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EPN Comments on
National Primary Drinking Water Regulation for Perchlorate
Docket ID No. EPA-HQ-OW-2024-0592
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The Environmental Protection Network (EPN) harnesses the expertise of more than 750 former Environmental Protection Agency (EPA) career staff and confirmation-level appointees from Democratic and Republican administrations to provide the unique perspective of former regulators and scientists with decades of historical knowledge and subject matter expertise.

Introduction

EPA is proposing to set the Maximum Contaminant Level Goal (MCLG) for perchlorate at 20 µg/L and an enforceable Maximum Contaminant Level (MCL) of 20 µg/L, 40 µg/L, or 80 µg/L. In addition, EPA is proposing requirements for water systems to monitor for perchlorate in drinking water; take mitigation actions if concentrations exceed the MCL; provide information about perchlorate for their customers through public notification and consumer confidence reports; and report to their respective primacy state agencies. The preamble states that the Administrator has determined the benefits of this regulation would not justify the costs, however, EPA is required to issue this rule in response to the D.C. Circuit's decision in NRDC v. Regan[1].

An MCLG is based on a reference dose (RfD). EPN will critique the RfD EPA has proposed for perchlorate and the MCLG EPA derived from it. An RfD is defined as 'the estimated maximum daily dose of perchlorate that can be consumed by humans for an entire lifetime without toxic effects.' EPA has calculated the RfD from a model-based estimate formulated from the known action of perchlorate to block the sodium-iodine transporter (NIS). This action at NIS reduces iodine uptake into the thyroid gland and disrupts thyroid hormone synthesis. The RfD presented by EPA is the result of an innovative two-step approach, coupling a Pharmacokinetic Perchlorate *Exposure* model with a Biologically Based Dose *Response* (BBDR) model using human data. Although the approach is novel, the development of a BBDR model laudable, and one external review was conducted, the model itself has not been formally validated nor has it received the scrutiny of peer review in the scientific literature.

EPA is now presenting for public comment in the Federal Register Notice (FRN), an MCLG based on a Point of Departure (POD) derived from a non-validated unpublished model, and a subsequent RfD calculation that has not undergone any external scientific peer review.

As far as EPN can discern, EPA has never issued a Response to Comments document following the 2018 review panel report, so we have no clear idea as to what, if any, alterations the Agency may have made as a result of the feedback received. EPN identifies a number of deficiencies in the proposed rule's uncertainty analysis that have significant impact on the RfD. EPN will bolster their support for a lower RfD citing additional recent mechanistic data from animal studies not available in earlier perchlorate assessments or taken into consideration in the current proposal. These data establish clear effects on fetal thyroid function in the absence of direct reflection in

[1] *NRDC v. Regan*, 20-1335 (D.C. Cir. 2023)

the maternal or the fetal serum hormone measures. EPN will also cite data demonstrating structural defects in the brains of offspring of rat dams exposed to perchlorate, data that were not available for consideration in earlier evaluations of animal data. Importantly, these defects in brain structure resulting from maternal exposure to perchlorate were greatly exacerbated under conditions of iodine deficiency.

Background Information

A lengthy history of actions exists on EPA's efforts to determine an acceptable protective health-based maximum concentration of perchlorate in drinking water.

In December of 1998, EPA published its first external review draft of the health and ecotoxicological assessments and proposed a harmonized RfD. Peer review a few months later endorsed the mode of action of iodine inhibition to disrupt thyroid hormones. In 2002, a second public peer review was published with an RfD of 0.03 µg/kg/day based on perchlorate's inhibition of radioactive iodine uptake (RAIU) into the thyroid in a study of n=7 healthy male and female adult volunteers[2] and animal studies of thyroid hormones, thyroid histopathology, and developmental neurotoxicity, and the subsequent application of an uncertainty factor (UF) of 10X for intrahuman variability, a UF of 10X for rodent studies, and an additional UF of 3 for database deficiencies. In 2003, a review by the National Academy of Science (NAS) was requested.

In 2005, the report from NAS review returned an RfD of 0.7 µg/kg/day which was subsequently adopted by EPA.[3] The RfD was based on the same Greer *et al.* study described above reporting a no-observed effect level (NOEL) of 7 µg/kg/day for perchlorate's inhibition RAIU and application of a single UF of 10 for intraspecies variability.

In 2008, based on the National Research Council-derived RfD of 0.7 µg/kg/day, EPA published a Health Advisory of 15 µg/L to provide guidance to state and local officials using the then current EPA default adult body weight (70 kg) and drinking water consumption rate (2 L/day) and a perchlorate-specific relative source contribution (RSC) from water of 62% for a pregnant woman.

