

Foth & Van Dyke

R E P O R T

Final

**Baseline Risk Assessment Plan
City of Delavan Superfund Project**

Scope ID: 91W54-1

*State of Wisconsin
Department of Natural Resources*

October 1991

Foth & Van Dyke

2737 S. Ridge Road
P.O. Box 19012
Green Bay, WI 54307-9012
414/497-2500
FAX: 414/497-8516

Foth & Van Dyke

Engineers

Architects

Planners

Scientists

October 9, 1991

2737 S. Ridge Road
P. O. Box 19012
Green Bay, WI 54307-9012
414/497-2500
FAX: 414/497-8516

Ms. Stephen M. Ales
Environmental Response and Repair Section
Bureau of Solid and Hazardous Waste Management
Department of Natural Resources
P. O. Box 7921
Madison, WI 53707

91W54

Dear Mr. Ales:

RE: Final Baseline Risk Assessment Plan for the Delavan Superfund Project

Foth & Van Dyke is pleased to submit this Final Baseline Health Risk Assessment Plan for the Delavan Superfund Project. Your comments on the draft copy of the Plan have been incorporated into this document.

Foth & Van Dyke values you as a client and encourages you to please call if you have any questions regarding this report.

Sincerely,

FOTH & VAN DYKE



Steven J. Laszewski, Ph.D.
Senior Environmental Toxicologist



Janis Schallhorn Kesey, C.P.G.
Manager, Environmental Investigations

SJL/lb

DISTRIBUTION LIST

No. of Copies

Sent To:

6

Stephen M. Ales
Environmental Response and Repair Section
Bureau of Solid and Hazardous Waste Management
Department of Natural Resources
P. O. Box 7921
Madison, WI 53707

**BASELINE RISK ASSESSMENT PLAN
CITY OF DELAVAN SUPERFUND PROJECT**

Prepared for:

**STATE OF WISCONSIN
DEPARTMENT OF NATURAL RESOURCES**

Prepared by:

**FOTH & VAN DYKE and Associates Inc.
2737 S. Ridge Road
P. O. Box 19012
Green Bay, Wisconsin 54307-9012**

OCTOBER 1991

REUSE OF DOCUMENTS

This document has been developed for a specific application and not for general use; therefore, it may not be used without the written approval of Foth & Van Dyke and Associates. Unapproved use is at the sole responsibility of the unauthorized user.

Foth & Van Dyke

2737 S. Ridge Road
P. O. Box 19012
Green Bay, Wisconsin 54307-9012
414/497-2500
FAX: 414/497-8516

**Copyright®, Foth & Van Dyke
1991**

TABLE OF CONTENTS

	PAGE
1.0 INTRODUCTION	1
1.1 Site Overview and Background	1
1.2 Risk Assessment Objectives	3
2.0 DATA EVALUATION	4
2.1 Site Characterization/Data	4
2.1.1 QA/QC	4
2.2 Treatment of Data	5
2.2.1 Statistical Evaluation	5
2.2.2 Qualified Data	5
2.2.3 Data Summary	6
2.2.4 Background Chemical Concentrations	6
2.2.5 Identify Chemicals-of-Concern	6
3.0 EXPOSURE ASSESSMENT	8
3.1 Receptor Identification	10
3.2 Exposure Pathway	12
3.2.1 Ingestion	12
3.2.2 Inhalation	13
3.2.3 Dermal	13
3.2.4 Environmental Fate and Transport	14
4.0 TOXICITY ASSESSMENT	15
4.1 Hazard Identification	15
4.2 Noncarcinogenic Effects	16
4.3 Carcinogenic Effects	16
5.0 RISK CHARACTERIZATION	17
5.1 Noncancer Risk Estimate	17
5.2 Cancer Risk Estimate	18
5.3 Assumptions and Uncertainties	18
5.4 Risk Expression and Communication	19
6.0 REFERENCES	20

LIST OF FIGURES

FIGURE 3-1	TCE Trend Analysis.....	9
FIGURE 3-2	Generic Intake Equation.....	11

LIST OF APPENDICES

APPENDIX A	Screening Methods for Inhalation Exposure
------------	---

1.0 INTRODUCTION

This document presents the methodology to be used in development of the baseline human health risk assessment (BHRA) for the City of Delavan Municipal Well No. 4 Superfund Project. The United States Environmental Protection Agency (USEPA) defines a BHRA as an evaluation of the potential adverse health effects from releases of hazardous substances at a site in the absence of any remedial action. A BHRA is often viewed as a human health evaluation of the no-action alternative. In this regard, the BHRA at this site may not be considered a true baseline risk assessment since remedial measures have been present at the site since 1984. However, for the remainder of this document and project, this initial study performed by Foth & Van Dyke for the WDNR will be termed a BHRA.

The principal guidance document which will be used for developing the risk assessment will be the Risk Assessment Guidance for Superfund: Human Health Evaluation Manual (Part A), 1989. Additional USEPA documents which may be used include: Superfund Exposure Assessment Manual (1988), Exposure Factors Handbook (1990), and a supplemental guidance document titled Standard Default Exposure Factors (1991). When necessary, the open literature may also be used for development of the risk assessment. A complete bibliography of the above references and others which will be used in the BHRA is present at the end of this document.

1.1 Site Overview and Background

A random sampling of municipal water supply systems by the WDNR in 1982 discovered elevated levels of trichloroethene (TCE) at Delavan Municipal Well No. 4. Following an investigation, the source of contamination was determined to be Sta-Rite Industries, Inc., a manufacturer of high-quality pumps, which is located approximately 400 feet east of City Well No. 4. A hazard assessment performed by the USEPA utilizing the Hazard Ranking System (HRS), resulted in a score of 28.9, sufficient to qualify the site for National Priorities List (NPL) nomination under Superfund. City Well No. 4 was nominated for the NPL in 1983 and listed in 1984.

Several studies have been performed at the site since the 1982 discovery of TCE in City Well No. 4. The studies revealed that the contamination was originating from Sta-Rite Plant No. 1, located approximately 1,000 feet north-northeast of City Well No. 4, and Sta-Rite Plant No. 2, located approximately 400 feet east of City Well No. 4. Generally, the plume associated with Plant No. 1 consists of groundwater impacted by TCE and 1,1,1-trichloroethane (TCA), while the plume associated with Plant No. 2 consists primarily of TCE and tetrachloroethene (PCE).

The studies also showed that approximately 400 feet of unconsolidated glacial deposits overlie bedrock at the site. The unconsolidated deposits consist of approximately seven feet of topsoil overlying 150 feet of coarse sands and gravel. Below the sand and gravel unit is approximately 265 feet of fine-grained glacial till. The water table surface occurs at an average depth of 35 feet. City Well No. 4 is approximately 115 feet deep with the lower 20 feet consisting of wire-bound well screen.

A pump test and hydraulic analysis conducted by Hydro-Search, Inc. in 1990 showed that impacts to City Well No. 4 were from Plant No. 2. The report concluded that Plant No. 1 was hydraulically isolated from City Well No. 4.

Active remediation is currently present at the site. A groundwater extraction system consisting of five pumping wells discharging to the storm sewer has been operating at Plant No. 1 since 1984. Two extraction wells have been operating at Plant No. 2 since 1985. In addition, an *in situ* soil vacuum extraction system has been in operation at Plant No. 2 since 1988.

A preliminary health assessment conducted by the Wisconsin Department of Health and Social Services in 1989 concluded that a public health concern exists at the location when City Well No. 4 is operating. The report recommended that City Well No. 4 should not be used except in emergency situations. The report also recommended collecting more data so that additional environmental and human exposure pathways could be properly evaluated.

1.2 Risk Assessment Objectives

The objective of the BHRA performed at Delavan is to quantitatively evaluate the current and future adverse health effects at the site due to human exposure to the released substances. The conclusions of the risk assessment, as well as information contained within the assessment, can be used to assist remedial action decisions at the site.

2.0 DATA EVALUATION

The objective of the data evaluation section is to develop a data set which will be appropriate for use in the BHRA. Data from the RI/FS may be used exclusively to construct the BHRA or be combined with data from previous on-site investigations. Factors used to evaluate the appropriateness of including past data are the similarity of laboratory methods used to obtain the data, QA/QC procedures followed and similarity in concentrations or concentration trends over time.

Decisions on including past data in the risk assessment analysis will be made following consultation with the WDNR project manager.

2.1 Site Characterization/Data

The data evaluation section will construct a data set to be used in the BHRA. Often an entire data set is not used as some results may not withstand the rigor of the QA/QC procedures. Other samples may be rejected if the sample quantitation limit (SQL) greatly exceeds positive reported results or if the data are semi-quantitative, e.g., organic vapor analyzer readings.

The Quality Assurance Project Plan (QAPP) developed for this project emphasizes approved formal procedures to ensure that quality data will be collected. Data Quality Objectives (DQO) within each Operable Unit (OU) are presented in the Sampling and Analysis Plan (SAP). Five DQO analytical levels have established for the project. Data intended for use in the risk assessment has been assigned the three highest DQO analytical levels (III, IV, and V).

2.1.1 QA/QC

The QAPP contains specific QA and QC procedures, which if followed, will help to ensure the collection of legally defensible data.

2.2 Treatment of Data

At times analytical data may be transformed in order to be better utilized in the BHRA. Examples of transformations which may be used on the data include, statistical tests and procedures, treatment of no detects, proper use of qualified (coded) data and background chemical information. Complete procedures with example calculations will be included with any data transformation.

2.2.1 Statistical Evaluation

Routine statistical procedures such as a determination of population distributions, means, range, standard deviations and 95 percent confidence intervals will be used with the data set. Results from these analyses will be presented in tabular format and separated by the environmental medium of concern. More sophisticated statistical tests, e.g., parametric and nonparametric analysis of variance (ANOVA), may be performed on the data set. These tests can help to show whether significant differences are present in contaminant concentrations over time and also between different OUs. This type of analysis can be incorporated into the BHRA to provide valuable information to the risk management remedial decisions for the entire site or within an individual OU.

2.2.2 Qualified Data

Questions regarding chemical identity, chemical concentration, or both, are frequently present in environmental data sets. Under the EPA contract laboratory program (CLP), analytical results with these types of concerns will be flagged through the use of various data qualifiers or codes. Laboratories without CLP accreditation will often use the same type of code on suspect data. All qualifiers will be handled in a consistent manner using the guidelines presented in RAGS (EPA 1989). In the event that the data qualifiers used by the laboratory are not defined in the data set, a request will be made to the laboratory for the exact meaning of the qualifier. Qualified data will not be used in the BHRA until its meaning is clearly understood.

2.2.3 Data Summary

The BHRA data set will be presented in a clear and concise manner. This will be accomplished through the use of tables which summarize the data by medium, time collected, statistical tests performed and by OU. Data within an individual medium may also be separated for the BHRA, i.e., surface soil exposure differs from subsurface soil exposure.

When appropriate, chemical concentration data from different mediums, but within the same OU will be presented together. This will aid in determining what types of relationships, if any, exist between chemical concentrations within adjacent mediums.

2.2.4 Background Chemical Concentrations

A comparison of site chemical concentrations within a medium to naturally occurring or anthropogenic chemicals in the environment can greatly aid risk management decisions at a site. Background chemical information can be obtained from a combination of off-site sampling and local, regional, or national data bases.

Another type of analysis related to a background evaluation is a comparison of the normal daily exposure and risk to a specific compound from the air, food, and water, to site chemical exposure. For example, TCE is present in the ambient air within the United States at concentration levels in the part-per-trillion range. This type of ambient exposure has a small but expressible risk associated with it. When presented properly, background information of this sort is often very useful in aiding site risk management decisions.

2.2.5 Identify Chemicals-of-Concern

At many locations where a release of hazardous substances has occurred, subsequent environmental investigations reveal the presence of a large number of different types of organic contaminants. Many of the compounds detected are either only

infrequently seen or are present in low concentrations and possess a low degree of toxicity. Inclusion of all the chemicals detected in an investigation into the BHRA may distract from the true risks presented at the site. A procedure exists, using chemical classes, frequency of detection, background chemical information data, essential nutrient information, and a concentration-toxicity screen to reduce the number of compounds taken through the BHRA.

Past data collected at the Delavan site showed that TCE, PCE, and TCA, comprise the compounds most frequently seen at the site. However, other VOCs detected at the site include 1,1-dichloroethane, trans-1,2-dichloroethene, 1,1-dichloroethane and toluene. Dependant on the RI/FS results, it may be appropriate to eliminate some contaminants from the BHRA.

A Technical Memorandum will be submitted to the WDNR which contains the identity and concentrations of the chemicals to be taken through the BHRA and any applicable or relevant and appropriate requirements (ARARs) of those compounds.

3.0 EXPOSURE ASSESSMENT

The objective of the exposure assessment is to estimate the type (inhalation, ingestion, and dermal) frequency, duration and magnitude of exposures at the site due to the releases of hazardous substances. The exposure assessment constitutes a critical section of the risk assessment where site-specific information is used to estimate the degree of chemical intake through the various exposure routes. This information will be later combined with the toxicity assessment information contained in another section of the risk assessment to evaluate the overall site hazard.

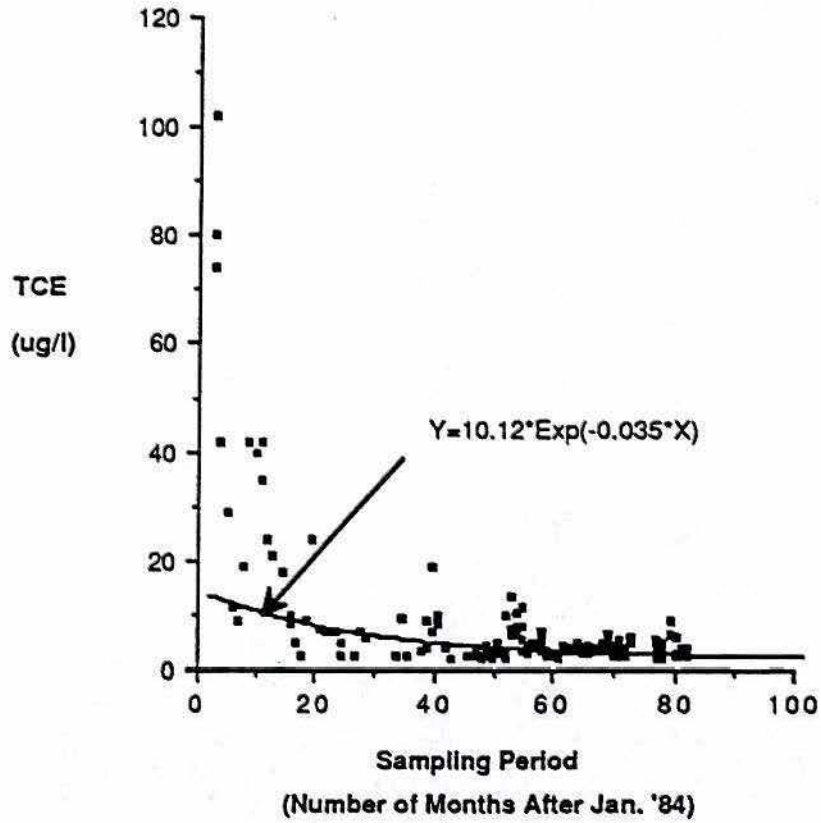
The media of concern at this site includes the soil, air, groundwater, surface water and sediments. Within each medium, the degree of human exposure will be evaluated through a series of exposure scenarios. An exposure scenario is formulated into an equation which solves for the chronic daily intake (CDI) of an individual compound in a particular medium. The scenarios will consider both current and future exposures.

Current exposures at the site will be evaluated using the data collected during the RI/FS. Future exposures will have to be based on models of future conditions which can be constructed through the use of existing monitoring data coupled with statistical and environmental fate models.

For example, sufficient characterization of TCE at a number of locations has been conducted so that a statistical trend analysis can be performed on the data (Figure 3-1). Assuming TCE data from the RI/FS falls within specific confidence intervals on the trend line than extrapolations with some degree of certainty can be made on future TCE concentrations at a given exposure point.

When constructing human health risk assessments at Superfund sites the USEPA embraces the concept of Reasonable Maximum Exposure (RME), which requires the risk assessor to develop exposure or CDI equations that reflect a conservative estimate on exposure. The idea is to estimate an exposure level which is above the "average case" but less than a "worst case" analysis through combining upper-bound and

Trend of TCE Measured at City Well 4



DELAVAN RISK ASSESSMENT PLAN		
FIGURE 3-1 TCE TREND ANALYSIS		
9	Scale: N/A	Date: OCTOBER, 1991
	Prepared By: Foth & Van Dyke	By: DHS

mid-range exposure factors into the CDI equations. Recently the USEPA published OSWER Directive-928.6-03, March 1991 which attempts to reduce unwarranted variability in the selection of exposure factors used to estimate a RME. An illustration of how upper-bound and mid-range exposure factors may be combined into a standard exposure intake calculation is presented in Figure 3-2. Use of site-specific information to generate exposure factors which deviate from RAGS or the recent OSWER directive are still allowed, however, the use of alternative exposure factors must be accompanied by supporting data with solid justification for their use.

In the exposure assessment CDIs are not summed across exposure pathways, i.e., simultaneous exposure to compounds in air and soil, rather this task is performed in the risk characterization section. Yet the RME concept at a site holds for exposures across pathways as well. Therefore, to arrive at an RME across pathways it may be necessary to combine the RME for one pathway with a more typical exposure analysis for another pathway. The key determination to be made is whether it is likely that the same individual would consistently face the RME by more than one pathway. If sufficient reasoning cannot be found to use RME across pathways, than the assessment should rely upon equations which combine average exposure parameters with conservative parameters.

A discussion on the uncertainty associated with the exposure assessment will be presented as part of the BHRA. Part of this discussion will include tables which highlight the exposure equation input variables, range of variable values, value chosen and reasoning for choosing that value.

