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Review of Technical Justification of Assumptions and Methods Used by the Environmental Protection Agency for Estimating Risks Avoided by Implementing MCLs for Radionuclides

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DISCLAIMER

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EXECUTIVE SUMMARY

The Environmental Protection Agency (EPA) has proposed regulations for allowable levels of radioactive material in drinking water (40 CFR Part 141, 56 FR 33050, July 18, 1991). This review examined the assumptions and methods used by EPA in calculating risks that would be avoided by implementing the proposed Maximum Contaminant Levels for uranium, radium, and radon. Proposed limits on gross alpha and beta-gamma emitters were not included in this review.

Table ES-1. Factors of overestimation of uranium

Source	Overestimation Factor
Absorption from gut	2-10 times
Carcinogenicity	unquantified
Chemical Toxicity	10-20 times
Population exposed	< 1.1 times
Water intake	1.8 times
Relative source contribution	1.4 times
Exposure duration (cancer only)	0-9 times
Overall uranium risk estimate from toxicity	2,800 times
Overall uranium risk estimate from radiocarcinogenicity	100-900 times

The approach taken and methods used by EPA were reviewed and evaluated. In some cases, the overall approach used to develop avoided risk-estimates was found to be flawed. The parameters needed to estimate risks were independently estimated with attention to uncertainty. For most parameters, this was done by determining a range or distribution of possible values. EPA's estimates invariably fell in the upper part of that range. The

following tables summarize the degree of overestimation found in EPA's estimates compared to the mean values of the ranges developed in this study.

Table ES-2. Overestimation of radium risk	
Parameter	Overestimation Factor
Dose response function	2.6 times
Water intake	1.7 times
Exposure duration	15 times
Overall radium risk estimate	13-17 times

Table ES-3. Overestimation of radon risk.	
Parameter	Overestimation Factor
Ingestion risk	3.8 times
Inhalation risk	2.5 times
Overall radon risk estimate	2.7 times

TABLE OF CONTENTS

1. INTRODUCTION

The Environmental Protection Agency (EPA) has proposed regulations for allowable levels of radioactive material in drinking water (40 CFR Part 141, 56 FR 33050, July 18, 1991). This review examined the assumptions and methods used by EPA in calculating risks that would be avoided by implementing the proposed Maximum Contaminant Levels for uranium, radium, and radon. Proposed limits on gross alpha and beta-gamma emitters were not included in this review.

The approach taken and methods used by EPA were reviewed and evaluated. The parameters needed to estimate risks were independently estimated and the results compared to the values used by EPA. A key factor in developing MCLs is the treatment of uncertainty in the data. Uncertainties range from extrapolating toxicity and pharmacokinetic results from animal studies to estimating the range of variability in the amount of water that people drink daily in different parts of the country. Over time, research can help to reduce these uncertainties, but can never eliminate them. To assure protection of public health, EPA must include a "factor of safety," thus requiring a higher degree of treatment than would be necessary if the risks were better understood. How big a factor of safety or how much the risk should be overestimated is a matter of judgment. The more the overestimation, it would seem, the more the public is protected. Costs of treatment, however, often increase more than linearly with the degree of treatment, so it is not in the public interest to exaggerate the safety factor unnecessarily. Given a range of possible values for a parameter, EPA has tended to select a value at or near the extreme. The overestimation of risk is often further exaggerated because many overestimated parameters are usually multiplied together in the calculation of risk. More recently, EPA has attempted to select more central estimates of risk, relying on an explicit factor-of-safety multiplier. This is a much improved approach, but, as will be seen, has not been completely applied in this case. Much of the difference between our estimates and EPA's lies in their repeated selection of parameter values from the upper part of a range of values.

2. URANIUM

EPA systematically chose high values from the range of reasonable values for each parameter considered in its analysis. Each parameter is discussed in detail below. Table 2-1 summarizes the general order of overestimates compared to a realistic mean value.

Table 2-1. Sources of overestimation of uranium risk.

Source	Overestimation Factor
Absorption from gut	2-10 times
Linear carcinogenic model	unquantified
Chemical toxicity	10-20 times
Population exposed	< 1.1 times
L/d water intake	1.8 times
Relative source contribution	1.4 times
Exposure duration (cancer only)	0-9 times
Overall (based on toxicity)	2,800 times
Overall (based on carcinogenicity)	100-900

Absorption through gut.

A key assumption was the percentage of ingested uranium absorbed through the gut into the bloodstream (pharmacokinetic transfer factor f_1). EPA reported "Absorption has ranged from 0.7 to 31 percent" and proposed a "reasonable mid-range estimate of ... about 5 percent of the intake" (Uranium, 1991, p. IV-4). In its toxicokinetic model, EPA used a range of 5-20% (Uranium, 1991, p. IV-23). EPA's basis for its absorption assumption was summarized in their Tables IV-1 (human studies) and IV-2 (animal studies). The actual ranges from the EPA tables are 0.3-31% for human studies and 0.01-4.5% for animal studies. Although it is possible that human absorption of uranium is remarkably different from other species, this goes against a wide body of scientific knowledge, especially given evidence of considerable similarity among species (LaTouche et al., 1987). Probably a more important difference is that the animal studies were generally more controlled than human studies and thus had smaller error. Of a total of 33 estimates of absorption reported in the two tables, only 5 exceed 5.2% (all 5 are among the 15 estimates from human studies). These 5 high estimates are highly questionable compared to the other estimates; they are discussed

individually below. Because of the questionable nature of these high estimates compared to the more "solid" lower estimates, 5% appears to be an upper end of the range, not a "reasonable mid-range estimate."

This view was supported by a committee of internationally known experts in this field, commissioned by EPA to review the metabolism of ingested uranium. They analyzed the literature available in the mid-1980s and recommended a value of 1.4% and noted, "None of the available experimental or environmental data support a fractional U absorption greater than about 5%, even at intakes of the order of 1 to 2 μg/day for Reference Man. A higher value for U absorption (about 20%) based on dietary U data from the British (Hamilton, 1972) and unpublished analyses of U in urine (M.H. Dean, quoted by Hursh and Spoor, 1973) seems unlikely on physiological grounds..." (Wrenn et al., 1985, pp. 626-627).

The 5% absorption rate value originally came from Report 30 of the International Commission on Radiological Protection (ICRP, 1979), but in a more detailed report, ICRP (1975) stated that "the oral absorption of soluble uranium by man lies between 0.5 and 5% of the ingested dose" (p. 415).

Of the high estimates reported by EPA (Uranium, 1991), two are based on fecal excretion (assume [intake-fecal excretion] / intake = absorption). Spencer et al. studied 4 subjects over 24-54 days. In contrast, the Study by Somayajulo et al. (1980) studied only 1 subject for 2 separate 24-hour periods, assuring even greater error. Theoretically, this is a correct approach, but in practice has been shown to involve a very high error rate. Spencer et al. (1990), for example, in reporting their results, state that, "Although the net absorption of the two uranium isotopes determined from the intake and fecal excretions ... averaging 26 and 23% respectively, the large error for the balance shown in their Table IV includes zero net absorption. *It is well known that it is not possible to determine uptake of trace elements quantitatively in this manner*, and uptake is estimated by other means later" [emphasis added]. Although Spencer et al. made the calculation for ^{234}U and ^{238}U separately, EPA reported only the higher number, even though it applied to ^{234}U, which constituted only about 1/10,000th of the total mass concentration of the intake.

The other three high estimates were based on calculations that combine "market basket" estimates of uranium content in foods for various cities or countries with measurements of uranium in urine in small samples. Not only do these estimates include errors associated with unknown differences between market basket surveys and actual intake by the subjects whose urine was analyzed, but they ignore the contribution to intake by drinking water, biasing absorption estimates upward. Spencer et al. (1990), showed that water may be a more important contribution than food. A review by Wrenn et al. (1985) addressed the calculations of Hursh and Spoor (1973), but did not include them in their analysis (see quote above from Wrenn et al.). Instead, they made their own calculation (the 7.7% value included in the EPA Table IV-1) based on the earlier data, but then excluded this data point from their final analysis, citing unresolved uncertainties.

There is some question about conditions that might cause increased uptake. Animal experiments indicate that overnight fasting increases uptake, raising a question about people who do not eat breakfast. The Wrenn committee (Wrenn et al., 1985) found a correspondence between the fasted animal data and the human data. More recent human findings at environmental exposure levels are consistent with this (Spencer et al., 1990; Singh et al., 1990). It is not clear why average human values match fasted animal values, but there is no evidence to support using the low absorption rates from animals fed *ad lib* (often below 0.1% of intake) as the basis for estimates of increased absorption among people who do not eat breakfast. Uptake for fasted animals is in the same range or lower than EPA's estimate for people.

Another condition leading to increased uptake that EPA (Uranium, 1991) raised is anemia due to iron deficient diet. In the study that supports this notion (Sullivan and Ruemmler, 1988), the researchers could not produce anemia in the animals despite an iron-deficient diet. To produce the anemic condition for the experiment, one-third of their blood volume was removed from the animals. Even this severe effect produced less effect on adsorption than fasting overnight. The principal usefulness of this line of investigation (including reports such as Sullivan et al., 1986), is to increase the understanding of factors affecting absorption, providing a better basis for interpreting the animal data.

On the basis of the information described above, we believe the best estimate is in the range 0.5-2%, with an overall range of 0.1-5%.

