

**Testimony Relevant to H.R. 1747  
To Amend the Safe Drinking Water Act to Require a National  
Primary Drinking Water Standard for Perchlorate**

**Testimony Provided by Gary L. Ginsberg, Ph.D.  
Connecticut Department of Public Health  
To the House Subcommittee on Environment and Hazardous  
Materials of the Committee on Energy and Commerce  
April 25, 2007**

Thank you for the opportunity to present testimony today on perchlorate health effects and the issue of a perchlorate Maximum Contaminant Level or MCL. My name is Gary Ginsberg. I am a toxicologist at the Connecticut Department of Public Health in Hartford CT where I am involved in environmental risk assessment and standard setting for drinking water, air quality and soil contamination. I am also an adjunct faculty member at Yale University and am an assistant clinical professor in the University of Connecticut School of Community Medicine. I currently serve on a National Academy of Science Panel on Improving USEPA's Risk Assessment Methods and recently completed service on the NAS Panel on Human Biomonitoring. Finally, I serve on the Children's Health Protection Advisory Committee, a group which reports directly to USEPA Administrator Johnson on that agency's efforts to protect children from environmental threats. My publication record is largely in the area of children's vulnerability to toxic chemicals. For more details, see my curriculum vitae (attached). I must also note that my testimony was prepared independently by me and does not represent the official position of the Connecticut Department of Public Health, Yale University or the University of Connecticut.

My testimony today can be summed up by saying: Its **smart** public health policy to regulate perchlorate as quickly as possible. I emphasize the word smart because perchlorate has the potential to make our children less smart and academically left behind. When environmental threats to intelligence have surfaced in the past, action has been taken to remove the threat: lead, mercury, and PCBs are prime examples. Perchlorate is another threat to human intelligence, one in which the evidence may not yet be as compelling as with the iron-clad cases of lead or mercury, but which still represents an important public health concern. It's a risk that warrants protecting public health via the establishment of a drinking water MCL.

Do we have direct evidence of a perchlorate effect on brain function? Yes, that is in laboratory animals where several studies showed that perchlorate dosing in pregnant rats leads to effects on the behavior of offspring (ARL, 1998; Bekkedal, 2000). These effects resulted from perchlorate interference in thyroid function. The National Academy of Science review in 2005 discounted the rat data because rats may be more sensitive to thyroid disruption than humans (NAS, 2005). However, after the NAS review, evidence has emerged that at low exposures common across our population, perchlorate appears to disrupt the thyroid gland in humans (Blount, et al., 2006). That CDC study found an association between perchlorate exposure in the general population and altered thyroid status in the direction you'd expect from an anti-thyroid agent (low thyroid hormone, high TSH). The effect was only seen in women and only in those women with low iodide intake. This increases the concern that pregnant women could be especially at

risk from perchlorate because of their gender and because pregnancy increases demands for many nutrients including iodide (Glinoe, 2001). One may ask why should anyone have low iodide intake when most table salt is iodized? Well the data show that iodide intake in the US population has decreased considerably from where it was in the 1970s (Hollowell, et al., 1998) and this roughly corresponds to our increasing consumption of fast food. Even though fast food is salty, the kind of salt used is not iodized (Wright, 2002). In the CDC study, approximately 36% of the women were in the low iodide, high risk group. So, we have many vulnerable women who, when they become pregnant are passing their vulnerability onto their developing baby in terms of low iodide intake and exposure to perchlorate.

This is important because the developing brain is sensitive to even small changes in thyroid hormone levels. We all know that large decreases in thyroid hormone cause cretinism, severe underdevelopment of the brain (Delange, 1996). Smaller deficiencies can cause more subtle effects, but effects that are still important and measured as lowered IQ and poorer performance in school (Smallridge and Ladenson, 2001). At typical environmental levels of exposure, we don't expect perchlorate to create cretins, but we have every reason to expect perchlorate, if not properly regulated, to erode brain development, learning and intelligence. These effects can be prevented by establishing a perchlorate drinking water standard that is protective of health effects in utero and also in babies once born.

