

PERCHLORATE, THE SAFE DRINKING WATER ACT AND PUBLIC HEALTH

Summary

Perchlorate — a naturally-occurring and man-made salt used in a variety of military, aerospace and industrial settings — is arguably one of the most studied chemicals currently under regulatory review. The scientific record regarding the human health effects of perchlorate involves more than 60 years of research, including during perchlorate's use worldwide as a drug to treat Graves' Disease. It is precisely because of the unique robustness of the scientific database on perchlorate that its health effects, or lack thereof at environmental levels, are so well understood.

Recent confirmation of the ability of perchlorate to form naturally in the atmosphere, in arid environments — and even on Mars — suggest that the compound has been a part of our natural environment for millennia. It is only as detection technologies have become more and more advanced in recent years that we have been able to detect perchlorate in minute traces, approaching the parts-per-trillion level.

Perchlorate at high (therapeutic) levels is known to block the transport of iodide from the bloodstream into the thyroid gland. Lower doses reversibly inhibit iodide uptake, an effect which scientists have concluded is non-adverse. Of note, perchlorate is only one of several compounds known to have this non-adverse effect. Others, principally including nitrate and thiocyanate, occur naturally in many of the foods we eat and together account for more than 99 percent of the iodine uptake inhibition (IUI) that takes place in the body. At the upper range of levels found in the environment, perchlorate accounts for less than one percent of IUI.

Scientists have also found a perchlorate level that has no measurable effect; doses below this no effect dose have no measurable effect on health. To the best of scientific knowledge to date, taking the dose that has no measurable effect in the body and reducing it 10-fold should protect the health of sensitive populations, including infants, fetuses, and pregnant women who may be iodine deficient.

In 2005, scientific studies and reports were reviewed by a group of independent scientists at the National Academies of Science (NAS), which concluded that a reference dose for perchlorate of 0.0007 milligrams per kilogram of body weight per day — roughly equal to 24.5 parts per billion (ppb) in drinking water — would be safe for even the most sensitive populations.

Since the NAS report was published, other authoritative bodies have independently concluded that the low levels of perchlorate found in the environment have no effect on human health, even when sensitive populations are considered. This is a reliable and, on the whole, strongly consistent body of scientific analysis. Based on the criteria EPA uses and has used in its evaluation of chemical agents, the perchlorate database is as strong, or stronger, than that of many chemicals previously assessed. In the first quarter of 2011, even as new studies confirmed

the findings of NAS and other researchers, EPA published a new *Regulatory Determination on Perchlorate*, concluding that perchlorate should be regulated under the federal Safe Drinking Water Act.

In order for the EPA to decide to regulate perchlorate, it must be demonstrated that perchlorate satisfies all three requirements set forth in the Safe Drinking Water Act. Specifically:

1. The compound must cause an adverse effect on human health.
2. The compound must be present in drinking water systems at a frequency and level of public health concern.
3. Regulating the compound must present a meaningful opportunity for health risk reduction.

However, the *Regulatory Determination on Perchlorate* does not demonstrate that perchlorate meets any, let alone all, of the three criteria and instead relies on very broad generalizations (*e.g.* “...if an MCL were to be set at 6 ppb, perchlorate exposures could be reduced to levels below 6 ppb) which do not satisfy the requirements of the Safe Drinking Water Act and, if used as a rationale, could in fact be used to justify regulating *anything*. EPA must give additional consideration to the overwhelming weight of the evidence that there is in fact no public health benefit in the regulation of perchlorate.

As the nation now faces a time of unprecedented pressure on public sector budgets, the allocation of limited federal resources on a chemical for which no adverse health effects have been documented in six decades of study, must be more closely scrutinized. In particular, the far reaching impacts of such a misallocation could have on other, much more needed, public health programs must not be overlooked.

Regulatory Background

In 1996, Congress amended the Safe Drinking Water Act to require EPA to compile a contaminant candidate list (CCL) and make a determination whether to regulate at least five contaminants from the CCL every five years.¹ The CCL is a compilation of contaminants that are known or anticipated to occur in public water systems and which may require regulation.² The current CCL contains more than 100 chemical contaminants, including perchlorate, and an additional 12 microbial contaminants.³

In 1997, perchlorate was detected in the Lower Colorado River for the first time.⁴ Perchlorate contamination was also discovered in several groundwater basins in California in 1997.

¹ 42 U.S.C. § 300g-1(b)(1)(B).

² *Id.*

³ 74 Fed. Reg. 51850 (Oct. 8, 2009).

⁴ State of Nevada, Nevada Division of Environmental Protection, Bureau of Corrective Action, *Southern Nevada Perchlorate Cleanup Project*, available at <http://ndep.nv.gov/bca/perchlorate05.htm> [accessed March 2011].

Perchlorate had been identified in groundwater as a result of sampling in the San Gabriel Valley in California as early as 1985.⁵ However, testing of public water supplies for perchlorate occurred only sporadically in the late 1980s and 1990s.⁶ It was not until March 1997, when the California Department of Health Services (DHS) refined the analytical method to achieve a detection limit of 4 micrograms per liter ($\mu\text{g/L}$), that California DHS began to sample for, and to find, perchlorate in groundwater in more locations.⁷

In March 1997, a non-profit risk assessment consulting firm, Toxicology Excellence for Risk Assessment (TERA), convened an independent peer review panel to assess perchlorate health risks.⁸ The peer review panel concluded that the existing scientific database for perchlorate was inadequate for development of a credible quantitative health risk assessment.⁹ As a result, another expert panel met in May 1997, and developed a testing strategy based on the known mode of action of perchlorate. An integrated approach to risk characterization was proposed to address data gaps and reduce uncertainties regarding possible health effects of low levels of perchlorate in drinking water.¹⁰

Following the recommendation of the expert panel, the Perchlorate Study Group (PSG) and Department of Defense (DOD) funded eight studies, including pharmacokinetic and mechanistic studies in animals and humans. In April 1998, the results from these eight studies were submitted to EPA. In December 1998, EPA's National Center for Environmental Assessment (NCEA) prepared a draft risk assessment for perchlorate based on the more comprehensive database, which included the information contained in the eight studies.¹¹ The eight studies confirmed that the health effects of perchlorate were related to the thyroid, and confirmed the absence of effects on other organs or systems. NCEA proposed an RfD of 0.0009 mg/kg-day,

⁵ Takata, K., *Request for CDC Assistance Regarding Potential Health Effects of Perchlorate Contamination at the San Gabriel Valley Superfund Sites* [memorandum with attachments to Don Hawkins] San Francisco, CA: U.S. Environmental Protection Agency, Superfund Records Center, Region 9; December 23, 1985, available at <http://www.epa.gov/ncea/perchlorate/references2/documents/2649.pdf> [accessed March 2011].

⁶ National Academy of Sciences, *Health Implications of Perchlorate Ingestion* (The National Academics Press 2005) at 12 (hereinafter, "NAS"), available at http://www.nap.edu/catalog.php?record_id=11202 [accessed March 2011].

⁷ Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Perchlorates* (Sept. 2008) at 205 (hereinafter, "ATSDR"), available at <http://www.atsdr.cdc.gov/toxprofiles/tp162.pdf> [accessed March 2011]. ATSDR is a federal public health agency in the U.S. Department of Health and Human Services

⁸ NAS, *supra*, n. 7, at 20.

⁹ *Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization Based on Emerging Information (External Review Draft) 1998 "Report Information,"* available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=23292> [accessed March 2011].

¹⁰ *Id.*

¹¹ *Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization Based on Emerging Information (External Review Draft) 1998* at 1-18.

which translated into a drinking water equivalent level of 32 ppb, using standard risk assessment assumptions.¹²

At the time the 1998 NCEA risk assessment was released, EPA stated that the “risk assessment activities regarding perchlorate have been a model for a full and open public process involving several EPA offices, programs and regions, many other Federal Agencies, States, industry and the public.”¹³ EPA also noted that the Interagency Perchlorate Steering Committee (IPSC), a working partnership among EPA, DOD, ATSDR, the National Institute for Environmental Health Sciences, and interested state, tribal and local governments, had identified issues and coordinated the exchange of scientific issues.¹⁴

In February 1999, EPA contracted with Research Triangle Institute, an independent research organization, to conduct a peer review of NCEA’s RfD. The peer review panel concluded that the 32 ppb drinking water equivalent level was likely to be conservative. The peer review panel also recommended the completion of additional studies to determine the RfD more precisely.¹⁵ In April 1999, PSG and DOD agreed to fund the additional studies, and, in July 2000, data from the additional requested studies were submitted to EPA.¹⁶

NCEA released a second draft risk assessment for perchlorate in January 2002.¹⁷ Based on an animal study, that was later found to be unreliable, NCEA proposed a new RfD of 0.00003 mg/kg-day.¹⁸ This translated into a drinking water equivalent level of 1 ppb, using standard risk assessment assumptions. In late 2003, at the urging of several stakeholders, EPA requested that NAS review the new NCEA risk assessment.

The Scientific Record on Perchlorate

A. The National Academy of Sciences Report

The National Academy of Sciences (NAS) is a society of distinguished scholars engaged in scientific and engineering research that investigates and reports upon scientific subjects when called upon to do so by any department of the federal government.¹⁹ The NAS perchlorate

¹² NAS, *supra*, n. 7, at 23.

¹³ *Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization Based on Emerging Information (External Review Draft) 1998 “Report Information,” supra*, n. 10.

¹⁴ *Id.*

¹⁵ NAS, *supra*, n. 7, at 22.

¹⁶ *Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization Based on Emerging Information (External Review Draft) 1998 “Report Information,” supra*, n. 10.

¹⁷ *Id.*

¹⁸ NAS, *supra*, n. 7, at 135.

¹⁹ 36 U.S.C. §150303.

review panel was comprised of 15 leading scientists and physicians with wide-ranging expertise in the relevant areas.²⁰ The NAS process unfolded over a 15-month period, and included an exhaustive review of a vast body of animal and human studies, public comment and written submissions. The NAS panel stated that “emphasis was given to studies with the soundest scientific methods.”²¹

In its 2005 report, *Health Implications of Perchlorate Exposure*, the NAS reached several conclusions. First, inhibition of iodide uptake (IUI) is the only consistently documented human health effect of perchlorate.²² Second, IUI is a reversible biochemical phenomenon and is not an adverse health effect.²³ Third, no adverse health effects will occur if IUI does not occur.²⁴ Fourth, changes in thyroid hormone levels are not necessarily adverse; rather, these levels change in response to numerous environmental stimuli seasonally, and even daily.²⁵ Fifth, as shown in the Greer Study and other clinical studies, IUI is only observed at doses in excess of 0.007 mg/kg-day.²⁶

This dose level (0.007 mg/kg-day), which correlates to a drinking water equivalent level of 245 ppb, was established as the no-observed-effect-level (NOEL), and was used by the NAS as its point of departure for developing a reference dose (RfD).²⁷ To derive the RfD, NAS divided the NOEL by an uncertainty factor (UF) of 10 to account for the most susceptible individuals in the population—hypothyroid or iodide deficient pregnant women and their developing fetuses.²⁸

Typically, an RfD is derived from a no-observed-*adverse*-effect-level (NOAEL) or a lowest-observed-*adverse*-effect-level (LOAEL). The NAS stated that: “Inhibition of iodide uptake by the thyroid is clearly *not* an adverse effect; however, if it does not occur, there is no progression to adverse health effects.”²⁹ The NAS further stated:

The committee emphasizes that its recommendation differs from the traditional approach to deriving the RfD. The committee is recommending using a non-adverse effect rather than an adverse effect as the point of departure for the perchlorate risk assessment.

²⁰ NAS, *supra*, n. 7, at 4.

²¹ *Id.*

²² *Id.* at 13.

²³ *Id.* at 14-17.

²⁴ *Id.*

²⁵ *Id.*

²⁶ *Id.*

²⁷ *Id.* at 15-16.

²⁸ *Id.*

²⁹ *Id.* at 183.

*Using a non-adverse effect that is upstream of the adverse effect is a conservative, health-protective approach to the perchlorate risk assessment.*³⁰

Given the natural compensation mechanisms, NAS determined that it was “highly likely” that, in people with normal iodide uptake, reduction of iodide uptake by 75% for several months or longer would be required for thyroid hormone production to decrease enough to cause adverse effects.³¹ In adults, that would require *sustained* exposure to 0.4 mg/kg-day of perchlorate for a 70 kg person.³² This dose level (0.4 mg/kg-day) translates into a drinking water equivalent level of 14,000 ppb, using standard risk assessment assumptions. Perchlorate is water soluble, but is not metabolized by the body and is quickly removed from the bloodstream by the kidneys.³³ The half-life of perchlorate in the body is about eight hours.³⁴ Because of this short duration, exposure to perchlorate must essentially be continuous for any level of perchlorate to remain in the body.³⁵

In reaching its conclusions, the NAS relied principally on the Greer Study and on “four other studies in which healthy adults were given perchlorate.”³⁶ The NAS stated that in addition to these studies, studies of long-term treatment of hyperthyroidism, occupational studies and studies of “environmental exposure add confidence to the overall database.”³⁷ The NAS relied exclusively on human studies, stating that, when available and reliable, human studies are preferred over animal studies.³⁸

B. The Greer Study and Other Clinical Studies

The NAS RfD was based upon five key clinical studies in which various doses of perchlorate were given to healthy adults for various periods of time and the effects on iodide uptake and on thyroid hormones were determined.³⁹ In the most comprehensive study (the Greer Study), 37 healthy adults were given four different doses of perchlorate for 14 days: (1) 0.007 mg/kg-day; (2) 0.02 mg/kg-day; (3) 0.1 mg/kg-day; and (4) 0.5 mg/kg-day.⁴⁰ Thyroid uptake of iodide was measured 8 and 24 hours after the first administration of perchlorate, on the final day (14th day)

³⁰ *Id.* at 15.

³¹ *Id.* at 66-67.

³² *Id.*

³³ *Id.* at 220.

³⁴ *Id.* at 62.

³⁵ *Id.* at 62-66.

³⁶ *Id.* at 177.

³⁷ *Id.*

³⁸ *Id.* at 169.

³⁹ *Id.* at 62-64.

⁴⁰ *Id.*

of perchlorate administration, and 15 days thereafter.⁴¹ On the 14th day, iodide uptake was 98.2% of the baseline value (a statistically insignificant decrease) in the subjects given the lowest dose—0.007 mg/kg-day of perchlorate.⁴² Greer concluded that at doses of 0.007 mg/kg-day, and below, perchlorate would have no effect on the uptake of iodide into the thyroid gland.⁴³

The NAS concurred with Greer’s conclusion, and noted that the results of all five of the clinical studies were “remarkably consistent.”⁴⁴ The study subjects were all healthy adults.⁴⁵ They were all free-living, eating a self-selected diet.⁴⁶ The baseline values of thyroid hormones varied somewhat among the subjects, but all were within the normal range.⁴⁷ Although each individual study group was small, the results were highly consistent within each treatment group.⁴⁸ The effects of similar doses of perchlorate on iodide uptake were also very similar across all five studies.⁴⁹ At a daily dose of 0.007 mg/kg-day, no effect was found in two of the studies.⁵⁰ A daily dose of 0.020 mg/kg-day had a small effect in one of the studies. And, daily doses of 0.030 and 0.040 mg/kg-day had no effect in two of the studies.⁵¹ The NAS stated that the reproducibility of the results across all five of the clinical studies, coupled with the results of studies of long-term treatment of hyperthyroidism and studies of environmental and occupational studies strengthened its confidence in the NOEL value determined by Greer.⁵²

C. The Crump Study and Other Studies of Environmental Exposure on Fetuses, Newborns and Children

A number of environmental studies have been conducted comparing populations exposed to varying levels of perchlorate in drinking water. The most important of these, the Crump Study, was published in 2000.⁵³ In the Crump Study, thyroid hormone levels and the frequency of thyroid disease were studied in newborns and children in three cities in northern Chile with

⁴¹ *Id.*

⁴² *Id.*

⁴³ *Id.* at 62.

⁴⁴ *Id.* at 65.

⁴⁵ *Id.* at 63.

⁴⁶ *Id.*

⁴⁷ *Id.* at 66-67.

⁴⁸ *Id.*

⁴⁹ *Id.*

⁵⁰ *Id.*

⁵¹ *Id.*

⁵² *Id.* at 177.

⁵³ *Id.* at 67; Crump, C., P. Michaud, R. Tellez, C. Reyes, G. Gonzalez, E.L. Montgomery, K.S. Crump, G. Lobo, C. Becerra, and J.P. Gibbs, 2000, *Does Perchlorate in Drinking Water Affect Thyroid Function in Newborns or School-Age Children?* J. Occup. Environ. Med. 42(6):603-612.

differing concentrations of perchlorate in their municipal water supplies.⁵⁴ The three cities were Antofagasta (no detectible perchlorate), Chanaral (5 to 7 ppb perchlorate) and Taltal (100 to 120 ppb perchlorate).⁵⁵ The Crump Study concluded that continual exposure to perchlorate at levels up to 120 ppb had no effect on the thyroid health of fetuses, newborns or children.⁵⁶

The NAS found the Crump Study to have “important strengths” including: (1) perchlorate concentrations were actually measured at the children’s homes and schools; (2) the rate of participation in the study was high; (3) thyroid function, other end points and potential risk factors were obtained in a systematic fashion and adjusted for important covariates; (4) all assessments were done at the same laboratory; (5) all assessments and measures were done by observers unaware of the children’s city of origin; and (6) all newborn tests in Chile are done at a single facility.⁵⁷ NAS also reviewed criticisms of the Crump Study and concluded that the criticisms were adequately addressed by the co-authors of the Crump Study, were common to all environmental studies, or were without merit.⁵⁸

Several years later, a team led by Tellez studied 184 pregnant women from the same three cities in northern Chile.⁵⁹ The Tellez Study found no significant association between levels of perchlorate in drinking water and levels of thyroid hormones measured early in pregnancy (16.1 weeks) or late during pregnancy (32.4 weeks).⁶⁰ In addition, Tellez evaluated neonatal weight, length, head circumference, length of gestation, and thyroid hormones and perchlorate in umbilical cord blood.⁶¹ The Tellez Study found no significant differences among the fetal development indicators or thyroid hormone levels among pregnant women and newborns from the different cities.⁶² The Tellez Study concluded that even drinking water levels as high as 114 ppb do not inhibit iodide uptake sufficiently to affect thyroid hormone levels either during gestation or among newborns.⁶³

More recently, a team led by Amitai evaluated newborns of mothers in three communities in Israel with differing exposure to perchlorate in drinking water.⁶⁴ The communities were: Morasha (up to 340 ppb perchlorate); Ramat Hasharon (42 to 94 ppb); and Hertzlia (less than 3

⁵⁴ NAS, *supra*, n. 7, at 99-100.

⁵⁵ *Id.*

⁵⁶ *Id.*

⁵⁷ *Id.* at 101-102.

⁵⁸ *Id.* at 101-105.

⁵⁹ ATSDR, *supra*, n. 8, at 58.

⁶⁰ *Id.* at 58-59.

