Comments on Dec. 2012 OEHHA draft Public Health Goal for PERCHLORATE in Drinking Water

Summary

1) OEHHA's PHG for perchlorate in drinking water is based only on the response of the applied doses from the Greer et al., 2002 critical study. The background goitrogen exposure (perchlorate, nitrate and thiocyanate) has been omitted.
   If these exposure doses would not have been omitted, a **PHG of 45 ppb** perchlorate would have been found for drinking water free of other goitrogens, instead of 1 ppb found by OEHHA.
2) In order to take the goitrogen nitrate into account (which OEHHA does not) the perchlorate PHG for drinking water should be modified into a combined nitrate + perchlorate PHG on the following way:

   PE PHG = [NO3-/150] + [ClO4-] = 45 ppb PEC with PE PHG = Perchlorate equivalent PHG expressed in perchlorate equivalent concentration (PEC).

3) In California, perchlorate's relative contribution to the total goitrogen load in drinking water is only about 3.5% (median). Further the relative contribution of the total perchlorate load (drinking water + food) to the total goitrogen load exposure (drinking water + food) in the US is no more than 0.7% (mean).
   Yet 100% of the risk assessment of potential iodine uptake inhibition at the NIS and the derived PHG is concentrated on perchlorate in drinking water and none on the other goitrogens and environmental sources. In our view this is not justified as such approach is contrary to the principle of the optimization of allocation of resources in order to obtain the best possible health protection for the population.

The Greer et al.,2002 study on perchlorate in adults was used by OEHHA as a critical study to derive a lower bound benchmark dose (BMDL) for adults and from there a public health goal(PHG) for drinking water (DW) (it has also been used by the NRC (National Research Council) as a critical study leading to a RfD (Reference Dose) of 0.7 µg/kg-d and by other states to arrive at their own PHG or MCL (maximum contaminant level for DW).
However the Greer study did not take into account the background level of perchlorate and other competitive inhibitors of iodine uptake such as nitrate and thiocyanate.

We will show that this background level has a significant impact on the OEHHA PHG for perchlorate and any other PHG or MCL for perchlorate based on the Greer study.

1. The importance of other inhibiting anions and the need of their associated evaluation when setting drinking water limits for perchlorate.

I refer to an article by Dr. AM Fan, California EPA, project Director and Final Reviewer of this OEHHA draft PHG for perchlorate[1].
In this article, when commenting on a study about nitrate as a competitive iodine uptake inhibitor at the NIS the following remark is made: [emphasis added] "…*The results are consistent with a* ***common***

[1] AM Fan, Nitrate and Nitrite in drinking water: A toxicological Review, CAL-EPA, 2011, *Encyclopedia of Environmental Health*, 2011 volume 4. , Elsevier, Burlington, pp. 137–145.

mode of action *by these anions of simple competitive interaction, in which* ***any one of the anions, either individually or in a mixture of the three, is indistinguishable from a concentration or dilution of either*** *one of the remaining two ions in inhibiting iodine uptake at the NIS. The current interest in perchlorate findings in drinking water, and the effects of perchlorate on inhibition of iodide uptake by the thyroid, has brought increased attention to comparing the effects on thyroid of nitrate and perchlorate and the* ***associated evaluation as drinking water contaminants and setting drinking water limits."***

The author of this article and final reviewer of the OEHHA's proposed PHG for perchlorate is thus convinced that there is no distinction to be made in the mode of action of those 3 anions except for their inhibitive potency, each contributing in a simply additive (cumulative) fashion to the total goitrogen load.

Indeed the effect of perchlorate cannot be distinguished from the effects of other NIS iodine uptake inhibiting anions[2] [3] [4].

In short the author defends a holistic approach rather than assessing one goitrogen after the other as suggested by OEHHA but which can have unintended consequences as we will show.

2. What could be the reason why the background level has been overseen?

The Greer study was a longitudinal dose/response study (i.e. a series of increasing doses administered to each person participating in the trial) which is exceptional as a tool for estimating maximum allowed exposure and intake levels of environmental contaminants. This kind of clinical studies is normally used for testing pharmaceuticals or for studies on animals and not on humans as was the case here.