In considering whether to set an MCL, USEPA must review the toxicology and exposure data. Apparently the Agency considers the toxicology data adequate because it has established a reference dose or RfD on its IRIS database. This RfD is the same as that recommended by the 2005 NAS report, and it has been used by EPA's Office of Solid Waste and Emergency Response (OSWER) to establish a Preliminary Remediation Goal at waste sites of 24.5 ug/L. I agree with the Agency that there is a great deal of toxicology data in both animals and humans for the establishment of a health benchmark for perchlorate. However, I disagree with the RfD chosen as it is based upon a small number of human subjects in limited testing (Greer, et al., 2002) and in which it appears that some individuals may have been more sensitive, but that sensitivity was not considered in the RfD derivation. Our commentary published in 2005 critiqued the NAS/EPA RfD , making a case for why it should be lower and more health protective. That commentary is included as an attachment (Ginsberg and Rice, 2005). The October 2006 CDC study is more powerful than the study used by NAS and EPA to set the RfD as it involved thousands of subjects rather than only 30, it divided the population based upon known risk factors including low iodide intake, and it included a reliable exposure measure, urinary levels of perchlorate. That study appears to bear out our concerns about the RfD as the association between perchlorate exposure and impaired thyroid function occurred at background population exposures that are 10 fold below the RfD. This is analyzed in our more recent publication (Ginsberg, et al., 2007), also presented as an attachment. These findings of perchlorate effects in a key subgroup of the population (the 36% of women with low iodide intake) indicates that the stakes are higher than originally thought with perchlorate and that it would be highly imprudent to not regulate

the public's drinking water exposure. The CDC data also make a strong case for an overhaul of the RfD so that it more fully reflects the human epidemiology and laboratory data. Do the data exist to do this? Yes, I believe they do. Are there uncertainties with relying on a single epidemiology study as the primary driver in establishing dose response? Perhaps, but these uncertainties are no greater than those present in the current RfD which is based upon a single study in humans in which the sensitivity of the individuals and of the test method to detect a low dose perchlorate effect is questionable.

The other main component in setting a perchlorate MCL is exposure. One needs to know about exposure to make sure there is enough of it from drinking water to merit USEPA action vis-à-vis setting an MCL. Additionally, one needs to know the various sources of exposure to know what percentage of the daily dose is coming from food and what percentage can be allowed to come from drinking water. The goal of the MCL is to keep the total daily exposure below the RfD. On the issue of data sufficiency for determining the need for an MCL, USEPA's Unregulated Contaminant Monitoring Rule in 2001-2005 required the testing of several thousand public water supplies across the country for perchlorate and found detections averaging approximately 10 ppb in 160 such systems in 26 states. These systems serve 5-17 million people, depending upon how one calculates the distribution of water from these supplies (USEPA, FR 4/11/07). Results from that screening program are summarized in the following figure:

# UCMR Detections in Drinking Water

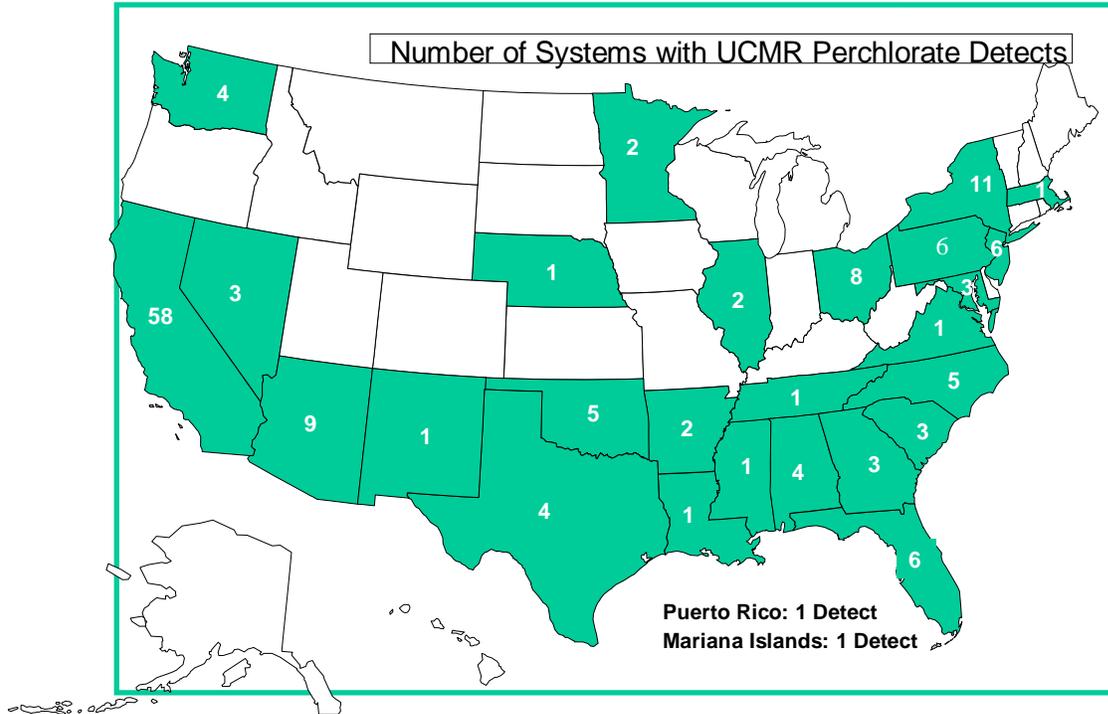


Figure from USEPA Region 9

While these detections are not very common, they are common enough to expose large numbers of people to risky levels of perchlorate. This is only a screen of large public supplies. Smaller systems may be more vulnerable to hot spot groundwater contamination. So the UCMR data may underestimate the number and in some cases, the severity of exposure to perchlorate. For example, data collected in 2004 by the Massachusetts Dept of Environmental Protection (MassDEP) showed that 9 smaller public supplies around the state were contaminated with perchlorate with the highest detection in Boxboro at 1300 ppb. The table below shows this and the other 8

