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Protection of Human Health from Mixtures of Radionuclides and Chemicals in Drinking Water[1]

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Abstract. This study was undertaken to develop a common scale for evaluating health risks from contaminated drinking water. For different agents, many unrealistic models of risk have been used. By intent, regulatory toxicology depends on "data-sparse, model-intensive" analogies from exotic animal genetics and novel exposures (NCRP 1989). The question is, does a risk evaluation so derived have any predictive validity? Absence of data prevents answer because regulatory toxicology rationalizes in step-by-step logic, which we call *absolute* (i.e., predicts cases of disease in a population). Absolute models ensure safety, but do so at the cost of realism. In contrast, we make *relative* comparisons in the manner of horsepower or RBE from radiation biology. All pollutants are assumed to contribute to toxic injury. Next, relative potencies are linked to the most credible standards. Thus, experience is transferred from well-studied chemicals to the new chemical by "data-intensive, model-sparse" methods. This logos provides much relative precision. Then, pollutants are compared with: (1) common foodstuffs, (2) ambient radiation background, or (3) utility-pure drinking water. Finally, an assessment is made for a waste disposal area.

This study was charged to develop a common scale for mixed wastes. Two common-scale methods are offered—one *risk* based, the other *safety* based. The approach depends on the belief that classification as a "noncarcinogen" is tentative, depending on how a particular expert committee evaluates the contemporary weight of evidence (for carcinogenicity).

Background

Radiological and chemical hazards are evaluated and regulated independently. Chemical carcinogens have been hypothesized as having different mechanisms of action from classical pharmacological/toxicological hazards. Also, variable levels of risk have been used to establish statutes, depending on whether the evaluation was from epidemiological or laboratory data, *e.g.*, a more protective analogy is used for animal-based estimates, but that more protective model has on occasion been used with a 10-fold less protective level of permissible risk (EPA 1986b).

At least four methods having at least a similar number of hazard control levels (4 × 4) are currently used by rulemakers. Also, there has been no rational, objective way to express the composite hazard from multiple pollutants or complex mixtures of carcinogens and noncarcinogens. Thus, risk management has relied on decisions of expensive proportions from incoherent analogies.

We are using those historical, regulatory methods where possible, but they are supplemented with additional data and methods to produce common scales. Figure 1 shows how diverse data and models are integrated. Two concepts, *viz.*, risk and safety, used to calibrate the different common scales, are shown where the box on "Rapid Screening of Hazard" feeds boxes on "QRA Models" and "generally recognized as safe" (GRAS). One common scale is calibrated to risk, and others to reference exposures which are practically unavoidable.

RADRISK was used; the model was developed for the EPA (Sullivan *et al.* 1981), to estimate health risks from radionuclides. Dose calculations were coupled with a life-table methodology. The cohort risk baseline included competing risks based on the U.S. population. Lifetime exposure to a unit concentration of 154 radionuclides by ingestion or inhalation was assumed.

The EPA considers noncarcinogenic chemicals in the classical pharmacological/toxicological sense. That approach assumes a no-observable-adverse-exposure-level (NOAEL) can be established for chemicals that act through toxification/detoxification mechanisms. Exposures below the

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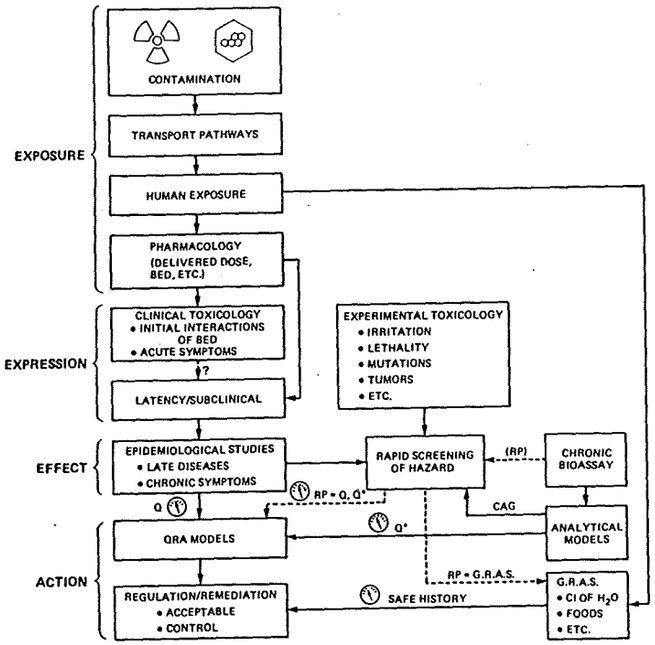

Fig. 1. Integration of the various issues affecting the evaluation of potential health hazard relevant to human exposure to toxic chemicals and radionuclides.

threshold are postulated to represent no hazard—even for concomitant exposures with carcinogens.

