A Multimedia, Multiple-Exposure Pathway Methodology for Deriving Risk-Based Standards for Tetrachloroethylene (PCE) in Soil

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This paper was prepared for publication as a chapter in the Proceedings of the Fifth Annual Conference: Hydrocarbon Contaminated Soils September 24–27, 1990, Amherst, MA

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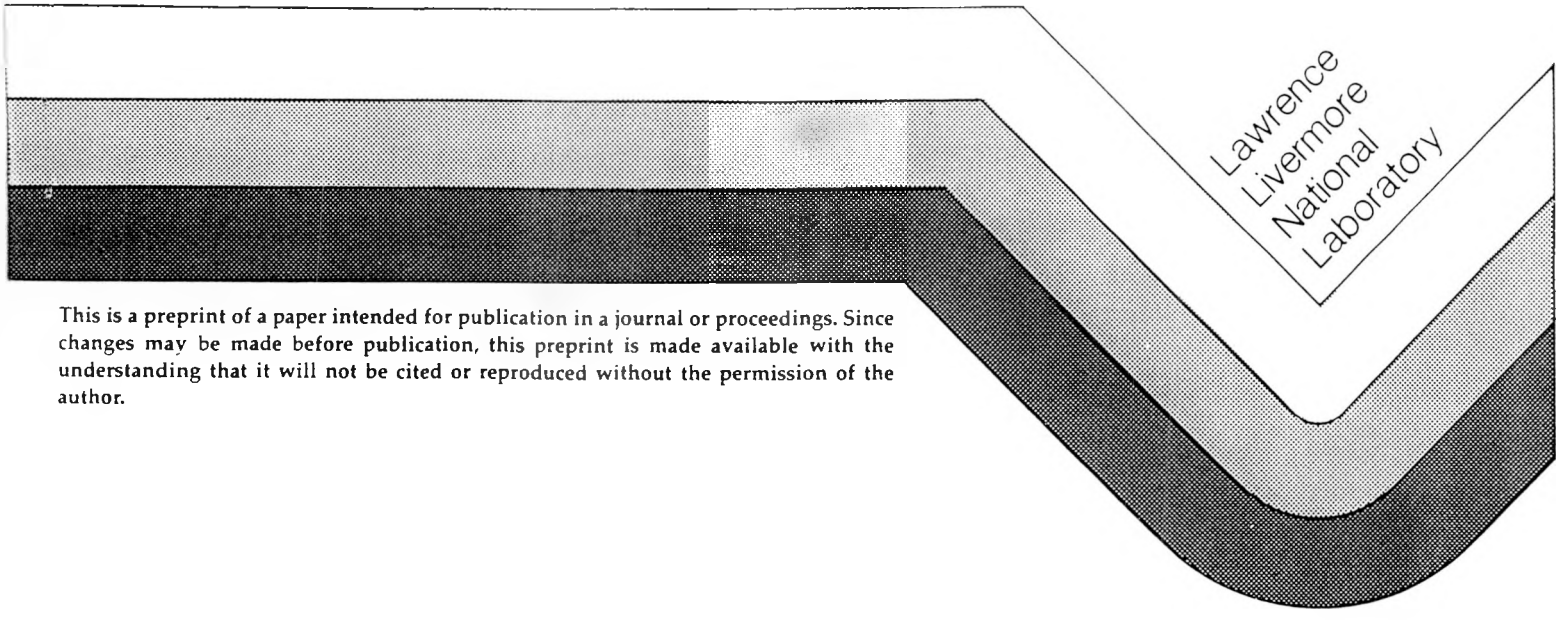

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ABSTRACT

For contaminants of concern at hazardous-waste sites, the State of California develops exposure criteria referred to as Applied Action Levels (AALs). An AAL is the concentration of a contaminant in a source medium that, when exceeded, is expected to pose an unacceptable risk to human health. Typically, AALs are calculated separately for individual environmental media (e.g., air, water, or soil) and only for a limited number of routes of exposure. This approach fails to recognize the multimedia character of many soil contaminants, and by excluding significant exposure routes, could underestimate risk. Using tetrachloroethylene (perchloroethylene, PCE) in soil as an example, we describe an alternative methodology for deriving risk-based AALs from estimates of intake that account for multimedia, multiple-pathway exposure.

