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Henry J. Schuver
USEPA

Chapter 29

ASSESSING THE PUBLIC HEALTH SIGNIFICANCE OF SUBSURFACE-CONTAMINANT VAPORS INTRUDING INTO INDOOR AIR

By Henry J. Schuver[§]
USEPA, OSW, Ariel Rios Bldg (MC-5303W), 1200 Pennsylvania Ave. NW, Washington, DC 20460

ABSTRACT

To assess the public health significance of exposures via the vapor intrusion pathway for exposure a risk assessment was conducted for a common VOC trichloroethylene (TCE) in an unbiased sample of all the contaminated sites within a large geographic setting (northern New Jersey). Probabilistic methods were used to minimize the impact of single point-estimate input values and to help assess the impact of variability and uncertainty in input parameters. Central-tendency probabilistic methods were used to provide an estimate of the most likely exposure point concentrations. The exposure assessment involved 709 TCE-contaminated groundwater sites with 29,856 groundwater samples from 11,210 monitor wells in the state's Hazsite database. The groundwater mapping component focused on the 78 sites with one or more TCE-contaminated wells located on land classified as residential. The extent of groundwater contamination beyond the monitor well locations was estimated (mapped) using generic GIS-based Inverse Distance Weighted methods on a natural-log scale and additional hypothetical 'clean wells.' The risk assessment focused on the 38 sites with one or more hypothetical residences overlying groundwater with a house-plot averaged concentration greater than 2.7 ug/L. The attenuation of vapors generated from the upper-most groundwater was estimated for the 883 hypothetical overlying residential structures using the USEPA's national empirical database of vapor attenuation factors. Receptor characteristics based on county-level statistics are used to estimate adult individual and childhood age-specific exposures using probabilistic "age at move in" techniques and with possible in-utero and lactation exposures. The exposure estimates are combined with central-tendency probabilistic estimates of toxicity (primarily based on NYDOH, 2006a) to estimate central-tendency risks for the cancer and non-cancer outcomes under study (Non-Hodgkin's Lymphoma and Central Nervous System effects). In general the

[§] Corresponding Author: Henry J. Schuver, U.S. EPA – OSW, Ariel Rios Bldg (MC-5303W), 1200 Pennsylvania Ave. NW, Washington, DC 20460, Email: 703-308-8656, Fax: 703-308-8609

*This is a personal perspective and does not represent Agency policies or positions.

risks are low and highly skewed. Only those few individuals at the highest level of exposure are estimated to be subject to risks of typical concern. However, the methods used include limitations and these results are not likely to be representative of some other areas of the country. Sensitivity and two-dimensional analyses indicate the inputs for vapor attenuation and groundwater concentration dominate the risk estimates.

1. INTRODUCTION

The recent legislative proposal ("TCE Reduction Act of 2007") reported that "exposures to volatile organic compound vapors from migration to indoor air have become a concern throughout the United States." However, the threat to public health from vapor intrusion remains largely unknown because no study has summarized the frequency and magnitude of vapor intrusion exposures and risks across a broad spectrum of contaminated sites over a large geographic area.

This study conducted an assessment of the risks possible due to vapor intrusion in a full and unbiased sample of all the contaminated sites within a large geographic area. Vapor intrusion exposures have been assessed in an increasing number of contaminated-site investigations, however, typically only at sites suspected to have a high potential for vapor intrusion. Because these 'high potential' sites investigated for vapor intrusion to date are not likely representative of all contaminated sites, the possible impact of vapor intrusion exposures on public health has not been fully assessed. The purpose of the risk assessment, to assess the public health significance of exposures via the vapor intrusion pathway, necessitated a scope broad enough to include a sufficient number of sites potentially affected by vapor intrusion from a full and unbiased sample of all the contaminated sites within a large geographic setting.

2. METHODS

This risk assessment is based on current risk literature and U.S. EPA risk assessment guidance (e.g., USEPA, 1989; 2005a; 2005b), including EPA guidance on probabilistic risk assessments (USEPA, 2001a). However, differing from the EPA's typical methods for estimating upper-bound risks, the method used here was designed to produce typically expected (i.e., central tendency) results (as has been recommended by the Office of Management and Budget (OMB, 2002, 2007)). This risk assessment uses probabilistic ('Monte Carlo') techniques (Cullen and Frey, 1999) for randomly selecting individual point-estimate input values from distributions of input values in each of multiple model iterations to obtain a distribution of individual risks. Distributions are used for both the exposure and the dose-response inputs. Models are presented describing the expected distribution for dose-response relationships and the distribution of exposures for the exposed population. These distributions are integrated into risk estimates in the probabilistic risk model using Crystal Ball software, version 7.2.1 (Decision Engineering, 2006). Probabilistic methods are used to help minimize the impact of single point-estimate input values where there are high levels of variability and uncertainty (USEPA, 2001a).

Probabilistic methods are also used to assess the impact of variability and uncertainty in risk input values on the resulting risk estimates.

This risk assessment uses the environmental evidence from groundwater investigations of contaminated sites in the state of New Jersey (NJ) that was available in electronic formats as of June 2004 (estimated to be approximately 2/3 of groundwater samples collected (Defina, 2004)).

The study area is defined by the political and hydrologic boundaries of the nine watershed management areas (WMA 01 to 09) making up the northern portion of the state of New Jersey. To focus the risk assessment and normalize the dose-response component, a single indicator compound of vapor-forming chemicals was selected (Trichloroethylene (TCE)). In summary, this quantitative risk assessment estimates typical population-wide vapor intrusion exposures and risks for a selected indicator VOC (Trichloroethylene (TCE)), which is present as a contaminant in groundwater beneath residents of northern New Jersey.

2.1 Hazard Assessment

Trichloroethylene or TCE (Cl2C=CHCl; CASRN 79-01-6) is a once widely used chlorinated-solvent VOC (USEPA, 2001b) that has a long history of animal and human studies indicating an association with various health outcomes. These outcomes include non-cancer effects to the central nervous system, liver, and kidney, as well as reproductive and developmental effects, and several cancers such as liver, kidney, lung, and testicular, as well as lymphoma and leukemia (Schiotz, 1938; Waters 1977; and NRC, 2006). The historical industrial use of TCE was substantial (e.g., 145,000,000 kg in 1994 (USEPA, 2001b)), and releases to the subsurface have resulted in TCE being the fourth most commonly detected contaminant in the nation's groundwater (Zogorski et al., 2006) as well as the substance with the most commonly completed exposure pathways at Superfund sites (45% of 1,356) (Johnson, 2002; NRC, 2006). TCE is one of a class of contaminants (chlorinated solvents) that are relatively resistant to bio-degradation and persistent in the subsurface and thus are more likely to be present through a complete vapor migration pathway into indoor air. In summary, trichloroethylene is a generally recognized hazard for its potential to increase the risk for cancer and other adverse health effects and can present a hazard via the vapor intrusion exposure pathway.

2.2 Exposure Assessment

The exposure assessment consisted of five major elements: 1) the preparation of the groundwater contaminant data, 2) the prediction of groundwater concentrations underlying specific residences, 3) the prediction of indoor air concentrations, 4) the descriptions of receptors, and 5) the calculation of average daily exposures. For this study the TCE-contaminated groundwater is the source of the TCE vapors potentially intruding into the indoor air of overlying residences and this study used analytical results (concentrations) from groundwater samples (and 26 associated parameters) that were made available in an electronic format from the state of New Jersey's Department of Environmental Protection (NJDEP) Site Remediation Program's Hazsite Database Submittal System (HDSS) (NJDEP 1999a; 1999b). The data used in this risk assessment was from groundwater samples collected over an approximately 10-year period between October 1993 and April 2003, but the majority of the

samples were collected between 2000 and 2003. The data was reviewed and preliminarily prepared for use in the risk assessment. The preparation included the removal of inappropriate or inconsistent data and the addition of fields for units-corrected result concentration values and adjusted estimates for the depths to water. The data used from Watershed Management Areas 01 to 09 for the risk assessment included 29,856 groundwater samples analyzed for TCE from 11,210 monitoring wells at 709 contaminated sites (see Figure 1).

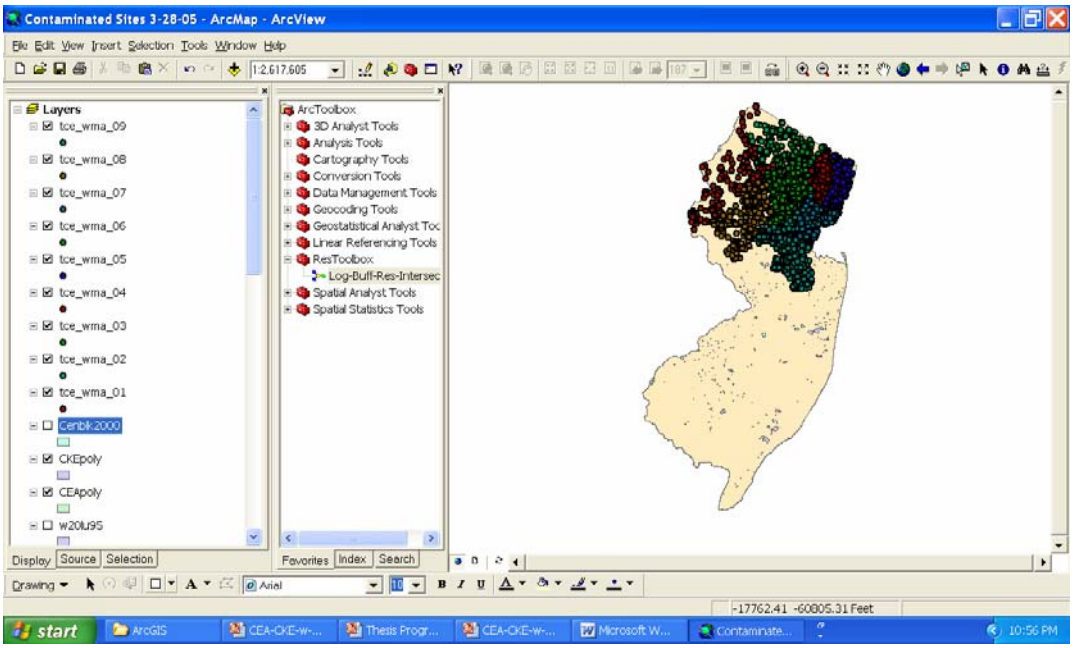

Figure 1. Map of New Jersey showing available groundwater samples indicating the location of study area (Watershed Management Areas 01 to 09)

Spatial modeling (mapping) of the groundwater concentrations was performed to estimate the concentration of TCE in the upper-most groundwater under individual hypothetical residential structures. This was completed using a GIS mapping software, specifically ArcInfo version 9.1 (ESRI, 2006). Preliminary GIS analysis identified those projects that had one or more TCE-contaminated wells located on land that was observed to be residential (NJDEP 2007a) and contained a mean concentration of TCE greater than the lower limit of concern for this study (2 ug/L). Only 78 of 709 contaminated site projects (11 %) were carried forward for prediction of the groundwater plume extent.