⁶¹ *Id.* at 72.

⁶² *Id.* at 72.

⁶³ *Id.* at 71.

⁶⁴ *Id.* at 73.

ppb).⁶⁵ Neonatal thyroid hormone levels, which were obtained from the National Screening Program database, were virtually identical in the three communities.⁶⁶ Moreover, neither birth weight nor gestational age was significantly different among the three groups.⁶⁷ The Amitai Study concluded that exposure to perchlorate at levels up to 340 ppb in drinking water had no effect on thyroid health among developing fetuses and newborns.⁶⁸

D. Additional Reports from Federal Authoritative Bodies

Two additional comprehensive reviews of perchlorate have been published by authoritative bodies in the federal government since the NAS published its 2005 report. In 2007, ATSDR published its *Toxicological Profile for Perchlorates*.⁶⁹ And in April 2010, the EPA Office of Inspector General (OIG) published its *Scientific Analysis of Perchlorate*.⁷⁰

1. *Agency for Toxic Substances and Disease Registry (ATSDR) Report*

In its profile, ATSDR stated that: (1) exposure to perchlorate can occur by ingestion of food or water that contains perchlorate; (2) the main target organ for perchlorate toxicity is the thyroid gland and perchlorate has been shown to partially inhibit the thyroid's uptake of iodide; (3) "although not demonstrated in humans, it is anticipated that people exposed to excessive amounts of perchlorate for a long time may develop a decreased production of thyroid hormones;" and (4) other chemicals, such as thiocyanate (in food and cigarette smoke) and nitrate (in some food), are also known to inhibit iodide uptake.⁷¹

ATSDR also concurred with the NAS-recommended RfD of 0.0007 mg/kg-day for perchlorate.⁷² ATSDR stated that its:

*decision was made after a careful evaluation of the NAS report
and of studies that have been published after the NAS (2005)*

⁶⁵ Tarone, Robert E., Lipworth, Loren, and McLaughlin, Joseph, 2010, *The Epidemiology of Environmental Perchlorate Exposure and Thyroid Function: A Comprehensive Review*, J Occup. Environ. Med. 52(6):653-60 at 657.

⁶⁶ *Id.* at 657-58.

⁶⁷ ATSDR, *supra*, n. 8, at 73.

⁶⁸ *Id.*

⁶⁹ ATSDR, *supra*, n. 8.

⁷⁰ U.S. Environmental Protection Agency, Office of Inspector General, *Office of Inspector General Scientific Analysis of Perchlorate*, Report No.10-P-0101 (April 19, 2010); (hereinafter, "OIG"); available at <http://www.epa.gov/oig/reports/2010/20100419-10-P-0101.pdf> [accessed March 2011].

⁷¹ ATSDR, *supra*, n. 8, at 6-8.

⁷² *Id.* at 22.

*report. The results from newer studies do not change the bottom-line.*⁷³

ATSDR further noted that the Greer Study, upon which the RfD was based, was supported by other clinical studies, worker studies and environmental studies.⁷⁴

ATSDR also discussed the simultaneous joint effects of perchlorate and other competitive inhibitors of iodide uptake.⁷⁵ ATSDR stated that nitrate and thiocyanate are widely distributed in nature, and because both of these anions also inhibit iodide uptake, they must be included in any discussion of iodide inhibition.⁷⁶ Nitrates are a natural constituent of green leafy vegetables and are used as a preservative; thiocyanates are present in certain common foods and in tobacco.⁷⁷ ATSDR noted that the effects of thiocyanate on thyroid function have long been known.⁷⁸ ATSDR calculated that exposure to nitrate in drinking water at the current nitrate MCL level would cause inhibition of iodide uptake equivalent to 300 ppb of perchlorate in drinking water.⁷⁹

ATSDR also stated that the most sensitive populations to iodide uptake inhibition are fetuses and pre-term newborns.⁸⁰ Importantly, there are no cases linking perchlorate to adverse health effects in fetuses or newborns in the scientific literature. The expected high sensitivity of these populations is due to the important role played by thyroid hormones during development. Low thyroid hormone levels during embryonic or fetal development can lead to mental deficits, impaired motor skills and hearing and speech impediments.⁸¹ This is because the fetus is dependent upon maternal thyroid hormones until about 16 to 20 weeks of gestation, when the fetal thyroid begins to produce its own thyroid hormones.⁸²

⁷³ *Id.* at 22-23.

⁷⁴ *Id.* at 24.

⁷⁵ *Id.* at 132.

⁷⁶ *Id.*

⁷⁷ *Id.*

⁷⁸ *Id.* at 133.

⁷⁹ *Id.* at 132.

⁸⁰ *Id.* at 134.

⁸¹ *Id.*

⁸² *Id.*

2. EPA Office of Inspector General Report

In its April 2010 report, the OIG stated that: (1) perchlorate acts by blocking iodide uptake into the thyroid; (2) dietary exposure to thiocyanate and nitrate also inhibits iodide uptake; and (3) iodide deficiency itself directly impacts iodide uptake.⁸³ OIG concluded that it is the combined effect of iodide deficiency and exposure to thiocyanate, nitrate and perchlorate that accounts for decreased iodide uptake in humans.⁸⁴

OIG determined that a single-chemical risk assessment would not effectively address any public health issue associated with decreased iodide uptake.⁸⁵ As a result, OIG conducted a cumulative risk assessment, following the concepts in EPA's *Framework for Cumulative Risk Assessment*, to address the public health issues associated with low iodide uptake.⁸⁶ OIG concluded that:

*the nature of the public health issue not only meets the requirements to be addressed by a cumulative risk assessment approach, but the complexity of the interactions among the three NIS inhibitors and the iodide nutritional level requires the use of a cumulative risk assessment approach to accurately characterize the nature of the problem and to identify an effective and cost-efficient solution(s) to the problem.*⁸⁷

With regard to iodide deficiency, OIG stated that if the diet is poor in iodide, the amount of iodide uptake into the thyroid will be low regardless of exposure to thiocyanate, nitrate and perchlorate.⁸⁸ Lack of iodide in the diet results in the same outcome as exposures to substances that inhibit the uptake of iodide.⁸⁹ It is the combined effect of all four stressors on the thyroid (exposure to thiocyanate, nitrate and perchlorate, and lack of iodide in the diet) that determines the amount of iodide uptake.⁹⁰

OIG stated that the public health issue, which it characterized as one of subtle mental deficits in children, is caused by an insufficient amount of iodide uptake in pregnant women, fetuses and infants.⁹¹ While theoretically, any one of the four stressors (lack of iodide, or excess thiocyanate,

⁸³ OIG, *supra*, n. 71, at 10, 31.

⁸⁴ *Id.* at 10.

⁸⁵ *Id.* at 11.

⁸⁶ *Id.* at 12.

⁸⁷ *Id.* at 28.

⁸⁸ *Id.* at 48.

⁸⁹ *Id.*

⁹⁰ *Id.* at 39.

⁹¹ *Id.* at 175.

nitrate or perchlorate) could result in lowered iodide uptake.⁹² OIG emphasized that focusing on only one of the four stressors would not address the public health issue in question.⁹³

OIG pointed out that a cumulative risk assessment would allow all of the necessary issues, many of which are outside EPA's legislative mandate, to be identified and addressed in cooperation with other agencies and organizations.⁹⁴ For example, OIG observed that while EPA has a statutory framework pursuant to which it can regulate contaminants in drinking water, addressing iodide nutrition is outside of EPA's legislative mandate and, in OIG's view, addressing iodide deficiency is essential to addressing the underlying public health issue.⁹⁵

Safe Drinking Water Act Mandated Criteria for Regulation

In order to be regulated, a contaminant on the CCL must satisfy three requirements, as specified in the Safe Drinking Water Act.⁹⁶ Perchlorate does not satisfy any of these three requirements.

- First, perchlorate does not cause adverse health effects at levels found in the environment. The Regulatory Determination refers to levels of perchlorate found in the environment as “low-level perchlorate exposure.”⁹⁷
- Second, perchlorate does not appear with a frequency or at levels of concern in public water supplies. The Regulatory Determination relies on an unrepresentative set of data that is 8 to 10 years old. Since that data was collected, levels of perchlorate in the Colorado River (by far the largest water source that contains perchlorate) have decreased by 80 percent due to cleanup activities. Actions by several states have also resulted in significant decreases in the frequency and levels of perchlorate.
- Third, a drinking water regulation for perchlorate will result in no public health benefit. At environmental levels, perchlorate has a miniscule impact on the uptake of iodide — its only known health effect. Lack of iodide in the diet and exposures to thiocyanate and nitrate overwhelm whatever impacts perchlorate might have.

A. Perchlorate Does Not Cause Adverse Health Effects At Levels Found In The Environment

The first finding that must be made before a determination to regulate is made under the Safe Drinking Water Act is whether “the contaminant may have an adverse effect on the health of persons.”⁹⁸ The Regulatory Determination purports to make the finding by posing the question: “May perchlorate have an adverse effect on the health of persons?” and answering the question,

⁹² *Id.*

⁹³ *Id.*

⁹⁴ *Id.* at 176.

⁹⁵ *Id.* at 177.

⁹⁶ 42 U.S.C. §300g-1(b)(1)(A).

⁹⁷ 76 Fed. Reg. 7763.

⁹⁸ 42 U.S.C. §300g-1(b)(1)(A)(i).

“Yes.”⁹⁹ There are, however, a number of serious problems with respect to this first finding. First, the analysis contained in the Regulatory Determination does not support the finding. Second, there are no known instances of adverse effects of perchlorate at environmental levels. Third, the environmental studies that directly address this question reach the opposite conclusion of EPA. Finally, perchlorate is only one stressor regarding iodide uptake and is at most an immaterial stressor.

1. The Regulatory Determination Does Not Support The Finding

The analysis regarding the “health effects” finding is contained in a single nine-sentence paragraph in the Regulatory Determination.¹⁰⁰ Most of the nine sentences recite well-understood and non-controversial facts about thyroid function and do not mention perchlorate. For example, the fourth sentence states that transfer of iodide into the thyroid is an essential step in thyroid hormone synthesis.¹⁰¹ The seventh sentence states that poor iodide uptake has been linked to developmental problems.¹⁰² Several of the sentences recite unchallenged facts about the mode of action of perchlorate. For example, the second sentence states that perchlorate can inhibit iodide uptake, and the third sentence states perchlorate competes with iodide for uptake into the thyroid because it has a similar shape and electrical charge.¹⁰³

Once these unremarkable sentences are peeled away, the analysis offered in support of the “health effects” finding amounts to a single sentence—the sixth sentence in the paragraph:

*Because the developing fetus depends on an adequate supply of maternal thyroid hormone for its central nervous system development during the first and second trimester of pregnancy, iodide uptake inhibition from low-level perchlorate exposure has been identified as a concern in connection with increasing risk of neurodevelopmental impairment in fetuses of hypothyroid mothers.*¹⁰⁴

However, and as is clear from the text itself, (1) “identifying” perchlorate (2) as a “concern” (3) “in connection with” (4) “increasing risk” falls far short of actually finding that perchlorate may have an adverse effect on the health of persons.

Given the countervailing scientific information available, the Regulatory Determination stated all that can be said about the health effects of low-level perchlorate exposure. And, it falls well short of what is required by the Safe Drinking Water Act to regulate perchlorate. Current

⁹⁹ 76 Fed. Reg. 7763.

¹⁰⁰ *Id.*

¹⁰¹ *Id.*

¹⁰² *Id.*

¹⁰³ *Id.*

¹⁰⁴ *Id.*

science establishes that low-level perchlorate exposure is insignificant in light of the other more important thyroid stressors—thiocyanate, nitrate and iodide deficiency. Moreover, the NAS concluded that the “only effect” that low-level perchlorate exposure would cause is minor and transient inhibition of iodide uptake and that inhibition of iodide uptake is not an adverse effect.¹⁰⁵ Particularly in light of these facts, it is telling that the only sentence in the Regulatory Determination that attempts to actually address the health effects finding is stated in such tentative terms. The Regulatory Determination simply does not support a finding that low-level perchlorate exposure has an adverse effect on human health.

2. *There Are No Known Instances of Adverse Effects from Low-Level Perchlorate Exposure*

Inhibition of iodide uptake is the only effect that has been consistently documented in humans from low-level perchlorate exposure.¹⁰⁶ However, the inhibition of iodide uptake and the resultant transient changes in thyroid hormone production are not adverse health effects.¹⁰⁷ Rather, they are biochemical changes that may (or may not) precede adverse effects.¹⁰⁸ No cases of adverse effects from exposure to perchlorate at environmental levels in the United States have been documented.

The mode of action model set forth in the NAS report contains the following steps, each progressing from the prior step: (1) perchlorate exposure; (2) perchlorate in blood; (3) inhibition of iodide uptake; (4) changes in levels of thyroid hormones; (5) thyroid hypertrophy or hyperplasia; (6) hypothyroidism; and (7) abnormal fetal and child development and/or adverse metabolic changes (at any age).¹⁰⁹ Others have suggested that abnormal fetal development can precede hypothyroidism.¹¹⁰ The NAS stated that the first three events in this sequence have been observed in humans as a result of low-level perchlorate exposure.¹¹¹ However, none of the events in Steps 4 through 7 have been clearly observed in humans exposed to perchlorate (although they are biologically plausible in the absence of adequate compensation).¹¹²

The NAS further explained that “thyroid hormone production must fall substantially and, more importantly, must remain low for a prolonged period for adverse effects to occur.”¹¹³ Continuing, the NAS also stated that the “minimum extent and duration of the fall in thyroid

¹⁰⁵ NAS, *supra*, n. 7, at 13-15.

¹⁰⁶ *Id.*

¹⁰⁷ *Id.*

¹⁰⁸ *Id.*

¹⁰⁹ *Id.* at 13-16.

¹¹⁰ OIG, *supra*, n. 71, at 48.

¹¹¹ NAS, *supra*, n. 7, at 13-14.

¹¹² *Id.*

¹¹³ *Id.* at 51.

hormone production that has adverse effects are not known.”¹¹⁴ The NAS then concluded that it would likely take essentially therapeutic doses of perchlorate—well out of the range of concentrations that have been observed in the environment—to cause an adverse effect on the health of persons. NAS stated:

*[I]t is highly likely that in people with a normal iodide intake that doses of perchlorate would have to reduce thyroid iodide uptake by at least 75% for a sustained period (several months or longer) for iodide uptake and thyroid hormone production to decline enough to cause adverse health effects (equivalent to reducing dietary iodide by 75%). In adults, that is likely to require sustained exposure to more than 30 mg of perchlorate per day (0.4 mg/kg per day for a 70-kg person), on the basis of the clinical studies in healthy subjects and the studies of long-term treatment of hyperthyroidism.*¹¹⁵

That level of perchlorate exposure would be the equivalent to a drinking water standard of 14,000 ppb.

The Regulatory Determination correctly isolates the issue of whether “iodide uptake inhibition from **low-level perchlorate exposure**” will have an adverse effect on human health.¹¹⁶ However, there is no evidence that such low-level perchlorate exposure (i.e., exposure to concentrations found in the environment) will have an adverse effect on human health. It should be noted that several cross-sectional studies have reported associations between low-level perchlorate exposures and thyroid hormone levels in certain study subsets.¹¹⁷ However, the effects noted were not clinically significant and the gender differences noted in the studies do not appear to be biologically plausible.¹¹⁸ Other cross-sectional studies found no associations.¹¹⁹

The Greer Study and the other clinical studies reviewed by the NAS demonstrate that low-level perchlorate exposure will **not** have an adverse effect on the health of persons. The NAS developed an RfD based on a no-observed-effects-level and applied an uncertainly factor of 10 to account for the most susceptible subpopulations. The RfD developed by the NAS translates to a drinking water equivalent level of 24.5 ppb. At this low level of exposure, not only are there no adverse effects—there are no effects at all.

¹¹⁴ *Id.*

¹¹⁵ *Id.* at 66-67.

¹¹⁶ 76 Fed. Reg. 7762 (emphasis added).

¹¹⁷ E.g., Blount BC, Pirkle JL, Osterloh JD, et al., 2006. *Urinary Perchlorate and Thyroid Hormone Levels in Adolescent and Adult Men and Women Living in the United States*. Environ. Health Perspect. 114(12):1865-1871.

¹¹⁸ ATSDR, *supra*, n. 8, at 59.

¹¹⁹ E.g., Pearce EN, Lazarus JH, Smyth PP, He X, Dall’amico D, Parkes AB, Burns R, Smith DF, Maina A, Bestwick JP, Jooman M, Leung AM, Braverman LE, 2010. *Perchlorate and Thiocyanate Exposure and Thyroid Function in First-Trimester Pregnant Women*, J Clin Endocrinol Metab, v. 95, no. 7:3207-3215.

3. *The Environmental Studies That Directly Address This Issue Conclude That Low-Level Perchlorate Exposures Do Not Have An Adverse Effect on the Health of Persons*

Three studies have examined the health effects of varying levels of perchlorate in drinking water on otherwise similar populations. Two of the studies were conducted in three closely proximate cities in northern Chile: Taltal, where the local water supply contains 100 to 120 ppb of perchlorate; Chanaral, where the local water supply contains 5 to 7 ppb; and Antofagasta, where perchlorate was below the detection limit. One of the studies was conducted in three closely proximate communities in Israel: Morasha, where the local water supply contained upwards of 340 ppb of perchlorate; Ramat Hasharon, where the water contained 42 to 94 ppb; and Hertzlia, where the water contained less than 3 ppb.¹²⁰

In the Crump Study, published in 2000, the researchers found no significant differences in thyroid hormone levels or incidence of thyroid disease among children aged 6 to 8 who were life-long residents of their respective cities in northern Chile.¹²¹ The researchers also found no significant differences in measures of thyroid health among the newborns of mothers resident in the different cities.¹²² The NAS found the design of the Crump Study to have “significant strengths” and the criticisms of the Crump Study to be without merit.¹²³

In the Tellez Study, published in 2005, the researchers studied the effects of environmental perchlorate on the thyroid status of pregnant women and newborns in the same three northern Chilean cities.¹²⁴ Maternal and neonatal thyroid hormones were measured, along with neonatal birth weight, length and head circumference.¹²⁵ The researchers found that perchlorate in drinking water at levels up to 114 ppb did not cause changes in newborn thyroid function or cause fetal growth retardation.¹²⁶

In the third study, the Amitai Study, published in 2007, researchers studied populations exposed to high (up to 340 ppb), moderate (50-95 ppb) and low (below 3 ppb) levels of perchlorate in three closely proximate communities in Israel.¹²⁷ No differences were found among the suite of thyroid health indicators measured.¹²⁸

¹²⁰ NAS, *supra*, n. 7, at 100-101.

¹²¹ *Id.*

¹²² *Id.*

¹²³ *Id.* at 101-105.

¹²⁴ *Id.*

¹²⁵ ATSDR, *supra*, n. 8, at 72-73.

¹²⁶ *Id.* at 73.

