Rocket Fuel in Drinking Water: New Studies Show Harm From Much Lower Doses

Perchlorate was first discovered to affect the thyroid 50 years ago, but only recently has research focused on its effects at low levels. The trend in the findings is both clear and ominous: The more scientists look, the lower they find the threshold for adverse effects.

Perchlorate's main effect is on the thyroid gland, which is responsible for controlling growth, development and metabolism. Perchlorate inhibits the thyroid's ability to take up iodide, a necessary nutrient, which is important in the production of thyroid hormones. An underactive thyroid gland in adults can lead to fatigue, depression, anxiety, unexplained weight gain, hair loss, and low libido.

More serious, however, are the effects of thyroid hormone disruption in the developing fetus and child: Small changes in maternal thyroid hormone levels during pregnancy have been associated with reduced IQs in children. Fetuses, infants and children who experience more significant changes in hormone levels may suffer mental retardation, loss of hearing and speech, abnormal testicular development or deficits in motor skills. In older children, depressed thyroid levels have been associated with lower motivation to learn and attention deficit disorder. (Haddow et al. 1999, Pop et al. 1999.)

As recently as 1998, only one study had shown any adverse effect of perchlorate at levels as low as 0.01 micrograms per kilogram of drinking water a day. More recent research has shown that even at this very low dose level, perchlorate induces significant effects on the levels of thyroid hormones in the mother, fetus and offspring. (Crofton 2001.) And similar low dose studies have found "a large number of significant effects" on brain structure and thyroid structure. (Argus 2001, EPA 2002.)

A clear trend among the most recent perchlorate studies is more profound effects were seen in laboratory animals who were exposed in utero. This is unsurprising, given that research has shown that marginal iodine deficiency has

significant effects on the fetal thyroid even if no effects are seen in the mother. One study, for instance, found that the thyroids of pregnant rats were able to compensate for marginal iodide deficiency in the diet but that such compensation did not take place in the fetal offspring which showed a 50 percent decrease in iodide uptake. (Versloot et. al. 1997.)

One recent study found thyroid tumors in second-generation exposed rats at extremely young ages. In fact, out of thirty rat pups which were exposed to perchlorate both in utero and as pups, two developed thyroid tumors after just 19 weeks. (Argus 1999.) These types of thyroid tumors are extremely rare, especially when seen this early: The National Institute for Environmental Health Sciences (NIEHS) had estimated the probability of this occurring at less than two-tenths of one percent. (EPA 2002.) Although the dose tested was significantly higher than what might be consumed in drinking water, the findings are troubling because they point to the possibility of in utero programming. This phenomenon, in which fetal exposure leads to a higher susceptibility to hormone perturbation during development and adulthood, has been seen in other cases of endocrine disruption. (Prins et al. 2001, Phillips et al. 1998. Seckl 1997.)

Because iodide is concentrated not only by the thyroid gland, but also by the mammary gland, one of the questions lingering after the peer review of EPA's 1998 provisional reference dose was whether perchlorate would be found or concentrated in breast milk. Recent technological improvements made it possible to look into this question and the results were troubling. Perchlorate was found in the milk of rats that were given very low doses of perchlorate in drinking water (0.01 mg/kg-day), leading the EPA to conclude that rat pups "are in fact exposed to significant levels of perchlorate through the maternal milk." (Yu 2000, EPA 2002.)

Of several epidemiological studies looking at the effects of perchlorate exposure on newborn infants, only two were funded by entities that did not have a significant stake in the outcome of the results. One study, conducted by the Arizona Department of Health Services, found differences in infant thyroid hormone levels among babies in Arizona born to mothers who had consumed

perchlorate-tainted Colorado River water as compared to mothers who had not been exposed to perchlorate during pregnany. (Brechner et al. 2000.)
These findings were confirmed by another recent study of California newborns which found that concentrations of perchlorate at about the same level proposed as a drinking water standard may affect infant thyroid hormone levels. The looked at the hormone levels of all infants born in California in 1996 and compared the hormone levels of infants whose mothers had drunk perchlorate-contaminated water to those whose mothers had not. The study found a statistically significant effect on infant thyroid hormone levels from perchlorate exposure by the mother of only 1 to 2 ppb. The effects were more pronounced at higher dose levels. (Schwartz 2001.)

