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Using the RESRAD computer code to evaluate human health risks from radionuclides and hazardous chemicals

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Abstract

A pathway analysis computer code called RESRAD was developed at Argonne National Laboratory for the U.S. Department of Energy (DOE) for the evaluation of sites contaminated with residual radioactive materials. The DOE and its contractors have used RESRAD to calculate radiation doses and cleanup criteria. Recently, the RESRAD code has been improved so that it can calculate the excess cancer incidence risk from radiation exposure by using the slope factors recommended by the U.S. Environmental Protection Agency. This new feature is discussed in detail in this paper. The method for predicting health risks caused by hazardous chemicals is similar to that for predicting risks caused by radionuclides. The feasibility of applying RESRAD to chemical risk assessment is examined in this paper. The results show that after modification, RESRAD can be used for risk assessment of some classes of hazardous chemicals, for example, metals. Expansion of the RESRAD database to include chemical compounds and the addition of applicable exposure pathways (e.g., inhalation of volatile vapors) will increase RESRAD's capability to handle chemical risk assessments.

1. Introduction

The RESRAD computer code was developed at Argonne National Laboratory for the U.S. Department of Energy (DOE) for the evaluation of sites contaminated with residual radioactive materials [1, 2]. The RESRAD code can be used to calculate potential radiation doses to an on-site resident or worker. It can also be used to derive soil cleanup criteria if the site is to be decontaminated. Soil cleanup criteria are derived on the basis of DOE's dose requirement and analysis to keep the potential dose to a level that is as low as reasonably achievable (ALARA) below the applicable limit. The DOE primary dose limit, that is, the effective dose equivalent from external radiation plus

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the committed effective dose equivalent from internal radiation, to a member of the critical population is 100 mrem/y (1 mSv/y) from all sources and pathways [2]. Therefore, cleanups are expected to achieve control that limits doses to a fraction of 100 mrem/y. However, according to U.S. Environmental Protection Agency (EPA) guidance [3], the evaluation of human health risk should be developed on the basis of age-averaged lifetime excess cancer incidence risk per unit intake of radionuclide (or per unit of external exposure from the radionuclide). This paper discusses the enhancement of the RESRAD code to predict the excess cancer incidence risk based on EPA's guidance.

Radiological surveys have shown that many radioactively contaminated sites are also contaminated by toxic chemicals. To perform a parallel risk analysis of coexistent toxic chemicals, software that uses methodologies consistent with those used in RESRAD for radiological contaminant assessment is desirable and is the basis for enhancing RESRAD. The environmental transport process of nonvolatile chemical contaminants is similar to that of radiological contaminants except that chemical contaminants do not undergo radioactive decay and do not cause external exposure from penetrating radiation. Chemical contaminants are, however, subject to chemical and biological degradation, which may reduce their hazard potencies. This paper examines and demonstrates the applicability of RESRAD for risk assessment of nonvolatile chemicals and identifies additional pathways and databases that are necessary for RESRAD to perform a complete chemical risk assessment of both nonvolatile and volatile hazardous chemicals.

2. Health risk prediction for radionuclides

2.1 Calculation of intake rates

The calculation of the total effective dose equivalent and the soil cleanup guideline for residual radioactive materials in RESRAD is based on a pathway analysis method known as the concentration factor method [4–7]. In this method, the total effective dose to a member of a critical population is expressed as the product of the soil concentration and the pathway sum, the latter being the sum of the products of the "pathway factors." The pathway factors describe the relationships among the environmental compartments through which radionuclides can be transported or radiation transmitted. Pathway factors are primarily steady-state concentration ratios of adjoining compartments, they are sometimes conversion factors for converting a radionuclide concentration to a radiation level, or they are use and occupancy factors that affect exposure. Each term in the sum corresponds to a pathway of connected compartments. A pathway factor can be modified without affecting the other pathways or pathway factors. Because of this structural feature of pathway factors, RESRAD can easily be modified or tailored to use alternative models for any given situation so that additional pathways can be incorporated.

