August 26, 2019

Comments from the Natural Resources Defense Council
On EPA's Proposed National Primary Drinking Water Standards for Perchlorate
Docket ID No. EPA-HQ-OW-2018-0780-0147
84 FR 30524 June 26, 2019[1]

The following comments are being submitted on behalf of the Natural Resources Defense Council (NRDC) and our over three million members and online activists. NRDC combines the expertise of some 600 scientists, lawyers, and policy advocates to work for the rights of all people to clean and affordable water to drink, safe air to breathe, and a flourishing environment including wildlife and wild places. NRDC has no direct or indirect financial or fiduciary interest in the manufacture or sale of perchlorate or any chemical that would be the subject of this proposed rule.

With this FR Notice, the Environmental Protection Agency (EPA) is proposing to set both the enforceable drinking water Maximum Contaminant Level (MCL) for the perchlorate regulation and the perchlorate MCLG at 0.056 mg/L (56 µg/L, or parts per billion, ppb). The EPA also states that it may set the MCL at 18 ppb, 90 ppb, or withdraw it altogether, leaving toxic rocket fuel in the water and foods of Americans.

NRDC strongly opposes this proposal, on the following grounds:

- The best available peer-reviewed science supports the conclusion that the Agency's proposal will cause harm at the population level;
- Failure to include an adequate margin of safety violates the clear requirements of the Safe Drinking Water Act and will ensure that the MCL Goal (MCLG) and the proposed MCL will not be public health protective;
- The most vulnerable subpopulations, pregnant women and their offspring, will be permanently harmed.

- EPA has unlawfully failed to meaningfully consider many of the quantifiable and non-quantifiable benefits of establishing a standard for perchlorate of lower than 18 ppb, such as a standard equivalent to the Massachusetts standard of 2 ppb.

In these comments, NRDC demonstrates that for these reasons, EPA's proposal fails to meet the legal requirements of the Safe Drinking Water Act (SDWA).

Perchlorate is a hazardous chemical component of explosives that is used in rocket fuel, and is still used in fireworks, air bags and in food packaging. It also occurs naturally in Chilean soil nitrate, which has historically been used as fertilizer here in the US. It also frequently occurs in hypochlorite, which is often used to treat drinking water—a source that would not be found in source water monitoring, and which EPA's Unregulated Contaminant Monitoring in some cases would not have detected because it is often added after the intake or even after the point of entry into the distribution system (due to booster chlorination etc.).[2] EPA's analysis fails to consider this type of exposure.

According to a 2010 GAO report, "Perchlorate has been found in water and other media at varying levels in 45 states, as well as in the food supply, and comes from a variety of sources."[3] EPA reported that approximately 4% of 3865 public water supplies tested – serving over 16 million people- had detections of perchlorate at or above 4 ppb (the lowest level that was looked for) or higher.[4] FDA found it in well over half of food samples it analyzed, including baby foods and infant formula.[5] It is also in human breast milk. [6] [7] [8] For an acute contaminant like perchlorate where even short-term exposures can have health consequences for vulnerable populations, consumers need reliable information about the health risks associated with perchlorate, what routes of exposure are of greatest concern, and how to prevent exposures.

NRDC recommends that EPA set the MCL at no higher than 2 ppb

NRDC is recommending that EPA move forward without delay, to set a health-protective MCL that is no higher than 2 ppb, consistent with the best available science, and with the legal requirements under SDWA. An MCL of 1-2 ppb is consistent with the scientific peer reviewed determinations of recent state actions:

- California has an enforceable drinking water standard of 6 ppb, and a public health goal (PHG) of 1 ppb perchlorate in drinking water, (updated from 6 ppb in 2004), based on perchlorate's adverse impacts on the thyroid gland. A healthy thyroid gland is necessary for prenatal and postnatal growth and development (CAL EPA 2015).[9]
- Massachusetts has an enforceable drinking water standard of 2 ppb (Mass DEP 2006), based on the Reference Dose (RfD) of 0.00007 mg/kg-day recommended by the National Academies (NAS 2005),[10] a relative source contribution of 20% from water, and an Uncertainty Factor (UF) of 300X. While the drinking water standard must be set to protect all members of the public including vulnerable subpopulations, the state also emphasizes that *no one* should consume water with perchlorate levels that exceed 18 ppb.[11]

Following lengthy rigorous public processes that included public comment opportunities and scientific peer review, a dozen states have established advisory levels or health-based goals that range from 1 to 18 ppb in drinking water (GAO 2010).[12]

EPA's current proposal to set an MCL and MCLG at 56 ppb or higher, or even withdraw it altogether, is a clear outlier, presumably a gift to polluters – mainly the Department of Defense (DOD) and military contractors – that are responsible for clean-up. Although perchlorate occurs naturally at low levels in arid regions such as the southwest US, over 90% of domestically produced perchlorate is used in the defense and aerospace industries, [13] so EPA's decade-long delay has already benefited these polluters.

EPA's current proposal will continue to harm the public from a neurodevelopmental toxicant for which there may be no safe level.

DETAILED COMMENTS

The best available science supports the conclusion that the Agency's proposal will cause harm at the population level.

EPA's proposal does not comply with requirement in the Safe Drinking Water Act that the agency must use the use of best available, peer-reviewed science, as mandated by SDWA § 1412(b).
The Act specifies that, when EPA acts under § 1412 to regulate drinking water contaminants, "to the degree that an Agency action is based on science, the Administrator shall use . . . the best available, peer-reviewed science and supporting studies conducted in accordance with sound and objective scientific practices . . . ." 42 U.S.C. § 300g-1(b)(3)(A)(i). As EPA is proposing to regulate perchlorate under § 1412, this requirement to use "best available, peer-reviewed science" applies to every aspect of that action that is based on science.

Below we detail some of the significant ways that this proposal fails to follow best available science, with the result that it's proposed MCL/MCLG underestimates risk.

