Human Health Risk Assessment and Development of Remedial Action Levels for Cypress Creek Sub-Area III Memphis, Tennessee

REVISED REPORT

Prepared for:

Memphis Environmental Center
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Prepared by:

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29 June 2006

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June 29, 2006

Mr. J. M. Apple, Director
Tennessee Department of Environment and Conservation
Division of Solid Waste Management
L & C Tower, 5th Floor
401 Church Street
Nashville, Tennessee 37243-1535

Re: Revised Report on Human Health Risk Assessment and Development of Remedial Action Levels for Cypress Creek Sub-Area III
Velsicol Chemical Corporation, Memphis Facility
Facility Identification No. TND 00 702 4664
Tennessee Permit No. TNHW-109

Dear Mr. Apple:

Enclosed is Velsicol's *Revised Report on Human Health Risk Assessment and Development of Remedial Action Levels for Cypress Creek Sub-Area III, Memphis, Tennessee.* This revision incorporates all of the changes the were made in response to comments received from the U.S. Environmental Protection Agency (EPA) and the Tennessee Department of Health (TDH) on the May 10, 2006 version of the report. We understand that TDEC, EPA and TDH have reviewed and have accepted all of Velsicol's responses to those comments and related report revisions, which have been communicated over the past few weeks. We look forward to receiving TDEC's approval of this report.

I certify under penalty of law that this document and all attachments were prepared under my direction or supervision in accordance to a system designed to assure that qualified personnel properly gather and evaluate the information submitted. Based on my inquiry of the person or persons who manage the system, or those persons directly responsible for gathering the information, the information submitted is, to the best of my knowledge and belief, true, accurate, and complete. I am aware that there are significant penalties for submitting false information, including the possibility of fine and imprisonment for knowing violations.

Sincerely,

Memphis Environmental Center, Inc.

Gary J. Hermann, P.E.
Senior Environmental Projects Manager

Enclosure

c: Mike Apple, TDEC (three hard copies, one electronic copy)
Jon Johnston, EPA
Bonnie Bashor, Tennessee Department of Health
Norm LaChapelle, Memphis and Shelby County Health Department
Paul Patterson, Memphis Public Works
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29 June 2006

Mr. Gary Hermann, P.E.
Memphis Environmental Center, Inc.
5909 Shelby Oaks Drive, Suite 146
Memphis, TN 38134-7311

Re: ***Human Health Risk Assessment and Development of Remedial Action Levels for Cypress Creek Sub-Area III, Revised Report***
Velsicol Chemical Corporation, Memphis, Tennessee

Dear Mr. Hermann:

Please find enclosed 15 bound and 1 unbound hard copies of the revised report entitled *Human Health Risk Assessment and Development of Risk-Based Remedial Action Levels for Cypress Creek Sub-Area III,* dated 29 June 2006. Reviewers from the US Environmental Protection Agency and the Tennessee Department of Health provided comments on the 10 May 2006 version of this report. The enclosed revised report has been modified to be consistent with GeoSyntec's responses to those comments provided in our letter dated 6 June 2006, as well as further clarifications provided via email on 22 June 2006.

If you have any questions regarding this revised report, please do not hesitate to give me a call.

Sincerely,

Christopher Saranko, Ph.D., DABT

Enclosures

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LIST OF ACRONYMS

AF	Adherence Factor
AT	Averaging Time
ARARs	Applicable or Relevant Appropriate Requirements
BW	Body Weight
CAP	Corrective Action Program
CERCLA	Comprehensive Response, Compensation, and Liability Act
COPC	Chemical of Potential Concern
CSF	Cancer Slope Factor
CEM	Conceptual Exposure Model
EF	Exposure Frequency
EPC	Exposure Point Concentration
FDA	Food and Drug Administration
FDEP	Florida Department of Environmental Protection
HI	Hazard Index
HQ	Hazard Quotient
K_{ow}	Organic Carbon Partition Coefficients
LOAEL	Lowest Observed Adverse Effect Level
MEC	Memphis Environmental Center
NCP	National Contingency Plan
ND	Not Detected
NHANES	National Health and Nutrition Examination Survey
NOAEL	No Observed Adverse Effect Level
OSWER	Office of Solid Waste and Emergency Response
PEF	Particulate Emission Factor
Q/C	Term for measure of air dispersal
RAGS	Risk Assessment Guidance for Superfund
RAL	Remedial Action Level
RCRA	Resource Conservation and Recovery Act
RfD	Reference Dose
RME	Reasonable Maximum Exposure
SA	Skin Surface Area
SSG	Soil Screening Guidance
TDEC	Tennessee Department of Environment and Conservation
TDOH	Tennessee Department of Health
TERA	Toxicology Excellence for Risk Assessment
UCL	Upper Confidence Limit
EPA	United States Environmental Protection Agency
VF	Volatilization Factor

1.0 INTRODUCTION

The Tennessee Department of Environment and Conservation (TDEC) directed Velsicol Chemical Corporation (Velsicol) to develop soil cleanup or removal levels for pesticides in Sub-Area III of Cypress Creek (TDEC, 2004). In response to that request, Velsicol has developed a technical approach to establish a remedial action level (RAL) for the cleanup of pesticides in the soils. This report presents the results of a risk-based evaluation of all relevant surface soil data collected from Sub-Area III to date. It also incorporates changes and refinements to Velsicol's technical approach in response to the input received from TDEC, the U.S. Environmental Protection Agency (EPA), the Tennessee Department of Health (TDOH), the Memphis-Shelby County Health Department, and the non-profit organization Toxicology Excellence for Risk Assessment (TERA). This analysis indicates that selection of a single RAL based on the indicator compound dieldrin, with consideration of secondary contributors to risk, will address potential risks from cyclodiene chemicals in Sub-Area III.

Multiple risk evaluations conducted during the various phases of the Cypress Creek investigation (MEC, 2002a, 2002b, 2003) consistently indicate that dieldrin is a significant contributor to potential risk associated with exposure to Sub-Area III soils. For this reason, a comprehensive analysis was performed to evaluate the use of dieldrin as an indicator compound for establishing a risk-based RAL. The results of this analysis support the use of dieldrin for this purpose. In addition to a RAL based on dieldrin, a second criterion was also developed for other chemicals of concern to ensure that any RAL selected based on dieldrin will also provide a safe level of exposure to other chemicals. The use of dieldrin in this manner will significantly streamline and expedite the remediation process. It will also foster a consistent approach to corrective action throughout Sub-Area III.

The remainder of this document is organized as follows:

Section 2 presents background information on the Cypress Creek Sub-Area III investigation;

Section 3 presents the methodology used in developing RALs based on dieldrin;

Section 4 presents a conceptual exposure model for the evaluation of theoretical human health risk focused on complete exposure pathways including a discussion of the potential for uptake of cyclodiene chemicals by vegetables;

Section 5 presents the equations and factors used to develop the RALs and the methodology used to calculate cumulative risks associated with exposure to chemicals of potential concern at each property;

Section 6 provides a discussion of the selection and implementation of RALs; and

Section 7 provides a list of references cited in the report.

2.0 BACKGROUND INFORMATION

Beginning in 2001, Velsicol has performed a series of soil investigations along the Cypress Creek Stormwater Channel downstream of Velsicol's chemical manufacturing facility located at 1199 Warford Street in Memphis, Tennessee (MEC, 2002a). These investigations are a component of an ongoing Resource Conservation and Recovery Act (RCRA) Corrective Action Program (CAP) and have focused on understanding the distribution of chlorinated cyclodiene pesticides and related chemicals.

The ultimate purpose of these investigations is to support the evaluation of potential human health risks associated with exposure to these chemicals. This is done through a regulatory process called *Risk Assessment*, which uses information about the toxicity of chemical substances to estimate a theoretical level of risk for people who might be exposed to those substances. This report focuses on the use of the risk assessment process to develop RALs for residential properties within Sub-Area III.

The most recent investigative phase has focused on the Sub-Area III Study Area, which is the concrete-lined, 2.4-mile long section of Cypress Creek from Jackson Avenue to Evergreen Street. There are approximately 170 properties adjacent to Cypress Creek in Sub-Area III. Land use at these properties is predominately residential, with a lower proportion of commercial, industrial, and open space properties. In 2003, Velsicol's Memphis Environmental Center (MEC), supported by the EPA and TDEC, collected a total of 164 surface soil samples from 84 of these properties.

In most cases, two five-point composite surface soil samples were collected at each property to gain a better understanding of the lateral distribution of soil contamination at properties near Cypress Creek. The first composite sample "A" was collected in a narrow strip of land adjacent to the creek roughly corresponding to the original construction easement; the second composite sample "B" was collected from a generally larger portion of the property outside of the construction easement. The samples were analyzed for 28 or 29 chemicals, depending on which laboratory processed the samples, all of which are chlorinated pesticides, intermediates of pesticide synthesis, or pesticide breakdown products. Full details of the sample collection methodology are provided in reports prepared by MEC (2003, 2005) and Premiere Environmental Services (2005).

Results of the 2003 sampling identified elevated levels of chlorinated cyclodiene chemicals at a number of individual properties. Based on these results, at TDEC's direction, Velsicol submitted an Interim Measures Work Plan (MEC, 2004) that proposed sampling of

additional properties adjacent to those where concentrations of dieldrin exceeded a screening level of 0.7 mg/kg. This sampling was conducted in the summer of 2004. The 2004 sampling added 76 samples from 50 properties. In 2005, Additional soil sampling was performed at 7 properties adjacent to those where concentrations of dieldrin exceeded 3 mg/kg.

With the completion of the 2005 sampling event, approximately 141 Sub-Area III properties have been sampled. This represents approximately 83% of the properties in the study area. