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California Environmental Protection Agency
1515 Clay St., 16th floor
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Re: Proposed Public Health Goal for Perchlorate in Drinking Water

To Whom It May Concern:

The Association of California Water Agencies (ACWA) appreciates the opportunity to comment on the proposed public health goal (PHG) for perchlorate in drinking water. ACWA represents nearly 450 public water agencies in California that collectively supply 90% of the water delivered in California for domestic, agricultural and industrial uses. Our state enjoys some of the highest water quality in the world and local water agencies are responsible for meeting many stringent federal and state water quality regulations to ensure the water they deliver is clean and safe, including the existing drinking water standard of 6 ppb for perchlorate.

We have the following specific comments on the proposed PHG for perchlorate in drinking water.

Initial comment period

We are disappointed that the proposed revision to the PHG for perchlorate published in January, 2011 does not appear to respond to any of ACWA’s comments or stated concerns as presented in our letter to you dated August 29, 2008. These concerns primarily address the lack of evidence to lower the current PHG, which we feel is health protective of Californians, including sensitive subpopulations. Therefore we reiterate those comments to you below.

Overall comment

Perchlorate is a goitrogen, a chemical that blocks the uptake of iodide to the thyroid, resulting in hypertrophy of the thyroid, i.e. goiter. Sustained iodide deficiency can cause additional effects beyond goiter, including hypothyroidism and, of greatest concern, hypothyroxinemia. Maternal iodide deficiency and hypothyroxinemia during pregnancy and nursing can result in neurodevelopmental deficits in children, historically referred in its more extreme forms as
cretinism. It is this health endpoint that is the key to the determination of the current PHG. OEHHA determined that exposure to drinking water containing less than 6 ppb of perchlorate did not pose an excess public health risk of this outcome and we still believe this is accurate.

**Exposure to Perchlorate through Drinking Water**

The 2007 study by Pearce et al.\(^1\) indicates that perchlorate is actively transported into milk by nursing mothers. While this is certainly true, the study also indicates “Breast milk iodine content was significantly correlated with urinary iodine per gram creatinine and urinary cotinine, but was not significantly correlated with breast milk or urinary perchlorate.” The letter also cites Kirk et al. 2007 which did not find any correlation between perchlorate concentrations in breast milk and iodide concentrations, as did the earlier work by Kirk et al 2005\(^2\). Most interesting, Kirk et al. 2007 reported that drinking water did not appear to be a significant vector for exposure to perchlorate. The study concludes, “The fact that higher levels of perchlorate were present in milk samples from subjects’ drinking water treated by reverse osmosis indicates that drinking water is not necessarily the principal vector for perchlorate exposure.” Moreover, one of these participants (E) used a reverse osmosis system connected to a municipal water supply, which we have repeatedly analyzed: The perchlorate concentration in the feed water ranged from 0 to 4 μg/L, with rare excursions > 2 μg/L. Clearly, her perchlorate intake through drinking water would not account for the observed expression in breast milk. This fact—that drinking water is not generally an important vector for perchlorate exposure—is consistent with measurements of urinary perchlorate versus drinking-water perchlorate reported by Valentin-Blasini et al. 2005\(^3\) (emphasis added).\(^3\) These studies would not indicate that the PHG estimated by OEHHA 5 years ago in any way underestimated the risk.

**Blount study results**

The NGO letter submitted in 2008 cites prominently one specific study, Blount 2007\(^4\), an analysis of the NHANES 2001-2002 study. This study showed a negative association between urine perchlorate concentrations (uncorrected for creatinine) and T4 serum concentrations in women with low urine iodide concentrations. It was not shown that this actually lowered the T4 serum concentration outside of normal concentration range (5 – 12 mcg/dL). Further, Blount reports that the mean serum T4 concentration was 8.4 mcg/dL with a 95% confidence interval covering 7.97 – 8.58 mcg/dL for women aged 12 and over. This means that about 95% of the women in this study had T4 serum concentrations within 5% of the mean and well within the normal range. The study did not provide any indication that any women were hypothyroxinemic, or if they were that these women had lower iodide or higher perchlorate

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concentrations than the other women in the study. Interestingly, Blount found no correlation between iodide urine concentrations and perchlorate concentrations, which is consistent with the breast milk studies cited above. Once more, while informative, this study does not indicate that the current PHG is an underestimate or fails to provide adequate public health protection.

The Blount study found perchlorate in all participants across the United States and numerous studies indicate perchlorate is widely distributed in various food sources and ubiquitous in human exposure studies. However, the USEPA’s Information Collection Rule found perchlorate in relatively few drinking waters (Kimbrough & Parekh 2007) and generally in very small concentrations. This indicates, as research above supports, drinking water is a relatively minor source of human exposure to perchlorate.