<u>EPA failed to use SAB recommended population-based approach, thereby ignoring important public-health related data.</u>

In 2012 EPA requested review of its previous assessment from the Science Advisory Board (SAB), as required by SDWA § 1412(d). At the SAB meeting, EPA presented – and SAB approved – an approach that EPA termed the 'population-based approach' which involved evaluating the shift in the proportion of the population for whom perchlorate exposure would result in a reduction in thyroid hormone levels to below normal (hypothyroxinemia).[14]

However, in this proposed rule, EPA is now rejecting its own previous proposal and SAB recommendations, instead proposing an MCL/MCLG based on a 'two-stage approach' that clearly underestimates the hazard and risk. In its Charge Questions to the Peer Reviewers in 2018, EPA describes its process as: "Using the output from the revised BBDR [Biologically Based Dose Response] model (stage 1 of the analysis) and the quantitative relationships between thyroid hormone levels and neurodevelopmental effects from the published epidemiological studies (stage 2 of the analysis), EPA characterized the relationship between perchlorate exposure on fT4 levels in pregnant mothers during early gestation and the potential for changes in neurodevelopmental outcomes in their offspring."[15] For the second stage, linking maternal perchlorate exposure to outcomes in offspring, EPA is here proposing to use a dose-response function from its own 'EPA independent analysis' of IQ loss extrapolated from a modeled first-trimester fetal exposure to perchlorate derived from a single epidemiologic study (Korevaar et al 2016).[16]

EPA identifies three significant advantages that the population-based approach has over the two-stage approach that it ultimately selected. First, the population-based approach is based on an endpoint of hypothyroxinemia, which is well-established in the published scientific literature to be causally associated with an elevated risk of adverse neurodevelopmental outcomes. Second, because the population-based approach is based on risk of neurodevelopmental outcomes instead of a single outcome (the two-stage approach is based only on IQ loss) it is a more accurate representation of the suite of risks associated with early-life perchlorate exposure. Third, the population-based approach avoids the uncertainty of quantifying the dose-response relationship between maternal free T4 levels and the magnitude of adverse impacts.

By relying on the two-stage model instead of the population-based approach, EPA is introducing significant uncertainties into its assessment, as discussed below.

<u>EPA failed to follow External Peer Review recommendations to address study limitations and uncertainties</u>

For the 2018 external peer review, EPA included a charge question asking the reviewers to make recommendations to EPA on its use of five epidemiologic studies with endpoints relevant to neurodevelopment: "Charge Question 10a: Assumptions, strengths and limitations of focusing on five studies and their associated neurodevelopmental endpoints" (2018 Peer Review Report, p. 5).[17] The Peer Review final report identified important limitations with the study set, including that there were no studies of a US population and a small number of studies. To address these limitations, the Peer Review final report recommended that EPA take the following steps, all of which EPA disregarded in this proposed rule:

- Use more studies;
    - *What the Peer Reviewers recommended*: The Peer Reviewers recommend using additional studies to improve the information that is used for the quantitative assessment of the relationship between maternal thyroid hormone levels (fT4) and neurodevelopmental outcomes. Using more studies will tend to address individual study limitations. For example, Peer Reviewers noted that the five studies that EPA proposed to use all used different ways to assess urinary iodine concentrations, making it difficult to interpret iodine status in a given individual. "Iodine status is much more reliably measurable on a population level". [18]
    - *What EPA did*: EPA used its own analysis of a single study, Korevaar et al (2016). Relying on a single study set means that any errors, limitations, or biases with the study design or results will have a much greater influence on the final risk predictions. For example, Korevaar et al (2016) assessed urinary iodine on only a subset of 672 women, and the study was conducted in the Netherlands. Peer Reviewers expressed reservations about its use as the sole data source. As one reviewer stated, "whenever a single study is used there is uncertainty whether the study population adequately represents other populations of interest/concern."[19]
- Use studies that address more adverse outcomes;
    - *What the Peer Reviewers recommended*: The Peer Review final report recommends that EPA include additional studies with a broader range of neurodevelopmental outcomes, including for example ADHD and autism "as they are now realized to be associated with early insufficiencies of thyroid hormone". [20]

- *What EPA did*: EPA based its proposed rule on only a single endpoint, loss of IQ points. This fails to address all the neurodevelopmental outcomes of interest, and is not sensitive enough to address risks of ADHD and autism, both of which can occur in the absence of measurable IQ loss. [21]

Despite the Peer Reviewer recommendations in response to EPA's charge question on how to address limitations with the studies – use more studies, more data, and more endpoints of concern - EPA's proposed rule goes in the opposite direction, using a single study and a single outcome (IQ loss).

<u>EPA relied on an insensitive endpoint from a least-sensitive study</u>

EPA and its expert peer reviewers identified five epidemiologic studies that it determined met the criteria for use in dose-response modeling of the relationship between maternal thyroid hormone levels (fT4) and offspring neurodevelopment. EPA used the upper bound of the 95th percent confidence interval (95%CI), either extracted directly from the study, or derived with additional analysis (84FR 30532). In the FR Notice, Table III-2 EPA lists the studies it has identified as options to use for dose-response modeling, and EPA's derived dose-response for a 1%, 2% and 3% decrease in the specified (study-dependent) neurodevelopmental effect and corresponding perchlorate dose.

Despite putting what must have been a very large commitment of time and resources to evaluate five studies, and generate seven dose-response analyses, EPA selected the second least protective analysis (Korevaar et al, 2016, with EPA additional analysis) as the sole basis for its proposed MCL/MCLG. And, compounding EPA's failure to protect the public, it opted to use a 2% IQ loss (that is, a loss of 2 IQ points), instead of a more protective 1% loss.

EPA explains its choice to use the Korevaar study and the endpoint of IQ loss; the study was large, adequately addressed potential confounders, and used an appropriate endpoint that is "straightforward to interpret" (84FR 30534). However, EPA provides no discussion for why it didn't pool data from multiple studies, especially those that also evaluated IQ loss, to generate a meta-analysis, as recommended by the expert Peer Reviewers (discussed above). By relying on a single study, EPA failed to follow best available science as recommended by its expert peer reviewers, thereby weakening the scientific basis for the MCL and giving the appearance of 'cherry picking' the scientific evidence. Minimally, this introduces uncertainties into EPA's analysis that must be addressed with appropriate UFs (discussed below).

EPA selected a 2% loss of IQ (which is permanent over a lifetime) as its point of departure. The EPA Benchmark Dose Guidance (2012) states, ''[a] BMR of 1% has typically been used for quantal human data from epidemiology studies'' (USEPA 2012; 84 FR 30536).[22] Thus, EPA failed to follow its own Guidance. EPA's rationale is that it, "made a policy decision to use a 2 IQ point decrement in the population distribution of IQ for the sensitive population" (84 FR 30536). EPA does not justify this departure from its Guidance, or its decision to select a less protective endpoint, or identify any written policy upon which it based its decision to select a less-protective adverse effect level. A point of departure of 2-point IQ loss will not prevent adverse impacts in vulnerable populations.