The spatial models used to predict of the extent and concentrations of groundwater plumes involved a number of techniques intended to provide a reasonably confident estimate of the concentration under any specific residential location, given the available information. The low number of monitor wells located on residential properties and the lack of critical site-specific parameters, such as the groundwater gradient, hydraulic conductivity, and aquifer thickness, prevented the use of more complex (site-specific) groundwater plume fate and transport models. In general, it was assumed that the contaminated groundwater plume extent was largely defined

by the existing monitor wells. However, none of these wells were expected to be located immediately beneath residential structures and thus it was necessary to estimate the concentrations and extent of contamination between and beyond the monitor wells. The concentrations of TCE in the groundwater beyond the monitor wells were estimated using a generic (non-site specific) assumption that the concentrations decreased in all directions on a log scale away from locations with documented concentrations (monitor wells). The approach used Inverse Distance Weighted (IDW) techniques (ESRI, Geostatistical Analyst) with concentrations expressed in natural-log units. Additional hypothetical 'clean wells' (with assumed zero concentration of TCE) were added to the spatial models to further bound the concentrations in the estimated up- and cross-groundwater gradient directions.

It was assumed that the general direction of groundwater flow could be approximated by the surface topography (USGS, 1983). More specifically, it was assumed that the groundwater flow direction can be approximated using the surface topography from a NJDEP produced Digital Elevation Model (DEM) with a 10 meter cell size that was based on a United States Geological Survey (USGS) data (NJDEP, 2007b). The mapping technique developed to estimate the groundwater flow direction involved the use of the "Least-Cost" raster path (aka "Rain Drop") tool (within the Spatial Analyst program, ESRI 2006) to approximate the down-gradient groundwater 'flow line' direction based on expected surface run-off. An assumed 300 ft buffer was then added to both sides of this central down-gradient flow line and hypothetical clean (0 ug/L TCE concentration) wells were added at 50 ft intervals along the 300 ft buffer lines to bound the contamination in the up- and cross-groundwater gradient directions (see Figure 2).

The approach developed to determine 'shallow' samples (which can generate vapors for possible vapor intrusion) used the minimum "START_DEPTH" (NJDEP, 1999a; 1999b) value from any sample in the entire site (or adjacent sites with wells within the area) and then added 10 feet (one typical monitor well screen-length interval) to that value to arrive at a preliminary 'shallow' limit value for the site. Histograms (Geostatistical Analyst, Data Explorer, ESRI, 2006) of both the minimum and the mean sample START_DEPTH values per well were then inspected to assess whether this preliminary 'shallow' limit value captured the population of samples most likely intended to represent the upper-most waters and also to be distinct (e.g., via an expected clean break) from samples intended to represent deeper portions of the aquifer.

In general, the shallowest histogram category (calculated using 'natural breaks' statistics (ESRI, 2006)) that included the preliminary 'shallow' limit value was used as the final 'shallow' limit value for the site. This analysis involved some hydro-geologic interpretations, particularly when there was substantial topographic relief within the area of the site's wells, included or excluded specific samples to ensure the sample population represented the quality of the upper-most water. In general, only samples with START_DEPTH values less than the final 'shallow' limit value were used to represent the upper-most groundwater (i.e., only the acceptably shallow data were exported as "shallow" files for mapping the distributions of contaminant concentrations, and depth to water).

Finally, the hypothetical homes were assumed to be located at the centroid of regularly spaced average lot-sized areas for the specific land-use density classification, as documented in the NJDEP's 1995/97 Landuse/Landcover files (NJDEP 2007a). Further, it was assumed that the

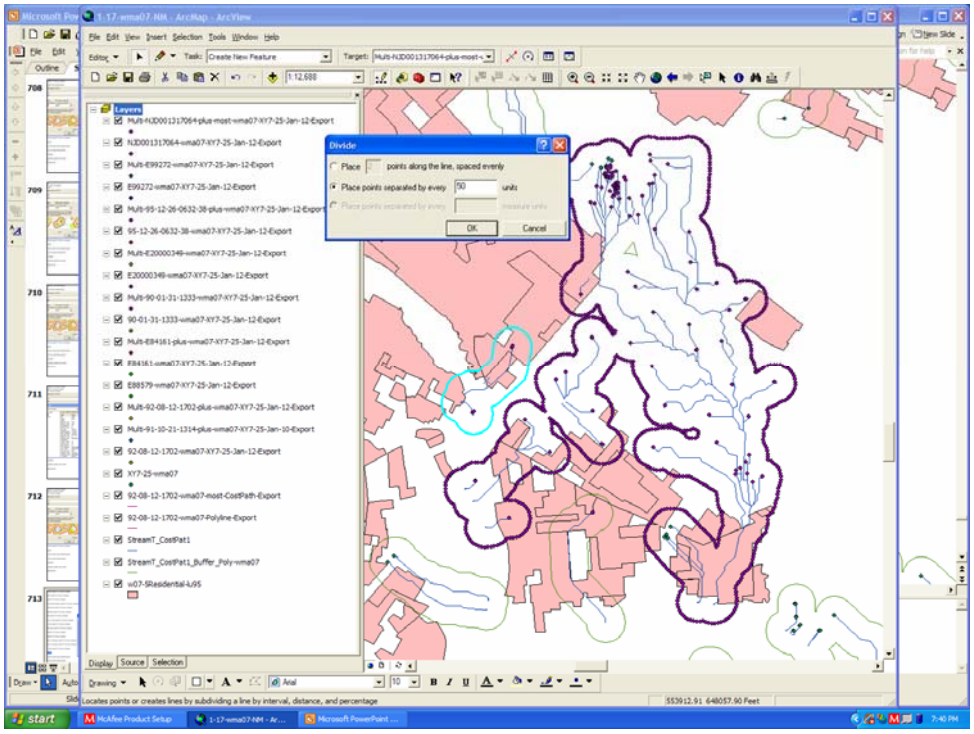

Figure 2. Hypothetical clean wells being added at 50 ft intervals along the 300 ft buffer lines surrounding monitor wells and flow lines (multiple site's wells shown here). Note buffer lines highlighted in light blue tone indicates a monitor well that is located on residential land.

groundwater concentration representing the vapor source for an overlying building could be estimated by using the mean groundwater concentration for the estimated 30 x 30 ft footprint of the home. Only projects with one or more hypothetical residential structures located at the centroid of their lot sizes that had 30 x 30 ft house plot area averaged estimated groundwater concentrations greater than one natural log unit (2.7 ug/L) (see Figure 3 below) were considered further in the risk assessment (38 of the 78 mapped projects).

The prediction of indoor air concentrations in the hypothetical residential structures overlying TCE-contaminated groundwater was based on the estimated residence-specific groundwater concentrations, the vapor partitioning coefficient (Henry's Law), the resulting estimated soil-gas concentration expected to be present immediately above the groundwater table, and a national empirical vapor attenuation data set.

Prior to being used as input into the risk calculation models, the individual predicted hypothetical residence-specific shallow groundwater concentrations (derived by the mapping above) were first depth-adjusted. Predicted residence-specific groundwater concentrations where the depths to water were estimated to be < 5 ft below ground surface (bgs) were adjusted to account for the lower attenuation expected due to the shorter distance for attenuation and likelihood that the contaminated groundwater could be in direct contact with building materials ('wet basement' scenario; USEPA, 2002a). Approximately 40% of the building-specific

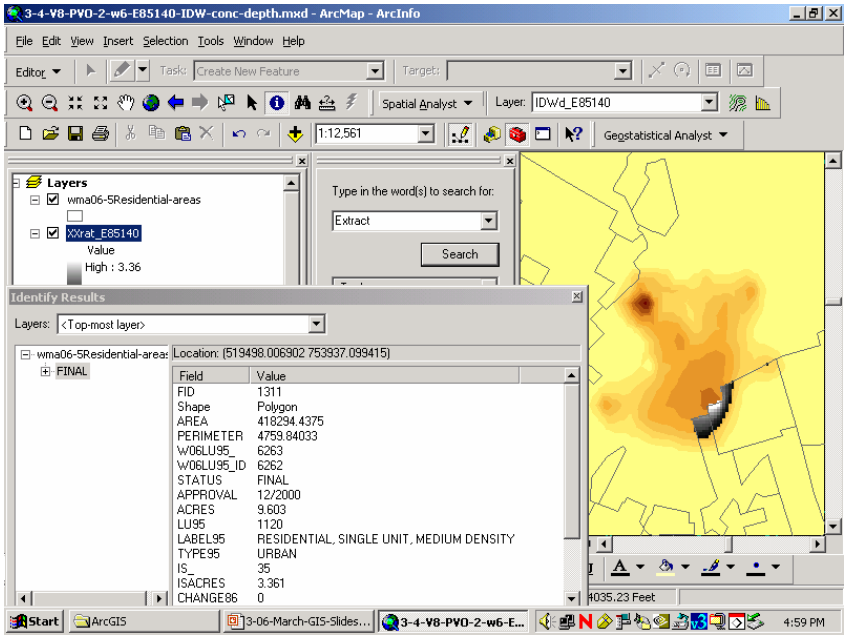

Figure 3a. Predicted groundwater concentrations and impacted residential land (showing concentrations for all 30 x 30 ft potential building plots (concentrations up to 3.36 ln)).

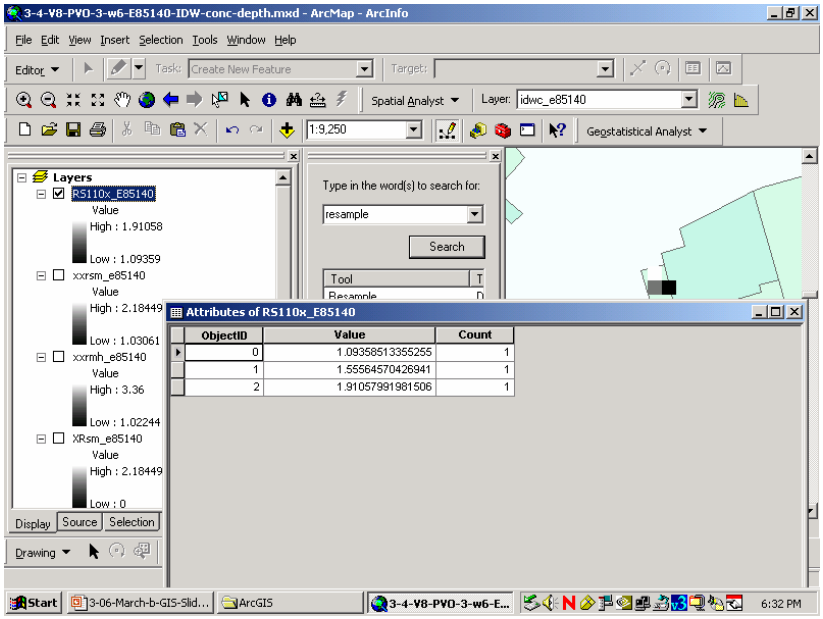

Figure 3b. Re-sampling of all potential building plots resulted in three lots with residential building-specific shallow groundwater concentrations greater than one log resulting from the placement of the (30 x 30 ft) building sites at the centroid of the applicable lot size (here 110 ft by 110 ft).

groundwater estimates were found to be shallow (i.e., <5 ft bgs) and their concentrations were multiplied by a factor of 3.3x, and the 13% of the building-specific groundwater estimates that

were found to be very shallow (<3.3 ft bgs) and were multiplied by an additional 3.0x factor. This approach provided location-specific adjustments for the expected reduction in attenuation from shallow sources where direct contact with building materials is possible.

The distribution of predicted groundwater concentrations, including the values that were depth-adjusted, were then fit to a continuous distribution using Crystal Ball software with Chi-Square fitting criteria. Randomly selected values from the best-fit distribution of (depth-adjusted) groundwater concentrations were the initial and primary input into the calculation of probabilistic estimates of the indoor air concentration of TCE due to vapor intrusion.

The concentration of TCE in the soil-gas immediately above the contaminated groundwater (vapor source) was estimated using a unit-less Henry's Law partitioning coefficient for an average shallow groundwater temperature for northern New Jersey of approximately 52 degrees F (11.1 degrees Celsius) (USEPA, 2004a). The unit-less Henry's Law partitioning coefficient for this temperature has a value of 0.216 using the Office of Solid Waste and Emergency Response (OSWER) method (USEPA, 2004a). TCE dissolved in the upper-most portions of the groundwater was assumed to fully partition at the predicted Henry's Law equilibrium level (as is needed to be consistent with the practice used in calculating the empirical attenuation factors) and has been observed in the field (Wertz, 2006; MADEP, 2000). Each hypothetical residence-specific groundwater concentration estimate was multiplied by the Henry's Law partitioning coefficient for TCE (and a unit conversion of 1000 liters/m^3) to estimate the concentration TCE vapors (in ug/m^3) immediately above the upper-most groundwater underlying each residential structure.

The reduction (attenuation) of vapor concentrations between the vapor source (e. g., concentration in soil gas immediately above the groundwater) and the indoor air has been described by an attenuation "factor" (Johnson and Ettinger, 1991) which is defined as the ratio of concentrations in indoor air to the subsurface vapor source (immediately above the groundwater). For ease of communication, and to allow whole numbers to be entered in the risk software, for this study an attenuation 'value' was defined as the ratio of the subsurface vapor source concentration divided by the indoor air concentration (i.e., 1/attenuation factor). This nomenclature allows high amounts of attenuation to be represented by high attenuation 'values' and low amounts of attenuation to be represented by low numerical attenuation 'values.' This terminology is similar to the "dilution factor" term used by others (ITRC, 2007).

The distribution of expected groundwater to indoor air attenuation values is based on empirical data collated by the USEPA. Over the last six years the USEPA has been building a national database of paired data including measured groundwater concentrations and measured indoor air concentrations that were collected under the authority of various federal and state regulatory authorities (Dawson, Hers, and Truesdale, 2007). While many variables can influence the attenuation values observed for an individual building and paired data set, the overall distribution of observed attenuation values from groundwater has remained somewhat stable over the last few years with a median groundwater attenuation 'value' of approximately 10,000 (attenuation factor of 1/10,000). For this study the distribution of national empirical attenuation values was filtered to include only those attenuation values where the vapor source (soil-gas) concentration term was greater than 100 times the 90th percentile of recent literature indoor air "background" values (i.e., 0.5 ug/m^3; Dawson, 2007). This distribution was assumed to

approximately represent the range and distribution of possible attenuation of intruding vapors that could be expected in northern New Jersey. A best-fit curve for the distribution of observed attenuation factors in the USEPA vapor intrusion database (from 1,020 sample pairs) was used in the Crystal Ball models. The curve representing the distribution of observed attenuation values was log-normal with a mean value of 51,648, a minimum attenuation value of 23.5, and a maximum attenuation of 2,080,000. In summary, the calculation divided the vapor concentration at the source (i.e., in the soil-gas immediately above the groundwater table) by the randomly selected attenuation 'value' to estimate the indoor air concentration.

The population occupying the residential structures overlying the TCE contaminated groundwater plumes was expected to be similar to the rest of the population of these counties and of New Jersey. Current statistics for populations in the northern counties of New Jersey, and state-wide, show approximately 2.7 persons per household (US Census, 2004). For the purpose of this risk assessment there was expected to be one adult male and one adult female occupant and 0.7 children (under 18) per household/residence. Given the exposure assessment identified 883 hypothetical residences expected to overlie shallow TCE-contaminated groundwater and the average occupancy rate (2.7 persons / residence), the population of potential receptors would be approximately 2,384 persons at a given point in time.

Given that people move in and out of homes with a median of every nine years (USEPA, 1997) and the 30 year period of this study (1985-2015), it can be expected that approximately 3.3 (30/9) different families/groups would occupy each of the homes over the 30 year period of this study. The total potential receptor population could be expected to be approximately 7,867 (3.3 x 2,384) persons.

State-wide and northern-county New Jersey statistics show approximately 75% (approximately 2/2.7) of the population is over age 18, and 25% (approximately 0.7/2.7) is under 18 (US Census, 2004). Thus, of the 7867 expected occupants over the 30 year period, approximately 5900 are adults and approximately 2000 are children. Given the northern-county approximate average of 14 live births per 1000 population per year (over the period from 1982-2002; NJDHSS, 2002) it can be estimated that approximately 1000 children would be born to residents in these homes over the 30 year study period (14 live births/1000/yr x 2,384 residents at any year = 33 births/yr x 30 yrs = approximately 1000 births from parents in residences over plumes). Assuming that approximately one-half of the children expected to be born in these residences are not already included in the population estimates, it is possible that another 500 children could be born and raised (for some period of time) in these residences (for a possible total of approximately 2500 children residents over the 30 year period of the study).

The calculation of average daily exposures for occupants of the hypothetical residential structures overlying TCE-contaminated groundwater was based on the estimated indoor air concentrations due to vapor intrusion, the exposure duration, exposure frequency, and exposure time, as well as the appropriate averaging time for the potential outcomes. The calculation of average daily exposure was performed similarly in each of the two adult risk models: adult-cancer, and adult-non-cancer (see Appendix 1 and 2). The calculations for the child-cancer and child-non-cancer models were somewhat different to account for child-specific factors and the age-specific time of exposure (see Appendix 3 and 4). The calculations for adults will be discussed first.

The general equation for characterizing inhalation exposure, when using either units of Reference Concentrations (RfC) for non-cancer risks or Inhalation Unit Risks (IUR) for cancer risks, is presented below, as modified after USEPA (1994):

$$I = \frac{C_{vi} \times EF \times ED}{AT}$$

Where:

I = effective long-term avg. daily exposure (ug/m^3)

C_{vi} = concentration of contaminant in indoor air due to VI (ug/m^3)

EF = exposure frequency (in 24hr-days/yr) spent inside residence

ED = exposure duration (yrs) residing at that location

AT = averaging time (days)

The predicted concentration of TCE contaminants in residential indoor air from vapor intrusion (C_{vi}) is derived from procedures described above (and shown in Appendix 1 and 2 on row 6).

For this probabilistic assessment the exposure frequency (EF) normally expressed in terms of days/yr spent at residence, and exposure times (ET) normally expressed in hrs/day actually spent in the residential structure, were combined and expressed as a single population of exposure frequency (EF in 24-hr-days/yr). This is equivalent to the number of full 24-hr-days actually spent inside the residence per year. This was done to improve the accuracy of the probabilistic calculations using distributions of exposures.

The distribution of 24-hr-day exposure frequencies for adults was modeled with a triangular distribution with 240 24-hr-days/yr as the most likely value. This 24-hr-days/yr value represents mean residential indoor air exposures of 948 minutes (15.8 hrs) per day (USEPA, 1997) for 365 days per year. The distribution's minimum exposure frequency value was from the fifth percentile of time in residence (equivalent to 137 24-hr-days/yr) and the maximum value for all adult age groups (>18 years old) was equivalent to 365 24-hr-days/yr) from EPA (USEPA, 1997).

The distribution of exposure duration (ED) values for the duration of time (years) that adults would reside in a specific residence overlying the contaminated groundwater (ED in years are based on USEPA guidance (1997) and literature values. The distribution of Exposure Durations (residence times) was modeled with a triangular distribution having nine (9) years as the most likely value and an assumed practical minimum (adult) value of one year in residence and maximum of 40 years.

The exposure averaging times (AT in years) for cancer effects in adults were presented as single point estimates for this risk assessment (70 years; USEPA, 1997). The averaging time for non-cancer effects in adults is based on the individual's (randomly selected) exposure duration.

For children, the derivation of exposure durations and frequencies were similar but more complicated than for adults (see Appendix 3 and 4). The exposure duration values used for children (ED in years) were similar to those for adults; however, they included a possible shorter minimum period (min. 0.1 years) as these short exposures may still be relevant for children, and the child's distribution was capped at maximum of 18 years (USEPA, 2002a). Additionally, because children could be of any age at the time the family moves into the home, could be born any time within the family residence duration and/or could attain the age of 18 (i.e., no longer be expected to be at home) prior to the family moving out, the models needed to account for the actual age of the child at 'move in' and the time of birth within the family residence duration. The child's age at 'move in' was designed to be randomly selected from a uniform distribution with ranges up to 18 years at the time of family 'move in' and to be as young as being born up to 10 years after the family 'moves in' (see Appendix 3 and 4, rows 8 to 11). The calculations in Appendix 3 and 4, rows 9 to 11, calculate the total child residence duration. The calculations in rows 12 and 13 provide the individual child's exposure durations within the specific 0-2 years of age, and 2-18 years of age, periods. In summary, the individual children's exposure durations, including that of newborn infants, were modeled using a probabilistic structure so that children could be born up to 10 years after family move-in.

For children, the USEPA reports the central tendency estimate (median) of exposure time (in hours per day) spent in the residential structure is 21 hours per day for ages 0-4 (years) and approximately 16.4 hours per day (approximately the same as adults) for aged 5-17 (EPA, 2002a, Table 9-41; and 1997, Table. 15-131). For this assessment, children 0-2 yrs of age were estimated to have a central tendency exposure time of 21 hours per day which relates to an approximate central tendency exposure frequency of 300 24-hr-days per year. The distribution of exposure frequencies for children aged 0-2 was assumed to be triangular with a minimum of 150, a most likely value of 300, and a maximum of 365 24hr-days per year.

For children aged 3-18 the exposure times for children ages 0-4 (21 hr/day) and ages 4-18 (16.4 hrs/day) are combined with the exposure frequency value for children aged years 7-18 of 240 24-hr-days/yr (i.e., the same as adults). This resulted in a combined estimate of 268 24hr-days per year for children of ages 3-18 years. The distribution of exposure frequencies for children was modeled with a triangular distribution having 268 24-hr-days/yr as the most likely value and the minimum value of the fifth percentile of time in residence (148 24-hr-days/yr) and maximum of the 95% for all age groups <18 years old (365 24-hr-days/yr) as the minimum and maximum values (EPA, 2002a; Table 9-41).