¹²⁷ *Id.*

¹²⁸ *Id.*

Each of the three studies determined that the varying levels of perchlorate had no measureable effect on the thyroid status of the populations studied. Significantly, the levels of perchlorate in drinking water in Taltal, Chile (100 to 120 ppb) and Morasha, Israel (up to 340 ppb) are well above environmental levels encountered in the United States. There are no well-designed studies comparing similar populations to varying levels of perchlorate in local drinking water that produce contrary results. These studies are compelling and indicate that the answer to the statutory question of whether perchlorate “may have an adverse effect on the health of persons,” is undeniably: “No.”

4. *Perchlorate is Only One of Several Stressors Affecting Iodide Uptake and is an Immaterial Stressor*

There is general agreement in the scientific community that stressors affecting iodide uptake include: (1) lack of iodide in the diet (iodide deficiency); (2) thiocyanate; (3) nitrate and (4) perchlorate.¹²⁹ The last three of these stressors act in the same way—thiocyanate, nitrate and perchlorate all compete with iodide for uptake into the thyroid gland, thereby decreasing iodide uptake. Lack of iodide is a more direct stressor—if the diet lacks sufficient iodide, there is less iodide available for uptake into the thyroid gland in the first place.

OIG performed a cumulative risk assessment that took into account all four stressors. OIG concluded that iodide deficiency “is the dominant stressor in this public health issue.”¹³⁰ Stated differently, whether an individual has adequate iodide is determined more by the amount of iodide in their diet than by exposure to any of the other stressors (thiocyanate, nitrate or perchlorate). The only real cure for a lack of iodide in the diet is iodide supplementation (or a better diet).¹³¹ In its report, NAS recommended that iodide be added to all prenatal vitamins and that the federal perchlorate Interagency Working Group (IWG) address this issue.¹³² OIG indicates that these recommendations have not yet been acted upon.¹³³

OIG concluded that thiocyanate “at typical exposure concentrations is a *medium impact* stressor in this public health issue.”¹³⁴ OIG also concluded that nitrate “at typical exposure concentrations is a *weak impact* stressor.”¹³⁵ Finally, OIG concluded that at the RfD exposure level (about 24.5 ppb in drinking water), perchlorate “is a *very weak impact* stressor.”¹³⁶ OIG notes several findings supporting these conclusions. Among them, the Food and Drug Administration’s total diet study indicates that “perchlorate is present at very low levels in food,”

¹²⁹ ATSDR, *supra*, n. 8, at 8; OIG, *supra*, n. 72, at 175; 76 Fed. Reg. at 7766.

¹³⁰ OIG, *supra*, n. 71, at 176.

¹³¹ *Id.* at 177.

¹³² NAS, *supra*, n. 7, at 18.

¹³³ OIG, *supra*, n. 71, at 177.

¹³⁴ *Id.* at 181 (emphasis added).

¹³⁵ *Id.* at 182 (emphasis added).

¹³⁶ OIG, *supra*, n. 71, at 183 (emphasis added).

primarily in dairy and vegetables.¹³⁷ In addition, modeling indicates that at median exposure levels observed in biomonitoring data collected by the Centers for Disease Control, perchlorate would account for only 0.09% of the iodide inhibition experienced in the general population, with thiocyanate and nitrate accounting for essentially all of the rest.¹³⁸ Even at perchlorate levels corresponding to the RfD level, perchlorate would account for 0.9% of the iodide inhibition experienced, again with thiocyanate and nitrate accounting for essentially all of the rest.¹³⁹

In sum, the amount of iodide in the diet is the most important determinant of iodide uptake into the thyroid. Setting aside the most important determinant and looking only at the other three stressors, perchlorate is still a “very weak” stressor, accounting for less than 1 percent of the iodide inhibition experienced. Perchlorate does not affect the most important determinant of iodide uptake—the amount of iodide in the diet. And perchlorate does not have a material impact on the other determinant of iodide uptake—exposure to iodide inhibitors. There is no evidence that a 1 percent (or less) component of a secondary set of stressors on iodide uptake can have any effect on human health.

B. Perchlorate Does Not Appear With A Frequency Or At Levels Of Concern In Public Water Supplies

The Regulatory Determination’s conclusion that perchlorate occurs with a frequency and at levels that justify regulation is not supported by the available evidence. First, the UCMR1 data upon which the Regulatory Determination relies is compromised by the large amount of unrepresentative data it contains. Second, perchlorate levels in the Colorado River—the single largest source of drinking water that contains perchlorate—have declined substantially since the UCMR1 data was collected. Third, data from very large public water systems show that perchlorate occurs at significantly lower concentrations than what is reflected in the UCMR1 data set. Finally, several states have taken various corrective measures, and adopted regulatory and advisory levels for perchlorate, all of which have caused perchlorate concentrations to decline since the collection of the UCMR1 data. Chief among these is California, which has stated as long ago as 2004 that essentially no water systems in that state are purveying water above 4 or 6 ppb.

1. Data Regarding the Occurrence of Perchlorate in the Environment

As a result of its discovery in drinking water supplies in the Colorado River and other locations in the southwestern United States, perchlorate was added to the Contaminant Candidate List (CCL) in 1998 and to the monitoring program under the Unregulated Contaminant Monitoring

¹³⁷ *Id.*

¹³⁸ *Id.*

¹³⁹ *Id.*

Rule (UCMR) in 1999.¹⁴⁰ Under the first cycle of the UCMR, known as UCMR1, eleven contaminants, including perchlorate, were monitored at numerous public water systems.¹⁴¹ Although the UCMR1 data was collected over a five-year period from 2001 and 2005, most of the sampling took place between January 2001 and December 2003.¹⁴² In order to detect concentrations of contaminants in drinking water systems, samples were to be collected at “entry points to the distribution system.”¹⁴³

Approximately 1.9% (637) of the 34,331 samples collected pursuant to UCMR1 had positive detections of perchlorate at levels greater than or equal to the detection limit of 4 µg/L.¹⁴⁴ However, nearly one-third (31%) of the UCMR1 samples were collected from untreated source water upstream of the water distribution systems and not from the entry point to the distribution systems.¹⁴⁵ Given the high percentage of untreated source water samples in the database, the UCMR1 data does not actually represent the levels of perchlorate in public water systems in the 2001 to 2003 time frame.

2. *The UCMR1 Data Set Is Not Representative of Conditions Existing in 2001-2003 and is Now Out of Date*

The Regulatory Determination acknowledges that the data upon which it relied in reaching its conclusion as to the occurrence of perchlorate in public water systems is drawn from the UCMR1.¹⁴⁶ The Regulatory Determination treats the UCMR1 data as though it is representative of the quality of water in public water systems from 2001 to 2003. However, the UCMR1 data is not representative of conditions at that time. Nearly one-third (31%) of the samples in the UCMR1 database were collected not from the entry point to the drinking water distribution system, but from untreated source water upstream of the point of entry.¹⁴⁷

¹⁴⁰ U.S. Environmental Protection Agency, *Unregulated Contaminant Monitoring Rule 1 Fact Sheet*; available at <http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/factsheet.cfm> [accessed March 2011].

¹⁴¹ U.S. Environmental Protection Agency, *Unregulated Contaminant Monitoring Regulation: Monitoring for List 1 Contaminants by Large Public Water Systems (January 2001)*, available at http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/upload/2005_08_12_ucmr_ucmr1_fs_ucmr1_monitoring_large_sys.pdf [accessed March 2011]; U.S. Environmental Protection Agency, *Unregulated Contaminant Monitoring Regulation: Monitoring for List 1 Contaminants by Selected Small Public Water Systems (January 2001)*, available at http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/upload/2005_08_12_ucmr_ucmr1_fs_ucmr1_monitoring_small_sys.pdf [accessed March 2011].

¹⁴² *Id.*, Exhibit G at 2, Exhibit H at 2.

¹⁴³ *Id.*

¹⁴⁴ Brandhuber, Philip, *A Review of Perchlorate Occurrence in Public Drinking Water Systems*, *Journal AWWA*, vol. 11, issue 11 (Nov. 2009) at 67.

¹⁴⁵ *Id.*

¹⁴⁶ *Id.*

¹⁴⁷ Brandhuber, Philip, *A Review of Perchlorate Occurrence in Public Drinking Water Systems*, *supra*, n. 10, at 67.

It is also unclear from the UCMR1 database whether the untreated source water sampled was treated before distribution (which would remove essentially all of the perchlorate), blended with other water (which would lower the perchlorate concentrations), or directly purveyed. It is known that the frequency of detections in untreated source water (2.7%) in the UCMR1 database was almost double the frequency of detections in water sampled at the entry point to the distribution systems (1.5%).¹⁴⁸ Presumably, the levels of perchlorate found in the untreated source water samples were also higher than in the water sampled at the entry point to the distribution systems. For these reasons, it is apparent that the UCMR1 database does not represent the frequency and levels of occurrence of perchlorate in the nation’s drinking water in the 2001 to 2003 time period.

It is also undeniable that the UCMR1 data regarding perchlorate is eight to ten years old. The available evidence indicates both that the UCMR1 data set is unrepresentative of the 2001 to 2003 time period in which it was collected (as explained above), and that perchlorate levels have declined substantially since that time. As a result, the UCMR1 data does not accurately reflect the current levels or frequency of occurrence of perchlorate, making this data an unreliable basis from which to reach any regulatory determination. The staleness of the UCMR1 data might not have been cause for concern if there was an indication that perchlorate concentrations had remained stable over the intervening decade rather than declining substantially.