Chemical Toxicity of Uranium.

For chemical toxicity risk, EPA chose to base its estimates on a 30-day study of rabbits from 40 years ago (Maynard and Hodge, 1949), from which EPA selected 0.01 g/kg/day as a LOAEL and then applied a safety factor of 1000. This study included a 30-day, 1-year, and 2-year study of rats, a 30-day and 1-year study of dogs, and a 30-day study of rabbits. Sixty-three pages were devoted to the rat and dog studies, providing great detail on the methods and the results. Slightly over 3 pages were devoted to the rabbit studies, of which nearly half was devoted to comparisons among rats, dogs and rabbits. The lowest exposure level in rabbits was 0.01 g/kg/day; this caused detectable weight depression, although weight depression was considered "minimal." Maynard and Hodge (1949, p. 274) reported that renal damage was "moderate" at this dose level and EPA selected this as a LOAEL. Although EPA restates kidney damage as "moderate" on p. VIII-4 (Uranium, 1991), it thereafter refers to the LOAEL as being based on "moderately severe renal damage" (pp. VIII-5, 6, and 7 of Uranium, 1991).

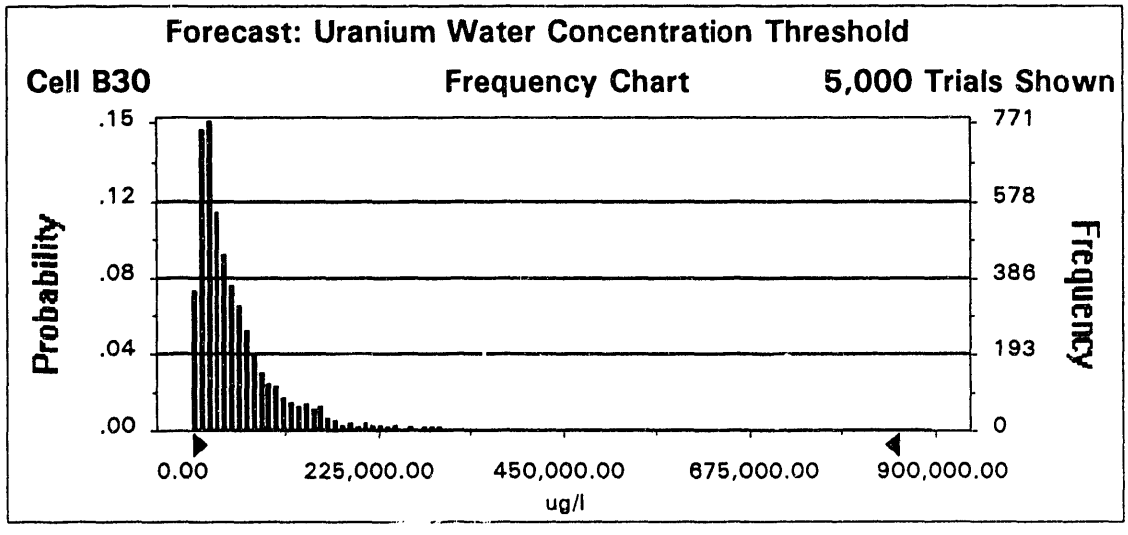
EPA justified its selection of a safety factor of 1000 on the basis that the study chosen did not determine a NOAEL (Uranium, p. VIII-4). This is a narrow approach that ignores the bulk of available literature. The decision would make sense if this single, highly limited, rabbit study was the only health effects data on uranium available. But this is not the case. There are a multitude of animal

studies that *do* determine a NOAEL. Moreover, there are massive amounts of human data available.The Committee on Metabolism and Dosimetry of High LET Radionuclides for the National Workshop on Radioactivity in Drinking Water (Wrenn et al., 1985) recommended "that based on the NAS [1877] definition, U should be assigned an uncertainty factor of 10-100" (pp. 612-632). It then selected 50 as a safety factor "that should provide a high margin of safety" (pp. 632-633). Using the EPA guidelines for selection of a safety factor, the National Research Council's Safe Drinking Water Committee selected an uncertainty factor of 100 (NAS, 1983, p. 96). Both these expert committees were established by EPA; their members were leaders in the field, and did an extensive review of the published material. EPA cavalierly chose 1000 with no discussion of why it did not follow the recommendations of these earlier groups. This automatically makes the EPA limit in this area more stringent, by a factor of 10 to 20, than these expert groups thought necessary. This is probably the biggest source of unnecessary conservatism.

Although EPA went to great effort to model the pharmacokinetics of ingested uranium to obtain organ-specific doses for its estimates of radiologically induced cancer, much against the advice of its Science Advisory Board (SAB), it did not use this information to estimate the kidney concentration of uranium for estimating chemical toxicity. It simply took the relationship between ingested dose and kidney damage in rabbits and applied it to man (with a safety factor of 1000). By substituting available information for arbitrary safety factors, a more rational and realistic value for the allowable drinking water level might have been obtained. This could be important since a rabbit's diet is much different from man's and the rate of absorption through the gut might be expected to be different (although Tracy et al., 1992, found that gut absorption for rabbits did not differ from that for rats).

We developed probability distributions explicitly characterizing the uncertainty associated with the parameters necessary to calculate uranium toxicity (absorption through gut, uptake in kidney, biological half-time in kidney), toxicity threshold in terms of kidney concentration of uranium, and drinking water intake rates). The quantitative characterization of these distributions is detailed in Appendix 2-1. Calculating backwards through these distributions in a Monte Carlo analysis from the distribution of uranium toxic threshold level in the kidney resulted in a distribution of toxic water concentration level (Figure 1) with a mean of 56.7 mg/l (5 percentile level of 7.7 mg/l and 95 percentile level of 165 mg/l). EPA's proposed maximum concentration level of 20 µg/l is 2800 times more stringent than the mean and 385 times more stringent than the 5 percentile level of this result. We take 2800 as the mean level of overestimation of risk in the proposed MCL for uranium.

Figure 1. Distribution of drinking water concentration of uranium associated with chemical toxicity on a chronic exposure basis.



Size of Population exposed.

EPA estimated the concentration of uranium in ground water by the size of water distributions based on measurements from a 2% sample of all ground water systems. EPA then extrapolated from this sample to all water systems, whether from ground-water or surface-water sources, even though no direct data on uranium in drinking water from surface-water supplies was available. Available data on surface waters cited by EPA suggest uranium content is somewhat lower in surface waters than in ground water. Because high uranium concentrations are primarily in small water systems, however, the extrapolation only increases the total estimated population exposure by about 30%, so any overestimate of effect from this source is probably small compared to other potential sources of error.

Amount of Water Drunk.

EPA assumed drinking water intake of 2 L/day (Uranium, p. III-16). This was in accordance with an SAB recommendation to maintain consistency with previous EPA assessments (apparently, EPA used 0.66 L/day in an earlier draft reviewed by SAB). This was a mistake. Two liters/day has always been an overestimate, especially for estimating risk from tap water. The 2 L/day assumption, if it had any reality, included water in bottled drinks, etc., that do not come from the drinking water source being investigated. A nation-wide survey showed the 50 percentile mid-range consumption of tap water to be 0.6 L/day with a mean of 1.2 L/day (Ershow and Cantor, 1989; Roseberry and Burmaster, 1992).

Relative Source Contribution (RSC).

In addition to drinking water, people are also exposed to uranium through food (including milk) and inhalation (although the latter is negligible). EPA defines the RSC as the "fraction of total intake accounted for by drinking water as a source" (FR, 1991, p. 33068). Once the Reference Dose (RfD) is converted to the Drinking Water Equivalent Level (DWEL), i.e., the concentration in drinking water that would, by itself, produce the "safe" RfD, it is multiplied by RSC to account for water's proportionate share (otherwise, in order for the total exposure to meet RfD, one assumes food would have to be reduced to zero). There are problems with this approach. Ideally, perhaps, EPA should look at all sources of uranium exposure and design its regulations to reduce each to the level that would achieve the most cost-effective way to meet the desired total exposure limits. EPA, however, regulates only one medium at a time. Moreover, the percentage of exposure from different sources varies sufficiently from place to place that a comprehensive approach might be difficult anyway. The RSC approach is designed to reduce the medium being addressed (here drinking water) according to its proportionate share of the total reduction required to achieve the total exposure limit. The difficulty arises when, for the sake of conservatism, water is assumed to have a smaller RSC than it actually has. This leads to forcing a greater than proportional share reduction in the water contribution. If, for example, the actual contribution of drinking water to total dose is 50% and EPA assumes 20%, the MCL will be 40% overly restrictive (0.2/0.5). EPA recognizes that median dietary uranium intake from food is generally low (FR, 1991, p. 33068). The mean value of RSC was estimated at 36.6% (Uranium, p. III-9). Canadian drinking water standards are based on an RSC of 90% (Health & Welfare Canada, 1989, p.3). That may be too high, although Spencer et al. (1990) conclude that, despite almost equal intake from food and water, that gastrointestinal uptake "...from diet other than water appears small" (p. 94).

EPA's selection of 20% as the RSC is based on the belief (without evidence) that some areas of the country may have undocumented higher soil and water uranium levels and that "These areas may need lower water contributions to maintain total uranium intakes low enough to ensure safety from kidney toxicity" (FR, 1991, p. 33068). Indeed, a recent study in Utah found higher uranium intake in both water and food (Singh et al., 1990). RSC value for drinking water based on this study would be 33%. Since food anywhere in the United States is likely to come largely from national markets, one would expect that the fractional contribution to total intake from water would be higher in areas with higher natural uranium levels.