Ten chemicals are carcinogenic from epidemiology: acrylonitrile, arsenic, benzene, benzidine, beryllium, cadmium, chromium VI, coke oven emissions, nickel refinery dust, and nickel subsulfide (EPA 1987). Risk coefficients are from linear models like those used in RADRISK. Risk coefficients for the 49 other EPA carcinogens have additional margins of safety [by factors up to 10,000 (Barnard 1987)] to allow for differences between animal and human data.

Method

Chemicals vary in potency by 10^7-fold. For this and other reasons described, a chemical-scoring system was developed for the U.S. Air Force, currently being used in the Environmental Restoration Program of the Department of Defense (1987, 1989). The method was called RASH (*R*apid *S*creening of *H*azard). The need to compare biological test data (across pollutants two-by-two) required a definition of relative potency that is only a slight editing of the radiological RBE, *viz.*, the relative potency of agent A with respect to the reference agent, B, is defined in terms of the treatments, T_A, and T_B, which produce equal biological effects. For equal responses, *relative potency* is then T_B/T_A. Relative potency, like RBE, is unit-

Table 1. "Immature" or interim guidance values projected from the relative potency of a chemical and the statutory values for other chemicals. The table illustrates how such experiences can be used to identify a composite reference level, which is then used to predict initial permissible concentrations that may be proposed for currently unregulated chemicals. Expected accuracy is about an order of magnitude

Chemical	EPA-RfD (mg/L)	Relative potency[a]	Column 2 × Column 3[b]
Mercury	0.002[d]	0.40	0.0008
Lead	0.05	0.092	0.0046
Toluene	2.0[d]	0.0038	0.0076
Pentachlorophenol	0.2[d]	0.11	0.022 = Median[c]
Cresol	2.0	0.015	0.030
Phenol	4.0	0.027	0.11
Barium	5.0[d]	0.030	0.15

[a] Potency relative to B[a]P as estimated from RASH
[b] Based on this simple comparison, it appears the RfD value for mercury is significantly more cautious than for other chemicals shown here
[c] For any currently unregulated chemical in drinking water, projections of initial regulations can be made from RfD_i = (0.022 mg/L)/RP_i, where RP_i is from Table III of Jones et al. 1988
[d] EPA 1989

Table 2. Median value of hazard for chemicals having no statutory risk coefficients is obtained from epidemiologically based risk coefficients modified by relative potency values

Chemical	Risk coefficient[a] from CAG (mg/kg/D)$^{-1}$	Risk coefficient[b] adjusted for oral intake (mg/kg/D)$^{-1}$	Potency[c] relative to B[a]P	(Col. 3/Col. 4)[d]
Beryllium oxide	7.0 (w)	0.014[e]	0.29E+01[e]	0.0048[e]
Chromium VI	41. (w)	8.2	0.44E+02	0.19
Acrylonitrile	0.24 (w)	0.23	0.11E+01	0.21
Nickel subsulfide	1.7 (w)	1.4	0.50E+00	2.8
Coke oven emissions	2.16 (w)	4.0	1.00E+00	4.0
				← Median
Nickel refinery dust	0.84 (w)	0.70	0.13E+00	5.4
Arsenic	1.5 (h)[f]	1.5	0.16E+00	9.4
Benzene	0.029 (w)	0.062	0.50E−02	12.
Cadmium	6.1 (w)	0.92	0.79E−01	12.
Benzidine	234 (w)	222	0.60E+01	37.

[a] From EPA/600/8-84/026F. w = human occupational exposures; h = human drinking water exposures; [b]Column 2 × $A_{oral}/A_{inhal.}$; [c]Jones *et al.* (1988) and Owen and Jones (1990); [d]Because the risk coefficient is directly proportional to the potency of a chemical, the risk coefficient divided by the relative potency should be constant if all data were equally valid and if regulatory guidelines were consistent (Owen and Jones 1990). Note: permissible concentrations for a constant level of risk are inversely proportional to potency values as shown in Table 3; [e]The CAG value for beryllium oxide was adjusted according to the absorption coefficient and relative potency for beryllium; [f]Not corrected for absorption because risk coefficient is based on oral dose

