

Estimating risk to human health

Trichloroethylene in drinking water is used as the example

C. Richard Cothorn
William A. Coniglio
William L. Marcus

Environmental Protection Agency
Office of Drinking Water
Washington, D. C. 20460

The decision to regulate the maximum level of an environmental contaminant allowable in air, water, or food is predicated on the ability of the substance to alter life processes and on the degree of exposure that creates a significant risk of adverse effects to a sensitive population. For more than four centuries, scientists have tried to understand the adverse effects of exposure to environmental contaminants.

In general, any contaminant can cause damage if exposure to it creates high concentrations within a cell, and if accumulated cellular damage can result in disease or death. Environmental regulations are developed to reduce exposure to a level that precludes such health effects.

Drinking-water regulations are being developed for many contaminants, some of which are known to cause cancer in laboratory animals. One group, the volatile organic compounds (VOCs), is currently being assessed for the development of national drinking-water standards. Reference 1 contains an explanation of the risk assessment studies being used to evaluate VOCs.



SL 034464

Levels of human exposure to VOCs found in drinking water and the level of cancer risk imposed by their presence have been estimated as part of the process of developing regulations. Estimates include the health benefits to be derived from different levels of national regulation. They couple the estimate of carcinogenic potency, derived from an extrapolation of a dose-response curve developed from high-level exposure of experimental animals, with estimates of the number of persons exposed to contaminated drinking water. The estimates include the level of daily intake—oral, respiratory, and dermal—resulting from the presence of these chemicals in water (Figure 1).

Estimates of the number of cancers resulting from currently unregulated conditions and of those cancers that will be prevented or allowed by setting standards at different levels can be included in the regulatory process. Current knowledge about the health effects of contaminants has its limits: Much is known about some aspects; little or

nothing is known about others. But there is uncertainty in all areas.

The *population risk estimate* is an assessment of the total expected excess number of cancers in the United States during a lifetime from a given contaminant. This is an estimate and not a statement of perfect knowledge. Therefore, a level of uncertainty or a range of values will be included to describe the estimate. If the frequency distribution of the contributing factors controlling exposure and toxicity were known, the propagation of errors could be determined by use of standard methods. Frequency distributions often are not known, however, and a range of values often must be used to approximate the situation.

This article describes some of the data on which regulatory judgments are made. It also describes the assumptions introduced to complete the assessments, the results, and their uncertainty. We take as our example the VOC trichloroethylene (TCE).

Recent research has shown that TCE

can be transformed in water to vinyl chloride, a known human carcinogen (2). The cancer risk associated with the presence of vinyl chloride in drinking water also has been assessed (1).

TCE in drinking water

There are approximately 60,000 public water supplies in the United States, serving some 220 million persons. Three major federally sponsored monitoring surveys have been conducted nationwide to obtain information on the concentration of selected VOCs appearing as pollutants in potable water provided to the nation's cities and towns.

By using the survey data in conjunction with the multinomial approach for projecting national occurrence, it was estimated that 97% of groundwater systems of all sizes contain either no TCE or concentrations $<0.5 \mu\text{g/L}$ (Table 1). It is not possible to estimate how many systems in this category are contaminated with TCE.

Of the estimated 1632 systems expected to have levels $>0.5 \mu\text{g/L}$, 421 (0.9% of the total number of groundwater systems) are expected to have concentrations $>5.0 \mu\text{g/L}$, and 133 systems (0.3%) nationally have concentrations $>50 \mu\text{g/L}$. Although a greater percentage of the large systems is expected to be contaminated, because of the large number of small groundwater systems it is likely that in absolute numbers more small systems are contaminated.

In a similar analysis of surface water supplies, it was estimated that 96% either have no TCE present or that levels are $<0.5 \mu\text{g/L}$. It is estimated that 498 surface water systems have levels $\geq 0.5 \mu\text{g/L}$ (4.5% of total surface water systems); 9 ($<0.1\%$) are expected

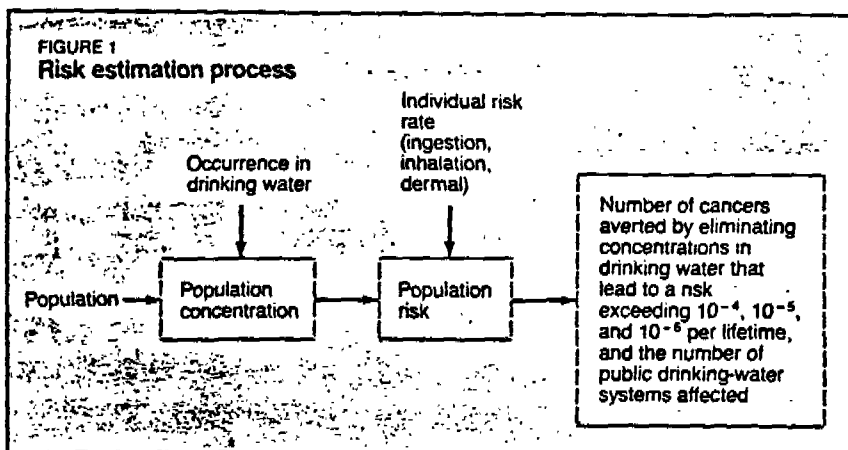


TABLE 1
Trichloroethylene contamination of groundwater systems

System size (population served)	No. of systems in U.S.	Estimated number of systems by concentration (µg/L)													
		<0.5*	0.5-5	>5-10	>10-20	>20-30	>30-40	>40-50	>50-60	>60-70	>70-80	>80-90	>90-100	>100	
25-100	19,125	18,506	465	26	52	0	26	0	26	0	0	0	0	26	
101-500	15,674	15,166	381	21	42	0	21	0	21	0	0	0	0	21	
501-1,000	4,877	4,719	118	7	13	0	7	0	7	0	0	0	0	7	
1,001-2,500	4,400	4,257	107	6	12	0	6	0	6	0	0	0	0	6	
2,501-3,300	891	862	22	1	2	0	1	0	1	0	0	0	0	1	
3,301-5,000	1,065	1,031	26	1	3	0	1	0	1	0	0	0	0	1	
5,501-10,000	1,168	1,130	28	2	3	0	2	0	2	0	0	0	0	2	
10,001-25,000	835	775	34	11	4	0	0	8	0	0	4	0	0	0	
25,001-50,000	290	269	12	4	1	0	0	3	0	0	1	0	0	0	
50,001-75,000	64	59	3	1	0	0	0	1	0	0	0	0	0	0	
75,001-100,000	14	13	1	0	0	0	0	0	0	0	0	0	0	0	
>100,000	55	41	14	0	0	0	0	0	0	0	0	0	0	0	
Total	48,458	46,828	1,211	80	132	0	64	12	64	0	5	0	0	64	

*Calculated as the difference between the number of systems expected to have $\geq 0.5 \mu\text{g/L}$ TCE and the total number of systems in that size category. This group includes those having no contamination and those with levels $<0.5 \mu\text{g/L}$.

to have levels $>5 \mu\text{g/L}$; none is estimated to have TCE $>40 \mu\text{g/L}$.

Using the combined data from surface water and groundwater supplies, it was estimated that nearly 190 million persons (88.3% of the population served by public drinking-water systems) receive uncontaminated water or water contaminated with TCE at levels $<0.5 \mu\text{g/L}$. Of the 25 million persons (11.7%) receiving water that contains TCE at levels $\geq 0.5 \mu\text{g/L}$, an estimated 1.8 million (0.9%) are exposed to levels $>5 \mu\text{g/L}$. About 212,000 persons (0.1%) are probably exposed to levels $>50 \mu\text{g/L}$, and 42,000 ($<0.1\%$) are estimated to be exposed to levels $>100 \mu\text{g/L}$.