Radio-carcinogenicity of Uranium.

Authoritative bodies, including EPA's Science Advisory Board, have recommended that regulation be based on chemical toxicity and not carcinogenicity (SAB, 1991; NAS, 1980, p. 177; Wrenn et al., 1985, p. 610). Since

our findings show that the risk of chemical toxicity from uranium in drinking water is much lower than claimed by EPA, it is necessary to re-examine the risk from radiological effects. EPA's analysis of carcinogenic effects indicated that a drinking water concentration of 170 pCi/L would be within the 10^{-4} lifetime mortality risk criteria used for the MCL (FR, 1991, p33076). This suggests that, were the MCL to be based on EPA's estimate of radio-carcinogenic effects, it is 170/20 = 8.5 times too low.

Despite almost universal exposure to natural uranium, including large numbers of people occupationally exposed over 50 years and, according to EPA's survey described in (Uranium, 1991), large numbers of people exposed to relatively high levels in drinking water, there is no human evidence of carcinogenicity associated with uranium. EPA goes into great detail to estimate increased cancer by organ associated with uranium intake, based on linear extrapolation from high doses from effects of radionuclides other than uranium. EPA then made downward adjustments on leukemia risk to accommodate comments from the SAB (Uranium, 1991). Comparison of the EPA risk factor with a simpler approach using whole-body lifetime effective committed dose based on the approach of ICRP reports 30, 60 and 61 (ICRP, 1979, 1991a, 1991b) found little difference.

The lack of data suggests that these calculations may overestimate the risk of low doses of radiation from uranium in drinking water but at the same time, precludes any means of credibly calculating a lower value. We can only assert a commonly held belief that use of the linear model to extrapolate to low doses is frequently considered a "conservative" approach and in this case may be particularly so. The possibility that the risk of drinking water with uranium concentrations as found in the survey may be zero cannot be excluded.

The dose-response function and the organ distribution factors are not the only contributors to the overall estimate of carcinogenic risk. The estimates of L/day of water intake, absorption from the gut, and relative source contribution described in the toxicity analyses above, apply to the radiological effects in the same way as they do to toxic effects. Our estimate of the mean value of water consumed is 1.2 L/day compared to EPA's estimate of 2 and our estimate of absorption through the gut is 1% compared to EPA's estimate of 5%. We also estimate that the EPA overestimated the RSC by a factor of 1.4. These factors combined would increase the EPA estimate of the drinking water concentration associated with a 10^{-4} lifetime risk from 170 to 2000 pCi/L, 100 times higher than the proposed MCL.

In the case of toxic effects, a toxic concentration could accumulate in the kidney over a relatively short time, given a sufficiently high dose. The effective dose-commitment and the lifetime risk of radiogenic cancer, however, are dependent on the duration of exposure. The previous analysis assumed a lifetime exposure. Average residence time for rural areas in the U.S. is 7.8 (± 1.17) years (Israeli and Nelson, 1992). Since committed effective dose varies with age at exposure, the point in one's lifetime when the exposure occurs makes a difference.

The average effect of an exposure duration of 7.8 years, however, would overestimate the water concentration associated with a 10^{-4} lifetime risk by (70/7.8) or 9 times, making the overall overestimation of the MCL 900 times, were the risk calculated on the radiological effect.

Results and Conclusions

Our analysis shows that EPA systematically chose values in its analysis that tended to overestimate the risk of uranium in drinking water. The extent of these overestimates was summarized in Table 2-1. Given uncertainties that invariably exist, it is certainly prudent for a regulatory agency to build into its standards a factor of safety to protect public health. In this instance, however, the overall degree of safety would appear to be high given the potential risk.

It would also appear that EPA's thinking is not simply to provide ample protection for the people in these high-uranium areas, but to provide it primarily through regulations on drinking water. This appears to reflect limitations and contradictions of EPA's regulatory process that focuses on one medium at a time. Further, it does not seem reasonable to impose additional, unnecessary control costs on the entire country to "protect" these potentially high-exposure areas if these few people are indeed at high risk of kidney toxicity from uranium in the water and food, a national drinking water standard seems an expensive and ineffective approach to deal with the problem. The whole need for such protection is, of course, hypothetical. There is no evidence of any kidney damage at any of the measured levels of uranium in drinking water.

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Appendix 2A. Quantitative Characterization of Uranium Risk Parameters

Metabolic/Pharmacokinetic Parameters

EPA models organ doses for carcinogenic effects of uranium, but not for toxic effects. For the latter, EPA compares administered dose to rabbits directly with toxic effect, extrapolating to humans on a mg U/kg body weight basis. We use a pharmacokinetic model for both radiocarcinogenic and toxic effects. In the latter, we compare kidney concentration of uranium with a threshold level. We calculate backwards, from the toxic threshold concentration in the kidney, through the pharmacokinetic model and the amount of water drunk per day, to obtain the equivalent threshold level in drinking water that can be compared with the EPA value.

Uptake (gut to blood)

We followed the approach of Wrenn et al. (1985) and LaTouche et al. (1987). We included more recent reports and analyzed them in three groups: overnight fasting (animals and humans), ad lib feeding, and human studies with environmental doses (Table 2A-1). The ad lib feeding group had considerably lower uptake, but the two other groups were similar. We based our estimate on the environmental group and characterized the results as lognormal, with geometric mean = 1.03%, standard deviation = 1.78, 95% bounds from 0.0033 to 0.0317, and total range of 0-0.05. The upper bound on the range was based on the upper estimate of ICRP 30.

Blood to Kidney

We take the general approach of Wrenn et al. (1985), drawing on their results, supplemented with new results from rats and rabbits (Tracey et al., 1992). The latter authors report that, although there are differences in uptake from the gut, once in the blood uranium uptake and retention by the kidney are similar among rats, rabbits, and humans. We model the two distributions as lognormal distributions with mean and standard deviations taken directly from the data of Wrenn et al. and Tracy et al. The 95% confidence intervals of our distributions for kidney uptake and biological half-life are 1.84-21.4% and 4.70-26.2 days, respectively. We assume the two distributions are inversely correlated with a correlation coefficient of 0.7.

EPA uses the ICRP 30 model with two elimination rates. Fractional uptake from blood to kidney is assumed to be 12%, of which 99.6% is retained with a 6-day half-time and 0.4% with a 1500 day half-time (Uranium, p. IV-23). The 12% uptake is at the 85th percentile level of our distribution.

Table 2A-1. Data used for estimation absorption through gut.

Data used to estimate uranium uptake through gut (file 56-10T).				
Full citations not included here are in Wrenn et al., 1985				
Fasted Group from Wrenn et al., 1985, with LaTouche and Wrenn 89 added				
Data Set		Species	ug/kg/d	%Absorb
A2-1	Harrison and Stather, 81	hamster	630	0.77
A2-3	Fish et al., 60	dog	700	1.55
A2-4	Larson et al., 84	baboon	0.5	1.2
A2-5a	Butterworth, 58	man	6700	0.73
A2-5b	Hursh et al, 69	man	132	1.4
A5-2	Yamamoto et al., 68; Masuda, 1971 a-d	man	0.15	1.6
A5-3	Fisher et al., 83	man	0.34	0.76
A5-4	Somayajula et al, 1980	man	0.76	2.2
LaT-1	Latouche et al., 87	rat	30	0.78
LaT-2	Latouche et al., 87	rat	300	1.08
LaT-3	Latouche et al., 87	rat	3000	1.78
LaT-4	Latouche et al., 87	rat	3.00E+04	0.64
LaT-5	Latouche et al., 87	rat	4.50E+04	2.82
Wr89	Wrenn et al., 89	man	3.57E+00	0.6
Environmental Exposure Studies of Humans				
Data Set			ug/kg/d	%Absorb
Dang	Dang et al., 92	man	0.011	1.6
Masuda-T	Masuda, 71	man	0.131	1.61
Masuda-N	Masuda, 71	man	0.080	1.32
Masuda-A	Masuda, 71	man	0.056	0.69
Masuda-U	Masuda, 71	man	0.025	0.34
Spencer	Spencer et al, 90	man	0.063	1.5
Singh	Singh et al, 90	man	0.067	1
"Fed" Group from Wrenn Table A-2, with Tracy rabbits added				
Data Set			ug/kg/d	%Absorb
Tracy	Tracy et al., 92	rabbit	3.90E+04	0.06
6a	Hamilton, 48	rat	3.00E+02	0.35
6b1	Sullivan, 80	rat	2.3	0.06
6b2	Sullivan, 80	rat	4.00E+03	0.06
6c1	Sullivan, 83	rat	5.10E+03	0.044
6c2	Sullivan, 83	rat	1.30E+04	0.044
6c3	Sullivan, 83	rat	2.50E+04	0.088
6d	Tracy et al., 1983	rat	3.30E+04	0.035
'6e1	Maynard et al, 53	rat	2.00E+04	0.052
'6e2	Maynard et al, 53	rat	9.60E+04	0.059
'6e3	Maynard et al, 53	rat	2.00E+05	0.06
'6e4	Maynard et al, 53	rat	1.20E+05	0.038
'6e5	Maynard et al, 53	rat	4.70E+05	0.078
'6e6	Maynard et al, 53	rat	9.70E+05	0.04

Model Formulation Uncertainties

Although uncertainty in individual parameters of the intake-to-kidney concentration model has been characterized, there is no direct method to estimate uncertainties associated with the specific formulation of the model used. As a practical approach to account for this uncertainty, we assume that the range of uncertainty from this source is about a factor of two. We achieve this by specifying a lognormal distribution with geometric mean 1 and geometric standard deviation 1.4.