Table 3. Risk coefficients and permissible concentrations that can be used to assess ORNL RAP chemicals as carcinogens or as noncarcinogens

		For control as a carcinogen					For control as a noncarcinogen	
RAP Chemical	Relative Potency[a] from RASH	Risk coefficient[b] based on RASH (mg/kg/d)$^{-1}$	Risk coefficient[c] from CAG (mg/kg/d)$^{-1}$	RASH[d] (μg/L)	RASH modified by absorption coefficient (μg/L)	EPA-CAG[e] (μg/L)	Projection of interim regs[e] from RASH (mg/L)	EPA water quality[f] (mg/L)
Arsenic	0.16E+00	0.75E+00	0.15E+01	0.19E+00	0.19E+00	0.23E+00		
Barium chloride	0.51E−01	0.24E+00		0.59E+00	0.59E+01		0.43E+00	5.0[g]
Barium (II) nitrate (1:2)	0.86E−02	0.40E−01		0.35E+01	0.35E+02		0.26E+01	(Barium)
Benzene	0.50E−02	0.24E−01	0.29E−01	0.60E+01	0.60E+01	0.12E+02		
Beryllium	0.29E+01	0.14E+02	0.70E+01	0.10E−01	0.10E+02	0.50E−01		
Cadmium	0.79E−01	0.37E+00	0.61E+01	0.38E+00	0.63E+01	0.50E+01[g]		
Chloroform	0.50E−02	0.24E−01	0.81E−01	0.60E+01	0.60E+01	0.43E+01		
Chromium	0.44E+02	0.21E+03	0.41E+02	0.68E−03	0.14E−01	0.85E−02[h]		
Copper	0.10E+00	0.47E+00		0.30E+00	0.60E+00		0.22E+00	
Cresol	0.15E−01	0.70E−01		0.20E+01	0.20E+01		0.15E+01	
1,1-dichloroethylene	0.14E−01	0.66E−01	0.12E+01	0.21E+01	0.23E+01	0.30E+00		
2,4-dimethylphenol	0.58E−02	0.27E−01		0.52E+01	ND		0.38E+01	
Di-*n*-butyl phthalate	0.23E−03	0.11E−02		0.13E+03	0.15E+03		0.95E+02	
Ethylbenzene	0.23E−02	0.11E−01		0.13E+02	0.16E+02		0.95E+01	0.70E+00[g]
Lead	0.92E−01	0.43E+00		0.33E+00	0.33E+01		0.24E+00	0.50E−01
Mercury—elemental	0.40E+00	0.19E+01		0.75E−01	0.75E+03		0.55E−01	
—inorganic	0.48E+00	0.23E+01		0.63E−01	0.42E+00		0.45E−01	
—organic	0.23E+00	0.11E+01		0.13E+00	0.14E+00		0.95E−01	0.20E−02
Methylene chloride	0.22E−02	0.10E−01	0.14E−01	0.14E+02	0.14E+02	0.25E+02		
Naphthalene	0.48E−02	0.23E−01		0.63E+01	0.63E+01			
Nickel	0.13E+00	0.61E+00	0.84E+00	0.23E+00	0.46E+01	0.42E+00		
PCBs	0.33E−02	0.16E−01	0.77E+01	0.91E+01	0.96E+01	0.50E+00[g]		
Phosphorus (phosphoric acid)	0.12E−01	0.56E−01		0.25E+01	0.28E+01		0.18E+01	
Selenium (selenious acid)	0.41E+00	0.19E+01		0.74E−01	0.12E+00		0.54E−01	0.50E−01
Toluene	0.38E−02	0.18E−01		0.79E+01	0.79E+01		0.58E+01	0.20E+00[g]
Vinyl chloride	0.31E−03	0.15E−02	0.23E+01	0.97E+02	0.11E+03	0.15E+00		
Xylene	0.35E−02	0.16E−01		0.86E+01	0.86E+01		0.63E+01	0.10E+02[g]
Zinc	0.56E−02	0.26E−01		0.54E+01	0.11E+02		0.40E+01	

[a] Jones et al. 1988 or from additional evaluations; [b]From method shown in Table 2. Value given is not adjusted for oral intake—see column 6 for that adjustment; [c]From the "Health Assessment Document for Beryllium." EPA/600/8-84/026F. Values are not adjusted for differential absorption (EPA and CAG do not usually incorporate absorption coefficients in estimates); [d]Computed according to (0.03 μg/L)/RP_i, where RP_i is the potency of the chemical relative to B[a]P and 0.03 μg/L is for PAH's in drinking water. See page 116 of Jones et al. 1988; [e]From Table 1; [f]Values from 40 CFR and EPA Documents; [g]Values from EPA 1989; [h]EPA 1989 is proposing 100 μg/l