In RESRAD, the following equation is used to calculate the effective dose equivalent of a radionuclide:

$$(\text{Dose})_{j,p}(t) = DCF_{j,p}(t) \times ETF_{j,p}(t) \times SF_{ij}(t) \times S_i(0) \quad (1)$$

where $(\text{Dose})_{j,p}(t)$ (mrem/y) is the effective dose equivalent of radionuclide j from exposure pathway p at time t(y) corresponding to the existence of radionuclide i at $t=0$; $(\text{DCF})_{j,p}$ (mrem/pCi) is the dose conversion factor; $\text{ETF}_{j,p}(t)$ (g/y) is the environmental transport factor (for ingestion and inhalation pathways it is defined as the ratio between the annual intake rate, in pCi/y, of radionuclide j and the soil concentration, in pCi/g, of radionuclide j at time t); and $SF_{ij}(t)$ is the source factor, which is greater than 0 when $j=i$, or when radionuclide j is a decay product of radionuclide i. The source factor is defined as a ratio of the soil concentration of radionuclide j at time t to the soil concentration of radionuclide i at $t=0$. Two factors affect the soil concentrations of radionuclides: the ingrowth and decay process and the leaching process caused by percolating water. The soil concentration of radionuclide i at time 0 is $S_i(0)$. For radionuclide i, which has a nonzero initial concentration at time 0, the total effective dose is the sum of the dose from its decay products j and itself, that is,

$$(\text{Dose})_{i,p}(t) = \sum_{j=1}^{N} (\text{Dose})_{j,p} \quad (2)$$

where N is the total number of radionuclides in the decay chain of radionuclide i, including radionuclide i.

The calculations of $ETF_{j,p}(t)$ and $SF_{ij}(t)$ in eq. (1) are performed independently in RESRAD. Therefore, their values can be used in the calculation of the intake quantities for radionuclides. The total intake quantity of radionuclide j from pathway p is obtained by the following equation:

$$(\text{Intake})_{j,p}(t) = \sum_{i=1}^{M} ETF_{j,p}(t) \times SF_{ij}(t) \times S_i(0) \quad (3)$$

The summation is performed over index i rather than index j, and M is the number of initially existent radionuclides. The calculated total intake quantity of a radionuclide does not include contributions from its decay products. However, the intake rate of a daughter radionuclide includes the contribution of ingrowth from all its initially existent parent radionuclides. Intake rates can be calculated for both the inhalation and ingestion pathways but not for the external exposure pathway.

2.2 Calculation of excess cancer incidence risk

The excess cancer incidence risk is predicted by using slope factors [3]. The slope factors for radionuclides are characterized as the best estimates of the age-averaged lifetime total excess cancer incidence risk per unit intake or exposure, which is not expressed as a function of body weight. On the basis of

EPA guidance for human health risk assessment [3], the total excess cancer incidence risk for radionuclide j and pathway p can be expressed as

$$(\text{Cancer risk})_{j,p}(t) = (\text{Intake})_{j,p}(t) \times Sf_{j,p} \times ED$$

$$= \sum_{i=1}^{M} ETF_{j,p} \times SF_{ij}(t) \times S_i(0) \times Sf_{j,p} \times ED \tag{4}$$

where $Sf_{j,p}$ is the slope factor for radionuclide j and pathway p, and ED (y) is the exposure duration. For the external radiation pathway, Sf is expressed as (risk/y)/(pCi/g); for the inhalation and ingestion pathways, Sf is expressed as risk/pCi. The slope factor for external radiation is developed on the basis of the unit volume concentration, 1 pCi/g, of a contaminated site with an infinitely large area and depth [8]. Realistically, however, the size and depth of a contaminated site are finite, and the site may be irregular in shape rather than cylindrical. The site may also be covered with clean soil material, which provides additional shielding. Therefore, a correction must be made for the difference between real circumstances and the ideal. For the external radiation pathway, the environmental transport factor, $ETF_{j,p}(t)$, is calculated as the product of the factors used to accommodate such a difference. These factors include the cover and depth, shape, area, and occupancy factors [1].

Equation (4) can be used to calculate the excess cancer incidence risk for an individual radionuclide and pathway. The risk calculated by using this equation for a radionuclide, however, does not include the contribution from decay products generated during transport in the environment. To include contributions from decay products, the total cancer risk can be calculated for radionuclide i and pathway p as follows:

$$(\text{Total cancer risk})_{i,p}(t) = \sum_{j=1}^{N} ETF_{j,p}(t) \times SF_{ij}(t) \times S_i(0) \times Sf_{j,p} \times ED \tag{5}$$

The total cancer risk for radionuclide i from all pathways is therefore

$$(\text{Total cancer risk})_i(t) = \sum_{p=1}^{P} (\text{Total cancer risk})_{i,p}(t)$$

$$= \sum_{p=1}^{P} \sum_{j=1}^{N} ETF_{j,p}(t) \times SF_{ij}(t) \times S_i(0) \times Sf_{j,p} \times ED \tag{6}$$

where P is the total number of pathways.

The grand total cancer risk from all radionuclides and pathways can then be calculated as

$$(\text{Grand total cancer risk})(t) = \sum_{i=1}^{M} (\text{Total cancer risk})_i(t)$$

$$= \sum_{i=1}^{M} \sum_{j=1}^{N} \sum_{p=1}^{P} ETF_{j,p}(t) \times SF_{ij}(t) \times S_i(0) \times Sf_{j,p} \times ED \tag{7}$$

Equation (6) can be used to derive risk-based cleanup criteria for radionuclide i.

3. Using RESRAD for chemical risk assessment

3.1 Pathway consideration

The RESRAD code considers nine environmental transport pathways: external exposure, inhalation of dust, ingestion of plants, ingestion of meat, ingestion of milk, ingestion of aquatic foods, ingestion of water, ingestion of soil, and inhalation of radon. Of these pathways, the inhalation of contaminated dust particles and the ingestion of plant, meat, milk, fish, water, and soil are also applicable to chemical risk assessment. Intake rates for the inhalation or ingestion pathway can be obtained from RESRAD output. External radiation exposure and inhalation of radon are unique to radionuclides; results from these pathways are irrelevant to chemical risk assessment.

Although RESRAD considers the dust inhalation and food and soil ingestion pathways it does not consider other pathways characteristic of hazardous chemicals. These pathways include absorption through dermal contact while taking showers or swimming and inhalation of volatile vapors from chemicals in the soil or in water drawn from a nearby pond or well. These two pathways may be important for some hazardous chemicals and should be included in chemical risk assessment.

3.2 Selection of chemicals

RESRAD's current database consists of 52 radionuclides in their elemental forms. To perform risk assessment for chemical compounds as well as for radionuclides, the database needs to be expanded. To demonstrate RESRAD's applicability to nonvolatile chemical risk assessment, elements that are known or suspected to cause adverse health effects or cancers in the human body were selected [8]. These materials are manganese, nickel, and antimony.

Manganese was found to cause respiratory symptoms and psychomotor disturbances in workers when contaminated dust was inhaled. The reference concentration that the EPA has established for manganese for inhalation is 4.0×10^{-4} mg/m^3, which converts to a reference dose of 1.1×10^{-4} mg/kg·d [9]. For ingestion, the reference dose that the EPA has established on the basis of the NRC's [10] "adequate and safe" standard of 2 to 5 mg/d for adults, is 1.0×10^{-1} mg/kg·d [9]. The reference dose is defined as an estimate of the daily exposure level for the human population, including sensitive subpopulations, that is unlikely to have appreciable risk of deleterious effects during a lifetime [3]. Nickel was found to reduce body and organ weights in experiments with rats fed nickel sulfate constantly for two years. The EPA oral reference dose for nickel is 2.0×10^{-2} mg/kg·d [9]. Nickel is also regulated as a known human carcinogen on the basis of evidence from occupational exposure to nickel refinery dust. The inhalation slope factor is 0.84 $(\text{mg/kg} \cdot \text{d})^{-1}$ [9]. Experiments

in which antimony potassium tartrate was put in the drinking water of rats have shown that antimony can decrease the life span and alter blood chemistry. The reference dose for oral ingestion of antimony is 4.0×10^{-4} mg/kg·d [9].

3.3 Calculation of intake rates

Slope factors are the best estimates of age-averaged lifetime excess cancer incidence risks for radionuclides. For chemicals, however, reference doses or slope factors are based on daily exposure level and unit body weight. Therefore, when predicting the excess cancer incidence risk or the noncancer hazard quotient/index, that is, the sum of the hazard quotients, for chemicals, the average (or lifetime average) daily intake rate divided by the average body weight must be used.

When considering the toxicity of hazardous materials rather than the radiation intensity of radionuclides, the ingrowth and decay process is not applicable. Instead, chemical and biological degradation may take place. Because manganese, nickel, and antimony are stable in soil, the influence of degradation on the soil concentration was not considered. In RESRAD, users can modify the decay half-life of elements. Setting the decay half-life to a large number suppresses the influence from ingrowth and decay.

The average daily intake rate of chemicals can be obtained by using the output intake value from RESRAD and multiplying it by $1/(70 \times 365)$. The number 70 is used on the basis of the assumption that the average (male) body weight is 70 kg; 365 is used to convert the annual intake rate to the daily intake rate. If the soil concentration, $S_i(0)$, is expressed in mg/g for chemicals, then the unit of the intake rate will be mg/kg·d. For carcinogens, the lifetime average daily intake rate can be derived by multiplying the average daily intake rate by $ED/70$; ED is the exposure duration, that is, the time in years a person will live on-site, and 70 years is the average life span [11].