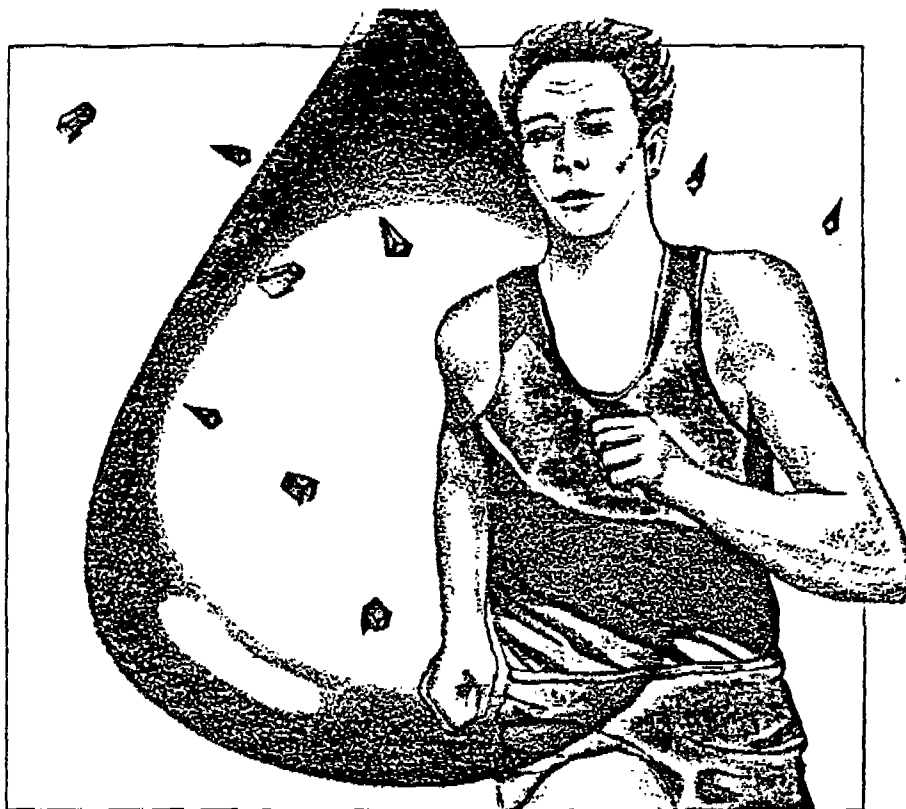
Of the approximately 23 million persons exposed to levels ranging from $0.5 \mu\text{g/L}$ to $5 \mu\text{g/L}$, 76% obtain water from surface water supplies. However, of the 1.8 million persons exposed to levels $>5 \mu\text{g/L}$, 62% use groundwater sources. All exposure to TCE in drinking water in levels $>40 \mu\text{g/L}$ is projected to be from groundwater sources.

Several assumptions have been made during the assessment of TCE contamination of drinking water, among them that sites selected in surveys are representative of drinking-water supplies across the nation and that grab samples are representative of water quality throughout the water supply system.

Using scientific judgment, the uncertainties in these estimates were analyzed. The uncertainties include those in the measurements themselves and those involved in projecting the existing data to estimate natural occurrence. In general, the overall uncertainty is less than an order of magnitude.

Human exposure to contaminants

The average adult in the United States uses 48–55 gal of water each day (3, 4). This means that a household of two children and two adults uses ap-



proximately 255 gal of water each day (5). Water pollutants pass through the home in water that is used for laundering clothing, washing dishes, removing wastes, bathing, cooking, and drinking.

The level of exposure to any pollutant present in drinking water is the result of many personal daily choices and several factors over which we have very little direct control. Where we live, what we eat and drink, and how old we are all have a profound influence on the magnitude of exposure. Also, the physical and chemical characteristics of the pollutant govern the amount that stays in the water and that which is transported into indoor air.

Table 2 provides an example of the potential differences in exposure of

family members to a VOC resulting from personal choices. This table has been prepared for a theoretical VOC, similar in chemical characteristics to TCE. We assumed that 100% of the compound is absorbed for all oral intake, 50% for respiratory intake, and a very small amount is absorbed in direct proportion to water flux across the skin.

Cancer risks projected for TCE are based on assumed lifetime ingestion of 0.03 L of drinking water for each kilogram of body weight and on the assumption that 100% of the ingested chemical is absorbed. The factor for ingestion was developed using an intake standard of 2 L/70 kg adult weight. Individuals may experience many times this intake rate. Formula-fed infants and young children, for example, have average intake rates that are as much as eight times greater than those of average adults.

Adults in tropical areas may consume twice as much liquid as the average, as may athletically inclined adults when engaged in strenuous physical activity. Persons who are ill also may consume much more water than the average. Social behavior ritualized around the drinking of tea or coffee may lead to increased water consumption, although boiled water may contain smaller amounts of contaminants. A recent Canadian study shows that children under 5 years old, 10% of the children 6–17 years old, and 2% of the adult population consume more than 0.03 L/d of drinking water for every kilogram of

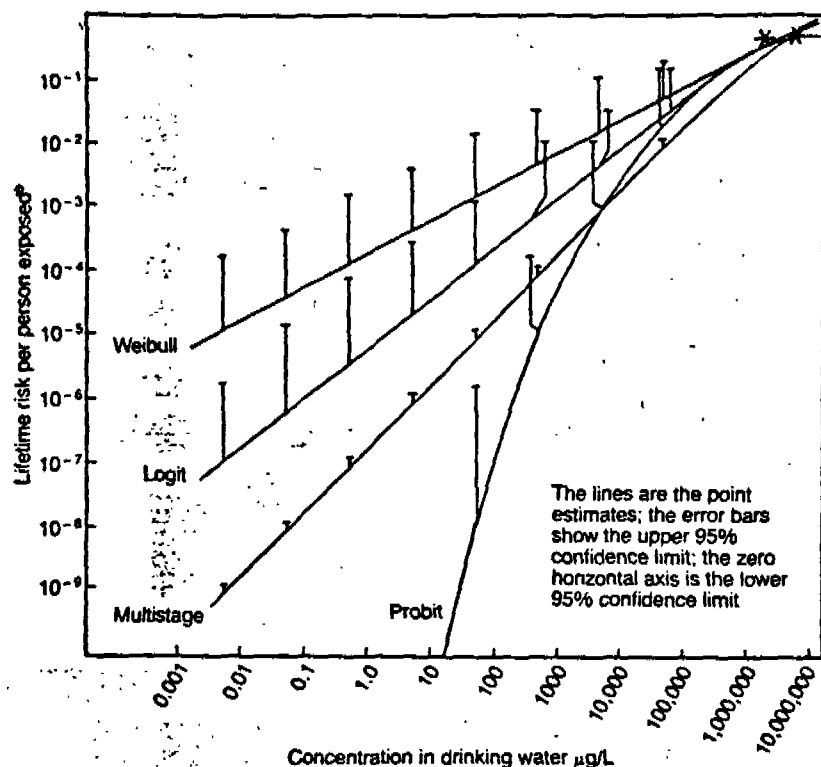
TABLE 2
Comparative model of absorbed dose from a volatile pollutant in drinking water^a

Exposure route	Absorbed dose ($\mu\text{g/d}$)			
	Formula-fed Infant (4 kg)	Pretreen (32 kg)	Adult female (60 kg)	Adult male ^b (70 kg)
Fluid ingestion	80	150	200	200
Inhalation of enriched indoor air	10	10	70	50
Inhalation of enclosed shower air	—	100	300	200
Dermal absorption				
Bathing	.02–.06	—	?	?
Swimming	—	10–300	—	—
Total absorbed dose	20 $\mu\text{g/kg/d}$	10–20 $\mu\text{g/kg/d}$	10 $\mu\text{g/kg/d}$	7 $\mu\text{g/kg/d}$

^a100 $\mu\text{g/L}$.

^bShowering adult male

FIGURE 2
Bioassay data and model extrapolations for exposure
to TCE through ingestion*



*Bioassay data are the stated points in the upper right-hand corner. The lifetime risk per person exposed was calculated assuming that the risk due to inhalation and dermal exposure is equal to that due to ingestion exposure. The error introduced by this simplifying assumption is less than the widths of the lines shown.
*Through ingestion, inhalation, and dermal exposure, assuming that ingestion equals inhalation plus dermal

body weight (6).

Each of the VOCs considered, including TCE, has been shown to transfer from water into air if the water is heated or aerated. Monitoring data indicate that this process is continuous within the home and leads to an immediate enrichment of respirable air at the point of water use and to a diffusion throughout the home. Showers taken within an enclosed bathroom, using

20-30 gal of water, result in the liberation of all or a portion of the VOCs into the air. A person who showers inhales air containing a highly enriched level of the chemical while inside the bathroom. VOCs from other sources as well mix with general home air, resulting in higher levels of the pollutants being released into the indoor air.

Very few data are available on the enrichment of general indoor air with

VOCs. However, research has been conducted on the radon enrichment of indoor air, and it is assumed that the fraction of radon released is about the same as that for VOCs. These data indicate that an enrichment of indoor air (the amount of radon in a liter of air) is about 10^{-4} to the concentration found in a liter of drinking water (7-10). This model assumes an indoor air change rate of one per hour.

For our analysis it has been assumed that respiratory exposure to VOCs in drinking water is roughly equivalent to that from ingestion. To compare the relative exposures from inhalation and ingestion, assume that for both pathways 100% of the VOCs that enter the body reach the bloodstream. Then if water with a concentration of $X \mu\text{g/L}$ is ingested, the daily intake to the bloodstream is $2X \mu\text{g/d}$. For example,

$$X \mu\text{g/L} \times 2 \text{ L/d} = 2X \mu\text{g/d} \quad (1)$$

If water with a concentration of $X \mu\text{g/L}$ transfers all of the VOC to indoor air and it all goes to the bloodstream, then the intake to the bloodstream is again $2X \mu\text{g/d}$. For example,

$$\begin{aligned} & \frac{X \mu\text{g}}{\text{L}} \times \frac{10^{-4} \text{ L (water)}}{\text{L (air)}} \\ & \times \frac{20 \text{ m}^3 \text{ (air)}}{\text{d}} \times \frac{10^6 \text{ cm}^3 \text{ (air)}}{\text{m}^3 \text{ (air)}} \\ & \times \frac{\text{L (air)}}{1000 \text{ cm}^3} = 2X \mu\text{g/d} \quad (2) \end{aligned}$$

Under these conditions exposure from ingestion and inhalation is equal.

Individual risk rates

Estimates of individual risk rates are based on bioassay data derived from animal experiments. These data are converted to a continuous human equivalent exposure for use in risk estimates. The bioassay data selected to estimate carcinogenic potency of VOCs in drinking water are from experiments

TABLE 3
Population risk estimates for current TCE levels in drinking water

Mean drinking-water concentration (µg/L)	Number of persons served	Total lifetime individual risk for the mean concentration*		Lifetime population risk
		Low (probit)	High (Weibull)	
0.25	1.9×10^4	$< 10^{-10}$	2.4×10^{-4}	$< 1-45,600$
2.75	2.3×10^7	$< 10^{-10}$	7.3×10^{-4}	$< 1-16,790$
7.5	4.3×10^5	$< 10^{-10}$	1.3×10^{-3}	$< 1-559$
15	2.1×10^5	$< 10^{-10}$	1.7×10^{-3}	$< 1-367$
35	7.4×10^5	7×10^{-4}	2.3×10^{-3}	$< 1-1,702$
45	2.6×10^5	3×10^{-7}	2.6×10^{-3}	$< 1-676$
55	4.2×10^4	4×10^{-7}	2.8×10^{-3}	$< 1-117$
75	1.3×10^5	6×10^{-7}	3.2×10^{-3}	$< 1-416$
100	4.2×10^4	1.2×10^{-8}	3.7×10^{-3}	$< 1-155$
Total*				$< 1-70,000$

*The total individual risk was determined by assuming that the risk due to inhalation is equal to that due to ingestion.

*Rounded to one significant figure

SL 034467

reviewed and selected by the National Academy of Sciences and the EPA Carcinogen Assessment Group (11, 12). When the carcinogenic data were from inhalation experiments, they were converted to ingestion values.

The bioassay data are fit to four analytical models: logit, multistage, probit, and Weibull. These four models are chosen somewhat arbitrarily and are representative of models currently in use. It is well-known that other models could be used to fit the same data (13-16). There are no biologically based criteria for choosing one model over another. The models are in the form of analytical expressions that can be used to fit the high-dose data mathematically.

The dose-response curves for the models are fit to equivalent human dose-response data converted using the relative surface area of animals and humans. The curves are extrapolated to the lower environmental levels. A log-log plot of the entire region shows the two nonzero data points for TCE (Figure 2). The solid lines are the point estimates, and the error bars show the upper 95% confidence limits. The GLOBAL computer program was used for the multistage model, and the Krewski computer program was used for the logit, probit, and Weibull models.

The curves shown in Figure 2 are not the only models that could be fit to the data. Additionally, there is no assurance that the actual curve is in the range of those shown. It is possible, although highly unlikely, that the actual curve lies outside those shown. For example, the probit model generally decreases extremely quickly relative to dose, whereas the Weibull model decreases very slowly.

Although the dose-response curves projected by each model in Figure 2 start at the same points, they diverge significantly at lower dose levels. At a concentration of 50 $\mu\text{g/L}$ TCE in drinking water, the Weibull model provides a risk estimate approximating 1×10^{-2} , whereas the probit model provides an

estimate of 10^{-10} . These estimates provide a range of uncertainty equivalent to not knowing whether one has enough money to buy a cup of coffee or pay off the national debt.

Population risk calculations

The population at risk of health damage due to TCE in drinking water was estimated. The population risks are determined by a product of the number of people affected and the individual risk rate (Table 3). In each case the concentration is in the average of the range. The lowest concentration ($<0.5 \mu\text{g/L}$) represents the minimum reported concentrations for the national surveys and does not necessarily represent the detection limit of the chemical in drinking water. Although we have used the estimated average of $0.25 \mu\text{g/L}$ for this category, it is possible that all of the persons in this category could be exposed to $0 \mu\text{g/L}$ or that all of them could be exposed to the maximum of $0.5 \mu\text{g/L}$. For each chemical the individual lifetime risks were determined from the model fits.

In each case the curves in Figure 2 giving the lowest and the highest individual risks were used to determine the range of population risks. The uncertainty in choice of model in Figure 2 is by far the largest uncertainty of any involved in the estimates of risk. Thus it alone was used to calculate the range of risk. When calculations yielded less than one person, the number was listed as a whole number rather than as the fractional number of persons.

The risk reduction analysis (Table 4) is a determination of the number of cancer cases averted if the standard is set corresponding to individual lifetime risk rates of 10^{-4} , 10^{-5} , or 10^{-6} . For these individual risk rates, the corresponding range of concentration and the cases averted are listed. The concentration range was determined from the graph shown earlier in Figure 2. The population risk estimates are listed as ranges; the range is indicative of the uncertainty in this estimate.