Table 4. Derivation of GRAS baseline for toxic chemicals from (1) daily consumption of utility processed but otherwise pure drinking water or (2) the composite value from utility water and one reference meal

	T-bone steak	Bread	Lettuce	Tea	Water	Total
Consumption per day	10 oz. (280 g)	2 slices (57 g)	1.5 oz. (43 g)	1 cup (250 mL)	2 liters	—
Chemical used for comparison	B[a]P	B[a]P	B[a]P	Fluoride	Fluoride and chlorinations products	— — —
Concentration	50 ng/g	1 ng/g	1.6 ng/g	100 ppm	1 ppm and 28 μg/L	
Equivalent Toxic Dose of B[a]P[a]	14 μg	57 ng	69 ng	1200 μg[c]	92 μg[d]	1.3 mg
Dose of B[a]P (mg/kg/d)[b]	2.0E−04	8.1E−07	9.9E−07	1.7E−02	1.3E−03	1.9E−02
Risk coefficient from EPA-CAG $(mg/kg/d)^{-1}$	11.5	11.5	11.5	11.5	11.5	11.5
GRAS Index Value (Dose × Risk Coefficient)	2.3E−03	9.3E−06	1.1E−05	2.0E−01	1.5E−02	2.2E−01
GRAS Value for Hazard Index Equation					0.015	0.22

[a] The equivalent dose of B[a]P is computed from the product of the dose and the potency of that compound relative to B[a]P. Many other xenobiotics are present, but only the one in Row 2 is used for simplicity

[b] Based on daily consumption and a 70 kg reference body weight

[c] Fluoride contribution = (250 g) $(10^{+6}$ μg/g) $\frac{(10^{-6})}{\text{ppm}}$ (100 ppm) (0.046) ≈ 1200 μg

[d] Contribution from Cl products = (21 μg/L) (0.005) + (6 μg/L) (0.0065) + (1.2 μg/L) (0.021) = 0.17 μg/L, and fluoride = 46 μg/L

less. The second step is to couple relative potency to one or more reference exposures. *Note:* Neither the traditional wording of RBE nor our relative potency version make any requirements regarding biological mechanisms of response or biochemical pathways leading to the effect being compared. A common specious criticism is that relative comparisons should be limited to-agents having similar mechanisms of action—this assurance is not possible even for ionizing radiations.

RASH, a part of Figure 1, is well documented for 278 chemicals (Jones *et al.* 1988). The objective is to use robust statutory concentrations to transfer experience from one chemical to another. In so doing, a more complete picture derives from a series of incomplete overlays.

The RADRISK code is based on absorbed dose to organ tissues at risk, but statutes for chemicals are based on concentrations in air, water, or food. In some instances, the EPA considers an agent carcinogenic only if the agent has been demonstrated to cause cancer by the identical route of exposure. It is assumed that any carcinogenic agent can increase cancer in any organized tissue with which it comes in contact.

Each nucleated cell—in any tissue—contains the genetic code for the entire body. Only phenotypic expression distinguishes a nucleated cell in one organ from a nucleated cell in another organ. Gene function for cell proliferation is necessary for initiation of precarcinogenic lesions and clonal expansion into frank cancer. Evidence is ubiquitous (Jones *et al.* 1983). Lack of data for a particular route of exposure seems to be a specious way to identify carcinogens (Jones and Glass 1989). Only an understanding of metabolic pathways justifies assumed detoxification. Absorption coefficients for chemicals evaluated in this paper (Owen 1990) have been used by Owen and Jones (1990) to explore variability in statutory concentrations.

Historically, noncarcinogens have been controlled individually and without concomitant interactions with carcinogens (EPA 1986c). If a chemical is lacking one intrinsic trait needed to be a carcinogen, it has not been permitted to borrow that trait from other pollutants. But, "noncarcinogens" are commonly used experimentally to promote carcinogenesis initiated by other agents. A better logic would be to allow chemicals to borrow and lend properties of toxicity (Jones *et al.* 1983, NCRP 1989, ACGIH 1989).