This therefore is contrary to the requirements of the SDWA, which as noted earlier requires EPA to establish an MCLG at a level at which *no known or anticipated adverse effects* will occur, *with an adequate margin of safety.* As the loss of even 1 IQ point is an effect level, not a NOAEL, it is inappropriate to use as a departure point under the plain meaning of the statute. EPA must use the

most sensitive endpoint as its point of departure. Additionally, at a minimum and in the alternative, its lack of protection should be addressed with appropriate UFs as described below.

EPA failed to follow established risk assessment best practices to address population variability

The preferred way to assess population risk is to replace the single-point factors with a series of realistic distributions to represent likely real variability in the factors now represented by single-point. Had EPA employed best-practices, it would have generated realistic distributions, and then the overall implications of these distributions for population risks can then be assessed using a "Monte Carlo" analysis which uses thousands of individual "trials" of random values selected from the distributions to fairly and transparently explore the likely distribution of population outcomes for IQ and/or other affected outcomes or the population of exposed fetuses and young children. This type of methodology for addressing and reducing variability between individuals (intraspecies variability) is now reasonably standard in risk assessment. The EPA analysis as it stands is arbitrary and cannot substitute for a serious variability-based analysis.

The variability between individuals across a population supports the intraspecies UF of at least 10X.

Uncertainty in the toxicity database, toxicokinetic information, and dose-response relationship

EPA identified numerous areas of remaining uncertainty, particularly in the dose-response relationship between exposure and neurodevelopmental outcomes (84 FR 30537). This include but are not limited to:

- "There are very few toxicokinetic calibration data available for the perchlorate to thyroid hormone relationship described in the BBDR model." This is a significant uncertainty, given how much the model depends on this relationship. Too few data points makes it more difficult to detect a link between perchlorate and thyroid levels at low doses, thus leading to an underestimate of risk.
- EPA identified uncertainties in the toxicodynamic side of the BBDR model that include:
    - lack of data on competitive inhibition between perchlorate and thyroid hormone at the NIS (sodium iodide symporter), which plays a fundamental role in thyroid function by mediating the transport of the iodine molecule (anion) into the thyroid (follicle) cells;
    - rate of depletion of stored iodide under different physiological states;
    - clear data-driven descriptions of compensation mechanisms by the TSH feedback loop.

  Lack of data makes it more difficult to identify measurable impacts at low doses of perchlorate, leading to an underestimate of risks at low doses.
- EPA identified uncertainties linking maternal free T4 (fT4) to offspring IQ effects. This includes that there is no US-based study and a "lack of information on the iodine intake status of the population for which the dose-response information is available". This was also a concern of the Peer Reviewers and is relevant given that diet, iodine-consumption, and other factors are likely to differ significantly across geographic regions, cultural practices, and dietary trends.
- EPA identified uncertainties around the methods used to assess maternal fT4 during pregnancy. Less sensitive methods will underestimate risk.

There are also uncertainties that EPA has identified regarding the, "lack of information linking incremental changes in infant thyroid hormone levels to adverse neurodevelopmental outcomes" (84 FR 30537). Lack of data for this relationship will lead to an underestimate of risk at lower exposures, and

for neurodevelopmental outcomes like autism and ADHD, which can occur without any measurable loss of IQ.[23]

EPA acknowledged this database deficiency: "The difficulty in estimating the likelihood and magnitude of the potential implications of perchlorate's mode of action on expressed neurodevelopmental health effects in humans exposed to perchlorate during development is the lack of robust epidemiological studies" (84 FR 30527) These database deficiencies support the use of additional UFs, discussed below.

While we generally agree with EPA that the fetus may be highly sensitive to small short-term fluctuations in iodine availability, given its limited ability to store iodine compared with an adult (FR p. 30537), this is a much more complicated issue with many data gaps and uncertainties in our understanding. First, up until week 12 or so, the fetus can't concentrate iodide, so what really matters is maternal fT4. Second, in contrast, the newborn must have adequate dietary iodine because it must synthesize the T4 it needs each day. With no hormone storage and a half-life of fT4 only about three days (instead of 10-12 in adults), the neonate is highly vulnerable to low iodine. These complexities contribute to uncertainties in the impact of even transient or low-levels of perchlorate exposure during later gestation when the fetus is reliant on its own ability to produce thyroid hormones. As EPA notes, "the immature fetal HPT axis has very limited capacity to increase output of thyroid hormones … so the [fetus] may not be able to adjust output in the face of reduced maternal fT4 supply and perchlorate exposure" (FR, p. 30537). Failing to model the potential harm from small or brief exposures to perchlorate during second and third trimester fetal development will overlook, and therefore underestimate risk during vulnerable life stages.

These and other database deficiencies support the use of additional UFs, discussed below.

Failure to include an adequate margin of safety will ensure that the proposed MCL will not be public health protective.

EPA's proposed MCLG is improper because it does not include the "adequate margin of safety" mandated by SDWA § 1412(b)(4)(A), which requires that EPA set a MCLG "at the level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety." Because SDWA does not define "margin of safety," the words take their plain meaning. Smith v. United States, 508 U.S. 223, 228 (1993) ("When a word is not defined by statute, we normally construe it in accord with its ordinary or natural meaning."). The plain meaning of "margin" indicates a gap, buffer, or distance between two things. Thus, the "margin of safety" SDWA § 1412(b)(4)(A) requires is a gap between the no-adverse-effect level and the MCLG. Specifically, EPA must set the MCLG some measure below the no-adverse-effect level.

Additionally, as the legislative history of the Act makes clear, establishment of the MCLG is to involve 4 steps. First, the known adverse effects are to be compiled. Second, EPA is to consider whether there may be any adverse effects that "can be reasonably anticipated, even though not proved to exist." Third, EPA "must consider the possible impact of synergistic effects, long-term and multi-media exposures, and the existence of more susceptible groups in the population." And finally, EPA must establish the MCLG at a level "set to prevent the occurrence of any known or anticipated adverse effect. It must include an adequate margin of safety, unless there is no safe threshold for a contaminant. In such a case, the [MCLG] should be set at the zero level." H.R. Rept. 93-1185 part 2, 93rd Cong. 2d Sess. 1974, at 20.