For TCE, the lower estimate for each

concentration range is less than one. The sum of the upper population risk estimates is 70,000, when rounded off to one significant figure. The largest portion of the population risk is contributed by the lowest concentration category. This category represents calculations below the detection limit and may be an artifact of the mathematical analysis technique used to estimate the population risk. The range is largely a reflection of the uncertainty in choice of the mathematical model selected to estimate individual risk. The contribution to uncertainty from other factors is negligible by comparison.

If a drinking-water standard—a maximum contaminant level—for TCE were established at $7.5 \mu\text{g/L}$, nationwide compliance would result in the avoidance of anywhere from <1 to 4000 cases.

Explanation

The development of regulations covering contaminants in drinking water requires the assembly of data from many different sources. One important piece of information needed is the estimate of the expected number of health effects and how many of these can be prevented by regulation. Risk assessment usually involves using skimpy data concerning the levels of a contaminant in drinking water and using health effects data from animal experiments conducted at exposure levels much higher than those usually found in the environment.

The risk can be estimated using TCE in drinking water as the example, although the range of uncertainty is quite large. If there were no data, the range of estimated health effects would be between none and all the people. Using the available data for TCE in drinking water, the estimated range of health effects lies between <1 and 70,000. If all exposure to levels $>7.5 \mu\text{g/L}$ were eliminated, as few as one or less of these health effects could be prevented, or perhaps as many as 4000. These estimates are the best representation of the existing data for TCE in drinking water. Their value to the regulatory decision-making process must be judged when placed alongside the other information used in that process.

Acknowledgment

We thank Joseph A. Cotruvo for his encouragement and helpful discussions on the work described here.

The thoughts and ideas expressed in this article are those of the authors and are not necessarily those of the EPA.

References

- (1) Cothorn, C. R.; Coniglin, W. A.; Marcus, W. L. "Techniques for the Assessment of Carcinogenic Risk to the U.S. Population Due to Exposure from Selected Volatile Or-

TABLE 4
Risk reduction analysis*

Maximum allowable drinking-water concentration ($\mu\text{g/L}$)	Approximate individual risk rate for maximum concentration	Cumulative cases averted*
100	1×10^{-7} to 1×10^{-2}	<1 -200
45	1×10^{-9} to 1×10^{-2}	<1 -1,000
7.5	$<1 \times 10^{-10}$ to 6×10^{-3}	<1 -4,000
2.75	$<1 \times 10^{-10}$ to 4×10^{-3}	<1 -20,000

*For limiting TCE concentration in drinking water

*Number of cases averted for concentration shown in first column

ganic Compounds from Drinking Water via the Ingestion, Inhalation and Dermal Routes." EPA 570/9-85-001; EPA: Washington, D.C., 1985.

- (2) Vogel, T. M.; McCarty, P. L. *Appl. Environ. Microbiol.* 1985, 49, 1080-83.
- (3) Watson, K. S.; Farrell, R. P.; Anderson, J. S. *J. Water Pollut. Control Fed.* 1967, 30(12), 2039-54.
- (4) Lenawever, F. P., Jr. In "A Study of Flow Reduction and Treatment of Wastewater from Households"; Bailey, J. R., Ed.; General Dynamics report to the Federal Water Quality Administration: Washington, D.C., 1969.
- (5) Bailey, J. R. et al. "A Study of Flow Reduction and Treatment of Waste Water from Households," Water Pollution Control Research Series 11050FKE: General Dynamics report for the Federal Water Quality Administration; Department of the Interior: Cincinnati, Ohio, 1969.
- (6) "Tap Water Consumption in Canada," 82EHD80; Canadian Environmental Health Directorate, Ministry of National Health and Welfare: Ottawa, Ont., 1981.
- (7) "Sources and Effects of Ionizing Radiation," Report to the General Assembly of the United Nations by the United Nations Scientific Committee on the Effects of Atomic Radiation, 1977.
- (8) Hess, C. T.; Weiffenbach, C. V.; Norton, S. A. *Environ. Int.* 1982, 8, 59.
- (9) Kahlos, H.; Asikainen, M. *Health Phys.* 1980, 39, 108.
- (10) Nazaroff, W. W. et al. "Potable Water as a Source of Airborne Radon-222 in U.S. Dwellings: A Review and Assessment,"

LBL 18154; Lawrence Berkeley Laboratory: Berkeley, Calif., 1985.

- (11) *Drinking Water and Health*; National Academy of Sciences: Washington, D.C., 1982.
- (12) Anderson, E. L. et al. *Risk Anal.* 1983, 3, 277-95.
- (13) Van Ryzin, J. J. *Occup. Med.* 1980, 22, 321-26.
- (14) Van Ryzin, J.; Rai, K. In *The Scientific Basis of Toxicity Assessment*; Witschi, H., Ed.; North-Holland Biomedical Press: New York, N.Y., 1980; p. 273.
- (15) Krewski, D.; Van Ryzin, J. In *Statistics and Related Topics*; Csorgo, M. et al., Eds.; North-Holland: New York, N.Y., 1981; p. 145.
- (16) Munro, T. C.; Krewski, D. R. *Food Cosmet. Toxicol.* 1981, 19, 549-60.



C. Richard Cothorn is a physicist with EPA. He received his Ph.D. in physics

from the University of Manitoba. He is involved in setting standards for radionuclides in drinking water and in assessing risks that result from drinking-water contaminants.



William A. Coniglio (l.) is a biologist with EPA. He has an M.S. in economic biology from Rutgers University and received public health training at the University of Alabama. He develops background information on the occurrence of and exposure to drinking-water contaminants.

William L. Marcus (r.) is a toxicologist with EPA. He received his Ph.D. in toxicology and pharmacology from Howard University. He reviews and analyzes toxicological reports and data and advises on toxicological matters.

CIVIL ENGINEERS ENVIRONMENTAL ENGINEERS CHEMICAL ENGINEERS HYDROGEOLOGISTS

Dames & Moore, one of the world's largest firms in engineering and applied earth sciences, is continuing to expand its practice in waste management and water resources engineering. Our Suburban Washington, D. C. office has several immediate vacancies:

- Civil Engineers—1 to 5 years experience in all aspects of civil engineering with a concentration in water resources and hazardous waste management;
- Chemical and Environmental Engineers—2 to 15 years experience in hazardous waste remedial investigations and alternative technologies studies, RCRA Part B permitting, and advanced wastewater treatment systems development;
- Hydrogeologists—2 to 10 years experience in design and review of groundwater sampling and monitoring programs, groundwater modeling and analysis of contaminant transport.

All positions require excellent communication skills. Hydrogeology positions require a minimum of a BS in geology or hydrogeology. Engineering positions require a minimum of BS in Engineering and P.E. or eligibility.

Interested candidates should send their resumes to:

Dames & Moore

7101 Wisconsin Avenue
Bethesda, MD 20814
Attention: Personnel Dept. CEM

EOE/AA



TANK LEAK? LINE LEAK? NO LEAK?

**GET THE ANSWER FAST,
WITHOUT EXCAVATION!**

Ask us to prove our "edge" in accuracy, speed, trained personnel, economy; national program capability, state-of-art technology. Meets NFPA 329 standards. Call today!

leak lokator

Hunter

ENVIRONMENTAL SERVICES, INC.

