

HEALTH RISK ASSESSMENT ON RESIDENTS EXPOSED TO CHLORINATED HYDROCARBONS CONTAMINATED IN GROUNDWATER OF A HAZARDOUS WASTE SITE

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We conducted this study to estimate residents' chronic hazard and carcinogenic risk in a groundwater-contaminated community after on-site remediation in Taiwan during 1999–2000. We followed guidelines for assessing hazardous waste sites of the U.S. Environmental Protection Agency (EPA) and used empirically measured contaminant levels and exposure parameters to perform health risk assessment on seven chlorinated hydrocarbons. We measured groundwater concentrations of vinyl chloride, tetrachloroethylene, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane, cis-1,2-dichloroethylene, and 1,1-dichloroethane in 49 off-site residential wells by gas chromatography/mass spectrometry. Exposure parameters were mainly derived from our field survey of 382 residents, and partially from U.S. EPA default values. Total exposure dose estimation included routes of inhalation during showering and dermal absorption of showers and other activities involved with hand–water contacts. The ingestion route of water was not included because most residents drank boiled water with negligible contaminants. We calculated a hazard index (HI) for all seven chlorinated hydrocarbons and carcinogenic risks for known human carcinogen of vinyl chloride and probable human carcinogens of tetrachloroethylene and trichloroethylene, which had the same target organ, the liver. The HI values for reasonable maximal exposure (RME) and average exposure were 14.3 and 0.2, respectively. The cancer risks based on RME and average exposure (in parentheses) for vinyl chloride, tetrachloroethylene, and trichloroethylene were 8.4×10^{-6} (7.3×10^{-9}), 1.9×10^{-4} (1.3×10^{-7}), and 1.4×10^{-4} (1.2×10^{-6}), respectively. We applied Monte Carlo simulations to the sensitivity analysis, which showed that the contaminant levels, exposure duration, and time for showers were major determinants of health risks. We concluded that the contaminated groundwater was still unsafe for use even after the contaminated site underwent remediation by extraction and treatment in 1997.

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Uncontrolled hazardous waste sites have emerged as a major environmental and public health concern in many countries. The Taiwan Environmental Protection Administration (EPA) has identified more than 160 hazardous waste sites (Taiwan EPA, 1999). In 1994, a hazardous waste site contaminated by a former factory involved in the manufacturing of electronic appliances was found in Taoyuan City, Taiwan (designated as the R factory). The factory was built and in operation between 1970 and 1992. Previous site investigations revealed that on-site soils and groundwater in the factory were contaminated by about 12 different chlorinated hydrocarbons for more than 10 yr (Bechtel, 1995; Target, 1995). Eight of them were also found in the off-site groundwater of the downstream area, namely, 1,1-dichloroethane (1,1-DCA), 1,2-dichloroethane (1,2-DCA), 1,1-dichloroethylene (1,1-DCE), *cis*-1,2-dichloroethylene (*cis*-1,2-DCE), 1,1,1-trichloroethane (1,1,1-TCA), tetrachloroethylene (PCE), trichloroethylene (TCE), and vinyl chloride (VC) (ITRI, 1994; Geomatrix, 1995). The highest contaminant level in an off-site residential well was 4800 µg/L for PCE, which was adjacent to the factory's source area with soil concentration up to 1100 mg/kg of PCE (Geomatrix, 1995; Bechtel, 1995). The Taiwan EPA declared the R factory a hazardous waste site in 1994 and requested remediation action on the contaminated soils and groundwater within the factory during 1996–1998 (Taiwan EPA, 1994–1997).

An extraction and treatment system for restoring contaminated groundwater had been operated for 6 mo in 1997 and failed to meet the requirements of World Health Organization (WHO) drinking-water guidelines (Geomatrix, 1998). There are about 10,000 residents currently living in the village downstream of the R factory. Many residents living in the vicinity of the factory relied mainly on locally extracted groundwater from residential wells as their primary source of domestic water supply until 1994. Some of them still continued to use these wells for showering, washing vegetables and dishes, or other cleaning activities, even though tap water was supplied. Concerned about these residents' potential health risks from chronic exposures to the contaminated groundwater, we initiated a comprehensive study entitled "Epidemiological Study and Health Risk Assessment for Residents Living Near a Contaminated Site Polluted by Volatile Chlorinated Compounds" during 1999–2000. This study included measurements of well waters, toxicological studies with animal models, epidemiological investigations, and health risk assessment for chlorinated hydrocarbons. We present here only part of the risk assessment. We demonstrate that a well-planned and carefully carried-out risk assessment can help evaluate potential health problems for a contaminated site and identify important exposure parameters for risk management.

METHODS

We conducted this site-specific risk assessment by following the detailed guidance for developing risk information at hazardous waste sites published

by the U.S. Environmental Protection Agency (U.S. EPA, 1989, 1998a), which included the following processes.

Hazard Identification of Contaminants in Groundwater

The hazard identification was mainly based on the considerations of contaminants' toxicity, environmental persistence, and concentrations. Chlorinated hydrocarbons with the characteristics of more toxic, persistent, mobile, or higher concentrations were our top choices of target chemicals of concern. In addition, we also considered degradation products from the chemicals used in the R factory as our target pollutants because many contaminants had been present for more than 10 yr (Bechtel, 1995). For example, PCE can lead to the production of seven chlorinated hydrocarbons, including TCE, *cis/trans*-1,2-DCE, 1,2-DCA, 1,1-DCE, 1,1-DCA, and VC. And 1,1,1-TCA can form 1,1-DCA, 1,1-DCE, and subsequently VC through anaerobic reductive dehalogenation (Sims et al., 1991). Although all of these chemicals can be determined by our analytical methods, described later, we only conducted quantitative risk assessment on the chemicals that exceeded the maximum contaminant levels (MCLs) of the U.S. EPA (1999). This practice was in line with the U.S. EPA's recommendations in the Risk Assessment Guidance for Superfund (RAGS) (U.S. EPA, 1989). Moreover, these chemicals' toxicological endpoints and target organs were obtained by reviewing epidemiologic studies and animal bioassays on these chlorinated solvents from the literature, such as the TOMES database (Micromedex, 1998), the Agency for Toxic Substances and Disease Registry Toxicological Profiles (ATSDR, 2000), Medline databases, etc. In order to perform quantitative risk assessments on noncarcinogenic health effects, we reviewed systemic toxicities, including hepatotoxicity, nephrotoxicity, and central nervous system depression, which resulted from chronic exposure to these chlorinated solvents. For conducting quantitative carcinogenic risk assessment, we selected only chemicals classified by the International Agency for Research on Cancer (IARC) as Group 1 or 2A (IARC, 1995). We summarized carcinogenic risks across chemicals that affected the liver as their major target organ based on animal studies (U.S. EPA, 2001a; Rhomberg, 2000).

Exposure Assessment

Groundwater Sampling and Analysis We conducted groundwater sampling from off-site residential wells that were still in use for domestic purposes. According to the local groundwater hydrogeology, the off-site groundwater flow direction was generally toward the north and northeast in the first aquifer (Geomatrix, 1998). Residential wells in the nearby communities were identified by a door-to-door survey. The wells were located from about 50 m up to 1 km away from the factory. For the downstream wells to the north of the factory, we collected a total of 69 samples from 44 wells with 1 sample per well for 32 wells and 2 to 3 samples per well for the other 12 wells. For the upstream community, we collected two groundwater samples of water from two wells to the south of the factory and

another three samples from three wells to the west of the factory. In total, we had 74 groundwater samples to describe environmental concentrations of chlorinated hydrocarbons surrounding the hazardous site.

We followed the standard procedures of sampling groundwater. In brief, water in the wells was drawn directly from the wells with a bailer and then stored in 30-ml brown bottles containing 30 mg preadded ascorbic acid. The bailer was washed with the well water twice before each collection. Each sampling bottle was filled completely and sealed immediately to ensure water samples without bubbles. All water samples were stored at 4°C before analysis, and the analytical work was completed within 2 wk after sampling.

Nineteen chlorinated volatile organic compounds (VOCs) were measured in this study, including VC, PCE, TCE, 1,1-DCE, *cis/trans*-1,2-DCE, 1,1,1-TCA, 1,1-DCA, 1,2-DCA, chloroethane, trichlorofluoromethane, methylene chloride, chloroform, benzene, bromodichloromethane, toluene, 1,1,2-trichloroethane, chlorobenzene, and 1,1,1,2-tetrachloroethane. The VOC concentrations were determined by using a method modified from U.S. EPA Method 524.2 (U.S. EPA, 1992a). Briefly, a purge and trap device (Tekmar 3000) was used to collect the analytes, which were subsequently analyzed by a gas chromatograph/mass spectrometer (HP6890 and HP5972, Hewlett Packard, USA) equipped with an RTX-Volatiles capillary column (60 m × 0.32 mm × 1.5 μm). Calibration was performed by the internal standard method using 50 μg/L fluorobenzene and 4-bromofluorobenzene in each sample for quantification. The detection limit of each VOC was calculated by 7 different analyses of the VOC standards at 0.4 μg/L. Based on threefold the standard deviation of the mean standard concentration, the detection limits for these 19 VOCs ranged from 0.01 to 0.1 μg/L. Nondetected (ND) values were replaced by half of their detection limits when calculating the geometric and arithmetic mean of concentrations. The reproducibility was evaluated with nine sets of groundwater samples containing VOCs and was calculated from two duplicate analyses. The relative mean deviation of these duplicates was within 20% for most of the target VOCs.

Water Exposure Parameters We used empirical data from our field survey to obtain exposure parameters for a more realistic risk prediction. Residents in the nearby communities were invited to participate in a complete physical examination including a detailed structured questionnaire. We collected data on body weight, height, exposure-related activities such as drinking water, showering, food and cloth washing, etc., as well as exposure duration and time. However, for ventilation rate, chemical-specific permeability coefficient, and average time, which were not collected in our study, the default values recommended by the U.S. EPA were adopted (U.S. EPA, 1992b, 1997a).