In 2012, building on these previous analyses, EPA determined a range of potential MCLGs based on life stages that ranged from 2 µg/L for bottle-fed infants to 18 µg/L for women of child-bearing age. The 2 µg/L MCLG is consistent with the 2015 Public Health Goal of 1 µg/L for bottle fed infants derived by The Office of Environmental Health Hazard Assessment, California EPA.[4]

In 2019, to focus on the population of concern, EPA developed a two-step approach for modeling the neurodevelopmental effects in offspring of women who are exposed to perchlorate

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[2] Greer MA, Goodman G, Pleus RC, Greer SE. Health effects assessment for environmental perchlorate contamination: the dose response for inhibition of thyroidal radioiodine uptake in humans. Environ Health Perspect. 2002 Sep;110(9):927-37. doi: 10.1289/ehp.02110927. Erratum in: Environ Health Perspect. 2005 Nov;113(11):A732. PMID: 12204829; PMCID: PMC1240994.

[3] NRC. (2005). Health implications of perchlorate ingestion. Washington, DC: National Academies Press.

[4] California Environmental Protection Agency. Office of Environmental Health Hazard Assessment, Pesticide and Environmental Toxicology Branch. 2015. *Perchlorate in Drinking Water.* https://oehha.ca.gov/water/chemicals/perchlorate

in early pregnancy. This approach contained two main components: (1) a BBDR model of how perchlorate's inhibition of iodine uptake into thyroid gland affects thyroid hormone production in early pregnancy in women with low iodide intake; and (2) a pharmacodynamic model, which describes the relationship between decreased thyroid hormone (thyroxine [T4]) in early pregnancy and later neurodevelopmental effects (e.g. IQ decrease) in the offspring.

In 2026, based on the output of these models, EPA derived a new POD of 3.1 µg/kg/day and a new RfD of 1 µg/kg/day based on a decrease of 1 IQ point in offspring of pregnant women with low iodine intake, free thyroxine (fT4) levels, and intact thyroid-stimulating hormone (TSH) feedback effectiveness. EPA now proposes to set the MCLG for perchlorate at 20 µg/L, 40 µg/L, or 80 µg/L.

In the sections below, EPN will provide comments on key elements of the proposed rule, a rule based on an unvalidated, unpublished model. EPN will focus on the woefully inadequate appraisal of the uncertainty factors applied to EPA's model-derived POD of 3.1 µg/kg/day. EPN will also provide comments on EPA's choice of the default 'ceiling' value of the RSC. In the past, this component of the MCLG calculation has ranged from 20-62% of exposure coming solely from drinking water. In the present assessment, EPA has chosen a value of 80% which significantly increases the calculated value of the MCLG.

RfD Derived from Two-Stage Model

The novel EPA two-stage model approach advances the science beyond that available when the RfD was previously developed based upon iodide uptake inhibition in an underpowered study of n=7 adult male and female volunteers.[5] This study was not designed for the purposes of setting a safe level of exposure, but rather to derive a rate constant to parameterize the original rodent-based pharmacokinetic model to extrapolate to humans. The current RfD focuses more appropriately on neurodevelopmental outcomes in children of exposed pregnant women. As such, it is a reasonable alternative approach that informs about the potential for effects in pregnant women, which Greer *et al.* clearly does not. However, despite these improvements, the new model is fraught with uncertainties that have not been adequately addressed. EPN contends that given the complexity of the hybrid models, the limitations of IQ as an endpoint, the restriction to consideration only of early pregnancy, and the meager quantity of available human data, the total UF of 3 selected by EPA to yield an RfD of 1 µg/kg/day is neither appropriate nor sufficient. Furthermore, although this complex modeling approach has undergone one external peer review in 2018,[6] a number of issues raised during that review have not been formally addressed. Neither is there evidence that the derivation of the RfD based on that model has ever been formally peer reviewed. EPN summarizes the rationale for application of two 10X UF, one for Intrahuman and one for a toxicokinetic/toxicodynamic Modifying Factor as described below.

Uncertainty Analysis Based on Human Variability:

[5] Greer, M. A., Goodman, G., Pleus, R. C., & Greer, S. E. (2002). Health effects assessment for environmental perchlorate contamination: the dose response for inhibition of thyroidal radioiodine uptake in humans. *Environmental health perspectives*, *110*(9), 927–937. https://doi.org/10.1289/ehp.02110927

[6] EPA-HQ-OW-2016-0439-0012.

This UF (UF_H) is applied to ensure protection of sensitive subpopulations. EPA's default UF for Human Variability is 10, to account for both pharmacokinetic and pharmacodynamic differences. And yet EPA picked 3. Why just 3? The justification for selection of an UF of 3 was never clearly articulated by EPA and requires a well-reasoned, well-supported, and appropriately documented rationale. Despite the fact that the POD was derived from human data on the developmental effects of children born to women exposed to perchlorate, an UF of 3X based on a limited empirical model output is not sufficient to account for *pharmacokinetic* differences among pregnant women or fetal kinetics. EPN agrees that this component of 3 for pharmacokinetic uncertainties is warranted. However, pharmacodynamic differences were not adequately incorporated into the derivation of the RfD. The pharmacodynamic portion of the two-step model was limited to fitting data during the first trimester of pregnancy. EPA deemed this to be 'the most sensitive period' for the potential effects of perchlorate exposure and concluded that no additional uncertainty factor was warranted to account for possible *pharmacodynamic* differences. EPN disagrees and contends this UF of 3 is woefully inadequate and should be increased based on the following reasoning:

1) The use of data from Korevaar *et al.*[7] as the critical study results in a *de facto* assumption by EPA that the first trimester is *the most sensitive stage* for thyroid hormone to impact IQ function in the continuum from conception to birth. At this early stage of pregnancy, the fetus is solely dependent on the mother for its thyroid hormone. As pregnancy progresses, the fetal thyroid gland is formed and begins to function and contribute to the hormonal needs of the developing fetal brain. It is in these later stages of pregnancy, as the full complement of fetal thyroid function matures, that the neurological substrates underpinning brain functions assessed in standard tests of IQ are formed. As such there is a complete disconnect of what is measured, when, and in whom (mother versus fetus), and outcome dependent on that measured change. This was a significant challenge also raised by external reviewers in 2018.[8]
2) Restricting the analysis to the first trimester of pregnancy also fails to consider the direct effects of perchlorate on the fetal thyroid gland that develops as pregnancy progresses into the second and third trimesters. The current model "accounts" for the fetus only as a marginal increase in maternal body weight. EPA's analysis assumes that protecting a first trimester fetus from alterations in maternal fT4 will protect the fetus throughout pregnancy, an assumption also challenged in the External Peer Review in 2018.[9] At around week 12 of pregnancy, the fetal thyroid gland is formed and as it matures begins to synthesize and release hormones necessary for fetal growth and development, reducing reliance on maternal sources.[10] EPA's restriction to first trimester pregnancy fails to consider the combined action of perchlorate on the maternal and fetal thyroid gland as pregnancy progresses and the subsequent impact

[7] Korevaar, T. I., Muetzel, R., Medici, M., Chaker, L., Jaddoe, V. W., de Rijke, Y. B., Steegers, E. A., Visser, T. J., White, T., Tiemeier, H., & Peeters, R. P. (2016). Association of maternal thyroid function during early pregnancy with offspring IQ and brain morphology in childhood: a population-based prospective cohort study. *The Lancet. Diabetes & Endocrinology,* 4(1), 35–43. https://doi.org/10.1016/S2213-8587(15)00327-7
[8] EPA-HQ-OW2016-0439-0012.
[9] *Id.*
[10] Rovet J. F. (2014). The role of thyroid hormones for brain development and cognitive function. *Endocrine development*, *26*, 26–43. https://doi.org/10.1159/000363153

on neurodevelopment.[11] Because generating direct human data relating fetal hormone levels with developmental outcomes is not ethically possible, EPA, as in the past, should consider use of animal data when informative as a surrogate or complementary. Recent rodent studies reveal that perchlorate administered through the maternal drinking water passes through the placenta to the fetal serum via the same transporter system present in the thyroid gland and in sufficient quantities to disrupt thyroid hormone synthesis in the fetal thyroid gland.[12]

In view of these uncertainties, as currently applied, the two-step model is likely to underestimate the potential effects of perchlorate exposure on the fetus and neonates. At a *very minimum*, EPN strongly recommends an increase from EPA's 3X value to the full 10X intrahuman UF (UFH) to account for intrahuman variability.

Application of a Chemical Specific Modifying Factor Based on Uncertainties in Data Used to Derive the POD

A modifying factor (MF) is applied to account for deficiencies in the data available for parameterization of the complex two-step model and level of confidence in its application. EPN identifies the following concerns that warrant consideration of this additional factor to capture uncertainty not reflected in the UF for intrahuman variability.

1) Uncertainties arise when modeling the relationship of depletion of iodide stores from perchlorate-induced NIS inhibition under different iodine intake levels and physiological states. There are very limited data available to calibrate the pharmacokinetic aspects of the model, particularly during the first trimester of pregnancy. As such, estimates of perchlorate and iodide absorption, metabolism, and excretion remain uncertain. Neither are there data to calibrate the joint effect of varying perchlorate and iodide serum concentrations on thyroid uptake of iodide and subsequent impact on production of T4 hormone levels from onset of pregnancy until gestation week 16.
2) Uncertainty also exists in the critical mechanistic characteristics of the effect and use of an empirical model to address the magnitude of the impact of iodine insufficiency on the relationship of maternal fT4 levels and offspring IQ as it relies *exclusively* on one study. The modeled study population was in the Netherlands and a comparable dataset on maternal fT4 and childhood IQ is not available for a U.S. population. The Netherlands represents a country with optimal iodine status owing to the Dutch iodine fortification policy where urinary iodine concentrations in a sample of >1500 pregnant women were well in excess of the recommended level.[13] Although iodine status in the United States is considered "sufficient" at the population level, intake varies widely across the population,