115 DEWALT AVE. N.W., CANTON, OHIO 44702
800-523-4370 (In Ohio: 216-453-1800)

Estimating risk to human health

Trichloroethylene in drinking water is used as the example

C. Richard Cothorn
William A. Coniglio
William L. Marcus
*Environmental Protection Agency
Office of Drinking Water
Washington, D.C. 20460*

The decision to regulate the maximum level of an environmental contaminant allowable in air, water, or food is predicated on the ability of the substance to alter life processes and on the degree of exposure that creates a significant risk of adverse effects to a sensitive population. For more than four centuries, scientists have tried to understand the adverse effects of exposure to environmental contaminants.

In general, any contaminant can cause damage if exposure to it creates high concentrations within a cell, and if accumulated cellular damage can result in disease or death. Environmental regulations are developed to reduce exposure to a level that precludes such health effects.

Drinking-water regulations are being developed for many contaminants, some of which are known to cause cancer in laboratory animals. One group, the volatile organic compounds (VOCs), is currently being assessed for the development of national drinking-water standards. Reference 1 contains an explanation of the risk assessment studies being used to evaluate VOCs.



SL 034470

Levels of human exposure to VOCs found in drinking water and the level of cancer risk imposed by their presence have been estimated as part of the process of developing regulations. Estimates include the health benefits to be derived from different levels of national regulation. They couple the estimate of carcinogenic potency, derived from an extrapolation of a dose-response curve developed from high-level exposure of experimental animals, with estimates of the number of persons exposed to contaminated drinking water. The estimates include the level of daily intake—oral, respiratory, and dermal—resulting from the presence of these chemicals in water (Figure 1).

Estimates of the number of cancers resulting from currently unregulated conditions and of those cancers that will be prevented or allowed by setting standards at different levels can be included in the regulatory process. Current knowledge about the health effects of contaminants has its limits: Much is known about some aspects; little or

nothing is known about others. But there is uncertainty in all areas.

The *population risk estimate* is an assessment of the total expected excess number of cancers in the United States during a lifetime from a given contaminant. This is an estimate and not a statement of perfect knowledge. Therefore, a level of uncertainty or a range of values will be included to describe the estimate. If the frequency distribution of the contributing factors controlling exposure and toxicity were known, the propagation of errors could be determined by use of standard methods. Frequency distributions often are not known, however, and a range of values often must be used to approximate the situation.

This article describes some of the data on which regulatory judgments are made. It also describes the assumptions introduced to complete the assessments, the results, and their uncertainty. We take as our example the VOC trichloroethylene (TCE).

Recent research has shown that TCE

can be transformed in water to vinyl chloride, a known human carcinogen (2). The cancer risk associated with the presence of vinyl chloride in drinking water also has been assessed (1).

TCE in drinking water

There are approximately 60,000 public water supplies in the United States, serving some 220 million persons. Three major federally sponsored monitoring surveys have been conducted nationwide to obtain information on the concentration of selected VOCs appearing as pollutants in potable water provided to the nation's cities and towns.

By using the survey data in conjunction with the multinomial approach for projecting national occurrence, it was estimated that 97% of groundwater systems of all sizes contain either no TCE or concentrations $< 0.5 \mu\text{g/L}$ (Table 1). It is not possible to estimate how many systems in this category are contaminated with TCE.

Of the estimated 1632 systems expected to have levels $> 0.5 \mu\text{g/L}$, 421 (0.9% of the total number of groundwater systems) are expected to have concentrations $> 5.0 \mu\text{g/L}$, and 133 systems (0.3%) nationally have concentrations $> 50 \mu\text{g/L}$. Although a greater percentage of the large systems is expected to be contaminated, because of the large number of small groundwater systems it is likely that in absolute numbers more small systems are contaminated.

In a similar analysis of surface water supplies, it was estimated that 96% either have no TCE present or that levels are $< 0.5 \mu\text{g/L}$. It is estimated that 498 surface water systems have levels $\geq 0.5 \mu\text{g/L}$ (4.5% of total surface water systems); 9 ($< 0.1\%$) are expected

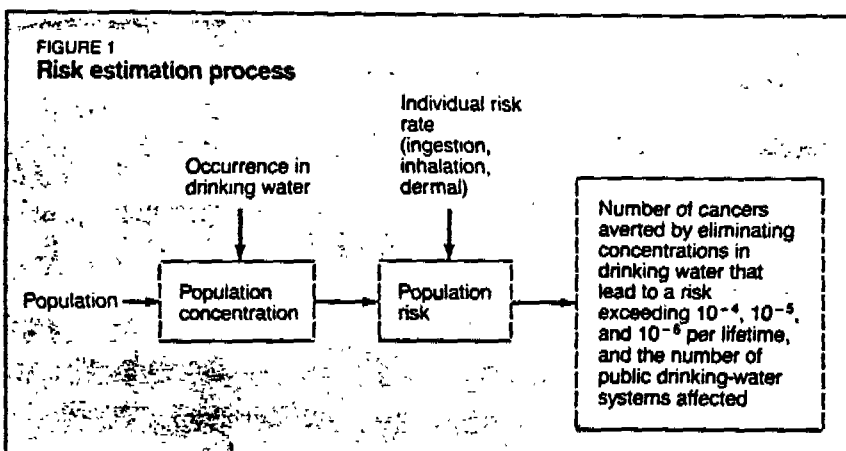


TABLE 1
Trichloroethylene contamination of groundwater systems

System size (population served)	No. of systems in U.S.	Estimated number of systems by concentration ($\mu\text{g/L}$)												
		$< 0.5^*$	0.5-5	$> 5-10$	$> 10-20$	$> 20-30$	$> 30-40$	$> 40-50$	$> 50-60$	$> 60-70$	$> 70-80$	$> 80-90$	$> 90-100$	> 100
25-100	19,125	18,506	465	26	52	0	26	0	26	0	0	0	0	26
101-500	15,674	15,166	381	21	42	0	21	0	21	0	0	0	0	21
501-1,000	4,877	4,719	118	7	13	0	7	0	7	0	0	0	0	7
1,001-2,500	4,400	4,257	107	8	12	0	6	0	6	0	0	0	0	6
2,501-3,300	891	862	22	1	2	0	1	0	1	0	0	0	0	1
3,301-5,000	1,065	1,031	26	1	3	0	1	0	1	0	0	0	0	1
5,501-10,000	1,168	1,130	28	2	3	0	2	0	2	0	0	0	0	2
10,001-25,000	835	775	34	11	4	0	0	8	0	0	4	0	0	0
25,001-50,000	290	269	12	4	1	0	0	3	0	0	1	0	0	0
50,001-75,000	64	59	3	1	0	0	0	1	0	0	0	0	0	0
75,001-100,000	14	13	1	0	0	0	0	0	0	0	0	0	0	0
$> 100,000$	55	41	14	0	0	0	0	0	0	0	0	0	0	0
Total	48,458	46,828	1,211	80	132	0	64	12	64	0	5	0	0	64

*Calculated as the difference between the number of systems expected to have $\geq 0.5 \mu\text{g/L}$ TCE and the total number of systems in that size category. This group includes those having no contamination and those with levels $< 0.5 \mu\text{g/L}$.

to have levels $>5 \mu\text{g/L}$; none is estimated to have TCE $>40 \mu\text{g/L}$.

Using the combined data from surface water and groundwater supplies, it was estimated that nearly 190 million persons (88.3% of the population served by public drinking-water systems) receive uncontaminated water or water contaminated with TCE at levels $<0.5 \mu\text{g/L}$. Of the 25 million persons (11.7%) receiving water that contains TCE at levels $\geq 0.5 \mu\text{g/L}$, an estimated 1.8 million (0.9%) are exposed to levels $>5 \mu\text{g/L}$. About 212,000 persons (0.1%) are probably exposed to levels $>50 \mu\text{g/L}$, and 42,000 ($<0.1\%$) are estimated to be exposed to levels $>100 \mu\text{g/L}$.