3. The Colorado River Is The Largest Source of Water Containing Perchlorate and Levels In The River Have Been Declining For A Decade

The Colorado River is a mega-source of drinking water, providing water to over 20 million people—mostly in California, Arizona and Nevada.¹⁴⁹ Perchlorate was discovered in the Colorado River in 1997, and the Colorado River is by far the single largest source of drinking water that contains perchlorate.¹⁵⁰ The discovery of perchlorate in the Colorado River led the Nevada Division of Environmental Protection (NDEP) and others to initiate the Southern Nevada Perchlorate Cleanup Project over a decade ago.¹⁵¹

As a result, concentrations of perchlorate in the Colorado River have been declining for over a decade as well. Perchlorate concentrations in the Las Vegas Wash have “decreased by more than 90 percent since 1997,” and sampling data from the Willow Beach, Arizona sampling point on the Colorado River, approximately 11 miles downstream from Hoover Dam, “shows a reduction from a height of 9.7 ppb in June 1999 to 1.8 ppb in May 2008.”¹⁵² The concentrations of

¹⁴⁸ Intertox, Inc., *Comments in Response to the EPA Notice [EPA-HQ-OW-2009-2097; FRL-8943—9] Drinking Water: Perchlorate Supplemental Request for Comments* (Oct. 8, 2009) at 24.

¹⁴⁹ U.S. Environmental Protection Agency, *Perchlorate in the Pacific Southwest*, available at http://www.epa.gov/region9/toxic/perchlorate/per_nv.html [accessed March 2011].

¹⁵⁰ *Id.*

¹⁵¹ State of Nevada, Nevada Division of Environmental Protection, Bureau of Corrective Action, *Southern Nevada Perchlorate Cleanup Project*, *supra*, n. 5.

¹⁵² *Id.*

perchlorate in the Colorado River—and therefore to the public water systems that draw from the Colorado River—have declined by more than 80 percent¹⁵³ since the onset of UCMR sampling.

Because the Colorado River is piped to many municipal purveyors, this 80 percent decline necessarily impacts perchlorate concentrations in many water systems across Southern California, Nevada and Arizona.

In sum, because the Regulatory Determination relies on water quality data from UCMR1 which predates nearly all of the 80 percent decline in Colorado River perchlorate concentrations, the Regulatory Determination cannot possibly be taking this decline into account. Given the sheer size of the Colorado River as a water source (serving upwards of 20 million people), this single omission, in and of itself, undermines the entire validity of the second finding.

4. *The Large Public Water Systems Identified as Having Elevated Concentrations in the UCMR1 Data Have Also Experienced Significant Decreases in Perchlorate Concentrations.*

The UCMR1 data identified 11 public water systems with either median or 90th percentile perchlorate concentrations in excess of the current RfD drinking water equivalent level of 24.5 ppb.¹⁵⁴ A summary of the UCMR1 perchlorate data is presented in Appendix A of *National Cost Implications of a Potential Perchlorate Regulation*, published by the American Water Works Association in July 2008.¹⁵⁵ Of the 11 water systems reporting perchlorate concentrations over 24.5 ppb, the three largest were Riverside, Redlands and Pasadena.¹⁵⁶ All of these systems are located in California.¹⁵⁷

Riverside, the largest public water system among the three, serves water to approximately 300,000 people.¹⁵⁸ The UCMR1 data for Riverside shows median perchlorate concentrations for 23 different locations. The *median* values of these 23 locations ranged from below the detection limit up to 42.0 ppb.¹⁵⁹ In sharp contrast, the consumer confidence reports for Riverside show that during the 2001 to 2003 time period when UCMR1 data was collected, the *highest* concentration of perchlorate detected in the Riverside water system was 12 ppb.¹⁶⁰ Even more

¹⁵³ *Perchlorate Concentration and Specific Conductance, Colorado River – Willow Beach AZ Sampling Point*, available at http://ndep.nv.gov/bca/file/perchlorate_willow_beach.xls [accessed March 2011].

¹⁵⁴ American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008) at Appendix A, available at http://www.awwa.org/files/GovtPublicAffairs/PDF/Perchlorate_Treatment_Costs.pdf [accessed March 2011].

¹⁵⁵ *Id.* at Appendix A.

¹⁵⁶ *Id.*

¹⁵⁷ *Id.*

¹⁵⁸ U.S. Environmental Protection Agency, *Where You Live: Your Drinking Water Quality Reports Online*, available at <http://cfpub.epa.gov/safewater/ccr/index.cfm> [accessed March 2011].

¹⁵⁹ American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008), *supra*, n. 111, at Appendix A.

¹⁶⁰ City of Riverside Public Utilities, *Water Quality Reports (2001-2009)*, *supra*, n. 116.

significant, the Riverside Consumer Confidence Reports show that *no perchlorate was detected* in the Riverside system in 2008 or 2009.¹⁶¹ The Consumer Confidence Reports for Riverside report the following perchlorate levels over the last decade:

2001 Average: 6.4 ppb. Range: <4 to 12 ppb.
2002 Average: 4.6 ppb. Range: <4 to 11 ppb.
2003 Average: 2.3 ppb. Range: <4 to 7.2 ppb.
2004 Average: 1.7 ppb. Range: <4 to 4.8 ppb.
2005 Average: 2.6 ppb. Range: <4 to 4.5 ppb.
2006 Average: 2.2 ppb. Range: < 4 ppb.
2007 Average: 2 ppb. Range: ND to < 4 ppb.
2008 Average: ND. Range: ND.
2009 Average: ND. Range: ND.¹⁶²

The data for 2010 is not yet available.¹⁶³ Public water system operators are required to provide Consumer Confidence Reports to all of their customers each calendar year, setting forth the range and average concentrations in the water purveyed.¹⁶⁴

The Riverside Consumer Confidence Reports clearly show steadily declining concentrations of perchlorate in the 2001 to 2009 time period.¹⁶⁵ Two things are therefore clear. First, the UCMR1 data is not consistent with the data in the Riverside consumer confidence reports during the 2001 to 2003 time period. Second, and even more important for purposes of whether or not the determination to regulate was warranted, there is currently no perchlorate in the Riverside system—the largest public water system in which perchlorate was reported to be present at concentrations over the RfD level of 24.5 ppb in the UCMR1 data. Consumer Confidence Reports for Redlands and Pasadena also show levels of perchlorate lower than reported in UCMR1.¹⁶⁶

For Redlands, the UCMR1 data shows median perchlorate concentrations for 16 different locations.¹⁶⁷ The *median* values ranged from below detection limits up to 62.0 ppb.¹⁶⁸ In

¹⁶¹ *Id.*

¹⁶² City of Riverside Public Utilities, *Water Quality Reports (2001-2009)*, available at <http://www.riversideca.gov/utilities/water-wqr.asp> [accessed March 2011].

¹⁶³ *Id.*

¹⁶⁴ 42 U.S.C. §300g-3(c)(4); 40 CFR §141.151, *et seq.*

¹⁶⁵ *Id.*

¹⁶⁶ Pasadena Water and Power, *Water Quality Reports (2001-2009)*, available at <http://ww2.cityofpasadena.net/waterandpower/waterquality/default.asp> [accessed March 2011]; City of Redlands, Redlands Municipal Utilities Department, *Consumer Confidence Reports (2001-2010)*, available at http://www.ci.redlands.ca.us/utilities/water_report.htm [accessed March 2011].

¹⁶⁷ American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008), *supra*, n. 111, at Appendix A.

¹⁶⁸ *Id.*

contrast once again, the consumer confidence reports for Redlands show that during the 2001 to 2003 time period when UCMR1 data was collected, the **highest** concentration of perchlorate detected in the water system was 9 ppb.¹⁶⁹ Again, the UCMR1 data is not consistent with the Redlands consumer confidence reports. In addition, the consumer confidence reports from 2008, 2009, and 2010 show that perchlorate in the Redlands system ranged from below detection limits to 4.7 ppb, values significantly below those contained in the UCMR1 database for Redlands.

For Pasadena, the UCMR1 data shows median perchlorate concentrations for 10 different locations.¹⁷⁰ The **median** values ranged from 2.5 to 31.5 ppb.¹⁷¹ By contrast, the consumer confidence reports for Pasadena show that the **highest** concentration of perchlorate recorded in Pasadena over the last decade was 13.2 ppb.¹⁷² Once again, this evidence indicates both that the UCMR1 data is inconsistent with other data collected during the same time period, and that the UCMR1 data is not an accurate reflection of current conditions.

The data from the three largest public water systems for which UCMR1 data showed perchlorate concentrations in excess of 24.5 ppb indicates that the frequency and levels of perchlorate in drinking water systems is significantly lower than indicated by the UCMR1 data. It appears that the UCMR1 data did not accurately represent perchlorate frequency and levels in the 2001 to 2003 time period. In any event, the concentrations reported in the consumer confidence reports are significantly lower than what appears in the UCMR1 database. As stated previously, water purveyors are required to sample their water and provide annual reports to consumers of the levels of contaminants detected in purveyed water.¹⁷³ As the EPA has access to these reports, the information is readily available and should have been reviewed for purposes of making its regulatory determination. Indeed, EPA hosts a webpage on which one can search for consumer confidence reports nationwide.¹⁷⁴ Regulatory Determinations cannot be based on 8 to 10 year old information, particularly when more recent, more accurate, and thus more relevant, information is readily available. This is especially the case where the most recent data tends to show that federal regulation is in fact not necessary as perchlorate is **not** present at a frequency and levels of concern in public water systems.

¹⁶⁹ City of Redlands, Redlands Municipal Utilities Department, *Consumer Confidence Reports (2001-2010)*, *supra*, n. 118.

¹⁷⁰ American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008), *supra*, n. 111, at Appendix A.

¹⁷¹ *Id.*

¹⁷² Pasadena Water and Power, *Water Quality Reports (2001-2009)*, *supra*, n. 118.