We considered all the potential exposure routes, including ingestion, inhalation, and dermal absorption, in the exposure assessment. First, we conducted an experiment to measure VOCs in boiling water to examine

whether ingestion of boiled water was a potential exposure pathway because the survey showed that 99% of the residents drank boiled water. Second, the inhalation exposure from volatilization in showers was estimated by using the model developed by Little (1992) and the physical characteristics of shower system in Taiwan reported by Kuo et al. (1998). The parameters of such shower rooms were 1.2 m³ of air in the shower, water flow rate of 5 L/min, air flow rate of 50 L/min, air exchange rate of 2.5 times/h, water temperature of 44°C, and shower-head height of 1.8 m. Based on two-resistance theory, we can obtain overall mass-transfer coefficients with a liquid-phase basis ($K_{OL}A$) for PCE and TCE that were 7.2 L/min, and 9.2 L/min, respectively (Little, 1992). The values of $K_{OL}A$ for the other 5 chlorinated hydrocarbons were assumed to be 9.2 L/min because experimental data were not available. The values of absorption efficiency of chlorinated hydrocarbons in the alveoli were assumed around 50%, because TCE and 1,1,1-TCA were estimated to have inhalation absorption efficiency in the range of 30–78% (Pleil et al., 1998; Byard, 1989). Third, the dose from dermal absorption was estimated for showering and hand exposure-related activities, such as washing vegetables, washing dishes, and doing laundry by hand. The fractions of skin in contact with water were assumed to be 80% for showering, and 8% for washing activities involved with hand exposure, respectively. The chemical-specific permeability coefficients were derived from a report of dermal exposure assessment of the U.S. EPA (1992b).

The site-specific exposure parameters used in our study were obtained as follows: The exposure duration was defined as the period in years between the year of initial exposures after 1970 and the year at the end of exposures. The exposure time during showering was defined as the minutes per day that an individual spent for a shower using groundwater. A default value of 20 min/d was assumed for the exposure time of hand exposure-related activities. The skin surface area was estimated by using a formula (surface area = $0.0239H^{0.417}W^{0.517}$) proposed by the U.S. EPA (1997a) and the heights (H) and weights (W) of 382 individuals measured by the registered nurses.

Our risk assessments were based on two exposure scenarios: the reasonable maximum exposure (RME) as a precautionary estimate, and the average exposure proposed by the U.S. EPA (1989, 1998a, 2001b). For RME, we used the upper-bound estimate of each input variable, which were the 95% upper confidence limit of the arithmetic mean for chemical concentrations and the 95th percentiles of exposure parameters for their corresponding distributions. For average exposure, we used the geometric means of contaminant concentrations and the arithmetic means of exposure parameters to estimate health risks.

Chronic daily intakes were calculated both for inhalation while showering and for dermal absorption from showers and hand exposure-related activities. Lifetime intake was defined as the exposure dose per unit body

weight and per unit time, averaged over an assumed lifetime of 70 yr. The equations for calculating chronic daily intakes are displayed as follows:

Dermal contact:

$$\text{Chronic daily intake (mg/kg-d)} = \frac{C \times SA \times F \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

(U.S. EPA, 1989, 1997a), where C is chemical concentration in ground-water (mg/L), SA skin surface area (m^2), F fraction of skin in contact with water (unitless), PC Permeability coefficient (cm/h), ET exposure time (h/d), EF exposure frequency (d/yr), ED exposure duration (yr), and AT average time (d) using lifetime intake for carcinogen = 70×365 and chronic intake for noncarcinogen = $ED \times 365$, BW body weight (kg), and CF conversion factor (10^{-1} L/m^3).

Inhalation:

$$\text{Chronic daily intake (mg/kg-d)} = \frac{C_{\text{air}} \times VR \times AE \times ET \times EF \times ED \times CF}{BW \times AT}$$

(Kuo et al., 1998; U.S. EPA, 1989, 1997a), where C_{air} is concentration in air (mg/L), VR ventilation rate (m^3/h), AE absorption efficiency in alveoli (unitless) = 50%, ET exposure time (h/d), EF exposure frequency (d/yr), ED exposure duration (yr), AT average time (d) using lifetime intake for carcinogen = 70×365 and chronic intake for noncarcinogen = $ED \times 365$, BW body weight (kg), and CF conversion factor (10^3 L/m^3).

Dose Response/Risk Characterization

Chronic hazards and carcinogenic risks were characterized by incorporating exposure assessment and toxicity values. Hazard index (HI) was used as an indicator of noncarcinogenic risk, which summarized the ratios of the chronic exposure doses to the chemical-specific reference dose (RfD) values. Carcinogenic risk was incremental probability of getting cancer over a lifetime, which was calculated from lifetime intake multiplied by slope factor for individual chemical carcinogen. The primary sources of the toxicity values, including RfD and slope factors, were from the U.S. EPA databases. We followed a hierarchy of preferred sources, namely, Integrated Risk Information System (IRIS), Health Effects Assessment Summary Tables (HEAST), and other provisional values that were available in the online database of the Risk Assessment Information System (ORNL, 2001). As weighting evidence of carcinogenic potential (U.S. EPA, 1996), we assessed carcinogenic risk for human carcinogen of VC and probable human carcinogens of PCE and TCE. The total organ HI was calculated by summarizing over all chlorinated hydrocarbons assuming additive effects of multiple chemicals (U.S. EPA, 1989).