INTRODUCTION

As a result of its widespread use as an industrial solvent, PCE is a common contaminant of soil and groundwater in many urban areas of California. The chemical PCE is a chlorinated organic compound that volatilizes into the atmosphere from surface soil or surface water. Once in the atmosphere, PCE is subject to relatively rapid chemical or photolytic degradation. The U.S. Environmental Protection Agency (EPA) (U.S. EPA, 1985) has estimated PCE's residence time in the atmosphere to be ≤1 y. In groundwater, PCE is degraded through hydrolysis and biotransformation; these processes occur relatively slowly, however, and estimates of the half-life of PCE in groundwater range from months to several years (Mallon, 1989).

The U.S. EPA (1985) classified PCE as a Possible Human Carcinogen (Group C) based on the positive results of a National Cancer Institute (NCI, 1977) bioassay. In that study, PCE was administered by gavage to mice. Data from a later study (National Toxicology Program (NTP), 1986) indicated that PCE was carcinogenic to both mice and rats when administered by inhalation over a two-year exposure period. The State of California also includes PCE on a list of "Chemicals Known to the State to Cause Cancer or Reproductive Toxicity" (California Department of Health Services (CDHS), 1990). To calculate a range of risk-based AALs for PCE that incorporate multimedia, multiple-pathway estimates of exposure, we first use the environmental fate and transport model, GEOTOX, to estimate the equilibrium

concentration of PCE in five different environmental media. The environmental concentrations of PCE predicted by GEOTOX are based on a steady-state source of PCE in soil in a 100 km^2 landscape that is typical of a region in California. Second, pathway-exposure factors (PEFs) are developed that incorporate information on chemical properties, human physiology, and human behavior patterns, and are used to translate the PCE concentration in each environmental medium into estimates of human exposure. Third, total exposure for a specific exposure pathway is computed by summing the products of the PEFs and the corresponding medium-specific concentrations of PCE for that pathway. Fourth, quantitative estimates of the carcinogenic potency of PCE for different exposure pathways are developed. Fifth, the pathway-specific cancer-potency factors (CPFs) are multiplied by the exposure attributable to each exposure pathway to yield an estimate of pathway-specific carcinogenic risk; these pathway-specific risks are then summed to obtain an estimate of total risk for multimedia equilibrium concentrations of PCE. Finally, the ratio of acceptable risk (such as 10^{-6}) to the total calculated risk is determined, and this ratio is used to scale the GEOTOX-derived soil concentration of PCE to a soil concentration that corresponds to the specified level of acceptable risk. The resulting soil concentration of PCE is the AAL.

MULTIMEDIA PARTITIONING OF PCE USING GEOTOX

The presence of a contaminant such as PCE in soil results in contamination of other environmental media including air, surface water, and groundwater. Environmental concentrations of PCE in air, water, and soil that result from soil contamination can be estimated using the multimedia transport and transformation model GEOTOX (McKone, 1981; McKone et al., 1983; McKone and Kastenberg, 1986; McKone and Layton, 1986; McKone et al., 1987; Layton et al., 1986). GEOTOX is a computer program designed to calculate time-varying chemical concentrations in air, soil, groundwater, and surface water. Table 1 summarizes the processes by which contaminants are exchanged and lost among compartments of the GEOTOX model.

The GEOTOX model uses two sets of input data, one providing the properties of the environment or landscape receiving the contaminants and the other describing the properties of the contaminants. The information we need to construct a landscape-data set include meteorological data, such as average annual

Table 1. Summary of the processes by which contaminants are exchanged and lost among the seven compartments of the GEOTOX model.

Compartment	Gains	Losses
Air (gas phase of the troposphere)	Diffusion from soil and surface water.	Diffusion to soil; diffusion to surface water; washout by rainfall; and convection losses.
Air Particles (atmospheric dust)	Resuspension of deposited soil particles.	Deposition from atmosphere to soil; deposition from air to surface water; and convective losses.
Upper-Soil Layer (surface-soil layer)	Diffusion from air; washout by rainfall; deposition of air particles; and irrigation from groundwater.	Diffusion to air; infiltration (leaching); resuspension of deposited soil particles; soil-solution runoff; and erosion.
Lower-Soil Layer (vadose zone)	Infiltration from upper-soil layer.	Infiltration to groundwater zone.
Groundwater Zone	Infiltration from lower-soil.	Discharge to surface-water irrigation.
Surface Water	Diffusion from air; washout by rainfall; deposition of atmospheric particles; soil-solution runoff; erosion (mineral runoff); diffusion from sediment; and sediment resuspension.	Sediment deposition; diffusion to sediment; and surface-water outflow.
Sediment Layer	Diffusion from surface water; and sediment deposition (from surface water).	Diffusion to surface water; and sediment resuspension.