¹⁷³ 42 U.S.C. §300g-3(c)(4).

¹⁷⁴ U.S. Environmental Protection Agency, *Where You Live: Your Drinking Water Quality Reports Online*, available at <http://cfpub.epa.gov/safewater/ccr/index.cfm> [accessed March 2011].

5. *State-Based Efforts To Reduce and Remove Perchlorate From Public Water Supplies Since The Late 1990's Have Reduced Perchlorate Levels and the Frequency of Perchlorate Detection.*

A number of states – based on their local circumstances - have taken regulatory action on perchlorate already. Arizona, California, Maryland, Massachusetts, Nevada, New Mexico, New York and Texas, have all adopted advisory or regulatory levels for perchlorate and/or taken other corrective measures.¹⁷⁵

In March 2004, the California Office of Environmental Health Hazard Assessment (OEHHA) published a public health goal for perchlorate of 6 ppb.¹⁷⁶ California later established a state MCL of 6 ppb.¹⁷⁷ Also in March 2004, California OEHHA published an advisory containing “frequently asked questions” regarding perchlorate and the new public health goal. One of the questions addressed in the advisory was: “Does perchlorate make California’s drinking water systems unsafe?” California OEHHA answered, “No,” and then explained:

*Drinking water suppliers are monitoring their water for perchlorate as required by DHS, which has the authority to order suppliers not to provide water with unsafe levels of perchlorate. Since January 2002, DHS has had an “action level” (an advisory level for contaminants for which there is no drinking water standard) of 4 ppb for perchlorate. (DHS has aligned the action level to the 6 ppb PHG.) While perchlorate has been detected at or above the 4 ppb level in almost 350 drinking water sources (primarily ground water wells) statewide, **drinking water providers have taken action to ensure that very few drinking water systems are providing water with perchlorate levels exceeding 4 ppb.** Perchlorate levels in the Colorado River, a major source of drinking water for Southern California, currently [in 2004] range from 4 to 6 ppb (U.S. EPA, Region 9, Perchlorate Monitoring Results, Henderson, Nevada to the Lower Colorado River, December 2003 report), which are within the level of the PHG. The water is blended with water from other sources to reduce perchlorate levels prior to delivery to the public.¹⁷⁸*

A review of the UCMR1 data indicates that more samples were collected from California water systems than from any other state.¹⁷⁹ It appears that approximately half or more of the UCMR1

¹⁷⁵ *State Perchlorate Advisory Levels as of 4/20/05*, available at <http://www.epa.gov/fedfac/pdf/stateadvisorylevels.pdf> [accessed March 2011].

¹⁷⁶ California Office of Environmental Health Hazard Assessment, *Frequently Asked Questions about the Public Health Goal for Perchlorate*, (March 11, 2004) (hereinafter, “OEHHA Fact Sheet”), available at http://oehha.ca.gov/public_info/facts/pdf/PerchlorateFAQfinal.pdf [accessed March 2011].

¹⁷⁷ *State Perchlorate Advisory Levels as of 4/20/05*, *supra*, n. 107.

¹⁷⁸ OEHHA Fact Sheet, *supra*, n. 108, at 6.

¹⁷⁹ American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008), *supra*, n. 111, at Appendix A.

samples that detected perchlorate were collected in California.¹⁸⁰ However, California OEHHA stated that, by 2004, very few water systems were providing water with perchlorate at concentrations exceeding 4 ppb.¹⁸¹ And again, although it cannot be discerned with certainty, it appears that there are hundreds of samples from California on the UCMR1 database at levels well above 4 ppb, including many above 10 ppb and some as high as 66.6 ppb.¹⁸² Thus, it is clear that the data upon which the Regulatory Determination is based does not accurately reflect current conditions.

In addition to California, a number of states have published perchlorate advisory and reporting levels, and have taken steps to monitor, and ultimately reduce and/or remove perchlorate from their water supplies.¹⁸³ Most of these activities took place after the onset and/or completion of the UCMR1 data collection effort. It is to be expected that these state efforts were at least partially effective in decreasing perchlorate concentrations in drinking water. However, because the Regulatory Determination relied only on UCMR1 data, none of these state-based efforts—or their impacts — were taken into consideration when assessing the second finding regarding the frequency and levels of perchlorate detection.

C. A Drinking Water Regulation For Perchlorate Will Result In No Public Health Benefit

The third finding that must be made before a determination to regulate is reached under the Safe Drinking Water Act is whether “in the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.”¹⁸⁴ The Regulatory Determination purports to make this finding.¹⁸⁵ There are, however, a number of serious problems with respect to this finding. First, the Regulatory Determination contains no analysis to support the finding. Second, the comprehensive study undertaken by the NAS demonstrated that perchlorate causes no effect at environmental levels. Third, because perchlorate is only a very weak stressor regarding iodide uptake, regulating perchlorate in isolation will provide no public health benefit.

1. *The Regulatory Determination Provides No Support For The Third Finding.*

Table 2 in the Regulatory Determination contains estimates, based on the UCMR1 data, of the number of people who were purportedly exposed to perchlorate in drinking water above various levels (4 ppb, 6 ppb, 9 ppb, 14 ppb, 19 ppb and 23 ppb) on at least one occasion.¹⁸⁶ For example,

¹⁸⁰ *Id.*

¹⁸¹ OEHHA Fact Sheet, *supra* n. 108, at 6.

¹⁸² American Water Works Association, *National Cost Implications of Potential Perchlorate Regulations* (July 2008), *supra*, n. 111, at Appendix A.

¹⁸³ *State Perchlorate Advisory Levels as of 4/20/05*, *supra*, n. 107.

¹⁸⁴ 42 U.S.C. §300g-1(b)(1)(A)(iii).

¹⁸⁵ 76 Fed. Reg. 7765.

¹⁸⁶ *Id.*

Table 2 purports to show that, using a central value estimate, approximately 400,000 people were exposed to perchlorate above 23 ppb on at least one occasion between 2001 and 2003; and approximately 3 million people were purportedly exposed to perchlorate above 6 ppb on at least one occasion.¹⁸⁷ (High end estimates are higher.) The Regulatory Determination then states that:

EPA has determined that a [drinking water regulation] for perchlorate [1] could reduce perchlorate exposures for these populations to levels below the potential alternative HRLs [levels] that EPA has identified as levels of public health concern for purposes of this determination, and [2] that such exposure reductions present a meaningful opportunity for the reduction of health risks for persons served by PWSs [public water systems].¹⁸⁸

This is not an excerpt—this is the *entire* analysis offered to support the third finding. Although the language used is overly complex, the concepts are simple. The first clause above means that, for example, if an MCL were to be set at 23 ppb, perchlorate exposures could be reduced to levels below 23 ppb; and if an MCL were to be set at 6 ppb, perchlorate exposures could be reduced to levels below 6 ppb. This is an unremarkable observation and is simply a statement of the general effect of establishing and enforcing an MCL.

The second clause states that reducing perchlorate exposures to levels below 23 ppb (or one of the other levels appearing in Table 2) presents “a meaningful opportunity for the reduction in health risks.”¹⁸⁹ This second clause is wholly conclusory and does not follow from the first clause. What is required to make the third finding (“meaningful opportunity for the reduction of health risks”) is some analysis of: (1) the current health risks posed by exposure to perchlorate through drinking water; (2) whether those risks would be reduced by establishing an MCL; and (3) whether this potential reduction would be meaningful. No such analysis appears in the Regulatory Determination. The remainder of the text in the Regulatory Determination regarding the third finding simply describes how the UCMR1 data is presented.¹⁹⁰ There is no discussion as to whether regulating perchlorate will reduce health risks. Nor is there any discussion as to whether, even if such a regulation might theoretically reduce health risks, that reduction would be meaningful.

The observation that setting an MCL at a certain level could reduce exposures above that level, coupled with a conclusory statement that “such exposure reductions present a meaningful opportunity for the reduction in health risks” does not satisfy the third finding from the Safe

¹⁸⁷ *Id.*

¹⁸⁸ *Id.*

¹⁸⁹ *Id.*

¹⁹⁰ *Id.*

Drinking Water Act.¹⁹¹ Indeed, this one-two combination of: (1) a statement of the obvious and (2) a non-sequitur stating the ultimate conclusion, could be used to justify regulating anything.

2. *The Comprehensive NAS Study Demonstrates That Perchlorate Causes No Adverse Effect to Human Health at Environmental Levels.*

As addressed above, in a comprehensive report published in 2005, the NAS concluded that perchlorate has no effect whatsoever in healthy adults at doses of 0.007 mg/kg-day.¹⁹² This conclusion was based on the results of five clinical studies, the results of which were “remarkably similar,” with further support provided by environmental and worker studies.¹⁹³ This dose (0.007 mg/kg-day) translates into a drinking water equivalent of 245 ppb, using standard risk assessment assumptions. The 245 ppb level is not the level at which adverse effects begin to occur—it is the level at which *any effect at all* begins to occur. The effect in this case is the inhibition of iodide uptake into the thyroid gland. This effect itself is not adverse—it is an effect for which the body fully compensates through several well understood mechanisms.