[11] Brent G. A. (2014). Perchlorate exposure in pregnancy and cognitive outcomes in children: it's not your mother's thyroid. *The Journal of clinical endocrinology and metabolism*, *99*(11), 4066–4068. https://doi.org/10.1210/jc.2014-3673

[12] Gilbert, M. E., Hassan, I., Wood, C., O'Shaughnessy, K. L., Spring, S., Thomas, S., & Ford, J. (2022). Gestational Exposure to Perchlorate in the Rat: Thyroid Hormones in Fetal Thyroid Gland, Serum, and Brain. *Toxicological sciences : an official journal of the Society of Toxicology*, *188*(1), 117–130. https://doi.org/10.1093/toxsci/kfac038

[13] Ghassabian, A., Steenweg-de Graaff, J., Peeters, R. P., Ross, H. A., Jaddoe, V. W., Hofman, A., Verhulst, F. C., White, T., & Tiemeier, H. (2014). Maternal urinary iodine concentration in pregnancy and children's cognition: results from a population-based birth cohort in an iodine-sufficient area. *BMJ open*, *4*(6), e005520. https://doi.org/10.1136/bmjopen-2014-005520

and the percentage of women of childbearing age with iodine deficiency is increasing.[14] In a study of 464 pregnant women in Michigan, 23% had inadequate iodine intake.[15] Unlike the Netherlands, no such systematic fortification program exists in the U.S., increasing the probability that the model fails to accurately account for both the incidence and level of iodine deficiency in women in the U.S. exposed to perchlorate. Although a well-powered and robust dataset, it remains a single study, in a European population, and is limited to one stage of pregnancy.

3) EPA's consideration of only first trimester pregnancy, a point in time when the fetal thyroid gland is not yet functional, accounting for fetal exposure through allometric scaling of a slight increase in maternal body weight is unacceptable. It is unacceptable not only because it ignores the 'rest of pregnancy' and the postnatal period of brain development (the outcome of concern and covered in the intrahuman UF), but also because it fails to account for toxicodynamic differences that exist between maternal and fetal gland responses to direct perchlorate exposure. Perchlorate passes through the placenta via the NIS and is transported from the fetal blood into the fetal thyroid gland. Given that the fetal brain is dependent on a compromised maternal supply of thyroid hormone and the fetal gland is only newly developed, the fetus faces two potential sources of hormone insufficiency. One from perchlorate induced reductions in maternal sources, the other from depletion of stored hormone and iodine in the colloid of its own developing gland. Recent mechanistic studies in a gestational rodent model indicate ~2-fold greater sensitivity of the fetal to maternal thyroid hormone synthesis machinery to the effects of perchlorate at low concentrations administered during the equivalent of the 2nd trimester of human pregnancy. Maternal exposure to perchlorate through drinking water significantly reduced thyroid hormone synthesis products by ~50% in the fetal thyroid gland at concentrations that were without any effect in the gland of the dam in late gestation.[16]
4) EPA has argued that TSH-mediated feedback in response to small perchlorate-induced perturbations in serum thyroid hormones in the pregnant woman would be sufficient to maintain blood concentrations of thyroid hormones in the normal range. This may be true for the pregnant woman, but as detailed above, as pregnancy progresses, considerable uncertainty exists in the ability of the nascent fetal TSH feedback loop to compensate for perchlorate-induced perturbations in thyroid function that are available to the mature hypothalamic-pituitary-thyroid (HPT) axis of the pregnant woman.
5) Uncertainty exists due to the lack of evaluation of outcome measures that are more sensitive than global IQ, i.e. more refined neuropsychological tests of cognition and executive function. As reported in the peer reviewed scientific literature, behavioral studies based on congenital hypothyroidism and residual effects in congenital hypothyroid children who were "properly treated" show more sensitivity in higher cognitive domains

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[14] Niwattisaiwong, S., Burman, K. D., & Li-Ng, M. (2017). Iodine deficiency: Clinical implications. *Cleveland Clinic journal of medicine*, *84*(3), 236–244. https://doi.org/10.3949/ccjm.84a.15053; Hatch-McChesney, A., & Lieberman, H. R. (2022). Iodine and Iodine Deficiency: A Comprehensive Review of a Re-Emerging Issue. *Nutrients*, *14*(17), 3474. https://doi.org/10.3390/nu14173474

[15] Kerver, J. M., Pearce, E. N., Ma, T., Gentchev, M., Elliott, M. R., & Paneth, N. (2021). Prevalence of inadequate and excessive iodine intake in a US pregnancy cohort. *American journal of obstetrics and gynecology*, *224*(1), 82.e1–82.e8. https://doi.org/10.1016/j.ajog.2020.06.052

[16] Gilbert, M. E., Hassan, I., Wood, C., O'Shaughnessy, K. L., Spring, S., Thomas, S., & Ford, J. (2022). Gestational Exposure to Perchlorate in the Rat: Thyroid Hormones in Fetal Thyroid Gland, Serum, and Brain. *Toxicological sciences : an official journal of the Society of Toxicology*, *188*(1), 117–130. https://doi.org/10.1093/toxsci/kfac038

than observed with standardized tests of IQ.[17] This issue also was of concern to the External Reviewers of the two-step model in 2018.[18] Recent animal data further corroborate neurotoxicity induced by maternal exposure to perchlorate neurodevelopment in offspring. Perchlorate-induced neurophysiological impairments in brain regions critical for cognition, as well as structural defects in the brain, were observed and greatly exacerbated under conditions of iodine deficiency[19].

6) Finally, uncertainty exists in EPA's selection of a linear function to fit the data of fT4 and child IQ, contrasting to polynomial function selected by Korevaar (2016) to model the same data. As highlighted in both EPA's external review and in the New Jersey Department of Environmental Protection review, this selection of the linear over the polynomial function results in an underestimation of the effects on IQ at small decrements in maternal fT4.[20] At the very least, EPA must justify why it chose a linear, rather than a polynomial, function to model the fT4 and child IQ data.

In view of these uncertainties, the two-step model, as currently applied, is likely to underestimate the potential effects of perchlorate exposure on the fetus and newborn child. EPN strongly recommends that in addition to the 10X UF for intrahuman variability, a composite 10X MF be added to the model output 3.1 ug/kg/day POD to derive an RfD.

Application of Two Sets of UFs to Define the RfD

EPA has long-employed guidance which describes the following uncertainty factors and default values for use when agent-specific data are not available: interspecies (10X), intrahuman (10X), Lowest Observed Adverse Effect Level (LOAEL) to No Observed Adverse Effect Level (NOAEL) (3X or 10X), subchronic to chronic (3X or 10X), database deficiencies (case specific), and modifying factor (case specific). As the POD was derived from modeling human data with exposure during pregnancy, several of these factors do not apply (ie interspecies, LOAEL to NOAEL, subchronic to chronic). However, based on concerns outlined above, EPN considers two 10X UFs appropriate to apply to the POD of 3.1µg/kg/day derived from the EPA model. Many of these concerns were also identified in the New Jersey Department of Environmental Protection Science Advisory Board's 2020 Review Of Proposed EPA Maximum Contaminant Level for Perchlorate (2020), and in the Public Comments provided by New Jersey, California, Massachusetts, the NRDC, and EPN in response to EPA's FRN in 2020.[21]

In conclusion, EPN believes that the RfD for perchlorate should be 0.031 µg/kg/day, based upon a model output POD of 3.1 µg/kg/day and the application of a total uncertainty factor of 100 (UFH = 10X; MF=10X) as summarized below:

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[17] Rovet J. F. (2014). The role of thyroid hormones for brain development and cognitive function. *Endocrine development*, *26*, 26–43. https://doi.org/10.1159/000363153; Willoughby, K. A., McAndrews, M. P., & Rovet, J. F. (2014). Effects of maternal hypothyroidism on offspring hippocampus and memory. *Thyroid : official journal of the American Thyroid Association*, *24*(3), 576–584. https://doi.org/10.1089/thy.2013.0215; Wheeler, S. M., Willoughby, K. A., McAndrews, M. P., & Rovet, J. F. (2011). Hippocampal size and memory functioning in children and adolescents with congenital hypothyroidism. *The Journal of Clinical Endocrinology & Metabolism*, *96*(9), E1427-E1434.

[18] (EPA-HQ-OW-2016-0439-0012)

[19] Gilbert et al 2008; 2023; 2024a; 2024b

[20] EPA-2019HQ-OW2016-0439-0012; NJ Department of Environmental Protection, 2020.

[21] (EPA-HQ-2018-0780-0304)

Intrahuman UF of 10X for limiting the assessment to first trimester pregnancy only, lack of consideration of the vulnerability of fetal thyroid gland to the direct action of perchlorate, low iodine and hormone storage capacity and immaturity of compensatory mechanisms of the fetal thyroid system relative to the pregnant women.

Modifying Factor of 10X for limited data to parameterize the complex two-step BBDR model, reliance on data from a single study on a different continent in an iodine sufficient population, deficiency in the model to adequately represent the iodine status of pregnant women in the US, reliance on a single study relating maternal T4 to IQ, the lack of consideration of sensitivity differences in the response to perchlorate in the fetal vs the maternal thyroid gland, insensitivity of standardized tests of global IQ relative to more refined tests of cognitive performance, and application of a linear vs a polynomial function to fit IQ data with small deviations of maternal fT4.

From this RfD, an MCLG can then be calculated.