Finally, consistent with the previous legislative history, in 1996 the statute was amended to explicitly require that this margin of safety is to consider not just the general population, but the most vulnerable subpopulations. As the Act states, the agency is required to consider the "effects of the contaminant on the general population and on groups within the general population such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other subpopulations that are identified as likely to be at greater risk of adverse health effects due to exposure to contaminants in drinking water than the general population." SDWA §1412(b)(3)(C)(i)(V). Thus, the MCLG must include a margin of safety not just for the general population but for subpopulations of individuals who may be at greater risk.

Intraspecies UF at least 10X is justified – EPA reduced it to 3, failed to follow its Guidance

EPA has reduced the standard 10X uncertainty factor to adjust for human-to-human variability (intraspecies UF) to 3X, despite noting that its own Guidance say that this factor can only be reduced from 10 if, "data are sufficiently representative of the exposure/dose-response data for the most susceptible population(s)" (84 FR 30537, USEPA 2002, p. xvii). In fact, the full paragraph from which the FR Notice is quoting emphasizes that it should only be reduced from 10 only in very extreme cases:

> "In general, the Technical Panel reaffirms the importance of this UF, recommending that reduction of the intraspecies UF from a default of 10 be considered only if data are sufficiently representative of the exposure/dose response data for the most susceptible subpopulation(s)."[24]

EPA failed to present a convincing data-supported rationale for reducing the intraspecies UF. EPA says it reduced the UF to 3, "because the modeled groups within the population that are identified as likely to be at greater risk to perchlorate in drinking water (i.e. the fetus of the iodide deficient pregnant mother) and has selected model parameters to account for the most sensitive individuals in that group (i.e. muted TSH feedback, low fT4 values, low-iodine intake)." (84 FR 30537). In fact, EPA's own analysis establishes the opposite – that EPA's model has many uncertainties with regard to model parameters including fT4 values, iodine intake, and low-dose parameters (84 FR 30537):

- "There are very few toxicokinetic calibration data are available for the perchlorate to thyroid hormone relationship described in the BBDR model.";
- "The ability of the TSH feedback loop to compensate for perturbations in thyroid function each have their own uncertain features";
- "uncertainties around the methods used to assess maternal fT4 measurements during pregnancy";
- "uncertainties related to the true distribution of fT4 for a given iodine uptake".

EPA acknowledges that the revised BBDR model fails to address the impact of other goitrogens, including nitrate and thiocyanate, a critical data gap that was discussed at length by the 2013 SAB (p. 21-22), and raised again by the Peer Reviewers in 2018 (pages 15, 45, 55). EPA wrongly supposes that this may be indirectly addressed in the BBDR model because "the model predictions for zero perchlorate exposure are calibrated to [NHANES] data, which are from a population with an exposure distribution to other goitrogens that is assumed to be independent of perchlorate". [25] However, this is a mistaken assumption, since the NHANES data do not explicitly address this data gap, are a limited random sampling so cannot be expected to include localized exposures, and do not include data from infants. Peer Reviewers noted that this remains, "a limitation of any exposure model"[26] It is odd that both the

2013 SAB and the 2018 Peer Review discusses this data gap , and suggests potential data sources that EPA could have used for thiocyanate (2018 Peer Review, p. 55; 2013 SAB p. 21-22), but EPA did not do this.[27] This remains a significant data gap, and uncertainty. The SAB noted that, "The contributions to NIS inhibition from other NIS inhibitors (e.g., thiocyanate, nitrate) could also be incorporated in the modeling, but may be addressed as qualitative uncertainties at this time"(SAB, p. 18).

Additionally, we describe above that EPAs population variability analysis has not followed standard best practices, and as it stands is arbitrary and cannot substitute for a serious variability-based analysis.

These and other uncertainties discussed in these comments and the FR Notice directly contradict EPA's stated rationale for reducing the intraspecies UF. Additionally, the model does not address the limitations pointed out by the Peer Reviewers (discussed above) that EPA relied on limited data from a single study in a non-US population for many of these physiological parameters.

Moreover, EPA Guidance identifies situations where an UF of greater than 10 may be justified: "At the other extreme, a 10-fold factor may sometimes be too small because of factors that can influence large differences in susceptibility, such as genetic polymorphisms" (underline not in original).[28] Although EPA does not mention it in the FR Notice, there are almost 100 genetic polymorphisms in the NIS gene.[29] How all these genetic polymorphisms may affect the critical role of the NIS in thyroid hormone synthesis is a very significant data gap. This is especially relevant given that one of the many uncertainties that EPA identified is the lack of data on competitive inhibition between perchlorate and thyroid hormone at the NIS. How large is the variation in this critical model parameter across individuals when the almost one hundred different variations in the gene are also considered? An intra-human UF of more than 10X may be warranted.

An additional UF of 3 for use of a LOAEL is justified – EPA reduced it to 1, failed to follow its Guidance

EPA eliminated the UF factor (reduced from 10X to 1X) for the use of a LOAEL (lowest observable adverse effect level) instead of a NOAEL (no observable adverse effect level). EPA's rationale is that it has used a more sophisticated BBDR model of changes in IQ extrapolations using linear regression so that the Point of Departure "would not be expected to represent an adverse effect" (84 FR 30538).

EPA's use of a measurable IQ deficit – whether 2 IQ points as EPA proposes, or even 1 IQ point loss, is an adverse effect in an individual and across a population (see economic arguments below). Further, EPA's approach will not address adverse effects that occur in the absence of measurable IQ loss, such as ADHD and autism (discussed above). The statute and legislative history are clear: if there is evidence of a known or anticipated adverse effect, the MCLG must be established at a level that ensures that such adverse effect will not occur, with an adequate margin of safety. EPA's selection of a 2 IQ point loss and failure to consider a 1 IQ point loss, and failure to consider other adverse effects such as ADHD and autism, is a manifest failure to comply with the SDWA's commands. The agency must establish an MCLG that ensure that no such adverse effect would occur, with an adequate margin of safety.