Incomplete carcinogens may become carcinogens based on epidemiology. DDT, benzene, and arsenic may be examples. It is proposed that a threshold for noncarcinogens reflects "immature" regulatory science, because noncarcinogens may increase cancer in humans. This is supported by the NCRP's position (1989) that promoters induce cell proliferation and the ACGIH's note (1989) that physical irritation may initiate, promote or accelerate physical impairment through interaction with other chemical or biologic agents. Many noncarcinogens may eventually be regulated as carcinogens. Estimates for currently noncarcinogenic compounds are given in Table 1.

Potencies in Table 1 are relative to BaP. Table 1 chemicals were selected as benchmarks to transfer both qualitative and quantitative statutory considerations to currently unregulated chemicals. By the same process, projections for forthcoming statutes can be made (Jones *et al.* 1988). The EPA-CAG risk coefficients (based on epidemiology) define the standard of risk, shown as the median in Table 2. All chemical agents are linked to that standard by relative comparisons. But to assess relative hazard or safety, it is useful to adjust comparisons to a different standard, *e.g.*, chloroform (ubiquitous after water chlorination) instead of BaP.

The product of (1) relative potency, (2) absorption coefficient, and (3) statutory concentration should be constant across pollutants, ignoring uncertainty and agency-intended variations in risk. But the hazards associated with statutes for chemicals in Table 2 vary by 10000-fold (Owen and Jones 1990, Barnard 1987). Because it is not possible to select a "best" standard, the median is the most prudent measure of central tendency. The median would correspond to a

Table 5. Concentrations of chemicals computed to be of toxicity equal to per capita consumption of 2 L of utility pure water daily or consumption of water and a reference meal consumed daily

Chemical	GRAS-equivalent[a,b] based on drinking water (mg/L)	GRAS equivalent[c] based on drinking water and a reference meal (mg/L)
Arsenic	0.71	10
Barium chloride	22	320
Barium (II) nitrate (1:2)	130	1900
Benzene	22	320
Beryllium	38	550
Cadmium	24	350
Chloroform	22	320
Chromium (VI)	0.05	0.73
Copper	2.2	33
Cresol	7.5	110
1,1-Dichloroethylene	8.6	120
2,4-Dimethylphenol	19	280
Di-*n*-butylphthalate	560	8200
Ethylbenzene	58	860
Lead	12	180
Mercury		
elemental	2800	41,000
inorganic	1.5	22
organic	0.50	7.4
Methylene chloride	53	770
Naphthalene	23	330
Nickel	17	250
PCBs	35	510
Phosphorus (phosphoric acid)	10	150
Selenium (selenious acid)	0.46	6.8
Toluene	29	430
Vinyl chloride	390	5700
Xylene	33	480
Zinc (zinc oxide)	40	590

[a] Computed from Dose = Risk/[(Risk coef.) × A_{oral})], 70 kg body weight, and 2 L/day

[b] Risk is 0.015 based on Table 4

[c] Risk is 0.22 based on Table 4

hypothetical chemical with an intrinsic toxicity between coke oven emissions and nickel refinery dust. This is the benchmark to which the relative potency scale will be calibrated. The median risk coefficient is 4.7 × RP_i $(mg/kg/d)^{-1}$, and can be used:

$$\text{Risk} = 4.7 \times RP_i\ (mg/kg/d)^{-1} \times D_i\ (mg/kg/d) \quad [1]$$

for dose D_i of any chemical, with potency RP_i relative to BaP. Calculated risk can be decreased by modifying D_i by the oral absorption coefficient. Eqn [1] can also be used to estimate the daily dose that results in a specific level of risk, *e.g.*, 10^{-5}:

$$D_i\ (mg/kg/d) = \text{Risk}/[4.7 \times RP_i\ (mg/kg/d)^{-1}]. \quad [2]$$

D_i can be converted to concentration in water or air using appropriate assumptions.

Risk coefficients and 10^{-5} per capita lifetime risk concentrations are listed in Table 3 along with the derived concentrations based on EPA models. The EPA values for noncarcinogens and for animal-based carcinogens may be subject to major changes (Barnard 1987). Thus, for purposes of long-range planning, the concentrations based on columns 2 and 3 may be more realistic. Column 2 is based on the composite toxicity of each chemical, whereas the statutory value is usually based on only one bioassay. From Table 3, RASH-derived values (column 6) and EPA-derived values (column 7) are in close agreement for 7 of 11 comparisons. Elemental, inorganic, and organic mercury all seem to have similar levels of intrinsic toxicity (column 2), but a low absorption efficiency makes elemental mercury 1000-fold less hazardous (column 6). The *rightmost* value in Table 3 would be both safe and acceptable by consensus.