Maximum Contaminant Level Goal (MCLG)

A noncancer MCLG is designed to be protective of noncancer effects over a lifetime of exposure with an adequate margin of safety, including for sensitive populations and life stages, consistent with Safe Drinking Water Act (SDWA) section 1412(b)(3)(C)(i)(V) and 1412(b)(4)(A). The inputs for a noncancer MCLG include an oral noncancer toxicity value (RfD = POD/UF), body weight-adjusted drinking water intake (DWI-BW), and a RSC, as presented in the equation below**:**

$$MCLG\left(\frac{\mu g}{L}\right) = \left(\frac{POD/UF}{DWintake}\right)X\ RSC$$

RfD = chronic reference dose – an estimate of daily oral exposure of the human population to a substance that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is derived from POD from the model output with application of appropriate UF. Incredulously, EPA selected a single UF of 3 for intrahuman variation. Based on concerns outlined above, EPN proposes composite UF of 100, 10X for intrahuman variation and a 10X modifying factor UF to account for uncertainties in the toxicokinetics and toxicodynamics in establishing a health protective RfD upon which to base an MCLG.

DWI-BW = An exposure factor for the 90th percentile body weight-adjusted drinking water intake value for the identified population, in units of liters of water consumed per kilogram body weight per day (L/kg bw-day). EPA applied a value of 0.0354 DW intake. EPN concurs.

RSC = relative source contribution – the percentage of total exposure attributed to drinking water sources (U.S. EPA, 2000a), with the remainder of the exposure allocated to all other routes or sources. EPA applied an RSC 0.80, the highest default value with little explanation. This choice, which differs from previous values implemented by EPA, requires additional justification. In 2008, EPA set the RSC for perchlorate intake from drinking water to 0.62. In 2019 EPA set the perchlorate RSC to 0.56 (based on 90th % BW-adjusted). In the present analysis EPA applied the maximum default ceiling RSC of 0.80 based on mean perchlorate intakes. There appears little justification for the selection of the mean rather than upper percentile values applied in previous

assessments and Health Advisories issued by EPA.[22] Assessments in Massachusetts,[23] New Jersey,[24] and California[25] have used values ranging from 0.20-0.73. EPN contends a more appropriate RSC may be 0.56 as previously applied in EPA documents for perchlorate intake through drinking water in pregnant women.

Calculation of MCLG

EPN's intermediate MCLG proposal based on a composite UF of 100X (UFH of 10X and MF of 10X for toxicokinetics/toxicodynamics), and RSC set to 0.56

$$\left(\frac{3.1\mu g/100}{0.0354\,L}\right) X\ 0.56 =\ 0.49\left(\frac{\mu g}{L}\right)$$

This MCLG value of 0.49µg/L is almost two orders of magnitude lower than the lowest EPA proposed value of 20 µg/L. Furthermore, this MCLG value calculated by EPN is consistent with recent determinations made by several other regulatory bodies who have considered the outputs of this model or other approaches to define a POD and determine an RfD (e.g., New Jersey Department of Environmental Protection[26]; Health Canada[27]; Massachusetts[28]; California[29]).

Maximum Contaminant Level (MCL)

SDWA requires that EPA set the MCL as close to the MCLG as is feasible or, if the Administrator determines the health benefits of the MCL do not justify the cost, at the level where the cost is justified by the benefits. EPA is seeking comment on possible MCLs set at 20 µg/L, 40 µg/L, 80 µg/L or any other MCL because the Administrator has determined there is no MCL at which the benefits of treatment at a limited number of systems justify the costs of monitoring across systems where perchlorate is not expected to occur at levels of concern.

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[22] EPA 2002 635/R-02/003 Toxicological Review and Risk Assessment; EPA 2008 822-R-08-025 Interim Drinking Water Health Advisory for Perchlorate

[23]Massachusetts Department of Environmental Protection - Perchlorate Background Information and Standards https://www.mass.gov/lists/perchlorate-background-information-and-standards#perchlorate---final-standards-

[24] New Jersey Department of Environmental Protection. Science Advisory Board. Public Health Standing Committee. 2020. *Review of Proposed EPA Maximum Contaminant Level for Perchlorate.* https://hdl.handle.net/10929/69038

[25] California Environmental Protection Agency. Office of Environmental Health Hazard Assessment, Pesticide and Environmental Toxicology Branch. 2015. *Perchlorate in Drinking Water.* https://oehha.ca.gov/water/chemicals/perchlorate

[26] New Jersey Department of Environmental Protection. Science Advisory Board. Public Health Standing Committee. 2020. *Review of Proposed EPA Maximum Contaminant Level for Perchlorate.* https://hdl.handle.net/10929/69038

[27] Health Canada. 2020. Drinking water screening value for perchlorate–Technical summary. https://www.canada.ca/en/services/health/publications/healthy-living/drinking-water-screening-value-perchlorate.html

[28]Massachusetts Department of Environmental Protection - Perchlorate Background Information and Standards https://www.mass.gov/lists/perchlorate-background-information-and-standards#perchlorate---final-standards-

[29] California Environmental Protection Agency. Office of Environmental Health Hazard Assessment, Pesticide and Environmental Toxicology Branch. 2015. *Perchlorate in Drinking Water.* https://oehha.ca.gov/water/chemicals/perchlorate

An MCL is promulgated on a final health benchmark of an established RfD. EPN contends that the concerns outlined above in EPA's calculation of an RfD from the model derived POD and the lack of peer review of EPA's current RfD makes the derivation of an MCL untenable. In this light however, EPN also offers the following concerns for monitoring and treatment requirements that would be impacted in the absence of a more appropriate MCL.