In the case of perchlorate however, a longitudinal study could be applied i.e. incremental doses could be administered to humans because the health effect at those doses was known to be a non-adverse effect (and which is not the usual effect from which maximum environmental doses are derived).

Thus, since such longitudinal studies are normally (ethically and legally) not possible, the health risk assessors normally start from a cross-sectional epidemiological dose-response study where the doses are not incremental on top of the background level but where the doses are the background levels themselves.

This may be the reason why background exposure has been overseen in calculating the PHG for perchlorate.

3. Goitrogen background level

3.1. Background perchlorate exposure

3.1.1. *Background perchlorate exposure from food*

Using data from U.S. FDA's Total dietary Survey for 2005-2006 , Murray et al. , 2008[5] estimated an average perchlorate intake from food for adults (which is the sub-population participating in the Greer study) of 0.10 µg/kg-d . Those same data have been used by OEHHA (see table 3, page 10 of the PHG draft).

[2] G. M. Bruce et al., 2013, Urinary Nitrate, Thiocyanate, and Perchlorate and Serum Thyroid Endpoints Based on NHANES 2001 to 2002, *American College of Occupational and Environmental Medicine.*

[3] RE Tarone et al., 2010, The epidemiology of environmental perchlorate exposure and thyroid function: *a comprehensive review. J Occup Environ Med.* 2010; 52;653-660.

[4] B. De Groef et al., 2006, Perchlorate versus other environmental sodium/iodide symporter inhibitors : potential thyroid-related health effects. *Eur J Endocrinol*. 2006; 155;17-25.

[5] CW Murray et al., 2008, US Food and Drug Administration's Total Diet Study: Dietary intake of perchlorate and iodine. *J Expo Sci Environ Epidemiol*. 18 (6):571-80.

3.1.2. Background perchlorate dose from drinking water

Blount *et al*., 2010 [6] estimated an average perchlorate intake from tap water for adults of 0.004 µg/kg-d.

3.1.3. Total background perchlorate dose

Assuming the average results are representative of the typical US adult, these data can be used to estimate the average background dose of perchlorate at the NOEL/BMDL dose of the critical Greer study as follows:

Food background + drinking water background = Total perchlorate background

0.10 µg/kg-d + 0.004 µg/kg-d = **0.104** µg/kg-d

3.2. Background nitrate (in perchlorate-equivalent) exposure estimated from dietary and urinary data

3.2.1. Background nitrate dose from food

The food intake rates from the CSFII (USDA 2010[7]) were combined with literature values for nitrate concentrations in specific foods which resulted in estimated average dietary intakes of nitrate = 1,023 µg/kg-d, for adults > 25y (Table 1 in addendum below).
By comparison, in a population of women age 55 to 69 in Iowa, the median intake of nitrate from dietary sources was estimated to be 1,787 µg/kg-d as nitrate, assuming a 70 kg body weight(Ward et al. 2010[8]), based on a food frequency questionnaire and concentrations reported in the published literature. The mean dietary intake of the highest quartile of participants was 3,457 µg/kg-d as nitrate (Ward et al. 2010[8]).
The average adult intake of nitrate from food in the United States has been estimated at 571 to 1,429 µg/kg-d assuming a 70 kg body weight (OEHHA 1997[9]).
Thus the 1,023 µg/kg-d can be considered as conservative.

3.2.2. Background nitrate dose from drinking water

The geometric mean nitrate level measured in tap water during NHANES 2005-2006 was 1,452 µg/L (Blount et al. 2010[6]). The estimated mean tap water consumption rate for the US adult population (age ≥ 20 yrs) was 11.6 mL/kg-d. Using individual tap water consumption data and body weight, Blount et al. (2010) estimated the median nitrate dose attributable to tap water for this population as 11.3 µg/kg-d (95th percentile intake 290 µg/kg-d).