Safe or *safety* to the FDA "means that there is a reasonable certainty in the minds of competent scientists that the substance is not harmful [in] the intended . . . use. It is impossible . . . to establish . . . harmlessness . . . of any substance. Safety may be determined by scientific procedures or by general recognition . . . the following factors shall be considered: (1) the probable consumption . . . and (2) the cumulative effect . . . taking into account any chemically or pharmacologically related substance or substances . . . General recognition of safety may be based on the views of experts . . . The basis of such views may be either (1) scientific procedures or (2) in the case of a substance used in food prior to January 1, 1958, through experience based on common use in food . . ." (Delaney 1985). From this, it is proposed to use comparisons of relative toxicity to define GRAS-type concentrations that are consistent with evaluations of the FDA.

Table 4 offers different *safe* benchmarks for drinking water (Owen and Jones 1990). One baseline or GRAS index can be balanced against a reference meal plus 2 L of utility-pure water. A second can be adjusted to the daily consumption of water alone. The product of the chemical dose and the risk coefficient is taken as the GRAS index. At great expense, industrial compounds, natural pollutants, and purification byproducts in drinking water could be reduced to ever-decreasing concentrations. But, xenobiotics from foods and cooking processes dominate exposure (Ames 1989) so that, from a health perspective, cost of cleanup should be weighed against potential gain.

Some may question values in Table 4 because fluoride is highly toxic, having been used in rat poison and insecticides. The Commissioner of Food and Drugs has concluded that it is in the interest of the public health to limit the addition of fluorine compounds to (1) fluoridation of public water, (2) bottled water, and/or (3) to that authorized by 40 CFR Part 180 (FDA 1985). However, most of society desires fluoridation of drinking water. To avoid a fluoride debate, GRAS-like values can be normalized to residual contaminants from chlorination only—or any other preferred benchmark. In this manner, Table 4 is used to compute two composite index values (in the bottom line) to estimate GRAS-equivalent concentrations in Table 5.

Risk values for radionuclides (Sullivan *et al.* 1981), a risk coefficient of 2 × 10^{-6} fatal cancers per Gray, a dose of 4 × 10^{-4} Sv (40 mrem) per year, and a 70 year lifespan were used to compute a risk of 5.6 × 10^{-4} fatal cancers per person. We subjectively chose 40 mrem/y for whole-body natural background-ignoring internal exposures, cosmic radiations, and radon. The baseline of 5.6 × 10^{-4} was used to compute GRAS-equivalent doses for radionuclides in Table 6.

Results

The Hazard Index is for exposure to multiple agents (EPA 1986c). Many ratios contribute to

$$HI = E_1/C_1 + E_2/C_2 + \ldots + E_n/C_n \quad [3]$$

where E_i is the exposure and C_i is the corresponding statutory concentration. Our method develops consistency and permits combination of agents for reasons given previously. That combination is logically correct only if one of our

Table 6. Development of risk-specific concentrations and GRAS-equivalent index values for radionuclides

Nuclides	Deaths in 10^5 cohort[a] $(Bq/year)^{-1}$	Risk coefficient[b] (Bq/L)	Concentration for 10^{-5} chance of death[c] (Bq/L)	GRAS-equivalent (natural background equivalent concentration)[d,e] (Bq/L)	51FR34859 (4 mrem/year) (Bq/L)
^{3}H	4.86E−6	3.55E−8	2.8E+2	1.6E+4	3E+3
^{60}Co	3.35E−4	2.45E−6	4.1	2.3E+2	7
^{90}Sr	5.86E−3	4.27E−5	2.3E−1	1.3E+1	2
	3.95E−4	2.89E−6	3.5	1.9E+2	3E+1
^{99}Tc	5.19E−5	3.78E−7	2.6E+1	1.5E+3	2E+2
^{106}Ru	8.89E−4	6.49E−6	1.5	8.6E+1	1E+1
^{137}Cs	2.49E−3	1.82E−5	5.5E−1	3.1E+1	4
^{154}Eu	1.88E−4	1.37E−6	7.3	4.1E+2	4E+1[f]
^{232}Th	1.07E−2	7.78E−5	1.3E−1	7.2	
^{233}U	3.46E−4	2.52E−6	4.0	2.2E+2	
	1.39E−2	1.02E−4	9.8E−2	5.5	
^{235}U	3.57E−4	2.61E−6	3.8	2.1E+2	
	1.14E−2	8.30E−5	1.2E−1	6.7	
^{238}U	3.14E−4	2.29E−6	4.4	2.4E+2	
	1.16E−2	8.43E−5	1.2E−1	6.6	
^{239}Pu	7.84E−3	5.73E−5	1.7E−1	9.8	1E−1
	7.62E−2	5.57E−4	1.8E−2	1.0	
^{240}Pu	7.84E−3	5.73E−5	1.7E−1	9.8	
	7.62E−2	5.57E−4	1.8E−2	1.0	
^{241}Am	7.68E−2	5.59E−4	1.8E−2	1.0	1E−1
^{244}Cm	4.57E−2	3.32E−4	3.0E−2	1.7	