wind speed, deposition velocities, air temperature, and depth of the mixing layer; hydrological data, such as annual rainfall, runoff, soil infiltration, groundwater recharge, and surface-water depth and sediment loads; and soil properties, such as bulk density, porosity, water content, erosion rates, and root-zone depth. The GEOTOX model also requires that we know or estimate solid/liquid phase partition coefficients (between the soil and soil water, between groundwater and aquifer material, and between surface water and sediments); the air/liquid partition coefficients; and the diffusion coefficients of substances in air and water.

We used GEOTOX with a steady-state input of PCE to the soil layer to estimate concentrations in the adjacent air and water compartments of a 100-km^2 landscape. This 100-km^2 landscape represents a typical region of California; specific landscape parameters used in GEOTOX were based on the national land-classification system developed for the U.S. Department of the Interior and the U.S. Department of Agriculture (Bailey, 1980). Table 2 lists the environmental concentrations of PCE predicted by GEOTOX for multiple environmental media, scaled to a soil concentration of PCE of 1 ppm (mg/kg).

PATHWAY-EXPOSURE FACTORS

We have developed an approach that links the concentration of a chemical in each environmental medium to human exposure through the use of pathway-exposure factors (PEFs). Each PEF incorporates information on human

Table 2. Concentrations of PCE in a 100-km^2 landscape, assuming a soil-based steady-state source of contamination. Concentrations are predicted using the GEOTOX model, and are scaled to a soil concentration of PCE of 1.0 mg/kg.

Media (abbreviation; concentration units)	PCE concentration
Soil (C_s; mg/kg)	1.0
Air gases (C_a; mg/m^3)	1.4
Air particles (C_p; mg/m^3)	~ 0.0[a]
Potable water (C_w; mg/L)	0.21
Surface water (C_r; mg/L)	0.27

[a] There is some partitioning of PCE to particules in air; however, the majority of PCE in the air compartment is in the gaseous phase, and the very small amount present on particles is insignificant.

physiology, human-behavior patterns, and environmental transport into a term that translates a unit concentration in a specified environmental medium (e.g., air in mg/m^3, soil in mg/kg, or water in mg/L) into daily exposure in mg/kg-d for a specified route of exposure (inhalation, ingestion, or dermal contact). For example, the PEFs for inhalation of air gases (F_{ah}), and air particles (F_{ph}), which translate the atmospheric concentration of gaseous- and solid-phase PCE, respectively, into estimates of exposure, can be calculated from the following expressions:

$$F_{ph} = 16\left[\frac{12}{16} \times 0.75 + \frac{4}{16}\right]\left(\frac{BR}{BW}\right)_a + 8 \times 0.75\left(\frac{BR}{BW}\right)_r, \text{ and} \quad (1)$$

$$F_{ah} = 16\left(\frac{BR}{BW}\right)_a + 8\left(\frac{BR}{BW}\right)_r, \quad (2)$$

where 16 is the number of hours per day that an adult or child is assumed to be active; 8 is the number of hours per day that an adult or child is assumed to be resting; 12/16 is the representative fraction of active hours that an adult or child spends indoors; 4/16 is the fraction of active hours that an adult or child spends outdoors; 0.75 is the level of suspended particulate matter indoors relative to that outdoors as reported by Hawley (1985); $(BR/BW)_a$ is the active breathing rate per unit body weight, 0.021 m^3/kg-h; and $(BR/BW)_r$ is the resting breathing rate per unit body weight, 0.0070 m^3/kg-h.

We have developed PEFs for nine pathways that are linked to PCE concentrations in five environmental media. Table 3 contains the matrix of PEFs that link exposure to PCE concentrations in the different environmental media. The derivation of each of these PEFs is described in detail by McKone and Daniels (1990).

HUMAN EXPOSURE TO PCE

The PEFs listed in Table 3 are used in conjunction with the GEOTOX-derived concentration data (Table 2) to calculate pathway-specific exposure to PCE. The relationship between a PEF and an exposure is given by the following expression:

$$e_{pathway}^{medium} = C_{medium} \times F_{pathway}, \quad (3)$$

Table 3. Matrix of pathway-exposure factors (PEFs) for concentrations of a contaminant in specific environmental media.