3.2.3. Background endogenous nitrate

There is, besides an oral exposure, also an endogenous exposure to nitrate estimated at 45% of total nitrate exposure[10].

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[6] BC Blount et al., 2010, Perchlorate, nitrate, and iodide intake trough tap water. *Environ Sci Technol*. 44 (24):9564-70.

[7] USDA (US Department of Agriculture), 2010, Continuing Survey of Food Intakes by Individuals 1994-96, 1998. *Agricultural Research Service, Beltsville Human Nutrition Research Center, Food Surveys Research Group (Beltsville, MD).* http://www.ars.usda.gov/Services/docs.htm?docid=14392

[8] MH Ward et al. 2010. Nitrate intake and the risk of thyroid cancer and thyroid disease. *Epidemiology*. 21(3):389-95.

[9] OEHHA (Office of Environmental Health Hazard Assessment), 1997. Public health goal for nitrate and nitrite in drinking water. *California Environmental Protection Agency, Sacramento, CA.* http://www.oehha.ca.gov/water/phg/pdf/nit2_c.pdf

[10] Nitrate and Nitrite in Drinking Water, page 38, Subcommittee on Nitrate and Nitrite in Drinking Water, Commission on Life Sciences, NRC (National Research Council). Washington, DC, *National Academy Press, (ISBN-10: 0-309-08370-2).*

3.2.4. Total nitrate background dose (exposure)

The total background dose of nitrate is thus estimated as:

Food background + drinking water background + endogenous background

(1,023 + 11.3=1,034) + 1,034/0.55= 1,880 µg/kg-d.

3.2.5. Total nitrate background dose(exposure) in perchlorate equivalent

*1,*880 µg/kg-d / 150) = 12.53 µg/kg-d PEC (perchlorate equivalent concentration)

Note: In molar terms the potency equivalency of perchlorate compared to nitrate is 240/1 (AM Fan, Nitrate and Nitrite in drinking water[1]). Here we apply the weight based equivalency of 150/1 as all data are expressed in such units and not in moles.

Note: Based on urinary measurements from the NHANES 2001-2002 dataset, the geometric mean urinary concentrations of nitrate for adults, ages 20 years and older and excluding lactating women and including smokers was 46,776 µg/g creatinine (n = 1,508) or 45,252µg/g creatinine excluding smokers (n=1,132).This translated into a geometric mean calculated dose of 1,410 µg/kg-d (including smokers) or 1,315 µg/kg-d (excluding smokers) (Table 3 in addendum below). We select the first number as the Greer study did not exclude smokers. This then translates into 1,410 / 150= 9.4 µg/kg-d PEC

Averaging this with the 12.53 µg/kg-d arrived at above we arrive at **10.96** µg/kg-d PEC.

3.3. Background thiocyanate exposure (perchlorate-equivalent) estimated from dietary and urinary data

3.3.1. Background thiocyanate dose from food

The food intake rates from the CSFII (USDA 2010[7]) were combined with literature values for thiocyanate concentrations in specific foods which resulted in estimated average dietary intakes thiocyanate of 7.55 µg/kg-d for adults > 25y (Table 2 in addendum below).

Note: Thiocyanate background exposure estimated from diet is underestimated because of endogenous formation of thiocyanate from cyanide[11] and because of the biological half-life (residence time) of thiocyanate is significantly longer compared (several days) to perchlorate and nitrate (<24h).

3.3.2. Background thiocyanate dose from drinking water

Thiocyanate levels in drinking water are negligible.