[a] Values converted from Table 7 of Sullivan et al. (1981). Second value for isotope is for least soluble compounds
[b] Example calculation ^{3}H: Risk Coef. $(pCi/L)^{-1}$ = (4.86E-6 deaths)/[(10^5 persons) × (1 Bq/y) (1 y/365 d) (1 d/2 L)]
[c] Concentration (Bq/L) = Risk/Risk Coefficient
[d] 200E−6 fatal cancers per person-cGy; 40 mrem/y; and lifespan of 70 years were used to compute a risk of 5.6E−4 fatal cancer/person
[e] Risk of 5.6E−4 used with footnote "c"

common-scale logos is used for C_n's. Statutes containing various margin of safety lead to an incoherent evaluation of the Hazard Index Model.

Example Assessment

Two groundwater samples from inside a radioactive low-level, solid waste disposal area, operated since 1968, are given in Table 7. Well T-92 was 4.12 m below the surface and T-257 was 3.60 m deep (Solomon *et al.* 1988). Only an abbreviated inventory of pollutants is shown. From either well T-92 or well T-257 measured concentrations exceeded statutory concentrations for 11 of 25 pollutants listed. But, no member of the public can be exposed to these concentrations because the site has controlled access. Also, compliance is mandated at site boundaries. However, assuming that someone could build a home on-site and sink a well—except for the fact that the groundwater yield at the site is too low to support a residential well—the exposure could be undesirable.

Measured concentrations at this site have been compared with EPA-standards. Six organic compounds were above the Safe Drinking Water Act Maximum Contamination Levels (SDWA-MCLs), barium and nickel were 2-fold higher, and ^{3}H, ^{14}C, and ^{137}Cs were greater than proposed MCLs (Trabalka 1987). The concentrations used by Trabalka corresponded to 100 mrem/y compared with 4 mrem/y promulgated by the EPA for drinking water.

In contrast, the GRAS-type comparisons suggest that only strontium-90 in well T-92 would be a hazard above commonly accepted foods and utility-pure water, even though levels were 400 times permissible concentrations calculated as the Hazard Index. Also, these calculations suggest that well T-257 is about 10-fold less toxic than well T-92. This comparison is based on concentrations for the indicated sample dates and could change (Solomon *et al.* 1988).

Conclusions

Data in Table 4 and EPA statutes were used to compare hazards (Figure 2). Data are based on toxicity, not on risk. All values are normalized to a pack of cigarettes (Owen and Jones 1990). Each substance was evaluated on daily consumption rate, and each water pollutant was evaluated on the basis of the EPA concentration. With respect to toxicological potential, the EPA statutory concentrations compare with a wide variety of GRAS-type foods. It should be noted that the ordinate is in $\log_{10}$ hazard units. As illustrated, (1) the regulatory standards for vinyl chloride and PCBs in drinking water may be less hazardous than consumption of lettuce which is grown near industry, by 100-fold and (2) EPA controls BaP in drinking water 100-fold below that consumed in only one 10 oz. T-bone steak. The statute in water would compare with eating only 1/10 oz of steak, daily—one steak in 3 months. These and similar comparisons have caused us to develop *relative* methods to evaluate the realism of *absolute* methods as used by regulatory agencies.