Uncertainty/Sensitivity Analysis

Monte Carlo analysis was used with the risk calculations to incorporate individual input distributions of exposure variables to produce probability distributions of the health risk estimates and to assess uncertainties in the risk assessment (U.S. EPA, 1997b). The simulations were performed with Crystal Ball (Decisioneering, 2000) in conjunction with the spreadsheet program Microsoft Excel 2000. Health risk was calculated through 5000 iterations using randomly selected values derived from individual probability distributions of the corresponding exposure model parameters. The simulation model was also used for sensitivity analysis in which the relative importance of each input variable was assessed by calculating its contribution to variance.

RESULTS

The hazard identification step identified seven chlorinated hydrocarbons, which met our selection criteria, as target pollutants for health risk assessment. Table 1 summarizes the toxicological characteristics, health concerns, and groundwater concentrations of VC, PCE, TCE, 1,1-DCE, 1,1,1-TCA, *cis*-1,2-DCE, and 1,1-DCA. Hepatic effects were their common noncarcinogenic chronic hazards. The three chemicals with carcinogenic effects, VC, PCE, and TCE, had the liver as their major target organ. There were wide ranges of groundwater concentrations among these seven chlorinated hydrocarbons. Geometric mean (GM) concentrations ranged from 0.03 µg/L for VC to 15.26 µg/L for TCE. The 95% upper-bound concentrations ranged from 7.61 µg/L for VC to 889.65 µg/L for PCE. There was also significant spatial variation in groundwater concentrations as shown by an example result for TCE in Figure 1. High levels of TCE above the MCL (5 µg/L) were found in 29 out of 44 wells (66%) in the downstream community. The highest concentrations of contaminants were found in the wells closest to the R factory. An excessively high TCE concentration of 192 µg/L was found even in the well located about 1 km northerly from the factory. In contrast, all 19 VOCs were either at trace or nondetected (ND) levels in the 2 upstream wells to the south of the factory.

A total of 529 adult residents aged above 15 yr participated in our field survey, including 382 persons in the downstream community and 147 from the upstream community. The exposure-related characteristics of the 382 downstream residents are summarized in Table 2. The prevalence of hepatitis B virus (HBV) carriers was 13.6%. The average body weight and skin surface area used in this study were 61 kg and 1.66 m², respectively, which were less than the U.S. EPA anthropometrical default values for assessing risks on adults. The average durations of key water exposure parameters were 13.7 yr of taking showers and 13.0 yr of involving hand exposure-related activities. The average time for each shower was about 13.9 min, which was less than the U.S. EPA default value. The results of residuals in water at temperatures of 4, 45, 65, 85, and 100°C and at 1 min after 100°C

TABLE 1. Concentrations and Frequency Distribution of Chemicals of Potential Concern in the 44 Downstream Residential Wells

| Chemicals of potential concern | Major target organ ^a | | | Percentage above MCL/PRG ^b | Geometric mean (range) (µg/L) | Arithmetic mean (95% UCL) ^c (µg/L) |
|----------------------------------|---------------------------------|------------------------------------|------------------------------|---------------------------------------|-------------------------------|---|
| | IARC cancer group | Carcinogenic effect (slope factor) | Noncarcinogenic effect (RfD) | | | |
| Vinyl chloride | 1 | Liver | Hepatic | 29.5%/31.8% | 0.03 (ND ^d -72.3) | 3.75 (7.61) |
| Tetrachloroethylene | 2A | Liver | Hepatic | 45.5%/45.5% | 2.63 (ND-5228.3) | 519.57 (889.65) |
| Trichloroethylene | 2A | Liver/lung | Hepatic/renal | 65.9%/77.3% | 15.26 (ND-1790.7) | 253.41 (404.68) |
| 1,1-Dichloroethylene | 3 | — | Hepatic | 27.3%/72.7% | 1.01 (ND-1240.4) | 111.41 (200.79) |
| 1,1,1-Trichloroethane | 3 | — | Hepatic | 11.4%/0% | 0.57 (ND-1504.4) | 82.68 (163.23) |
| <i>cis</i> -1,2-Dichloroethylene | — ^e | — | Hepatic/blood | 15.9%/15.9% | 1.16 (ND-1376.0) | 92.25 (181.06) |
| 1,1-Dichloroethane | — | — | Renal | —/0 | 0.61 (ND-227.9) | 16.60 (31.42) |

^aToxicity values are derived from animal bioassays based on which target organ is primarily affected.

^bMaximum contaminant level (MCL) is the water quality standard set by U.S. EPA (1999). Region 9 Preliminary Remediation Goal (PRG) is the risk-based contaminant level in residential tap water from inhalation exposure (U.S. EPA, 2000).

^c95% Upper confidence limit (UCL) is the upper-bound estimate of the contaminant distribution, which is the input for conservative estimation of health risk under reasonable maximal exposure (RME) assumptions.

^dND (nondetected) value was replaced by (1/2 × detection limit) as calculating the geometric/arithmetic mean.