3.3.3. Total thiocyanate background dose(exposure) in perchlorate equivalent

The total perchlorate equivalent background dose of thiocyanate was thus estimated as:
(7.55 µg/kg-d /8.8) = 0.86 µg/kg-d PEC

Note: Based on urinary measurements from the NHANES 2001-2002 dataset, the geometric mean urinary concentrations of thiocyanate for adults, ages 20 years and older and excluding lactating

[11] DE. Kimbrough, Relative Source Contribution of Perchlorate and Other Goitrogens in Newborn Thyroid Function, 2011, *American College of Occupational and Environmental Medicine*.

women and including smokers was 1,585 µg/g creatinine (n = 1,508) or 1,096 µg/g creatinine excluding smokers (n=1,132). This translated into a geometric mean calculated dose of 30.1 µg/kg-d (including smokers) or 20.1 µg/kg-d (excluding smokers) (Table 3 in addendum below). We select the first number as the Greer study did not exclude smokers.
Since thiocyanate is a metabolite of cyanogenic glucosinolates that occurs naturally in many vegetables (cabbage and other Brassica sp, sweet potatoes almonds apricots, corn etc.[11]) significant amounts could not be accounted for via the estimated diary intakes.
Therefore we select the urinary NHANES data.
This then translates into 30.1 µg/kg-d / 8.8= **3.4** µg/kg-d PEC

Note: In molar terms the potency equivalency of perchlorate compared to thiocyanate is 15/1[1]. Here we apply the weight based equivalency of 8.8/1 as all data are expressed in such units and not in moles.

3.4. Total goitrogen background exposure in perchlorate equivalent (TGBE)
The total goitrogen background exposure (TBGE) from all 3 sources =0.104 µg/kg-d+10.96 µg/kg-d+3.4 µg/kg-d **=14.46** µg/kg-d PEC (about 0.7% from perchlorate, 76 % from nitrate and 23 % from thiocyanate).

4. Effect of the goitrogen background exposure on the PHG of perchlorate

The Greer 2002 critical study on which OEHHA's lower bound benchmark dose (BMDL),estimated maximum daily dose(ADD), estimated public health-protective concentration(C) and proposed public health goal(PHG) for perchlorate in drinking water is based did not take into account this background dose. Instead OEHHA's BMDL, ADD, C and PHG were based only on the *incremental* doses administered by Greer et al.

Therefore the true ADD = Total goitrogen background exposure in perchlorate equivalent (TGBE) + lower bound benchmark dose/uncertainty factor.

ADD = TGBE +BMDL/UF = 14.46 µg/kg-d +3.7 µg/kg-d/10 = **14.83** µg/kg-d PEC.

Note that no UF (uncertainty factor) should be applied on the background dose. Indeed the UF is intended to compensate for uncertainty in the dose-response relationship leading to the BMDL in order to take into account different and potentially more vulnerable population segments. The BMDL has been derived from the Greer data (increasing doses and corresponding IUI (iodine uptake inhibition) responses). However the background exposure is not a data point in this Greer dose-response.

5. Perchlorate equivalent PHG

Consequently the health-protective concentration (C) for perchlorate in drinking water calculated by OEHHA should be corrected as follows:
C= true ADD µg/kg-d X BW/WC X RSC assuming no other goitrogens are present in drinking water.
where:

BW (body weight), WC (daily water consumption) and RSC (relative source contribution) are kept the same as in the OEHHA draft.

This leads to C and thus **PHG** = 14.83 µg/kg-d x 4.2kg-day/L x0.73= 45.47 µg/L = **45 µg/L(45ppb)** perchlorate instead of 1 µg/L perchlorate.

PHG = 45 ppb perchlorate

A PHG of 45 ppb perchlorate for drinking water (DW) assumes that no other goitrogens causing IUI are present in such water.

This is in fact what OEHHA assumes.

Of course in reality DW contains nitrate. The US mean is 1,452 µg/L nitrate[9]. This is equivalent to about 10 ppb perchlorate which in that case leaves 35 ppb for perchlorate alone.
Thus to be consistent in terms of IUI, the PHG, which now is valid for perchlorate and nitrate and is therefore the PE (perchlorate equivalent) PHG, should read as follows:

PE PHG = [NO3-/150] + [ClO4-] = 45 ppb PEC instead of 1 ppb perchlorate according to OEHHA.

This also implies that, If OEHHA's heath risk assessment would be adopted, the maximum nitrate level allowed in DW to protect against should be 45 x 150/1000= 6.75 ppm and then only when no perchlorate is present.

6. Discussion

6.1. Adding the background goitrogen load is necessary but not enough

The current OEHHA 6 ppb perchlorate MCL and the proposed 1 ppb PHG would thus allow a maximum of 39x 150/1000=5.85ppm or 6.60 ppm nitrate respectively. This implies that the current nitrate MCL of 45 ppm which is based on the OAEL (observed adverse effect) for Methemoglobinemia[1], should be lowered to that level if OEHHA would be consistent in its approach.

Yet if OEHHA would not take into account the background goitrogen load as is the case for this draft PHG then, to remain consistent , the maximum level for nitrate in DW could not even exceed 0.15 ppm nitrate (1 PPB PEC or 150 ppb nitrate) and then only when no perchlorate is present.

As an example assume a 0.5 ppb perchlorate drinking water sample.
This is already on itself about the average concentration released by bleach added to DW and was the sole reason why Massachusetts increased its MCL for perchlorate from 1 to 2 ppb.
Yet 0.5 ppb is also already 57% lower than the US median of 1.16 ppb in tap water[6].
Still it would imply that the maximum level of nitrate in that sample could not be higher than 0.5 x 150/1000= 0.075 ppm in order for the sample to stay at ≤ 1 ppb PEC which is clearly impossible as the current US median for nitrate is 0.76 ppm or 10 times higher[6] and even the purest water contains a higher level (through precipitation of atmospheric nitrate from natural nitrogen cycle).

Moreover, the RSC of DW for lactating women --and thus very relevant for infants(the OEHHA target subpopulation) as well -- is not 0.73 as assumed by OEHHA and assumed up to now in all other publications(i.e. supposed to be similar to that reported for pregnant women) but is recently found to be = 0.05 as a mean (0.01 µg/kg-d out of a total of 0.19 µg/kg-d or 5.3% (table 2, ref. 12)) even though pregnant women consume on average more water than the standard amount of 2l/adult-day [12]. This then implies that the PHG for nitrate should be still further lowered well below 0.075 ppm followed by a further corresponding lowering of the perchlorate PHG. Such pristine water is simply not found on the globe.

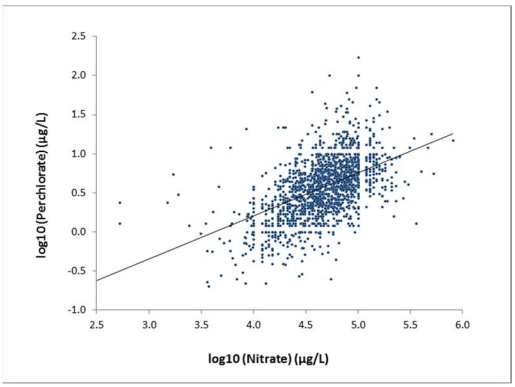
[12] A. Kirk et al., 2013, Relative Source Contributions for Perchlorate in a Lactating Human Cohort, *Science of the Total environment, Elsevier*.

On the contrary it is known that perchlorate levels in DW in California are higher than the national average. And the reality is that this is also the case for nitrate: in California the median concentration of nitrate in drinking water sources from 31,464 samples taken between 1994 to 2003 was 16 ppm(107 ppb PEC) and the average 21 ppm(140 ppb PEC)[11]. Spot samples show that these numbers have not decreased in the meantime. Nitrate was detected at least once above its MCL (300 ppb PEC) in 731 sources in California.
From 2000 to 2011, the median nitrate concentration in the Tulare Lake Basin and Salinas Valley public water supply well samples was 23 mg/L(23 ppm, 345 ppb PEC) and 21 mg/L(315 ppb PEC)[13], respectively, and in all reported non-public well samples, 23 mg/L and 20 mg/L, respectively[13]. In public supply wells, about one in ten raw water samples exceeds the nitrate MCL (675ppb PEC)[13].

Still, according to Blount et al., 2010[6] significant positive correlations were found between nitrate levels and perchlorate levels ($r=0.25$, $p<0.0001$) in tap water.