Table 7. Demonstration of the risk-based composite hazard index and the composite-Gras index for water samples from two wells inside a solid waste storage area. Values in the table are upper limit estimates in that the "<" symbol in columns 2 and 6 are ignored. NOTE: One column is omitted for Well T-257 because the hazard index is <1 in column 7

Agent	Concentraion Well T-92[a]	Hazard Index[b]	Hazard Index[c]	Hazard Index[d]	Concentration Well T-257[a]	Hazard Index[b]	Hazard Index[d]
Benzene	<0.0044	2.0E−4	1.4E−5	3.7E−1	<0.0044	2.0E−4	3.7E−1
Ethylbenzene	<0.005	9.4E−5	6.4E−6	7.1E−3	<0.005	9.4E−5	7.1E−3
Toluene	<0.005	1.7E−4	1.2E−5	2.5E−2	0.035	1.2E−3	1.8E−1
Chloroform	<0.0016	7.3E−5	5.0E−6	3.7E−1	<0.005	2.3E−4	1.2E+0
1,1-dichloroethylene	<0.0028	3.3E−4	2.3E−5	9.3E+0	<0.005	5.8E−4	1.7E+1
Methylene Chloride	<0.0028	5.3E−5	3.6E−6	1.1E−1	<0.005	9.4E−5	2.0E−1
Xylenes	<0.005	1.5E−4	1.0E−5	5.0E−4	0.011	3.3E−4	1.1E−4
Vinyl chloride	<0.01	2.6E−5	1.8E−6	6.7E+1	<0.01	2.6E−5	7.1E−2
Naphthalene	0.051	2.2E−3	1.5E−4	3.6E−1	<0.01	4.3E−4	7.1E−2
Di-*n*-butyl phthalate	<0.01	1.8E−5	1.2E−6	7.7E−5	<0.01	2.0E−5	7.7E−5
Arsenic	<0.005	7.0E−3	5.0E−4	2.2E+1	<0.10	1.4E−1	4.3E+2
Cadmium	<0.005	2.1E−4	1.4E−5	1.0E−3	0.0012	5.0E−5	2.4E−4
Chromium[e]	<0.04	0.8E+0	5.5E−2	2.4E+2	0.0049	9.8E−2	2.9E+1
Copper	<0.02	9.1E−3	6.1E−4	6.7E−2	<0.002	9.1E−4	6.7E−3
Lead	<0.01	8.3E−4	5.6E−5	2.0E−1	<0.020	1.7E−3	4.0E+0
Mercury[f]	<0.0002	4.0E−4	2.7E−5	1.0E−1	<0.0002	4.0E−4	1.0E−1
Nickel	0.06	3.5E−3	2.4E−4	1.4E+2	0.014	8.2E−4	3.3E+1
Zinc	<0.02	5.0E−4	3.4E−5	5.0E−3	0.44	1.1E−2	1.1E−1
PCBs	NA[g]				NA[g]		
^{3}H	2.2E+3	1.4E−1	1.4E−1	7.9E+0	3.9E+3	2.4E−1	1.4E+1
^{60}Co	0.22	9.6E−4	9.6E−4	5.4E-2	1.4	6.1E−3	3.4E−1
^{90}Sr	86	6.6E+0	6.6E+0	3.7E+2	NA		
^{99}Tc	<100	6.7E−2	6.7E−2	3.8E+0	<100	6.7E−2	3.8E+0
^{137}Cs	2.6	8.4E−2	8.4E−2	4.7E+0	2.1	6.8E−2	3.8E+0
^{235}U	NAL[h]				NAL[h]		
Total		7.7	7.0	866		0.64	537
Total (w/o chromium and ^{90}Sr)		0.32	0.30	256		0.54	508

[a] Chemical concentrations in mg/L and radionuclide concentrations in Bq/L
[b] Concentration in water sample divided by the GRAS-equivalent based on drinking water. Values for chemicals are from Table 5
[c] Concentration in water sample divided by the GRAS-equivalent based on drinking water and a reference meal. Values for chemicals are from Table 5
[d] Concentration in water sample divided by the right-most value in the relevant row of Table 3 or column 4 of Table 6 (i.e., P.C. to indicate "permissible concentration")
[e] Treated all chromium as hexavalent
[f] Treated all mercury as organic
[g] NA = Analysis not performed
[h] NAL = Analysis not performed because gross activity was low

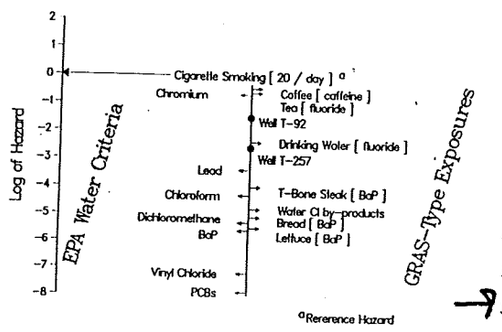

Fig. 2. Hazards from US EPA drinking water statutes compared with consumption of xenobiotics in foods. Also, shown are groundwater samples from a low-level solid waste disposal area

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