Synergistic Effects

Another important issue is the appearance of other NIS inhibitors such as thiocyanate and nitrate that might interact with perchlorate and iodine to affect thyroid hormone levels. Blount et al. 2006 found not just perchlorate but nitrate and thiocyanate in considerable concentrations. This is indeed important as the H&SC states “(C) To the extent information is available, the public health goal shall take into account each of the following factors: (i) Synergistic effects resulting from exposure to, or interaction between, the contaminant and one or more other substances or contaminants.” Both of these chemicals are also goitrogens, just like perchlorate, albeit less potent. Nitrate and thiocyanate are both goitrogens which have been shown to occur in almost all of the subjects in the NHANES 2001-2002 study (Blount et al. 2007) and occur widely in food products. Nitrate is nearly ubiquitous in drinking water (Kimbrough & Parekh 2007). Thiocyanate is thought to be about 1/10th as potent as perchlorate but has a half-life that is considerably longer, 8 hours for perchlorate (Greer et al. 2002) vs. 1–6 days (Junge 1985; Schulz et al. (1979) for thiocyanate. Blount reports that the geometric mean concentration of thiocyanate among study participants was 1,200 mcg/L (95% CI 1,080 – 1,330), while the geometric mean concentration of perchlorate was 2.84 mcg/L (95% CI 2.54 – 3.18). The ratio of the geometric means is 422:1 and converting the thiocyanate into a “perchlorate equivalent concentration” (PEC), the ratio would be 42:1 thiocyanate to perchlorate. Tonacchera et al. (2004) determined the relative potency of perchlorate vs. nitrate to be 1:240 and for the effects of multiple goitrogens to be additive. Blount (2007) reported the geometric mean concentration of nitrate in the NHANES 2001-2002 study to be 38,000 mcg/L (95% CI 35,900 – 40,300) so the ratio of the geometric means of nitrate to perchlorate would be 13,000:1.

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7 Tonacchera, M.; Pinchera, A.; Dimida, A.; Ferrarini, E.; Agretti, P.; Vitti, P.; Santini, F.; Crump, K.; Gibbs, J. Relative potencies and additivity of perchlorate, thiocyanate, nitrate, and iodide on the inhibition of radioactive iodide uptake by the human sodium iodide symporter. Thyroid 14, 1012-1019, 2004
Correcting for the relative potency of nitrate, the PEC ratio of nitrate to perchlorate would be 56:1. Based on the NHANES 2001-02 study results as presented by Blount, the overall “goitrogenic burden” from perchlorate is less than 1% as compared to nitrate and thiocyanate, most of which does not come from drinking water as noted above. It is clear that co-occurring contaminants with similar health effects may have contributed more to the observed outcomes than perchlorate alone. These data do not suggest that the current PHG is excessively high.

The following comments specifically address the January, 2011 draft revised PHG for perchlorate.

Any use of ecological studies (Steinmaus et al. 2010, Buffler et al. 2006, etc.) in OEHHA’s analysis is inappropriate because these studies, by their nature, are greatly flawed in their analysis. Utilizing occurrence data from the CDPH files to assign drinking water perchlorate levels to various California populations is not appropriate because even CDPH indicates the early data is “...helpful in identifying areas in which perchlorate has affected sources of drinking water (principally wells), but they should not be interpreted as representative of water being served by public water systems.” Therefore, the authors could not have accurately taken these data, many of which were not treated or blended waters or even sources in service, and even remotely accurately assigned them to populations. This misunderstanding by the authors on how the monitoring results were obtained and how water systems operate has led to flawed studies that cannot be relied upon.

Additionally, ACWA objects to OEHHA increasing the uncertainty factor applied to infants from 3 to 10. This would seem arbitrary and unwarranted because OEHHA has not demonstrated actual adverse health effects correlated with perchlorate exposure. All of the connections are between perchlorate exposure and hypothesized adverse health effects. Further, there are a large number of epidemiological studies where no adverse health effects were found. The proposed PHG is based entirely on studies like Steinmaus, Greer, and Blount9 where some physiological change is measured, iodide uptake or changes in TSH levels, neither of which is adverse in and of themselves. Even in Blount and Steinmaus, the amount of change in TSH concentration measured was not outside of the normal clinical range, it was just different as compared to controls. By not addressing studies where no effects were found and the inclusion of studies showing only non-adverse health effects, several layers of uncertainty have already been built in to the calculation.

Because OEHHA has also dramatically increased the infant drinking water intake per body weight in this analysis, it is inappropriate to increase the uncertainty factor for infants when OEHHA has already accounted for so much uncertainty. Therefore, ACWA recommends that OEHHA recalculate the health protective concentration for infants as follows:

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8 http://www.cdph.ca.gov/certlic/drinkingwater/Pages/Perchlorate.aspx
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\frac{3.7 \text{ ug/kg-day}}{3} = 1.23 \text{ ug/kg-day} \\
1.23 \text{ ug/kg-day} \times 4.3 \text{ kg-day/L} \times 0.73 \text{ (RSC)} = 3.86 \text{ ug/L} \quad \text{rounded} = 4 \text{ ug/L}
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Lastly, ACWA supports the comments made by East Bay Municipal Utility District (EBMUD) regarding the incorporation of microbiological risks associated with current disinfection practices and the health risk trade-off associated with the production of perchlorate during sodium hypochlorite storage.

ACWA and its member agencies’ highest priority continues to be protecting public health while ensuring a reliable water supply for consumers. We look forward to working with you and the appropriate stakeholders as OEHHA and the California Department of Public Health address this very important issue.

If you have questions, please contact me at 916-441-4545 or danielleb@acwa.com.

Sincerely,

Danielle Blacet
Regulatory Advocate