A Technical Memorandum will be submitted to the WDNR which contains the exposure pathways considered for inclusion in the BHRA, assumptions used in the exposure equations, and results of any environmental fate model.

3.1 Receptor Identification

Current and future individuals or receptors who have the potential to be exposed to substances from the site will be identified. If the probability of exposure occurring

$$\text{Intake} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

C = Concentration of the chemical in each medium (conservative estimate of the media average contacted over the exposure period).

IR = Intake/Contact Rate (upper-bound value)

EF = Exposure Frequency (upper-bound value)

ED = Exposure Duration (upper-bound value)

BW = Body Weight (average value)

AT = Averaging Time (equal to exposure duration for non-carcinogens and 70 years for carcinogens).

DELAVAN RISK ASSESSMENT PLAN	
FIGURE 3-2 GENERIC INTAKE EQUATION	
Scale:	N/A
Date:	OCTOBER, 1991
Prepared By:	Foth & Van Dyke
By:	DHS

is very low, the professional judgement of the risk assessor in conjunction with WDNR guidance will be used to determine if the receptor and exposure pathway should be included in the BHRA.

3.2 Exposure Pathway

An exposure pathway describes the course a contaminant may take from an identified source to a potential receptor. Key elements of an exposure pathway include: contaminant source and release mechanism(s), transfer media, exposure point and an exposure route. Each element must be present or have a significant potential of being present, e.g., future use of groundwater in an impacted area, for the exposure pathway to be included in the BHRA.

3.2.1 Ingestion

The potential for receptor ingestion of contaminated soil, groundwater and surface water exists at the Delavan site. Inadvertent soil ingestion and groundwater ingestion have a greater potential than surface water ingestion to form a complete exposure pathway at this site.

Two major surface water bodies, Delavan and Comus Lakes, are in the vicinity of the site, however, only Comus Lake, a probable discharge area for groundwater flow from the site, has the potential for site-related compound exposure. Previous work (Donohue 1984) did not reveal TCE or TCA in Comus Lake. RI/FS results from OU-1D will help to show if future exposure to contaminants in Comus Lake is probable.

The groundwater extraction system and site surface runoff water enter the municipal storm sewer which ultimately discharges into an intermittent stream feeding Swan Creek. Surface water and sediment contaminant characterization at the discharge location will take place as part of the OU-2E investigation. Assuming receptors are present at this location, the data from OU-2E will be used in the development of an exposure pathway.

Receptors exposed to contaminated groundwater include the Delavan residents who use water from City Well No. 4. Since water use from City Well No. 4 is only reserved for peak demand times and water from City Well No. 4 is blended with clean water, information collected in OU-2E of the RI/FS will be needed to estimate exposure. The estimate of the VOC concentrations at the tap will be obtained through an analysis of VOCs in the municipal distribution system receiving water from City Well No. 4. An assumption will be made that the VOC concentrations in the distribution system are equal to the VOC concentrations at the tap. This information will be compared with exposure to City Well No. 4 water at the wellhead in order to better evaluate the risk associated with using City Well No. 4.

3.2.2 Inhalation

Exposure to chemicals-of-concern in the ambient air have the potential to occur at several exposure points including: groundwater extraction well discharge points, soil vapor extraction emission locations, volatilization from soil, storm sewer discharge areas and volatilization from contaminated groundwater during residential use. Estimations on the exposure concentrations will be made through a combination of ambient air monitoring, emission measurements with dispersion modeling and emission modeling with dispersion modeling. Simple screening models will be used in a first approximation to estimate ambient air concentrations of the chemicals-of-concern (Appendix A). If necessary, more sophisticated models may be selected if the screening models indicate that the inhalation route is an exposure route of concern.

3.2.3 Dermal

Potential dermal exposure routes at the site include dermal contact with: site soils, groundwater, e.g., showering or bathing with City Well No. 4 water, surface waters and sediments. Dermal exposure CDI equations differ from most other exposure equations in that the equations consider the absorbed dose as compared to the administered dose. Absorption factors are used to account for the binding of the compound to soil or

sediments and absorption of the compound across the dermal barrier. Absorption factors will be obtained from the open literature.

3.2.4 Environmental Fate and Transport

Following release of a chemical into the environment several different fate mechanisms, such as volatilization, biodegradation, chemical or abiotic degradation, adsorption and bioaccumulation, may affect the concentration of a compound. A compound's fate in the environment is dependent on its physical/chemical properties. When applicable these properties will be used in environmental fate models, e.g., first-order biodegradation expressions, to predict future contaminant concentrations at exposure points.

4.0 TOXICITY ASSESSMENT

The objective of a toxicity assessment is to estimate the potential for site contaminants to cause adverse health effects. The two separate steps used to accomplish this task are hazard identification and dose-response. Hazard identification describes qualitatively the types of adverse health effects which may occur following exposure to the chemicals-of-concern. The dose-response discussion is a quantitative evaluation of the dose of a chemical likely to cause carcinogenic and/or noncarcinogenic health effects. Toxicity information will be obtained from the integrated risk information system (IRIS), health effect assessment summary tables (HEAST) and if unavailable from either of these sources, other USEPA references or the open literature.

Currently, no approved mechanism exists for incorporating hazard identification information into dose-response information, yet an appreciation of their interrelationship is important in viewing site risk. For example, a high carcinogenic risk estimate at a site may be driven by a compound which has only demonstrated tumorigenicity in a select rodent strain, but not in humans. Failure to consider this type of information in risk management decisions may lead to an overestimation of the site hazard.

4.1 Hazard Identification

The toxicity information on a substance is collected from epidemiologic, clinical and animal studies and used in an evaluation of the hazard of the substance. If present, toxicity information should be presented which best matches the conditions and exposure characteristics of the study site. Additional supporting data which the USEPA considers in the toxicity assessment of a chemical are short-term genotoxicity tests such as tests for chromosomal aberrations and DNA damage and repair. The present trend within the agency is for a greater incorporation of supporting toxicity information into risk assessments and risk management decisions.

4.2 Noncarcinogenic Effects

A reference dose, or RfD, will be used as the noncarcinogenic toxicity value to assess the likelihood of adverse health effects to receptors. A RfD is an estimate of the daily intake of a chemical which would be free from adverse health effects. The use of an RfD implies that a threshold dose or exposure limit occurs, below which no response is seen. RfDs are obtained from the USEPA and exist for most chemicals. In the event a RfD is not present for a chemical, one may be derived using EPA methodology.

Subchronic RfDs are also available and should be used to evaluate short-term exposures, i.e., two weeks to seven years. All RfDs incorporate a degree of uncertainty and should not be viewed as an absolute dividing line separating toxic from nontoxic exposures.

4.3 Carcinogenic Effects

A slope factor will be used as the carcinogenic toxicity value to assess the carcinogenic risk to the receptors. The slope factor is the upper 95 percent confidence limit of the slope of the carcinogenic dose/response curve. Use of the slope factor will generate an upper-bound lifetime probability of an individual contracting cancer following a set exposure to a chemical. Sometimes the cancer risk estimate is incorrectly viewed as the probability of dying from a cancer. It should be emphasized that the slope factor only indicates the probability of developing a cancer.

A weight-of-evidence classification will accompany each chemical and slope factor. This classification indicates the potency or strength of the carcinogen. The most likely tumor site will be presented for each carcinogen listed.

A Technical Memorandum will be submitted to the WDNR which highlights the information to be used in the toxicity assessment section of the BHRA.

5.0 RISK CHARACTERIZATION

The objective of the risk characterization section is to assimilate the exposure and toxicity assessment information into a quantitative risk expression. For an individual pathway and chemical, a cancer risk and noncancer hazard quotient estimate will be calculated. If multiple chemicals are present within an individual pathway, the risk is summed for the chemicals to arrive at a total cancer risk and noncancer hazard index. At some locations, an individual might be exposed to chemical(s) through more than one pathway, e.g., ingesting contaminated drinking water and inhaling air contaminants, if so, risks will be combined across pathways. Risks across pathways will only be calculated at the site if a clear justification for doing so is present. This decision will rely upon the professional judgement of the risk assessor in collaboration with the WDNR project manager.

The key assumptions used in the risk assessment will also be presented in this section, with some discussion as to how these assumptions impact the risk expression at the site. The risk characterization section acts as the link between the risk assessment and risk management portions of a project and as such, is important to the overall remedial decisions at the site.

A Technical Memorandum which describes the results of the risk characterization will be prepared and delivered to the WDNR.

5.1 Noncancer Risk Estimate

Unlike a cancer risk expression, a noncancer risk expression is not presented as a probability statement. Rather, the CDI is divided by the RfD to produce a hazard quotient. If the quotient exceeds one there is the potential for adverse noncarcinogenic effects to occur to exposed individuals. The CDI/RfD ratio is not a probability statement, however, the greater the value is above one, the more concern should exist at the site.