3.4 Calculation of health risk

The excess cancer incidence risks of carcinogenic chemicals can be calculated by using RESRAD output values. The user can first choose to modify the inhalation or ingestion slope factors, then multiply the excess cancer risk in the output file by $1/(70 \times 365)$ and $1/70$; the first 70 accounts for average life span in years and the second 70 accounts for average body weight in kg. The noncancer hazard quotient of hazardous chemicals is calculated by dividing the average daily intake rate in terms of mg/kg·d by the reference dose. This calculation is different from that for the excess cancer risk, which is the product of the lifetime average daily intake rate and the slope factor. However, if the value of the inverse of the reference dose is input into the slope factor input field in RESRAD, the excess cancer risk value in the output file can be used and multiplied by $1/(70 \times 365)$ and $1/ED$ to obtain the hazard quotient for individual chemicals and pathways directly; this multiplication is necessary because RESRAD's calculation is for the annual intake rate rather than for the

daily intake rate. Hazard indexes, that is, the sum of the hazard quotients across chemical species and/or pathways, can then be calculated with the obtained hazard quotients.

4. Examples and results

4.1 Radionuclides

A hypothetical site with an area of 10,000 m^2 is contaminated with strontium-90 and cesium-137. The thickness of the contaminated zone is 1 m with a homogeneous concentration of 1 pCi/g for both radionuclides. No cover material exists above the contaminated zone, and the groundwater table is 5 m below the ground surface. A pond is located at the edge of the contaminated site, and the pump intake depth below the groundwater table is 10 m for a well located at the downgradient edge of the site.

It is assumed that a farm family lives on the contaminated site. The family raises vegetables and livestock for its needs and consumes fish caught from a nearby pond. Of the plant food consumed by the farm family, it is assumed that 50% is grown on-site. The adjacent pond provides 50% of the family's aquatic food, and an on-site well provides 100% of its drinking water and irrigation water. It is further assumed that family residents spend 50% of their time indoors on-site, 25% outdoors on-site, and 25% off-site. The exposure duration for the farm family is assumed to be 30 years. The indoor dust level is assumed to be 40% of the outdoor dust level [12], and it is assumed that the building reduces external radiation by 30%. All pathways except the radon pathway are considered because radon is not a decay product of strontium-90 or cesium-137. In addition to the parameters mentioned previously, RESRAD default values were used for the other parameters in this example (see Table 1).

Figure 1 shows the total dose summed over all pathways for cesium-137. Figure 2 shows the change of dose with time from the water-independent pathways for strontium-90. The effective dose equivalent from the water-independent pathways decreases with time because of radioactive decay and the loss of radionuclides due to the leaching process in the contaminated zone. The dose contribution from the water-dependent pathways is zero before the breakthrough time of groundwater contamination and then gradually increases to a maximum value at the rise time; after that it diminishes to zero. The overall effect of the water-independent and water-dependent pathways on the total dose can be seen in Fig. 1 for cesium-137. The water-independent pathways dominate the total dose throughout the considered time frame because the breakthrough time of the groundwater contamination is 8,000 years, with a soil–water distribution coefficient of 1,000 cm^3/g, and the half-life of cesium-137 is only 30.17 years. Therefore, when cesium-137 reaches the groundwater table, its activity has already decayed to a negligible level. According to RESRAD calculational results, the breakthrough time of groundwater contamination for strontium-90 is 241 years. This time is also much longer than the

TABLE 1

Input parameters for the resident farmer scenario

Parameter	Unit	Value
Area of contaminated zone	m^2	10,000
Thickness of contaminated zone	m	1[a]
		2[b]
Length parallel to aquifer flow	m	100
Cover depth	m	0
Density of contaminated zone	g/cm^3	1.6
Contaminated zone erosion rate	m/y	0
Contaminated zone total porosity	–	0.4
Contaminated zone effective porosity	–	0.2
Contaminated zone hydraulic conductivity	m/y	10
Contaminated zone b parameter	–	5.3
Evapotranspiration coefficient	–	0.6
Precipitation	m/y	1.0
Irrigation	m/y	0.2
Irrigation mode	–	overhead
Runoff coefficient	–	0.2
Watershed area for nearby stream or pond	m^2	1,000,000
Density of saturated zone	g/cm^3	1.6
Saturated zone total porosity	–	0.4
Saturated zone effective porosity	–	0.2
Saturated zone hydraulic conductivity	m/y	100
Saturated zone hydraulic gradient	–	0.02
Saturated zone b parameter	–	5.3
Water table drop rate	m/y	0
Well pump intake depth (below water table)	m	10
Model: Nondispersion (ND) or Mass-Balance (MB)	–	ND
Number of unsaturated zone strata	–	1
Unsaturated zone thickness	m	4.0
Unsaturated zone soil density	g/cm^3	1.6
Unsaturated zone total porosity	–	0.4
Unsaturated zone effective porosity	–	0.2
Unsaturated zone soil specific b parameter	–	5.3
Unsaturated zone, hydraulic conductivity	m/y	100
Distribution coefficients for all zones	cm^3/g	
Cesium-137		1,000
Strontium-90		30
Manganese		200
Nickel		1,000
Antimony		140
Inhalation rate	m^3/y	8,400
Mass loading for inhalation	g/m^3	0.0002
Dilution length for airborne dust, inhalation	m	3
Shielding factor, inhalation	–	0.4
Shielding factor, external gamma	–	0.7
Fraction of time spent indoors	–	0.5
Fraction of time spent outdoors (on-site)	–	0.25
Shape factor, external gamma	–	1.0