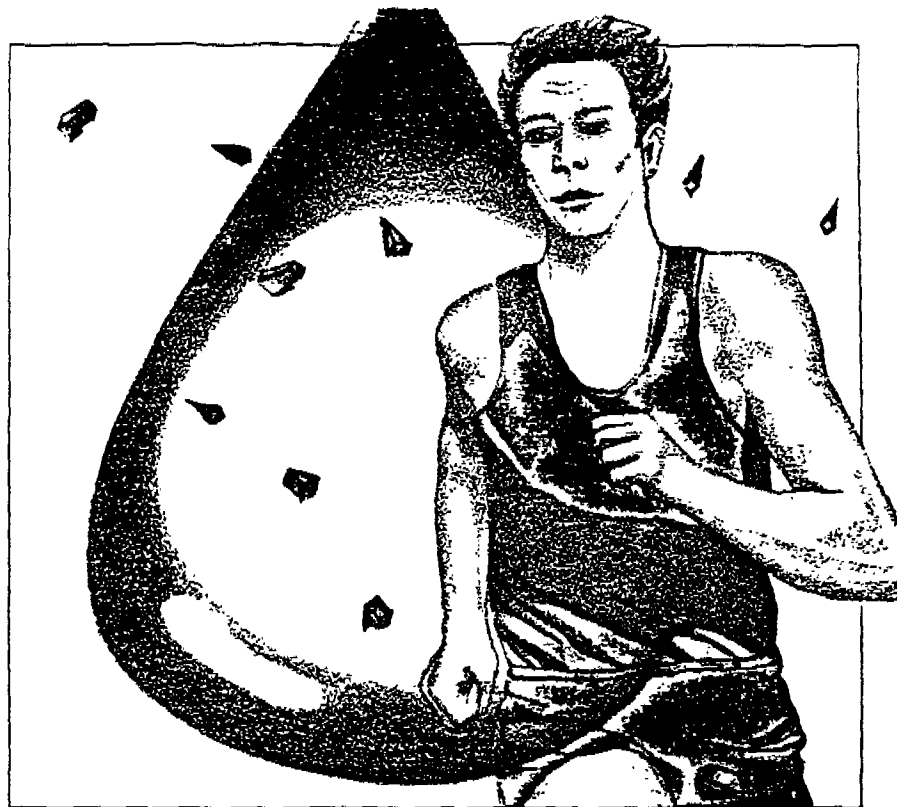
Of the approximately 23 million persons exposed to levels ranging from $0.5 \mu\text{g/L}$ to $5 \mu\text{g/L}$, 76% obtain water from surface water supplies. However, of the 1.8 million persons exposed to levels $>5 \mu\text{g/L}$, 62% use groundwater sources. All exposure to TCE in drinking water in levels $>40 \mu\text{g/L}$ is projected to be from groundwater sources.

Several assumptions have been made during the assessment of TCE contamination of drinking water, among them that sites selected in surveys are representative of drinking-water supplies across the nation and that grab samples are representative of water quality throughout the water supply system.

Using scientific judgment, the uncertainties in these estimates were analyzed. The uncertainties include those in the measurements themselves and those involved in projecting the existing data to estimate natural occurrence. In general, the overall uncertainty is less than an order of magnitude.

Human exposure to contaminants

The average adult in the United States uses 48–55 gal of water each day (3, 4). This means that a household of two children and two adults uses ap-



proximately 255 gal of water each day (5). Water pollutants pass through the home in water that is used for laundering clothing, washing dishes, removing wastes, bathing, cooking, and drinking.

The level of exposure to any pollutant present in drinking water is the result of many personal daily choices and several factors over which we have very little direct control. Where we live, what we eat and drink, and how old we are all have a profound influence on the magnitude of exposure. Also, the physical and chemical characteristics of the pollutant govern the amount that stays in the water and that which is transported into indoor air.

Table 2 provides an example of the potential differences in exposure of

family members to a VOC resulting from personal choices. This table has been prepared for a theoretical VOC, similar in chemical characteristics to TCE. We assumed that 100% of the compound is absorbed for all oral intake, 50% for respiratory intake, and a very small amount is absorbed in direct proportion to water flux across the skin.

Cancer risks projected for TCE are based on assumed lifetime ingestion of 0.03 L of drinking water for each kilogram of body weight and on the assumption that 100% of the ingested chemical is absorbed. The factor for ingestion was developed using an intake standard of 2 L/70 kg adult weight. Individuals may experience many times this intake rate. Formula-fed infants and young children, for example, have average intake rates that are as much as eight times greater than those of average adults.

Adults in tropical areas may consume twice as much liquid as the average, as may athletically inclined adults when engaged in strenuous physical activity. Persons who are ill also may consume much more water than the average. Social behavior ritualized around the drinking of tea or coffee may lead to increased water consumption, although boiled water may contain smaller amounts of contaminants. A recent Canadian study shows that children under 5 years old, 10% of the children 6–17 years old, and 2% of the adult population consume more than 0.03 L/d of drinking water for every kilogram of

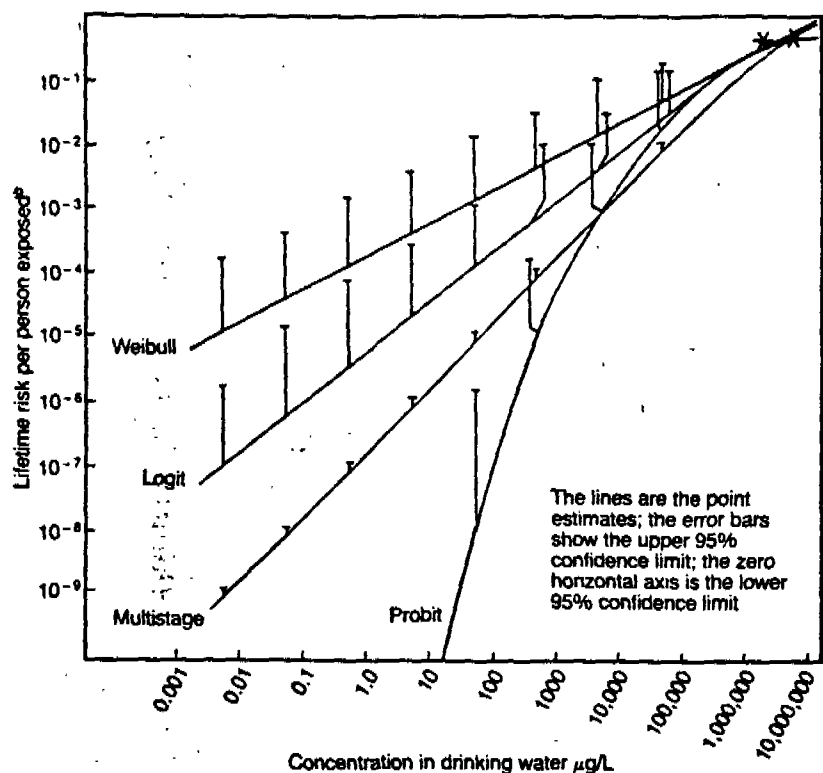
TABLE 2
Comparative model of absorbed dose from a volatile pollutant in drinking water^a

Exposure route	Absorbed dose ($\mu\text{g/d}$)			
	Formula-fed infant (4 kg)	Preteen (32 kg)	Adult female (60 kg)	Adult male ^b (70 kg)
Fluid ingestion	80	150	200	200
Inhalation of enriched indoor air	10	10	70	50
Inhalation of enclosed shower air	—	100	300	200
Dermal absorption				
Bathing	.02–.06	—	?	?
Swimming	—	10–300	—	—
Total absorbed dose	20 $\mu\text{g/kg/d}$	10–20 $\mu\text{g/kg/d}$	10 $\mu\text{g/kg/d}$	7 $\mu\text{g/kg/d}$

^a100 $\mu\text{g/L}$

^bShowering adult male

FIGURE 2
Bioassay data and model extrapolations for exposure
to TCE through ingestion*



*Bioassay data are the starred points in the upper right-hand corner. The lifetime risk per person exposed was calculated assuming that the risk due to inhalation and dermal exposure is equal to that due to ingestion exposure. The error introduced by this simplifying assumption is less than the widths of the lines shown.
*Through ingestion, inhalation, and dermal exposure, assuming that ingestion equals inhalation plus dermal

body weight (6).