perchlorate detections. The use of perchlorate in blasting projects was clearly the largest risk factor in this limited database.

Since perchlorate has no odor or taste, monitoring programs are needed to uncover contamination. **Without an MCL, there is no monitoring requirement** and so the pregnant women in Boxboro would not know that they are exposing themselves and their developing babies to dangerous levels of this contaminant. Fortunately, in this particular case the state government required proactive sampling and uncovered these perchlorate hotspots and required mitigation.

**Perchlorate Occurrence Monitoring**  
March – October 2004 - Public Water Supply Data Only (693 systems)

| Town         | Maximum (µg/L) | Source?   |
|--------------|----------------|-----------|
| Boxboro      | 1,300          | Blasting  |
| Chesterfield | 8.9            | Fireworks |
| Hadley       | 3.8            | Unknown   |
| Millbury     | 45             | Blasting  |
| Southbridge  | 3.1            | Unknown   |
| Tewksbury    | 3.3            | Industry  |
| Westford     | 3.7            | Blasting  |
| Westport     | 3              | Fireworks |
| Williamstown | 10             | Fireworks |

Massachusetts Department  
of  
ENVIRONMENTAL PROTECTION 

Data from MassDEP Website <http://www.mass.gov/dep/brp/dws/percinfo.htm>

The other side of the exposure coin is how much is coming from drinking water and how much from food. The current database is far from complete but indicates substantial contamination can exist in certain produce as well as in dairy. There are still

datagaps for a variety of other foods. Exactly how this adds up to a dietary background of exposure can take considerable research and time to iron out. It is inappropriate to wait for all types of exposure information before setting an MCL. The standard default assumption when there is an indication of extensive exposure via non-water sources is that 80% of the RfD comes from these non-water sources, thus allowing 20% to come from drinking water. This sets the relative source contribution or RSC at 0.2, a reasonably conservative default that has worked quite well in protecting public health from drinking water contaminants like perchlorate for decades.

Several states have already moved forward and developed a statewide drinking water target for perchlorate. The state MCL in Massachusetts is 2 ug/L, the Public Health Goal in California is 6 ug/L and the target level in New Jersey is 5 ug/L. In each case, exposure and risk experts in state government grappled with the issue of the RSC and were able to make an informed decision on how to proceed. I encourage USEPA to do the same in an expeditious fashion given that undetected and unregulated exposures are ongoing as we speak.

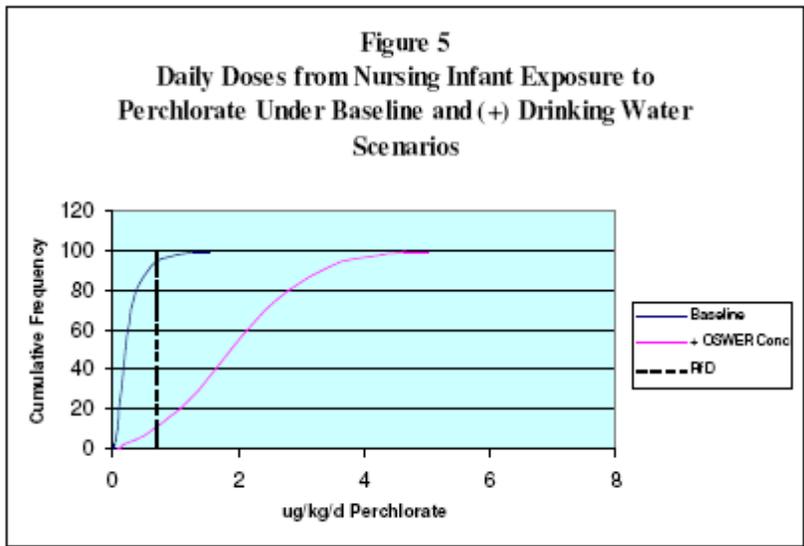
In fact, I point USEPA to our analysis of the CDC data that was published in *Environmental Health Perspectives* in early 2007 (Ginsberg, et al., 2007). In that paper (also attached) we demonstrate the utility of the CDC data in showing the background dietary exposure of the population to perchlorate as converted from the urinary biomonitoring data. We demonstrate that approximately 30% of the current RfD comes from the diet for adults but that because of the greater exposure in nursing infants,

approximately 80% of the RfD dose comes to the infant via the mother's diet. Thus, our analysis supports the default RSC of 0.2, which by the way is the value used in Massachusetts and New Jersey. It is important to remember that if the RfD itself were lowered because of the new CDC data as I describe above, then the RSC would accordingly shift – diet would then take up a larger portion allowing less to come from drinking water. In my judgement, the reasonably conservative and historically accepted default for the RSC of 0.2 is appropriate to enable standard setting to move forward.

It's also important to recognize the special vulnerability of the nursing infant to perchlorate. This was the main subject of our 2007 publication in *Environmental Health Perspectives* (Ginsberg, et al., 2007, attached). We spent considerable effort summarizing the literature that shows that after birth the brain is still growing rapidly and that it is still highly dependent upon thyroid function for proper development. However, in this case it is the infant's own thyroid that is needed as there is virtually no thyroid hormone in breast milk – so the infant is on its own. The baby's thyroid gland does not have a store of thyroid hormone to count on and so must continually make new hormone to keep up with the demands of a rapidly growing being. This keeps the baby's thyroid very busy. To support this, the nursing infant gets all of its iodide needs from breast milk. Perchlorate does two things to interfere with this rather delicate arrangement. First, perchlorate gets pumped into breast milk by the same type of transporter that pumps it into the thyroid gland. This causes the nursing infant to get a substantial dose of perchlorate that can potentially interfere with the baby's thyroid gland and brain development. But the effect of perchlorate is compounded by its inhibition of iodide movement into breast milk. This creates a double jeopardy for the nursing infant – lower

iodide intake at the same time that it is getting a risky level of perchlorate. As we point out in our 2007 paper, it is imperative to fully consider nursing infants when establishing a perchlorate MCL.

Finally I come to the matter of the OSWER PRG for perchlorate of 24.5 ug/L, set by that branch of USEPA in January, 2006. While this is only a preliminary remediation goal, it also takes on the authority of the only federal groundwater/drinking water reference value for use in making site determinations. It effectively says that if the groundwater at a site is below 24.5 ug/L there is little need to analyze the situation further or clean it up. Given the complex array of contaminants and exposure pathways common at Superfund sites, perchlorate will likely not be addressed if its below the OSWER PRG. This federal perchlorate level is also a main subject of our 2007 publication. Our analysis shows that it is set too high to be protective of the developing fetus in utero or the nursing infant. The following figure from our paper shows how the OSWER PRG is likely to push many nursing infants above the RfD, and remember this is the RfD established by the NAS and that doesn't take into account the new CDC data.



The figure shows that under baseline conditions where the only exposure of the mother is to perchlorate in the diet, most of the nursing infants are below the RfD. However, the red line shows the case where mothers are allowed to drink tap water at the OSWER PRG in addition to their background exposure. This pushes most infants well above the RfD. This shows that the OSWER PRG is far from protective of the nursing infant and further, our analysis shows that it is not protective of the developing fetus.

Based upon these risks the OSWER PRG needs to be lowered. In fact, the current situation is about as bad from a public health perspective as possible – not only don't we have a federal MCL, but the only federal guideline we do have is a value from OSWER that puts brain development in the fetus and nursing infant at risk. The vacuum created by not having a federal MCL for perchlorate creates the following public health problems:

- Lack of sampling and detection: without an MCL there is no requirement for public water supplies to test; literally millions of U.S. residents are exposed to perchlorate at potentially adverse levels without the knowledge of drinking water customers or regulators;
- No unifying standard to determine the need for mitigation: in the spotty cases where sampling will occur, results may come in from local areas of contamination; the lack of an MCL requires state or local authorities to develop their own standard; this creates a patchwork of differing values across the country which causes uneven protection of public health and confusion/loss of confidence on the part of the public;
- The default value that currently exists is the OSWER PRG – a value that is clearly inadequate to protect public health.

In summary, thank you for this opportunity to present my perspective on the public health issues surrounding the lack of an MCL for perchlorate. While I would normally support USEPA's science gathering and deliberative process to run its course, I feel that in this case a rich biomonitoring and health effects database is available to move forward towards an MCL. Further, given the immediacy of the public exposures and potential health effects, it is imperative to move forward in a timely manner. While an MCL established in the near future will contain a degree of uncertainty, this would likely be no greater than the uncertainty associated with most other MCLs that are currently in place.

Therefore, I support the bill before the House of Representatives (H.R. 1747) to require the development of an MCL for perchlorate in a timely manner.

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