Indeed plotting nitrate against perchlorate data from NHANES 2001-2002 data shows the following relationship:

Association between perchlorate and nitrate based on urinary measurements from adults ages 20 and older in NHANES 2001-2002



This means that an increase in perchlorate exposure above say 0.5 ppb will be accompanied by a much greater increase in nitrate PEC in absolute terms since as calculated above (§3.4), perchlorate only represents < 1% of the total goitrogen load and nitrate about 76 %(see also intercept in above graph: +2.5 for nitrate against -0.5 for perchlorate (log10 scale)).

Thus one cannot even argue that higher perchlorate level might, hopefully, be compensated by lower nitrate levels. On the contrary the IUI effect of sources with higher perchlorate levels will be multiplied by higher nitrate levels in those same sources.

All this implies that, if OEHHA would hold on to its risk assessment of perchlorate leading to the PHG of 1 ppb it will eventually have to impose infinitesimally low levels for nitrate and ever lower levels for perchlorate, if it would wish to remain consistent in its future goitrogen health risk assessment.

OEHHA (DR. AM Fan, Cal EPA/OEHHA, 2011[1]) already has suggested a future review of the nitrate MCL for drinking water to take into account health effects other than Methemoglobinemia (a) (b)

[13] Center for Watershed Sciences University of California, Davis, 2012, Addressing Nitrate in California's Drinking Water With a Focus on Tulare Lake Basin and Salinas Valley Groundwater, *Report for the State Water Resources Control Board Report to the Legistature.*
(a) The MCL for Methemoglobinemia has been established based on a study from 1947 and hasn't been reviewed since.
(b) An IUI Greer type study on humans has never been done for nitrate.

such as IUI as described above[1]. If so it will then be forced to review again the PHG of perchlorate and raise it significantly to remain consistent.

Why wait? Why not now?

When OEHHA mentions[14] that "*It is argued that it is unfair to regulate perchlorate at a level that does not cause reduction in thyroidal iodide uptake but at the same time allow exposures to nitrate and thiocyanate at levels where such an effect may occur*", we don't think that this has anything to do with "fairness" but with rationality and optimization of allocation of resources to obtain the best possible health protection for the population.
I quote from[2]: "*Assessing risk from multiple chemicals with similar mode of action in risk assessment is not a new concept and is one that has been endorsed by the National Academy of Sciences[15], US Environmental Protection Agency[15] and World Health Organization"[16].*

Indeed California law (California Safe Drinking Act of 1996) requires consideration of the effects of multiple chemicals that act similarly to the extent the information is available[14].
The information *is* available and the 3 anions do not even act similarly but have an identical, well understood and simple mode of action.

6.2. The use and misuse of relative source contribution (RSC) coefficients

The use of RSC coefficients such as the one used in the health-protective concentration(C)equation in §5, in order to weigh the contribution of drinking water as a proportion of overall exposure ,is of course justified.
However it contains a build-in contradiction: the less the contribution of the drinking water source to the overall exposure the more stringent the PHG or MCL of drinking water will end up being.
The rationale behind this is that the higher the exposure from other sources (e.g. food) the lower additional sources such as drinking water should be kept particularly since controlling levels is much easier in water than in e.g. food.

This is rational but only up to a point because when driven to its extreme, an infinitesimal contribution of drinking water will force an infinitesimally low MCL which will cause the relative contribution of drinking water in the decrease of the overall exposure to tend to zero as well but at the price of an infinitesimally high effort because of the law of diminishing returns.
Because of this build-in counter-effect, US EPA and California EPA limit RSCs to 0.2(20 %) and when no data are available apply a default RSC of 0.2 as well.

Unfortunately this principle is not applied when dealing with the goitrogen load

Indeed, as calculated in §3.4 , not only does perchlorate's total background exposure (food + water) in the US represent only about 0.7 % of the total goitrogen load (expressed in PEC) but perchlorate's share in drinking water itself is very low. For example in California(1997-2003) the water sources that contain > 4 ppb perchlorate(reporting limit at that time) contain a median concentration of 7.3 ppb perchlorate and a median nitrate concentration of 31,000 ppm[11] or 207 ppb PEC which means also there perchlorate does not represent more than about 3.5 % of the total PEC load in drinking water either.

