Code §96.3(d) raises the manganese limit for coal mining discharges from 1.0 mg/l to the federally allowable limit of 2.0 mg/l at the point of discharge, aligning Pennsylvania with other states and the federal regulations. Manganese discharges from coal mining operations are, based on a survey of PCA operating members and consultants, on average greater than 40 miles from a PWS intake. When factoring for the federal discharge limit of 2.0 mg/l, assimilation, and with the added protection of the required reasonable potential analysis found in 40 CFR §122.44 and incorporated at 25 Pa. Code §92a.44, the discharge of this naturally occurring element from mining facilities will not affect any water supply withdrawals, will protect aquatic life from harmful chemical over-treatment, and will provide a positive economic benefit to the coal industry, watershed associations, and other organizations that treat mine water or acid mine drainage from abandoned legacy sites.

The Department requested specific information in three subject matter areas: (1) the compliance point for the manganese standard, (2) the adequacy of the existing PWS Chapter 93 standard, and (3) the development of standards for other protected uses. We address each of these subjects below.

I. INFORMATION REGARDING THE CHAPTER 96 COMPLIANCE POINT FOR THE Mn STANDARD

Changing the compliance point as directed by Act 40 will not change the maximum concentration of manganese permissible at a PWS point of intake because federal and state law limit the discharge from coal mining facilities to 2.0 mg/l on a daily average.

FEDERAL EFFLUENT LIMITATION GUIDELINES

Discharges from coal mining operations have been subject to federal effluent limitations guidelines and standards at 40 CFR Part 434 (ELGs) for over 40 years. EPA's coal mining ELGs apply to discharges from active coal mines, as well as coal preparation plants and associated areas. The coal mining ELGs cover six separate subcategories of coal mining operations, five of which could apply to coal mining activities in Pennsylvania. The coal mining ELGs are technology-based limits aimed at preventing pollution by requiring effluent quality attainable using demonstrated treatment technologies.

Manganese is one of only two nonconventional pollutants that EPA chose to regulate as part of the coal mining ELGs. Based on manganese removals achieved by the best available technology economically achievable (BAT) for certain coal mining operations, EPA imposed a 2.0 mg/l manganese effluent limitation, based on an average of daily values for 30 consecutive days. This manganese BAT limitation has been in place since 1985, and coal mining operations in

Pennsylvania and across the country must meet this limit before discharging to a receiving water. Coal mining operations have been, and will continue to be, subject to EPA's 2.0 mg/l end-of-pipe effluent limitation for manganese. Therefore, moving the compliance point for the Commonwealth's manganese water quality criteria to PWS intakes would not mean that coal mining operations would be free to discharge unrestricted levels of manganese into receiving waters.

OTHER COAL STATES

Pennsylvania's application of the 1.0 mg/l PWS criterion for manganese at coal mining discharges is more restrictive than any other coal mining state, and, as previously detailed, more restrictive than federal regulations. Specifically:

In Ohio, designated uses are provided in Chapter 3745-1-07 of the Ohio Administrative Code. Section (B)(3)(a) designates that "*... all surface waters within five hundred yards of an existing public water supply surface water shall be classified as 'Public Water Supply.'*" Further, Ohio does not have a PWS standard, an aquatic life standard, or a human health standard for manganese. Rather, it regulates mine discharges consistent with 40 CFR §434.

In Kentucky, all streams, according to 401 Kentucky Administrative Regulations 5:026, are designated for warm water aquatic habitat and primary and secondary contact recreation. "*The designation for domestic water supply is applicable only at points of intake.*" Further, Kentucky does not have a PWS standard, an aquatic life standard, or a human health standard for manganese. Rather, it regulates mine discharges consistent with 40 CFR §434.

In Illinois, per Title 35, §303.202 "*...waters of the State shall meet the public and food processing water supply standards . . . at any point at which water is withdrawn for treatment and distribution as a potable supply or for food processing.*"

In Indiana, Title 327, Article 2, "*All waters that are used for public or industrial water supply must meet the standards for those uses at the points where the water is withdrawn.*" Further, Indiana does not have a PWS standard or an aquatic life standard for manganese.

In West Virginia, "*The manganese human health criterion shall only apply within the five-mile zone immediately upstream above a known public or private water supply used for human consumption.*" [citation]

MINE WATER TREATMENT COST

The Department asked for information relating to the financial and economic impacts, and the cost or savings to the regulated community, including small businesses and state and local governments, of changing the point of compliance for manganese.

Coal mine discharges are located mostly in remote extreme headwater reaches far away from public drinking water supplies. As previously mentioned, based on a survey of PCA operating members and consultants, discharge sites are on average greater than 40 miles from a PWS intake. Given that all waters of the state do not serve, either legally or practically, as public drinking water supplies, the application of the PWS standard at such distances is overly restrictive and nonsensical. Water quality criteria for public drinking water withdrawals are protective of the use if the criteria are met at the point where the water is withdrawn for treatment for human consumption. The Department will employ its reasonable potential analysis to determine on a case-by-case basis whether a manganese limit is necessary for a permit.

Applying the federal technology-based 2.0 mg/l standard at the discharge, and the 1.0 mg/l PWS standard at the withdrawal, would result in significant chemical cost savings to coal mining operations. Discharge flow rates vary based on a number of factors, including the type of operation, season, and precipitation. However, as a reasonable estimate, treating coal mine discharges to 2.0 mg/l costs approximately \$.00065 per gallon per minute (gpm) to treat manganese. If an average discharge rate is 200 gpm, the chemical cost savings for caustic addition at one discharge would be over \$68,000 annually. Considering there are hundreds of National Pollution Discharge Elimination System permits for coal mining operations in Pennsylvania, including facilities being operated by watershed associations and other non-profit organizations to treat acid mine drainage from legacy operations, the economic impact could be upwards of a million dollars.

II. INFORMATION RELATING TO THE ADEQUACY OF THE EXISTING PWS CHAPTER 93 Mn STANDARD

FEDERAL REGULATIONS FOR DRINKING WATER

On June 3, 2002, EPA published a preliminary notice not to regulate manganese with a National Primary Drinking Water Regulation (NPDWR):

“The Agency has made a preliminary determination not to regulate manganese with a NPDWR because it is generally not considered to be very toxic when ingested with diet because drinking water accounts for relatively small proportion of manganese intake.”¹

The EPA finalized its initial determination on July 18, 2003:

“After reviewing the best available public health and occurrence information, EPA has made the determination not to regulate manganese with a NPDWR at this time, because it would not

¹ 67 Fed. Reg. 38235 (June 3, 2002), pages 38235-38236

present meaningful opportunity for health risk reductions for persons served by PWS.”²

EPA has established .05 mg/l for manganese as a secondary maximum contaminant level (SMCL) in National Secondary Drinking Water Regulations. SMCLs are established to provide guidance to public water systems in managing drinking water for aesthetic considerations such as taste, color and odor and are not considered to present a risk to human health. Contrary to the Department’s ANPR, SMCLs are not enforceable, and public water systems only test for SMCLs on a voluntary basis.³ Manganese is listed as a SMCL for aesthetic reasons such as laundry staining, and organoleptic effects like taste.

PCA believes that EPA’s conclusions are sound and that there is no reason to revisit the SMCL or the underlying data on which it is based.

STATE REGULATIONS AND TECHNICAL GUIDANCE DOCUMENT

The effluent limitation for manganese related to coal mining activities, found in 25 PA Code Chapters 87 (bituminous surface), 88 (anthracite), 89 (bituminous underground), and 90 (coal refuse) and mirroring 40 Code of Federal Regulations §434, is 2.0 mg/l average daily value for 30 consecutive days and 4.0 mg/l daily maximum.

25 Pa Code, Chapter 93 establishes criteria for a Potable Water Supply (PWS) which, for manganese, is 1.0 mg/l. It is important to note that PWS is defined in 25 Pa Code Chapter 93 as “Used by the public as defined by the Federal Safe Drinking Water Act, 42 U.S.C.A §300F, or by other water users that require a permit from the Department under the Pennsylvania Safe Drinking Water Act (35 P.S. §631-641), after conventional treatment, for drinking, culinary, and other domestic purposes, such as inclusion into foods, either directly or indirectly.” Thus, the 1.0 mg/l manganese criterion is not a human health criterion, it is taste and odor criterion that applies after conventional treatment of water. Therefore, there should not be additional water-quality-based permitting for Mn, beyond what is regulated in 25 Pa Code, Chapters 86 - 90 and 40 CFR Part 434 in discharges of water from an area disturbed by coal mining activities that do not have a reasonable potential to interfere with a public water supply intake. However, at present, the Department is applying the aforementioned PWS standard, a standard for drinking water, directly at the outfalls from permitted coal mining treatment facilities and sedimentation ponds.

After the Environmental Quality Board promulgates a regulation establishing that the water quality criteria for manganese be met consistent with the exception in 25 Pa Code §96.3(d), consistent with 40 CFR §122.44 and 25 Pa. Code §92a.44, permit writers will use the

² 68 Fed. Reg. 42898 (July 18, 2003), pages 42903-42904

³ Secondary Drinking Water Standards: Guidance for Nuisance Chemicals

<https://www.epa.gov/dwstandardsregulations/secondary-drinking-water-standards-guidance-nuisance-chemicals>

Department's Technical Guidance Document (TGD) 563-2112-115, *Developing National Pollutant Discharge Elimination System (NPDES) Permits for Mining Activities*, to evaluate whether a water-quality-based effluent limitation for manganese or another constituent is necessary in a coal mining activity permit to ensure that the water quality criteria in Section 93.7 will be met at the specified point of compliance in Section 96.3, which includes public water supply intakes. . This is known as a reasonable potential analysis. If there is a reasonable potential to exceed §93.7 levels, a more stringent and appropriate effluent limit may be applied to the NPDES permit to protect existing and designated surface water uses.

III.

INFORMATION RELATED TO THE DEVELOPMENT OF ADDITIONAL CHAPTER 93 Mn STANDARDS FOR OTHER PROTECTED USES

FEDERAL REGULATIONS FOR AQUATIC LIFE

There are no federally recommended acute or chronic criteria for manganese toxicity or freshwater organisms.

HUMAN HEALTH

Manganese is a naturally occurring element that constitutes approximately 0.1% of the Earth's crust. Manganese occurs naturally at low levels in soil, water, and food and is essential for normal physiological functioning in humans and all animal species.⁴ Manganese is an element essential to the proper functioning of both humans and animals, as it is required for the functioning of many cellular enzymes and can serve to activate many others.⁵

Like many water quality constituents, in excess manganese can be toxic, however, deficiencies may also prove harmful. Foods high in manganese include mussels, clams, nuts, pumpkin seeds, pineapple, whole wheat bread, tofu, beans, fish, spinach, whole grains, and black tea. Dining on 6 ounces of mussels results in the ingestion of 11.6 mg of manganese; add 100 g of whole wheat bread to the meal and another 2.174 mgs is ingested. Manganese is a nutritionally essential mineral necessary for antioxidant function, bone development, and metabolism.⁶ A review of typical Western and vegetarian diets found average adult manganese intakes ranging from 0.7 to 10.9 mg/day, with the upper range manganese intake value of 11.0 mg/day from dietary studies is considered a no observed-adverse effect level (NOAEL). It is not believed that this amount of manganese in the diet represents an overexposure to the element.⁷

⁴ 67 Fed. Reg. 38235 (June 3, 2002), pages 38235-38236

⁵ World Health Organization. Manganese in Drinking-water: Background document for development of WHO Drinking-water quality. World Health Organization 2011; page 1.

⁶ Oregon State University. Linus Pauling Institute Micronutrient Information: Manganese

⁷ World Health Organization. Manganese in Drinking-water: Background document for development of WHO Drinking-water quality. World Health Organization 2011; page 4.

The World Health Organization's Guidelines for Drinking-water Quality lists manganese as a naturally occurring chemical that has no adverse health effects,⁸ but does provide acceptability aspects for taste, odor and appearance.⁹

EPA's Integrated Risk Information System summary indicates that while average levels of manganese in various diets have been determined, no quantitative information is available, and environmental, biological, and host factors such as alcohol consumption, anemia, liver function, and general nutritional status can significantly influence an individual's manganese status. Further, of the one study describing toxicologic responses in humans consuming large amounts of manganese, it was determined that the concentration of manganese exposure was as high as 28.0 mg/l¹⁰, which is fourteen times the criterion in 25 Pa Code Chapters 86-90 and 40 CFR §434.

Applying the criterion for manganese regulated under 25 Pa Code Chapters 86 - 90 and 40 CFR §434 would have no adverse impact on human health. In addition, the Department's Technical Guidance Document (TGD) 563-2112-115, *Developing National Pollutant Discharge Elimination System (NPDES) Permits for Mining Activities* already requires a reasonable potential analysis to ensure that a coal mine discharge does not increase the constituent concentration above the listed requirements of 25 PA Code §93.7 for public water supply intakes. Therefore, promulgating this regulation would have no adverse impact on human health.

AQUATIC LIFE

While manganese has low toxicity to aquatic life, its treatment and removal can be highly dangerous for fish and invertebrates due to the tremendous increase in pH required for manganese removal. This is evident based on review of an Eh-pH diagram for manganese as compared to iron.¹¹ Removal of manganese from mine drainage requires either high pH (generally greater than 9.0, often at 10.5 or 11.0) or strong oxidation combined with near-neutral pH. Because of the difficulties in obtaining strong oxidation sufficient to remove manganese, pH adjustment is necessary.

Treating manganese to accomplish a limit of 1mg/l requires significant caustic addition to achieve high pH levels in treatment ponds. High pH levels in the discharge can cause a more significant adverse harm to the receiving stream's aquatic life than a manganese concentration in the discharge of up to 2.0 mg/l, which is the best available technology (BAT) standard. The national recommended criteria for pH is limited to 6.5 to 9.0 due to the impact on aquatic life. A pH range of 6.5 to 9.0 protects fish and aquatic life, which Pennsylvania has adopted as a water quality criterion in Chapter 93. Outside of this range, fish suffer adverse physiological

⁸ World Health Organization. Guidelines for Drinking-water Quality: Fourth edition incorporating the first addendum. Chemical aspects; pages 155-210

⁹ World Health Organization. Guidelines for Drinking-water Quality: Fourth edition incorporating the first addendum. Acceptability aspects; pp. 219-23

¹⁰ U.S. Environmental Protection Agency. Integrated Risk Information System Chemical Assessment Summary: Manganese, Reference Dose for Oral Exposure; page 7.

¹¹ *Methods for Passive Removal of Manganese from Acid Mine Drainage*, Rose et al.

effects increasing in severity as the degree of deviation increases until lethal levels are reached.¹² Further, while aluminum is relatively insoluble at pH 6 to 8, the solubility of aluminum increases under alkaline conditions. Thus, increase in pH for treatment of manganese at 1.0 mg/l results in soluble aluminum, $Al^{+4} (OH)_4$, which is toxic to aquatic life.

Other states have acknowledged, and US EPA has concurred, that an aquatic life criteria for manganese is not necessary. Until the mid-1990s, West Virginia maintained a water quality criterion of 1.0 mg/L for manganese in streams classified as either public drinking water supplies or aquatic life uses. In 1997, after an exhaustive review of technical information and supporting scientific data, the West Virginia Environmental Quality Board deleted the aquatic life criterion for manganese. EPA Region III subsequently approved the deletion of the aquatic life criterion for manganese.

In 1995, a Penn State University professor, Dr. Dean Arnold, assisted by Penn State graduate students, began monitoring the benthic macroinvertebrate community in Otter Run in Lycoming County for impairment from exposure to manganese. Later, in 1998, Normandeau Associates took over monitoring, and in 2000 a new sampling and data analysis methodology was developed by a work group that included consultants, the PA Fish and Boat Commission, and US Geological Survey, which was put in effect in August 2000 and continues today. This methodology, which is used in determining a significant loss of biota, has resulted in the determination that the benthic macroinvertebrate community is not considered impaired at the manganese levels measured, which frequently exceeded 2mg/l, often by more than double.¹³

Further, while the vast majority of research in US EPA'S ECOTOX database for aquatic life toxicity was conducted on species not native to Appalachia or in some instances the United States and not appropriate for use in a criteria calculation, ECOTOX does indicate that manganese has low toxicity to aquatic life.¹⁴

CONCLUSION

Commenters have expressed concern that the implementation of this regulation would mean that manganese discharges would be unregulated. This is simply untrue. As stated above, technology-based effluent limitation guidelines for manganese are well established, both under state and federal regulations, at 2.0 mg/l for activities specific to coal mining.