	PEFs associated with environmental medium concentration, C_i, where i is the type of medium[a]				
Pathway	Air (gas phase; m^3/kg-d), C_a	Air (particles; m^3/kg-d), C_p	Soil (kg/kg-d), C_s	Drinking water[b] (L/kg-d), C_w	Surface water (L/kg-d), C_r
Inhalation	F_{ah} (0.39)	F_{ph} (0.31)	F_{sh} (9.2×10^{-9})	F_{wh} (0.11)	-- --
Ingestion					
Water	--	--	--	F_{ww} (3.4×10^{-2})	--
Fruits and vegetables	F_{av} (1.6×10^{-4})	F_{pv} (14.0)	F_{sv} (1.1×10^{-3})	--	--
Grains	F_{ag} (2.5×10^{-4})	F_{pg} (22.0)	F_{sg} (8.0×10^{-4})	--	--
Meat	F_{at} (5.7×10^{-6})	F_{pt} (2.8×10^{-2})	F_{st} (5.4×10^{-7})	F_{wt} (1.9×10^{-6})	--
Milk	F_{ak} (4.0×10^{-6})	F_{pk} (2.9×10^{-2})	F_{sk} (5.2×10^{-7})	F_{wk} (1.2×10^{-6})	--
Fish	--	--	--	--	F_{rf} (2.1×10^{-2})
Soil	--	--	F_{ss} (1.5×10^{-6})	--	--
Dermal contact	--	--	F_{sd} (2.6×10^{-6})	F_{wd} (3.8×10^{-2})	--

[a] Subscripts refer to the source media (a = air gases, p = air particles, s = soil, w = drinking water, and r = surface water) and pathways (h = inhalation, w = water ingestion, v = vegetables, g = grain, t = meat, k = milk, f = fish, s = soil ingestion, and d = dermal contact).

[b] Drinking-water concentrations are obtained by arithmetically averaging the concentrations in surface and groundwater so as to reflect the mix in a local-water supply. For the AAL calculation we assume half of the drinking water comes from groundwater and the other half from surface water.

where $e_{pathway}^{medium}$ is the lifetime-equivalent human exposure for an individual due to the presence of PCE at a concentration denoted C_{medium} in a specific environmental medium and $F_{pathway}$ is the PEF for a specific pathway.

Table 4 presents a matrix of estimated exposures to PCE. Each exposure value given in this table was calculated according to a formula that is similar in form to that shown in Eq. 3. The total multimedia-PCE exposure for a particular pathway is determined by summing the exposure values for that pathway across all media. The total estimated exposure for each particular exposure pathway (e.g., inhalation (E_h), ingestion (E_g), and dermal contact (E_d)) is given in the far right column of Table 4.

CARCINOGENIC POTENCY OF PCE

Data from rodent bioassays in which PCE was administered by gavage (National Cancer Institute (NCI), 1977) or inhalation (National Toxicology Program (NTP), 1986) have provided evidence that PCE is carcinogenic to animals. According to reviews by the U.S. EPA (1985) and Bogen et al. (1987), PCE is metabolized in mammals to one or more reactive metabolites, and extensive evidence exists that it is a product of PCE metabolism, rather than the parent compound itself, that is responsible for PCE's carcinogenicity in laboratory animals. Estimates of the carcinogenic potency of PCE as a function of metabolized dose have been calculated from the data of the NCI (1977) and NTP (1986) by the U.S. EPA (1985, 1986) and more recently, by Bogen et al. (1987). The term carcinogenic "potency" is used here to refer to the quantitative expression of increased tumorigenic response per unit dose rate at very low doses. The cancer potency estimates of Bogen et al. (1987) were adjusted to include data on animal body weight, metabolite elimination, and other factors not incorporated into the U.S. EPA calculations (U.S. EPA, 1985, 1986). We therefore relied on potency estimates derived by Bogen et al. (1987) and not on those derived by the U.S. EPA (1985, 1986).

Bogen et al. (1987) used the term $q_1^*(M)$ to specify the 95% upper-confidence limit (UCL) estimate of potency that relates low levels of metabolized dose, M, to predicted upper-bound increased tumor risk, R*, by the linear approximation $R^* \sim q_1^*(M) \times (M)$. A summary of the values of $q_1^*(M)$ for PCE calculated by Bogen et. al. (1987) for each of the NCI (1977) and NTP (1986) tumor-incidence sets is presented in Table 5.