For children the exposure averaging times for cancer effects (during childhood) were presented as single point estimates for the entire childhood of 18 years (Appendix 3, row 35). The averaging time for non-cancer effects in children is equal to the individual's (randomly selected) exposure duration (Appendix 3, row 31).

In summary, the exposure calculations for each of these receptors and appropriate exposure factors are performed within the probabilistic Crystal Ball risk calculator software. The model structures and formula are described above, shown in detail in Appendices 3 and 4.

Several versions of childhood age-adjusted exposures were also calculated based on the growing volume of research on and evidence for the potential heightened effects for exposure

during critical periods of growth and development (e.g., USEPA, 2005b, 2006; Barton, et al., 2005). The age-period-specific adjustments to exposure are used as surrogates for possible differential response (toxico-kinetic and -dynamic effects) due to exposures during critical periods of a child's development.

For cancer risks to children, the use of the single point estimate exposure averaging period of 18 years, discussed above, established an effective minimum age-adjustment factor of approximately 3.3 (18/70) relative to adult cancer risks for all cancer risks to children (i.e., ages $\leq$ 18 yrs). This is generally consistent with EPA guidance for exposures to children up to 16 years of age, for exposures to chemicals acting through a mutagenic mode of action (USEPA, 2005b). Note, while TCE is not commonly considered to act through a mutagenic mode of action, the USEPA has only recently proposed a definition of what is meant by a mutagenic mode of action (USEPA, 2007), and TCE is known to have multiple complex and not fully defined modes of action (Chiu et al., 2006) particularly for NHL (NYSDOH, 2006a). Additionally, recent evidence indicates cancers (particularly childhood cancers) may be influenced by the interaction of genetic and epigenetic processes such as "alterations in gene expression, DNA repair, cell cycle control, genome stability and genome reprogramming" (Preston, 2006). Barton et al. (2005) report a "reasonable expectation that children are more susceptible to some carcinogenic agents than adults" and a geometric mean ratio of early-life to adult cancer potencies of 3.4 for lifetime exposures for six chemicals known to act through a non-mutagenic mode of action. There have been some observed associations that indicate a heightened cancer response in children can occur for exposures to chemicals outside of those typically considered to have a mutagenic mode of action, e.g., for VOCs such as TCE (e.g., Cohn et al. 1994; Costas, Knorr, and Condon, 2002; Infante-Rivard et al. 2005) as well as a still incomplete, but suggestive, study (ATSDR, 2003) where additional and possibly continuing exposures due to vapor intrusion have only recently recognized (ATSDR, 2007). Additionally, this 3.3x factor can be considered to compensate for the use of the IUR as the toxicity metric, because the IUR values do not consider the body weight or breathing rate of children. Recent studies have indicated an approximate two-fold higher dose for Category 3 gases in the pulmonary region for children (Ginsberg, Foos, and Firestone, 2005), and the "peak concentration of an inhaled VOC depends greatly on cardiac output and ventilation rate" (Bushnell et al., 2005). Finally, this 18 yr averaging-period approach helps avoids the discontinuities possible when randomly selected exposure durations of < 10% of 70 yrs (e.g., 6 yrs) would otherwise indicate the use of sub-chronic exposure assessment techniques for those individuals.

For cancer effects in children four versions of age adjustments for exposures for children aged 0-2 yrs were calculated. The risk without additional age adjustment (i.e., assuming no more heightened risk than was already included in the 18 yr averaging time (a 3.3x factor)) was considered the baseline. In addition, three types of adjustments to the exposures during the first two years of life were made. The first version of age-adjusted exposure, termed "1x+," adds a distribution around the most likely value of 1.0. The distribution has a minimum of 0.9, a maximum of 3.0 and a most likely value of 1.0. The second version of age-adjusted exposure, termed "3.0x," uses a single point estimate multiplier of 3.0 for all exposures in the 0-2 age period (a value that is consistent with USEPA's (2005b) total 10x adjustment). The third version of age-adjustment for child cancer, termed "3.0+x," uses the same 3.0 point estimate multiplier for exposures during the 0-2 age period, but also adds the possibility of additional exposures bio-

transferred from the mother, if she was exposed more than one year prior to birth and she breast-fed the child (Appendix 3 and 4, rows 24-25).

Unlike cancer risks, for non-cancer risks to children no inherent child age-adjustment factor was included in the baseline case. However, a variety of non-cancer effects, including neurological, have been found to be elevated or unique to children such as *in utero* exposures and should be considered when using typical adult-animal or adult-human based toxicological reference concentrations values (e.g., ATSDR, 1999; White et al., 1997). Thus, in addition to the baseline model without any age adjustment (i.e., assuming no heightened risk), four additional types of adjustments to the exposures during childhood were made. A quantitatively-minor multiplicative age adjustment (i.e., 1.25) was assumed possible for children 2-18 yrs (versus exposures to adults), a higher adjustment (i.e., 1.5) was assumed appropriate for exposures in the 0-2 age period, and an even higher adjustment (i.e., 1.75) was assumed to be appropriate for exposure during the *in utero* period (age -3/4 to 0 yrs). Finally, exposures possibly bio-transferred from the mother, if she was exposed more than 1 year prior to birth and she breast-fed the child, was assumed to be an additional factor of 1.5 times (Appendix 3 and 4, rows 17-19).

In summary, a variety of age-adjustment factors were explored in this risk assessment. These ranged from the typically assumed default value of 1 (i.e., assuming no effect due to age at exposure) to various distributions of values at different ages as well as the possibility of pre-natal exposures and the bio-transfer of contaminants in mother's milk.

2.3 Dose-Response Assessment

Over the last 40+ years there has been a substantial amount of research on the dose-response relationships for cancer and non-cancer effects associated with TCE exposures. Much of this work has been summarized and reviewed since 2000, including a 'state of the science' TCE-dedicated issue of *Environmental Health Perspectives* in 2000 (Scott and Cogliano, 2000), a draft TCE risk assessment by the USEPA (2001c), a review by the USEPA's Science Advisory Board (USEPA, 2002c), a summary of the recent evidence by the USEPA (2004a, 2005c), a TCE-dedicated mini-monograph (Chiu et al., 2006), a review of the critical issues by the National Academy of Sciences (NRC, 2006), and updated toxicity values by NYSDOH (2006a), as well as a number of other newly published studies, summary reviews, and meta-analyses.

This quantitative risk assessment focused on two specific health outcomes: those believed to have a sufficient number of consistent observations of an association with TCE inhalation exposures and those where low-level environmental exposures may be relevant to the public's health. This risk assessment considered one cancer, Non-Hodgkin's Lymphoma (NHL), and one non-cancer, Central Nervous System (CNS), health outcome.

The results of the dose-response assessment are represented as distributions in the probabilistic models. For both cancer and non-cancer effects this relationship is represented in the probabilistic model using a triangular frequency distribution with the central tendency risk as the most likely value, the lower-bound confidence interval (CI) values as the estimated 2.5 percentile or minimum response value, and the upper CI as the estimated 97.5 percentile value or maximum response value.

For non-cancer effects, TCE has been long recognized to interrupt the transmission of nerve/pain signals in the human central nervous system, and TCE was introduced as a narcotic in 1911 and was being widely used for surgical anesthesia by the 1950s (Waters, Gerstner, and Huff, 1977). While the exact mechanisms of action were not fully understood, TCE was used an anesthetic until as recently as the 1970s, when awareness of some of the undesirable side effects of these very high-level TCE exposures (approximately 2,000 ppm) grew (Waters, Gerstner, and Huff, 1977).

TCE is also recognized for central nervous system (CNS) effects in humans at much lower concentrations than used for anesthesia, albeit typically with longer exposure durations. The ATSDR (1997) has summarized the observations of a variety of CNS effects in humans associated with occupational-level TCE exposures (e.g., low 100s ppm), including headaches, drowsiness, confusion, dizziness, nausea, loss of facial sensation, nerve damage, and reduced scores on a variety of neurological function tests. A number of community-based studies have also documented some associations with CNS effects in environmental TCE-exposed populations (ATSDR, 1999, White et al., 1997). Research on possible impacts of low concentrations on public health continues (Barton and Clewell, 2000) and has focused on bio-chemical markers of neurotoxicity (Bushnell, et al., 2005).

This dose-response assessment has focused on two well-conducted studies of animals exposed to low levels of TCE in air showing a variety of CNS effects that, while subtle, may also be important for public health. First, Arito, et al. (1994) reported statistically significant associations for decreased wakefulness and heart rate. Using continuous polygraph recordings, Arito, et al. (1994) found that "exposure to all levels of TCE resulted in a statistically significant, dose-related decrease in the amount of time spent in wakefulness ($p<0.01$) during the 8-hour exposure period. Rats exposed to 50 ppm or higher [i.e., all doses] also had statistically significant decreases in time averaged heart rates during stages of wakefulness ($p<0.05$), slow wave sleep ($p<0.01$) and paradoxical sleep ($p<0.01$) during the 22-hour post exposure period" (NYDOH, 2006a). The NYDOH suggested that this could be due to "TCE-induced disruption of wakefulness and its circadian rhythm" (NYDOH 2006a). Arito et al. (1994) also observed at higher doses spontaneous bradyarrhythmia epidsodes in older (20-26 months) rats. Second, Briving et al. (1986) observed statistically significant associations for biochemical changes in the brains of gerbils (as did Haglid et al. (1981) and Kyrklund et al. (1984)). Specifically, Briving et al. (1986) documented significant changes in the amounts of brain proteins (glutamate and GABA [*gamma*-amino butyric acid], as well as GSH [Growth Stimulating Hormone] at higher doses) that are believed to be indicators of neuronal damage.

The NYSDOH (2006a) has provided the most recent quantitative analysis of both animal and worker evidence for CNS effects associated with TCE inhalation exposures. After consideration of a wide variety of both animal and human evidence, the NYSDOH considered a human-based occupational study (Rasmussen, Arlien-Soborg, and Sabroe, 1993) to best represent the high toxicity end of the TCE exposure to CNS response relationship for humans. In summary, Rasmussen, Arlien-Soborg, and Sabroe, (1993) found 33/99 (33%) metal degreasers primarily using TCE having at least one abnormal motor coordination score and the mean number of abnormal coordination tests (six tests) illustrating a dose-related trend. Clinical evidence of cranial nerve dysfunction was also found in the more highly exposed workers of that study. The abnormal motor coordination tests from that study and the physiologically-based pharmo-kinetic

(PBPK) model derived Human Equivalent Concentrations (HEC) calculated by NY State (NYSDOH, 2006a) were used to develop NYDOH's high-toxicity low-response percentile (RfC) value of 11 ug/m^3.