EPA's Guidance, referenced in the FR Notice and discussed above, says, "a UF (default 10) is typically applied to the LOAEL when a NOAEL is not available. The size of the LOAEL-to-NOAEL UF may be altered, depending on the magnitude and nature of the response at the LOAEL"[30] EPA often reduces the LOAEL UF to 3X when it employs a biological model that it believes can partially address aspects of absorption, distribution, metabolism, or excretion (ADME) information relevant to modeling a dose response

relationship. However, by eliminating it altogether – as EPA has done in this proposed rule – EPA is failing to follow its own practices and policy Guidance.

EPA should re-instate an UF of at least 3X for LOAEL-to-NOAEL extrapolation, consistent with its policies and practices, and acknowledging that any loss of IQ points is an adverse effect.

Database UF of at least 10X should be applied, due to acknowledged study limitations, model uncertainties, and data gaps

EPA states that it removed the Database UF (reduced from 10 to 1X) because, "the mode of action of perchlorate toxicity is well understood" (84 FR 30538). However, this is a misreading of EPA's Guidance, which is not about whether or not the mechanism of toxicity is understood, but whether there are or are not deficiencies in the toxicity data set including magnitude of toxicity, and considerations of toxicity at vulnerable life stages.

"The database UF is intended to account for the potential for deriving an underprotective RfD/RfC as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a lower reference value might result if additional data were available. Consequently, in deciding to apply this factor to account for deficiencies in the available data set and in identifying its magnitude, the assessor should consider both the data lacking and the data available for particular organ systems as well as life stages." (underline added for emphasis) [31]

In these comments we identify data gaps and data uncertainty for critical toxicity components of EPA's model and analysis. Many of these were identified by EPA in this FR Notice (84 FR 30537 and discussed in these comments), by the SAB (2013), and by the Expert Peer Reviewers (2018). For example, EPA states, "The difficulty in estimating the likelihood and magnitude of the potential implications of perchlorate's [toxicological] mode of action on expressed neurodevelopmental health effects in humans exposed to perchlorate during development is the lack of robust epidemiological studies" (84 FR 30527) The uncertainty is not in whether perchlorate affects thyroid hormone – it clearly does. Rather, the uncertainty is in characterizing all the conditions under which perchlorate affects thyroid hormone, and setting an MCL that is will protect the most vulnerable Americans – with an adequate margin of safety – from harm.

The main database deficiencies relevant to characterizing the toxicity of perchlorate include:

- Toxicity information is lacking to support toxicokinetic calibration of model (84 FR 30537);
- A lower reference value would likely result if additional data were available on ADHD, autism and other neurodevelopmental effects that occur without IQ loss (84 FR 30527);
- lack of data quantifying the magnitude of adverse impacts (84 FR 30527);
- lack of information relevant to linking incremental changes in infant thyroid hormone levels to adverse neurodevelopmental outcomes, including ADHD and autism and other effects that may occur at exposures below those that cause measurable IQ deficits

Some of the above data gaps and data uncertainties were introduced by EPA's choice of the two-stage model instead of the population-based model (as described above in these comments, and by EPA in the FR Notice). Some database uncertainties were introduced by EPA's choice to rely on a single study and single endpoint to model neurodevelopmental effects (as detailed above in these comments). Some uncertainties are data gaps due to the difficulty in collecting precise data on complex cognitive and

behavioral outcomes due to pre-birth exposures, and then trying to extrapolate to predict population effects.

Again, in order to comply with the SDWA and its legislative history, EPA should apply a database UF of at least 10X, given the incomplete, deficient, or absent data sets relevant to characterizing perchlorate toxicity.

Summary: EPA should apply a total UF of at least 300X – EPA's proposed 3X leaves population at risk

EPA Guidance notes that the total UFs should be limited to no more than 3000X. NRDC argues a total UF of at least 300X is justified:

- 10X for intraspecies variations, including almost 100 genetic polymorphisms in the NIS gene, which is critical for thyroid hormone production. This is a significant uncertainty in the model.
- 10X for database uncertainties, as detailed above and identified by EPA and the Peer Reviewers. Many of these data gaps will lead to underestimating toxicity due to the lack of data at low doses or for short periods of time during critical windows of development.
- 3X for using a LOAEL instead of a NOAEL, given that even a 1-2 point IQ loss is a measurable adverse effect, and will not address adverse effects that occur in the absence of measurable IQ loss, such as ADHD and autism.

EPA should apply a total database UF of at least 300X, given the incomplete, deficient, or absent data sets identified by SAB (2013), the external Peer Reviewers (2018) and EPA (84FR 30537). If EPA did this, and selected the PoD of 2%IQ loss, it would result in an MCLG of 0.55 ppb. If EPA did this, but selected the more protective PoD of 1% IQ loss, as NRDC is recommending, it would result in an MCLG of 0.18 ppb. In either case, the MCLG should effectively be zero, if EPA were to apply a total UF of 300X, consistent with EPA policies and practices. Such UFs and an MCLG at this level is critical to ensuring that no known or anticipated adverse effects would occur with an adequate margin of safety.

Below is a Table demonstrating what the resulting MCLG would be for the 3 PoD values presented by EPA, and the Total UF value of 3X proposed by EPA, and 300X proposed by NRDC. All calculations follow EPA proposed formulas, inputs and assumptions as presented in 84FR 30538, 30540. EPA is proposing an MCLG of 56 ppb (in yellow, below); NRDC is proposing an MCLG of 0 (in green, below).

Table 1: RfD with no UFs, using Korevaar et al with EPA analysis (EPA's proposal)

	PoD for 1% IQ loss ug/kg-day	PoD for 2% IQ loss ug/kg-day	PoD for 3% IQ loss ug/kg-day
UF 1X (see 84FR Table III-2)	3.1	6.7	10.8
UF 3Xintra (see 84FR30538)	1 MCLG=18 ppb (RSCw 56%)	2.2 MCLG=55 ppb (RSCw 80%) ***(EPA's proposal***)	3.6 MCLG=90 ppb (RSCw 80%)
UF 30 (3Xintra, 10Xdatabase)	0.10 MCLG=1.8 ppb	0.26 MCLG=5.5 ppb	0.36 MCLG=9.0 ppb

UF 300 (10Xintra, 10Xdatabase, 3XLOAEL)	0.010 MCLG=0.18 ppb (***NRDC's proposal***)	0.026 MCLG=0.55 ppb	0.036 MCLG=0.90 ppb

Note, for the values used in the Table above, EPA calculated MCLG values as described in 84FR 30540, Table III-5, using the following standard formula: MCLG=(RfD/DWI)xRSCw where: DWI=0.032, the 90th percentile bodyweight adjusted drinking water ingestion rate (L/kg-day) for non-pregnant, non-lactating women aged 15-44 yrs old. RSCw= the relative source contribution of drinking water to overall perchlorate exposure. Note that for RfD of 1, EPA used a 56% RSCw, whereas for subsequent RfD values, EPA used the standard 80% RSCw; EPA has not offered any explanation in the FR Notice for why it used different RSCw values.