[14] D. Ting, RA Howd, AM Fan, and GV Alexeeff, 2006, Development of a Health-Protective Drinking Water Level for Perchlorate, *EHP, Environmental Health Perspectives*, page 23.
[15] MA Callahan et al., 2007, If cumulative risk assessment is the answer, what is the question?, *Environ Health Perspect*. 2007; 115; 799-806.
[16] ME Meek et al., 2011, Risk Assessment of combined exposure to multiple chemicals: a WHO/IPCS framework. *Regul Toxicol Pharmacol*. 2011 April 2

Summarized:
In California, perchlorate's relative contribution to the total goitrogen load in drinking water is only about 3.5% (median). Further the relative contribution of the total perchlorate load (drinking water + food) to the total goitrogen load exposure (drinking water + food) in the US is no more than 0.7% (mean).

Yet 100% of the risk assessment of potential iodine uptake inhibition at the NIS and the derived PHG is concentrated on perchlorate in drinking water and none on the other goitrogens and environmental sources. In our view this is not justified as such approach is contrary to the principle of the optimization of allocation of resources in order to obtain the best possible health protection for the population.

Addendum

Table 1. Nitrate intakes based on the USDA CSFII and nitrate concentrations in specific foods

Food Description	Average US Consumption, Adults 25+ (g/d)[a]	Average Nitrate Concentration (µg/g)[b]	Nitrate Intake Rate (µg/d)
Asparagus, fresh/frozen, boiled	1.2	44	52.8
Beets, canned	0.89	2,400	2,136.0
Bologna (beef/pork)	3.15	105 (ham)	330.8
Broccoli, fresh/frozen, boiled	6.23	740	4,610.2
Brussels sprouts, fresh/frozen, boiled	0.28	120	33.6
Cabbage, fresh, boiled	3.45	520	1,794.0
Cantaloupe, raw/frozen	6.96	360	2,505.6
Carrot, baby, raw	2.98	200	596.0
Carrot, fresh, peeled, boiled	3.25	200	650.0
Cauliflower, fresh/frozen, boiled	1.42	480	681.6
Celery, raw	1.48	2,300	3,404.0
Coleslaw, mayonnaise-type, from grocery/deli	4.84	520	2,516.8
Collards, fresh/frozen, boiled	2.16	800	1,728.0
Corn, canned	3.03	45	136.4
Corn, fresh/frozen, boiled	5.55	45	249.8
Corn/hominy grits, enriched, cooked	3.66	45	164.7
Cucumber, peeled, raw	3.84	110	422.4
Eggplant, fresh, peeled, boiled	1.14	270	307.8
Frankfurter (beef/pork), boiled	4.30	77 (German sausage)	331.1
Green beans, canned	3.61	340	1,227.4
Green beans, fresh/frozen, boiled	5.07	340	1,723.8
Ham, cured (not canned), baked	3.74	150	561.0
Lettuce, iceberg, raw	15.82	1,700	26,894.0
Lettuce, leaf, raw	1.72	1,700	2,924.0
Lima beans, immature, frozen, boiled	1.33	54	71.8
Luncheon meat (chicken/turkey)	4.01	105 (ham)	421.1
Luncheon meat (ham)	5.12	105	537.6
Mushrooms, raw	1.00	160	160.0
Onion, mature, raw	4.36	170	741.2
Peas, green, frozen, boiled	3.64	28	101.9
Pepper, sweet, green, raw	2.61	120	313.2
Pork bacon, oven-cooked	1.79	52	93.1
Pork sausage (link/patty), oven-cooked	2.91	77 (German sausage)	224.1
Potato, boiled (w/out peel)	10.06	110	1,106.6
Potato, mashed, prepared from fresh	15.20	110	1,672.0
Potato, sweet, canned	3.53	46	162.4

