Maximum Exposure Guideline

for

Radon in Drinking Water

CAS Registry Number: 10043-92-2

October 2, 2006

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1. Introduction

This document describes the basis for a Maximum Exposure Guideline (MEG) for radon in drinking water. MEGs represent the Maine Center for Disease Control and Prevention's (ME-CDC) recommendations for concentrations of chemicals in drinking water below which there are minimal risks of adverse health effects from lifetime ingestion. MEGs are guidance levels; they are not regulatory standards. ME-CDC has established procedures for developing MEGs that are largely consistent with U.S. Environmental Protection Agency procedures (DHHS, 2000). For a number of reasons discussed in this text, the derivation of an MEG for radon departs from these procedures.

In this document 'radon' refers specifically to ^{222}radon, a naturally occurring radioactive gas found throughout Maine. Radon is radioactive, meaning that it decays into other molecules and releases radiation in the process. The molecules into which radon decays are known as 'radon daughters', and these are also radioactive.

Radon in water can get into the human body via two distinct pathways- ingestion and inhalation. When someone drinks water that contains radon, a radiation dose is delivered to internal organs. Additionally, radon, as a gas, moves from water to air when the water is being used. When radon gas is inhaled, the radon daughters bind to the lung tissue and deliver a radiation dose to the surrounding tissue. Radon that is ingested in water is thought to create a cancer risk, primarily in the stomach. The dominant health concern with radon, however, is the lung cancer risk created when radon gas escapes water and is inhaled. The MEG for radon has been derived in consideration of both pathways of radon exposure.

This document is organized as follows.

1) A description of radon sources and exposure in Maine
2) A review of the evidence showing that radon can cause cancer
3) A review of cancer risk estimates for radon in water
4) A summary of radon guidance from other states and the EPA
5) A proposed MEG for Maine

Although radon gas also enters homes from the soil, this document is specifically focused on radon in water. In the discussion at the end of the document we briefly discuss the impact and treatment implications of total radon exposures.

2. Radon sources and exposures in Maine

Radon is a decay product of uranium-238, a naturally occurring radioactive element. Maine is part of a geological region that is relatively high in natural uranium-238. Radon in soil gas and in water is therefore common in Maine, and random sampling of Maine homes suggests that any location could have high radon from either source. For this reason ME-CDC recommends that every home in Maine with a private well should be tested for radon in both air and water. Since radon is a radionuclide, concentrations in air and water are reported in units of radioactivity: picoCuries per liter (pCi/l)[1].

Private well water in Maine is thought to have radon concentrations, on average, of around 11,000 pCi/l. This is based on data supplied to the Maine Radon Program and is not considered to be random. The data may be biased high due to self-selection bias (people who think they have a problem are more likely to test). The final sample set consists of 1,572 measurements with a range of 0 to 1,140,367 pCi/L, a mean of 11,420 pCi/L, a standard deviation of 56,589 pCi/L, and a geometric mean of 2,082 pCi/L.

Soil and water radon concentrations are not strongly related to each other, but it is possible to estimate the contribution of waterborne radon to air in a house. The NAS considered estimates of the water-to-air transfer coefficient from several sources, including measurements in Maine homes (NAS 1999b). These estimates were lognormally distributed with a mean value of 8.7E-5, a geometric mean of 3.8E-5, and a geometric standard deviation (GSD) of 3.3. The transfer coefficient was also modeled as a function of house volume, ventilation rate, water use, and transfer efficiency. The mean transfer coefficient estimated in this way was 1.2E-4; the geometric mean and GSD were 5.5E-3 and 3.5, respectively. The National Academy of Sciences (NAS) recommended using a transfer coefficient of 1E-4, midway between the estimates from measurements and modeling. This is equivalent to a water:air ratio of 10,000:1 (10,000 pCi/l in water will result in 1 pCi/l in air). This ratio is used to estimate the lung cancer risks of radon escaping to air from water (NAS 1999b). According to this ratio, the average Maine well water concentration of 10,000 pCi/l will add 1 pCi to each liter of air in the home. This would be in addition to radon entering the home from the surrounding soil.

3. Evidence for Health Effects from Radon Exposure

Studies of underground miners exposed to radon have consistently demonstrated an exposure-related increase in lung cancer risk; based on this evidence, radon is classified as a known human carcinogen[2]. In recent years, miner-based lung cancer risk estimates have been substantiated by studies of residential radon exposures. Although a cancer risk

[1] This unit measures radioactivity in terms of radioactive decays per second; 1 pCi is equivalent to roughly two radioactive decays per minute.

[2] For example, by the National Toxicology Program (http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s097zird.pdf) and the International Agency for Research on Cancer (http://monographs.iarc.fr/ENG/Monographs/vol78/volume78.pdf).

from ingested radon has not been observed in epidemiological studies, a recent model has incorporated information about the fate of ingested radon in the body to estimate this endpoint. The risks of radon in air and water have been extensively reviewed by the National Academy of Sciences (NAS 1999a, 1999b), as described below.

3.1 Lung cancer risk

The NAS has published a series of reports on radiation under the title Biological Effects of Ionizing Radiation, or BEIR. The sixth report (BEIR VI) was published in 1999 (NAS 1999a) and focused exclusively on the inhalation of radon gas. When BEIR VI was written the evidence of a lung cancer risk from residential exposures was not clear. To account for this lack of data, the BEIR VI authors used estimates of lung cancer risk from studies of occupationally exposed miners and extrapolated risks to the general population. Eleven major studies of miners were evaluated and it was estimated that 10-15% of lung cancer deaths in the US could be attributable to the inhalation of radon.

Since publication of BEIR VI the evidence for a domestic radon risk has become more compelling. These studies have the advantage of not relying on extrapolation from the relatively high occupational exposures of miners, and they are also based on a more appropriate study group (mixed sex, mixed age general public vs. adult male miners). Table 1 lists most of the available case-control studies to date. The primary weakness of the body of evidence available to the BEIR VI committee was that individual case-control studies had insufficient sample sizes to generate statistically precise risk estimates; this is apparent in fact that most of the results in Table 1 are not significantly positive (the 95% confidence intervals include zero). This problem has been addressed in newer analyses that pool the data from individual case-control studies; these analyses have been conducted with North American, European and Chinese data, as shown in Table 1 and described in more detail below.

The combined analysis of North American studies produced a significantly positive risk estimate very close to the projected risk estimate from the miner data (Krewski et al. 2005). Subjects who move less frequently are presumed to have better exposure estimates. When this analysis was restricted to subjects who only lived in one or two homes over the study period the excess odds ratio at 2.7 pCi/l increased from 0.11 to 0.15.

The combined analysis of 13 European studies also generated a risk estimate very close to that based on miner data (Darby et al. 2004). These authors noted a systematic bias in the radon measurement data; when this bias was corrected the excess odds ratio at 2.7 pCi/l increased from 0.08 to 0.16.

Lubin et al. (2004) combined the two Chinese studies and again generated a risk estimate very similar to that based on miner data. This analysis also restricted subjects to those living in only one home and in this case the excess odds ratio increased from 0.13 to 0.32.

In conclusion, there is a strong, well-established epidemiological literature, based on extensive studies of underground miners and multiple case-control studies of residential exposures, that shows increased lung cancer risks from inhalation of radon at levels typically found in homes.

3.2 GI cancer risk

Ingested radon diffuses into the tissues of the stomach and small intestine. From there it enters the bloodstream and is carried throughout the body. The majority of ingested radon is thought to be exhaled when the blood flow carries it to the lungs. The NAS (1999b) modeled the fate of ingested radon and its daughters in the body and estimated the associated cancer risk. It was determined that most of the radiation dose is delivered to the stomach as radon diffuses through the stomach wall. Most of the cancer risk from ingested radon is thus a stomach cancer risk, although there is a small additional risk of cancer in other tissues of the body.

There have been very few epidemiological studies of the cancer risk of ingested radon. Auvinen et al. (2005) conducted a case-cohort study of stomach cancer in Finland. Average radon concentrations in the water of study subjects (8,600 pCi/l) were similar to those found in Maine. Although the results of this study were consistent with risk estimates derived by the NAS (1999b), the study was essentially inconclusive. The hazard ratio at 2,700 pCi/l was calculated to be 0.68 (95% CI 0.29-1.59). This suggests that the stomach cancer risk at this concentration could be increased by up to 60% or decreased by up to 70%. By comparison, the NAS model would predict an increase in stomach cancer risk of around 2% at this concentration.

Kjellberg and Wiseman (1995) conducted an ecological correlation analysis of county-level radon concentrations and cancer rates in Pennsylvania. Radon was significantly correlated with stomach cancer mortality in both men and women, and with stomach cancer incidence in women. This study was not able to quantify a stomach cancer risk estimate, however, due in part to the variability in radon concentrations within each county. Wilkinson (1985) conducted a similar analysis of county-level stomach cancer rates in New Mexico and found that counties with significant uranium deposits had higher stomach cancer mortality rates. Again, although it is plausible that the cancer risk was increased by radon originating in the uranium deposits, a quantitative estimate of risk could not be derived from this study.