Adopting the regulation required by Act 40, which requires the water quality criteria for manganese to be met consistent with the exception in 25 Pa. Code §96.3(d) (relating to water quality protection requirements), will serve multiple, common sense purposes; it will protect aquatic life from harmful chemical over-treatment of manganese discharges; it will have no impact on human health; it will not increase cost for water treatment facilities; it will result in several millions of dollars in chemical cost savings to coal mining operations, watershed

¹² U.S. EPA Quality Criteria for Water, 1986. Page 180.

¹³ Attachment A.

¹⁴ Attachment B.

associations and other non-profit organizations that treat mine discharges; and, it will align Pennsylvania with other states and the federal government, which will allow Pennsylvania's coal producers to be more competitive with their out-of-state competitors. Given these considerations, we urge the Department and the EQB to move forward with the implementation of Act 40 as expeditiously as possible.

PCA appreciate the opportunity to comment on the Department's ANPR. Please direct any questions or comments to Rachel Gleason at gleason@pacoal.org at your earliest convenience.

Sincerely,

A handwritten signature in dark ink that reads "Rachel Gleason". The script is cursive and fluid, with the first name and last name clearly distinguishable.

Rachel Gleason
Executive Director
Pennsylvania Coal Alliance

Exhibit B

**Comments on the Proposed Pennsylvania
Ambient Water Quality Criterion for Manganese**

Gradient Corporation

July 28, 2020

Comments on the Proposed Pennsylvania Ambient Water Quality Criterion for Manganese

Prepared for
Pennsylvania Coal Alliance
212 North Third Street
Suite 203
Harrisburg, PA 17101

July 28, 2020



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Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criterion
IRIS	Integrated Risk Information System
MF	Modifying Factor
Mn	Manganese
NAWQA	National Water-Quality Assessment
NOAEL	No-Observed-Adverse-Effect Level
PADEP	Pennsylvania Department of Environmental Protection
PBPK	Physiologically Based Pharmacokinetic
ppm	Parts Per Million
RfD	Reference Dose
UF	Uncertainty Factor
US EPA	United States Environmental Protection Agency
USGS	United States Geological Survey
WHO	World Health Organization

1 Introduction

Gradient is providing comments on the Pennsylvania Department of Environmental Protection (PADEP) Proposed Manganese (Mn) Ambient Water Quality Criterion (AWQC).

Through its Proposed Rulemaking published on July 25, 2020 (PADEP, 2020), the Environmental Quality Board is soliciting comments on the Proposed Rulemaking related to the Mn AWQC and its implementation. The Proposed Rulemaking was drafted in response to an amendment that was added on October 30, 2017, to Section 1920-A of the Administrative Code of 1929 ("Act 40"; Pennsylvania General Assembly, 2017), which mandated that the Environmental Quality Board promulgate regulations within 90 days of its enactment that require the water quality criterion for Mn must "be met, consistent with the exception in 25 Pa. Code § 96.3(d) (relating to water quality protection requirements)." Section 96.3(d) requires that the water quality criteria for several constituents, now to include Mn, shall be met at least 99% of the time at the point of all existing or planned surface potable water supply withdrawals unless otherwise specified (PADEP, 2000). The Environmental Quality Board and PADEP are using the Proposed Rulemaking to collect broader information relating to the adequacy of the current Pennsylvania regulatory Mn criterion in 25 Pa. Code Chapter 93. PADEP, in its Advance Notice of Proposed Rulemaking, specifically requested "[p]eer-reviewed, published toxicological studies, reports, and data on human health effects resulting from exposure to Mn in water" (PADEP, 2018a, p. 4).

Based on information received during the development of the Proposed Rulemaking, and as presented in the July 25, 2019 meeting materials to the Water Resources Advisory Committee (PADEP, 2019a), PADEP is proposing to revise the current Mn AWQC of 1 mg/L to a value of 0.3 mg/L based on concern for possible health effects at 1 mg/L Mn. It should be noted that regardless of the proposed Mn AWQC, PADEP has adopted a secondary maximum contaminant limit for drinking water of 0.05 mg/L Mn (PADEP, 2006). Thus, whether the point of compliance for the Mn AWQC is at the point of discharge or the point of intake, a public water system (as defined in 25 Pa. Code Chapter 109) must supply drinking water (*i.e.*, from the tap) that meets the Secondary MCL of 0.05 mg/L Mn (25 Pa. Code § 109.202(b)(1)).

This report evaluates whether the current Mn AWQC (1 mg/L)¹ is protective for human consumption, and whether PADEP's proposed Mn AWQC (0.3 mg/L) is necessary for cases (albeit rare) in which consumers drink water containing Mn at concentrations above the secondary maximum contaminant level of 0.05 mg/L Mn. As described in the comments herein, **recent peer-reviewed scientific information indicates that a 0.3 mg/L Mn value is overly conservative and is not consistent with current science related to Mn, which indicates that 1 mg/L Mn in drinking water is not expected to lead to adverse health effects in people. Overall, based on the available studies, there is no conclusive evidence to suggest that exposure to Mn in drinking water at 2 mg/L is associated with adverse health effects. Therefore, the current 1 mg/L Mn AWQC is protective for human consumption.**

Our comments are summarized in the sections below. Section 2 provides background on sources of Mn and how PADEP derived the proposed Mn AWQC, Section 3 describes the most up-to-date science on Mn and critiques current regulatory Mn drinking water values and PADEP's proposed Mn AWQC, and Section 4 describes Gradient's critique of the current AWQC for Mn based on updated science and also calculates a surface water value that is protective for a non-drinking water scenario (*i.e.*, swimming and fish ingestion).

¹ We understand that the current AWQC of 1.0 parts per million (1 ppm, or 1 mg/L) was originally established based on taste and odor concerns and to prevent laundry staining (PADEP, 2018b).

Section 5 presents our conclusions. We provide a discussion and critique of PADEP's (2019b) Rationale for the proposed Mn AWQC in Appendix A.

2 Background Information

2.1 Sources of Mn

Manganese (Mn) is a naturally occurring element that serves as an essential nutrient in the human body (US EPA, 2002). Maintaining sufficient levels of Mn is important for a number of bodily functions, including proper bone and cartilage formation, cell energy production, and neuronal functioning in humans. Exposure to elevated levels of Mn, particularly *via* inhalation in occupational settings, may cause adverse neurological effects (ATSDR, 2012), as excess Mn accumulates in the brain (Aschner, 1999; Chen *et al.*, 2015). However, sustaining deficient levels of Mn in the body (typically below 1 mg/day) is also associated with adverse health effects, such as effects on bone development (IOM, 2001). Under the quality criteria in 25 Pa. Code §93.8c, several substances with human health criteria values, such as arsenic and cyanide, are toxic and generally are not considered to be essential nutrients in the human body. Unlike these toxic substances, maintaining appropriate concentrations of Mn in the body is critical for human health and does not cause adverse health effects under normal dietary exposures.

The primary source of Mn intake in the general population is through the diet, with adults typically consuming between 1 and 10 mg Mn/day, approximately 1-5% of which is absorbed in the gut (ATSDR, 2012; IOM, 2001). The greatest sources of Mn in the diet are typically from vegetables, such as grains, beans, and nuts; thus, vegetarians may have a higher intake of Mn than the average person (ATSDR, 2012). Vegetarian diets containing up to 20 mg/day Mn have not been shown to be associated with adverse health effects (Schroeder *et al.*, 1966; Greger, 1999).

Mn intake from water consumption is often much lower than Mn intake from food (US EPA, 2002; WHO, 2003). When Mn is ingested, either from food or water, a portion of the Mn becomes bioavailable; *i.e.*, Mn enters the blood stream and potentially becomes "available" to be absorbed into or interact with various tissues in the body. There is little evidence to suggest that Mn bioavailability differs as a function of food or water ingestion (US EPA, 2002). Mn bioavailability is further discussed in Section 3, as a number of recent studies have been published on this topic that provide insight into Mn bioavailability and toxicity.

2.2 PADEP's Derivation of the Proposed Mn AWQC

PADEP (2019a) relies on the United States Environmental Protection Agency (US EPA) (2002) Integrated Risk Information System (IRIS) assessment of Mn to derive the proposed Mn AWQC. This includes US EPA's identified no-observed-adverse-effect level (NOAEL), oral reference dose (RfD), and a modifying factor (MF) for drinking water ingestion pathways for Mn (US EPA, 2002). An RfD is typically derived from a NOAEL, which is defined as the highest dose of chemical (across all reliable studies available) that is associated with no adverse health effects. The NOAEL is often divided by uncertainty factors (UFs) or MFs to account for uncertainties, such as extrapolation from animals to humans or variability within the human population, to derive the RfD. In this way, an RfD is a dose metric that describes a daily oral exposure to a chemical that is unlikely to produce an appreciable risk of deleterious effects during a lifetime for the general population.

US EPA's oral RfD for Mn is equal to 0.14 mg Mn/kg-day (US EPA, 2002). According to US EPA (2002), this oral RfD is derived from composite data from several studies showing that chronic human consumption

of food containing up to 10 mg Mn per day was not associated with neurological effects.² Thus, US EPA's oral RfD takes into account Mn from dietary sources (*i.e.*, food only). Based on the Mn science available at the time (described further in Section 3), US EPA (2002) also recommended that an MF of 3 be applied to the oral RfD for drinking water and other non-food exposure pathways.

PADEP applied the US EPA oral RfD for Mn and the MF of 3 to obtain an oral RfD for drinking water: $0.14 \text{ mg Mn/kg-day} \div 3 = 0.05 \text{ mg Mn/kg-day}$ ³ (PADEP, 2019a). PADEP then applied assumptions related to both water and non-water sources of exposure to the target chemical, daily water intake, body mass, and other factors (*e.g.*, fish consumption and the bioaccumulative potential of the target chemical) to derive the proposed Mn AWQC (US EPA, 2000, 2015; PADEP, 2019a).

More specifically, PADEP (2017, 2019a) assumed a relative source contribution of 0.2 (*i.e.*, 20% of orally bioavailable Mn can be attributed to drinking water with 80% coming from other non-water sources, such as diet),⁴ an average adult body weight of 80 kg, an average daily drinking water intake of 2.4 L, an average daily fish intake of 0.022 kg, and a bioaccumulation factor of 1 L/kg (*i.e.*, no bioaccumulation).

PADEP's (2019a) proposed Mn AWQC is calculated as follows:

$$0.05 \text{ mg Mn/kg-day} \times 0.2 \times (80 \text{ kg} \div [2.4 \text{ L/day} + (0.022 \text{ kg/day} \times 1 \text{ L/kg})]) = \mathbf{0.3 \text{ mg/L Mn}}$$

As described in the next section, recent Mn scientific information indicates that an MF of 3 is no longer needed for derivation of a health-protective drinking water concentration for Mn, indicating that the PADEP proposed Mn AWQC is not consistent with the best available scientific information for Mn.

² US EPA (2002) calculated a NOAEL of 0.14 mg/kg-day from a NOAEL of 10 mg/day by dividing 10 mg/day by 70 kg, the assumed adult body weight before later updates to exposure inputs (US EPA, 2015). No UFs were applied to the NOAEL; thus, the oral RfD for Mn is equal to the NOAEL of 0.14 mg Mn/kg-day.

³ Note the value is rounded.

⁴ US EPA (2000, 2015) noted that the relative source contribution accounts for other sources of exposure, with a value of 0.2 assumed if no data are available to quantify the relative sources of exposure.

3 Critique of Mn Regulatory Drinking Water Values

As discussed, based on the scientific information available at the time for Mn exposure and toxicity, US EPA's (2002) IRIS profile for Mn recommends an MF of 3 be applied to the oral RfD for exposure to Mn in drinking water. The reasons that US EPA included an MF of 3 are generally described (US EPA, 2002) as follows: (1) there was concern regarding possible increased uptake of Mn from drinking water as well as possible adverse health effects following lifetime consumption of 2 mg/L Mn in humans, and (2) there was concern that infants, particularly those fed formula made with water containing Mn, may be at increased risk due to increased Mn bioavailability. US EPA's lifetime health advisory for Mn in drinking water of 0.3 mg/L includes an MF of 3 to account for these concerns (US EPA, 2004). Similarly, the World Health Organization (WHO) also has established a health-based value for Mn of 0.4 mg/L, which includes an MF of 3 to account for possible increased Mn bioavailability from water (WHO, 2017). However, US EPA's (2002) reasoning for including an MF of 3 is now 17 years old and not consistent with the current science for Mn. Since US EPA's (2002) assessment, several important studies, as discussed below, have been published that assuage concerns related to possible increased bioavailability of Mn in drinking water. These studies also apply to WHO's (2017) health-based value and other state regulatory bodies that applied an MF of 3 for Mn drinking water values based on similar reasons of concern to those stated above.

US EPA's (2002) recommended application of an MF of 3 for Mn in drinking water is based, in part, on findings from a study by Kondakis *et al.* (1989). Specifically, Kondakis *et al.* (1989) observed that neurological effects were associated with exposure to approximately ≥ 2 mg/L Mn from well water in people over 50 years old; however, it should be noted that US EPA (2002) concluded it was "impossible to estimate the total oral intake of manganese in this study" due to "uncertainty in the amount of manganese in the diet and the amount of water consumed." Kondakis *et al.* (1989) is also limited by its study design. Specifically, Kondakis *et al.* (1989) measured Mn drinking water exposure concentrations and neurological effects only once rather than collecting regular measurements over months or years, resulting in considerable uncertainty in using the data to establish a causal relationship between Mn exposure and neurological effects in the study population. In addition, Kondakis *et al.* (1989) considered few confounding variables; thus, it is unclear whether neurological effects were due to Mn exposure or another variable, such as a pre-existing condition. Although US EPA (2002) concluded that it would be inappropriate to use the Kondakis *et al.* (1989) to derive a Mn oral RfD, US EPA chose to use Kondakis *et al.*'s (1989) findings as reason for concern. The US EPA's (2002) IRIS profile for Mn only describes three additional human Mn oral studies where toxic effects were observed. One study evaluated very high exposures to Mn in drinking water (14-28 mg/L Mn) in several individuals following contamination of the drinking water source from dry-cell batteries. Two case reports were described that resulted in high levels of Mn exposure *via* parenteral exposure in a 62-year-old male and another involving an individual with end-stage liver disease. Given the limitations in these studies, the data cannot be used to inform quantitative Mn risk assessment.

Since US EPA's Mn assessment in 2002, several community Mn drinking water studies have been conducted that have reported possible associations between Mn in drinking water and intellectual impairment in children (*e.g.*, Bouchard *et al.*, 2011; Khan *et al.*, 2011, 2012; Oulhote *et al.*, 2014). However, as described recently by the Agency for Toxic Substances and Disease Registry (ATSDR) (2012) and Health Canada (2019), these studies have many limitations, including cross-sectional study design (meaning the study evaluates one point in time and does not consider exposures over time) and limited to no exposure evaluation for individuals in the studies so that adverse effects cannot be attributed to Mn exposure. In addition, there is the potential in most of the studies for other unmeasured factors to influence the study outcome (*e.g.*, exposure to arsenic or other possible contaminants in the drinking water, or

caregiver IQ). PADEP describes some of these studies, among others, in its Rationale for development of the Mn AWQC (PADEP, 2019b). See Appendix A for our discussion and critique of the studies cited by PADEP (2019b).

Overall, based on the available studies there is no conclusive evidence to suggest that exposure to Mn in drinking water at 2 mg/L is associated with health effects. Similarly, there is also no conclusive evidence to suggest that Mn bioavailability differs between food or water ingestion. In fact, US EPA's own assessment (US EPA, 2002) includes discussion of an unpublished study by Ruoff (1995) that evaluated the relative bioavailability of Mn in food and water and found no significant differences. Although US EPA (2002) discussed possible increased Mn uptake in fasted individuals as a source of concern and additional basis for the MF of 3, there are no published studies that provide support for this concern.

As discussed below, the reasons for including an MF in deriving Mn water values are based on an outdated understanding of Mn toxicity and Mn bioavailability. The updated science suggests not only that the amount of Mn absorbed from food or water does not differ, but also that children are no more at risk from Mn exposure in water than adults. In this regard, the reasons for concern stated by US EPA (2002), WHO (2017), and state regulatory agencies relying on the same reasoning for the inclusion of an MF in Mn drinking water value derivations are not founded in the most up-to-date science and should be re-evaluated.