To account for exposures to the most susceptible populations—pregnant women and their fetuses—the NAS included an uncertainty factor of 10.¹⁹⁴ This results in an RfD that translates into a drinking water equivalent of 24.5 ppb. In a report published in late 2010, OIG used a different methodology to reassess whether the 24.5 ppb level was protective of the most susceptible populations.¹⁹⁵ OIG concluded that 24.5 ppb level was conservative and health protective by a factor ranging from 3 to 10.¹⁹⁶

There has been some discussion as to whether the 24.5 ppb level should be adjusted for “relative source contribution.”¹⁹⁷ That is, whether the 24.5 ppb level should be adjusted downward to account for the fact that people are exposed to perchlorate not just through drinking water, but through food as well. EPA established a health reference level (HRL) of 15 ppb to account for a relative source contribution from food.¹⁹⁸ However, it is clear from reference to the study design for the Greer Study, and the other clinical studies, that an adjustment for relative source contribution is inappropriate. This is because the study subjects were “free living, eating a self-selected diet.”¹⁹⁹ This means that the study subjects were fully exposed to perchlorate (and thiocyanate and nitrate) found in food because they ate what they chose. If the study subjects

¹⁹¹ *Id.*

¹⁹² NAS, *supra*, n. 7, at 15.

¹⁹³ *Id.* at 16, n. 4.

¹⁹⁴ *Id.* at 16.

¹⁹⁵ OIG, *supra*, n. 71, at 49-60.

¹⁹⁶ *Id.*

¹⁹⁷ 73 Fed. Reg. 60276-60277 (Oct. 10, 2008).

¹⁹⁸ *Id.*

¹⁹⁹ NAS, *supra*, n. 7, at 65.

had eaten a prescribed diet free of perchlorate, then a relative source contribution adjustment might be necessary. Here, no adjustment is necessary and the 24.5 ppb number derived from the RfD represents a level at which no effects whatsoever occur in the most sensitive subpopulations.

According to the Regulatory Determination, it is estimated (central value estimate) that 400,000 people were exposed to perchlorate in drinking water at levels above 23 ppb at least one time.²⁰⁰ A more relevant question is: How many people are *currently* exposed to perchlorate in drinking water at levels above 24.5 ppb (a level at which no effects occur) for prolonged periods of time? As the NAS observed, “thyroid hormone production must fall substantially and, more important, must remain low for a prolonged period for adverse effects to occur.”²⁰¹

It is likely that no one knows the answer to this question. First, concentrations of perchlorate in drinking water have been declining since the UCMR1 data was collected. Wells have been taken out of service, treatment has been added, concentrations in the Colorado River (a mega-source of drinking water) have declined, state standards have been implemented, and key cities report perchlorate levels in their consumer confidence reports much lower than the UCMR1 data reflects. Second, the 400,000 number was based on one-time, worst-case sample results. It is not clear what the number would have been if it had been developed based on averages or some other number reflective of the fact that thyroid hormone production “must remain low for a prolonged period for adverse effects to occur.”²⁰² Third, the derivation of the 400,000 number is unclear, as attempts by independent consultants to reproduce it have been unsuccessful.²⁰³

The NAS established an RfD that is protective of the most susceptible populations. ATSDR has concurred with that RfD.²⁰⁴ That RfD translates to a drinking water equivalent of 24.5 ppb. OIG recently reaffirmed that the 24.5 ppb number is protective of the most susceptible populations.²⁰⁵ The 24.5 ppb number protects not just against adverse effects, but against any effects whatsoever. No one knows how many people are currently exposed to perchlorate in drinking water above 24.5 ppb for prolonged periods. It is known that the number is less than 400,000, and the number may be much lower than 400,000. It is plausible, given the corrective measures occurring over the past 8 to 10 years, that the number is close to zero. If essentially no one is being exposed to perchlorate in drinking water at levels above which it has no effect whatsoever, there can be no meaningful opportunity to reduce health risks by regulating it.

²⁰⁰ 76 Fed. Reg. 7765.

²⁰¹ NAS, *supra*, n. 7, at 50.

²⁰² *Id.*

²⁰³ Intertox, Inc., *Comments in Response to the EPA Notice [EPA-HQ-OW-2009-2097; FRL-8943—9] Drinking Water: Perchlorate Supplemental Request for Comments* (Oct. 8, 2009), *supra*, n. 175, at 24.

²⁰⁴ ATSDR, *supra*, n. 8, at 23-24.

²⁰⁵ OIG, *supra*, n. 71, at 49-60.

3. *Because Perchlorate Is Only a Very Weak Stressor Regarding Iodide Uptake, Regulating It In Isolation Will Provide No Public Health Benefit*

Perchlorate is a stressor with respect to iodide uptake. The Regulatory Determination states that perchlorate “can interfere with the normal functioning of the thyroid gland by inhibiting the transport of iodide into the thyroid, resulting in a deficiency of iodide in the thyroid.”²⁰⁶ The Regulatory Determination also “acknowledges that nitrate and thiocyanate have the same mode of action as perchlorate, and that the effects of combined exposure to perchlorate, nitrate and thiocyanate are additive.”²⁰⁷

There does not appear to be any scientific dispute that, despite their recognized potencies (with perchlorate being the most potent inhibitor) thiocyanate has the largest effect on inhibiting iodide uptake, nitrate the next largest effect and perchlorate a very small effect.²⁰⁸ This is because in normal environmental conditions, the high amounts of nitrate and thiocyanate present in the diet completely overwhelm perchlorate. To be specific, perchlorate only accounts for about one percent of iodide uptake inhibition in the general population, (if ingested in drinking water at a concentration of 24.5 ppb) with thiocyanate and nitrate accounting for the other 99 percent.²⁰⁹

The amount of thiocyanate in the blood can vary by a factor of seven (10 to 70 umol/L).²¹⁰ The amount of nitrate in the blood can vary by a factor of 14 (10 to 140 umol/L).²¹¹ As compared to these large variations in the more important iodine uptake stressors, a 1 percent variation in the amount of iodide uptake inhibition caused by exposures to perchlorate at the RfD level are insignificant and would be dwarfed by the other natural fluctuations.

One way to place the exposure levels of perchlorate into context is to look at the comparison made by OIG of the effects of perchlorate in drinking water to iodized salt intake. According to OIG, decreasing exposure to perchlorate in drinking water by 18 ppb (from 24.5 to 6.1 ppb), would have the equivalent effect as consumption of an additional 14 milligrams (mg) of iodized salt per day.²¹² A pinch of salt weighs just less than half a gram (460 mg). Thus, the equivalent of the posited decrease in perchlorate exposure would consist of consumption of 1/32 of a pinch of salt per day, or about an extra pinch of iodized salt per month.²¹³ This level of effect is so small that it would be completely lost among the daily and seasonal variations in intake of various vegetables as well as iodized salt consumption. Regulating effects that are on the same

²⁰⁶ 76 Fed. Reg. 7763.

²⁰⁷ 76 Fed. Reg. 7766.

²⁰⁸ OIG, *supra*, n. 71, at 176-84.

²⁰⁹ *Id.* at 183-184.

²¹⁰ *Id.* at 181.

²¹¹ *Id.* at 182.

²¹² *Id.* at 184.

²¹³ *Id.*

scale as small fractions of a pinch of salt cannot logically present a meaningful opportunity for the reduction of health risks.

There are even differences of opinion as to whether iodide deficiency is a public health issue in the United States. NHANES data appear to suggest it is not, and the NAS concluded that any iodine deficiency that may exist in the United States is likely to be mild.²¹⁴ However, one thing is clear: to the extent that iodine deficiency is a public health issue, it cannot be addressed through the regulation of perchlorate in drinking water. Any effect that may be available through this regulatory mechanism is dwarfed by the other iodide uptake stressors. The mechanism that is available to assess the underlying issue is a cumulative risk assessment. The four known stressors of iodide uptake (thiocyanate, nitrate, perchlorate and iodide deficiency) meet EPA’s risk assessment guidance requirements for conducting a cumulative risk assessment by the dose addition method.²¹⁵ In addition:

*EPA has committed to a drinking water strategy that outlines four principles to expand public health protection for drinking water. [citation omitted]. One of these principles is to address contaminants in groups.*²¹⁶

The four known stressors of iodide uptake fit within this drinking water strategy, as all four operate by the same mode of action in an additive fashion.

The Regulatory Determination states that “EPA does not believe that there are sufficient scientific data currently available to assess and characterize the combined risk of these contaminants.”²¹⁷ This statement is undermined by the OIG report, which assessed and characterized the risks of the four iodide uptake stressors and developed conclusions based on its assessment.²¹⁸ The Regulatory Determination’s answer to all of this is a conclusory statement that EPA “does not believe that regulatory action to address perchlorate should be further delayed.”²¹⁹ However, this reaction to the abundance of available scientific information misses the mark. First, the available scientific information does not provide a basis for a determination to regulate perchlorate in the first place. Second, moving forward with a single-constituent analysis in the face of overwhelming evidence that perchlorate is just one of several iodide uptake stressors (and one which accounts for less than 1 percent of total iodide inhibition in typical exposure scenarios) is contrary to EPA’s cumulative risk assessment policy and its policy on addressing drinking water contaminants in groups. Third, if the ultimate goal is to protect public health from risks of insufficient iodide uptake (the only known risk associated with

²¹⁴ NAS, *supra*, n. 7, at 173.

²¹⁵ OIG, *supra*, n. 71, at 186.

²¹⁶ 76 Fed. Reg. 7766.

²¹⁷ *Id.*

²¹⁸ OIG, *supra*, n. 71, at 129-174.

²¹⁹ 76 Fed. Reg. 7766.

perchlorate), then regulating perchlorate in isolation will not address the stated risk. The only way to address the potential public health risk of insufficient iodide uptake is to cumulatively assess all of the stressors that impact iodide uptake.

Conclusion

The best available science is clear and provides the data to demonstrate that perchlorate at environmental levels does not affect public health. Further, a close review of EPA's Regulatory Determination reveals that perchlorate in fact does not satisfy the requirements for regulation set forth in the Safe Drinking Water Act. Most importantly, the unintended consequences of regulation with no attendant public health benefits should be carefully considered, including sufficiency of federal resources to address real public health needs.

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