Water Intake

We take the distribution of water intake from Ershow and Cantor (1989), based on the 1977-1978 Nationwide Food Consumption Survey of the U.S. Department of Agriculture (USDA, 1984). We use a lognormal distribution with arithmetic mean and standard deviation of 1.203 and 0.689 L/d representing intake of tap water in the mid-west.

EPA uses a value of 2 L/d. This is at the 89th percentile of our distribution and introduces a mean overestimate of 1.7.

Kidney Concentration Threshold

Wrenn et al. (1985) suggest a threshold of 1 μg U/g. Kocher (1989) applied a safety factor of 10 to this to protect maximally exposed individuals in the public, using a threshold of 0.1 μg U/g. Several reports of animal experiments demonstrate effects in the range of 0.5 to 1 μg U/g. These effects are perhaps not as severe as those on which the original occupational standard of 3 μg U/g was based, but may be more appropriate end-points for chronic exposure to the public, which does not have the additional protection of routine medical surveillance and bioassay. A range of 0.1-1 μg U/g appears appropriate. The threshold value was characterized as a Weibull distribution with location parameter 0.1 to reflect a lower limit of 0.1 μg U/g, a scale parameter of 0.6 and shape parameter of 4. These produce a maximum value of about 1 μg U/g with a mean of 0.6.

Safety Factor

EPA introduces a safety factor of 1000 on its water concentration toxic threshold. This is presumably due to its being a LOAEL rather than a NOAEL. EPA ignored NOAEL effects found in other studies. Using the EPA guidelines for selection of a safety factor, the NAS/NRC Safe Drinking Water Committee selected an uncertainty factor of 100 (NAS, 1983, p. 96). The Committee on Metabolism and Dosimetry of High LET Radionuclides for the National Workshop on Radioactivity in Drinking Water (Wrenn et al., 1985), recommended, "that based on the NAS definition, U should be assigned an uncertainty factor of 10-100" (pp. 612-632). It then selected 50 as a safety factor "that should provide a high margin of safety" (pp. 632-633).

We do not apply an arbitrary safety factor, but express uncertainties explicitly in the input and results, allowing the degree of safety to be chosen as an explicit level of confidence.

3. RADIUM

EPA (1991a) claims that its risk assessments generally use best estimates rather than conservative values. Nevertheless, in its analyses of risks from radium ingestion, the EPA selected high values, as opposed to central tendencies (*i.e.*, averages) or reasonable upper bounds of ranges of values for parameters.

Table 3-1. Comparisons of Parameters Used in Estimating Cancer Fatality Risks from Radium in Drinking Water.

Parameters	EPA Value or Default	Distribution Average	EPA Overestimate
Risk Factors (death/pCi•L^{-1})			
^{226}Ra	2.2×10^{-6}	8.6×10^{-7}	2.6
^{228}Ra	1.9×10^{-6}	7.4×10^{-7}	2.7
Water Ingestion Rate (L/d)	2	1.2	1.7
Exposure Duration (years)	70	4.6	15.2
Total Fatality Risks			
^{226}Ra	4.4×10^{-6}	3.3×10^{-7}	13.3
^{228}Ra	3.8×10^{-6}	2.2×10^{-7}	17.3

Carcinogenicity of Radium.

There is extensive epidemiological evidence of carcinogenic effects of ingested ^{226}Ra and ^{228}Ra. The primary data comes from studies of radium dial painters (Rowland et al., 1978, 1983). Radium body burdens were measured in the dial painters and were used to calculate lifetime radium intake.

Ingestion of ^{226}Ra resulted in bone cancers (osteosarcomas) and cancers of the linings of cranial sinuses (head carcinomas). Ingestion of ^{228}Ra resulted in bone cancers. The dose-response function for bone cancer induced by ingestion of ^{226}Ra or ^{228}Ra is purely quadratic, with no excess cancers at lower doses. From a practical point-of-view the function exhibits a threshold at a dose to the skeleton that is well above the worst environmental exposures.

The data for head carcinomas can fit either a linear or a quadratic function. These carcinomas are attributed to radon-222, a daughter of radium-226. No

excess head carcinomas are associated with ^{228}Ra. The half-life of its daughter product, radon-224, is too short to allow for migration to and accumulation in cranial sinuses.

The SAB (1991) recommended that the EPA use the epidemiological evidence for bone and head cancers in radium dial-painters. The evidence for radium-induction of other soft-tissue cancers is equivocal (Stebbings et al., 1984). The EPA's risk factors are based on the RADRISK model, derived from ICRP effective dose equivalents (ICRP, 1977) that were modified to account for the specific metabolic behaviors of radioactive daughters. RADRISK incorporates a toxicokinetic model based upon alkaline earth intake, retention and excretion. RADRISK is a linear, no-threshold model that uses the sum of weighted organ-doses to arrive at a single dose coefficient for predicting the risk of getting a cancer or the risk of dying from cancer, while using a life-table analysis to adjust for age- and sex-specific mortality from competing risks. The ICRP weighting factors and risk coefficients are predominantly based upon studies of the effects of low LET external irradiation on A-bomb survivors. The majority of the weighting factors are for soft-tissue cancer mortality.

Weighting factors in RADRISK have been modified from those of the ICRP (EPA, 1991b) to calculate the risks for all cancers (fatal and nonfatal). "Ingested radium is estimated to distribute about 85% to bone and 15% to soft tissues (UNSCEAR, 1972)" (EPA, 1991b). The ICRP RBE of 20 for alpha particles was reduced to 8 by the EPA. The EPA has adjusted risk calculations to meet the SAB concerns about overprediction of leukemias (EPA, 1991a), but the RADRISK model still produces a majority (~ two-thirds) of the overall risk estimate for soft tissues, where either no evidence or marginal evidence exists for radium-induced cancers. [For example, increases in breast cancer and multiple myelomas correlate better with duration of employment, a surrogate for external dose of gamma irradiation, than with radium intake (Stebbings et al., 1984)]. The ratio of all cancer risks to the risks for bone and cranial cancers may be overestimated by a factor between two and five according to the EPA (1991a).

The estimates of tissue doses and cancer risks for ingestion of ^{226}Ra and ^{228}Ra are further complicated by the use of a short-lived isotope, radium-224, with a different route of exposure and different pharmacokinetics. EPA's linear model is largely based upon analytical results from patients with ankylosing spondylitis or tuberculosis who were injected with ^{224}Ra (Speiss et al., 1989).

EPA (1991b) states that overall uncertainty of its risk estimates may be an order of magnitude in either direction.

In the following sections, we will demonstrate:

1. The specific differences from EPA's risk estimates achieved by using alternatives to EPA's values for risk factors, tapwater intake, and exposure duration;

2. The overall differences in risk estimates achieved by the combined use of the three alternatives.

Risk Factors for ^{226}Ra and ^{228}Ra in Drinking Water.

The proposed EPA drinking water standard for radionuclides is based on a lifetime mortality risk of 10^{-4}. The EPA's calculated lifetime risks for daily ingestion (over a 70-year lifetime) of 2 L of water, containing 1 pCi/L of either ^{226}Ra or ^{228}Ra, are respectively 4.4×10^{-6} or 3.8×10^{-6}.

Table 3-2. Lifetime Risks for Cancer Fatality from ingesting water containing 1 pCi/L of ^{226}Ra or ^{228}Ra at 1 L/d for 70 years (derived from Table VIII-5, Section 4, in EPA, 1991b).

Type	^{226}Ra	^{228}Ra
Bone Sarcoma	4.7E-7	4.7E-7
Head Carcinoma	4.7E-7	0
Leukemia, high LET	1.1E-7	1.3E-7
Leukemia, low LET	4.8E-8	1.3E-7
All other	1.2E-6	1.2E-6
Total	2.2E-6	1.9E-6

Unit risk factors [cancer fatality/(pCi•L^{-1})] are calculated by dividing the EPA risk factors by 2 (Table 3-2). The unit risk factors for all cancer fatalities from ^{226}Ra or ^{228}Ra are respectively 2.2×10^{-6} or 1.9×10^{-6}. These values are assumed to be the upper limit of the 90% confidence interval.

Table 3-3. Unit Risk Factors [cancer fatality/(pCi•L^{-1})] for 1 pCi/L of ^{226}Ra or ^{228}Ra.

Parameter	^{226}Ra	^{228}Ra
Arithmetic Mean	1.5E-6	1.0E-6
Standard Deviation	9.0E-7	1.4E-6
Lower 90% Confidence Limit	9.4E-7	4.7E-7
Upper 90% Confidence Limit	2.2E-6	1.9E-6

Although we cannot exclude the possibility that ingested radium induces leukemias and soft-tissue cancers, based on the available data, we assume that the probability of these hazards is extremely small. Therefore, we have assumed the EPA's Total Lifetime Risks for Fatal Cancers (Table 3-1) to be a lognormal distribution of lifetime risks per pCi/L of drinking water. The unit risk factors for bone plus head sarcomas from ^{226}Ra, and risks for only bone sarcomas from ^{228}Ra are assumed to be the lower limit of the 90% confidence interval. The arithmetic means and standard deviations (Table 3-3) for the lognormal distributions were calculated by the methods described in Layton et al. (1987).