Salami, luncheon-meat type (not hard)	2.71	135 (summer sausage)	365.9
Spinach, fresh/frozen, boiled	2.26	1,800	4,068.0
Squash, winter (Hubbard/acorn), fresh/frozen, boiled	1.11	400	444.0
Summer squash, fresh/frozen, boiled	3.04	400	1,216.0
Tomato, raw	15.35	58	890.3
Turnip, fresh/frozen, boiled	0.39	390	152.1
Watermelon, raw/frozen	7.98	360	2,872.8
Total Intake (µg/d)			**71,596**
Total Intake (µg/kg-d)			**1,023**

a 1994-98 CSFII (USDA, 2010)
b NRC, 1981; Choi, 1985

Table 2. Thiocyanate intakes based on the USDA CSFII and thiocyanate concentrations in specific foods

Food Description	Average US Consumption, Adults 25+ (g/d)[a]	Average Thiocyanate Concentration (µg/g) [b]	Thiocyanate Intake Rate (µg/d)
Broccoli, fresh/frozen, boiled	6.23	17.917	111.6
Brussels sprouts, fresh/frozen, boiled	0.28	97.7351	27.4
Cabbage, fresh, boiled	3.45	16.4011	56.6
Carrot, baby, raw	2.98	1.6312	4.9
Carrot, fresh, peeled, boiled	3.25	1.6312	5.3
Cauliflower, fresh/frozen, boiled	1.42	89.1432	126.6
Celery, raw	1.48	0.4692	0.7
Coleslaw, mayonnaise-type, from grocery/deli	4.84	16.4011	79.4
Collards, fresh/frozen, boiled	2.16	1.194	2.6
Cucumber, peeled, raw	3.84	4.6999	18.0
Lettuce, iceberg, raw	15.82	3.9206	62.0
Lettuce, leaf, raw	1.72	3.9206	6.7
Onion, mature, raw	4.36	0.7843	3.4
Spinach, fresh/frozen, boiled	2.26	2.5488	5.8
Tomato, raw	15.35	0.8219	12.6
Turnip, fresh/frozen, boiled	0.39	13.28	5.2
Total Intake (µg/d)			**528.8**
Total Intake (µg/kg-d)			**7.55**

a 1994-98 CSFII (USDA, 2010)
b Sanchez et al. 2007, Olea Serrano et al. 1988, Chandra et al. 2004

Table 3. Mean Urinary Goitrogen Concentrations and Calculated Internal Doses, Adults ≥ 20 Years Old, NHANES 2001-2002

Goitrogen	n	Geometric Mean Urinary Concentration (µg/L) (95% CI)	Geometric Mean Urinary Concentration (µg/g Cr) (95% CI)	Geometric Mean Calculated Internal Dose (µg/kg-d)
Iodine	1,508 [a]	150.1 (139.7-161.2)	153.9 (141.9-166.8)	ND
	1,132 [b]	152.5 (141.9-163.9	162.3 150.9-174.6	ND
Perchlorate	1.508 [a]	3.56 (3.27-3.87)	3.65 (3.37-3.95)	0.0692 (0.0638-0.0752)
	1,132 [b]	3.63 (3.34-3.95(	3.86 (3.54-4.22)	0.0708 (0.0649-0.0771)
Nitrate	1,508 [a]	45,620 (43,655-47,674)	46,776 (44,482-49,188)	1,410 (1,335-1,489)
	1,132 [b]	42,509 (40,797-44,293)	45,252 (42,666-47,994)	1,315 (1,238-1,398)
Thiocyanate	1,508 [a]	1,546.2 (1,429.5-1,672.5)	1,585.4 (1,469.8-1,710.0)	30.1 (27.5-33.0)
	1,132 [b]	1,029.6 (983.9-1,077.5)	1,096 (1,043.1-1,151.7)	20.1 (18.9-21.3)
Total Goitrogen Load in Perchlorate Equivalence		395.5 [a]	405.6[a]	11.2 [a]

ND = Not determined

a Excludes lactating women but includes smokers

b Excludes lactating women and smokers (e.g., those who had smoked within 5 days of urine sample collection) and lactating women

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