TABLE 1. Continued

Parameter	Unit	Value
Fruit, vegetable, and grain consumption	kg/y	160
Leafy vegetable consumption	kg/y	14
Milk consumption	L/y	92
Meat and poultry consumption	kg/y	63
Fish consumption	kg/y	5.4
Other seafood consumption	kg/y	0.9
Soil ingestion rate	g/y	36.5
Drinking water intake	L/y	510
Fraction of drinking water from site	–	1.0
Fraction of aquatic food from site	–	0.5
Livestock fodder intake for meat	kg/d	68
Livestock fodder intake for milk	kg/d	55
Livestock water intake for meat	L/d	50
Livestock water intake for milk	L/d	160
Mass loading for foliar deposition	g/m^3	0.0001
Depth of soil mixing layer	m	0.15
Depth of roots	m	0.9
Drinking water fraction from groundwater	–	1.0
Livestock water fraction from groundwater	–	1.0
Irrigation fraction from groundwater	–	1.0

[a] For radionuclides.
[b] For chemicals.

decay half-life of 28.8 years for strontium-90. The maximum total dose will occur at time 0 for both strontium-90 and cesium-137. The minimum soil cleanup guideline is 32 pCi/g for cesium-137 and 39 pCi/g for strontium-90.

Table 2 lists the slope factors for cesium-137 and strontium-90. Intake quantities of these two radionuclides at time 0 are listed in Table 3. Comparing the intake rates from individual pathways, it is found that the intake rate from the plant ingestion pathway for strontium-90 is about 100 times greater than the intake rate for cesium-137. The plant–soil transfer factor for root uptake for strontium-90 is 2.0×10^{-1}, which is 100 times the value for cesium-137, that is, 2×10^{-3}, and explains the difference in the intake rates. The milk transfer factor from ingestion of fodder or water is 1.5×10^{-3} d/L for strontium and 5×10^{-3} d/L for cesium. The greater value for cesium reduces the difference in the magnitude of the milk intake rate for cesium and for strontium, causing the milk intake rate of strontium to be about 30 times greater than that of cesium. RESRAD default values for the transfer factors (e.g., vegetable/soil transfer factors for root uptake) were used in the calculation. These numbers can be modified, however, by the RESRAD user.

Excess cancer incidence risks for individual radionuclides and pathways at time 0 are listed in Table 4. The external radiation pathway is the most important one for cesium-137 and accounts for almost 100% of its total risk.