The potential for noncarcinogenic effects from multiple chemical exposures is evaluated through the sum of individual hazard quotients to arrive at a hazard index. Therefore, simultaneous exposure to multiple chemicals, none of which has a hazard quotient value greater than one could trigger concern at a site. This approach has several major limitations, including that the assumption of dose additivity is best applied to chemicals with either the same mechanism of action or effect. The USEPA recognizes the limitations associated with the noncarcinogenic assessment and allows a segregation of hazard indices to be performed at a site. This process segregates chemicals by effect or mechanism of action so that an overestimation of the site hazard is not presented. If necessary, a segregation of hazard indices will be performed as part of the risk characterization.

5.2 Cancer Risk Estimate

The CDI developed in the exposure assessment is multiplied by the slope factor from the toxicity assessment to arrive at the cancer risk. This equation is valid for risk levels below 0.01, if a greater calculated risk is present at the site than the alternative one-hit equation will be used to calculate the cancer risk. The risk estimate derived through EPA methodology is an upper-bound estimate and therefore, the real risk associated with a site is not likely to exceed this estimate.

Simultaneous exposure to more than one carcinogen is presently evaluated through a simple summation of each individual cancer risk estimate. This evaluation assumes that synergistic or antagonistic mechanisms are absent with the chemicals. This assumption may be incorrect leading to either over- or under-estimation of the actual site cancer risk. Improved methods for evaluating carcinogen interactions are becoming available and may be available for use at the site.

5.3 Assumptions and Uncertainties

A qualitative uncertainty analysis will be included in this section of the report. This analysis will identify the key assumptions used in the risk assessment and describe the potential impact these assumptions had on the expression of risk at the site. Common

areas leading to uncertainty at a site include the site data package, statistical treatment of the data, exposure scenario inputs and toxicity values used in the risk assessment. The uncertainty analysis will include but possibly not be limited to these key areas.

5.4 Risk Expression and Communication

The final portion of the risk assessment interprets the findings of the risk assessment in the context of the overall information collected at the site during the RI/FS process. The intent is to fully articulate the results of the risk assessment, which up to this point are often only presented as numerical estimates of risk. For example, the major factors driving the site risks will be discussed at this time. To the extent possible, the results of the baseline risk assessment will be presented graphically to help in the communication of the study results. The primary user of the risk assessment will be the site manager responsible for risk management decisions at the site. The risk assessment will furnish a risk estimate for the site which the risk manager will use along with other site factors, e.g., economic, legal, and social to help formulate acceptable remedial action alternatives.

6.0 REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). U.S. Public Health Service. Toxicological Profile for Trichloroethylene. ATSDR/TP-88/24. 1989.
- Agency for Toxic Substances and Disease Registry (ATSDR). U.S. Public Health Service. Toxicological Profile for 1,1,1-Trichloroethane. 1990.
- Agency for Toxic Substances and Disease Registry (ATSDR). U.S. Public Health Service. Toxicological Profile for Tetrachloroethylene. ATSDR/TP-88/22. 1990.
- Brown, L.P., Farrar, D.G., and De Rooij, C.G. Health Risk Assessment of Environmental Exposure to Trichloroethylene. Regulatory Toxicology and Pharmacology, 11:24-41, 1990.
- Donohue, Groundwater Monitoring and Remedial Action - Interim Report to DNR, Sta-Rite Industries, Water Equipment Division, Delavan, Wisconsin: Donohue & Associates, Inc., Sheboygan, Wisconsin. 1984.
- EPA Office of Emergency and Remedial Response. Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A). EPA/540/1-89/002. 1989.
- EPA Office of Remedial Response. Superfund Exposure Assessment Manual. EPA/540/1-88/001. 1988.
- EPA Office of Health and Environmental Assessment. Exposure Factors Handbook. EPA/600/8-89/043. 1990.
- EPA Office of Emergency and Remedial Response. Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". OSWER Directive 9285.6-03. 1991.
- EPA Office of Research and Development. Health Effects Assessment Summary Tables (HEAST), Annual FY-1991. OERR 9200.6-303 (91-1). 1991.
- EPA Office of Health and Environmental Assessment. Integrated Risk Information System (IRIS). 1991.
- EPA Office of Air Quality Planning and Standards. Air/Superfund National Technical Guidance Study Series, Volume IV - Procedures for Dispersion Modeling and Air Monitoring for Superfund Air Pathway Analysis. EPA-450/1-89-004. 1989.
- Kimbrough, R.D., Mitchell, F.L., and Houk, V.D., Trichloroethylene: An Update. Journal of Toxicology and Environmental Health, 15:369-383. 1985.
- McKone, T.E., Human Exposure to Volatile Organic Compounds in Household Tap Water: The Indoor Inhalation Pathway. Environ. Sci. Technol., 21(12): 1194-1201. 1987. Andelman, J.B., Human Exposures to Volatile Halogenated Organic Chemicals in Indoor and Outdoor Air. Environ. Health Perspectives, 62:313-318. 1985.

APPENDIX A

**Screening Methods for
Inhalation Exposure**

SCREENING METHOD FOR ESTIMATING INHALATION EXPOSURE TO VOLATILE CHEMICALS FROM DOMESTIC WATER

1. Introduction

The following discussion has been developed to provide a screening method for estimating the indoor air concentrations of volatile chemicals from indoor water uses and the resulting human inhalation exposures, with an emphasis on showers. A computerized model titled MAVRIQ (Model for Analysis of Volatiles and Residential Indoor-Air Quality), which is under development, may also be used to refine the exposure estimates since it more accurately accounts for human behavioral and water use patterns.

This procedure evolved from research done by Julian Andelman at the University of Pittsburgh under funding from the Exposure Assessment Group at US EPA in Washington, DC. The references given provide a more detailed description of these procedures and related work.

2. When is Inhalation Exposure of Concern?

In order to determine the significance of the inhalation pathway the ratio of the vapor inhalation exposure to the water ingestion exposure can be calculated. Using Henry's Law Constant to obtain the equilibrium concentration in air, and setting a ratio of < 0.1 as criteria, the equation can be derived as follow:

$$\frac{\text{max inhalation exposure}}{\text{water ingestion exposure}} < 0.1$$

$$\frac{H C_w \times (20,000 \text{ L/day})}{C_w \times (2 \text{ L/day})} < 0.1$$

$$H < 10^{-5}$$

(1)

(2)

(3)

This will probably change to 1.0

Where C_w = contaminant concentration in water (mg/L)
 H = Henry's Law Constant (unitless)

The unitless Henry's Law Constant can be calculated by using the following equation.

$$H = H'/RT$$

where H' = Henry's Law Constant in atm-m³/mol
 R = gas constant in atm-m³/mol °K
 T = temperature in °K.

*Received 7/22/91 at
 Foth + Van Dyke, from
 USEPA's Exposure
 Assessment Group.*

Assuming a typical water temperature in a shower scenario of 40°C, RT is 2.6×10^{-2} atm·m³/mol.

Equation (3) suggests that for compounds with Henry's Law Constants of $< 10^{-5}$, the inhalation exposure would not exceed ingestion and is probably much less, therefore the inhalation pathway may not be of concern when compared to ingestion. Caution should be used when applying this criterion. If the ingestion exposure is significant, the inhalation exposure, although orders of magnitude less, may also be significant when considered separately.

3. Showering Exposure

The derivations and assumptions of the equations used to estimate exposure through the showering scenario are included in Appendix 1.

The exposure equation below accounts for the exposure during the showering time and the exposure during the period subsequent to the shower where there is a decay of the chemical concentration.

$$E_i = [C_{aAVG1} B t_1]_{\text{shower}} + [C_{aAVG2} B t_2]_{\text{after shower}} \quad (4)$$

Where: E_i = exposure [mg]

C_{aAVG1} = average air concentration during shower [mg/L]

C_{aAVG2} = average air concentration after shower [mg/L]

B = breathing rate [L/hr]

t_1 = shower period [hr]

t_2 = after shower period [hr]

C_{aAVG1} and C_{aAVG2} are estimated using equations (5) and (6) and (7) below.

$$C_{aAVG1} = C_{aMAX}/2 \quad (5)$$

$$C_{aAVG2} = C_{aMAX} \quad (6)$$

$$C_{aMAX} = \frac{C_w \times f \times F_w \times t_1}{V_b} \quad (7)$$

Where: C_{aMAX} = maximum air concentration in bathroom [mg/L]

C_w = water concentration [mg/L]

f = fraction volatilization [unitless]

F_w = water flow rate [L/hr]

V_b = bathroom size [L]

Default values for the variables in these equations are tabulated in table 1.

Using equations (4) through (7) and the average or most probable values from Table 1, one

can estimate the exposure during showering.

Example:

Assumptions

$$f = .75$$

$$F_w = 600 \text{ L/hr}$$

$$t_1 = 0.08 \text{ hr}$$

$$t_2 = 0.2 \text{ hr}$$

$$V_s = 10,000 \text{ L}$$

$$C_{s,MAX} = \frac{C_w (0.75)(600\text{L/hr})(0.08 \text{ hr})}{(10,000 \text{ L})}$$

$$= 3.6 \times 10^{-3} C_w$$

$$C_{s,AVG} = 1.8 \times 10^{-3} C_w$$

$$E_i = 1.8 \times 10^{-3} C_w(833\text{L/hr})(0.08\text{hr}) + 3.6 \times 10^{-3} C_w(833\text{L/hr})(0.2\text{hr})$$

$$= 0.72(\text{L}) C_w$$

TABLE 1

Variable	Value or Range	Reference
Fraction Volatilization (f)	0.5 - 0.9 (typical=0.75)	1
Water Flow Rate (F_w) [L/hr]	600 - 1,800 (mean=600)	2
Shower Period (t_1) [hr]	0.08 - 0.3 (mean=0.08)	2
After Shower Period (t_2)	0.2 (typical)	1
Bathroom size (V_b) [L]	8,300 - 9,800	3
Breathing Rate (B) [L/hr]	833 (20m ³ /day)	4

1. Andelman, J., Total Exposure to Volatile Organic Compounds in Potable Water, Chapter 20, Significance and Treatment of Volatile Organic Compounds in Water Supplies

2. U.S. Department of Housing and Urban Development, Residential Water Conservation Projects, March 1984, Contract H-5230

3. Giardino NJ, Gumerman E, Andelman JB, Wilkes CR, Small MJ, Borraro JE, Davidson CI (1990), Real-Time Air Measurements of Trichloroethylene in Domestic Bathrooms using Contaminated Water

4. U.S. EPA Factors Handbook

4. Whole House Exposure

Similarly, a one-compartment indoor-air model may be used to describe the range of average indoor-air concentrations that are likely to be encountered from a volatile organic chemical. The equation does not address the time and space variations that will be encountered throughout the day in the home. The exposure estimates obtained using the air concentrations from equation (8) do not include those that would occur at the point of water use, such as during showering.

The air concentration can be estimated by using the equation below.

$$C_a = \frac{WFH C_w f}{HV ER MC} \quad (8)$$

where; C_a = concentration in air (mg/m^3)
 C_w = concentration in water (mg/L)
WFH = water flow rate in whole house (L/day)
HV = house volume (m^3)
ER = exchange rate (air changes/day)
MC = mixing coefficient (unitless)
f = fraction of contaminant that volatilizes (unitless)

Table 2 shows a list of the ranges of values that these variables can take. An example of the use of equation (8) is presented below.

Assumptions

WFH = 723 (L/day)
HV = 177.7 (m^3)
ER = 13.7 - 58.8 (air changes/day)
MC = 0.15 - 1.0 (unitless)
f = 0.5 - 1.0 (unitless)

$$C_a = (0.03 - 2.0 \text{ [L}/\text{m}^3]) C_w \text{ [mg/L]} \quad (9)$$

TABLE 2

Variable	Value or Range	Reference
Water Flow Rate (WFH) [L/day]	723 (typical)	1
House Volume (HV) [m ³]	177.7 (typical?)	2
Exchange Rate (ER) [air changes/day]	13.7 - 58.8	3
Exchange Rate (ER) [air changes/day]	21.6 - 84.0	4
Mixing Coefficient (MC) (unitless)	0.15 - 1.0	5
Fraction Volatilization (f) [unitless]	0.5 - 1.0	6

1. U.S. Department of Housing and Urban Development (1984) Residential Water Conservation Projects
2. Axley J (1988) Progress Toward a General Analytical Method for Predicting Indoor Air Pollution in Buildings: Indoor Air Quality Modeling Phase III Report. NBSIR 88-3814
3. Grimsrud D.T., Sherman M.H., and Sonderegger R.C. (1982) Calculating infiltration: Implications for a Construction Quality Standard. Proceedings - ASHRAE/DEO Conference on Thermal Performance of the Exterior Envelopes of Buildings, Las Vegas, NV, Lawrence Berkeley Laboratory Report, LBL-9416. (refers to new houses)
4. ASHRAE (1985) Natural Ventilation and Infiltration. ASHRAE Fundamentals Handbook, Chapter 22, ASHRAE Inc., Atlanta, GA. (refers to older houses)
5. U.S. EPA (1987); Exposure to Volatilized Drinking Water Contaminants Via Inhalation - Importance Relative to Ingestion; Office of Drinking Water, Criteria and Standards Division, Health Effects Branch.
6. Cantor, K.P., Christman R.F., Ram, N.M., Significance and Treatment of Volatile Organic

**Compounds in Water Supplies; Chapter 20 - Total Exposure to Volatile Organic Compounds
in Potable Water; Julian B. Andelman**

Note: The ranges represent the average value and the maximum value. For the range presented in reference #4, the first value represents the median. Values presented for mixing coefficients are based on judgment.

Appendix A: Derivation of Equations

Nature of Volatilization Process

To assess the potential for VOC's to volatilize from water used indoors, it is useful to consider the equilibrium and rate processes involved. The relevant relationship describing the volatilization of a chemical and its subsequent equilibrium between the air and water phases is Henry's law

$$H = C_a / C_w \quad (1)$$

where H is the dimensionless Henry's law constant, and C_a and C_w (mass/volume) are the concentrations of the volatilized chemical in the air and water phases, respectively, at equilibrium.

Table 5 is a list of H constants at 25 °C for several organic chemicals of environmental concern, along with their vapor pressures and solubilities, the values being approximate, either calculated or taken directly from the compilation by Mackay and Shiu [19]. The H constants shown there encompass a

range greater than five orders of magnitude. Their vapor pressures and water solubilities are also quite different. Since the H values are predicted fairly well by the ratio of the vapor pressure of the pure material to its aqueous solubility, compounds such as carbon tetrachloride and tetrachloroethylene, with quite different solubilities and vapor pressures, can nevertheless have similar H values. Also it is essential to recognize that even a low vapor-pressure chemical, by virtue of its low solubility in water, has the potential to volatilize to the same extent as a high vapor-pressure chemical.

The maximum extent to which a chemical may be expected to volatilize in the home from indoor water uses can be estimated by considering the average quantities of water used within a home, F_w (L/h), along with typical air flow or infiltration rates F_a (L/h). For a family of four a typical ratio of F_a/F_w may be taken as 10^4 [4]. The ratio of masses of volatilized chemicals, r , in the two phases is given by

$$r = (C_a/C_w) (V_a/V_w) \quad (2)$$

where V_a and V_w are the quantities of air and water, respectively, used in a given period of time in the home.

In the steady-state one can assume that V_a/V_w equals F_a/F_w , and r_{MAX} is the maximum expected value for r when C_a/C_w equals H , such that

$$r_{MAX} = H(F_a/F_w) = 10^4 H \quad (3)$$

This indicates that in the steady-state, as water is used within the typical home and air infiltrates through it, for a chemical with an H value as low as 10^{-4} , r_{MAX} is unity, or about 50% volatilization will occur. ~~Since all the chemicals in Table 5~~ have H values greater than 10^{-4} , in each case, assuming Henry's law equilibrium is attained, one would expect substantial volatilization to occur in the home from normal uses of contaminated water as it is exposed to the indoor air.

The H constant will increase with temperature. Munz and Roberts [20] showed that for several volatile organic chemicals the temperature effect is given by

$$\log H = A' - B'/T \quad (4)$$

where A' and B' are constants for each chemical, and T is absolute temperature. For chloroform the measured A' and B' values were found to be 4.990 and 1729, respectively; and for carbon tetrachloride, 5.853 and ~~1716~~, respectively, the measurements being taken over the range of 10 to 30 °C. For example, using this equation for chloroform, the H values are 0.076 and 0.19 at 10 °C and 30 °C, respectively. The comparable values for carbon tetrachloride are 0.606 and 1.52. Thus the maximum extent of volatilization ~~that can occur~~ will increase markedly with temperature.

As discussed by Mackay and Yeun [21], the rate of volatilization of a chemical from water is dependent on its molecular-diffusivity properties. Often a two-resistance model

is used to describe the process in which the volatilizing chemical has to first diffuse across a liquid film at the air water interface, followed by diffusion across the air film. Mackay and Yeun measured volatilization rates in a wind wave tank for 11 organic compounds with varying Henry's law constants. They confirmed the validity of the two-resistance model, and showed the effects of solute diffusivity and temperature. The chemicals studied included several halogenated VOC's, including chlorobenzene, carbon tetrachloride, 1,2-dibromoethane, and 1,2-dichloropropane, as well as benzene and toluene, and several ketones and alcohols. They showed that no interactions occur when solutes volatilize simultaneously, and concluded that the mass-transfer rate was predominantly liquid-phase resistant for many of these chemicals.

The two-resistance model expressing the mass flux, F_a (mol/m²s), can be written as

$$F_a = K(C_a - C_w/H) \quad (5)$$

where K is the overall, two-resistance mass-transfer coefficient (m/s), C_a is the solute concentration in air (mol/m³) and C_w that in water. The overall mass transfer is a product of the flux and the surface area exposed, so that, for example, small droplets in a shower with a greater surface area would be expected to have a greater rate of volatilization per unit time than would the same mass of larger droplets with a lower surface area/mass ratio.