3.1 Updated Science on Mn Bioavailability from Drinking Water

A number of studies using physiologically based pharmacokinetic (PBPK) models have been published since the US EPA's (2002) IRIS assessment on Mn. These models are useful tools in understanding the relative importance of Mn exposure *via* inhalation and diet, as well as age differences in Mn absorption (Schroeter *et al.*, 2011; Yoon *et al.*, 2011). Recently, Song *et al.* (2018) developed a human PBPK model to evaluate Mn bioavailability from drinking water, and validated the model through simulating published datasets of human consumption of drinking water containing Mn, showing that modeled bioavailability of Mn from food and drinking water in humans is similar. Importantly, the Song *et al.* (2018) PBPK model assumed normal daily Mn intake of up to 10 mg/day Mn (*i.e.*, the NOAEL identified by PADEP, 2019a). For the average adult, this suggests that Mn up to 0.14 mg/kg-day is absorbed into the blood stream similarly, regardless of whether Mn exposure occurred from a food or water source.

In regard to age-related differences in Mn absorption, a recent PBPK study compared the relative importance of both dietary sources of Mn (*i.e.*, food *vs.* water) and age on modeled Mn brain concentrations (Yoon *et al.*, 2019). Specifically, Yoon *et al.* (2019) updated the Song *et al.* (2018) model for different age groups and showed that the impact of Mn in drinking water on Mn brain concentrations was similar for both children and adults at 1 mg/L when simultaneously simulating Mn exposure from food, water, and ambient air.⁵ Importantly, simulated Mn brain concentrations for all age groups were within the range of normal (Ramoju *et al.*, 2017) across all Mn drinking water concentrations evaluated, including 1 mg/L. Further, modeled Mn brain concentrations for formula-fed infants (prepared with Mn in drinking water up to 1 mg/L), who are considered to be the highest risk group, were similar to that of breastfed children across the range of Mn water concentrations measured. With regard to Mn bioavailability, Yoon *et al.*'s (2019) findings suggest that infants are at no greater risk compared to adults, for increased brain levels of Mn, even when considering formula feeding as a source of Mn exposure. The results of these recent PBPK studies are further discussed in Appendix A as part of our critique of PADEP's Rationale (PADEP, 2019b).

Overall, the most up-to-date science on Mn bioavailability discussed above suggests the human body absorbs Mn in food and Mn in water similarly. Further, updated science on Mn suggests that infants,

⁵ Note that the Yoon *et al.* (2019) model did not evaluate Mn drinking water concentrations higher than 1 mg/L.

whether breastfed or formula fed, are at no greater risk than adults in their capacity to absorb Mn. The results from these studies had not yet been published when US EPA delineated its concerns regarding Mn bioavailability in people as the rationale for including an MF of 3. In conclusion, application of an MF of 3 to the Mn oral RfD is not consistent with the best available science for Mn, should not be applied to derivation of a Mn drinking water value, and any current Mn drinking water value (*e.g.*, US EPA, 2004; WHO, 2017) that includes an MF of 3 should be revised.

4 Critique of PADEP's Proposed Ambient Water Quality Criterion for Mn

4.1 Critique of the Proposed PADEP Mn AWQC

As described in Section 3 above, based on the available studies, there is no conclusive evidence overall to suggest that exposure to Mn in drinking water at 2 mg/L is associated with health effects. Nevertheless, PADEP incorporates an MF of 3 in its calculation of the proposed Mn AWQC (PADEP, 2019a). However, the reasons for including an MF of 3 were based on an outdated understanding of the science. Also as described above, because Mn bioavailability from food and water is similar (Song *et al.*, 2018), applying an MF of 3 to account for differences in Mn bioavailability is no longer founded. Further, concern related to increased Mn bioavailability in high-risk populations, such as formula-fed and breastfed infants, is also alleviated in light of updated findings. Infants displayed similar modeled Mn brain concentrations resulting from ingestion of formula prepared with drinking water containing 1 mg/L Mn when compared to adults ingesting 1 mg/L Mn in drinking water (Yoon *et al.*, 2019). Therefore, application of an MF of 3 is not consistent with the best available science for Mn, and a Mn AWQC of 1 mg/L is protective for human consumption for all age groups under standard exposure assumptions, particularly where followed by conventional treatment.

Keeping all other assumptions the same (as described in Section 2.2), recalculation of the PADEP Mn AWQC without the MF of 3, *i.e.*, use of the Mn RfD of 0.14 mg/kg-day without adjustment, is as follows:

$$0.14 \text{ mg Mn/kg-day} \times 0.2 \times (80 \text{ kg} \div [2.4 \text{ L/day} + (0.022 \text{ kg/day} \times 1 \text{ L/kg})]) = \mathbf{925 \text{ } \mu\text{g/L Mn}}$$

A Mn AWQC of 925 $\mu\text{g Mn/L}$ is nearly equivalent to the current PADEP Mn AWQC of 1 mg/L Mn as well as the 1 mg/L Mn concentration found to be health-protective in the Yoon *et al.* (2019) PBPK analysis. Therefore, the current Mn AWQC of **1 mg/L** is protective for human consumption (and fish ingestion).⁶

As discussed in Section 3, US EPA (2002) and WHO (2017) have derived health-based drinking water values for Mn; however, these values were derived using an MF of 3. Removing the MF adjustment from US EPA's (2002) and WHO's (2017) drinking water values, consistent with the best available science for Mn, would result in values that are similar to the current criterion.

Therefore, the current Mn AWQC of 1 mg/L for an existing or planned surface potable water supply withdrawal is safe for human consumption under the standard regulatory agency specifications listed above (*i.e.*, assuming 2.4 L of water and 22 g of fish are consumed per day), to be met at least 99% of the time at the point of all existing or planned surface potable water supply withdrawals as required by Act 40.

⁶ Note that the fish ingestion pathway contributes minimally to the Mn AWQC value. The value is driven almost entirely by Mn in drinking water.

4.2 Mn Surface Water Value Protective for a Swimming and Fish-Ingestion Scenario

To provide perspective on other possible exposure pathways upstream of the surface water body, Gradient derived a Mn surface water value that is protective for non-drinking water exposures in the surface water body (*i.e.*, swimming and fish ingestion).

It is extremely unlikely that an individual would consume all of his or her drinking water from untreated surface water. However, a possible scenario could be one in which an individual regularly swims in a body of water upstream of the Mn AWQC point of compliance and ingests fish caught from the body of water. To this end, Gradient calculated a safe concentration of Mn in surface water for an adult and a child who (1) regularly swim in and (2) ingest fish caught from the water body.

As a conservative estimate, we assume that an adult or child would swim 3 days per week for 12 weeks during summer each year. We also assume that a child would gulp 50 mL/hour and an adult would gulp 21 mL/hour of water (up to 1 hour per day) on each day of swimming (US EPA, 2011). These are standard US EPA exposure assumptions. In accordance with US EPA (2019), we assume a default body weight of 80 kg and an exposure duration of 20 years for an adult. For a child aged 0 to 6 years, Gradient assumes a default body weight of 15 kg and an exposure duration of 6 years (US EPA, 2019). An oral Mn RfD of 0.071 mg/kg-day was used for non-dietary sources. In determining the oral RfD for non-dietary sources of Mn, US EPA (2019) recommends that the typical amount of Mn consumed in the diet (5 mg/day) be subtracted from 0.14 mg/kg-day, which results in a non-dietary oral RfD for Mn of 0.071 mg/kg-day. US EPA (2019) further applied an MF of 3 to this RfD for the same uncertainty discussed in Section 3, resulting in a non-dietary oral RfD for Mn of 0.024 mg/kg-day (US EPA, 2019). However, because US EPA's (2019) reasoning to include an MF of 3 is based on outdated science, as described in this report, Gradient did not apply an MF of 3 and instead retained the original non-dietary oral RfD for Mn of 0.071 mg/kg-day. We also assumed ingestion of fish from the water body, applying the same conservative calculation as applied to the AWQC described above, assuming the fish ingestion rate for a child of 0.011 kg/day (*i.e.*, 50% the fish ingestion rate for an adult) and the dietary Mn RfD of 0.14 mg/kg-day. (See Appendix B for a summary of exposure assumptions and risk calculations for the swimming and fish ingestion scenarios.)

Based on our conservative calculations (*i.e.*, likely overly health-protective) for swimming and fish ingestion only, we estimate that a Mn surface water concentration of 92 mg/L would be protective for an adult swimmer and fish consumer, and a concentration of 41 mg/L would be protective for a child swimmer and fish consumer. These surface water concentrations are much higher than what would be typical in the US. Based on data from the United States Geological Survey (USGS) National Water-Quality Assessment (NAWQA) database, the median Mn surface water concentration in the US is 0.016 mg/L, with 99th percentile concentrations ranging from 0.4 to 0.8 mg/L (ATSDR, 2012). Further, it is noteworthy that these Mn concentrations are much higher than what would be expected in surface water bodies upstream of the surface potable water supply withdrawal, particularly in streams which receive treated discharges from coal mining operations, because the federal technology-based effluent limitations for the Coal Mining Point Source Category (40 CFR Part 434) limit the concentration of Mn in the discharge to 2 mg/L on a monthly average (US EPA, 2018).

This assessment illustrates that even in an unlikely scenario in which an individual regularly swims in and ingests fish caught from water upstream of an existing or planned surface potable water supply withdrawal (*i.e.*, the Act 40 Mn AWQC point of compliance), the concentration of Mn in water could be 40-fold higher than 1 mg/L Mn and still pose no risk to human health.

5 Conclusion

PADEP proposes a Mn AWQC of 0.3 mg/L (PADEP, 2019a). It is overly conservative. This is due to PADEP's (2019a) decision to apply an MF of 3 because of concern related to potential increased Mn bioavailability from drinking water or in populations of interest (*i.e.*, formula-fed infants). However, these concerns are based on an outdated scientific understanding of Mn, and recent advances in the understanding of Mn bioavailability from drinking water suggest that the addition of an MF of 3 in deriving a Mn drinking water value is unnecessary. Not only does the most up-to-date science suggest that for all age groups, including formula-fed and breastfed infants, Mn bioavailability is similar, but also that at 1 mg/L Mn in drinking water results in modeled Mn brain concentrations for all age groups that are within the normal range (Song *et al.*, 2018; Yoon *et al.*, 2019). These studies are the most current peer-reviewed studies that address potential adverse effects in humans resulting from exposure to Mn in water, which is what PADEP specifically requested in its Proposed Rulemaking. In Appendix A, we provide a discussion and critique of the studies cited by PADEP in its Rationale for the proposed Mn AWQC (PADEP, 2019b).

Overall, the best available scientific information for Mn indicates that the current PADEP Mn AWQC of 1 mg/L is protective for human consumption. We also calculated a human health-protective Mn surface water value for potential non-drinking water exposures upstream of the public water supply withdrawal point. Even in a conservative swimming and fish-ingestion scenario, Gradient determined that a Mn surface water concentration upstream from the public water supply withdrawal point could be 41 mg/L (well above the Mn AWQC) and still pose no risk to human health.

Further, as PADEP has adopted US EPA's secondary maximum contaminant limit of 0.05 mg/L Mn in drinking water, the likelihood that any consumer would regularly drink water exceeding 1 mg/L is extremely small. Therefore, maintaining a Mn AWQC of 1 mg/L at the point of intake for an existing or planned potable water supply is protective for human consumption, particularly after conventional treatment.

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Appendix A

Critique of PADEP's Rationale for the Development of a Mn Human Health Criterion

A.1 Introduction

As basis for its proposed manganese (Mn) Ambient Water Quality Criterion (AWQC), the Pennsylvania Department of Environmental Protection (PADEP, 2019b) provided a rationale ("Rationale: Development of the Human Health Criterion for Manganese") in which it discusses its evaluation of the scientific data related to Mn and human health. Gradient provides a critique of the cited studies with particular focus on studies related to bioavailability and potential health effects of Mn. The overall evaluation and conclusions of PADEP (2019b) do not change Gradient's conclusions, as discussed in the main report.

PADEP cited additional studies in its Rationale,¹ which Gradient did not critique because the studies were determined to be review articles or not directly relevant to evaluating either oral Mn bioavailability or potential health effects associated with oral Mn exposure.

A.2 Studies of Mn Bioavailability

PADEP (2019b) discussed increased Mn absorption in infants (relative to adults) as a point of susceptibility to Mn. Specifically, PADEP (2019b) discussed immature liver Mn excretion, increased Mn absorption in the digestive system due to increased expression of DMT-1 proteins, increased water intake (per unit of body weight), increased permeability of the blood-brain barrier to Mn, and increased retention of Mn relative to adults as points of concern for infants. However, as discussed by Yoon *et al.* (2011, and as cited by PADEP, 2019b), enhanced uptake of Mn likely reflects developmental requirements for Mn. For example, Yoon *et al.* (2011) discussed that Mn present in bone and muscle is redistributed into other tissues during early postnatal development. Mechanisms unique to infants and that subside during later development, including enhanced expression of receptors that selectively bind lactoferrin-bound Mn (*i.e.*, Mn in breastmilk) and increased active transport of Mn across the blood-brain barrier, may explain observed findings that are interpreted as increased Mn absorption in infants (Yoon *et al.*, 2011). Importantly, these mechanisms related to Mn absorption and distribution are specialized processes for normal brain development during infancy (Yoon *et al.*, 2011). Under conditions in which high Mn content from food or formula milk are ingested, Yoon *et al.* (2011) noted that human infants have a fully developed biliary excretory pathway.

The considerations in Mn absorption, distribution, and elimination discussed above are important in interpreting studies cited by PADEP (2019b), including Chen *et al.* (2015), Crossgrove and Zheng (2004), Brown and Foos (2009), and O'Neal and Zheng (2015). In general, Chen *et al.* (2015), Crossgrove and Zheng (2004), and O'Neal and Zheng (2015) are reviews that primarily discussed studies of the characteristics of Mn absorption, distribution, and elimination, particularly when Mn exposure occurs *via* inhalation and in occupational settings, and generally did not focus on Mn exposure *via* ingestion. Characteristics of infant Mn absorption are rarely (if at all) discussed, with the exception of O'Neal and Zheng (2015), who cited three studies reporting higher intestinal Mn absorption, higher central nervous system levels of Mn, and higher serum levels of Mn in infants relative to adults. As discussed above, these reported findings very likely reflect a normal part of infant development and increased physiological requirement for Mn.

As discussed in Section 3.1, updated science on Mn suggests that infants, whether breastfed or formula fed, are at no greater risk compared to adults in their capacity to absorb Mn. Using physiologically based pharmacokinetic (PBPK) modeling and the observed results discussed above related to infant Mn absorption, distribution, and excretion, Yoon *et al.* (2011) estimated similar and lower internal Mn levels

¹ Examples of other studies cited by PADEP in its Rationale that are not discussed in our critique: Aschner (2000), Holley *et al.* (2011), Erikson *et al.* (2007), Cordova *et al.* (2013), Lidsky *et al.* (2007), Santamaria (2008), Bouabid *et al.* (2016), Lanphear *et al.* (2015), Grandjean and Landrigan (2014), and Finley and Davis (1999).

in the infant brain compared to the adult brain for various air and diet Mn exposure scenarios in humans. As discussed in Section 3.1, Yoon *et al.* (2019) also used an updated model for different age groups and showed that the impact of Mn in drinking water on Mn brain concentrations was similar for both children and adults at 1 mg/L when simultaneously simulating Mn exposure from food, water, and ambient air.² Further, modeled Mn brain concentrations for formula-fed infants (prepared with Mn in drinking water up to 1 mg/L) were similar to that of breastfed children across the range of Mn water concentrations measured and were within the range of normal Mn brain concentrations. Results from these PBPK modeling studies, which incorporated observed differences in Mn exposure and absorption parameters from infants to adults, suggest infants are not more susceptible than adults to health effects following oral Mn exposure from drinking water. PADEP did not consider Yoon *et al.* (2019) or other PBPK studies of Mn bioavailability.