^e—, Not available.

for PCE, TCE, 1,1-DCE, and 1,1-DCA are shown in Figure 2. The concentrations decreased significantly as water temperatures increased. There were negligible concentrations in the water after boiling for 1 min. Based on this finding, we excluded water ingestion exposure to VOCs from our risk estimation.

Table 3 presents the results of chronic exposure doses and health risk estimates for the routes of dermal absorption and inhalation based on the RME scenarios. The overall HI values calculated from inhalation exposure were about 10-fold higher than those from dermal absorption. Inhalation exposure from showering accounted for 61% of total cancer risks. There were 4 contaminants with HI greater than 1.0, which were PCE, TCE, 1,1-DCE, and *cis*-1,2-DCE. The summation of HI over 7 contaminants was 14.3, which was around an order of magnitude greater than the acceptable level. The carcinogenic risks for VC, PCE, and TCE were 8.4×10^{-6} , 1.9×10^{-4} , and 1.4×10^{-4} , respectively. The latter two IARC Group 2A car-

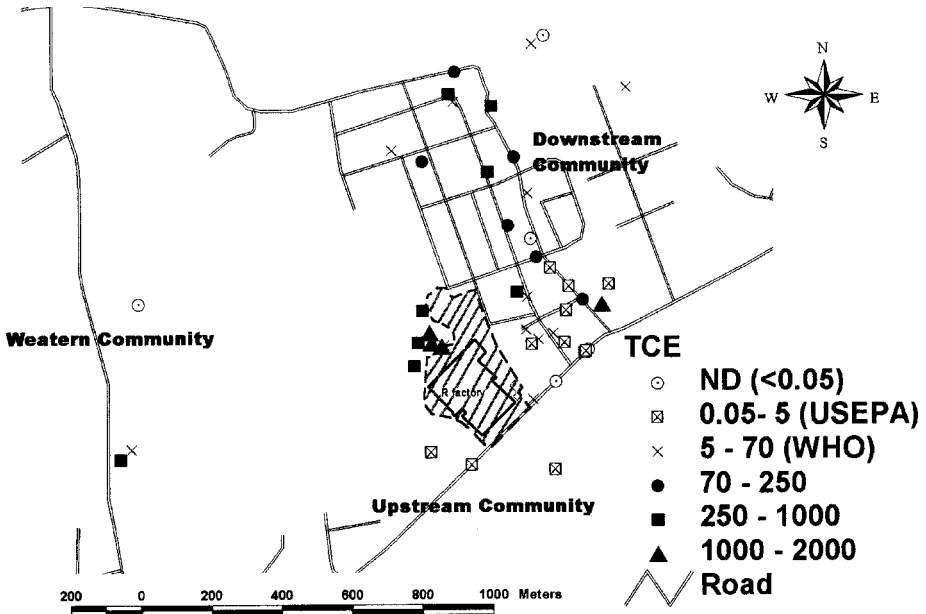


FIGURE 1. Geographic distribution of 49 residential wells near the R factory and levels of trichloroethylene (TCE, µg/L), one of the major contaminants in the groundwater.

TABLE 2. Summary of Exposure-Related Characteristics of 382 Residents Aged Over 15 yr in the Downstream Community

| Exposure-related characteristics ^a | Number | Downstream community |
|---|--------|----------------------|
| Gender | | |
| Male | 181 | 47.4% |
| Female | 201 | 52.6% |
| Age group (yr) | 382 | |
| 15–29 | 85 | 22.3% |
| 30–49 | 163 | 42.7% |
| ≥50 | 134 | 35.1% |
| Hepatitis B surface antigen positive | 52 | 13.6% |
| Body weight (kg) | 382 | 61.0 (± 10.7) |
| Body height (cm) | 382 | 160.1 (± 8.6) |
| Skin surface area (m ²) | 382 | 1.66 (± 0.17) |
| Exposure duration ^b (yr) | | |
| For showers | 336 | 13.7 (± 9.2) |
| For hand exposure-related activities | 330 | 13.0 (± 8.8) |
| Exposure time during showering (min) | 349 | 13.9 (± 6.9) |

^aCharacteristics are presented in percentage for nominal variables and arithmetic mean (± standard deviation) for continuous variables.

^bExposure duration was defined as the period in years between the year of initial exposures after 1970 and the year at the end of exposures.