Mackay and Yeun concluded that the mass-transfer coefficient in either the liquid or gas phase was most likely dependent on the Schmidt number, Sc , which is the dimensionless ratio of viscosity/(density x diffusivity), in the respective phase. The two-resistance model describes the K in terms of liquid and gas phase transfer coefficients, K_L and K_G , respectively, such that

$$1/K = 1/K_L + 1/HK_G \quad (6)$$

They showed that for their data K_L was proportional to $3.41 \times 10^{-3} Sc_L^{-0.5}$, while K_G was proportional to $4.62 \times 10^{-2} Sc_G^{-0.67}$. The Sc_G and Sc_L values for the 12 compounds did not differ greatly, ranging from 0.72-1.07 for Sc_G , and 939-1177 for Sc_L at 20 °C. However, the H values varied considerably by almost four orders of magnitude. For the smallest H -value compound, 1-butanol, the K_G term dominated to establish the overall K , while for the high H -value compounds like benzene and carbon tetrachloride, liquid-film transfer was the dominant rate-controlling step, the $1/HK_G$ term being negligible in Equation 8. The overall mass-transfer coefficients measured were thus quite different at these two extremes. For example, the ratio of mass-transfer coefficients for benzene to that of 1-butanol varied from 14 to 20. In contrast, for those compounds where K_L dominated, the K values did not vary much, as expected, since their Sc_L values were quite similar, and H no longer played a significant role in determining K . Thus, in one series of determinations of mass-transfer coefficients, Mackay and Yeun measured K values of 51.1, 51.1,

and 45.3 (10^6 m/s), respectively, for benzene, carbon tetrachloride, and 1,2-dibromoethane, their Sc_L values being 1021, 1062, and 1075, respectively.

This analysis indicates that one should be able to compare and predict the K values among compounds based on fundamental molecular properties and H values, to the extent that this two-resistance model applies to the volatilization from indoor water uses. They observed that the use of the K_L dependency on $Sc_L^{-0.5}$ predicts a 2.8% temperature increase in K per degree.

Equation 5 for the mass-transfer or flux at the water air-interface predicts that when the air concentration, C_a , is negligible, meaning a small buildup of chemical in the receiving air, then the rate of mass transfer is directly proportional to the concentration of volatilizing chemical in the water. This is of importance in that one could then extrapolate the percent volatilization at a high concentration in the feed water to predict the same fractional volatilization at a low-feed concentration. At the same time, even if the buildup in the air did occur, however, and its removal were first order in concentration, one could still extrapolate to the lower feed concentration.

There is independent evidence in laboratory studies that the mass-transfer coefficient may be reasonably constant over several orders of magnitude of concentration [22]. For 1,2-dichloroethane in the range of 1 g/L to 10 ug/L the coefficient

of variation of mass-transfer coefficient was found to be $\pm 6.31\%$; for 1,1,1-trichloroethane it was $\pm 5.42\%$ over a range of concentration of 0.05 g/L to 30 ug/L.

In summary, the H constant will limit the maximum volatilization that can occur in indoor water uses. However, except for a few still-water systems in the home, such as water in a toilet bowl, many water uses are flowing or are of short-term duration in which the rate of volatilization will be limiting and equilibrium not reached. In those instances the mass-transfer coefficients become the principal controlling factor for the relative releases of different volatile and semi-volatile chemicals. Even here, however, the H constant is of importance in that it will influence the magnitude of the mass-transfer coefficient, as well as the extent to which the flux for volatilization at the water-air interface will be reduced as the air concentration builds up.

Finally, the water-air interfacial areas and temperatures of the water uses are critical determining factors in the rate of mass transfer; and certainly the H constants will increase with temperature as well. Thus, one can expect that since the various indoor water uses involve different quantities and flows of water, residence times in the water appliances and uses, degrees of mixing and turbulence, and temperatures, the extents of volatilization among the water uses, even for a given chemical, should vary.

Values for transfer efficiencies among water uses in a typical home have been determined for radon by Prichard and Gessell [10]. As shown in Table 6, the transfer efficiencies (percent volatilization) were found to vary from 30 to 90% among the water uses, the volume use-weighted mean being about 50%.

Laboratory Shower Experiments

We have performed studies on volatilization of chemicals from laboratory and full-size shower and bath systems in which chemicals have been added to the water [3-8]. In our typical laboratory shower experiments with chloroform shown in Figure 1 [23], the concentration of the chemical in the air pumped from the chamber is measured continuously as the shower water flows, and continues to be measured after the chemical injection is terminated, but with the shower still flowing. The peak concentrations shown in Figure 1 occur shortly after terminating the injection of chemical. In these studies we have also monitored the drainwater leaving the shower chamber for mass-balance purposes.

For this system the equation describing the rate of change of air concentration, C_a (mg/L), can be expressed as [6]

$$V_a(dC_a/dt) = k(C_w - C_a/H) - F_a C_a \quad (7)$$

where V_a (L) is the volume of the shower chamber, C_w (mg/L) the concentration of the chemical in the feed water, F_a (L/min) the air flow rate through the chamber, and k (L/min) the

volatilization mass-transfer coefficient. When the feed concentration is terminated, the volatilization source term becomes zero and Equation 7 reduces to

$$V_a(dC_a/dt) = -F_a C_a \quad (8)$$

the integrated form being

$$\ln C_a = \ln C_{a,INITIAL} - (F_a/V_a)t \quad (9)$$

As expressed by Equation 7, we find that the volatilization source term $k(C_w - C_a/H)$ does indeed reduce significantly with time as C_a increases. For example, in the experiment with a chloroform feed of 1.84 mg/L shown in Figure 1, at 10, 30, and 50 min, the instantaneous fractional rate of volatilization, f , was 0.82, 0.70, and 0.62, respectively. This is consistent with our experimental observation that the C_a/C_w ratio for air and water leaving the chamber was found to be less than the H value for chloroform, but that the latter value of about 0.15 was gradually approached during the shower experiment [23], thus gradually inhibiting the volatilization rate.

We have also found in our experiments with both chloroform and trichloroethylene (TCE), that during the decay period (following the termination of the chemical in the shower feed) significant quantities of the volatilized chemical in the shower chamber air redissolves in the flowing water, as measured in the drainwater. Thus, Equations 8 and 9 are not quite accurate, since there is this additional decay route.

As shown in Figure 1 for chloroform, as expected the air concentration due to volatilization increases with temperature and concentration of the feed water in the shower experiments. Also as expected, we have found that increased air flow reduces the concentration of volatilized chemical in the chamber air and at the same time increases the rate of volatilization, since the rate of approach to Henry's law equilibrium is reduced. Rates of volatilization for chloroform and TCE ranged from about 50 to 90%, depending on temperature and other shower conditions, with chloroform volatilization typically lower than that for TCE.

Modeling Shower and Whole House Exposures

One can estimate the shower and whole house exposures by the use of simple, one-compartment modeling. For example, integrating Equation 7 and assuming that C_a/H is negligible compared to C_w , one obtains an expression for the change in C_a with time in a chamber

$$\ln (1 - C_a F_w / k C_w) = - (F_w / V_a) t \quad (10)$$

The assumption that C_a/H is negligible implies that the rate of volatilization in the shower is constant. In that case it can be shown that k equals $f F_w$, where f is the fraction of chemical that volatilizes from the feedwater whose flow rate is F_w (V/t).

Although, as noted above there is a gradual decrease in f values with time during the shower experiments, this will not substantially affect the estimated average values of C_a that will

be used to calculate exposures. Using Equation 10 one can calculate the maximum air concentration that will be achieved in a one-compartment shower or bath. For small values of $(F_s/V_s)t$ (the magnitude of which will be considered below), Equation 10 reduces to a simple linear form

$$C_s = ktC_w/V_s \quad (11)$$

Thus, after a given shower period, t , this is also the maximum concentration, C_{sMAX} ,

$$C_{sMAX} = ktC_w/V_s \quad (12)$$

Also, the average concentration, C_{sAVG} , would be $C_{sMAX}/2$ since C_s increases linearly with time

$$C_{sAVG} = ktC_w/2V_s \quad (13)$$

For the purpose of estimating possible shower exposures, it will be assumed that the concentrations during the shower period itself, as well as subsequently while a person remains in the bathroom, will be the same in the shower and bathroom. In fact, our measurements in a full-size shower show that there is indeed a difference between the two, and that the system should be more appropriately treated as a two-compartment system [24]. For precise modeling of the exposures, this difference should be considered, but as an approximation it will be neglected here.

Subsequent to the showering period there will be a decay of the air concentrations in the bathroom due to normal exchange of air. During this period the person in the bathroom will continue to be exposed to the volatilized chemicals in the air. The decay

of C_s is represented by

$$\ln (C_s/C_{sMAX}) = - (F_s/V_s)t \quad (14)$$

For small values of $(F_s/V_s)t$ this equation linearizes to

$$C_s = C_{sMAX} [1 - (F_s/V_s)t] \quad (15)$$

The average concentration during this period, C_{sAVG} , is

$$C_{sAVG} = (C_s + C_{sMAX})/2 \quad (16)$$

Combining Equations 15 and 16, one obtains

$$C_{sAVG} = C_{sMAX} [1 - F_s t / (2V_s)] \quad (17)$$

In many cases, the $F_s t / (2V_s)$ term in Equation 17 is likely to be substantially smaller than unity, so that as an approximation during the decay period one can assume that $C_{sAVG} = C_{sMAX}$, at least for the purposes of estimating the magnitude of inhalation exposures.