It is important to note that the factors that affect Mn absorption *via* inhalation are different from the factors that affect Mn absorption *via* oral drinking water. Therefore, Mn inhalation studies are not directly relevant to the Mn oral drinking water pathway. However, since PADEP (2019b) discussed the Mn inhalation pathway in its Rationale, we have critiqued that discussion here. PADEP stated that "most Mn intoxication cases have been associated with occupational exposure" and then stated that "[t]he increased level of toxicity associated with this exposure pathway is not unexpected since inhaled manganese has a direct pathway to the brain *via* the olfactory nerve (O'Neal 2015)" (PADEP, 2019b, p. 7). It is very important to point out that studies have suggested that only very small Mn particles (ultrafine, 0.2 microns or smaller) can be transported from the nasal cavity across the olfactory tract to the brain (Elder *et al.*, 2006), and these studies have all been conducted in nonhuman mammals. Further, PADEP stated that "[t]he human body tightly regulates the amount of *ingested* manganese that enters the circulatory system via intestinal absorption" (PADEP, 2019b, p. 7), suggesting the inhaled Mn is not regulated *via* homeostatic mechanisms. PADEP further stated that "the body will typically absorb 100% of the inhaled manganese" (PADEP, 2019b, p. 7). This statement is incorrect and not consistent with what is known about particle transport and deposition within the respiratory tract (including for Mn). Only respirable particles (2.5 microns or smaller) can reach the deep parts of the lung where they can be potentially absorbed into the bloodstream. Larger particles (>10 microns in diameter) become trapped in the upper respiratory tract where they are then typically removed *via* mucociliary escalation through coughing and sneezing and sometimes swallowed. Therefore, the statement that 100% of inhaled Mn is absorbed is not consistent with the current science regarding particle transport for Mn across the olfactory tract or within the respiratory tract. Further, as has been described through development of several Mn PBPK models (Schroeter, 2011; Yoon *et al.*, 2011, 2019), inhaled Mn (at typical human exposure concentrations), once absorbed into the blood, is regulated *via* homeostatic mechanisms.

A.3 Mn Epidemiology Studies

Overall, the epidemiology studies discussed by PADEP (2019b) as evidence of human health effects of Mn are generally limited by a number of factors. These limitations have been acknowledged by other health agencies (ATSDR, 2012; Health Canada, 2019) and include (1) cross-sectional study design (meaning the study evaluates one point in time and does not consider exposures over time), (2) limited exposure evaluation for individuals in the studies such that adverse effects cannot be attributed to oral Mn exposure, (3) potential for other unmeasured factors to influence the study outcome (*e.g.*, exposure to other possible contaminants in the drinking water, caregiver IQ, or socioeconomic status [SES]), (4) participant selection methods that may lead to a biased sample, and (5) in some cases, lack of a dose-response relationship³

² Note that the Yoon *et al.* (2019) model did not evaluate Mn drinking water concentrations higher than 1 mg/L. There is no indication that PADEP was aware of or considered Yoon *et al.* (2019) when it developed the Rationale or the Proposed Rulemaking.

³ A dose-response relationship is present when an outcome (*e.g.*, IQ scores) changes in an orderly fashion as the concentration of a chemical (*e.g.*, Mn) increases. The absence of a dose-response relationship suggests that the outcome may not be associated with the chemical of interest.

between Mn and the health endpoint of interest; however, it should be noted that the studies that did report a dose-response relationship were limited by several of the issues discussed above. The presence of any one or more of the limitations listed above in a given study introduces uncertainty into any reported health effects, making it difficult to attribute any effect to Mn exposure. Indeed, in its discussion of the potential susceptibility of children to Mn, the Agency for Toxic Substances and Disease Registry (ATSDR, 2012) concluded that these uncertainties "preclude the characterization of causal and dose-response relationships between the observed [neurological] effects and manganese exposure." PADEP did not reference these uncertainties and did not cite Health Canada (2019).

Despite the limitations discussed above, PADEP concluded that the "available epidemiological data on children suggests that exposure to elevated manganese levels may result in a variety of neurological and developmental deficits" (PADEP, 2019b, p. 6). PADEP (2019b) cited as evidence for its conclusions Henn *et al.* (2010), Khan *et al.* (2012), Kim *et al.* (2009), and Menezes-Filho *et al.* (2011), as well as other studies, which are briefly summarized in the Rationale document. It should be noted that although PADEP (2019b) does not discuss them at length, Henn *et al.* (2010), Khan *et al.* (2012), Kim *et al.* (2009), and Menezes-Filho *et al.* (2011) possessed similar limitations as those discussed above, which limit their ability to establish a causal association between oral Mn exposure and potential neurological effects. These include cross-sectional designs, lack of consideration for important confounders related to cognition (*e.g.*, parental IQ, quality of the home environment⁴, and SES), lack of information or relevance of the Mn exposure pathway, potential bias in participant selection, and co-exposures to other chemicals (*e.g.*, lead). It should also be noted that many of the studies reported blood Mn concentrations within normal levels: 4-15 µg/L (ATSDR, 2012). Critiques for individual studies discussed at length as cited by PADEP (2019b) are provided below.

PADEP referred to a review article by Smith *et al.* (2017) on the biochemistry of Mn, including its interaction with cells, enzymes, and other proteins in the human body. In a discussion of the role of Mn in dietary nutrition, Smith *et al.* (2017) referred to the Institute of Medicine's (IOM, 2001) Adequate Intake Level for adult women and men (*i.e.*, 1.8 and 2.3 mg/day, respectively) and Tolerable Upper Intake Level for Mn (*i.e.*, 11 mg/day).⁵ Smith *et al.* (2017) also referred to a no-observed-adverse-effect level (NOAEL) of 11 mg/day and a lowest-observed-adverse-effect level (LOAEL) of 15 mg/day, as identified by IOM (2001). However, the LOAEL reported by IOM is based on changes in serum Mn and lymphocyte Mn superoxide dismutase (MnSOD) levels, which are not adverse neurological effects, following 119 days of Mn supplementation (Davis and Greger, 1992). The serum level changes are likely more consistent with a marker of Mn exposure and homeostatic regulation of Mn in the body, consistent with no discussion of this endpoint as an adverse effect by ATSDR (2012) or the United States Environmental Protection Agency (US EPA, 2002). In fact, the study that IOM cites as the basis for the Mn serum and lymphocyte MnSOD level changes (Davis and Greger, 1992) reported Mn serum levels⁶ that are all generally within the normal range: <2.15 µg/L (Mayo Clinic Laboratories, 2020) or approximately 0.4-0.85 µg/L (ATSDR, 2012). In addition, Davis and Greger (1992) did not suggest that the Mn levels in serum are adverse and in fact evaluated Mn deficiency and effects of supplementation. The authors stated that "the fairly small response of lymphocyte MnSOD activity to manganese supplementation and the long time required for significant

⁴ Quality of the home environment is a measure of childhood cognitive stimulation and support as provided by caregivers in the home (Bradley *et al.*, 1989). Typically, quality of the home environment is assessed by an interview with caretakers and comprises subscales, such as parental responsiveness, child acceptance, organization of the environment, play materials, parental involvement, and variety of stimulation (Bradley *et al.*, 1989).

⁵ Note that when converted to mg/kg-day (assuming a 70 kg adult body mass), the Adequate Intake Level for women and men is 0.026 and 0.033 mg/kg-day, respectively. Further, the Tolerable Upper Intake Level is 0.16 mg/kg-day. US EPA's oral RfD for Mn is equal to 0.14 mg/kg-day Mn (US EPA, 2002).

⁶ Davis and Greger (1992) reported that serum Mn under baseline in women ranged between 15.3 to 19.3 nmol/L Mn, which is equivalent to 0.84 to 1.06 µg/L Mn. The authors also reported that serum Mn increased between 4 to 6.5 nmol/L Mn (*i.e.*, 0.22 to 0.36 µg/L Mn) after 119 days of Mn supplementation. Therefore, serum Mn levels after supplementation were calculated to be approximately 19.3 to 25.8 nmol/L Mn (*i.e.*, 1.06 to 1.42 µg/L Mn).

changes (89d) in this study suggest that manganese intake of these healthy young women approximated their requirements." Therefore, the study actually suggests that 15 mg/day Mn is a healthy intake level in women. IOM (2001) also stated that higher levels (20 mg/day) of Mn are present in vegetarian diets.

Therefore, there is no scientific basis for suggesting that 15 mg/day Mn would lead to adverse health effects in humans. As such, Smith *et al.* (2017) incorrectly stated that "such a narrow dose range between inadequate and excess intake...and only 5% oral absorption, small variation in absorption (to 2.5% or 10%) could substantially change the body burden." PADEP makes similar statements on pages 7 and 11 of its Rationale: *e.g.*, "[s]uch a narrow dose range exists between inadequate and excess intake that small variations in the body's absorption and handling of manganese could substantially change the body burden" (PADEP, 2019b, p. 11). Both Smith *et al.*'s and PADEP's statements are incorrect and not based on the available scientific evidence for safe levels of Mn intake. In addition, ATSDR (2012) stated that the average amount of Mn absorbed across the gastrointestinal tract in humans is on average 3 to 5%, and not 10%. In conclusion, PADEP should not consider the LOAEL of 15 mg/day Mn that was discussed by Smith *et al.* (2017) and IOM (2001) as reflecting an adverse effect.

Chung *et al.* (2015) evaluated the association between maternal blood Mn (mean: 22.5 µg/L) and mental and psychomotor development scores in 232 pairs of pregnant women and six-month-old infants in South Korea. Although the authors controlled for a number of potential confounding variables (*e.g.*, maternal age, monthly income, and birth weight), the authors did not control for other relevant confounders such as smoking, maternal intelligence, or quality of the home environment in their analyses. The authors found that blood Mn was associated with covariate-adjusted psychomotor development scores but was not associated with mental development scores. In addition, a dose-response relationship was not observed between blood Mn and psychomotor development scores. The authors did not measure oral sources of Mn exposure. Because of potential uncontrolled confounding, a lack of a dose-response relationship between Mn and development scores, and inadequate information on oral Mn exposure, this study cannot be used to draw conclusions regarding water Mn and cognitive endpoints.

Brown and Foos (2009) conducted a case study using hypothetical Mn exposure scenarios in children. The authors summarized research reporting that Mn levels in diluted powder-based infant formulas ranged from 34 to 169 µg/L Mn, with a median Mn concentration of 101 µg/L.⁷ PADEP (2019b) cited this paper in relation to the Mn absorption of formula-fed infants; however, Brown and Foos (2009) did not report data on Mn absorption. Therefore, this study should not be used to determine the bioavailability of Mn in infants.

Bouchard *et al.* (2007) conducted a cross-sectional study of associations between hair Mn (mean: 5.1 µg/g, range: 0.3 to 20 µg/g) and oppositional/hyperactive behavior in 46 Canadian children aged 6-15 years. Parents and teachers reported on children's behaviors using the Revised Conners' Rating Scale. Although the authors adjusted for some potential confounders (*i.e.*, age, sex, and income), the authors did not control for smoking, maternal intelligence, or quality of the home environment in their analyses. Children's hair Mn was associated with higher covariate-adjusted oppositional and hyperactivity scores (*i.e.*, indicating increased oppositional and hyperactivity behaviors) as reported by teachers. However, hair Mn was not associated with behavior scores as reported by parents. Given the difference between reporting by teachers and parents, lack of adjustment for potential confounders, and cross-sectional study design, the observed changes in behavior scores cannot be attributed to Mn. Health Canada (2019) did not include this study in its evaluation of Mn.

⁷ Brown and Foos (2009) reported Mn levels in formula as µg/5 fluid ounces Mn. Concentrations were converted to µg/L using 0.148 L = 5 fluid ounces.

Bouchard *et al.* (2011) conducted a cross-sectional evaluation of associations between Mn as measured in hair (median: 0.7 µg/g, range: 0.1 to 21 µg/g) and home tap water and IQ scores in 362 Canadian children aged 6-13 years. The authors reported that children's hair Mn was associated with estimated Mn intake from water consumption but not associated with estimated Mn intake from food. When the authors adjusted for a number of potential confounders (*e.g.*, maternal education, family income, alcohol and tobacco consumption during pregnancy, and quality of the home environment), hair Mn and estimated Mn intake from water (median: 8 µg/kg/month, range: 0 to 945 µg/kg/month) was inversely associated with full-scale IQ. However, approximately 33% of children in the study did not drink the tap water, suggesting that they were not exposed to Mn in the drinking water. In addition, there was no dose-response relationship between full-scale IQ and hair Mn or estimated Mn intake from water. Therefore, given the cross-sectional study design, lack of a dose-response relationship, and lack of a complete exposure pathway in 1/3 of the samples, the study provides limited evidence of an association between Mn in drinking water and adverse cognitive effects. ATSDR (2012) and Health Canada (2019) also discussed uncertainties of Bouchard *et al.* (2007, 2011), including that it was uncertain whether the observed effects were due to Mn or other drinking water or dietary components, that there was a lack of information about Mn levels in food and air, and that the study used a small sample size.

In a follow-up study of the same cohort of children, Oulhote *et al.* (2014)⁸ conducted a cross-sectional evaluation of associations between Mn (in hair and tap water) and memory, attention, motor function, and hyperactivity scores. The authors did not evaluate correlations between Mn intake and hair Mn. After adjusting for potential confounding variables (*e.g.*, maternal education and intelligence, family income, maternal tobacco and alcohol consumption during pregnancy, and tap water lead concentrations), the authors reported that children's hair Mn (mean: 1.4 µg/g, range: 0.1 to 20.7 µg/g) was associated with lower memory and attention scores but was not associated with motor function or hyperactivity scores. However, estimated Mn intake from water (mean geometric mean: 5.5 µg/kg/month, range: 0 to 1,059 µg/kg/month) was associated with reduced motor function but not memory, attention, or hyperactivity scores. The authors did not examine the quality of the home environment as a potential confounder. Because of the inconsistent associations reported between Mn and cognitive and behavioral function, cross-sectional study design, and lack of consideration for quality of the home environment as a potential confounder, this study provides limited evidence of an association between Mn and cognitive and behavioral outcomes. Health Canada (2019) discussed the uncertainties of both Bouchard *et al.* (2011) and Oulhote *et al.* (2014) and stated that the "risk of bias in these studies cannot be discarded." Specifically, Health Canada (2019) noted that few details on sample recruitment and retention were reported and that exposure misclassification was possible because: (1) water Mn was measured only once, (2) residing in one's current home for 3 months was sufficient for inclusion in the study, (3) hair Mn was used as a biomarker, (4) no information was reported regarding Mn in diet or soil, (5) no information was provided on the timing or duration of exposure during critical periods of development, (6) the authors did not report whether the investigators were blind to the exposure levels of the participants, and (7) the statistical error associated with effect estimates were large and borderline statistical significance was reported for many observed effects.

Haynes *et al.* (2015) conducted a cross-sectional evaluation of associations between blood (mean: 9.67 µg/L) and hair Mn (mean: 417 ng/g) and IQ in 404 children living in Ohio, aged 7-9 years. The Mn blood concentrations reported were within the normal range of 4-15 µg/L (ATSDR, 2012), and the authors found that blood Mn was not associated with hair Mn. The authors used a voluntary method of participant selection, which likely introduced sampling bias. Although the authors controlled for a number of potential

⁸ PADEP (2019b) cited Oulhote, Y., *et al.* (2014). "Age-Dependent Susceptibility to Manganese-Induced Neurological Dysfunction." *Environ. Health Perspect.* 122(12):1343-1350. However, this reference could not be identified. Based on the description of the study (PADEP, 2019b) and the cited journal volume and page numbers, Oulhote *et al.* (2014), titled "Neurobehavioral Function in School-Age Children Exposed to Manganese in Drinking Water," was identified as the referenced article.

confounders (*e.g.*, caregiver IQ, SES, serum cotinine [a marker for smoking], and blood lead), the authors did not measure the quality of the home environment. Haynes *et al.* (2015) reported that the highest quartile of hair Mn (*i.e.*, >747 ng/g) and blood Mn (*i.e.*, >11.2 µg/L) was associated with lower full-scale IQ scores. However, the authors reported a non-linear relationship between these biomarkers of Mn and full-scale IQ scores. In addition, the authors reported an association between serum cotinine (a marker for exposure to tobacco smoke) and child cognitive function. Because Haynes *et al.* (2015) did not evaluate exposure to Mn in drinking water, used a cross-sectional study design, did not consider quality of the home environment as a potential confounder, observed a non-linear relationship between Mn and cognition, and reported an association between serum cotinine and cognitive function, this study cannot be used to draw conclusions regarding water Mn and cognitive endpoints.

Khan *et al.* (2011) conducted a cross-sectional study of 201 Bengali children (aged 8-11 years) and examined associations between Mn and arsenic (in water and blood) and classroom behavior. Although the authors adjusted for confounding variables (*e.g.*, maternal education, sex, and body mass index), they did not adjust for the quality of the home environment, SES, or smoking. The authors found that water Mn (median: 650 µg/L, range: 40-3,443 µg/L) was associated with covariate-adjusted negative classroom behavior scores. Blood Mn (median: 14.6 µg/L, range: 6.3-33.9 µg/L) was not associated with classroom behavior. The median Mn blood concentration reported is within the normal range of 4-15 µg/L (ATSDR, 2012). The results of Khan *et al.* (2011) are limited by the cross-sectional study design, reliance on teacher-reported scores (which may introduce measurement error), and potential sampling bias (*i.e.*, children with lower water Mn were excluded due to their distance from the study region), which were also acknowledged as limitations by Khan *et al.* (2011). Because of these limitations, as well as not accounting for other confounders (listed above), including exposure to other chemicals in the water (*i.e.*, arsenic), this study cannot be used to draw conclusions regarding water Mn and behavioral endpoints.

Wasserman *et al.* (2006) examined cross-sectional associations between Mn and arsenic (in water and blood) and IQ scores in 142 Bengali children aged 10 years. After adjusting for confounder variables, such as maternal intelligence and house type (as a surrogate for SES), water Mn (mean: 795 µg/L) was associated with lower IQ scores. However, blood Mn (mean: 12.8 µg/L) was not associated with IQ scores. The Mn blood concentrations reported are within the normal range of 4-15 µg/L (ATSDR, 2012). Further, the authors reported that water Mn was not associated with blood Mn in a subset of 95 children. The authors did not control for other confounding variables, such as the quality of the home environment or smoking. Further, the authors also did not measure Mn in food, which could introduce exposure measurement error. ATSDR (2012) discussed uncertainties of Wasserman *et al.* (2006), including that it was uncertain whether the observed effects were due to Mn or other drinking water or dietary components, there was a lack of information about Mn levels in food and air, and the study used a small sample size. Because of the cross-sectional study design, inadequate Mn exposure assessment, small sample size, and inadequate control of confounding variables, this study cannot be used to draw conclusions regarding water Mn and cognition.

A.4 Mn Rodent Studies

In addition to the epidemiology studies discussed above, PADEP (2019b) relied on several Mn rodent studies in its Rationale (Kern *et al.*, 2010; Beaudin *et al.*, 2013; Moreno *et al.*, 2009). Overall, these studies cannot be used to draw conclusions regarding water Mn and potential health effects in humans due to a number of limitations. First, all three studies used concentrations of Mn in water that would be rarely encountered in humans. In general, the Mn exposure regimen used by these studies is associated with blood (or serum) Mn levels that are substantially higher than reported Mn levels in humans, which limits generalizability of the reported results to humans. Second, there is a lack of information about how rodent Mn requirements compare to human Mn requirements. Because of the essentiality of Mn, Mn dietary requirements in rodents would need to be considered in order to understand what doses were actually in

excess of the dietary requirements and of potential relevance to humans. Third, the authors often reported inconsistent behavioral findings. Specifically, in some cases, the authors would report that an endpoint (*e.g.*, fear behavior) was affected by Mn exposure using one procedure, but another procedure designed to assess the same or similar endpoint did not show an effect of Mn exposure. Fourth, in some cases, the authors did not observe a dose-response relationship between Mn exposure and behavior. This suggests that observed changes in behavior were due to a factor other than Mn. Finally, all three studies used only two doses of Mn, which limits their ability to establish dose-response relationships between Mn and behavior.

The first two limitations discussed above are important to keep in mind because the doses evaluated in the rodents in these studies are much higher than what is typical in humans. Across all three studies, the doses ranged from 4.4 to 50 mg/kg-day, compared to the upper limit (UL) in humans of 0.14 mg/kg-day calculated by US EPA (2002), based on upper intake Mn levels in the diet (10 mg/day). Further, since Mn is an essential nutrient, the oral reference dose (RfD) for Mn is applied based on the assumption that 50% of the intake would come from food (5 mg/day, which is a typical intake in adult humans) and that any additional Mn exposure should not result in exceedance of a total Mn intake of 10 mg/day (US EPA, 2019). However, there is a lack of information on rodent dietary requirements for Mn. Thus, the doses in these studies may be very high compared to normal dietary requirements in the rodents, and the effects reported may reflect dosing that is much higher than what would be expected in humans. Application of these rodent studies to derive an oral Mn toxicity value for humans would be highly uncertain.

ATSDR (2012) reached the same conclusion based on its review of the animal studies, stating, "However, inconsistencies in the dose-response relationship information across studies evaluating different neurological end points under different experimental conditions in different species, as well as a lack of information concerning all intakes of manganese (*e.g.*, dietary intakes plus administered doses), make it difficult to derive intermediate- or chronic-duration MRLs using standard MRL derivation methodology from the animal studies." ATSDR (2012) provided an interim guideline of 0.16 mg/kg-day based on a tolerable upper intake level of 11 mg/day in humans (similar to the US EPA [2002] approach for a Mn oral RfD).

Kern *et al.* (2010) conducted a study in neonatal rats to examine the effects of oral administration (*p.o. via* micropipette) of Mn (0, 25, and 50 mg/kg-day) on behavior and levels of brain dopamine. The authors stated that pre-weaning control rats consume approximately 70 µg/kg/day Mn from breast milk, which is approximately 100 times higher than normal human infant Mn intake from breast milk. The authors tested behavior in a number of procedures including an open arena, elevated plus maze, and radial arm maze. Kern *et al.* (2010) reported blood Mn concentrations of approximately 60, 150, and 210 µg/L for the control, 25, and 50 mg/kg-day groups, respectively. The authors reported that these procedures reflect fear and anxiety (open arena and elevated plus maze) and learning and memory (radial arm maze). Mn exposure dose-dependently increased fear behavior on the elevated plus maze but did not affect fear behavior in the open arena. The authors also observed that Mn exposure dose-dependently increased total memory errors upon initial testing, but after repeated testing all exposure groups demonstrated similar memory performance. This study is limited because only two doses of Mn were used, which is insufficient to generate a full dose-response curve. Further, this study is limited because the authors reported inconsistent effects on fear behavior. These inconsistent results, coupled with the reported findings that memory deficits recovered, make it uncertain whether behavior changes were due to Mn exposure in water. Finally, given that these doses are much higher than what humans would typically be exposed to in the diet, and that there is uncertainty with respect to how these doses compare to dietary requirements of Mn in rodents, the findings from Kern *et al.* (2010) cannot be extrapolated to humans.

Beaudin *et al.* (2013) studied neonatal rats to determine the effects of early-life and continuous exposure to oral Mn (0, 25, and 50 mg/kg-day) on sensorimotor performance. For early-life exposure, rats were administered Mn *via* oral gavage for the first 22 days after birth, which corresponded to blood Mn concentrations of 23.6, 186, and 267 µg/L in the 0, 25, and 50 mg/kg-day groups (respectively). For continuous exposure, rats were administered Mn *via* drinking water from birth to approximately 400 days of age, which corresponded to blood Mn levels of 5.81, 9.7, and 13.7 µg/L in the 0, 25, and 50 mg/kg-day groups (respectively). Behavioral testing began at 120 days of age for all groups. Using a procedure that required the rats to navigate a staircase and gather food pellets (the "staircase test"), the authors found that early-life exposure to 50 mg/kg-day Mn impaired fine motor control. Further, the authors observed that 25 mg/kg-day Mn delivered continuously impaired fine motor control but 50 mg/kg-day Mn did not impair behavior. This study is limited because it used only two doses of Mn, which is insufficient to generate a full dose-response curve. Further, several inconsistencies related to the exposure regimen and blood Mn concentrations introduced uncertainty in interpretation of the results. These include (1) using higher Mn water concentrations than what would be typically encountered in humans and (2) blood Mn concentrations were over 10 times lower in the rats who received Mn continuously relative to those who received Mn for 22 days in early life. This suggests a potential species difference between rats and humans that limits the generalizability of Beaudin *et al.*'s (2013) findings. Because of uncertainty due to the use of water Mn concentrations much higher than would be typically encountered in humans and a lack of information about rodent Mn requirements, this study should not be extrapolated to humans.

Moreno *et al.* (2009)⁹ conducted an experiment in C57BL/6 mice to determine the effects of juvenile and adult Mn exposure on motor behavior and neurotransmitter levels. Mice were administered Mn *via* oral gavage (0, 4.4, and 13.1 mg/kg-day¹⁰) as juveniles, adults, or as both. The authors reported serum Mn levels of 0.2-0.35 ppm (equivalent to 200-350 µg/L Mn). Motor ability was assessed using an open-field test similar to Kern *et al.* (2010). The authors reported that female mice behavior was not affected by Mn exposure as juveniles, adults, or both. Male mice who received 10 and 30 mg/kg-day Mn as juveniles spent less time on the periphery of the open field (*i.e.*, showed less fear/anxiety behavior), whereas male mice who received 10 and 30 mg/kg-day Mn as both juveniles and adults spent more time on the periphery (*i.e.*, show more fear/anxiety behavior). Mn exposure did not affect movement time in any group, with the exception of male mice receiving 30 mg/kg Mn as juveniles and adults, who displayed fewer movements per minute. Brain and serum Mn levels often did not display a dose-response relationship, such that Mn levels in controls were sometimes higher than Mn levels in Mn-exposed mice. Overall, this study is limited by (1) its use of high doses of Mn, which would not be typically encountered in humans, and (2) inconsistent behavioral findings in that Mn exposure was associated with both increased and decreased fear and anxiety behavior in male mice. These limitations, including a lack of information about rodent Mn requirements, prevent using this study to draw conclusions about Mn in water and potential health effects in humans.

A.5 Conclusions

Overall, PADEP's (2019b) Rationale was based on outdated science that possesses several limitations. These limitations preclude the use of these studies in making conclusions regarding Mn in water and potential health effects in humans. Similarly, PADEP (2019b) did not rely on the most up-to-date science regarding Mn bioavailability in breastfed and formula-fed infants (Yoon *et al.*, 2019), which demonstrates that these subpopulations are not at an increased risk of health effects from Mn following 1 mg/L Mn in drinking water. Therefore, the results from the studies discussed in PADEP's (2019b) Rationale do not

⁹ PADEP (2019b) cited Moreno, J.A., *et al.* (2009). "Neurobehavioral Function in School-Age Children Exposed to Manganese in Drinking Water." *Toxicol. Sc.* 112(2):394-404. However, this reference could not be identified. Based on the description of the study (PADEP, 2019b) and the cited journal volume and page numbers, Moreno *et al.* (2009), titled "Age-Dependent Susceptibility to Manganese-Induced Neurological Dysfunction," was identified as the referenced article.

¹⁰ Moreno *et al.* (2009) administered 10 and 30 mg/kg-day MnCl₂, which is equivalent to 4.4 and 13.1 mg/kg-day Mn.

change Gradient's conclusion that a Mn AWQC of 1 mg/L, which is based on the best available scientific information for Mn, is protective for human consumption.

Appendix B

Exposure Assumptions and Calculations for a Swimming and Fish-Ingestion Surface Water Value

Table B.1 Exposure Assumptions and Calculations for a Swimming and Fish-Ingestion Surface Water Value

Swimming and Fish-Ingestion Surface Water Value		=	$\frac{1}{SW_{(ing)}} + \frac{1}{SW_{(derm)}} + \frac{1}{SW_{(fish)}}$			=	41 Child	92 Adult	mg/L Mn
$SW_{(ing)} = \frac{THQ * RfD}{IF}$		=	216 Child	2,742 Adult	mg/L Mn				
$SW_{(derm)} = \frac{THQ * RfD}{IF * Kp \text{ (cm/hour)}}$		=	68 Child	117 Adult	mg/L Mn				
$SW_{(fish)} = \frac{THQ * RfD}{IF}$		=	191 Child	509 Adult	mg/L Mn				
Target Hazard Quotient (THQ) =		1							
Oral Non-diet Mn RfD (mg/kg-day) =		0.071							
GIAbs =		0.04							
Dermal Mn RfD (mg/kg-day) =		Oral Non-diet Mn RfD * GIAbs =	0.00284						
Oral Diet Mn RfD (mg/kg-day) =		0.14							
Kp (cm/hour) =		0.001							

Surface Water – Ingestion						
Intake Factor (IF) = $\frac{IR \times EV \times EF \times ED \times CF}{BW \times AT}$		=	3.3E-04 Child	2.6E-05 Adult	Basis	
IR	Incidental Ingestion Rate (mL/hour)		50	21	Mean ingestion rate value (US EPA, 2011)	
EV	Event Frequency (hours/day)		1	1	Professional judgment	
EF	Surface Water Exposure Frequency (days/year)		36	36	Professional judgment	
ED	Exposure Duration (years)		6	20	Default value for Resident (US EPA, 2019)	
CF	Conversion Factor (L/mL)		0.001	0.001		
BW	Body Weight (kg)		15	80	Default value for Resident (US EPA, 2019)	
AT	Averaging Time (days)		2,190	7,300		

Surface Water – Dermal Contact (assumes Mn is 100% soluble)						
Intake Factor (IF) = $\frac{SA \times ET \times EF \times ED \times CF}{BW \times AT}$		=	4.2E-02 Child	2.4E-02 Adult	Basis	
SA	Surface Area Exposed to Surface Water (cm²)		6,365	19,652	Default value for Resident (US EPA, 2014)	
ET	Exposure Time (hours/day)		1	1	Professional judgment	
EF	Surface Water Exposure Frequency (days/year)		36	36	Professional judgment	
ED	Exposure Duration (years)		6	20	Default value for Resident (US EPA, 2019)	
CF	Conversion Factor (L/cm³)		0.001	0.001		
BW	Body Weight (kg)		15	80	Default value for Resident (US EPA, 2019)	
AT	Averaging Time (days)		2,190	7,300		

Fish – Ingestion						
Intake Factor (IF) = $\frac{IR \times EF \times ED \times BAF}{BW \times AT}$		=	7.3E-04 Child	2.8E-04 Adult	Basis	
IR	Ingestion Rate (kg/day)		0.011	0.022	Default value for adult Resident (PADEP, 2019a); 50% of default value for child Resident	
EF	Ingestion Exposure Frequency (days/year)		365	365	Default value for Resident (PADEP, 2019a)	
ED	Exposure Duration (years)		6	20	Default value for Resident (US EPA, 2019)	
BAF	Bioaccumulation Factor (L/kg)		1	1	Default value for fish (PADEP, 2019a)	
BW	Body Weight (kg)		15	80	Default value for Resident (US EPA, 2019)	
AT	Averaging Time (days)		2,190	7,300		

Notes:

derm = Dermal; GIAbs = Gastrointestinal Absorption Factor; ing = Ingestion; Kp = Dermal Permeability Coefficient; Mn = Manganese; RfD = Reference Dose; SW = Surface Water.

Exhibit C

**Review of Manganese Issue
As It Relates to Surface Water Quality**

Tetra Tech, Inc.

August 11, 2020

REVIEW OF MANGANESE ISSUE AS IT RELATES TO SURFACE WATER QUALITY

PRESENTED TO

Pennsylvania Coal Alliance

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ES EXECUTIVE SUMMARY

The regulation of manganese in treated mine waters began with the 1972 Clean Water Act (CWA) and the specific requirements to regulate industry point source discharges using technology-based treatment. Based on these requirements, the U.S. Environmental Protection Agency (EPA) developed effluent limitation guidelines for various industry categories including the coal mining industry. In 1985, manganese best available technology (BAT) effluent limits for the coal mining industry were established at a 30-day average concentration of 2.0 mg/L and maximum daily concentration of 4.0 mg/L.

This evaluation of the EQB proposed 0.3 mg/L total manganese water quality standard examined various aspects of the proposed rulemaking. This included evaluation of:

- Sources of dissolved and particulate (total) manganese to surface waters.
- Fate & transport of dissolved manganese in surface waters.
- Cost implications on mine water treatment.
- Implications on treatment and treatment costs at downstream potable water treatment systems.
- Toxicity of manganese to aquatic life toxicity.

In surface waters, manganese can be found in both dissolved (Mn^{2+}) and particulate (Mn^{3+} and Mn^{4+}) forms that combined represent total manganese. Anthropogenic dissolved manganese sources in surface waters may include coal mining activities from chemical leaching/weathering associated with pyrite oxidation. This has been the primary focus of past discharge regulation of effluent manganese to surface waters. However, anthropogenic particulate manganese in surface water may be contributed by a variety of earth disturbance activities (e.g., non-coal mining, road construction, industrial/ commercial/residential development, urban stormwater runoff, and agriculture runoff) that generate runoff and contribute total suspended solids (TSS) with elevated particulate manganese. This manganese source is based on the presence of manganese in soils and surficial materials at varying concentrations throughout Pennsylvania (Shacklette and Boerngen 1984) where this runoff could lead to exceedances and reasonable potential of exceedances of the 0.3 mg/L total manganese at these discharge locations.

The fate & transport evaluation indicates treated mine water effluent manganese (as dissolved and not total) is likely to be diluted and oxidized to insoluble forms, and precipitated in the stream within a short distance from the discharge point with less than one mile for typical treated mine discharges to small streams and to larger streams. This indicates it is unlikely manganese from a treated discharge

can reach a potable intake in a dissolved form that would require additional treatment at the potable water treatment plant. If manganese is present at the intake of a potable water treatment system, it is more likely to be found in a particulate form that would not require additional treatment or from legacy mine sites that are not currently regulated and are the responsibility of DEP.

Evaluation of mine water treatment costs indicated implementation of the EQB proposed 0.3 mg/L water quality standard would result in double to tripling of chemical costs, increase sludge volumes and associated handling and disposal costs to the coal industry, require additional treatment to lower effluent pH, and potentially result in additional new treatment to also meet aluminum effluent limits (0.75 mg/L). Additional chemical production for treatment to comply with the 0.3 mg/L manganese water quality standard will cause carbon dioxide emissions in excess of 45,000 tons annually, and air pollutant emissions to the environment. The increase in annual treatment costs for the mining sector are expected to be \$44 to \$88 million. Treatment improvements in excess of \$200 million may be needed for pH control and additional treatment to address the conflicting effluent limits for manganese (pH > 10) and aluminum (pH < 9).

The evaluation of potable water treatment indicates conventional treatment systems, which are required for surface waters, have chemicals, feed systems (e.g., pre-chlorination), and treatment processes (sedimentation and filtration) that are capable of removing manganese from source waters requiring minimal, if any, process modification or new equipment. If all manganese in the source water is dissolved, the average household cost for any additional treatment is unlikely to exceed \$1.00 annually. Review of TMDL studies suggests the majority of manganese in these impaired surface waters is from legacy mine sites that are not currently treated and are the responsibility of DEP to restore and treat.

The review of aquatic life toxicological information indicated manganese is not very toxic and toxicity is hardness dependent. The lowest Acute Toxicity Value found was 8.9 mg/L at a hardness of 92 mg/L and the lowest Chronic Toxicity Value found was 4.6 mg/L at a hardness of 100 mg/L. This indicates the most sensitive aquatic species will be protected by established coal industry BAT limits (2.0 mg/L Monthly Average and 4.0 mg/L Maximum Daily).

1.0 INTRODUCTION

The regulation of manganese in treated mine waters began with the 1972 Clean Water Act (CWA) and the specific requirements to regulate industry point source discharges using technology-based treatment. Based on these requirements, the U.S. Environmental Protection Agency (EPA) developed effluent limitation guidelines for various industry categories including the coal mining industry. In 1985, manganese best available technology (BAT) effluent limits for the coal mining industry were established at a 30-day average concentration of 2.0 mg/L and maximum daily concentration of 4.0 mg/L.

With respect to Pennsylvania, the establishment of effluent limits for a point source discharge is based on protecting the designated uses of the receiving water. This protection is typically afforded through the NPDES program and imposing the established BAT effluent limits and, if necessary, water quality based effluent limits using water quality standards contained in Chapter 93, Title 25 of the Pennsylvania Code. Where a total maximum daily load (TMDL) analysis has been conducted for non-attaining surface waters, the Chapter 93 manganese standard of 1.0 mg/L sometimes has been applied as an effluent limit at the discharge point. This manganese standard was originally established with regard to treatment concerns to minimize aesthetic concerns (e.g., taste and odor, staining of sinks, etc.) of treated water for potable water supplies (PWS) and not for aquatic life protection. Act 40 of 2017 directed the Pennsylvania Environmental Quality Board (EQB) to amend §96.3(d) to include manganese as a parameter for which the Chapter 93 criterion is to be achieved at the point of an existing or planned surface potable water supply withdrawal. This change would establish the compliance point at the nearest downstream public water supply water intake and not at the discharge point or any location in a stream.

In response to Act 40, the Pennsylvania Department of Environmental Protection (DEP) reviewed the current Chapter 93 criterion for manganese and has proposed a human health (not aquatic life) criterion of 0.3 mg/L. The EQB voted on December 17, 2019 to move forward with a proposed rulemaking on this new criterion.

This report has been prepared to assist the Pennsylvania Coal Alliance (PCA) in understanding the issues related to instream manganese concentrations and the discharge of manganese. This report provides information related to toxicity of manganese to aquatic life, fate & transport of manganese in surface waters, cost implications of manganese removal from mine drainage, and approaches and implications of removal of manganese at potable water treatment plants that use surface waters.

2.0 BACKGROUND

Manganese is one of the most common elements in the earth's crust at an average concentration of about 0.1%. Manganese is ubiquitous in the environment and is found in soils, sediments, rocks and waters in various mineral forms including oxides, carbonates, silicates and sulfides. It is an important mineral that is used to produce various steel alloys and other metallic products (IMI 2020).

Manganese is also an essential element for life. Manganese is used by plants in photosynthesis to transpose light energy into carbon sources. It is also an essential element for animal life (including humans) in enzyme systems for metabolism, bone development, production of antioxidants, and reproductive health. The primary source of manganese is dietary through food and its importance is demonstrated by studies identifying food sources high in manganese (e.g., nuts, seafoods and dark green vegetables) and specific dietary manganese supplements. Generally, manganese consumption from drinking water is much less than dietary food intake. Throughout the world, manganese deficiency and toxicity in human populations is considered rare, although "more than 35% of the world population is possibly deficient" (INstiks 2017).

Anthropogenic activities (e.g., earth disturbance) can increase the concentration of manganese in surface waters. The presence of manganese in mine waters is typically a result of secondary weathering of mine spoil and coal deposits following the initial iron sulfide (i.e., pyrite) oxidation and acidity release. The secondary manganese weathering reactions result from 1) dissolution of manganese carbonate minerals, 2) cation exchange of manganese from clays and other minerals, and 3) acidic leaching of minerals. Concentrations of manganese can vary depending on site specific conditions, but in mine waters manganese typically ranges from 0.5 to 100 mg/L.

The concentration of dissolved manganese in surface waters is low due to its low solubility under oxidative conditions. Manganese is found in suspended solids in surface waters due to its common presence in soils, in oxidized and insoluble forms. Soluble manganese is more commonly found in groundwaters where natural reducing conditions cause stable soluble forms to remain in solution. Soluble manganese can also be present in the deeper anoxic zone of surface water impoundments where reducing conditions from seasonal lake stratification can solubilize manganese from soil sediments deposited in the impoundment from erosion. It is noteworthy that neither of the above can be attributed to mining activities but are instead environment processes occurring due to the common presence of manganese in the environment.

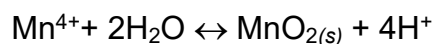
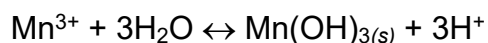
EPA has established a domestic drinking water secondary maximum contaminant level (SMCL) for manganese of 0.050 mg/L, which is based on aesthetics (i.e., complaints of taste and brown staining of sinks) and not based on human health effects. EPA has not developed any surface water quality criteria for manganese

where additional protection of human health or aquatic life would be established for surface waters if necessary.

3.0 TOTAL VS. DISSOLVED MANGANESE IN THE ENVIRONMENT

As previously indicated, manganese is a common metal in the earth's crust. It is the 6th most common metal world-wide. The average concentration reported in the earth's crust is about 0.1% (1,000 ppm) but there can be higher localized concentrations depending on the geology, rock formations and mineralogy (CRC 2017).

In aqueous environments manganese is found in three oxidation states including Mn^{2+} , Mn^{3+} , and Mn^{4+} . The solubility reactions for the three aqueous manganese oxidation states are:



Of the three, Mn^{2+} (manganous) is the soluble form typically found in natural water. Mn^{3+} (manganic) typically has some limited solubility but may have some solubility at acidic pH, typically less than 3. Mn^{4+} is considered insoluble in natural waters.

Dissolved Mn^{2+} is generally only found under reducing or low oxygen environments (e.g., groundwater, flooded soils and anoxic lake conditions). However, some mining conditions where pyritic oxidation and acidic conditions develop, dissolved Mn^{2+} may be released from chemical weathering (e.g., pyrite oxidation and acid leaching). This dissolved Mn^{2+} may remain in this soluble form for short periods or where the pH remains acidic but will be transformed to insoluble manganese where conditions support oxidation/precipitation, which are the typical conditions in most surface waters.

Manganese in soil and surficial materials, can also have implications on surface water manganese concentrations. In a USGS report by Shacklette and Boerngen (1984), soil and surficial material concentrations were evaluated in the conterminous United States that included Pennsylvania. Pennsylvania was found to have manganese (insoluble) ranging from 100 ppm to 5,000 ppm in soils and surficial materials depending on the location in Pennsylvania. This mineral manganese would mostly be in the Mn^{3+} and Mn^{4+} oxidation states and would be insoluble.

Various anthropogenic earth disturbance activities can release this insoluble manganese to surface waters in the form of suspended solids or particulate (total) manganese and not dissolved manganese. These earth disturbance activities that can release suspended solids and particulate manganese include non-coal mining,

road construction, industrial/commercial/residential development, urban stormwater runoff, and agriculture. The manganese is released from erosion of soil and surficial material and from breaking rock that exposes minerals to increased weathering. The concentration of manganese released from earth disturbance activities will depend on the concentrations found in the soils, surficial materials, and broken rock. Table 3-1 provides a matrix of total manganese potentially in runoff carrying total suspended solids (TSS) from earth disturbance activities.

Table 3-1. Matrix for the Range of Manganese Expected in Total Suspended Solids (TSS) Related to Earth Disturbance Activities in Pennsylvania.

TSS (mg/L)	Surficial Material Manganese Concentration (ppm)
25	0.002 - 0.12
50	0.005 - 0.25
75	0.008 - 0.38
100	0.01 - 0.50
200	0.02 - 1.00

As can be seen, this analysis indicates TSS produced from erosion can cause total manganese values that approach and exceed the proposed 0.30 mg/L manganese water quality standard. While regulations and erosion control may aid in lowering TSS, the analysis indicates the vast majority of earth disturbance activities have a “reasonable potential” to exceed the water quality standard.

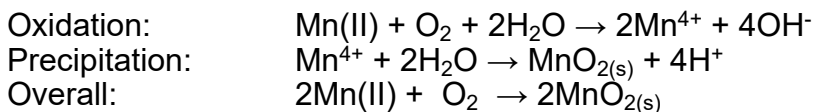
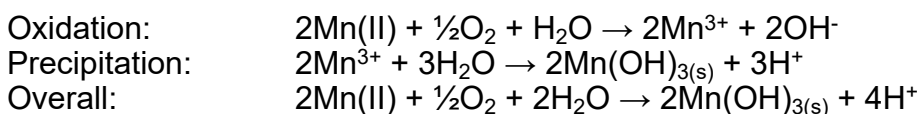
The USGS report by Shacklette and Boerngen (1984), shows a high degree of variability and no consistency of manganese concentrations in Pennsylvania soils and surficial materials. It is likely the underlying geology and minerals in the rock formations also have this high degree of uncertainty with variability within a region and a locality. However, since this analysis shows a “reasonable potential” could occur anywhere in Pennsylvania, additional material testing would be needed for each and every site proposed for non-coal mining, public infrastructure construction (e.g., roads, schools, hospitals, treatment facilities), and private development projects. If soil, surficial, and rock testing for manganese concentration is required for earth disturbance permitting, there could be added overall project costs and construction delays. Where there is a high likelihood of elevated total manganese in runoff from earth disturbance activities that could cause exceedances of the water quality standard and where control or treatment is not possible, the earth disturbance activity could be prohibited.

4.0 MANGANESE (Mn²⁺) FATE & TRANSPORT

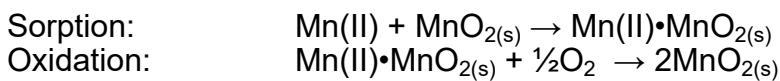
Manganese is a transition metal that can be found in aqueous solution (dissolved) principally as manganous ion (Mn²⁺), which is the reduced form of manganese. Other oxidation states of manganese are also found in aqueous environments under natural conditions, including Mn³⁺ and Mn⁴⁺, but both of these oxidation states have very low solubility at pH greater than 4. Soluble manganese, representing Mn²⁺ and complexes, collectively Mn(II), are commonly found at very low concentrations in natural surface waters, except where reducing conditions (e.g., stratified lakes and estuaries) are prevalent, and near groundwater discharges to surface waters. Concentrations of soluble manganese in natural surface waters are typically not detectable but can be higher if suitable reducing conditions prevail or there are nearby inputs of Mn(II). Groundwater can have higher Mn(II) due to reducing conditions and elevated P_{CO2} found in groundwater systems

Coal mining can also be a source of Mn(II) due to pyrite oxidation and associated chemical weathering of contact minerals including silicates, shales and carbonates. The elevated manganese from permitted mining sites is removed through treatment, where necessary, involving precipitation and oxidation of the Mn(II) to low concentrations. The following provides a discussion on the fate and transport of soluble Mn(II) found in surface waters from the water discharged from coal mining sites.

The low concentration of soluble manganese in surface waters is due to the relative instability of Mn(II) in circumneutral waters (pH = 7 ± 1), typical of surface waters. This is because under these conditions Mn(II) is oxidized to its insoluble forms including Mn³⁺ and Mn⁴⁺. The following reactions show both oxidation and precipitation of manganese.



The above oxidation (and precipitation) reactions are known as homogeneous manganese oxidation and precipitation reactions. However, there are additional manganese sorption and oxidation reactions occurring in the natural environment that can also oxidize and remove soluble manganese (Mn(II)) from water. These reactions have been described by Stumm & Morgan (1981), Hem (1981), and others. The following reactions describe this sorption, oxidation and subsequent precipitation of manganese, also known as heterogeneous manganese sorption/oxidation/precipitation:



While the above reaction equations demonstrate the reactions can occur, the actual rate of the reactions is more relevant to the removal of Mn(II) from oxygenated surface waters, which is known as kinetic or rate reactions. The combined homogeneous and heterogeneous aqueous reaction rates that lead to the removal of Mn(II) from waters are described by the following equation from Caughlin and Matsui (1975):

$$-d[\text{Mn}^{2+}]/dt = k_1 [\text{Mn(II)}] + k_2 [\text{Mn(II)}][\text{MnO}_{2(s)}]$$

The first homogeneous oxidation reaction and rate (k_1) has been found to be highly sensitive to pH with the rate increasing a 100-fold for every pH unit change over the pH range from 6 to 9. At circumneutral pH it is a relatively slow reaction. The second heterogeneous reaction is less sensitive to pH and more sensitive to the type and amount of manganese oxide solids involved in the sorption and subsequent oxidation. In other words, the rate is dependent on the concentration of insoluble manganese ($\text{MnO}_{2(s)}$) present. It is the later heterogeneous kinetic reaction that affects the Mn(II) removal rate in streams and rivers where the solids ($\text{MnO}_{2(s)}$) are present and accumulate in the stream and river bottom or substrate. This accelerated removal has been documented in the mine drainage affected sections of the Susquehanna River (Lewis 1976) and was also supported by later research conducted by Hem (1981). Hem (1981) suggested the rate of oxidation would be pseudo-first order and likely occur over an extensive and elongated area in the direction of stream flow.