For non-cancer effects this assessment used a Reference Concentration (RfC) metric (risk per ug/m^3 of TCE in air) and assumed a triangular distribution for frequency of risks with an estimated upper-bound risk (minimum RfC concentration level) of 11 ug/m^3 as the point estimate for subtle CNS effects in a low percent of the general population resulting from inhalation exposures to TCE (NYSDOH, 2006a). The estimated central tendency RfC value, general residential population was estimated to be 74 ug/m^3 and the maximum RfC value estimated to produce perhaps subtle, but nonetheless observable, CNS effects in a majority of the general population was estimated to be 110 ug/m^3.

For cancer effects, and in contrast to the long history of non-cancer effects, the data base of observations for TCE exposures and lymphoid cancers (such as NHL, Hodgkin's disease, multiple myeloma, and leukemia) has begun relatively recently, has been increasing rapidly, and some of the highest quality human observations have been completed only very recently. Observations for NHL were addressed in recent quantitative analyses and/or summary reviews by Wartenberg, Reyner, and Scott (2000), USEPA (2001c), USEPA's Science Advisory Board (2002b), Hansen, et al. (2001), Raaschou-Nielsen et al., 2003, Kelsh, et al. (2005), USEPA(2005c), Chiu et al., (2006), Scott and Chiu (2006), Mandel et al. (2006), and NYSDOH (2006a), but was not addressed by the NRC (2006).

In the most recently released quantitative toxicity value assessment of available occupational epidemiology studies, the State of New York's Dept. of Health chose to use the upper 95% confidence interval for NHL risk from the Hansen et al., (2001) study, with support from the exposure data from the Raaschou-Nielsen et al., (2002) study, to develop a potential lifetime air concentration criterion for NHL (NYSDOH, 2006a; Table 5-18). The lifetime air concentration value correlating to a 10^{-6} risk for upper-bound risks was reported to be 0.29 ug/m^3, which relates to an Inhalation Unit Risk (IUR) value of 3.5 x 10^{-6} risk per ug/m^3 (NYSDOH, 2006a, Table 5-18). The NYDOH reported a central estimate for the IUR of 1.3 x 10^{-6} based on mean risks for the middle category of exposure durations in the Hansen et al. (1980) study.

After a review of available animal and human studies, the State of New York's Dept. of Health chose to use the upper 95% confidence interval of a statistically significantly elevated risk of malignant lymphoma from a mouse inhalation study (Henschler et al. 1980) to develop its final air criteria for Non-Hodgkin's Lymphoma (NHL) (NYSDOH, 2006a; Table 5-39). The (non-age adjusted) Lifetime Average Daily Exposure (LADE) air concentration correlating to a 1.0 x 10^{-6} lifetime incremental increase in cancer risk for humans was reported as 0.3 ug/m^3 (NYSDOH, 2006a, Table 5-38) which relates to an Inhalation Unit Risk (IUR) value of approximately 3.5 x 10^{-6} risk per ug/m^3 of TCE. Note that this result is nearly identical to the value derived from the human-based studies discussed above.

This risk assessment used an Inhalation Unit Risk (IUR) metric (risk per ug/m^3 of TCE in air) with an assumed triangular distribution for the frequency of risks with an upper-bound value of 3.5 x 10^{-6} and the central tendency value of 1.3 x 10^{-6} developed by the State of New York based on human evidence (NYDOH, 2006a). A linear extrapolation from the NYDOH central

tendency risk estimate (1.3×10^{-6}) was made to estimate an approximate lower bound risk level of 4.7×10^{-7} per ug/m^3 of TCE.

2.4 Risk Characterization

The adult cancer calculations (model) will be described as an example. Appendix 1 illustrates the structure, example central tendency input values, and formula for calculating the adult cancer risks. The calculations for adult cancer risk begins with the lifetime average concentration, as described above, which is then multiplied by the randomly selected IUR value to produce the incremental lifetime cancer risk due to the vapor intrusion of TCE for the hypothetical individual person represented by the individual trial run. This process is repeated for a total of 5,900 times to produce a distribution of individual risks to the population of adult residents of buildings overlying these groundwater plumes. The model for adult non-cancer (CNS) risk is similar (Appendix 2), except that the average daily exposures are divided by a randomly selected Reference Concentration (RfC) value and the randomly selected exposure-averaging period. The children's risk models are similar to the adult models in that they use an average daily exposure level, although they also included, as described above, child-specific exposure durations in the home and a variety of age-adjustment factors (see Appendices 3 and 4).

3. RESULTS

The results of the GIS-based mapping of the estimated extent and concentrations of TCE-contaminated groundwater indicate that approximately one-half (38/78) of the 78 mapped sites, with one or more TCE-contaminated wells located on land that was classified as residential, have predicted concentrations of concern (> 2.7 ug/L) located under one or more of the hypothetical residential structures. Only these 38 sites were considered further in this risk assessment. The total area of mapped contaminated groundwater plumes (with concentrations > 2.7 ug/L) from these 38 sites was estimated to underlie 883 hypothetical residential structures. The groundwater concentrations estimated to underlie the 883 individual hypothetical residential structures (including depth-adjustments) were generally relatively low (median of 5.9 and a mean of 19.5 ug/L (ppb)). These concentrations were represented in the models by a highly skewed best-fit log-normal distribution with a mean of 19.5 and a standard deviation of 61.09.

The mean predicted indoor air concentrations of TCE due to vapor intrusion (Cvi) in the four primary models (adult and child cancer and non-cancer) ranged from 1.62 to 1.75 ug/m^3, and the median values for all models are 0.13 ug/m^3. The estimated average daily exposures for the receptor populations are presented as separate distributions for adults and children and for cancer and non-cancer outcomes and are summarized in Table 1 below.

In summary, these results show the adult and child average daily exposures are generally low and highly skewed, with a few individuals with higher levels of exposure. Additionally, in general, the average daily exposures for non-cancer effects are two to four times higher than

those for cancer effects which is expected to be largely due to the differences in the averaging periods (which included only the actual exposure period for non-cancer effects).

Table 1. Average Daily Exposures (ug/m^3)

Receptor	Cancer		Non-Cancer	
	Mean	Median	Mean	Median
Adult	0.28	0.02	1.15	0.08
Child	0.56	0.04	1.35	0.09
Child age-adj.	0.69	0.04	2.76	0.18

3.1 Cancer risk

Probabilistic estimates of the risks for NHL for the estimated 5900 adult residents of the 883 hypothetical residences overlying TCE-contaminated waters over the 30 years of this study are presented in Table 2 below. In general, the risks are low and highly skewed. The median risk is approximately 3 x 10-8, and the mean risk is approximately 5 x 10-7. The maximum individual risk was estimated to be approximately 1 x 10-4 risk. Only individuals at the highest percentiles of exposure (>90%) are subject to risks in the vicinity of levels of typical concern (i.e., at least 10-6 levels).

Table 2. Cancer Risk

Receptor	Median risk	Mean risk	Max. risk
Adult	3×10^{-8}	5×10^{-7}	1×10^{-4}
Child 1.0x	6.3×10^{-8}	1×10^{-6}	2.1×10^{-4}
Child 1+x	6.5×10^{-8}	1.1×10^{-6}	2.1×10^{-4}
Child 3.0x	7.3×10^{-8}	1.2×10^{-6}	2.2×10^{-4}
Child 3.0x+	7.5×10^{-8}	1.3×10^{-6}	2.2×10^{-4}

Probabilistic estimates of the risks for NHL for the estimated 2500 child residents of the 883 hypothetical residences overlying TCE-contaminated waters over the 30 years of this study for various age-adjustment scenarios are also presented in Table 2 above. Figure 2 shows the results for both Adults and Children (under the four age-adjustment scenarios).

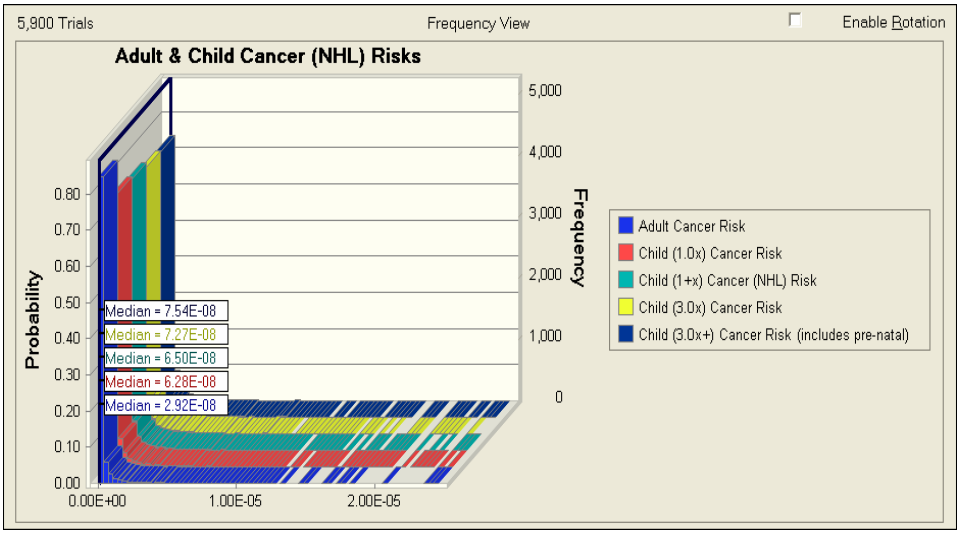

Figure 3. Overlay (frequency) chart for Adult and Child age-adjusted Cancer (NHL) risks.

3.2 Non-Cancer risk

Probabilistic estimates of the risks for CNS effects for the estimated 5900 adult residents of the 883 hypothetical residences overlying TCE-contaminated waters over the 30 years of this study are presented in Table 3 below. The non-cancer risks have a median value of approximately 0.001 and a mean risk of approximately 0.02. The maximum individual risk modeled has a Hazard Quotient (HQ) of 7.4. These non-cancer risks, which are typically compared to a HQ of 1.0, are mostly very low. Only adult residents at the highest percentiles of exposure (e.g., >>90%) are subject to risks above levels of concern (i.e., HQ > 1.0).

Table 3. Non-Cancer Risk

Receptor	Median risk	Mean risk	Max. risk
Adult	0.0013	0.02	7.4
Child (un-adj.)	0.0015	0.022	6.6
Child (age-adj.)	0.0028	0.052	30.8

Probabilistic estimates of the risks for CNS effects in the estimated 2500 child residents of the 883 hypothetical residences over the 30 years of this study are also presented in Table 3 above. When no age adjustments are considered, i.e., assuming that exposures during childhood (age period) had no higher effect on children than on adults, the median risk is approximately 0.0015, and the mean risk is 0.022. Only child residents at the highest percentiles of exposure

(i.e., >>90%) are subject to risks near levels of concern (e.g., 90% risks have a HQ of 0.03) and the maximum modeled risk has an HQ of 6.6.

The non-cancer risks to children when the age of exposures (age adjustment) is taken into account are presented in Table 3 above. The risks, considering various childhood periods (see Table 3), have a median value of approximately 0.003, and the mean risk is approximately 0.05. Only children residents at the higher percentiles of exposure (i.e., >>90%) are subject to risks in the vicinity of levels of concern (90% exposures have an HQ of only approximately 0.07), and the maximum risk has an HQ of approximately 31.0.

To compare the frequency and distribution of non-cancer (CNS) risks for both adults and children (under the two versions of childhood age adjustment), these risks are presented together in Figure 4. From this figure it is possible to see the gradual transition to slightly higher risks (and lower bar height for near-zero risks) beginning with adults and then for children without, and with, age adjustment. The median non-cancer risks (HQ) for all three receptors are in the low 10^{-3} range. In summary, the non-cancer risks to both adult and child receptors are generally low and highly skewed. Only individuals at the highest percentiles of exposure (>90%) are subject to risks above levels of typical concern (i.e., hazard indices of >1.0).

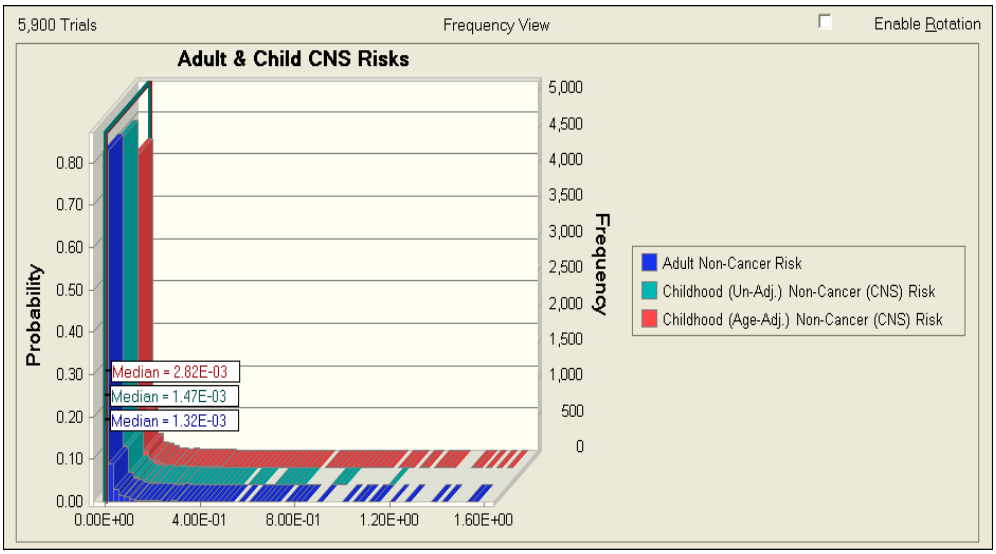

Figure 4. Overlay (frequency) chart for Adult and Child age-adjusted Non-Cancer (CNS) risks.

3.3 Secondary Analyses

Further analysis of the role of the inputs (assumptions) on the results was performed to better understand the leading causes of these risks and their distributions. These secondary analyses included sensitivity analyses and two-dimensional analyses.

Sensitivity analyses assess the impact of the input variables in the context of the sensitivity of the specific model structure and for the range of inputs tested. The "Sensitivity" analysis for

cancer (NHL) risks to adults shows that when all the inputs are allowed to vary, the risks are dominated by only two input variables: the attenuation of vapors value (-54% of the variance in risks) and the concentration of contaminant in water (36% of the variance in risks). These inputs are followed by the much smaller effects of the exposure duration (7%), the Inhalation Unit Risk (IUR) toxicity factor (2.5%), and the exposure frequency (0.5%). These values are summarized in Table 4 below. The results from another form of sensitivity analysis (one-at-a-time) also shows a very similar rank order and magnitude of effects of these same inputs (i.e., with attenuation and concentration having the most influence) for both adult and child cancer risks.

Table 4. Sensitivity of Cancer Risks

Input variable	Adult Cancer Contribution to Variance (%)	Child Cancer (3.0x) Contribution to Variance (%)
Attenuation	-54	-53
Concentration	36	35
Duration	7	0.6
Age at move in	-	-7.4
Toxicity	2.5	2.8
Frequency	0.5	0.5

The sensitivity analyses for non-cancer risks to both adults and children show results that are very similar to those for cancer risks, discussed above. As shown in Table 5 below, when all the inputs are allowed to vary, the risks are dominated by only two input variables: the attenuation of vapors value (-55 to -59% of the variance in risks) and the concentration of contaminant in water (38% of the variance in risks). These inputs are followed by the much smaller effects of the inhalation Reference Concentration (RfC) toxicity factor (-2.1%), the exposure frequency (1.2%), and the exposure duration (0.1%). The results from another (one-at-a-time) sensitivity analysis also shows a very similar rank order and magnitude of effects of these same inputs (i.e., with attenuation and concentration having the most influence) for both adult and child non-cancer risks.

Table 5. Sensitivity of Non-Cancer Risks

Input variable	Adult Contribution to Variance (%)	Child – age adjusted Contribution to Variance (%)
Attenuation	-59	-55
Concentration	38	38
Age at move in	-	4.4
Toxicity	2.1	3.1
Frequency	1.2	0.1
Duration	0.1	0.0

In summary, the results of the several forms of sensitivity analyses all suggest that the risks are largely influenced by the attenuation value and concentration of contaminant in groundwater input values. Furthermore, as modeled here, the risks are not significantly modified other variables such as age adjustments for exposures in early age or exposures prior to birth.

An additional form of sensitivity analyses ('Spider Charts') also indicate that these two inputs (attenuation and concentration) are most influential in the lower and upper ends of their distribution ranges (for all four models, adult example shown in Figure 5). The other inputs have nearly linear, lower-level influences across their ranges (with the only exception being the 'age at move in' input variable in the child models since it limits the duration of exposure possible during childhood (i.e., $\leq$ 18 yrs of age)).

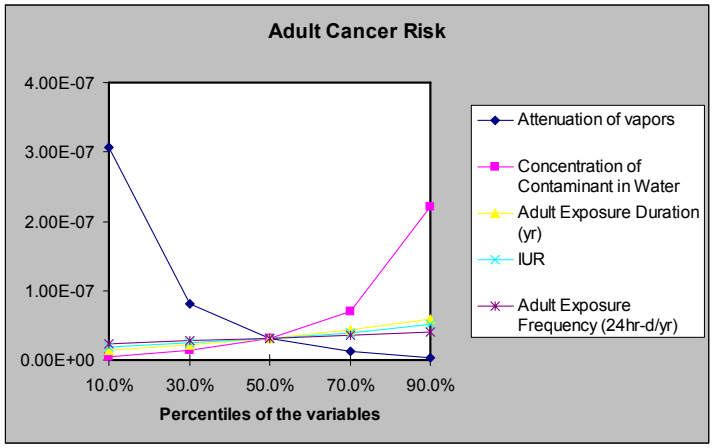

Figure 5. One-at-a-time sensitivity 'Spider Chart' (by percentiles) for Cancer - Adult

The risk estimates in this risk assessment included elements of both variability and uncertainty. That is to say the inputs to the models varied both due to recognized variability that occurs within a known range of values and also due to uncertainties (lack of knowledge) where, for example, both the range and the value of inputs can be unknown. Making a distinction between variability and uncertainty can be very important in part because uncertainty can, in theory, be reduced through research, whereas variability will remain as a natural feature of the problem. Furthermore, understanding the sources of uncertainty can help target research efforts towards reducing the most important uncertainties. Probabilistic methods allow analyses that can help to separate the impacts of input variability and uncertainty on risk predictions. The primary techniques are two-dimensional simulations that are comprised of a set of nested models that can help illustrate and differentiate between the influences of inputs that are primarily defined by uncertainty and those primarily defined by well-understood variability.

3.4 Two Dimensional Analyses

The two dimensional (2-D) methods used involve the random selection of a set of ten possible values for those input parameter(s) that are primarily defined by uncertainty. Each of these possible values (for the uncertain parameter(s)) is held fixed at the randomly selected value while a full model is run with all the other inputs; e.g., those primarily characterized by variability are allowed to vary across their expected distributions. Thus, for a 2-D simulation 10 full models (of 5900 iterations each) are run. One model is run for each of the ten randomly selected values of the uncertain parameter(s) that are held constant.

For this study the input values for attenuation, concentration, and toxicity (IUR or RfC) were considered to have the highest amount of uncertainty, and all the other input values (e.g., exposure duration, frequency, etc.) were allowed to vary across their expected (better understood, but variable) distributions. In a sense, this 2-D analysis is the opposite of the one-at-a-time sensitivity analyses discussed above because this 2-D approach fixes one (or more 'uncertain' inputs) and varies all others, whereas the one-at-a-time sensitivity approach discussed earlier fixed all but one parameter that was allowed to vary.

A number of 2-D simulations (each composed of 10 full model runs) were conducted with various combinations of inputs being considered to represent those with the most uncertainty. For each of the primary risk models (cancer and non-cancer, in adults and children), four versions of 2-D simulations were completed where the inputs that were considered to primarily represent uncertainty were 1) attenuation, 2) concentration, 3) attenuation and concentration, and 4) attenuation, concentration, and toxicity. This approach of running various combinations of fixed and varying inputs was used to provide insight into the effect of these inputs on risks. Thus, this approach could be considered a form of 'probabilistic sensitivity analysis' (Cullen and Frey, 1999). Two examples of the results from 2-D simulations for adult cancer are shown in Figure 6 below.