One can use these equations to estimate the C_{sAVG} values for various shower-water flow and bathroom characteristics. In an Australian survey of water uses, distributions of average shower-water flow rates and duration were reported for about 2,500 households [25]. The geometric mean for the shower flow rates, F_s , was about 8 L/min (about 500 L/h), and about 6 min for the shower duration, which will be specified as t_d , and typically taken as 0.1 h. These values will be utilized here to estimate C_s values using the above equations. In a study of modern houses in one heating season the geometric mean for air exchange rates was reported to be 0.53 h^{-1} [13]. This value will be used for the bathroom, along with a value for its size, V_s , of 10,000 L.

Thus, the F_s for the bathroom will be $0.53V_s$, or about 5,000 L/h. Thus, for a shower period of 0.1 h, or a decay period of 0.2 h, with $(F_s/V_s)t$ values of 0.053 and 0.115, respectively, the approximation of linearizing Equations 10 and 14 involves errors of less than one percent.

The above equations and data can be utilized to estimate the average air concentrations to which people are exposed in bathrooms during and after showering. As discussed earlier, the fractional volatilization rate in our shower experiments has been found to range from 0.5 to 0.9, depending on the specific chemical, water temperature, and other factors. For the purposes of estimating a typical value, we will use an f value of 0.75.

Using Equation 12 and the fact that k equals fF_s , yields

$$C_{MAX} = C_w f F_s t_d / V_s \quad (18)$$

One can use typical values for the variables indicated above to obtain

$$C_{MAX} = C_w (0.75) (500) (0.1) / 10^4 = 3.75 \times 10^{-3} C_w \quad (19)$$

The value for C_{MAX} would be one-half this, or $1.9 \times 10^{-3} C_w$. It is interesting to note that Prichard and Gesell [10] predicted that for a five-minute shower using 75 L of water and with 65% volatilization in a 30,000 L room, the average radon air concentration would be $1.6 \times 10^{-3} C_w$. Similarly, McCone [9] modeled several low molecular-weight organics volatilizing with multiple family use of a bathroom in the early morning hours and calculated typical bathroom air concentrations of $5 \times 10^{-3} C_w$.

Such predicted air concentrations will be highly dependent on a variety of factors, including the nature of the volatilizing chemical, geometry and air exchange between the shower and surrounding room, ~~water temperature~~, and water flow rate. Nevertheless, these can be assessed to determine the likely range of bathroom air concentrations that can be expected in homes.

It is also of interest to estimate the inhalation exposures in the shower and bathroom, ~~and compare them to the likely ingestion exposures~~. Inhalation exposure, E_i (mg), can be defined as the product of C_a , the breathing rate, B (L/h), typically 1,000 L/h for an adult, and the exposure time, t .

$$E_i = C_a B t \quad (20)$$

As an example, one can use this equation to estimate the exposures during a 0.1 h showering time, using the value of C_{avg} above of $1.9 \times 10^{-3} C_w$. Also as noted above, during a 0.2 h period subsequent to the ~~shower, the decay will not be significant~~, so that the C_{avg} during this period can be taken to be C_{max} , namely $3.75 \times 10^{-3} C_w$. Thus, one can calculate the E_i for the combined 0.1 h shower and 0.2 h subsequent period in the bathroom as the sum of two terms using Equation 20, to give

$$E_i = [C_{avg} B t]_{shower} + [C_{avg} B t]_{decay} \quad (21)$$

Inserting the appropriate values, one obtains

$$E_i = 1.875 \times 10^{-3} C_w (1000) (0.1) + 3.75 \times 10^{-3} C_w (1000) (0.2) \quad (22)$$

Thus, E_i has the value $0.94 C_w$, where the units of C_w are mass/L.

This is the inhalation exposure in the bathroom during the shower and subsequent to it while the bather remains in the bathroom, and is approximately equivalent to the exposure that would occur from ingesting one liter of the water. However, several occupants of a home may take a shower during a period when the volatile chemical air concentration in a bathroom has not decayed and builds up to levels higher than one would predict for a single bather. In that instance, the exposures could be substantially higher than would be predicted by the above relationship.

Similarly, we have used a simple predictive equation, based on a one-compartment indoor-air model, to describe the range of average indoor-air concentrations that are likely to be encountered from a chemical volatilizing at an average rate of 50% from all water uses, as discussed above to be a typical value for radon. The relationship we have obtained for the expected range of indoor-air concentrations is [7]

$$C_a = (0.1 \text{ to } 5) \times 10^{-4} C_w \quad (23)$$

where C_a is the average indoor-air concentration (mg/L), generated by the corresponding average water concentration, C_w (mg/L). Thus, for example, a water concentration of 1 mg/L would be expected to generate between 1×10^{-5} to 5×10^{-4} mg/L average air concentration in the home. This, of course, does not address the time and space variations that will be encountered throughout the day in the home. It is interesting to note that

Nazaroff et al. [13] have similarly made estimates of the likely indoor-air concentrations of radon for U.S. homes by the water volatilization route. The geometric mean in their factor applicable to Equation 23 is 0.65×10^{-4} , within our range of predicted values. Also, their range of one standard deviation around the mean corresponds to the following equation

$$C_a = (0.23 \text{ to } 1.87) \times 10^{-4} C_w \quad (24)$$

also within our predicted range. McKone [9] has similarly estimated household air concentration for several volatilizing chemicals, predicting an average C_a ranging from 2×10^{-3} to 1.2×10^{-4} mg/L in air for a C_w of 1 mg/L in water, also within the range of that predicted by Equation 23.

One can use these air concentration predictions to estimate the likely inhalation exposures, E_i , for an adult during a 24-hour residence period in a house. Combining Equations 20 and 23 one obtains

$$E_i = (0.1 \text{ to } 5) (10^{-4}) (1000) (24) C_w = (0.2 \text{ to } 10) C_w \quad (25)$$

Since the C_w units here are mass/L, a 1 mg/L water concentration corresponds to a range of inhalation exposures of 0.2 to 10 mg per day, in comparison to 2 mg per day for the ingestion of 2 liters of that water. It should be noted that these inhalation exposure estimates do not include those that would occur at the point of water use, such as during showering. As discussed above, the latter exposures can be comparable to those from direct ingestion.

There is a remarkable consistency in the above range of likely predicted average indoor-air concentrations from the totality of indoor water sources. Nevertheless, there are a number of factors to be considered in refining these estimates and developing a useful and simple predictive relationship that can be applied by those responsible for exposure assessments in specific situations. They can be categorized as follows:

- a) chemical characteristics that affect the rate and extent of volatilization, including soap and detergent use
- b) water use factors that affect the "source strength" and its time and location variability
- c) chemical characteristics that influence the behavior and interactions of the volatilized chemicals with "sinks", typically high surface area materials in the home; also the specific nature, amounts, and locations of these sinks
- d) house structure and indoor-air flow regimes that transport the volatilized chemicals throughout the home
- e) personal behavior and home occupancy factors that determine an individual's exposure.

The simple indoor-air models mentioned above generally are not sufficiently specific to address all the above factors, although they can and have been evaluated for some indoor-air pollution sources other than those from water [26].

The potential interactions between surfaces in homes and organic vapors released from water into indoor air have not been

studied and need to be evaluated. For some chemicals it may be appropriate to incorporate these interactions into the volatilization, indoor-air exposure model. One study of the interaction of volatile organic chemicals with materials used in the home examined three surfaces [27]: plywood, nylon carpeting, and wool carpeting. The study focused on twenty volatile organic chemicals, including alkanes, aromatics, alcohols, esters, ketones, aldehydes, terpenes, and chlorinated hydrocarbons. They showed clear interactions between the gaseous organic chemicals and the surfaces. For example, in one experiment wool carpeting became essentially saturated with lindane within about one day.

In order to determine the role of such "sink" interactions there are three broad questions that need to be addressed:

- 1) Which classes of organic/surface systems demonstrate significant sorption effects?
- 2) What are the appropriate equilibrium and kinetic models for the sorption process for the organic/surface systems of interest?
- 3) How can this equilibrium and kinetic information be incorporated into a water-volatilization, indoor-air quality model?

Summary and Conclusions

VOC's have the potential for causing substantial human exposures from indoor uses of contaminated water by non-ingestion

routes, namely inhalation following volatilization from water, as well as by skin contact. The latter exposures have been estimated to be comparable to those from direct ingestion of water, although published research in this area is scanty.

Measurements in homes have shown that VOC's can be detected in indoor air following the use of contaminated water. Scaled-down and full-size laboratory bath and shower studies for such VOC's as chloroform and trichloroethylene have shown that a variety of factors can affect the extent of volatilization, found to be typically in the range of 50 to 90%. These include the nature of the volatilizing chemical, water temperature, air and water flow rates, and nature of the water use (e.g., bath versus shower).