More recently, Scott *et al* (2002) conducted instream studies investigating Mn(II) removal in streams. This was a controlled study where the results demonstrated the importance of surface-catalyzed oxidation of manganese within the stream. The study provided a stream removal rate of 64 μmol of Mn(II) per day per meter of stream length. This rate may be of value in assessing implications of an upstream treated mine drainage discharge containing manganese (total) on a downstream potable water intake.

The above aqueous chemistry for manganese indicates that fate & transport of manganese is an important consideration in evaluating impacts of dissolved manganese on downstream uses such as potable water supplies. Table 4-1 and Table 4-2 provide the effects of fate & transport (i.e., dilution and reaction) on treated effluent manganese concentrations. Table 4-1 provides a representative surface mine discharge flow (or a collection of discharges from a surface mine site) into a small headwater receiving stream. Table 4-2 is a high flow example using a 3.0 MGD pumped deep mine discharge located at river mile 5.0 in the West Branch of the Susquehanna River. While the majority of NPDES mine discharges are likely closer to the Table 4-1 example, the two in combination show the range of discharge flow scenarios to be expected.

In the tables, the 10-year recurrence baseflow (a low flow condition) was developed using USGS (2020) by comparing watershed area, stream mile and

stream flow. The following equation was developed and used to convert river mile to streamflow.

$$\text{Stream Flow (cfs)} = 0.180 \times (\text{River Mile})^2 + 0.28 \times (\text{River Mile})$$

Table 4-1. Example Showing the Effects of Fate (i.e., Reaction) and Transport (i.e., Dilution) on Manganese for a Treated Mine Drainage Effluent (Average Monthly = 2.0 mg/L) for a Low Flow Mine Discharge Flow (Flow = 100 gpm or 0.22 cfs) in a Headwater Location.

	Initial Dilution Ratio (Discharge Flow : Stream Flow)							
	1:0		1:1		1:3		1:10	
Stream Mile	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L
Effluent	NA	2.00	NA	2.00	NA	2.00	NA	2.00
0	0.00	2.00	0.22	1.00	0.67	0.50	2.22	0.18
0.5	0.18	1.07	0.51	0.60	1.09	0.33	2.90	0.14
1.0	0.46	0.63	0.89	0.39	1.59	0.24	3.69	0.11
1.5	0.82	0.40	1.36	0.27	2.19	0.17	4.56	0.09
2.0	1.28	0.28	1.92	0.19	2.88	0.13	5.52	0.07
2.5	1.82	0.20	2.57	0.15	3.65	0.10	6.57	0.06

Table 4-2. Example Showing the Effect of Fate (i.e., Reaction) and Transport (i.e., Dilution) on Manganese for a Large Deep Mine Discharge Flow (Flow = 2.88 MGD or 4.46 cfs) in a larger stream.

	Initial Dilution Ratio (Discharge Flow : Stream Flow)							
	1:1.3 ¹		1:2		1:3		1:5	
Stream Mile	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L	Stream Flow cfs	Diss. Mn mg/L
Effluent	NA	2.00	NA	2.00	NA	2.00	NA	2.00
0.0	5.9	2.00	8.9	0.67	13.3	0.50	22.2	0.33
1.0	6.4	0.86	11.6	0.54	16.5	0.41	26.4	0.28
2.0	7.2	0.71	14.7	0.43	20.2	0.34	30.9	0.23
3.0	8.4	0.62	18.1	0.35	24.2	0.28	35.8	0.20
4.0	9.9	0.54	21.9	0.29	28.5	0.23	41.1	0.17
5.0	11.8	0.45	26.1	0.24	33.3	0.20	46.7	0.14
6.0	14.0	0.38	30.6	0.20	38.3	0.17	52.7	0.12
7.0	16.7	0.32	35.5	0.17	43.8	0.14	59.1	0.11
8.0	19.6	0.27	40.7	0.14	49.6	0.12	65.7	0.09

¹ Represents actual location of a large treated flow in the West Branch Susquehanna River.

Table 4.1 contains modeling results for low flow discharges in headwater locations, representative of discharges from surface and underground coal mines into small streams at common NPDES permit locations. The modeling shows how the in-stream concentration of manganese decreases rapidly from the discharge point,

which is due to the combined effects of reaction (fate) and dilution (transport). This analysis uses an NPDES effluent concentration of 2.0 mg/L, which the treated effluent must be below (as a monthly average) in order to comply with the EPA technology-based effluent limitations in the permit. In addition, the discharge flow from sedimentation ponds is likely to be dependent on receiving stream flow condition, where lower than permitted discharge flow occurs at lower (baseflow) stream flow conditions. Overall it is evident in the modeling analysis that even in effluent-dominated headwater streams (i.e., where the discharge from the coal mine operation provides essentially all of the stream flow), the in-stream concentration of manganese decreases to approximately 1 mg/L (the current Chapter 93 criterion) within one-half mile downstream of the discharge point. When the discharge flow to streamflow ratio is 1:1, 1:3 and 1:10, the in-stream manganese concentration decreases to well below 1 mg/L within one-half mile of the discharge. This analysis indicates treated coal mine discharges located in headwater locations have minimal effect on downstream manganese concentrations, typically less than one-half mile downstream of the discharge point.

Table 4.2 contains modeling results for high flow discharges that are more typically located in larger streams and rivers and representative of larger underground coal mines with discharges. These are a small number of coal mine NPDES permit locations. Similar to previous modeling for low flow discharges, the in-stream concentration of manganese decreases a short distance from the discharge point, which is due to the combined effects of reaction (fate) and dilution (transport). This analysis uses an NPDES effluent concentration of 2.0 mg/L, which the treated effluent must be below (on average) in order to comply with the EPA technology-based effluent limitations in the permit. Overall it is evident in the modeling analysis that under none of the conditions modeled does the in-stream concentration of manganese exceed the current Chapter 93 criterion of 1 mg/l at any point beyond one mile of the discharge location.

5.0 MINE WATER TREATMENT COST EVALUATION

Active treatment is the typical approach used for treatment of permitted coal mine drainage discharges. Active treatment is described in “Neutralization of Acid Mine Drainage” by EPA 1983. Most of the described treatment practices and processes are still in use today with some advancements to improve performance and sludge characteristics. In general, active mine drainage treatment involves use of caustic chemicals (e.g. lime) to raise the pH to neutralize acidity and precipitate metals including iron, aluminum and manganese. Aeration may be provided to promote the oxidation of ferrous to ferric, and its resulting precipitation as an iron oxyhydroxide. Normally a neutralization pH of 8.0 ± 0.2 is adequate for the precipitation of iron and aluminum to effluent limits.

Removal of manganese by neutralization will depend on a number of factors including the initial manganese concentration and iron concentration in the mine water. Investigation indicates initial manganese in a little over 90% of the mine discharges exceeds 1 mg/L but nearly 100% exceeds 0.3 mg/L. Manganese removal is more complex than iron removal due to the more complex chemistry of manganese where oxidation from Mn(II) to insoluble Mn(III) is slower, manganese will sorb to iron oxyhydroxide solids, and Mn(II) hydroxide has a higher solubility in water than either ferrous or ferric hydroxides. These factors will require a highly pH-controlled neutralization process depending on the effluent limit for manganese, which in turn will determine the lime dose required.

Figure 5-1. Example #1 Titration Results (Mine Water with Alkalinity)
Lime Dose & Dissolved Manganese Relationship

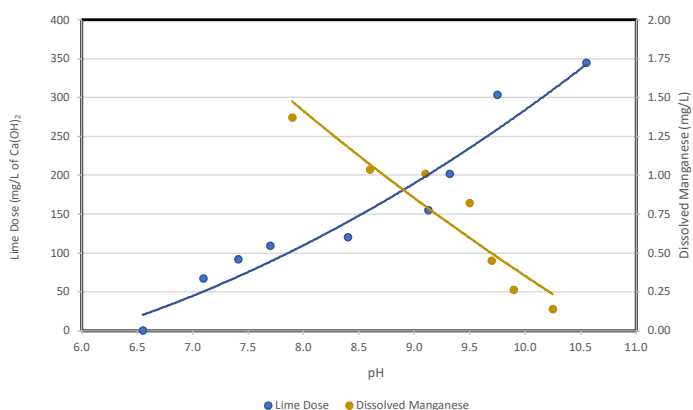


Figure 5-2. Example #2 Titration Results (Acidic Mine Water)
Lime Dose & Dissolved Manganese Results

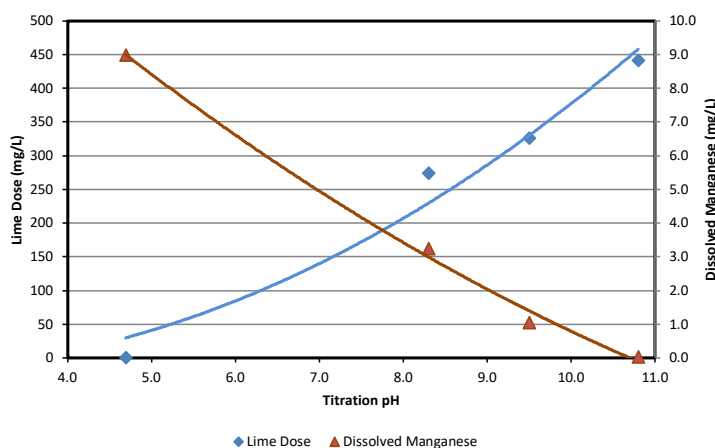


Figure 5-1 and Figure 5-2 are provided as real-world examples of titration curves related to lime dose, manganese removal, and endpoint pH. Figure 5.1 is for a mine discharge containing alkalinity, ferrous iron of about 40 mg/L, and an initial manganese concentration less than the 2.0 mg/L BAT-based effluent limit. Figure 5.2 is for an acidic mine discharge containing about 100 mg/L ferrous iron, 10 mg/L of aluminum, and about 8 mg/L of manganese.

As shown, dissolved manganese decreases nonlinearly with pH and approaches 0 mg/L at pH approaching 11. However, alkaline chemical (e.g., lime) dose increases nonlinearly with pH, which is due to the effect of calcium and magnesium precipitation as pH increases. Both figures show the pH and chemical dose must be increased from the BAT limit of 2.0 mg/L lime dose by more than 100 mg/L to achieve a 1.0 mg/L effluent limit, and by more than 200 mg/L to achieve a 0.3 mg/L limit. Using the two examples, lime use costs would increase by between \$0.10 to \$0.15 per 1,000 gallons treated and sodium hydroxide use costs would increase by between \$0.30 to \$0.45 per 1,000 gallons treated. In general, lime is used at higher flow discharges (> 200 gpm) and sodium hydroxide is used a lower flow discharges. Evaluating this on a coal industry-wide basis for the approximately 700 NPDES permits, and assuming approximately 200 gpm of mine discharge per NPDES permit, the total industry increase alkaline chemical cost would be between \$15 and \$40 million annually, depending on the percent of discharges treated with lime or sodium hydroxide.

In addition to lime costs, there would be an increase in several other operating costs including sludge disposal from the increased calcium and magnesium precipitation that will increase sludge volumes at the higher pH needed to meet the lower proposed manganese criterion. Sludge handling costs are about \$0.05 to \$0.10 per 1,000 gallons treated, based on calculations provided in AMDTreat, an OSMRE software product. If sludge volumes are merely doubled from the higher pH required to achieve the low manganese concentrations, this increased sludge handling would amount to an additional \$5 to \$10 million in treatment costs.

Additional treatment will also be required to meet the effluent pH of between 6 and 9. This will require acid addition or post-treatment aeration to lower the pH to the required effluent range. Acid addition for pH adjustment will require storage tanks and chemical feed systems with operating costs associated with acids and manpower, which will be similar in capital costs for a sodium hydroxide system used to raise pH for manganese removal. The estimated capital costs per location using AMDTreat is between \$30,000 and \$40,000 per year. Using the number of permit locations this results in a total capital cost of \$20 to \$40 million. Expected sulfuric acid doses to lower pH would range between \$0.05 and \$0.10 per 1,000 gallons treated resulting in an expected operating cost between \$4 and \$8 million annually.

There is one additional factor for some discharges that the alkaline chemical dose and pH for manganese removal does not capture. This is related to the conflict of

effluent compliance with a proposed low manganese effluent limit of 0.3 mg/L with the Chapter 93 aluminum criterion of 0.75 mg/L, which is established for the protection of aquatic life. Aluminum that is normally precipitated in treated mine water from neutralization between pH 8 and 9 will be resolubilized at pH higher than 9. The aluminum solubility indicates an effluent limit of 0.75 mg/L (the Chapter 93 criterion) will be exceeded at pH greater than about 9.5. This is a result of the formation of an aluminum hydroxide complex ($\text{Al}(\text{OH})_4^{1-}$) that will increase dissolved aluminum in the discharge as the pH is increased above 9.5, which would be required to meet the low manganese effluent limit. This situation would be applicable to all mine discharges with elevated aluminum in the untreated mine water. Addressing the removal of this dissolved aluminum is not simple. The decreased pH adjustment to less than 9 using acid described following high pH manganese removal would precipitate the aluminum in response to the lower pH, but the suspended solids would be less 5 mg/L and would not settle effectively. Because of this, higher aluminum discharges could require installation of a completely new treatment plant, addition of treatment components of equal size (i.e., a second stage), or addition of filtration components to existing plants. Based on equivalent cost of current treatment at half of the existing NPDES permits to address aluminum there is potentially capital cost of \$175 million with between \$20 and \$30 million in additional operating costs annually.

Based on the above preliminary analysis based on the treatment in the general mining sector, the proposed 0.3 mg/L water quality standard if applied at the discharge point could increase treatment costs by between \$44 and \$88 million annually. The wide range is due to generalizations and more refined estimates would require better understanding of flow, chemistry and treatment at each NPDES permit location. In addition to the increase in capital costs there is a potential additional capital costs in order to meet the 0.3 mg/L water quality standard. The capital costs could exceed \$200 million.

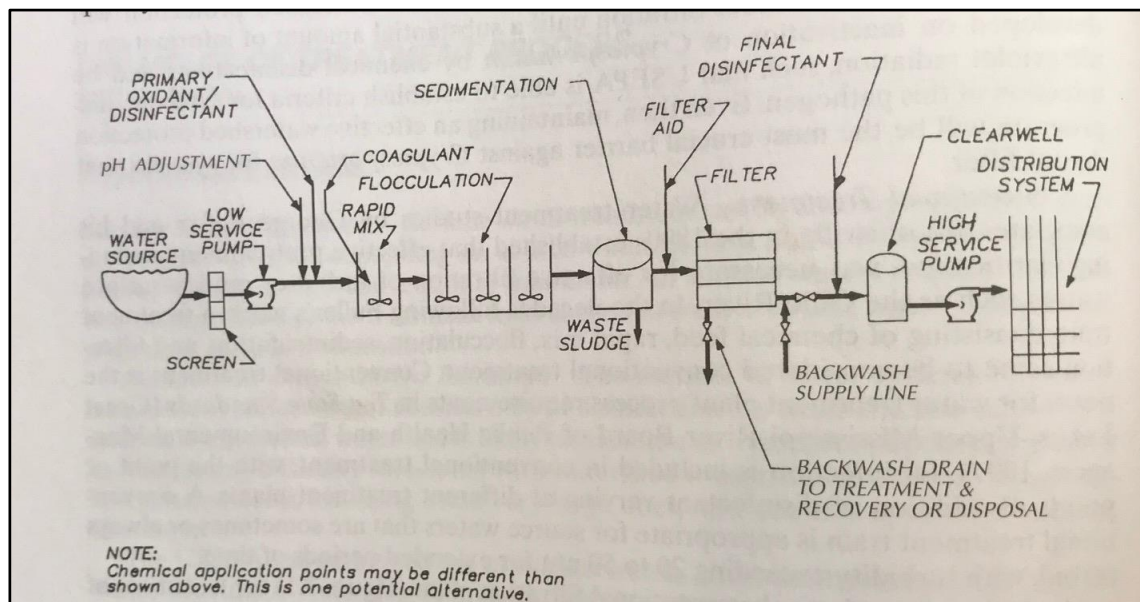
There are several other aspects to a lower manganese effluent limit that should be part of any assessment and this relates to the additional pollution that will occur as a result of promulgated the proposed 0.3 mg/L water quality standard. As indicated above, alkaline chemical addition will be expected to increase. Using lime as an example, the additional lime use will result in additional mining of limestone, production facilities to produce lime from the limestone, gas emissions (e.g., NO_x , SO_x , carbon dioxide, particulates) related to converting limestone to lime, and transportation (increased truck traffic) to deliver lime to each operating mine water treatment plant. Using lime as a basis the following is an assessment of the carbon dioxide emissions. The gas emissions from converting lime from limestone will include carbon dioxide, which is both evolved from the limestone and the energy used to heat the limestone in order to free the carbon dioxide from the limestone. For each ton of lime produced about 0.6 tons of carbon dioxide will be released from the limestone. The energy to heat the limestone will result in the release of between 0.20 and 0.45 tons of carbon dioxide, depending on the heating fuel used (GGP 2020).