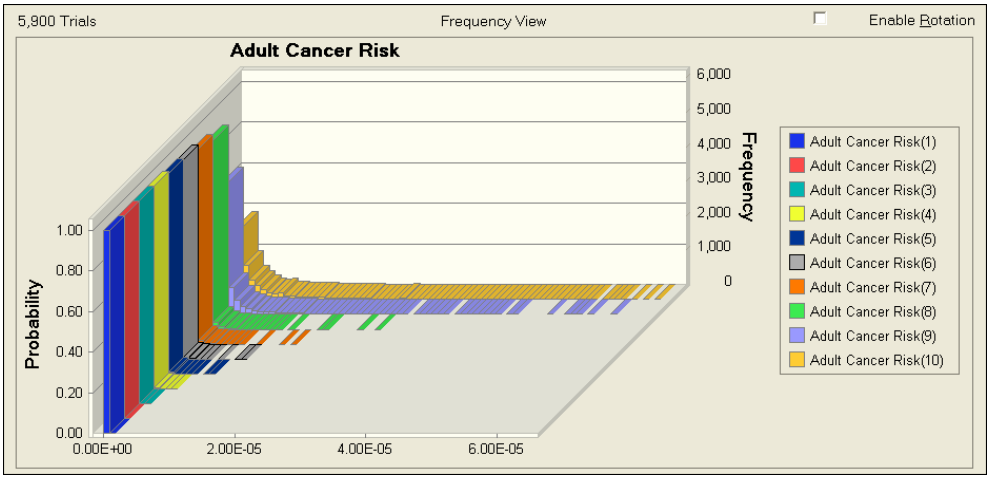

Figure 6a. 2-D Overlay-Frequency chart - Adult Cancer – Uncertainty = Attenuation

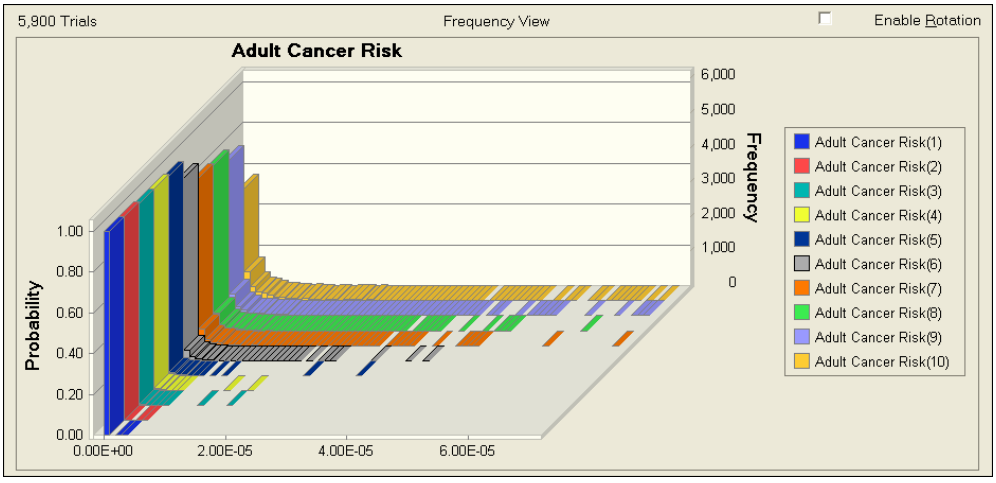

Figure 6b. 2-D Overlay-Frequency chart - Adult Cancer – Uncertainty = Concentration

In summary, the results of all the 2-D analyses, which involved 16 simulations (160 individual model runs), reconfirm the earlier observations that, across receptors (adults and children) and across outcomes (cancer and non-cancer), the risks 1) have central tendency values that are quite low; 2) have very few individual risks high enough to be within the range of typical concerns (i.e., cancer risks > 1 x 10^{-6} or hazards indices > 1.0); 3) are primarily influenced by the attenuation value and the concentration of the contaminant in groundwater (i.e., the vapor source term), particularly at the extremes of their input ranges.

4. DISCUSSION

The results of this assessment suggest that significant risks due to vapor intrusion of TCE in northern New Jersey are limited to a few individuals. There are, however, numerous limitations to the data and assumptions bridging data gaps in the methods used. For example, the electronic data set used was estimated to only represent 2/3 of the groundwater data collected in the study area and it was assumed this fairly represents all contamination in the study area. The generic (non-site-specific) groundwater plume mapping methods estimated the median shallow groundwater concentration (with depth adjustments) under the 883 residential structures was relatively low, i.e., only 5.9 ug/L (using the available evidence and the methods described above). Furthermore, the resulting risks presented here are central tendency probabilistic risks and these may be more comparable to other risks, e.g., groundwater-ingestion risks, if those risks were also calculated using central-tendency probabilistic methods (which is not commonly done).

Also note however, that several adverse health outcomes have been statistically associated with relatively low concentrations of TCE in groundwater that was used for tap-water (e.g., Cohn et al., 1994, where the maximum concentration category was only > 5 ug/L) and in some other cases reviewed by Bove et al., (1995) and Bove, Shim, and Zeitz (2002). Additionally, some associations have been observed in cases where vapor intrusion could be expected to be a contributing or primary exposure pathway. For example, observations of statistically significant associations with plausible outcomes in children and adults, with relatively low TCE vapor intrusion exposures have been found (e.g., NYSDOH, 2006b), and it is possible vapor intrusion may have played some contributory role in the proximity-based observations by Gerschwind, et al. (1992) which involved multiple sites across the state of New York. To improve their health studies' statistical power, and perhaps due to their experiences and informal observations at vapor intrusion sites, the New York State Dept. of Health has recently recommended a formal study of health associations at multiple sites with similar vapor intrusion exposures (NYSDOH, 2007). While it is likely that these NY sites could have somewhat higher exposures than the results of this study they could still have relatively 'low' central tendency probabilistic risks (compared to typical regulatory point-estimate reference values) and yet these exposures appear potentially relevant to public health.

5. CONCLUSIONS

Consideration of the methods and results of this risk assessment, as well as the inputs and secondary (sensitivity and two-dimensional) analyses, suggest that, while there are numerous limitations to the data and methods used, some observations can be made:

Significant risks due to vapor intrusion of TCE in northern NJ appear to be limited to a few individuals.

The data used indicate the most influential uncertain variable was the attenuation of the vapors between the subsurface vapor source and the indoor air.

The apparent second-most influential uncertain variable was the concentration of TCE in the groundwater actually beneath the home being assessed.

All other input variables had significantly less influence on the risks predicted.

There may be some concern that the monitor well data used in this study (reported only 2/3 of all data collected) may not be fully representative of the true groundwater vapor source-term concentrations in all residential areas of northern NJ. There may also be some concern that this study's generic groundwater modeling (IDW) methods may have underestimated the predicted groundwater vapor-source concentrations actually beneath some occupied residences. Nevertheless, the predicted population risks are so low that it appears unlikely that "the quality of the data or assessment methods is sufficiently poor that the true exposures might" miss significant population risks (Wilson and Crouch, 2001) in this portion of New Jersey.

However, New Jersey may not be representative of many states (e.g., its groundwater standard and screening criteria for vapor intrusion of TCE is the lowest in the nation (1 ug/L; NJDEP, 2007; Eklund et al., 2006), and other states have residential vapor intrusion screening criteria for groundwater up to 15,000 times higher (MIDEQ, 2006). That is to say, other states may have significantly more poorly assessed, or unknown, potential for vapor intrusion exposures and health risks.

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APPENDIX 1: ADULT – NHL MODEL STRUCTURE AND FORMULA

1a) Model Structure - Row# H

3 Conc(depth-adj) TCE in GWater	19.50	micrograms per liter (ug/liter)
4 Soil-gas Conc.@ Source	4212.00	micrograms per cubic meter (ug/m^3)
5 1/Attenuation Factor (GW-IAQ)	2280.82	unit-less
6 Indoor Air Conc. of TCE	1.85	micrograms per cubic meter (ug/m^3)
7 Exposure Frequency (adults)	240	days (full 24-hr) per year (24hr-day/yr)
8 Exposure Duration (adults)	9	duration of residence over plume (yrs)
9 Total Exposure	2160	days (full 24-hr) (24hr-day)
10 Background environ. exposures	0	Sharing metabolic pathways & mode of action
11 Background occupat. exposures	0	
12 Averaging Time (days)	25550	days in 70 yr lifetime (d)
13 Lifetime Average Conc.	0.156120746	micrograms per cubic meter (ug/m^3)
14 Inhalation Unit Risk - NHL	1.3E-06	Inhalation Unit (cancer) Risk per ug/m^3
Adult Cancer Risk	2.03E-07	unitless

1b) Model Formula - Row# H

3 Conc(depth-adj) TCE in GWater	19.5
4 Soil-gas Conc.@ Source	=Concentration*0.216*1000
5 1/Attenuation Factor (GW-IAQ)	2280.81779072092
6 Indoor Air Conc. of TCE	=H4/H5
7 Exposure Frequency (adults)	240
8 Exposure Duration (adults)	9
9 Total Exposure	=H8*H7
10 Background environ. exposures	0
11 Background occupat. exposures	0
12 Averaging Time (days)	25550
13 Lifetime Average Conc.	=(H6*H7*H8)/H12
14 Inhalation Unit Risk - NHL	0.0000013
Adult Cancer Risk	=H13*H14

APPENDIX 2: ADULT – CNS MODEL STRUCTURE AND FORMULA

2a) Model Structure - Row#	H	
3 Conc(depth-adj) TCE in GWater	19.50	micrograms per liter (ug/liter)
4 Soil-gas Conc.@ Source	4212.00	micrograms per cubic meter (ug/m^3)
5 1/Attenuation Factor (GW-IAQ)	2280.82	unitless
6 Indoor Air Conc. of TCE	1.85	micrograms per cubic meter (ug/m3)
7 Exposure Frequency (adults)	240	days (full 24-hr) per year (24hr-day/yr)
8 Exposure Duration (adults)	9	duration of residence over plume (yrs)
9 Total Exposure	2160	24 hr-days
10 Background environ. exposures	0	Sharing metabolic pathways & mode of action
11 Background occupat. exposures	0	
12 Averaging Time (days)	3285	Total duration of exposure in days (d)
13 Average Daily Exposure Conc.	0.156120746	micrograms per cubic meter (ug/m3)
14 Reference Conc. - CNS	7.4E+01	Inhalation RfC (non-cancer) Risk in ug/m^3
Adult Non-Cancer Risk (HQ)	2.11E-03	Unitless

2b) Model Formula - Row#	H
3 Conc(depth-adj) TCE in GWater	19.5
4 Soil-gas Conc.@ Source	=Concentration*0.216*1000
5 1/Attenuation Factor (GW-IAQ)	2280.81779072092
6 Indoor Air Conc. of TCE	=H4/H5
7 Exposure Frequency (adults)	240
8 Exposure Duration (adults)	9
9 Total Exposure	=H8*H7
10 Background environmental Exposures	0
11 Background occupational Exposures	0
12 Averaging Time (days)	=H8*365
13 Average Daily Exposure Conc.	=H6*(H9/H12)
14 Reference Conc. – CNS	74
Adult Non-Cancer Risk (HQ)	=H13/H14

APPENDIX 3: CHILD – CNS MODEL STRUCTURE AND FORMULA (W/ & W/O AGE-ADJUSTMENTS)