Studies looking at the ecological effects of perchlorate contamination also raise red flags. For example, one study exposed frog embryos to perchlorate at levels found in some surface and groundwaters. The frogs were found to have inhibited forelimb emergence, significantly lower percentages of animals completing tail resorption, reduced hind limb development, and a skewed sex ratio. (Goleman et al. 2002.) The study notes: "These effects were observed at concentrations at or below concentrations reported in surface waters contaminated with ammonium perchlorate, suggesting that this contaminant may pose a threat to normal development and growth in natural amphibian populations."

Despite all this new evidence, the Department of Defense and the aerospace and defense industry have been trying to get state and federal officials to base perchlorate risk assessments on the results of a single industry-funded human study. This study (Greer et al. 2002) was designed to characterize the effects of a *14-day exposure on adult males and non-pregnant females*. How much its results can be extrapolated to the real world is severely limited by several factors.

- First, the study subjects had healthy thyroids and high iodine intake levels relative to the normal population — whereas 15 percent of U.S. women of reproductive age may have low iodide consumption. (OEHHA 2002.)
- Second, adult humans have an extensive reserve of thyroid hormones which would limit the effects of a short-term exposure, but not the

harm from the lifelong perchlorate exposure that most people would experience.

- Third, the study was not conducted on the population of concern: pregnant women, children and infants. Because pregnancy puts stress on the thyroid, and the hormone reserves that exist in adults are smaller in children and virtually nonexistent in infants and neonates, these populations are much more susceptible to inhibition of iodide uptake (Delange and Ermans 1991.)
- Finally, although the lowest dose tested was designated as a no-effect level in this study, decreased iodide uptake was in fact observed. The EPA has concluded that the study was statistically underpowered to detect significant effects at the lowest dose level, due in part to the small sample size of just seven people. (EPA 2002.)

Despite these numerous and obvious flaws, the Pentagon and industry have used these results to argue for a drinking water standard of 200 ppb.

While state and federal authorities inch towards establishing the first enforceable drinking water standard for perchlorate, an array of proposed standards, action levels" and other proto-standards are circulating. Understandably, this has led to a considerable amount of confusion and frustration within the water-supply industry, which is left with contaminated water and little firm guidance on what level of perchlorate is safe to serve their customers. Unfortunately, none of the proposed standards are likely to be adequate to protect children's health.

California's Office of Environmental Health Hazard Assessment (OEHHA) is explicitly directed to draft Public Health Goals that protect public health including sensitive subpopulations "with an adequate margin of safety." Yet in its most recent draft, OEHHA relies solely on the results of the small industry-funded human study and uses an inadequate uncertainty factor of only three to account for such considerable uncertainties as the variability between individuals, extrapolating from short-term exposure to long-term exposure, extrapolating from the study population to the population of concern and questions of whether perchlorate concentrates in breast milk. (OEHHA 2002.) Of the 35 other Public Health Goals OEHHA has developed for other contaminants, only four have used uncertainity factors of less than 30. Of those four, one is an essential nutrient (copper), one is deliberately added to water (flouride), and the other two have extensive databases on chronic human effects (lead and nitrate).

In developing its provisional reference dose, the EPA has used a wide base of short-and medium-term animal studies which look at the populations of concern, rather than relying on the single short-term human used by OEHHA. The EPA has also proposed an uncertainty factor of 300 — which, given the considerable uncertainty remaining, still may not be large enough. However, the EPA continues to assert that infant body weight and drinking water figures do not need to be used in the calculation of a drinking water standard. (EPA 2002, EPA 2003.) This is unacceptable.

Neither the EPA nor OEHHA have taken into account the numerous common anti-thyroid chemicals that may worsen the effects of perchlorate, notably the drinking water contaminant nitrate. Neither the EPA nor OEHHA seem to be concerned that epidemiological studies have found effects on infant thyroid hormone levels at about the same perchlorate concentrations which have been proposed as a drinking water standard. And neither have the EPA nor OEHHA adequately taken into consideration the extra perchlorate that may be consumed by eating food grown with contaminated water or fertilizer. (EWG 2002.) To adequately protect the health of the nation's children a drinking water standard for perchlorate must be **set no higher than 0.1 part per billion.**

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