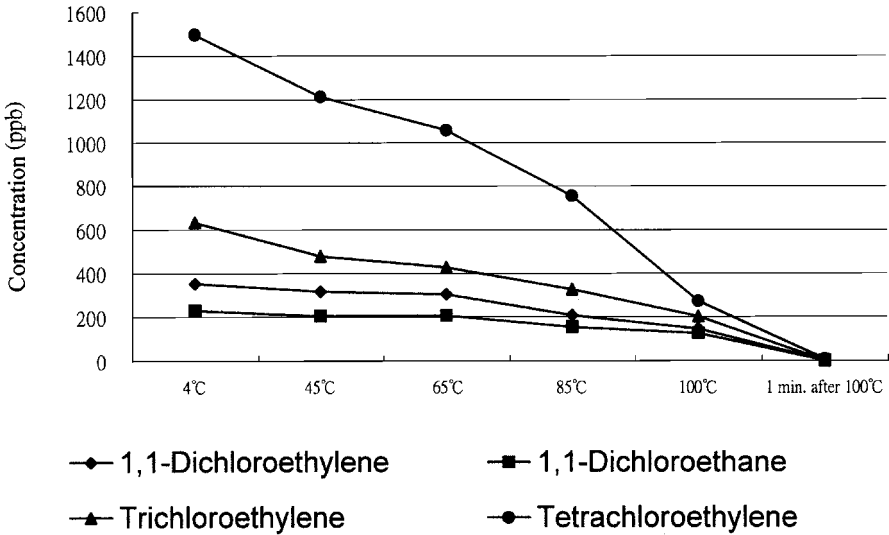


FIGURE 2. Concentrations of chlorinated hydrocarbons during the process of boiling, in an experiment on a water sample from a downstream contaminated well.

cinogens might pose cancer risks exceeding the upper limit of the acceptable range between 10^{-6} and 10^{-4} (U.S. EPA, 1990). The point estimates of health risks based on average exposure showed that HI was 0.2, and cancer risks of VC, PCE, and TCE were 7.3×10^{-9} , 1.3×10^{-7} , and 1.2×10^{-6} , respectively.

The input distributions of exposure-related variables, as well as assumed default values during Monte Carlo simulations, are summarized in Table 4. Figure 3 displays the cumulative probability distributions of simulated results and their associated sensitivity analyses for cancer risk (A) and HI (B). We found the simulated results were lognormal distributions. For cancer risk, the 95% upper-bound estimate was 7.3×10^{-5} , while the GM estimate was 2.2×10^{-6} . The sensitivity analysis showed that TCE and PCE contributed most to the variance of cancer risk estimates, while the exposure duration and time for showers accounted for another 9.9% and 8.1% of variance, respectively. For noncarcinogenic risk, the 95% upper-bound estimate was 8.9 and the GM estimate was 0.5. The variance of noncarcinogenic risk estimates was explained by TCE alone for 57.5% and exposure time of shower for 15.8%.

DISCUSSION

This is the first site-specific quantitative health risk assessment among more than 160 suspected hazardous waste sites throughout Taiwan. Based on the RME scenarios, we found that the community residents exposed to chlorinated hydrocarbons in the contaminated groundwater had an un-

TABLE 3. Calculated Health Risks, Both Noncarcinogenic and Carcinogenic, Based on Reasonable Maximal Exposure (RME)

| Exposure route | Chemicals of potential concern | Noncarcinogenic effects | | | Carcinogenic effects | | |
|--------------------------------|--------------------------------|--------------------------|--------------------------------|-----------------|---------------------------|------------------------|-------------|
| | | Chronic intake (mg/kg-d) | Reference dose (RfD) (mg/kg-d) | Hazard quotient | Lifetime intake (mg/kg-d) | Slope factor (mg/kg-d) | Cancer risk |
| Dermal absorption ^b | Vinyl chloride | 6.46E-06 | 3.00E-03 | 0.00 | 2.67E-06 | 7.20E-01 | 1.92E-06 |
| | Tetrachloroethylene | 4.97E-03 | 1.00E-02 | 0.50 | 2.05E-03 | 5.20E-02 | 1.07E-04 |
| | Trichloroethylene | 7.53E-04 | 9.00E-04 | 0.84 | 3.11E-04 | 7.33E-02 | 2.28E-05 |
| | 1,1-Dichloroethylene | 3.74E-04 | 9.00E-03 | 0.04 | | | |
| | 1,1,1-Trichloroethane | 3.23E-04 | 1.80E-01 | 0.00 | | | |
| | cis-1,2-Dichloroethylene | 2.11E-04 | 1.00E-02 | 0.02 | | | |
| Inhalation ^c | 1,1-Dichloroethane | 3.25E-05 | 1.00E-01 | 0.00 | | | |
| | Vinyl chloride | 1.01E-03 | 2.86E-02 | 0.04 | 4.19E-04 | 1.54E-02 | 6.45E-06 |
| | Tetrachloroethylene | 1.03E-01 | 1.71E-01 | 0.60 | 4.25E-02 | 2.00E-03 | 8.50E-05 |
| | Trichloroethylene | 4.65E-02 | 6.00E-03 | 7.75 | 1.93E-02 | 6.00E-03 | 1.16E-04 |
| | 1,1-Dichloroethylene | 2.64E-02 | 9.00E-03 | 2.93 | | | |
| | 1,1,1-Trichloroethane | 2.06E-02 | 6.29E-01 | 0.03 | | | |
| Health risk | cis-1,2-Dichloroethylene | 1.54E-02 | 1.00E-02 | 1.54 | | | |
| | 1,1-Dichloroethane | 3.11E-03 | 1.43E-01 | 0.02 | | | |
| | | | Hazard index | 14.3 | | | |
| | | | | | VC (IARC 1) | 8.4E-06 | |
| | | | | | PCE (IARC 2A) | 1.9E-04 | |
| | | | | | TCE (IARC 2A) | 1.4E-04 | |

^aHierarchy of toxicity data sources was listed in the preferred order as follows:

1. U.S. EPA Integrated Risk Information System (IRIS) online database.
2. U.S. EPA Health Effects Assessment Summary Tables (HEAST).
3. National Center for Environmental Assessment (NCEA)—provisional toxicity values developed by NCEA (U.S. EPA, 1995a, 1995b).