The most vulnerable subpopulations, pregnant women and their offspring, will be permanently harmed.

As noted, SDWA § 1412(b)(4)(A) does not simply require a "margin of safety": it requires an "adequate margin of safety." Something is "adequate" when it is sufficient to accomplish a given purpose. Part of Congress's purpose in amending the Safe Drinking Water Act in 1996 was to ensure that regulations would protect vulnerable subpopulations in addition to healthy individuals. 42 U.S.C. § 300g-1(b)(3)(C)(i)(V); 142 Cong. Rec. 21,472–73 (1996) ("The language in this bill requires that EPA drinking water standards be set at levels that take into account the special vulnerability of our children, our infants, pregnant women, our elderly, the chronically ill, and other groups that are at substantially higher risk than the average healthy adult."). Thus, a "margin of safety" is "adequate" only if it provides a margin between the no-adverse-effect level and the MCLG for the general population and vulnerable subpopulations.

EPA may not reverse its regulatory determination and refuse to issue an NPDWR for perchlorate; that would violate the SDWA and a court-approved Consent Decree

On February 11, 2011, EPA scientists and policy experts issued an FR Notice with its determination to regulate perchlorate in drinking water based on evidence that it occurs in public water systems with a frequency and at levels of public health concern that present a meaningful opportunity for health risk reduction through a drinking water regulation. As illustrated by the analysis above, it is clear that this determination was well-founded. Any EPA attempt to reverse such a finding is completely unwarranted by the evidence before the agency. Moreover, there is no authority in the statute to reverse such a finding, and in any event the provisions of the court-approved Consent Decree entered into between EPA and NRDC clearly states that EPA will promulgate a National Primary Drinking Water Regulation for perchlorate. Paragraph 5 of the Consent Decree states "No later than December 19, 2019, EPA shall sign for publication in the Federal Register a final MCLG and NPDWR for perchlorate."[32] Any agency attempt to do otherwise would violate the Act and our Consent Decree.

EPA's benefits assessment fails to comply with the Safe Drinking Water Act

<u>A loss in one IQ point is estimated to be $20,419, even if one inappropriately discounts the value of future IQ losses</u>

Using EPA assumptions for monetizing the impact of IQ loss as a result of mercury exposure, a published economic analysis by EPA authors reported that using EPA assumptions, a loss of 1 IQ point is associated with a lifetime earnings loss per person of 2.38% (see Griffiths et al 2007, Table 2).[33] Using EPA's methods, in 2017 dollars, at the 95th percentile, the mean value of a change in one IQ point is estimated to be $20,419 with a 3% discount rate (EPA 2019, Table 5-15). [34]

Unfortunately, EPA's economics benefits analysis reports the total annualized benefits of avoided lost IQ decrements at only its three proposed MCL limits: 18, 56, 90.[35] The values reported by EPA are as follows (84FR 30553-30555):

Table 2: Selected relevant information from EPA's economic analysis as provided by EPA in 84FR 30553-30555

	Avoided Lost IQ Decrements (upper/central estimate)	Annualized Benefits at 3% discount (Millions: 2017$) (upper/central)	Population served by systems with perchlorate at a given MCL level (Table VI-2)	Costs 3% discount (Table XII-14)
MCL 2 ppb (no data)	*EPA fails to provide information*	*EPA fails to provide information*	*EPA fails to provide information*	*EPA fails to provide information*
MCL 4 ppb (no data)	*EPA fails to provide information*	*EPA fails to provide information*	16,172,565	*EPA fails to provide information*
MCL 18 ppb (Table XII-10)	445/250	$6.56/3.68	701,180	$15.95
MCL 56 ppb (Table XII-7)	243/136	$3.57/2.00	64,733	$9.67
MCL 90 ppb (Table XII-13)	222/124	$3.26/1.83	25,972	$9.51

As can be seen from EPA's economic analysis as presented in the FR Notice, for MCLs at 56 and 90, the costs and benefits are fairly similar, but for the MCL of 18 ppb, both the costs and benefits are roughly double that of the less protective MCL values. Unfortunately, EPA fails to show the calculated costs and benefits over a range of MCL values, and – most frustrating- at the values of 1 ppb, 2 ppb, and 6 ppb identified by States as more appropriately protective drinking water limits. Without additional information, including the population served at lower more protective MCL values, this cannot be calculated.

EPA should provide the cost and benefit values for a wider distribution range of MCL values, including at 1 ppb (the California PHG), 2 ppb (the Massachusetts regulatory limit), and 4 ppb (what EPA apparently believes is the technical feasibility limit, despite an existing, implemented state standard at 2 ppb in Massachusetts). By failing to provide these additional relevant analyses, EPA leaves the public with too little information to provide informed comments on EPA's analysis and proposed MCL.

<u>EPA should not be using a discount rate</u>

The use of a discount rate is wholly inappropriate in the case of perchlorate. Discounting future benefits such as future IQ losses essentially means that long-term harms of IQ loss (and of other losses EPA has failed to quantify but should have) are essentially zero several years into the future. Moreover, any child born several years in the future's IQ loss or other adverse effects on their brains and learning will be discounted to the point of being essentially worthless. Why are future harms, and future generations' harms worth less than harms suffered today?

The use of a discount rate runs contrary to the preventative statutory goals espoused in the legislative history including the 1974 House Report, and provisions of the SDWA. The Act includes numerous specific requirements for the cost-benefit analysis in section 1412(b). If Congress believed a discount rate was appropriate, it could have explicitly required or authorized it, and did not do so. Moreover, as discussed below, by largely failing to base any of its standard on non-monetized benefits or benefits other than IQ loss, the agency has ignored the provisions of SDWA section 1412(b).

EPA's Economic Analyses Guidelines (EPA 2010) note that, "OMB's Circular A-4 (2003) requires the use of constant 3 and 7% for both intra- and intergenerational discounting for benefit cost estimation of economically significant rules but allows for lower, positive consumption discount rates, perhaps in the 1 percent to 3 percent range, if there are important intergenerational values."[36] First, it must be noted that those guidelines are not legally binding and cannot contravene statutory requirements such as those in the SDWA. Additionally, no discount rate is appropriate in the case of a perchlorate, where an MCL that avoided lost IQ decrements and other harms to developing brains would provide an immediate and long-term measurable benefit in healthy growth, development, learning, and adult income and quality of life for both current and future generations. As noted, in the case of harm to future generations' brain development, for example, it is inappropriate, and contrary to the preventive purposes and provisions of the SDWA to use a discount rate for perchlorate.

Failure to Meaningfully Consider Non-Quantified Benefits

EPA admits that its quantitative analysis fails to consider the substantial benefits for vulnerable children and others such as reducing the risk of ADHD, autism, schizophrenia, expressive language delay, reduced school performance and numerous other adverse effects of perchlorate exposure. The agency also admits it doesn't attempt to quantify impacts on cardiovascular disease risk, total cholesterol, LDL cholesterol, and triglycerides, or the potential relationship between increases in TSH and risk of fatal coronary heart disease. These are important impacts, particularly when paired with the potential contributions to NIS inhibition from other NIS inhibitors (e.g., thiocyanate, nitrate), as noted by the SAB.

Nor does EPA's quantitative analysis consider the co-benefits of removing other contaminants such as nitrate that EPA's-designated Best Available Technologies such as Ion Exchange or Granular Activated Carbon will remove. As EPA's HRRCA admits,

> In the Office of Inspector General: Scientific Analysis of Perchlorate (USEPA, 2008b), the EPA suggested that the best approach to conduct a risk assessment for perchlorate would include all four NIS stressors acting on the thyroid: thiocyanate, nitrate, perchlorate, and lack of iodide. Perchlorate is a strong NIS inhibitor; however, exposure to humans may be relatively low. In contrast, nitrate and thiocyanate are weak NIS inhibitors; however, exposure levels to these two chemicals are much greater than perchlorate (USEPA, 2008b). Consequently, reductions of the co-occurring contaminant nitrate could lead to additional health benefits. [37]

Despite this admission, EPA make no effort to estimate any of these additional health benefits. Nor does the agency otherwise meaningfully consider these benefits after offhandedly admitting they exist.

Additionally, EPA admits that there are innumerable other non-quantified benefits, as noted in the Federal Register proposal:

> There are a number of potential benefits of reducing perchlorate in drinking water that were not quantified as part of this analysis, which may result in an underestimate of actual benefits. As described by the SAB ''children exposed gestationally to maternal hypothyroxinemia (without hypothyroidism) show reduced levels of global and specific cognitive abilities, as well as increased rates of behavior problems including greater dysregulation in early infancy and attentional disorders in childhood (Man et al., 1991; Pop et al., 1999; Pop et al., 2003; Kooistra et al., 2006)'' (p. 10, SAB for the U.S. EPA, 2013). The EPA's literature review identified potential relationships between maternal thyroid hormone alterations and the risk of schizophrenia, ADHD, expressive language delay, reduced school performance and increased odds of autism, among others, none of which are being currently quantified in this assessment. Other potentially omitted benefits include risks associated with effects of thyroid disorders in adults, including cardiovascular disease risk; changes in thyroid hormone levels and their relationship with total cholesterol, LDL cholesterol, and triglycerides; as well as a possible relationship between increases in TSH and risk of fatal coronary heart disease. Treating for perchlorate in drinking water could also potentially remove nitrate, which is a co-occurring contaminant and a goitrogen. These additional potential health endpoints are not monetized in this benefits analysis.

84 Fed. Reg. at 3057.

While it is commendable that EPA admits that it has not quantified all of these extraordinarily impactful and worrisome adverse effects, the agency cannot merely mention them and then fail to make even the slightest attempt to quantify or actually seriously consider these non-quantified benefits. In its discussion weighing benefits and costs, little more than lip service is paid to these enormous adverse impacts. For example, according to the Centers for Disease Control and Prevention, about one in 59 children have autism spectrum disorder.[38] The total costs to society of paying for the special education and innumerable other costs of assisting these children and adults has been estimated by researchers in the peer-reviewed literature at $2.4 million.[39]

Similarly, cardiovascular disease is the number one killer of Americans. CDC estimates about 610,000 people die of heart disease in the United States every year–or 1 in every 4 deaths. Heart disease is the leading cause of death for both men and women.[40] According to a peer-reviewed article in the respected journal Circulation, 121.5 million adults in the U.S., or about 48 percent based on 2016 figures, have cardiovascular disease.[41] Heart disease was the No. 1 cause of death in the U.S. and stroke was No. 5. The costs of cardiovascular disease are staggering; the American Heart Association has estimated that in 2016, cardiovascular disease cost America $555 billion, and by 2035, the cost will double to $1.1 trillion.[42] Even a small reduction in these adverse health effects would have enormous economic and other societal benefits. EPA is remiss to not at least give the implications of the impact of perchlorate on such enormous societal costs serious and in-depth consideration.

EPA makes no attempt to meaningfully explain how it has weighed these enormous potential impacts after mentioning them. A mere mention does not constitute actual analysis and sober consideration of these issues. The lack of attention to this issue is arbitrary, capricious, and a clear failure of the agency to adequately consider relevant factors as required by the APA and SDWA section 1412(b).

EPA Fails to consider "willingness to pay" for reduced risk of brain harm from perchlorate

SDWA provides that EPA is to consider "valid approaches for the measurement and valuation of benefits under this subparagraph, including approaches to identify consumer willingness to pay for reductions in health risks from drinking water contaminants." SDWA §1412(b)(3)(C)(iii). Neither the EPA Health Risk Reduction and Cost Analysis (HRRCA) nor the EPA Federal Register notice makes a mention or an attempt to conduct a willingness to pay study or even to survey the literature on willingness to pay for safer drinking water.

Consumers clearly are willing to pay more for safe drinking water, including water that has been treated at a cost to remove perchlorate and other co-occurring contaminants. Both California and Massachusetts have adopted perchlorate standards at a cost to local consumers, and it is clear that these consumers have been willing to pay for that safety and security of mind. EPA appears to have made no effort to evaluate consumer willingness to pay for safer water in either of these states, despite the obvious availability of that information and clear public willingness to fund the costs of cleaning up their water in these states.