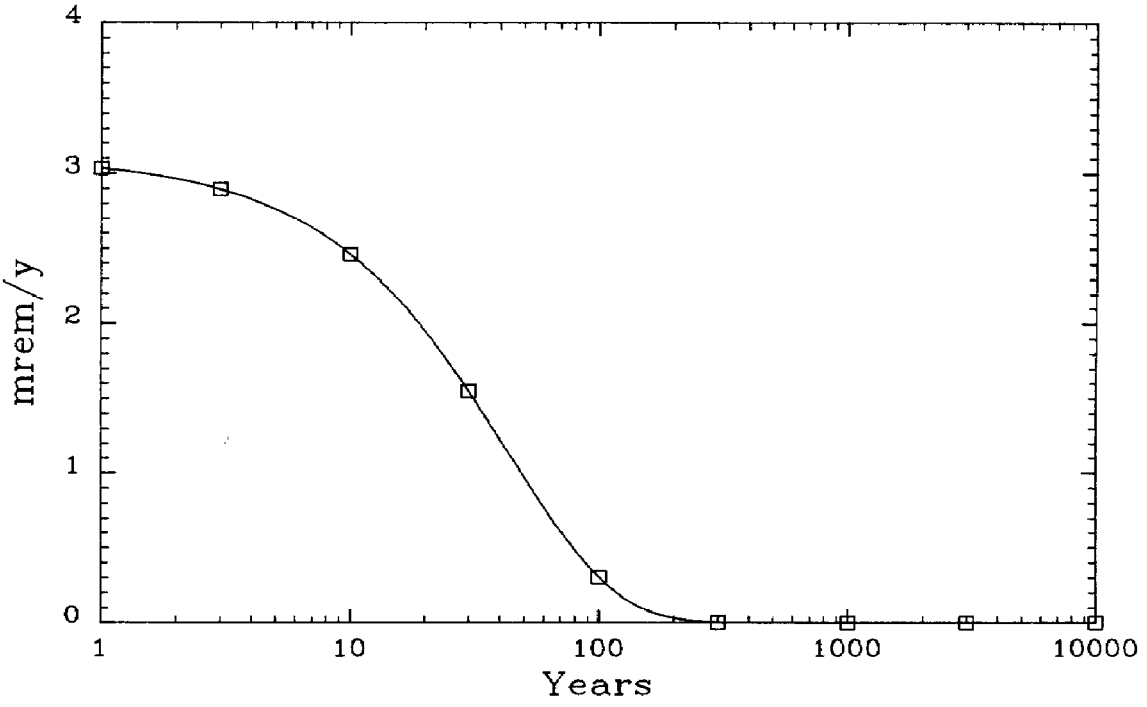

Fig. 1. Total dose for cesium-137.

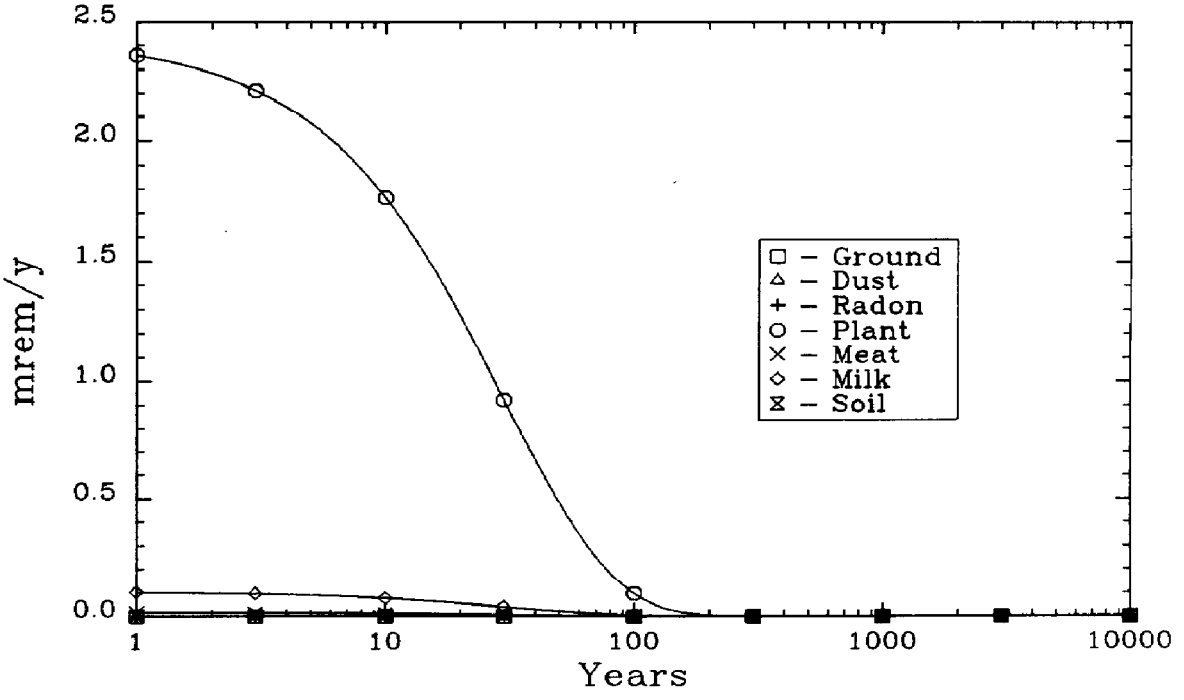

Fig. 2. Effective dose from individual water-independent pathways for strontium-90.

TABLE 2

Slope factors[a] for radionuclides

Radionuclides	Inhalation (risk/pCi)	Ingestion (risk/pCi)	External radiation (risk/y)/(pCi/g)
Cesium-137 + D[b]	1.9×10^{-11}	2.8×10^{-11}	2.0×10^{-6}
Strontium-90 + D[b]	6.2×10^{-11}	3.6×10^{-11}	0.00

[a] Slope factors taken from HEAST [8].
[b] Radionuclides noted by +D have slope factors including contributions from their associated decay daughters that have half-lives less than six months and are assumed to be in secular equilibrium with their parents.