Individual Lifetime Risks (ILR) for cancer fatality are calculated by the following equation:

$$ILR = RF \times WI \times F \times D \qquad (1)$$

where:

RF = Unit Risk Factor $\left(\frac{fatality}{pCi \bullet L^{-1}}\right)$;

WI = Water Ingestion Rate $\left(\frac{L}{d}\right)$; default = $\frac{2L}{d}$

F = Frequency; default = $\frac{365d}{y}$

D = Duration; default = 70y

To demonstrate the effects of using the unit risk factor distributions, the distributions for each isotope, as derived in the preceding paragraphs, were substituted for the EPA values in equation 1. Monte Carlo methods (Crystal Ball ® Decisioneering, Boulder, CO.) were used to calculate individual lifetime risks for daily ingestion of two liters of water over a seventy-year lifetime (Table 3-4).

Table 3-4. Comparisons of individual lifetime risks (ILR) for cancer mortality from ingesting either ^{226}Ra (1 pCi/L) or ^{228}Ra (1 pCi/L) for EPA and use of risk factor distribution[1].

	Statistics	ILR ^{226}Ra	ILR ^{228}Ra
EPA		4.4×10^{-6}	3.8×10^{-6}
Lognormal Distribution	Average	8.6×10^{-7}	7.4×10^{-7}
	SD	8.7×10^{-7}	7.6×10^{-7}
	Lower 90% Boundary	6.3×10^{-8}	5.4×10^{-8}
	Upper 90% Boundary	4.4×10^{-6}	3.8×10^{-6}

[1]Ingestion of two liters per day for 70 years (EPA 1991a)

Tapwater Intake.

The EPA uses a conservative daily water intake of two liters in its exposure calculations. Ershow and Cantor, 1989 calculated a national average tapwater intake of 1.2 L/d (SD, ± 0.7L). Tapwater intake includes beverages and foods prepared in the home using domestic tap water, as opposed to total water intake that includes purchased beverages and foods containing water from sources other than the home. For the general population, consumption of 2 L/d was observed at the upper 90th percentile level, although an adult male may drink more than 4 L/d. Roseberry and Burmaster (1992) reexamined the data of Ershow and Cantor, and found that the data fit a lognormal distribution.

Table 3-5 compares the lifetime individual risks (ILR) from a lognormal distribution of tapwater ingestion vs. the risks reported by the EPA for 2 L/d. For the risk calculations, tapwater intake during a 70 year duration is multiplied by the unit risk factors (^{226}Ra, 2.2×10^{-6}; ^{228}Ra, 1.9×10^{-6}) derived from the EPA values. Monte Carlo methods (Crystal Ball ®, Decisioneering, Boulder, CO) are used to calculate individual lifetime risks for ingestion of a lognormal distribution of water containing Ra.

Exposure Duration (Residence).

EPA's criteria document for radium only shows (committed) doses from 70 years of chronic exposure, although RADRISK produces yearly estimates (EPA, 1991b). This forces one to assume that an individual resides in one place and receives a uniform exposure over a 70-year lifetime (Table 3-2).

ILR in the preceding tables were calculated on the basis of 70 years of exposure. According to the EPA (1990), 9.4 years, the 50th percentile, represents the average duration of residence in one location by a homeowner, and the 90th percentile, 29.8 years, represents a reasonable worst case. A recent investigation reported that 7.8 years (SD, ±4.3 years) is the average total residence time for U.S. households in rural areas (Israeli and Nelson, 1992).

Table 3-5. Individual lifetime risks (ILR) for cancer mortality from ingesting either ^{226}Ra (1 pCi/L) or ^{228}Ra (1 pCi/L), using EPA risk factors for EPA water intake assumptions and water intake distribution[1].

	Tapwater Statistics	Tapwater Ingestion (L/d)[3]	ILR ^{226}Ra	ILR ^{228}Ra
EPA[1]	Default	2	4.4×10^{-6}	3.8×10^{-6}
Lognormal Distribution[2]	Average	1.2	2.6×10^{-6}	2.2×10^{-6}
	SD	0.7	1.4×10^{-6}	1.2×10^{-6}
	Lower 90% Boundary	0	2.9×10^{-7}	2.5×10^{-6}
	Upper 90% Boundary	4.2	9.0×10^{-6}	7.8×10^{-6}

[1]EPA (1991a)

[2]Based upon the data of Ershow and Cantor (1989)

[3]Seventy-year duration

To correct for exposures that are less than lifetime, 10 years are added to the duration of exposure (Table 3-6) to estimate a committed dose that is based on the retention function for Ra (ICRP, 1973). The committed dose is slightly overestimated, but the estimate is less conservative than the committed dose from a seventy-year exposure.

Risk Calculations For Less Than Lifetime Exposures.

D is a distribution of residence duration for rural areas (years of residence, Figure 3-1) derived from the data of Israeli and Nelson (1992). The distribution of residence duration, D, modified by the addition of 10 years, expressed as a fraction of total lifetime (D/70) is then multiplied by the ILR to calculate the adjusted individual lifetime risks (Table 3-7).

$$[(D + 10) \text{ years}] \times (\text{ILR}) \text{ deaths} / 70 \text{ years} = \text{adjusted ILR (deaths)} \qquad (2)$$

Figure 3-1. Distribution of rural residence duration

Table 3-6 Percentage of committed effective dose (CED) during and subsequent to one years' intake of radium, based upon the retention (R*) of ingested radium. Note that the number of digits is for intermediate calculation purposes and does not necessarily represent the accuracy of the estimate.

Year (t)	%CED
1	31.6324
2	12.4867
3	9.4791
4	7.9366
5	6.9568
6	6.2641
7	5.7411
8	5.3284
9	4.9920
10	4.7110
11	4.4718
12	0.0000

*$R(t) = 0.54t^{-0.52}$

Effects of Replacing Defaults with Distributions on Individual Risks for Cancer Fatality.

Replacement of EPA default values with the distributions cf parameters described above can reduce the risk estimates by an average exceeding one order of magnitude (Table 3-8). The EPA's proposed standards for radium in drinking water (20 pCi/L) are based on individual lifetime risks of 10^{-4}. These risks are reduced to less than 10^{-5}, if the distributions of the unit risk factors, water ingestion rates, and duration of exposure are substituted in the risk calculations.

Table 3-7. Comparisons of individual lifetime risks (ILR) from daily ingestion of water[1] containing Ra during a residence period that is less than a lifetime (70 years) duration to values based on lifetime exposure.

	Statistics for D[2]	^{226}Ra	^{228}Ra
Lifetime exposure	Default	4.4×10^{-6}	3.8×10^{-6}
Exposure based on distribution of residence duration	Average	8.6×10^{-7}	7.4×10^{-7}
	SD	8.7×10^{-7}	7.6×10^{-7}
	Lower 90% Boundary	6.3×10^{-8}	5.4×10^{-8}
	Upper 90% Boundary	4.4×10^{-6}	3.8×10^{-6}

[1]2 L/d

[2]D = Duration of Exposure = Residence (Years).

Table 3-8. Comparison of Individual Lifetime Risks (ILR)[1] , based on EPA defaults, to risks calculated by simultaneous substitution of distributions for three parameters (unit risk factor, water ingestion rate, and duration of exposure)[2] for the defaults.

	Parameter Values	^{226}Ra	^{228}Ra
EPA	Default	4.4×10^{-6}	3.8×10^{-6}
Distributions[2]	Average	3.3×10^{-7}	2.2×10^{-7}
	SD	4.4×10^{-7}	3.2×10^{-7}
	Lower 90% Boundary	3.2×10^{-9}	1.5×10^{-9}
	Upper 90% Boundary	5.1×10^{-6}	4.5×10^{-6}

[1]per $pCi \cdot L^{-1}$

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4. RADON

Radon in tap water leads exposure by ingestion and inhalation. Since radon quickly escapes to the air once water is taken from the tap, exposure by ingestion applies only to water drunk immediately from the tap without further handling. All other indoor exposure is by inhalation of radon that has escaped from water into the air. An important route from drinking water to inhaled radon is showering.

It is important to keep the potential inhalation exposure to radon from drinking water in perspective, considering the overall exposure to airborne radon in a home. EPA's own estimate is that radon in water accounts for only 1-5% of radon levels in indoor air in homes. Quite apart from the projected risk reduction from removing radon from drinking water, it must be remembered that even eliminating all radon from drinking water would make almost no difference in indoor exposures to radon.

EPA systematically chose for its assessments high values (often maximum values) from the range of reasonable values of parameters. Often selection of a maximum value is stated specifically, without regard for the important distinction between the need for high-quality information for risk assessment, included in which is uncertainty, and the need to protect the public, which usually includes a safety factor. Each specific overestimate is discussed in detail below. Table 4-1 summarizes the general order of overestimates compared to realistic, mean values. Both the factor overestimate in the individual parameter and the factor overestimate in the overall result (all other parameters unchanged) are given.

Table 4-1. Overestimation of radon risks in EPA proposed MCL.