The Henry's law equilibrium constants, H , predict that even chemicals with low vapor pressures may be expected to volatilize substantially, provided their water solubilities are also low. Thus, so-called semi-volatile organic chemicals have the potential to volatilize and cause inhalation exposures. Also, chemicals with varying H values may nevertheless volatilize at comparable rates.

Modeling and estimates of inhalation exposures to VOC's indicate that for the bather these exposures during and directly after a shower can be comparable to that from direct ingestion of the contaminated water. Also, when all water uses are considered, the inhalation exposures to all inhabitants of a home

may be substantially larger than that from direct ingestion, even without considering the inhalation exposures at the point of water use. However, additional research is required to more specifically and precisely quantify these exposures to encompass the full range of home characteristics, as well as personal water uses and occupancy factors.

Because the non-ingestion exposures to VOC's in indoor water uses are likely to be comparable to or greater than those from direct ingestion, it would be prudent to consider this in establishing regulatory limits in drinking water, as well as the need to restrict all indoor water uses when it is judged that there is a significant health risk from the direct ingestion of a contaminated water.

Acknowledgments

The author would like to acknowledge the fruitful discussions and collaborations with several students and colleagues which have provided useful perspective and data for much of this research. These include Nicholas Giardino, Thomas Jackman, Steve Myers, Lynn Wilder, Amy Couch, and William Thurston at the University of Pittsburgh; also, John Borrazzo and Cliff Davidson at Carnegie-Mellon University.

This research has been funded in part under cooperative agreement CR812761-02 between the U.S. Environmental Protection Agency and the Center for Environmental Epidemiology of the

University of Pittsburgh. This manuscript has not been subjected to EPA peer and administrative review policy, does not necessarily reflect its views, and no official endorsement should be inferred.

References

1. Wallace LA, ED Pellizzari, TD Hartwell, R Whitmore, C Sparacino, and H Zelon (1986) "Total Exposure Assessment Methodology (TEAM) Study: Personal Exposures, Indoor-Outdoor Relationships, and Breath Levels of Volatile Organic Compounds in New Jersey," Environment International 12:369-387.
2. Wallace, LA (1987) The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis: Volume I (Washington, DC: Office of Research and Development, U.S. Environmental Protection Agency).
3. Andelman, JB (1985) "Human Exposures to Volatile Halogenated Organic Chemicals in Indoor and Outdoor Air," Environmental Health Perspectives 62:313-318.
4. Andelman, JB (1985) "Inhalation Exposure in the Home to Volatile Organic Contaminants of Drinking Water," The Science of the Total Environment 47:443-460.
5. Andelman, JB, SM Meyers, and LC Wilder (1986) "Volatilization of Organic Chemicals from Indoor Uses of Water", in Chemicals in the Environment, JN Lester, R Perry, and RM Sterritt, Eds. (London: Selper Ltd.), 323-330.
6. Andelman, JB, A Couch, and WW Thurston (1986) "Inhalation Exposures in Indoor Air to Trichloroethylene from Shower Water," in Environmental Epidemiology, FC Kopfler, and GC Craun, Eds. (Chelsea, MI: Lewis Publishers, Inc.), 201-213.
7. Andelman, JB, LC Wilder, and SM Myers (1987) "Indoor Air Pollution from Volatile Chemicals in Water," in Proceedings of The 4th International Conference on Indoor Air Quality and Climate, INDOOR AIR '87, Volume 1 (Berlin: Institute for Water, Soil and Air Hygiene), 37-41.

8. Giardino, NJ, JB Andelman, JE Borrazzo, and CI Davidson (1988) "Sulfur Hexafluoride as a Surrogate for Volatilization of Organics from Indoor Water Uses," JAPCA 38(3):278-280.
9. McKone, TE (1987) "Human Exposure to Volatile Organic Compounds in Household Tap Water: The Indoor Inhalation Pathway," Environ. Sci. Technol. 21:1194-1201.
10. Prichard, HM, and TF Gesell (1981) "An Estimate of Population Exposures Due to Radon in Public Water Supplies in the Area of Houston, Texas," Health Physics 41:599-606.
11. Hess, CT, CV Weiffenbach, and SA Norton (1983) "Environmental Radon and Cancer Correlations in Maine," Health Physics 45:339-348.
12. Cross, FT, NH Harley, and W Hoffman (1985) "Health Effects and Risks from ^{222}Rn in Drinking Water," Health Physics 48(5):649-670.
13. Nazaroff, WW, SM Doyle, AV Nero, and RG Sextro (1987) "Potable Water as a Source of Airborne ^{222}Rn in U.S. Dwellings: A Review and Assessment," Health Physics 52(3):281-295.
14. Klaassen, CD (1980) "Absorption, Distribution and Excretion of Toxicants," in Toxicology, J Doull, CD Klaassen, and MO Amdur, Eds. (New York: Macmillan), 28-51.
15. Drinking Water and Health (1977) (Washington, DC: National Academy Press).
16. Brown, HS, DR Bishop, and CA Rowan (1984) "The Role of Skin Absorption as a Route of Exposure for Volatile Organic Compounds (VOCs) in Drinking Water," Am. J. Public Health 74:479-484.
17. Harris, RH, JV Rodricks, CS Clark, and SS Papadopoulos (1987) "Adverse Health Effects at a Tennessee Hazardous Waste Disposal Site," in Health Effects From Hazardous Waste Sites, JB Andelman, and DW Underhill, Eds. (Chelsea, MI: Lewis Publishers, Inc.), 221-240.
18. Wester, RC, M Mobayan, and HI Maibach (1987) "In Vivo and In Vitro Absorption and Binding to Powdered Stratum Corneum as Methods to Evaluate Skin Absorption of Environmental Chemical Contaminants From Ground and Surface Water," J. Tox. Env. Health 21:367-374.

19. Mackay, D, and WY Shiu (1981) "A Critical Review of Henry's Law Constants for Chemicals of Environmental Interest," J. Phys. Chem. Ref. Data 10(4):1175-1199.
20. Munz, C, and PV Roberts (1987) "Air-Water Phase Equilibria of Volatile Organic Solutes," J. AWWA 79:62-69.
21. Mackay, D, and ATK Yeun (1983) "Mass Transfer Coefficient Correlations for Volatilization of Organic Solutes from Water," Environ. Sci. Technol. 17(4):211-217.
22. Rathbun, RE, and DY Tai (1984) "Volatilization of Chlorinated Hydrocarbons from Water," in Gas Transfer at Water Surfaces, W Brutsaert, and GH Jirka, Eds. (Dordrecht, Holland: D. Reidel Publishing Company), 27-34.
23. Jackman, T (1988) "Volatilization of Chloroform and Trichloroethylene in a Model Shower System," MSc Thesis, University of Pittsburgh, Pittsburgh, PA.
24. Giardino, N (1987) "An Indoor Air Pollution Study Using Sulfur Hexafluoride Volatilized from a Model Shower System," MSc Thesis, University of Pittsburgh, Pittsburgh, PA.
25. James, IR, and MW Knuiam (1987) "An Application of Bayes Methodology to the Analysis of Diary Records From a Water Use Study," J. Amer. Stat. Assoc. 82:705-711.
26. Wadden, RA, and PA Scheff (1983) Indoor Air Pollution (New York: John Wiley and Sons).
27. Seifert, B, and HJ Schmahl (1987) "Quantification of Sorption Effects for Selected Organic Substances Present in Indoor Air," in Proceedings of The 4th International Conference on Indoor Air Quality and Climate, INDOOR AIR '87, Volume 1 (Berlin: Institute for Water, Soil and Air Hygiene), 252-256.

Appendix B: Assumptions and Uncertainties

- Equation (4) does not account for the concentration of the chemical in the air remaining from previous showers taken by other members of the family.
- The use of Equation (4) also assumes that $(F_a/V)t$, where F_a is the air flow rate, is small compared to unity, which implies that the relationship between concentration in air and time is linear.
- Equation (4) also assumes that C_s/H during the course of the shower is small compared to C_w ; which implies that the volatilization rate in the shower is constant.
- The use of Equation (6) assumes that $tF_a/2V_a$ is small compared to unity so that the concentration during the decay period after the shower, C_{AVERD} , can be approximated by C_{MAX} .
- The exchange between the air in the shower chamber and that in the bathroom is so rapid that the combined volume of these two compartments can be treated as a single chamber with a single concentration of volatilized chemical.
- Equation (4) does not account for the exchange rate that occurs when an exhaust fan is turned on. Modeling results using the Model for Analysis of Volatiles and Residential Indoor-air Quality (MAVRIQ) indicate that exposure is reduced by 20 % if exhaust fan is used.
- The range of volatilization fraction in Table 1 is based on experiments conducted with trichloroethylene, chloroform and dibromochloropropane. The relationship between these volatilization rates, Henry's Law Constant and molecular weight is not known yet. Summarized below are the experimental results for these three chemicals under approximately the same conditions.

<u>Chemical</u>	<u>T (°C)</u>	<u>H (unitless)</u>	<u>% Volatilized</u>
Trichloroethylene	46	1.14	81.8
Chloroform	42	0.35	56
Dibromochloropropane	42	0.03	22.8

- Equation (8) treats the whole house as one compartment model.