Each of the VOCs considered, including TCE, has been shown to transfer from water into air if the water is heated or aerated. Monitoring data indicate that this process is continuous within the home and leads to an immediate enrichment of respirable air at the point of water use and to a diffusion throughout the home. Showers taken within an enclosed bathroom, using

20–30 gal of water, result in the liberation of all or a portion of the VOCs into the air. A person who showers inhales air containing a highly enriched level of the chemical while inside the bathroom. VOCs from other sources as well mix with general home air, resulting in higher levels of the pollutants being released into the indoor air.

Very few data are available on the enrichment of general indoor air with

VOCs. However, research has been conducted on the radon enrichment of indoor air, and it is assumed that the fraction of radon released is about the same as that for VOCs. These data indicate that an enrichment of indoor air (the amount of radon in a liter of air) is about 10^{-3} to the concentration found in a liter of drinking water (7–10). This model assumes an indoor air change rate of one per hour.

For our analysis it has been assumed that respiratory exposure to VOCs in drinking water is roughly equivalent to that from ingestion. To compare the relative exposures from inhalation and ingestion, assume that for both pathways 100% of the VOCs that enter the body reach the bloodstream. Then if water with a concentration of $X \mu\text{g/L}$ is ingested, the daily intake to the bloodstream is $2X \mu\text{g/d}$. For example,

$$X \mu\text{g/L} \times 2 \text{ L/d} = 2X \mu\text{g/d} \quad (1)$$

If water with a concentration of $X \mu\text{g/L}$ transfers all of the VOC to indoor air and it all goes to the bloodstream, then the intake to the bloodstream is again $2X \mu\text{g/d}$. For example,

$$\begin{aligned} & \frac{X \mu\text{g}}{\text{L}} \times \frac{10^{-4} \text{ L (water)}}{\text{L (air)}} \\ & \times \frac{20 \text{ m}^3 \text{ (air)}}{\text{d}} \times \frac{10^6 \text{ cm}^3 \text{ (air)}}{\text{m}^3 \text{ (air)}} \\ & \times \frac{\text{L (air)}}{1000 \text{ cm}^3} = 2X \mu\text{g/d} \quad (2) \end{aligned}$$

Under these conditions exposure from ingestion and inhalation is equal.

Individual risk rates

Estimates of individual risk rates are based on bioassay data derived from animal experiments. These data are converted to a continuous human equivalent exposure for use in risk estimates. The bioassay data selected to estimate carcinogenic potency of VOCs in drinking water are from experiments

TABLE 3
Population risk estimates for current TCE levels in drinking water

Mean drinking-water concentration ($\mu\text{g/L}$)	Number of persons served	Total lifetime individual risk for the mean concentration ^a		Lifetime population risk
		Low (probit)	High (Weibull)	
0.25	1.9×10^6	$< 10^{-10}$	2.4×10^{-4}	$< 1-45,600$
2.75	2.3×10^7	$< 10^{-10}$	7.3×10^{-4}	$< 1-16,790$
7.5	4.3×10^6	$< 10^{-10}$	1.3×10^{-3}	$< 1-559$
15	2.1×10^6	$< 10^{-10}$	1.7×10^{-3}	$< 1-367$
35	7.4×10^5	7×10^{-8}	2.3×10^{-3}	$< 1-1,702$
45	2.6×10^5	3×10^{-7}	2.6×10^{-3}	$< 1-676$
55	4.2×10^4	4×10^{-7}	2.8×10^{-3}	$< 1-117$
75	1.3×10^5	6×10^{-7}	3.2×10^{-3}	$< 1-416$
100	4.2×10^4	1.2×10^{-6}	3.7×10^{-3}	$< 1-155$
Total ^b				$< 1-70,000$

^aThe total individual risk was determined by assuming that the risk due to inhalation is equal to that due to ingestion

^bRounded to one significant figure

SL 034473

reviewed and selected by the National Academy of Sciences and the EPA Carcinogen Assessment Group (11, 12). When the carcinogenic data were from inhalation experiments, they were converted to ingestion values.

The bioassay data are fit to four analytical models: logit, multistage, probit, and Weibull. These four models are chosen somewhat arbitrarily and are representative of models currently in use. It is well-known that other models could be used to fit the same data (13-16). There are no biologically based criteria for choosing one model over another. The models are in the form of analytical expressions that can be used to fit the high-dose data mathematically.

The dose-response curves for the models are fit to equivalent human dose-response data converted using the relative surface area of animals and humans. The curves are extrapolated to the lower environmental levels. A log-log plot of the entire region shows the two nonzero data points for TCE (Figure 2). The solid lines are the point estimates, and the error bars show the upper 95% confidence limits. The GLOBAL computer program was used for the multistage model, and the Krewski computer program was used for the logit, probit, and Weibull models.

The curves shown in Figure 2 are not the only models that could be fit to the data. Additionally, there is no assurance that the actual curve is in the range of those shown. It is possible, although highly unlikely, that the actual curve lies outside those shown. For example, the probit model generally decreases extremely quickly relative to dose, whereas the Weibull model decreases very slowly.

Although the dose-response curves projected by each model in Figure 2 start at the same points, they diverge significantly at lower dose levels. At a concentration of 50 $\mu\text{g/L}$ TCE in drinking water, the Weibull model provides a risk estimate approximating 1×10^{-2} , whereas the probit model provides an

estimate of 10^{-10} . These estimates provide a range of uncertainty equivalent to not knowing whether one has enough money to buy a cup of coffee or pay off the national debt.

Population risk calculations

The population at risk of health damage due to TCE in drinking water was estimated. The population risks are determined by a product of the number of people affected and the individual risk rate (Table 3). In each case the concentration is in the average of the range. The lowest concentration ($<0.5 \mu\text{g/L}$) represents the minimum reported concentrations for the national surveys and does not necessarily represent the detection limit of the chemical in drinking water. Although we have used the estimated average of $0.25 \mu\text{g/L}$ for this category, it is possible that all of the persons in this category could be exposed to $0 \mu\text{g/L}$ or that all of them could be exposed to the maximum of $0.5 \mu\text{g/L}$. For each chemical the individual lifetime risks were determined from the model fits.

In each case the curves in Figure 2 giving the lowest and the highest individual risks were used to determine the range of population risks. The uncertainty in choice of model in Figure 2 is by far the largest uncertainty of any involved in the estimates of risk. Thus it alone was used to calculate the range of risk. When calculations yielded less than one person, the number was listed as a whole number rather than as the fractional number of persons.

The risk reduction analysis (Table 4) is a determination of the number of cancer cases averted if the standard is set corresponding to individual lifetime risk rates of 10^{-4} , 10^{-5} , or 10^{-6} . For these individual risk rates, the corresponding range of concentration and the cases averted are listed. The concentration range was determined from the graph shown earlier in Figure 2. The population risk estimates are listed as ranges; the range is indicative of the uncertainty in this estimate.

For TCE, the lower estimate for each

concentration range is less than one. The sum of the upper population risk estimates is 70,000, when rounded off to one significant figure. The largest portion of the population risk is contributed by the lowest concentration category. This category represents calculations below the detection limit and may be an artifact of the mathematical analysis technique used to estimate the population risk. The range is largely a reflection of the uncertainty in choice of the mathematical model selected to estimate individual risk. The contribution to uncertainty from other factors is negligible by comparison.

If a drinking-water standard—a maximum contaminant level—for TCE were established at $7.5 \mu\text{g/L}$, nationwide compliance would result in the avoidance of anywhere from <1 to 4000 cases.

Explanation

The development of regulations covering contaminants in drinking water requires the assembly of data from many different sources. One important piece of information needed is the estimate of the expected number of health effects and how many of these can be prevented by regulation. Risk assessment usually involves using skimpy data concerning the levels of a contaminant in drinking water and using health effects data from animal experiments conducted at exposure levels much higher than those usually found in the environment.

The risk can be estimated using TCE in drinking water as the example, although the range of uncertainty is quite large. If there were no data, the range of estimated health effects would be between none and all the people. Using the available data for TCE in drinking water, the estimated range of health effects lies between <1 and 70,000. If all exposure to levels $>7.5 \mu\text{g/L}$ were eliminated, as few as one or less of these health effects could be prevented, or perhaps as many as 4000. These estimates are the best representation of the existing data for TCE in drinking water. Their value to the regulatory decision-making process must be judged when placed alongside the other information used in that process.

Acknowledgment

We thank Joseph A. Cotruvo for his encouragement and helpful discussions on the work described here.

The thoughts and ideas expressed in this article are those of the authors and are not necessarily those of the EPA.

References

- (1) Cothorn, C. R.; Coniglio, W. A.; Marcus, W. L. "Techniques for the Assessment of Carcinogenic Risk to the U.S. Population Due to Exposure from Selected Volatile Or-

TABLE 4
Risk reduction analysis*

Maximum allowable drinking-water concentration ($\mu\text{g/L}$)	Approximate individual risk rate for maximum concentration	Cumulative cases averted*
100	1×10^{-7} to 1×10^{-2}	<1 -200
45	1×10^{-8} to 1×10^{-3}	<1 -1,000
7.5	$<1 \times 10^{-10}$ to 6×10^{-3}	<1 -4,000
2.75	$<1 \times 10^{-10}$ to 4×10^{-3}	<1 -20,000

*For limiting TCE concentration in drinking water

*Number of cases averted for concentration shown in first column

ganic Compounds from Drinking Water via the Ingestion, Inhalation and Dermal Routes," EPA 570/9-85-001; EPA: Washington, D.C., 1985.

- (2) Vogel, T. M.; McCarty, P. L. *Appl. Environ. Microbiol.* 1985, 49, 1080-83.
- (3) Watson, K. S.; Farrell, R. P.; Anderson, J. S. *J. Water Pollut. Control Fed.* 1967, 30(12), 2039-54.
- (4) Lenaweaver, F. P., Jr. In "A Study of Flow Reduction and Treatment of Wastewater from Households"; Bailey, J. R., Ed.; General Dynamics report to the Federal Water Quality Administration: Washington, D.C., 1969.
- (5) Bailey, J. R. et al. "A Study of Flow Reduction and Treatment of Waste Water from Households," Water Pollution Control Research Series 11050FKE; General Dynamics report for the Federal Water Quality Administration; Department of the Interior: Cincinnati, Ohio, 1969.
- (6) "Tap Water Consumption in Canada," 82EHD80; Canadian Environmental Health Directorate, Ministry of National Health and Welfare: Ottawa, Ont., 1981.
- (7) "Sources and Effects of Ionizing Radiation," Report to the General Assembly of the United Nations by the United Nations Scientific Committee on the Effects of Atomic Radiation, 1977.
- (8) Hess, C. T.; Weiffenbach, C. V.; Norton, S. A. *Environ. Int.* 1982, 8, 59.
- (9) Kahlos, H.; Asikainen, M. *Health Phys.* 1980, 39, 108.
- (10) Nazaroff, W. W. et al. "Potable Water as a Source of Airborne Radon-222 in U.S. Dwellings: A Review and Assessment,"

LBL 18154; Lawrence Berkeley Laboratory: Berkeley, Calif., 1985.

- (11) *Drinking Water and Health*; National Academy of Sciences: Washington, D.C., 1982.
- (12) Anderson, E. L. et al. *Risk Anal.* 1983, 3, 277-95.
- (13) Van Ryzin, J. J. *Occup. Med.* 1980, 22, 321-26.
- (14) Van Ryzin, J.; Rai, K. In *The Scientific Basis of Toxicity Assessment*; Witschi, H., Ed.; North-Holland Biomedical Press: New York, N.Y., 1980; p. 273.
- (15) Krewski, D.; Van Ryzin, J. In *Statistics and Related Topics*; Csorgo, M. et al., Eds.; North-Holland: New York, N.Y., 1981; p. 145.
- (16) Munro, T. C.; Krewski, D. R. *Food Cosmet. Toxicol.* 1981, 19, 549-60.



C. Richard Cothorn is a physicist with EPA. He received his Ph.D. in physics

from the University of Manitoba. He is involved in setting standards for radionuclides in drinking water and in assessing risks that result from drinking-water contaminants.



William A. Coniglio (l.) is a biologist with EPA. He has an M.S. in economic biology from Rutgers University and received public health training at the University of Alabama. He develops background information on the occurrence of and exposure to drinking-water contaminants.

William L. Marcus (r.) is a toxicologist with EPA. He received his Ph.D. in toxicology and pharmacology from Howard University. He reviews and analyzes toxicological reports and data and advises on toxicological matters.

CIVIL ENGINEERS ENVIRONMENTAL ENGINEERS CHEMICAL ENGINEERS HYDROGEOLOGISTS

Dames & Moore, one of the world's largest firms in engineering and applied earth sciences, is continuing to expand its practice in waste management and water resources engineering. Our Suburban Washington, D. C. office has several immediate vacancies:

- Civil Engineers—1 to 5 years experience in all aspects of civil engineering with a concentration in water resources and hazardous waste management;
- Chemical and Environmental Engineers—2 to 15 years experience in hazardous waste remedial investigations and alternative technologies studies, RCRA Part B permitting, and advanced wastewater treatment systems development;
- Hydrogeologists—2 to 10 years experience in design and review of groundwater sampling and monitoring programs, groundwater modeling and analysis of contaminant transport.

All positions require excellent communication skills. Hydrogeology positions require a minimum of a BS in geology or hydrogeology. Engineering positions require a minimum of BS in Engineering and P.E. or eligibility.

Interested candidates should send their resumes to:

Dames & Moore

7101 Wisconsin Avenue
Bethesda, MD 20814
Attention: Personnel Dept. CEM

EEO/AA



TANK LEAK? LINE LEAK? NO LEAK?

**GET THE ANSWER FAST,
WITHOUT EXCAVATION!**

Ask us to prove our "edge" in accuracy, speed, trained personnel, economy, national program capability, state-of-art technology. Meets NFPA 329 standards. Call today!

leak lokator

Hunter

ENVIRONMENTAL SERVICES, INC.

115 DEWALT AVE. N.W. CANTON, OHIO 44702
800-523-4370 In Ohio 216-453-1800