To estimate the carcinogenic potency of PCE in terms of human-applied dose,

Table 4. Summary of estimated exposures[a] to PCE (mg/kg-d). Values were calculated using the environmental concentrations of PCE predicted by GEOTOX for a 100 km^2 area in a typical California region (Table 2) and the PEFs listed in Table 3.

	Exposure					
Pathway	Air (gases)[b]	Air (particles)[c]	Soil[d]	Potable water[e]	Surface water[f]	Totals
Inhalation	5.5×10^{-1}	0.00	9.0×10^{-9}	2.3×10^{-2}	--	5.7×10^{-1} (E_h)[g]
Ingestion						
Water	--	--	--	7.1×10^{-3}	--	
Vegetables	2.2×10^{-4}	0.00	1.1×10^{-3}	--	--	
Grains	3.5×10^{-4}	0.00	8.0×10^{-4}	--	--	
Milk	5.6×10^{-6}	0.00	5.2×10^{-7}	2.5×10^{-7}	--	
Meat	8.0×10^{-6}	0.00	5.4×10^{-7}	4.0×10^{-7}	--	
Fish	--	--	--	--	5.7×10^{-3}	
Soil	--	--	1.5×10^{-6}	--	--	
Ingestion total	5.9×10^{-4}	0.00	1.9×10^{-3}	7.1×10^{3}[h]	5.7×10^{-3}[h]	1.5×10^{-2} (E_g)[i]
Dermal uptake	--	--	2.6×10^{-6}	8.0×10^{-3}	--	8.0×10^{-3} (E_d)[j]

[a] Exposure = PEF (from Table 3) × Concentration (from Table 2).

[b] Concentration of PCE in the gaseous phase of air, C_a= 1.4 mg/m^3.

[c] Concentration of PCE in the particulate phase of air, C_p = 0.00 mg/m^3.

[d] Concentration of PCE in soil, C_s= 1.0 mg/kg.

[e] Concentration of PCE in potable water, C_w = 0.21 mg/L, which is the average concentration of PCE in both surface and groundwater.

[f] Concentration of PCE in surface water, C_r = 0.27 mg/L.

[g] E_h = Total multimedia exposure to PCE from the inhalation exposure pathway.

[h] Total ingestion exposure due to PCE in potable and surface water = 1.3×10^{-2} mg/kg-d.

[i] E_g = Total multimedia exposure to PCE from the ingestion exposure pathway.

[j] E_d = Total multimedia exposure to PCE from dermal contact.

Table 5. Predicted carcinogenic potency of PCE (from Bogen et al., 1987).

Study species strain	Tumor type[a]	Sex	Weight (kg)	Experimental applied dose or concentration	LTWA metabolized dose[b], M (mg/kg-d)	95%-UCL potency[c] of metabolized dose[d], $q_1^*(M)$, based on surface area[e] $(mg/kg\text{-}d)^{-1}$
NCI, 1977		m	0.030	536[f]	30.55	0.42
Mice	HC					
B6C3F1		f	0.025	386[f]	25.17	0.31
NTP, 1986		m	0.037	100[g]	43.29	0.19
Mice	HC					
B6C3F1		f	0.032	100[g]	46.74	0.095
NTP, 1986		m	0.037	100[g]	43.29	0.30
Mice	HAC					
B6C3F1		f	0.032	100[g]	46.74	0.13
NTP, 1986		m	0.44	200[g]	11.83	0.35
Rats	MLK					
F344/N		f	0.32	200[g]	14.03	0.24

[a] HC = hepatocellular carcinoma, HAC = hepatocellular adenoma, MLK = mononuclear-cell leukemia.

[b] Lifetime, time-weighted-average (LTWA) metabolized dose, M. See Bogen et al. (1987) for derivation of M as a function of applied dose or concentration.

[c] "Potency" means the low-dose, dose-response slope expressed by the value of the linear-multistage coefficient, q_1, such that at very low doses, risk = $q_1 \times$ dose, according to a multistage risk-prediction model (U.S. EPA, 1980; Anderson et al., 1983). UCL potency = one-tailed 95% upper-confidence limit of q_1.