4. The Risk Assessment Program (RAP) has contacted Superfund and has been given provisional values that should be used for DOE-ORR projects. This value should be clearly documented as provisional. For other projects, the Superfund Health Risk Technical Support Center should be contacted directly at (513)569-7300 (ORNL, 2001).

^bThe dermal chronic RfD and dermal slope factor are derived using the methods provided in U.S. EPA (1989).

^cThe inhalation chronic RfDs are derived from the inhalation chronic RfCs. The RfDs are calculated as shown in the following equation: $RfC \text{ (mg/m}^3\text{)} \times 20 \text{ m}^3/d/70 \text{ kg} = RfD \text{ (mg/kg-d)}$. The inhalation slope factors are derived from the inhalation unit risks. The inhalation slope factors are calculated as shown in the following equation taken from *Supplemental Guidance to RAGS: Region 4 Bulletins Human Health Risk Assessment*, November 1995: unit risk $(\mu\text{g/m}^3)^{-1} \times 70 \text{ kg} \times (20 \text{ m}^3/d)^{-1} \times 1000 \mu\text{g/mg} = SFI \text{ (mg/kg-d)}^{-1}$.

^dRoute extrapolation: provisional values of inhalation RfDs for TCE, 1, 1-DCE, and cis-1,2-DCE are assumed equal to their respective oral RfDs.

TABLE 4. Input Variables and Their Corresponding Distributions Used in the Probability Risk Calculations with Monte Carlo Simulations

| Input variable | Unit | RME parameter | Best-fitted distribution | Mean | SD | Minimum | Maximum | Likeliest |
|--|-------------------|---|---|-------|-------|---------|---------|------------------|
| Site-specific parameter | | | | | | | | |
| Body weight | kg | 60.9 | Lognormal | 60.9 | 10.5 | 40.0 | 95.0 | |
| Skin surface area | m ² | 1.66 | Lognormal | 1.66 | 0.17 | 1.30 | 2.14 | |
| Exposure time (shower) | min/d | 30 | Triangular | | | 1.1 | 41.7 | 10.0 |
| Exposure duration (shower) | yr | 29 | Lognormal | 14.69 | 15.25 | 1.0 | 29.0 | |
| Exposure duration (hand exposure) | yr | 27 | Lognormal | 13.87 | 13.93 | 0 | 29 | |
| Default assumption | | | | | | | | |
| Ventilation rate | m ³ /h | 0.84 | 0.7 | | | | | (Layton, 1993) |
| Fraction of skin in contact with water | unitless | 80% (Showering) 8% (Hand exposure) | | | | | | (Assumed) |
| Exposure time (hand exposure) | min/d | 20 | | | | | | (Assumed) |
| Permeability coefficient | cm/h | Chemical-specific value | | | | | | U.S. EPA (1992b) |
| Average time | d | 70 × 365 (carcinogen) ED × 365 (noncarcinogen) | 70 × 365 (carcinogen) ED × 365 (noncarcinogen) | | | | | U.S. EPA (1989) |

acceptable cancer risk. The total organ HI also indicated a potential health hazard. These RME risk levels were considered the high-end values on which remedial decision would be based, and provided the basis for developing protective exposure levels for future use as recommended by the U.S. EPA (2001b). Moreover, average risk estimates indicated a marginally significant cancer risk. Therefore, we must examine carefully the uncertainties involved in the processes of risk assessment before making any recommendation.

From our risk assessment, individual contaminants, including PCE, TCE, 1,1-DCE, and *cis*-1,2-DCE, posed an unacceptable chronic hazard to the liver, and PCE and TCE were probable human carcinogens involving the liver as the most sensitive tumor site. The potential health risks associated with the complex mixtures of chlorinated hydrocarbons might be greater than those of a single chemical because synergistic interactions may occur when mixtures are encountered (Seed et al., 1995). Moreover, a high HBV carrier rate of 15–20% was reported in the general population of Taiwan