Parameter	Overestimation factor
Ingestion	3.8 times
Inhalation	2.5 times
Total Risk: all factors	2.7 times

Consumption of Tap water.

Direct consumption of tap water refers to water drunk immediately from the tap without handling or processing. It excludes water used in preparing food and

drinks, from which radon is assumed to be lost to the air by diffusion. EPA reduced its estimate of water consumption from 2 to 1 liter/day to account for such losses, specifically stating that 1 liter/day is "a reasonable maximum," not an expected value.

As stated by the SAB/RAC, direct consumption of tap water is usually taken to be 0.66 liters/day. The Life Systems, Inc. assessment of radon exposure for EPA also stated that high loss rates of radon from water during food preparation and cooking ensure that only radon in water consumed directly from the tap (0.6-0.7 l/day) is ingested (Life Systems, 1991). These direct consumption estimates are consistent with estimates of total consumption of tap water averaging 1.2 liter/day in a national survey (Ershow and Cantor, 1989).

Assuming direct consumption of water is 1 liter/day instead of 0.66 liter/day introduces a 52 percent bias to this parameter, thus increasing total estimated risk from waterborne radon by 9 percent.

Dose-Response Function for Radon Ingestion

Crawford-Brown (1991) developed the dose-response function for ingested radon used by EPA. The most significant source of uncertainty in the Crawford-Brown estimate is in the value of the "quality factor," a constant that quantifies the relative effectiveness of high-LET alpha radiation in penetrating and damaging tissues compared to that of low-LET radiation. ICRP recommends a quality factor of 20 for alpha radiation, meaning that it is 20 times more damaging than low-LET radiation (ICRP 26, 1977). While this may be reasonable for a source in direct contact with sensitive tissues, the mucus layer and lining of the gut are thick and difficult to penetrate; for the stomach, the quality factor must necessarily be lower than 20. For the rulemaking, EPA assumed a more realistic factor of 8 rather than 20, producing a dose-response function of 1.5×10^{-7} excess cancer deaths per pCi/L radon ingested.

Even 8 is a high quality factor. Crawford-Brown states that there are no experimental data for ingested alpha emitters demonstrating a quality factor higher than about 3 (Crawford-Brown, 1992), and the expected value may be less (Sun, 1992). Thus, a quality factor of 3 probably represents an upper limit. A quality factor of 3 (or less) yields a dose-response function of 6×10^{-8} (or fewer) excess cancer deaths per pCi/L radon ingested.

Use of a dose-response function for ingested radon of 1.5×10^{-7} excess cancer deaths per pCi/L instead of 6×10^{-8} introduces a 150 percent bias to this parameter, which increases total estimated risk from waterborne radon by 25 percent.

Long-term Average Inhalation Exposure from Tap water.

EPA used the transfer factor method of estimating long-term average exposure indoors from radon in tap water. In this method, the long-term average

ratio of radon concentration in water to radon concentration in indoor air is assumed to be constant. A transfer factor of 10,000:1 means that 10,000 pCi/L radon in tap water will produce 1 pCi/L long-term average radon concentration in indoor air.

Drawing on its review of several reports, EPA estimated that this ratio had an overall mean between 10,000:1 and 15,000:1 and selected 10,000:1, the conservative end of this arbitrary range. It is not clear from where EPA obtained the value 10,000:1, which is only mentioned in the underlying reports as "the typically cited value of 1×10^4" (Nazaroff et al, 1987). The full range of measured transfer factors is about 2,600:1 to 33,000,000:1 (Life Systems, 1991). Nazaroff et al. (1987) reviewed the available data and calculated a geometric mean transfer factor of 15,400:1 with a 95% confidence range of about 1,900:1 to 122,000:1. The arithmetic mean of the Nazaroff, et al. data is 8800:1, which EPA incorrectly reports to be 9100:1 (Life Systems, 1991). Similarly, Becker and Lachajczyk calculated a "representative" transfer factor of 14,300:1 with a "reasonable" range of 2,860:1 to 58,800:1 (Becker and Lachajczyk, 1984). Thus these two independent reviews select about 15,000:1 as a reasonable average (Nazaroff et al, 1987; Becker and Lachajczyk, 1984).

A recent study of 28 houses by Lawrence, et al., completed after the EPA assessment was printed, compared measured concentrations of radon in houses with those predicted by the Nazaroff, et al. equation (Lawrence et al., 1992). The tap water in these houses had exceptionally high concentrations of radon. Using low and high assumptions about air exchange rates in the Nazaroff equation, estimated geometric standard deviation ratios were 8700:1 and 21,100:1, respectively, with geometric standard deviations of 1.8. The mid-point of these assumptions yields a geometric mean of 14,900:1. These results appear to support the conclusions of the earlier reviews, that a reasonable average is about 15,000:1. The authors conclude, however, that the Nazaroff model underestimates the concentration of radon gas indoors derived from water in some cases, so the average ratio might be slightly lower. The authors also conclude that the variability of indoor concentrations is so high that measurements alone have limited usefulness, and suggest that some long-term average model as that used by Nazaroff, et al. should be used.

The data from Lawrence, *et al.* also provide information on the relative importance of radon from water in areas having high concentrations of indoor radon from all sources. Figure 4-1 shows the distribution of estimated contributions of tap water for 28 high-radon houses. Clearly the estimated contribution to the concentrations of radon in these houses is much higher than the often stated 1 to 5 percent average and the 2 percent obtained by Nazeroff, et al.

Using a transfer factor of 10,000:1 instead of 15,000:1 introduces a 50 percent bias to this parameter, which increases total estimated risk from waterborne radon by 42%.

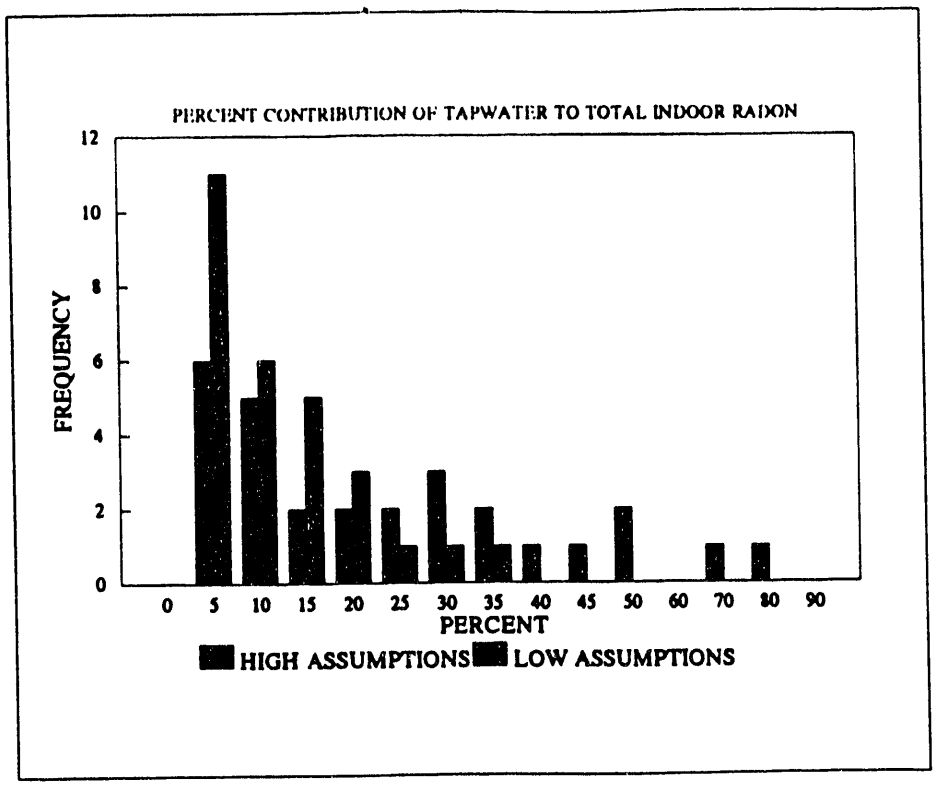

Figure 4-1. Distribution of contributions of water-borne radon to total indoor radon in high-radon houses (Source: Lawrence et al, 1992).

Dose-Response Function for Radon Inhalation

The EPA analyses used a dose-response function for inhaled radon progeny of 360 deaths per 10^6 working level months (WLM) exposure. This value is now obsolete. Based on the findings of BIER IV, EPA asked the National Research Council to reexamine the differences between underground miners and the general public with respect to characteristics affecting dose per unit exposure to inhaled radon progeny. The resulting NRC analysis, published in 1991 as a "Companion" to BIER IV, estimated that the dose per unit exposure is about 30% lower than previously assumed (NAS, 1991). In response, EPA has revised its "official" radon dose-response function; it is now 220 deaths per 10^6 WLM exposure to radon progeny (Puskin, 1992). This revised estimate was not used in the EPA rulemaking.

Use of a dose-response function for inhalation of radon progeny of 360 deaths per 10^6 WLM exposure instead of 220 deaths per 10^6 WLM exposure introduces a 64 percent bias to this parameter, which increases total estimated risk from waterborne radon by 53 percent.

Exposure to Radon from Showers.

Except for the systematic conservative biases mentioned above, the EPA analysis is based on standard assumptions about expected values of parameters (Life Systems, 1991). But the analysis includes only concentrations of radon gas, not exposure to radon progeny. This places excessive emphasis on exposures in showers, which have high radon gas concentrations, but low progeny concentrations and resulting radiation exposure.