Other than the formally promulgated MCLs in California and Massachusetts, another 12 states have science based perchlorate rules. They adopted health protective values ranging from 1 μg/L to 18 μg/L, as reported in current state websites. These are described in a confusing array of terms of unclear applicability such as various advisory levels, guidance levels, action levels, provisionary action levels, toxicity levels, groundwater chemical standards, etc. There are also site-specific cleanup values, as in Oregon and New Mexico, which do not appear to be state-wide. It is also a challenge to remain current or comprehensive on state regulations. For example, EPA's Office of Ground Water and Drinking Water published a fact sheet in 2020 with a table of state rules which has either missing or no longer valid information. A national enforceable standard is necessary to alleviate this patchwork for protecting public health.

It is most important that EPA take note that all of the state rules are below the indefensible 20 μg/L, the lowest level EPA has provided in the current proposal.

Timing of Perchlorate Monitoring

There is no requirement in EPA's proposal to evaluate the timing of peak perchlorate occurrence or to sample during the time of elevated risk of perchlorate contamination. This is a huge weakness in the rule. As designed, monitoring would be very likely not to detect peak perchlorate concentrations, and therefore water utilities that actually do have perchlorate in their source water probably will never find the peaks. Detecting the peak concentrations is important because the adverse effects seen in animal studies occur as the result of exposures during pregnancy. In the absence of data showing that repeated exposure for longer periods is necessary to elicit such effects, EPA must assume that a single, peak exposure could cause harm to a developing fetus.

As an example, a number of documented perchlorate peaks are associated with fireworks displays. If source water is located near fireworks displays, an appropriate time to look for peak concentrations would be July 5-8.

Per the rule, the initial perchlorate samples will be collected quarterly with antimony, arsenic, barium, beryllium, cadmium, fluoride, mercury, nickel, selenium, and thallium. These co-sampled contaminants are less likely to be dependent on discrete, infrequent high risk events for drinking water contamination. EPN recommends the final rule include a requirement to determine whether recurring (but infrequent) events, such as fireworks displays, present an elevated risk of perchlorate contamination and require sampling events to take place when peaks are expected to occur, e.g., within 48 hours of the event.

Monitoring Requirements

Annual monitoring of perchlorate in Public Water Supply (PWS) systems is warranted based on the rapidity of transport of perchlorate through groundwater and surface water, and would result in no significant cost increase to any PWS nationally.

Annual monitoring of other inorganic anions, such as nitrate and nitrite, is required by all PWS systems under 40 CFR §141.23. To minimize expenses to water supply systems, administrative, reporting, and waiver application procedures should be consistent with and no more complex than that for any other regulated inorganic analyte. EPA should coordinate with states with an existing perchlorate monitoring requirement to develop best methods of minimizing administrative workload on PWS systems. Obtaining samples for perchlorate is uncomplicated and easily incorporated into existing sampling programs. Aliquots of the currently requisite annual water quality samples can be used for perchlorate monitoring as well. No sample preservation and sample holding time limits for perchlorate are necessary.

Analysis of perchlorate for an annual sample would follow well-established and EPA-approved methods in use since 1996. These analytical methods are widely available throughout the United States and analytical costs to any individual PWS would be an insignificant addition to the system's operating and water quality monitoring budget. Widely available commercial analytical laboratories used for monitoring other chemicals, nitrate for example, are capable of perchlorate analysis. Analytical costs for perchlorate alone are less than $100 per sample and likely can be obtained for an order of magnitude less at a commercial laboratory.

Perchlorate travels readily in groundwater and with minimal dilution through mixing. Researchers use it as a non-reactive tracer to establish the rate of movement of the water itself to study the transport of other dissolved contaminants through groundwater and porous media. Perchlorate contamination will reach a groundwater well at the original concentration more rapidly than any other chemical. A delay in detection causes a longer period of potential exposure for this contaminant which displays toxic effects over relatively short time periods, such as pregnancy trimesters and neonatal development stages.

In 2018, there were 1,029 nitrate MCL violations issued to 507 systems, representing 7 percent of all of the health-based violations reported that year and impacting 0.3 percent of all systems and an even smaller percentage of the U.S. population.[30] The proposal cannot justify avoiding a monitoring requirement for perchlorate which threatens an equivalent order of magnitude of the nation's PWS and a greater portion of the population of the United States.

Treatment Requirements

Contaminant presence, source identification, and timely remediation activities performed by EPA and states are informed by a number of potential applicable or relevant and appropriate requirements including national drinking water standards. The decision to withdraw the regulatory determination has demonstrable bearing on actions related to mitigation measures on contaminated sites.