In conclusion, it is plausible that ingested radon creates a cancer risk, but it is not possible to quantify this risk based on currently available epidemiological observations. The NAS report, based on state-of-the-art models of physiology and dosimetry, is the best available way to estimate this risk.

3.3 Estimates of total cancer risk

The total cancer risk of radon in water includes the lung cancer risk of inhaled radon and the whole-body cancer risk of ingested radon, and both can be estimated using the National Academy of Sciences models described above. The risk of inhaled radon is complicated by the fact that radon and smoking interact. This results in a higher lung cancer risk estimate for smokers than for nonsmokers. Table 2 shows lung cancer risks derived by the NAS (1999b)[3].

Table 2. Lifetime lung cancer mortality risks from water radon escaping to air (NAS 1999b)

Concentration in water (pCi/l)	Risk to never-smokers	Risk to smokers	Mixed population risk
5,000	9.3E-4	4.8E-3	3.0E-3
10,000	1.9E-3	9.6E-3	5.9E-3
20,000	3.7E-3	1.9E-2	1.2E-2

The cancer risks associated with ingested radon are independent of smoking and can be represented with a single estimate for any given water concentration. The estimates of cancer risk from ingesting water with 5,000, 10,000, and 20,000 pCi/L radon are 4E-4, 7E-4, and 1E-3, respectively. It should be noted that approximately 87% of the ingestion risk is the estimated stomach cancer risk. Table 3 shows the estimated combined risk of inhaled and ingested radon. The estimate of the total lifetime cancer mortality risk for a mixed population, 6.6E-3 at 10,000 pCi/l in water, can be compared to the most recent EPA estimate of 6.3E-3 at the same concentration (US EPA 1999a). The fact that the two estimates are essentially identical reflects the reliance of the EPA on the NAS models- the risk associated with ingestion is the same in the two cases (EPA and NAS), with small differences in the estimated risk from the inhalation of liberated gas.

[3] These estimates are taken from Table ES-1 (NAS 1999b); risks presented at 1 Bq m^{-3} were converted to units of pCi/l (1 Bq m^{-3} = 0.027 pCi/l; NAS 1999b).

Table 3. Lifetime cancer mortality risk of ingested and inhaled radon (NAS 1999b)

Concentration in water (pCi/l)	Total risk: Never-smokers	Smokers	Mixed population
5,000	1.3E-3	5.2E-3	3.3E-3
10,000	2.6E-3	1.0E-2	6.6E-3
20,000	5.1E-3	2.1E-2	1.3E-2

It can be seen in Tables 2 and 3 that most of the total lifetime cancer mortality risk associated with radon in water is due to inhalation. Depending on smoking status, the ingestion of radon contributes between 7% (smokers) and 28% (nonsmokers) of the total risk

4. Other federal and state guidance for radon in water

Under the Safe Drinking Water Act, the US EPA has the responsibility to regulate public water supplies. For this purpose EPA uses standards called Maximum Contaminant Level Goals (MCLGs) and Maximum Contaminant Levels (MCLs). By law, all carcinogens (such as radon) must have MCLGs of zero. This is a non-enforceable goal. Most MCLs are promulgated as national standards and they allow for the consideration of the technical and economic feasibility of attaining the standard in addition to the predicted health risks. The EPA does not regulate private water supplies such as wells, and the technical and economic feasibility of treating public water is different from the feasibility of treating private water.

In 1999 the US EPA published a notice of proposed rulemaking for the National Primary Drinking Water Regulations for radon. The proposed rulemaking identified an MCLG (zero) and an MCL for radon. The EPA typically develops health-based guidelines for carcinogens such that the cancer risk is between 1E-4 and 1E-6. Based on a cancer mortality risk of 2E-5, the EPA proposed an MCL at 300 pCi/l (US EPA 1999b).

A complicating factor in dealing with radon is the fact that radon enters the home in soil gas and from outdoor air in addition to being liberated from water. In order to facilitate a more cost-effective approach to total radon reduction, the 1996 amendments to the Safe Drinking Water Act outlined the derivation of a third guideline known as the Alternative MCL (AMCL). Specifically, if the MCL established by EPA for radon in water is more stringent than necessary to reduce the contribution to radon in indoor air from drinking water to a concentration that is equivalent to the national average concentration of radon in ambient (outdoor) air, EPA is required to establish an AMCL. The AMCL is set at a level that would result in a contribution of radon from drinking water to radon levels in indoor air equivalent to the national average concentration of radon in outdoor air.