3a) Model Structure - Row#	H	
3 Conc(depth-adj) TCE in GWater	21.94	micrograms per liter (ug/liter)
4 Soil-gas Conc.@ Source	4738.67	micrograms per cubic meter (ug/m^3)
5 1/Attenuation Factor (GW-IAQ)	2280.82	unitless
6 Indoor Air Conc. of TCE	2.08	micrograms per cubic meter (ug/m^3)
7 Family Residence Duration	9	years (yrs)
8 Theoretical Child age at move in	0	constant distribution in years of age (-10 to 18)
9 Actual Child Age at move in	0	years
10 Child age at move out	9	years (max. age 18 at move out)
11 Tot. child residence duration	9	years
12 Child residence at 0-2 yr ages	2	years
13 Child residence at 2-18 yr ages	7	years
14 Exposure Frequency (0-2 yrs)	300	days (full 24-hr) per year (24hr-day/yr)
15 Exposure Frequency (2-20 yrs)	268	days (full 24-hr) per year (24hr-day/yr)
16 Total Childhood Exposure	2476	24 hr-days
17 Age >2 Period Adjustment Factor	1.25	Assumed 25% adj for higher response due to exposure in critical period
18 Age 0 to 2 Period Adj Factor	1.5	Assumed 50% adj for higher response due to exposure in critical period
19 Age -3/4 to 0 Period Adj Factor	1.75	Assumed 75% adj for higher response due to exposure in critical period
20 Breast feeding	1	Yes (1) - No (0) (unitless)
21 Age <-1 Period Adj. Factor	1.5	Assumed 50% (0-2 yrs) adj for bio-transfer from mother, if breast fed
22 Tot. Adjust. move in Age >2	1.25	
23 Tot. Adjust. move in Age 0 to 2	1.875	
24 Tot. Adjust. move in Age -3/4 to 0	3.28125	
25 Tot. Adjust. move in Age < -1	4.78125	
26 Individual move in Age Adj Factor	3.28125	Affects full exposure duration - to account for observed effects for longer durations
27 Tot. Adjusted Childhood Exposure	8124.375	24 hr-days
28 Background Exposures	0	Sharing metabolic pathways and mode of action

29 Maternal Exposure Adj Factor	0	
30 Paternal Exposure Adj Factor	0	
31 Averaging Time (days)	3285	total # days with possible exposure in childhood (d)
32 Childhood Avg. Conc. (Un-Adj.)	1.565962258	micrograms per cubic meter (ug/m^3)
33 Childhood Avg. Conc. (Age-Adj.)	5.13831366	micrograms per cubic meter (ug/m^3)
34 Inhalation RfC Non-Cancer Risk	7.4E+01	Non-Cancer reference concentration in ug/m^3
Childhood Unadj. Non-Cancer Risk	2.12E-02	Unitless
Childhood Age-adj. Non-Cancer Risk	6.94E-02	Unitless

3b) Model Formula - Row#	H
3 Conc(depth-adj) TCE in GWater	21.9382946446673
4 Soil-gas Conc.@ Source	=Concentration*0.216*1000
5 1/Attenuation Factor (GW-IAQ)	2280.81779072092
6 Indoor Air Conc. of TCE	=H4/H5
7 Family Residence Duration	9
8 Theoretical Child age at move in	0
9 Actual Child Age at move in	=IF(H8<0,0,H8)
10 Child age at move out	=IF(H9+H7>18,18,H9+H7)
11 Tot. child residence duration	=H10-H9
12 Child residence at 0-2 yr ages	=IF(H9<2,2-H9,0)
13 Child residence at 2-18 yr ages	=IF(H9>2,H11,H11-(2-H9))
14 Exposure Frequency (0-2 yrs)	300
15 Exposure Frequency (2-20 yrs)	268
16 Total Childhood Exposure	=(H12*H14)+(H13*H15)
17 Age >2 Period Adjustment Factor	1.25
18 Age 0 to 2 Period Adj Factor	1.5
19 Age -3/4 to 0 Period Adj Factor	1.75
20 Breast feeding	1
21 Age <-1 Period Adj. Factor	=1.5
22 Tot. Adjust. move in Age >2	=H17
23 Tot. Adjust. move in Age 0 to 2	=H18*H17
24 Tot. Adjust. move in Age -3/4 to 0	=H19*H18*H17
25 Tot. Adjust. move in Age < -1	=(H21*H20)+H24
26 Individual move in Age Adj Factor	=IF(H8>2,H22,IF(H8<-1,(H25),IF(H8>0,H23,H24)))
27 Tot. Adjusted Childhood Exposure	=H16*H26
28 Background Exposures	0
29 Maternal Exposure Adj Factor	0
30 Paternal Exposure Adj Factor	0
31 Averaging Time (days)	=H11*365
32 Childhood Avg. Conc. (Un-Adj.)	=H6*(H16/H31)
33 Childhood Avg. Conc. (Age-Adj.)	=H6*(H27/H31)
34 Inhalation RfC Non-Cancer Risk	74

Childhood Unadj. Non-Cancer Risk	=H32/H34
Childhood Age-adj. Non-Cancer Risk	=H33/H34

APPENDIX 4: STRUCTURE AND FORMULA FOR CHILD NHL MODEL (W/ & W/O AGE-ADJUSTMENTS)

4a) Model Structure - Row#	H	
3 Conc. (depth-adj) TCE in GWater	21.94	micrograms per liter (ug/liter)
4 Soil-gas Conc.@ Source	4738.67	micrograms per cubic meter (ug/m^3)
5 1/Attenuation Factor (GW-IAQ)	2280.82	Unitless
6 Indoor Air Conc. of TCE	2.08	micrograms per cubic meter (ug/m^3)
7 Family Residence Duration	9	years (yrs)
8 Theoretical Child Age at move in	-1.5	proportional distribution in years of age (-10 to 18)
9 Actual Child Age at move in	0	(w/o negatives)
10 Child Age at move out	9	years (up to max. age 18 at move out)
11 Tot. Child Residence Duration	9	Years
12 Child Residence at 0-2 yr Ages	2	Years
13 Child Residence at 2-18 yr Ages	7	Years
14 Exposure Frequency (0-2 yrs)	300	days (full 24-hr) per year (24hr-day/yr)
15 Exposure Frequency (2-18 yrs)	268	days (full 24-hr) per year (24hr-day/yr)
16 Total Childhood Exposure	2476	days (full 24-hr) (24hr-days)
17 0 to 2 Age Adj Factor (1.0x)	1	Assumes no higher response due to age
18 0 to 2 Age Adj Factor (1+x)	1	Adds variability for possible higher response for age
19 0 to 2 Age Adj Factor (3.0x)	3	Assumes 3.0x higher response due to age
20 Tot. Post-Natal Exposure (1.0x)	2476	days (full 24-hr) (24hr-days)
21 Tot. Adj. post-natal Exposure (1+x)	2476	days (full 24-hr) (24hr-days)
22 Tot. Adj. post-natal Exposure (3.0x)	3676	days (full 24-hr) (24hr-days)
23 Considering Pre-Natal Exposures?	1	Yes (1) - No (0) (unitless)
24 Age -3/4 to 0 Pre-Natal Adj Factor	1	Adds & adjusts for higher response due to age
25 Breast Feeding?	1	Yes (1) - No (0) (unitless)
26 Age < -3/4 Pre-natal Adj Factor	1	Adds & adjusts bio-transfer from mother, if breast fed
27 Total Possible Pre-natal Exposures	300	days (full 24-hr) (24hr-day/yr)
28 Total Childhood Exposure (1.0x)	2476	24 hr-days
29 Tot. Adj. Childhood Exposure (1+x)	2476	24 hr-days
30 Tot. Adj. Childhood Exposure	3676	24 hr-days

(3.0x)		
31 Tot. Adj. Childhood Exposure (3.0x+)	3976	24 hr-days - includes pre-natal exposures
32 Background Exposures	0	If sharing metabolic pathways or mode of action
33 Maternal Exposure Adj Factor	0	
34 Paternal Exposure Adj Factor	0	
35 Averaging Time (days)	6570	total # days in 18 yr childhood (d)
36 Childhood Average Conc. (1.0x)	0.782981129	micrograms per cubic meter (ug/m^3)
37 Childhood Average Conc. (1+x)	0.782981129	micrograms per cubic meter (ug/m^3)
38 Childhood Average Conc. (3.0x)	1.16245502	micrograms per cubic meter (ug/m^3)
39 Childhood Average Conc. (3.0x+)	1.257323493	ug/m3, "+" = includes pre-natal exposures
40 Inhalation Unit Risk (IUR)	1.3E-06	Cancer risk per ug/m^3
Child (1.0x) Cancer Risk	1.02E-06	Risk – Unitless
Child (1+x) Cancer Risk	1.02E-06	Risk – Unitless
Child (3.0x) Cancer Risk	1.51E-06	Risk – Unitless
Child (3.0x+) Cancer Risk	1.63E-06	Risk - Unitless ("+" includes pre-natal exposures)

4b) Model Formula - Row#	H
3 Conc. (depth-adj) TCE in GWater	21.9382946446673
4 Soil-gas Conc.@ Source	=Concentration*0.216*1000
5 1/Attenuation Factor (GW-IAQ)	2280.81779072092
6 Indoor Air Conc. of TCE	=H4/H5
7 Family Residence Duration	9
8 Theoretical Child Age at move in	-1.5
9 Actual Child Age at move in	=IF(H8<0,0,H8)
10 Child Age at move out	=IF(H9+H7>18,18,H9+H7)
11 Tot. Child Residence Duration	=H10-H9
12 Child Residence at 0-2 yr Ages	=IF(H9<2,2-H9,0)
13 Child Residence at 2-18 yr Ages	=IF(H9>2,H11,H11-(2-H9))
14 Exposure Frequency (0-2 yrs)	300
15 Exposure Frequency (2-18 yrs)	268
16 Total Childhood Exposure	=(H12*H14)+(H13*H15)
17 0 to 2 Age Adj Factor (1.0x)	1
18 0 to 2 Age Adj Factor (1+x)	1
19 0 to 2 Age Adj Factor (3.0x)	3
20 Tot. Post-Natal Exposure (1.0x)	=(H12*H14*H17)+(H13*H15)
21 Tot. Adj. post-natal Exposure (1+x)	=(H12*H14*H18)+(H13*H15)
22 Tot. Adj. post-natal Exposure (3.0x)	=(H12*H14*H19)+(H13*H15)
23 Considering Pre-Natal Exposures?	1
24 Age -3/4 to 0 Pre-Natal Adj Factor	1
25 Breast Feeding?	1
26 Age < -3/4 Pre-natal Adj Factor	1
27 Total Possible Pre-natal Exposures	=IF(H8<-

	1,(0.75*H14*H24)+(0.25*H14*H26*H25),0)
28 Total Childhood Exposure (1.0x)	=(H12*H14*H17)+(H13*H15)
29 Tot. Adj. Childhood Exposure (1+x)	=(H12*H14*H18)+(H13*H15)
30 Tot. Adj. Childhood Exposure (3.0x)	=(H12*H14*H19)+(H13*H15)
31 Tot. Adj. Childhood Exposure (3.0x+)	=(H12*H14*H19)+(H13*H15)+(H27*H23)
32 Background Exposures	0
33 Maternal Exposure Adj Factor	0
34 Paternal Exposure Adj Factor	0
35 Averaging Time (days)	=18*365
36 Childhood Average Conc. (1.0x)	=H6*(H28/H35)
37 Childhood Average Conc. (1+x)	=H6*(H29/H35)
38 Childhood Average Conc. (3.0x)	=H6*(H30/H35)
39 Childhood Average Conc. (3.0x+)	=H6*(H31/H35)
40 Inhalation Unit Risk (IUR)	0.0000013
Child (1.0x) Cancer Risk	=H36*H40
Child (1+x) Cancer Risk	=H37*H40
Child (3.0x) Cancer Risk	=H38*H40
Child (3.0x+) Cancer Risk	=H39*H40