Overall, the production of 1 ton of lime will result in about 1 ton of carbon dioxide emissions. In the above examples and based on a 1 MGD flow basis, carbon dioxide emissions just from the production of lime will be about 45,000 tons annually to meet the 0.3 mg/l water quality standard. The above analysis represents just the carbon dioxide emissions and as indicated above, there would be additional pollutants released to the environment for the mining industry to comply with the 0.3 mg/L water quality standard if applied at the discharge point.

6.0 POTABLE SURFACE WATER TREATMENT EVALUATION

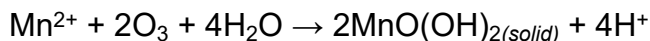
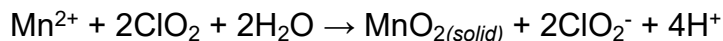
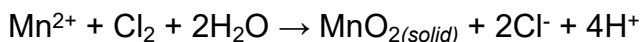
The treatment of surface water for drinking water must comply with the EPA Surface Water Treatment Rule, which generally requires conventional treatment of the raw water. For raw water drawn from larger turbid sources, filtration is required in the treatment process. A generalized process schematic for conventional potable water treatment is provided in Figure 6-1. Overall conventional water treatment consists of: 1) screening of the intake to remove large debris; 2) chemical feed of oxidants, disinfectants, pH adjustment chemicals (e.g., lime), coagulants and flocculants; 3) rapid mix to disperse the chemicals into the water; 4) flocculation to increase the size of the coagulated particles; 5) sedimentation to remove the large flocculated suspended solids; 6) filtration to remove fine particles and provide removal of pathogens (e.g., cryptosporidium oocysts); and 7) post-chlorination of the treated drinking water to maintain a disinfectant residual throughout the distribution system. The overall objective of conventional treatment is to provide a clean water free of suspended solids and pathogens.

Figure 6-1. Conventional Treatment, Surface Water. From AWWA 1999



As shown in Figure 6-1, one of the components in the initial treatment is the addition of primary oxidant/disinfectant chemicals. This chemical for surface waters often includes chlorination (or ozone), known as pre-chlorination, and is a component in the initial disinfection of the raw water and to prevent slime growth in the treatment systems including flocculators, pipes, clarifiers, and filters. While the primary function is disinfection and slime control, these chemicals will also act

as oxidants to oxidize inorganics, such as sulfurs, iron and manganese. The following reactions demonstrate the oxidation of manganese by these chemical oxidants (Benefield *et al* 1982 and AWWA 19999).



The first two are reactions with chlorine or chlorine dioxide, two forms of chlorine that are commonly used in water treatment. The third reaction is for ozone, which can also be used as a pre-treatment disinfectant/oxidant in water treatment.

In water treatment, greensand filtration is used for removing manganese but is generally used in the treatment of groundwater sources and not surface waters. Chlorine is frequently used for the oxidation of dissolved manganese prior to the greensand filters, where the oxidized and precipitated manganese is removed. In practice, conventional water treatment plants are not specifically designed to address dissolved manganese. However, dissolved manganese would be oxidized by the pretreatment oxidant/disinfectant chemical addition in conventional treatment systems and then the particulate manganese would be removed in subsequent treatment process including sedimentation and filtration. At most, any slight increase in dissolved (or reduced) manganese in intake water would require a modest increase in chemical (chlorine) use as part of pre-chlorination.

For example, 1 mg/L of dissolved manganese (Mn^{2+}) would require a chlorine dose of 1.3 mg/L, based on the equation provided above. *Note, if manganese is as a particulate, which would be the case for suspended solids in the raw water, no chlorine dose would be required and the particulate manganese would be removed as part of suspended solids removal.* This chlorine dose would result in a chlorine use at a surface water treatment plant of about 0.01 lbs per 1,000 gallons treated. The current market for chlorine depends on the cylinder (lbs to tons) purchased. Assuming an average cost of \$1.10 per pound across all cylinder sizes results in an annual chemical cost of about where there is 1 mg/L of dissolved manganese present in the intake water. If the 0.3 mg/L manganese is imposed at the discharge point for any discharger, and assuming 0.3 mg/L of dissolved manganese (and not particulate manganese) occurs at the intake of the potable water treatment, the regulation would result in a maximum treatment savings of less than \$0.007 per 1,000 gallons water treated at the treatment plant. The average household cost savings created by the 0.3 mg/L water quality standard would be only about \$0.40 to \$1.00 per year.

The above cost analysis does not consider the fate and transport of dissolved manganese provided in Section 4.0 above that shows it is unlikely that dissolved manganese from a treated mine discharge would reach a downstream potable

water intake. It is more likely that the majority of manganese that reaches the intake from a permitted mine discharge would be insoluble and would be present only during high flow events, when particulate manganese is scoured from the stream banks and bed. This manganese would be part of the suspended solids, which would be removed by the conventional treatment in sedimentation and filtration. This particulate manganese would not add to chemical costs and would be minor in the overall suspended solids likely to be removed by the water treatment plant.

The above analysis does not address current situations where dissolved manganese is in the surface water at the potable water intake and chemical oxidant is added. The source of this dissolved manganese is likely from upstream historic mining activities, but unrelated to NPDES discharge points from mining. Lewis (1978) studied the Susquehanna River basin and found manganese concentrations in basins with mining was significant. However, this manganese was not from permitted mining sites where treatment removes manganese to established effluent limits but from past abandoned mine sites where the mine water is left untreated. Review of two recent watershed reports including the “Moshannon Creek Watershed TMDL” prepared by DEP (2009) and the “Acid Mine Drainage TMDLS for the Kiskimintas-Conemaugh River Watershed, Pennsylvania” prepared by the EPA (2010b) indicates the vast majority of mine drainage loading (and manganese) in these mining affected basins is from abandoned legacy sources. It is these abandoned untreated sources that have resulted in TMDL implementation and would be the sources affecting downstream potable water intakes and, not the regulated and treated NPDES mine water sources. Treatment of these legacy discharges would be needed to comply with the 0.3 mg/L water quality standard, and this would be at the burden of the state and the state taxpayers. Unfortunately, watershed restoration goals for nearly all of the legacy sites do not include manganese removal. There may be some manganese removal but it is typically very limited because of the additional costs to achieve this manganese removal that can double treatment costs, limited available funding, and the minimal benefit of manganese removal at achieving watershed restoration objectives that primarily focus on pH, iron, aluminum and acidity/alkalinity for aquatic life restoration and recreational benefits.

7.0 AQUATIC LIFE TOXICITY

As previously indicated, there are no “Quality Criteria for Water” for manganese established by EPA to protect aquatic life, which is due to the near absence of manganese toxicity to fish and aquatic life. An evaluation was conducted to obtain, review and summarize published peer reviewed manganese aquatic life toxicity information.

Pollutants, such as dissolved metals, can be either acutely toxic causing mortality or have long term effects related to survival, growth and reproduction. The U.S. EPA publishes criteria documents that States may choose to adopt as water quality standards. These documents recommend criterion maximum concentrations (CMC) to prevent short term or acute toxicity impacts and criterion continuous concentrations (CCC) to prevent long term chronic toxicity impacts to aquatic life (or human health) in surface water. In the case of manganese, the EPA has not published any criteria document for manganese.

A literature search was conducted for publicly available publications related to manganese toxicity to aquatic life. The information available was somewhat limited. This is, in part, due to the relatively low toxicity of manganese to aquatic life and therefore lack of interest on the part of researchers.

The following Table 1 summarizes acute aquatic life toxicity data and Table 2 summarizes chronic aquatic life toxicity data from toxicological information obtained from journals, reports, and theses. Only moderate hardness test water conditions are provided in the table, which represents mid-range manganese toxicity as manganese toxicity is highly hardness dependent. Several studies documented this hardness dependency that indicates the aquatic life toxic concentration of manganese increases as hardness increases. The available aquatic life toxicity information included acute toxicity (e.g., 96-hr LC50) and chronic toxicity (e.g., LOEC) for a number of aquatic species including mollusks, crustaceans, insects and fish. A number of these aquatic species are known to be sensitive to pollution.

As can be seen in Table 1, the most sensitive reported aquatic species with acute toxicity to dissolved manganese is the freshwater scud (*Hyalella azteca*) with an acute toxicity concentration of 8.6 mg/L. Manganese in a concentration range of 13 to 20 mg/l has been reported to have acute toxic effects on some salmonid species. Because the hardness concentration of surface water affected by mine drainage is much greater than the laboratory test conditions reported in these studies, the concentration at which manganese is acutely toxic to aquatic species in that environment will be greater than shown in Table 1. Overall, it is evident that the BAT effluent limits of 2.0 mg/L average monthly and of 4.0 mg/L daily maximum, as total manganese, provide adequate protection for freshwater fish

and aquatic life, even at the low hardness concentrations of the laboratory test water.

Table 7-1. Acute Aquatic Life Toxicity Data for Dissolved Manganese.

Species	Endpoint	Life Stage	Effect Concentration mg/L	Test Hardness mg/L	Reference
Water Flea <i>Ceriodaphnia dubia</i>	48-hr LC ₅₀	< 24-hr old	14.5	92	Lasier <i>et al</i> 2000
Scud <i>Hyalella azteca</i>	96-hr LC ₅₀	7-day old	8.6	92	Lasier <i>et al</i> 2000
Longfin Dace <i>Agosia chrysogaster</i>	96-hr LC ₄₀	Juvenile	84	218	Lewis 1978
Sowbug <i>Asellus aquatica</i>	48-hr LC ₄₀	Adult	771	50	Martin & Holdich 1986
Amphipod Crangonyx pseudogracillis	48-hr LC ₄₀	Adult	1389	50	Martin & Holdich 1986
Rainbow trout <i>Oncorhynchus mykiss</i>	96-hr LC ₅₀	Early Life Stage	20.7	100	Reimer 1999
Coho salmon <i>Oncorhynchus kisutch</i>	96-hr LC ₅₀	Early Life Stage	13.2	100	Reimer 1999
Scud <i>Hyalella azteca</i>	96-hr LC ₅₀	Adult	19.1	100	Reimer 1999
Water Flea <i>Daphnia magna</i>	48-hr LC ₅₀	Adult	26.2	100	Reimer 1999
Midge <i>Chironomid tentans</i>	96-hr LC ₅₀	Adult	40.8	100	Reimer 1999
Clam <i>Lampsilis siliquoidea</i>	96-hr LC ₅₀	Adult	40.8	90	EPA 2010a
Mussel <i>Megaloniais nervosa</i>	96-hr LC ₅₀	Adult	31.5	90	EPA 2010a

Table 7-2. Chronic Aquatic Life Toxicity Data for Dissolved Manganese.

Species	Endpoint	Life Stage	Effect Concentration mg/L	Test Hardness mg/L	Reference
Brown trout <i>Salmo trutta</i>	Growth & Survival IC ₂₅	Early Life Stage	4.59	150	Stubblefield <i>et al</i> 1997
Algae <i>Scenedesmus quadricauda</i>	Growth EC ₅₀	NA	4.98	Not Available	Fagasova <i>et al</i> 1999
Water Flea <i>Ceriodaphnia dubia</i>	Brood - IC ₅₀	< 24-hr old	8.5	92	Lasier <i>et al</i> 2000
Rainbow trout <i>Oncorhynchus mykiss</i>	EC ₅₀	7-day Early Life Stage	20.7	100	Reimer 1999
Coho salmon <i>Oncorhynchus kisutch</i>	EC ₅₀	7-day Early Life Stage	13.2	100	Reimer 1999
Water Flea <i>Daphnia magna</i>	21-day LOEC	Adult	6.9	100	Reimer 1999
Algae <i>Selenastrum capricornutum</i>	72-hr Growth IC ₅₀	Adult	8.29	100	Reimer 1999

As can be seen in Table 2, the aquatic species with the most sensitive chronic toxicity to dissolved manganese is brown trout (*Salmo trutta*) with a growth effect concentration of 4.6 mg/L. This species was followed closely by an algae (*Scenedesmus quadricauda*) with a growth effect of manganese at 5.0 mg/L. Similar to aquatic life acute toxicity, aquatic life chronic toxicity is found to be hardness dependent and the hardness values in mine water affected surface waters are likely to be much greater than the values shown in Table 2. Overall, it is evident that the BAT effluent limit of 2.0 mg/L average monthly, as total manganese, provides adequate protection for freshwater fish and aquatic life, again even at the low hardness concentrations of the laboratory test water.

8.0 SUMMARY

This evaluation of the EQB proposed 0.3 mg/L water quality standard examined fate & transport of dissolved manganese in surface waters, potential cost implications on mine water treatment, evaluation of treatment at downstream potable water treatment systems, and aquatic life toxicity. The study findings include:

- In surface waters, manganese can be found in both dissolved and total (Mn^{3+} and Mn^{4+}) forms. Anthropogenic dissolved manganese in surface waters may include coal mining activities and the chemical leaching/weathering associated with pyrite oxidation. Anthropogenic particulate manganese in surface water may also be contributed by a variety of earth disturbance activities (e.g., non-coal mining, road construction, industrial/commercial/residential development, urban stormwater runoff, and agriculture) that generate runoff and contribute TSS to levels that may require manganese regulation from these sources.
- The fate & transport evaluation indicates treated mine water effluent manganese (as dissolved and not total) is likely to be diluted and precipitated in the stream in a short distance from the discharge point with less than one-half mile for typical treated mine discharges (e.g., surface mines) to small streams and less than 1 mile for large flow discharges (e.g., deep mines) to larger streams. This indicates it is unlikely manganese from a treated discharge can reach a potable intake in a dissolved form.
- Evaluation of mine water treatment indicated implementation of the EQB proposed 0.3 mg/L water quality standard would result in:
 - Double to tripling of chemical use to lower effluent manganese from compliance with an effluent limit of 2.0 mg/L to an effluent limit of 0.3 mg/L.
 - Other operating issues, such as sludge handling and disposal, would also increase proportionally.
 - Additional pH adjustment treatment will be needed to lower pH to effluent limits (pH 6 to 9).
 - High aluminum discharges will exceed aluminum effluent limits (0.75 mg/L) because of the increasing solubility of aluminum at pH greater than 9.5 that will likely require substantial upgrades and new treatment to meet effluent limits.
 - The additional chemical use to comply with the 0.3 mg/L manganese water quality standard will cause carbon dioxide emissions associated with the additional use of alkaline chemicals (i.e., production of lime from limestone) to increase by more than 45,000 tons annually.
- The anticipated increase in costs associated with the above changes in treatment to comply with the EQB proposed 0.3 mg/L water quality standard are estimated to be:
 - Annual treatment cost increase for the coal mining sector are expected to be \$44 to \$88 million.
 - Required treatment improvements for the coal mining sector will exceed \$200 million due to pH control and additional or changes in treatment

needed to address the conflicting effluent limits for manganese (pH > 10) and aluminum (pH < 9).

- Evaluation of potable water treatment indicates:
 - Conventional treatment is required because of the surface water source.
 - Conventional treatment systems for surface waters have chemicals, feed systems (e.g., pre-chlorination), and treatment processes (sedimentation and filtration) capable of removing manganese from source waters that will require minimal, if any, process modification or new equipment.
 - The potential impact to an average household is unlikely to exceed \$1.00 annually and this would only be in waters affected by mining discharges that are primarily from abandoned mine lands or non-NPDES discharge locations.
- The review of aquatic life toxicological information indicated manganese is not very toxic and toxicity is hardness dependent with the most sensitive species protected by established BAT limits (2.0 mg/L Monthly Average and 4.0 mg/L Maximum Daily).
 - Lowest Acute Toxicity Value = 8.9 mg/L @ 92 mg/L Hardness
 - Lowest Chronic Toxicity Value = 4.6 mg/L @ 100 mg/L Hardness

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