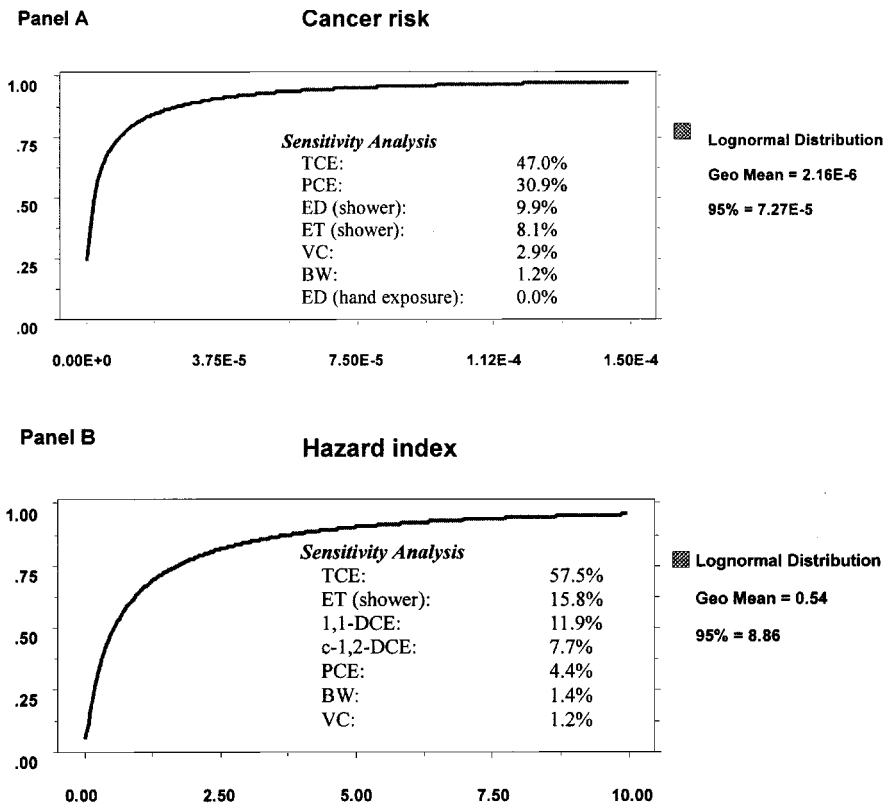


FIGURE 3. Cumulative probability distributions of health risks and associated sensitivity analysis using Monte Carlo simulations and assuming additive effects.

(Chen & Sung, 1978; Sung et al., 1984). The physical examination also disclosed a similar prevalence (13.6%) of HBV carriers among the downstream residents, who might be more susceptible to hepatic carcinogens such as aflatoxin (Wang et al., 1996). Therefore, the lack of a proper synergistic assessment model between these chlorinated solvents and chronic hepatitis B infection (Du & Wang, 1998) might further aggravate the magnitude of underestimation of risk.

Inhalation exposure from showering contributed most to health risk estimates in our study. Traditionally, ingestion of drinking water was considered a primary route of exposure to contaminated water (McKone, 1989). However, our empirical data indicated that the route of water ingestion was negligible. Moreover, research has shown that VOCs inhaled from tap water may exceed exposure via direct ingestion (McKone & Knezovich, 1991). Since we did not exhaustively include all the pathways of other indoor air exposure, such as boiling water, cooking, dishwashing, toilets, etc., the calculated health risk might be likely to be an underestimate.

As the sensitivity analysis revealed, the environmental concentrations and exposure duration for showers were the two key contributors to the total variances in the exposure modeling. It also indicated that TCE was the most important pollutant in determining health risk at this site, which could be used as an indicator for remediation. However, uncertainties associated with assumptions and scientific knowledge in our risk characterization must also be addressed. One limitation in our estimation of inhalation doses was that the K_{OLA} values for TCE and PCE were obtained from experimental data (Little, 1992), but those for the other five chemicals were assumed. We performed sensitivity analysis to assess the degree of impact from the assumption of K_{OLA} values. The results showed that the estimates of cancer risk and HI increased by less than 0.4%, and 5%, respectively, as the K_{OLA} values for the other five chemicals were all increased up to 10-fold. Therefore, the magnitude of impact from K_{OLAs} of these chemicals on health risk estimates was relatively small and it would not influence our conclusion. For dermal exposure, the exposure duration of doing laundry by hand was difficult to define because most residents could not recall exactly how much time they spent on doing laundry by hand. By performing sensitivity analysis, we found that dermal absorption from hand exposure-related activities accounted for less than 5% of cancer risk estimates even if an assumed 40-min exposure time was used. Moreover, this exposure duration contributed less than 0.1% of the total variance of cancer risk, as shown in Figure 3A. Thus, the assumed 20 min did not significantly bias the risk estimates. Due to the scope of this study, the target organ doses were not estimated in our study using physiologically based pharmacokinetic (PBPK) models (Roy et al., 1996; Bogen & Gold, 1997). The results of this study must be interpreted with some caution. Further studies are warranted to provide toxicity values based on PBPK models for any refined risk assessment.

Our results indicated that chlorinated hydrocarbons in groundwater may tend to be persistent pollution for decades due to their physical characteristics of dense nonaqueous-phase liquids (DNAPLs) (Huling & Weaver, 1991), and vinyl chloride, a more toxic and persistent chemical under anaerobic conditions, may be produced from degradation of PCE and TCE. The current levels of contaminants, especially PCE and TCE, were shown to be much higher than the water quality standards set by the U.S. EPA (1999). However, most people in the downstream community were still unaware of potential health effects of the groundwater contamination. Some of them had consistently used the contaminated groundwater for domestic water supply until 2000 because of economic incentives. Therefore, risk management is urgently needed to educate the residents, based on our risk assessment.

CONCLUSION

According to our risk assessment, increased chronic and carcinogenic risks on the liver may still be a potential public health concern for people living downstream, especially for sensitive subpopulations, such as HBV carriers. We recommend that all the community people should discontinue using the contaminated groundwater for any purpose. The environmental health advisories have to continue the medical surveillance and follow-up on the community residents. Further research will be needed to address the actual health impact on the residential population.

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