EPA's conclusion that the dose per unit concentration of radon gas would be lower in a shower than in the general home environment is correct. We present a more complete analysis of this exposure here.

The EPA analysis of exposure to indoor radon from showers is based on the model of McKone (1987) applied to radon-222 gas (Life Systems, 1991). This model predicts that concentrations of radon gas can become exceedingly high in a shower (Figure 4-2). From this, EPA concludes that total daily exposure to radon gas is dominated by short-term, high-level exposures while showering. While this conclusion may be true for radon gas, it is not the gas that is harmful to human health; it is the particulate decay products that are harmful. The concentration of radon progeny, measured in working levels (WL), is much slower to develop and remains much longer in any particular environment than does the parent radon gas.

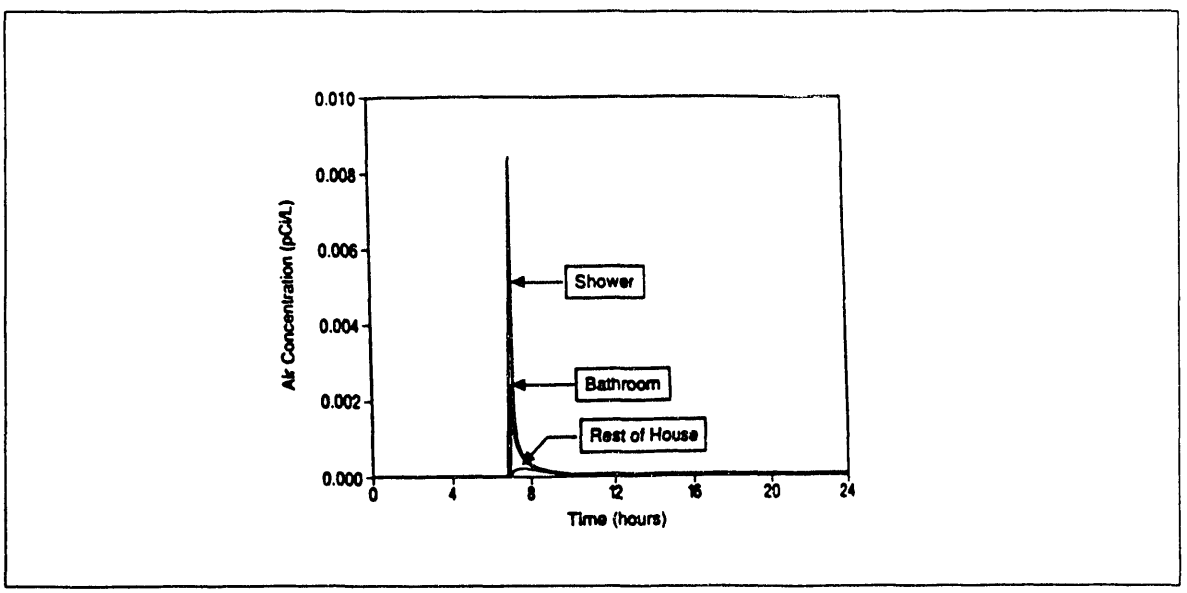

Figure 4-2. Predicted radon concentration in air of shower, bathroom and rest of house attributable to 1 pCi/L radon in shower water: basic exposure scenario (Source: Life Systems, 1991).

To account for all of the decay products of radon gas, we developed a three-compartment mass-balance model, much like that of McKone's, that includes

formation, deposition, and decay of all radon progeny (Figure 4-3). For this analysis, the three compartments represent a shower, bathroom, and the rest of a house; all characteristics of the compartments and the rate constants are the same as those used by EPA (Life Systems, 1991). The only required additions to the EPA assumptions are decay constants and deposition rates for radon progeny. Deposition is assumed to be 1.5%/min., the average for an indoor environment, for all progeny (Rowe, 1992). This is probably low for the aerosol-laden air of a shower and bathroom, so it yields an overestimate of the concentrations of particulate radon progeny.

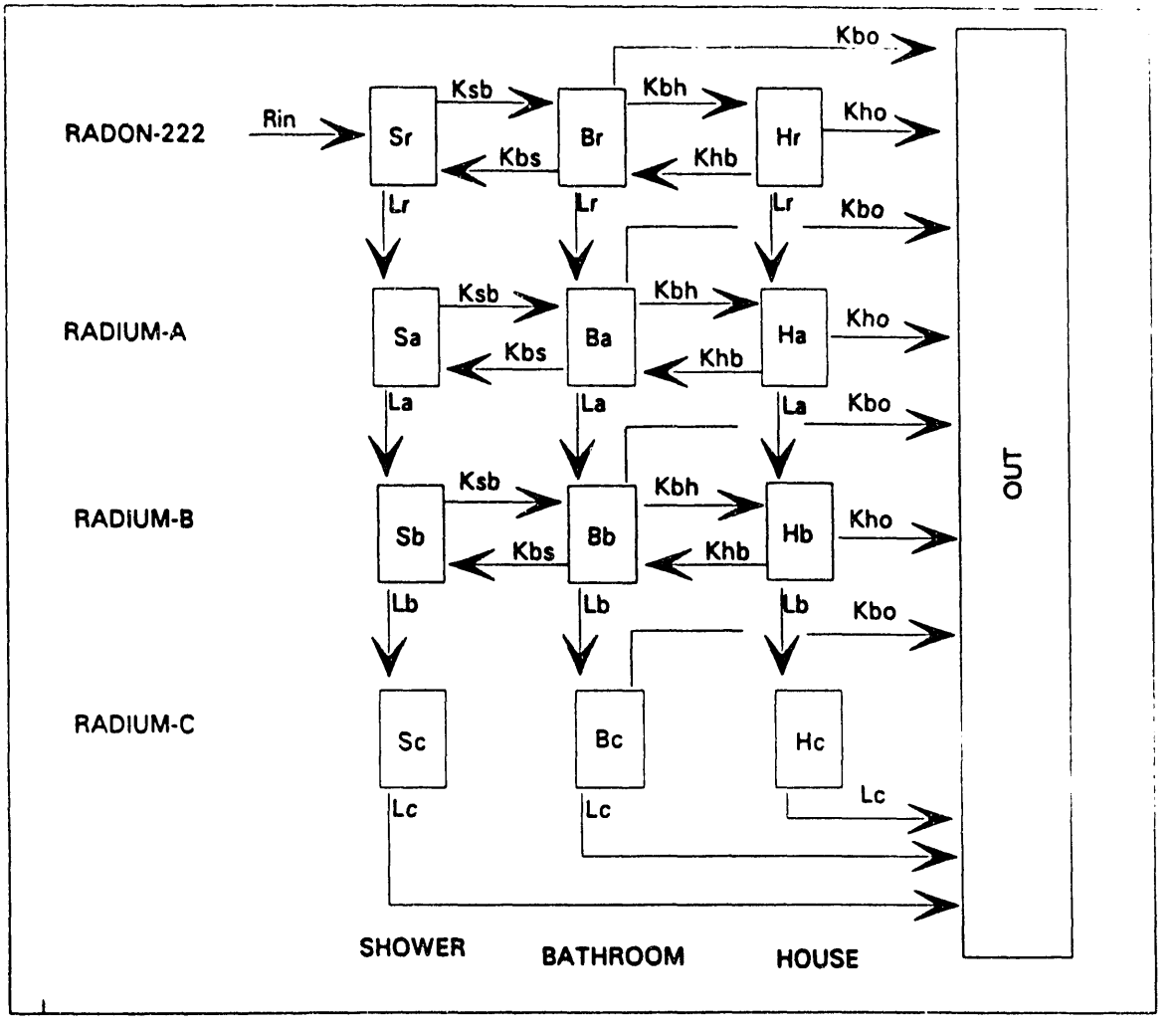

Figure 4-3. Three-compartment model of distribution, deposition, and decay of radon progeny indoors.

Figure 4-4 shows the predicted concentration of radon gas in the model shower, bathroom, and house under the conditions modeled by EPA (Figure 4-2). Parameters used in the analysis are given in Table 4-2. Figure 4-5 shows the resulting concentrations of radon progeny in the three compartments. These distributions differ from the results of the EPA model in two significant ways:

- The differences between the concentrations in the shower and the bathroom are much smaller, and probably not worth worrying about. The shower and bathroom can be modeled as a single compartment.
- The delay in formation of radon progeny produces a peak concentration in the shower and bathroom after the bather is likely to have left. Persons following the first bather are at greater risk.

Figure 4-6 shows the time-dependent concentration of progeny for an average individual spending a total of 20 minutes in the bathroom, with 8 minutes in the shower (EPA, 1990). An informal survey of 14 people in our office yielded 7 who spend more time in the bathroom before showering than after (mostly men), and 7 who spend more time in the bathroom after showering (mostly women).[1] We therefore assumed that the 20 minutes spent in the bathroom include 6 minutes before showering, 8 minutes in the shower, and 6 minutes following the shower.