Based on 50 measurements of radon in outdoor air (one in each state, generally around the state capital), the average ambient air concentration was estimated to be 0.4 pCi/l (NAS 1999b). Given the water-to-air transfer factor described above, a water concentration of 4,000 pCi/l would produce the same concentration (0.4 pCi/L) in indoor air. The AMCL for radon was accordingly set at 4,000 pCi/l. These proposed standards have not been finalized by the EPA. It is important to point out that the AMCL is based on a mandate from Congress and is driven more by risk management considerations than strict risk assessment/ risk acceptability considerations.

There are two reasons why the EPA AMCL may not be a suitable conceptual basis for Maine well water guidelines. First, while the estimate of 0.4 pCi/l is considered the best national estimate of outdoor air radon by the NAS (1999b), it is a very uncertain estimate and it may not be appropriate for individual states. For example, Steck et al. (1999) demonstrated that the average outdoor concentration of radon in Iowa was significantly higher than the national average (0.8 pCi/l based on 111 readings). Maine is one state with relatively extensive outdoor radon monitoring. Hess (1982 aci NAS 1999b) measured radon using etched track monitors at 51 sites in Maine during 1980-1981. Measurements were taken in sheltered outdoor locations (garages and porches) at a height of 1 meter from the ground. Concentrations ranged from 0.08 to 4.3 pCi/l with a mean of 0.7 pCi/l and a geometric mean of 0.48 pCi/l. This data set was not random and may be biased toward higher values. The second problem with the derivation of the EPA AMCL is in the way multiple sources of radon are implicitly treated. The total indoor air concentration of radon will be roughly equal to the sum of three sources- soil gas, water, and outdoor air (Hess 2006, pers. com.). Allowing the water to contribute an amount equal to background concentrations effectively allows for a doubling of background concentrations inside the home, even before considering exposure to radon entering the home in soil gas. The implication that radon from water is acceptable because the same amount of radon at background levels is acceptable is problematic in light of the fact that the two sources will coincide and combine.

Since the EPA does not regulate private water supplies, states have developed their own radon guidelines. Like the federal guidance, all state guidelines were derived with the awareness that reducing the cancer risk associated with radon to traditionally acceptable levels (e.g., less than 1 in 100,000) would not be feasible. Table 4 lists guidelines from New England states, including the previous Maine MEG. Note that the Rhode Island guideline is the same as the proposed EPA AMCL. Table 4 also shows the cancer risks associated with each guideline according to the estimates outlined in section 3 above. These numbers can be compared to the traditional ‘acceptable risk’ range of 1E-6 to 1E-4.

Table 4. State water guidelines for radon and associated cancer risks

Water guideline (pCi/l)		Lifetime cancer mortality risk		
		Never-smokers	Ever-smokers	Mixed Population
Maine	20,000	5E-3	2E-2	1E-2
Massachusetts	10,000	3E-3	1E-2	7E-3
Connecticut	5,000	1E-3	5E-3	3E-3
Rhode Island	4,000	1E-3	4E-3	3E-3
New Hampshire	2,000	5E-4	2E-3	1E-3

5. Proposed MEG for Maine

Maximum Exposure Guidelines developed by ME-CDC for carcinogens are typically set at an incremental lifetime cancer risk of 1E-5. In the case of radon, however, we are faced with a risk management problem in which this degree of risk reduction may not be practically attainable. Specifically, the overwhelming majority of drinking water wells in Maine have radon concentrations above 300 pCi/L (the MCL established by the EPA based on a lifetime cancer risk of 2E-5); an MEG of this magnitude would require ME-CDC to recommend water treatment for virtually every home with a private well. An alternative benchmark for setting the MEG can be found in guidance for air radon since the majority of the radon dose from drinking water is through inhalation. The EPA has an action level for radon in air of 4 pCi/L but also states that homes with levels greater than 2 pCi/L should consider remediation[4]. At 2 pCi/L the incremental lifetime cancer mortality risk for a mixed population of smokers and nonsmokers will be roughly 1E-2. As treatment companies can now effectively treat air down to 2 pCi/l, ME-CDC will be giving increased emphasis to remediating homes that exceed this level. The water concentration corresponding to this air concentration is 20,000 pCi/L according to the transfer factor described above. MEGs for non-carcinogens typically adjust such a benchmark with a Relative Source Contribution, representing the fraction of the total chemical intake that is allowed to come from a drinking water source. This methodology is used to ensure that one contaminant source does not dominate or drive exposure above a threshold. The default fraction, following EPA guidance, is 20% (ME CDC 2000, EPA 1990). In this case, with a carcinogen, the assumption is that 80% of ones exposure to radon will be coming from soil gas, the remaining 20% comes from a water source. Adjusting 20,000 pCi/L with the default RSC gives a candidate MEG of 4,000 pCi/L.