[30] USEPA Office of Ground Water and Drinking Water, November 30, 2020. https://www.epa.gov/sites/default/files/2021-01/documents/wsg_213_nitrate_wsg_11-30-2020_signed_508-compliantfinal.pdf

Very high levels of perchlorate were identified at a Superfund site in California in 1990.[31] The development of a reliable analytical method to 4 μg/L in 1997 confirmed widespread occurrence and threats to public water supplies. Yet there were no remediation or planning activities undertaken for many years until enforceable standards were established in several states. Current extraordinarily successful mitigation efforts have been undertaken after 2005 in the absence of national primary drinking water regulations only due to preliminary MCLGs or enforceable state standards for perchlorate.

Most states do not currently provide any enforceable monitoring or regulatory mechanism. Perchlorate use and contamination often originate at military-related manufacturing and testing facilities and other explosive use and fireworks sites. These are located nationwide, not only in states with perchlorate regulations. Preemptive monitoring waivers are unwarranted.

Although the SDWA's criteria for establishing National Primary Drinking Water Regulations do not explicitly include consideration of cleanup standards for contaminated sites, it does have the primary goal of protecting human health from any water-borne pollutants. Centuries-old practice is to identify and mitigate contamination sources, for example cholera bacteria, not to merely restrict use of untreated water at the distribution point.

Three general treatment methodologies are presented in the proposal, all of which have capital and operating costs dependent on site specific conditions. This accounts for the wide range of potential costs presented. Non-treatment options to assure drinking water protectiveness are ignored as well. EPA avers that the range of treatment costs on a national basis are too high. Further analysis is warranted.

Reliable and cost-effective treatment systems to remove perchlorate from water supply sources have been demonstrated operationally at a range of scales for several decades since several states established regulatory levels.

The proposal does not consider the use of nontreatment compliance options which have often been the first response when perchlorate has been detected in PWS systems of a range of sizes. Strategies such as shifting to an uncontaminated source, blending with an uncontaminated source, or connecting to another water utility have been used at least temporarily. This provides time for assessing and planning for options which typically include mitigation of the source. These strategies can result in significant cost savings and are frequently the first option considered by water utilities due to their low operation and maintenance cost after implementation. Excluding these from the compliance options unnecessarily drives up the cost estimates for the rule.

The most remarkable aspect of the decades of perchlorate treatment experience is that neither the capital nor operating costs are being borne by the PWSs. Perchlorate is an uncommon pollutant that is readily traced to an undisputed source. Detection of the contaminant by a water supply monitoring program rapidly resulted in identification of the responsible party in every situation nationally since the 1990s. By means of enforcement of a legal regulation, the originators of the pollutant are held liable. Typically the treatment efficiently removes perchlorate at the PWS and at

[31] National Academies of Sciences, Engineering, and Medicine. 2005. Health Implications of Perchlorate Ingestion. Washington, DC: The National Academies Press. https://doi.org/10.17226/6020.

the originating source as well, for example at the former Olin flare facility in rural San Martin, CA[32] and the former Kerr-McGee manufacturing facility in Henderson, Nevada.[33]

The proposal's analysis of treatment expenses is a major factor in an argument against establishing a protective enforceable standard. This forces the PWSs and their customers to bear the health risks while absolving the polluters from potentially substantial expense unless they are in a state with a robust regulatory mechanism. The conclusion offered in the proposal to avoid regulation runs counter to the primary purpose of EPA as established by Congress.

Benefits & Costs

Based on the benefit-cost analysis, the Administrator makes the determination that the quantified and unquantifiable benefits of the proposed rule do not justify the costs.

The benefits of protecting human health from perchlorate are on the same order of magnitude as to the protection from any other potential chemical toxins regulated under SDWA, such as nitrate. And, in both cases (perchlorate and nitrite), the health goal is to protect particularly susceptible and sensitive subpopulations-the fetus and infant.

The proposal overstates the cost of monitoring for perchlorate which should be incorporated into existing programs for sampling and analysis. The proposal apparently evaluated the cost for monitoring perchlorate as if it were an entirely separate process.

The cost for treatment, if borne by the PWS systems, is indeed substantial. However, in no identified instance have treatment expenses fallen to the public to any extent. All quantified treatment costs thus far have resulted from enforcement of a legal requirement or credible threat of a regulation. The proposal demonstrably protects the polluters, not the general population, nationwide except in states which already have enacted an enforceable requirement. The current proposal increases the actual and potential costs to the public and does not provide any adequate protection of the public's drinking water quality. The Administrator's determination runs exactly counter to the intentions of the SDWA.

[32] https://geotracker.waterboards.ca.gov/profile_report?global_id=SL0608756247
[33] https://ndep.nv.gov/environmental-cleanup/black-mountain-industrial-bmi-complex/perchlorate