Moreover, consumers have been voting with their wallets for bottled water and point of use filters due to concerns about the safety of tap water. Bottled water sales in the United States in 2017 topped $18.5 billion dollars[43], clearly showing consumer willingness to pay for water perceived as safer than tap water. And safety of tap water is indeed a leading reason that consumers have been turning to bottled water, according to published, peer-reviewed science. For example, an in-depth study published in 2011 found that "U.S. consumers are more likely to report bottled water as their primary drinking water source when they perceive that drinking water is not safe. Furthermore, those who give lower ratings to the quality of their ground water are more likely to regularly purchase bottle water for drinking and use bottle water as their primary drinking water source."[44]

Moreover, published willingness to pay studies have demonstrated consumer willingness to pay for safer tap water. For example, a recent consumer willingness to pay for safer drinking water study in Jacksonville, Florida was published in a peer-reviewed journal by Florida researchers.[45] After an environmental group had published a study suggesting contamination of the local water supply with certain contaminants (at levels below EPA standards), researchers surveyed local residents as to how much they would be willing to pay to "improve the quality of your water?" The researchers found that the average consumer was willing to pay $6.22 per month for improved quality of their water, or about $75 per year, with no violations of standards alleged or any official indication that the water was unsafe. Indeed, the local water utility vehemently publicly denied that there was any significant health risk from the contamination. Thus, if there were a violation of a federal standard, and associated authoritative statements about the health risks posed by the contamination, it would be reasonable to assume the willingness to pay would have been substantially higher.

This $75 per year average willingness to pay for water that would be of better quality but was not necessary to comply with a federal health standard is substantially higher than EPA's estimated cost of even the most stringent standard the agency considered of 18 ppb, which the agency estimated would

cost an average household about $38 to $46 dollars. 84 Fed. Reg at 30553. And clearly the cost to households served by larger systems with the economies of scale would be lower (more like $18-$24/year). Id.[46]

It is also worth noting that the Jacksonville Florida study's findings are confirmed by other published, peer-reviewed studies making similar findings in other locales. This is so even in lower income communities, such as Parral, Mexico where researchers determined "households are willing to pay from 1.8% to 7.55% of reported household income above their current water bill for safe and reliable drinking water services."[47] Similarly, in Bangladesh, consumers were willing to pay for water that contained safe levels of arsenic, despite the endemic poverty in the region. The authors of this peer-reviewed, published paper found that "Regardless of economic class, most of the households (75%) were willing to pay" the equivalent of "2–6% of their respective monthly income to access safe drinking water."[48]

These academic findings are readily confirmed by actual evidence in states that have adopted stricter standards. For example, Massachusetts' standard of 2 ppb and California's standard of 6 ppb have been implemented for several years, with no reports of consumer outrage or refusals to pay for safer drinking water that is not contaminated with excessive levels of perchlorate. This is the strongest evidence that there is a consumer willingness to pay for a strict drinking water standard for perchlorate of 2 ppb as we propose. The agency's utter failure to consider evidence regarding a key statutory criterion for assessing the benefits of adopting a drinking water standard of 2 ppb (or, in California, 6 ppb) is arbitrary, capricious and contrary to law.

Conclusion

EPA's proposal for this hazardous drinking water contaminant found in the drinking water of millions of Americans is deeply troubling. It would leave fetuses and young children largely unprotected and would threaten the health of older children and adults. The agency's proposal to set a perchlorate Maximum Contaminant Level (MCL) at 56 ppb, is nearly 4 times what EPA said is safe in its previous Lifetime Health Advisory and ignores the best available science and advice of its own SAB peer reviewers. The agency's suggestion that it may set a standard as high as 90 ppb, or might set no standard at all, is completely unsupported by the science and clearly would violate the law.

State health officials have undertaken detailed assessments of the studies of perchlorate and set drinking water standards based on the best available science of 2 ppb (in Massachusetts) and 6 ppb (in California).Additionally, in its 2015 reevaluation, California issued a revised Public Health Goal of 1 ppb, based on updated values of infants' water consumption, and new evidence that even very small reductions in thyroid hormones or iodine levels could cause significant adverse impacts on offspring brain development and function.

The agency's proposed MCLG is legally required to protect vulnerable populations from any known or anticipated adverse effects with an adequate margin of safety. Instead of following this statutory mandate, EPA proposes to set the MCLG based on the level of perchlorate that would cause a loss of 2 IQ points. The agency ignores that even a 1% decrease in IQ is also an adverse effect and has failed to use an adequate margin of safety as required by SDWA.

EPA's analysis of benefits fails to consider many of the non-quantified benefits that are likely to accrue from a stringent MCL for perchlorate, including many health benefits for children such as reduced

likelihood of ADHD and autism, as well as many other benefits for children and adults. The agency also failed to consider the consumer willingness to pay for better drinking water, a factor EPA is required to consider under the Act, and the co-benefits of reduced nitrate and other contaminants from treating for perchlorate.

For the reasons discussed above, we recommend that EPA scrap the proposal and establish an MCLG of zero, and an MCL of 2 ppb.

Respectfully,

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[2] Massachusetts Department of Environmental Protection DRAFT REPORT: The Occurrence and Sources of Perchlorate in Massachusetts, Updated April, 2006, available online at https://www.mass.gov/files/documents/2016/08/ra/percsour.pdf, accessed August 25, 2019; EPA, Office of Pesticide Programs, Hypochlorite, Calcium Hypochlorite, and Potassium Hypochlorite: Interim Registration Review Decision: Case Numbers: 0029 and 5076, March 2018, available online at https://www.regulations.gov/document?D=EPA-HQ-OPP-2012-0004-0032 (both incorporated by reference). While EPA suggested label precautions to potentially minimize perchlorate levels in hypochlorite, the agency admitted that "The practicality of these label amendments is based on the varying feasibility of different drinking water utilities to implement the suggested best management practices, such as geographic location and facility logistics; these label amendments are, therefore, advisory." Id. at 19. Thus, there is no enforceable assurance that perchlorate levels from hypochlorite use will be reduced, absent a protective MCL enforceable at the tap.

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[19] Final Peer Review Report - Perchlorate MCLG Approach. March 2018. Comments of Stephen Roberts, p. 84

[20] Final Peer Review Report - Perchlorate MCLG Approach. March 2018, p. 5

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