[4] http://www.epa.gov/radon/healthrisks.html

An MEG of 4,000 pCi/l would coincidentally be consistent with the EPA AMCL for radon in public water if the proposed AMCL were finalized without revision. An MEG of 4,000 pCi/l is also more consistent with guidance in other New England states (Table 4). Consistency between public water and private water guidelines and among states is recommended to reduce confusion in the general public and among testing laboratories.

In conclusion, ME-CDC is establishing a new interim MEG for radon in water of 4,000 pCi/l. This concentration is associated with a total incremental lifetime cancer risk of 3E-3 (3 in 1,000) as shown in Table 4.

6. Discussion: Radon in water and air

This document describes the basis for an MEG for radon in drinking water. As discussed above, however, radon also enters the home as soil gas and from outdoor air, and the soil gas radon is the dominant source of exposure in most homes. This being the case, ME-CDC does not recommend remediating water without first testing indoor air radon levels. The new interim MEG of 4,000 pCi/l is therefore not intended to trigger immediate action, but rather to serve as a flag for a potential problem. Cost-effective mitigation for a home will potentially include air and/or water treatment, and we anticipate that many homeowners will be confused about what mitigation strategy to pursue. Some cases are relatively straightforward- a home with water and air radon concentrations below the respective guidelines would not require remediation, for example, and a home with water radon concentrations above 20,000 pCi/l and air concentrations above 4 pCi/l would be a good candidate for both air and water treatment. Many homes, however, are in a gray area where one treatment system may be sufficient to address total radon exposure. In these situations the best course of action should be determined by the homeowner and ME-CDC. Hence the recommendation would be for calling the state radon program to discuss the need for further testing (e.g., a long term test) and cost effective modes of risk reduction.

Table 1. Case-control studies and combined analyses of lung cancer and radon in air.

Region	Cases (N)	Controls (N)	Average radon concentration (pCi/l)	Excess OR at 2.7 pCi/l	95% CI
Individual studies[5]					
NJ	480	442	0.70	0.56	-0.22-2.97
Winnipeg	738	738	3.20	0.02	-0.05-0.25
MO-1	538	1183	1.70	0.01	<0-0.42
MO-2	512	553	1.51	0.27	-0.12-1.53
IA	413	614	3.43	0.44	0.05-1.59
CT	963	949	0.89	0.02	-0.21-0.51
UT-ID	511	862	1.54	0.03	-0.2-0.55
Stockholm	201	378	3.45	0.16	-0.14-0.92
Sweden	1281	2576	2.89	0.10	0.01-0.22
S. Finland	291	495	5.75	0.28	-0.21-0.78
Finland	517	517	2.59	0.11	-0.06-0.31
SW England	982	3185	1.51	0.08	-0.03-0.20
Italy	384	404	2.59	0.14	-0.11-0.46
E Germany	1192	1640	2.00	0.08	-0.03-0.20
W Germany	1449	2297	1.35	-0.02	-0.18-0.17
Sweden (nonsmokers)	258	487	2.13	0.28	-0.05-1.05
France	688	1428	3.46	0.05	-0.01-0.12
Czech Rep.	210	12004	13.74	0.09	0.02-0.21
Shenyang China	308	356	2.30	-0.05	<0-0.08
Gansu China	768	1659	6.02	0.19	0.05-0.47
Combined analyses (alternative risk estimates in italics)					
7 North American studies	3662	4966	NA	0.11 *0.15*[6]	0.00-0.28 *0.01-0.37*
13 European studies	7148	14208	2.81	0.08 *0.16*[7]	0.03-0.16 *0.05-0.31*
2 Chinese studies	1050	1996	NA	0.13 *0.32*[8]	0.01-0.36 *0.07-0.91*
Miner data[9]				0.12	0.02-0.25

[5] Information on individual studies drawn from a similar table presented by Krewski et al. (2005).
[6] Restricted to subjects living in no more than two homes during the study period.
[7] Corrected for measurement error.
[8] Restricted to subjects living in one home for 30 years or more.
[9] This summary of the miner data is from BEIR VI and makes use of the low-exposed miners (<50 Working Level Months); similar risk projections were made from risk models using the full range of miner data (NAS 1999a, Lubin et al. 1997).

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