TABLE 3

Intake rates for individual radionuclides and pathways (pCi/y, $t=0$)

Radionuclide	Water-independent pathway				
	Dust	Plant	Meat	Milk	Soil
Cesium-137	7.3×10^{-1}	1.8×10^{2}	1.3×10^{2}	2.6×10^{1}	1.6×10^{1}
Strontium-90	7.3×10^{-1}	1.7×10^{4}	1.3×10^{2}	7.6×10^{2}	1.6×10^{1}
Radionuclide	Water-dependent pathway				
	Water	Fish	Plant	Meat	Milk
Cesium-137	0.0	0.0	0.0	0.0	0.0
Strontium -90	0.0	0.0	0.0	0.0	0.0

TABLE 4

Excess cancer risks for individual radionuclides and pathways ($t=0$)

Radionuclide	Water-independent pathway					
	Ground	Dust	Plant	Meat	Milk	Soil
Cesium-137 + D	3.6×10^{-5}	4.2×10^{-10}	1.5×10^{-7}	1.1×10^{-7}	2.2×10^{-8}	1.4×10^{-8}
Strontium-90 + D	0.0	1.4×10^{-9}	1.9×10^{-5}	1.4×10^{-7}	8.2×10^{-7}	1.8×10^{-8}
Total	3.6×10^{-5}	1.8×10^{-9}	1.9×10^{-5}	2.5×10^{-7}	8.4×10^{-7}	3.2×10^{-8}
Radionuclide	Water-dependent pathway					
	Water	Fish	Plant	Meat	Milk	All pathways[a]
Cesium-137 + D	0.0	0.0	0.0	0.0	0.0	3.6×10^{-5}
Strontium-90 + D	0.0	0.0	0.0	0.0	0.0	2.0×10^{-5}
Total	0.0	0.0	0.0	0.0	0.0	5.6×10^{-5}

[a] Sum of water-independent and water-dependent pathways.

The cancer risk for strontium-90 is 0 from the external radiation pathway, however, because the EPA-recommended slope factor [8] is 0 (risk/y)/(pCi/g). The greatest cancer risk for strontium-90 is caused by the ingestion of plant food, followed by the ingestion of milk. The maximum excess cancer risk is associated with exposure to radionuclides at time 0, with a total risk of 3.6×10^{-5} for cesium-137 and 2.0×10^{-5} for strontium-90. If a total excess cancer incidence risk of 1.0×10^{-4} is assumed for deriving soil cleanup criteria, then the guideline values for cesium-137 and strontium-90 would be 2.7 pCi/g and 5.1 pCi/g, respectively.

4.2 Chemicals

The same hypothetical site as that for radionuclides is adopted for chemicals and is assumed to be contaminated by manganese, nickel and antimony, with a soil concentration of 1 mg/g, respectively. The contaminated zone is 2 m deep, and the groundwater table is 6 m below the ground surface. The farm family scenario is again assumed, and all pathways except the external radiation and radon pathways are selected. RESRAD default values were used for the other parameters in this example (see Table 1).

RESRAD calculational results show that the breakthrough times of groundwater contamination were 1, 600 and 1,100 years (rounded to the nearest 10 years) for manganese, nickel, and antimony, respectively. Both the oral and inhalation intake rates for these chemicals have maximum values at time 0. The intake rate from the plant ingestion pathway contributed the most to the total oral intake rate throughout the 1,000-year time frame. Details of the calculated intake rates are listed in Tables 5 and 6.

TABLE 5

Reference dose and average oral daily intake rate (mg/kg·d) for soil concentration of 1 mg/g[a]

Time (y)	Metal		
	Manganese	Nickel	Antimony
RfD[b]	1.0×10^{-1}	2.0×10^{-2}	4.0×10^{-4}
0	1.2×10^{-1}	7.3×10^{-2}	4.1×10^{-2}
1	1.2×10^{-1}	7.3×10^{-2}	4.1×10^{-2}
10	1.1×10^{-1}	7.3×10^{-2}	4.1×10^{-2}
100	1.1×10^{-1}	7.2×10^{-2}	3.8×10^{-2}
250	9.9×10^{-2}	7.1×10^{-2}	3.3×10^{-2}
500	8.4×10^{-2}	6.8×10^{-2}	2.6×10^{-2}
750	7.2×10^{-2}	6.6×10^{-2}	2.1×10^{-2}
1,000	6.2×10^{-2}	6.4×10^{-2}	1.7×10^{-2}

[a] Note: All values are reported to two significant figures.
[b] RfD = reference dose; data for the RfDs for manganese, nickel, and antimony are from Ref. [9].

TABLE 6

Reference dose and average inhalation daily intake rate (mg/kg·d) for soil concentration of 1 mg/g

Time (y)	Metal		
	Manganese	Nickel	Antimony
RfD[a]	1.1×10^{-4}[b]	ND[c]	ND[c]
0	2.9×10^{-5}	2.9×10^{-5}	2.9×10^{-5}
1	2.9×10^{-5}	2.9×10^{-5}	2.9×10^{-5}
10	2.9×10^{-5}	2.9×10^{-5}	2.9×10^{-5}
100	2.7×10^{-5}	2.8×10^{-5}	2.6×10^{-5}
250	2.5×10^{-5}	2.8×10^{-5}	2.3×10^{-5}
500	2.1×10^{-5}	2.7×10^{-5}	1.8×10^{-5}
750	1.8×10^{-5}	2.6×10^{-5}	1.5×10^{-5}
1,000	1.5×10^{-5}	2.5×10^{-5}	1.2×10^{-5}

[a] RfD = reference dose; the RfD for manganese was obtained by multiplying the reference concentration (mg/m^3) listed in Ref. [9] by an inhalation rate of 20 m^3/d and dividing the product by a body weight of 70 kg.
[b] Data for the RfD for manganese are from Ref. [9].
[c] ND = no data available for inhalation RfD.

TABLE 7

Hazard quotients[a] for individual chemicals and pathways ($t = 0$)

Metal	Water-independent pathways					
	Dust	Plant	Meat	Milk	Soil	All pathways[b]
Manganese	2.6×10^{-1}	1.0	1.3×10^{-1}	3.0×10^{-3}	6.4×10^{-3}	1.4
Nickel	–[c]	3.2	8.0×10^{-2}	3.2×10^{-1}	3.2×10^{-2}	3.7
Antimony	–[c]	9.4×10^{1}	6.9	2.0	1.6	100

[a] Note: Hazard quotients for water-dependent pathways are 0 at $t = 0$, because the breakthrough times are greater than 0. All values are reported to two significant figures.
[b] Sum of water-independent pathways.
[c] A hyphen indicates that no hazard quotient was calculated due to lack of reference dose information.

The hazard quotients were calculated from RESRAD output results following the procedure mentioned in the discussion of the calculation of health risks. Table 7 gives the hazard quotients for individual chemicals and pathways at time 0. The ingestion of contaminated plant foods is the greatest threat to human health and is consistent with the prediction from the intake rate calculation. In addition to the ingestion of plant foods, inhalation of dust and

ingestion of meat are also critical pathways for manganese. The ingestion of milk results in the second greatest hazard quotient for nickel among the oral exposure pathways. Among the different ingestion routes for antimony, the ingestion of meat has a hazard quotient approximately three to four times that for the ingestion of milk and soil. The maximum hazard indexes calculated are 1.4 (inhalation and oral ingestion) for manganese, 3.7 (oral ingestion) for nickel, and 100 (oral ingestion) for antimony. On the basis of the hazard indexes, when the soil concentrations are 1 mg/g for each chemical at the hypothetical site, the exposed resident farmer might develop adverse health effects. The maximum excess cancer risk caused by inhalation of dust contaminated with nickel, as calculated by RESRAD, is 3.1×10^{-6}.

5. Conclusion

The enhancement of the RESRAD code for the calculation of intake rates and the prediction of excess cancer incidence risks for radionuclides was demonstrated for cesium-137 and strontium-90. The excess cancer incidence risks were tabulated for individual radionuclides and pathways as well as for initially existent radionuclides with contributions from decay daughters. This enhancement increases RESRAD's capability to analyze the health risks from exposure to radioactive materials.

This study also shows that RESRAD can be used in the risk analysis of hazardous chemicals. Expanding the RESRAD database to include chemical compounds would expand RESRAD's capability to handle risk assessments of hazardous chemicals. The addition of pathways applicable to chemical contaminants, such as the absorption of chemicals through dermal contact and the inhalation of volatile chemical vapors, would give users a tool to assess chemical risks from every potential exposure route. Including chemical or biological degradation models specific to hazardous chemicals would improve prediction accuracy; however, it may also increase prediction uncertainty.

The modified RESRAD code provides a consistent methodology for risk analysis of hazardous chemicals comparable to that for radioactive materials in a site contaminated with mixed waste. The results obtained provide a clearer picture of the total cost/risk of cleaning up a chemically/radiologically contaminated site.

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