It is clear from Figure 4-6 that, although the concentration in the bathroom reaches a peak of twice that in the rest of the house, the duration of that exposure is so short relative to the retention time of radon progeny in the rest of the house that its relative contribution to the total exposure is small. Under the conditions modeled, the house is essentially cleared of radon from a single shower within six hours, and only 10 percent of the total daily exposure is attributable to time spent in the bathroom and shower. Neither EPA's analysis nor this analysis included the estimate of exposure from showers in calculations. EPA used its high estimate of exposure from showering as one justification for using a conservative estimate of indoor inhalation exposure. In contrast, our analysis shows that the contribution of showering to overall indoor exposure is small, supporting our lower estimate of indoor radon concentration per unit concentration in water.

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[1] Following Gregor Mendel, the survey was stopped when the desired relationship had been obtained.

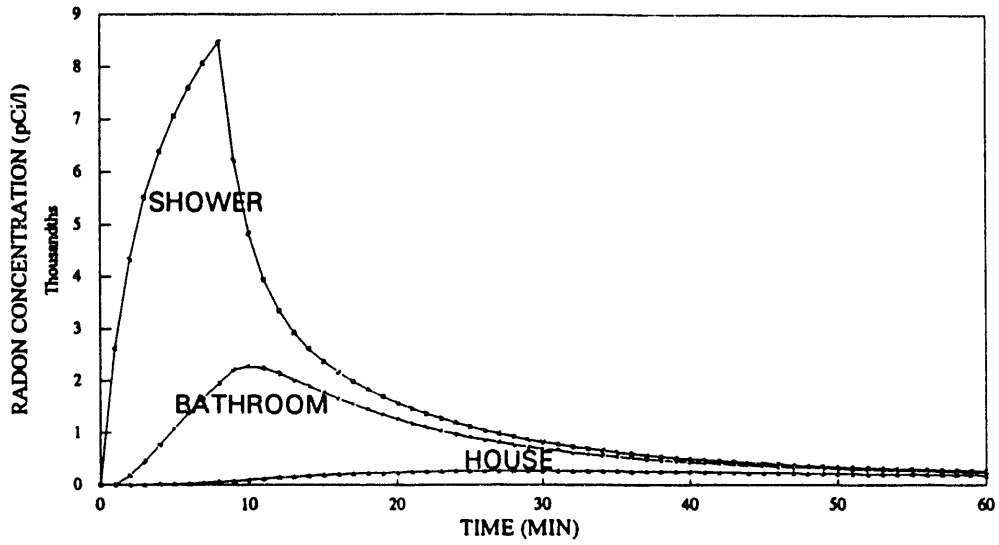

Figure 4-4. Estimated indoor concentration of radon gas from 8 minute shower with water containing 1 pCi/L radon..

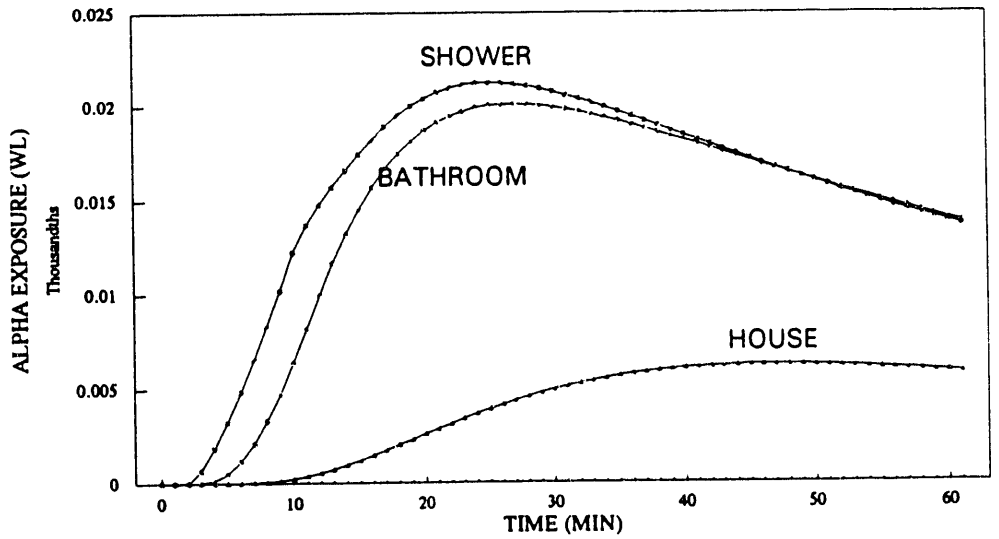

Figure 4-5. Estimated alpha exposure by radon progeny from 8 minute shower with water containing 1 pCi/L radon.

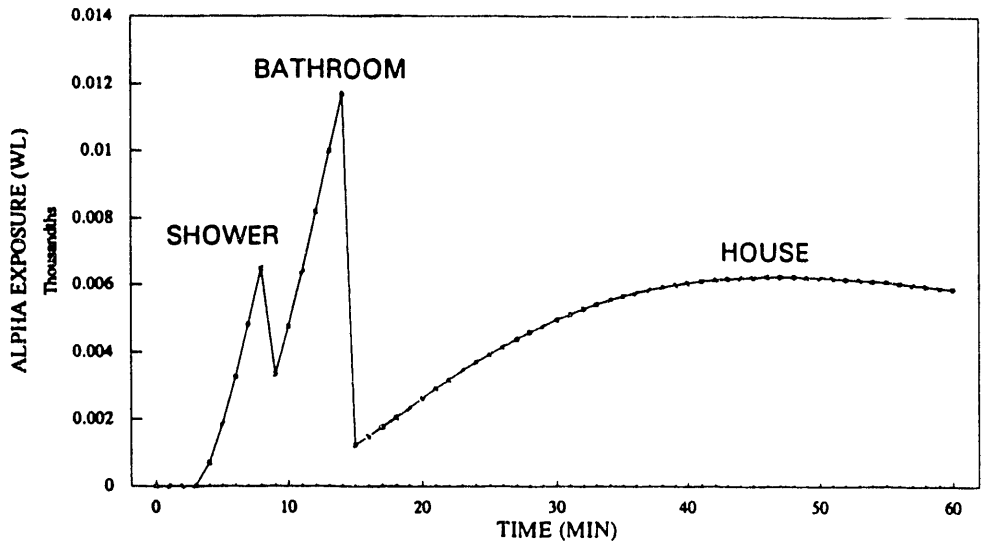

Figure 4-6. Time-dependent exposure to alpha radiation from 8 minute shower with water containing 1 pCi/L radon followed by 6 minutes in bathroom.

Conclusion

Table 4-3 summarizes the differences between EPA's conservative assumptions and our best-estimate assumptions. Table 4-4 shows the amount of overestimation. The overestimation factor for parameters is the individual bias for each parameter; the overestimation factor for total risk is the effect of the overestimation of that parameter on the estimated total risk, holding everything else constant. Because the total is a sum of two terms, rather than a product, and the terms are not equally important (inhalation provides more than 80 percent of the total risk), the total is not a linear function of the individual terms. The greatest bias introduced by EPA's conservatisms is in the dose-response function for ingestion. The bias in this function, however, has less impact on total estimated risk than other biases because ingestion is a small portion of the total risk. The largest impact of EPA's conservative biases is from the inhalation dose-response function.

The total effect of EPA's conservative biases is to increase estimated risk from waterborne radon by a factor of 2.7. Thus, EPA's estimate of total risk is 2.7 times what it would be were the best estimates of the values of parameters used instead of conservative estimates.

Table 4-2 . Model coefficients.

Concentration of radon in water	1 pCi/L
Radon transfer rate	0.7
Shower time	8 min
Bathroom time	20 min
Water volume	60 L
Shower stall volume	2 m^2
Bathroom volume	10 m^2
Remaining house volume	86 m^2
Transfer coefficients:	
Shower to Bathroom	0.347/min
Bathroom to Shower	0.0694/min
Bathroom to House	0.0926/min
Bathroom to Outdoors	0.0067/min
House to Bathroom	0.01077/min
House to Outdoors	0.0196/min
Radioactive decay rates:	
Radon-222	0.000126/min
Radium-A	0.2278/min
Radium-B	0.0258/min
Radium-C	0.0352/min
Particle deposition rate	0.015/min

Table 4-3. Summary of assumptions that differ between the EPA analysis and this study.

Parameter	Units	EPA Estimate	This Report
INGESTION			
Direct consumption of tap water	L/day	1	0.66
Ingestion dose-response function	Lifetime deaths per pCi/L	$1.5*10^{-7}$	$6.0*10^{-8}$
Total risk from ingestion	Lifetime deaths per pCi/L	$1.5*10^{-7}$	$3.9*10^{-8}$
INHALATION			
Indoor radon concentration per unit concentration in water	[pCi/L]/[pCi/L]	$1.0*10^{-4}$	$6.7*10^{-5}$
Inhalation dose-response function	Lifetime deaths/10^6 WLM	360	220
Total risk from inhalation	Lifetime deaths/pCi/L	$4.87*10^{-7}$	$1.98*10^{-7}$

Table 4-4. Overestimation of radon risks in EPA proposed MCL.		
Parameter	Overestimation factor	
	For Parameter	For Total Risk
INGESTION		
Direct consumption of tap water (l/d)	1.5 times	1.1 times
Ingestion dose-response	2.5 times	1.3 times
Total ingestion	3.8 times	1.5 times
INHALATION		
Indoor radon concentration per unit concentration in water	1.5	1.4
Inhalation dose-response function	1.6	1.5
Total Inhalation	2.5	2.2
Total Risk: all factors		2.7

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