[d] Human-equivalent lifetime, time-weighted-average metabolized dose, M, in mg/kg-d.

[e] Surface Area interspecies dose-extrapolation method:

$$M_{human} = M_{animal}^{[(animal\ weight)/70\ kg]^{1/3}}.$$

[f] Average daily dose administered by gavage (mg/kg-d) for 5 d/wk over 78 wk of a 90-wk bioassay.

[g] Average exposure by inhalation (ppmv) administered for 6 h/d, 5d/wk during a 2-yr bioassay.

A, which is necessary for calculation of an acceptable-exposure limit because exposure is derived in terms of applied and not metabolized dose, we multiplied the term $q_1^*(M)$ by f, the fraction of the applied dose that is metabolized (i.e., $q_1^*(A) = q_1^*(M) \times f$). Bogen et al. (1987) estimated $q_1^*(A)$ initally based on the relationship that f = 0.04 at a very small applied dose of PCE. Subsequently, Bogen and McKone (1988) used the data on human metabolism of PCE of Ikeda et al. (1972) and Ohtsuki et al. (1983) to demonstrate that between 5% and 34% of a respired or dermally acquired dose of PCE, f^*_{mr}, is metabolized. Bogen (1988) developed an equation to calculate the fraction metabolized, f^*_{mo}, of very low doses of an orally applied volatile organic compound such as PCE. The solution of that equation (see Bogen, 1988), based on the Bogen and McKone (1988) analysis of the human metabolism data of Ikeda et al. (1972) and Ohtsuki et al. (1983), indicates that approximately 7% (Ohtsuki et al., 1983) to 46% (Ikeda et al., 1972) of an ingested dose of PCE is metabolized by humans.

The revised estimates of the extent of human metabolism of PCE indicate that the value of f used to derive $q_1^*(A)$ varies from 0.05 to 0.46, depending on the source of the data and on the route of exposure. We calculated the arithmetic means of f^*_{mo} and f^*_{mr} using the respective ranges of values for these parameters developed by Bogen and McKone (1988) from the data of Ikeda et al. (1972) and Ohtsuki et al. (1983). Table 6 presents estimates of $q_1^*(A)$ based on the lowest and highest values of $q_1^*(M)$ listed in Table 5 and based on the mean values of f^*_{mo} (26%) and f^*_{mr} (20%).

DERIVATION OF RISK-BASED SOIL CONCENTRATIONS OF PCE AS AALs FOR SOIL

To derive a range of risk-based AALs for PCE in soil, we use pathway-specific estimates of exposure, pathway-specific estimates of cancer potency, and three levels of excess individual-lifetime cancer risk–1 per 10,000 (i.e., 10^{-4}), 1 per 100,000 (i.e., 10^{-5}), and 1 per 1,000,000 (i.e., 10^{-6}), which represent the range of alternatives considered by regulatory agencies as acceptable levels of total risk (see U.S. EPA, 1990 and CDHS, 1989).

For a 100-km^2 landscape in a typical California region, the total calculated risk associated with 1 ppm of PCE in soil [R_{TC}(1 ppm)] is determined by first multiplying each multimedia pathway-specific intake [i.e., inhalation, E_h; ingestion, E_g; and dermal-contact, E_d (see Table 4)] by its corresponding maximum or minimum

Table 6. Range of carcinogenic-potency estimates for PCE for ingestion, respiration, or dermal exposure.

$q_1^*(M)^b$ (mg M/kg-d)$^{-1}$	$q_1^*(A)^a$ (mg A/kg-d)$^{-1}$	
	Ingestion[c]	Inhalation or dermal uptake[d]
0.095	0.025	0.019
0.42	0.11	0.084

[a] 95%-UCL potency of human applied dose, A, based on surface-area, interspecies dose-extrapolation method. Note that $q_1^*(A) = q_1^*(M) \times f$, where $f = f^*_{mo}$ or f^*_{mr}.

[b] Range of 95%-UCL potency of human metabolized dose, M, based on surface-area, interspecies dose-extrapolation method. Values from Bogen et al. (1987).

[c] Calculated using the arithmetic mean of $f^*_{mo} = 0.26$.

[d] Calculated using the arithmetic mean of $f^*_{mr} = 0.20$. As noted in the text, it is assumed that the fraction of applied dose that is metabolized is the same for inhalation and dermal contact (see Bogen, 1988).

pathway-specific cancer-potency factor (CPF) (see Table 6). The products of each pathway-specific CPF and multimedia, pathway-specific intake are then summed to obtain the value of $R_{TC}(1\ ppm)$. Accordingly,

$$R_{TC}(1\ ppm) = (E_h \bullet CPF_h) + (E_g \bullet CPF_g) + (E_d \bullet CPF_d), \quad (4)$$

where

$R_{TC}(1\ ppm)$ = the total *calculated* risk associated with 1 ppm of PCE in soil;
E_h = inhalation intake related to multimedia exposure resulting from a soil-based PCE concentration of 1 ppm (mg/kg-d);
E_g = ingestion intake related to multimedia exposure resulting from a soil-based PCE concentration of 1 ppm (mg/kg-d);
E_d = dermal contact related to multimedia exposure resulting from a soil-based PCE concentration of 1 ppm (mg/kg-d);

CPF_h = maximum or minimum cancer-potency factor for the inhalation pathway (1/[mg/kg-d]);

CPF_g = maximum or minimum cancer-potency factor for the ingestion pathway (1/[mg/kg-d]); and

CPF_d = maximum or minimum cancer-potency factor for the dermal-contact pathway (1/[mg/kg-d]).

We calculate the concentration of PCE in soil corresponding to a specified level of total acceptable risk, R_{TA} (e.g., 10^{-6}), for consideration as an AAL by multiplying the steady-state concentration of PCE in soil, C_s(1 ppm), by the ratio of R_{TA}(AAL) to R_{TC}(1 ppm):

$$C_s(AAL_{ar}) = C_s(1\ ppm) \times [R_{TA}(AAL)/R_{TC}(1\ ppm)], \quad (5)$$

where

$C_s(AAL_{ar})$ = concentration of PCE in soil (C_s; mg/kg) that represents an applied action level based on an acceptable level of risk (AAL_{ar});

C_s(1 ppm) = GEOTOX- generated concentration of PCE in soil (C_s) normalized to 1 ppm (i.e., mg/kg) for a California landscape of 100 km^2;

R_{TA}(AAL) = total *acceptable* risk for a soil-based PCE concentration; and

R_{TC}(1 ppm) = total *calculated* risk for multimedia, multiple-pathway exposure to a 1 ppm soil-based concentration of PCE for a California landscape of 100 km^2, and either maximum or minimum bounding estimates for pathway-specific CPFs.

The $C_s(AAL_{ar})$ for low- and high-potency estimates and R_{TA}(AAL) values ranging from 10^{-4} to 10^{-6} are presented in Table 7. Note that for any specific level of acceptable total risk, there is an inverse relationship between a CPF and the calculated $C_s(AAL_{ar})$ value. Consequently, the lowest $C_s(AAL_{ar})$ representing a

Table 7. Risk-based concentrations of PCE in soil for consideration as applied action levels (AALs)[a].

Level of risk[a]	Soil concentration (mg/kg) Low potency	High potency
10^{-4}	9×10^{-3}	2×10^{-3}
10^{-5}	9×10^{-4}	2×10^{-4}
10^{-6}	9×10^{-5}	2×10^{-5}

[a] Risk is the incremental probability of an individual developing cancer over a 70-y lifetime as a consequence of multiple-pathway exposure (i.e., from ingestion, inhalation, and dermal contact) to soil-based concentrations of PCE in multiple enviromental media (i.e., soil, air, and water).

[b] The terms "low" and "high" refer to the range of cancer-potency estimates for PCE listed in Table 6.

potential AAL will be associated with the highest pathway-specific CPFs. Alternatively, the highest calculated $C_s(AAL_{ar})$ value will be associated with the lowest pathway-specific CPFs. These relationships result because the CPF is the upper-bound estimate of the probability of a carcinogenic response per unit intake of a chemical over a lifetime, expressed in units of $(mg/kg\text{-}d)^{-1}$.

CONCLUSION

The calculated $C_s(AAL_{ar})$ values appearing in Table 7 constitute a range of risk-based concentrations of PCE in soil for consideration as the soil-based AAL. It is the responsibility of the risk manager to select the AAL from among these values or to use the methodology described here to develop other more or less conservative values for consideration. However, the final decision will be judgmental and should be based on criteria such as likelihood of public exposure, size and overall susceptibility of the population at risk, and principal exposure pathway, as well as the uncertainties in the calculated AALs.

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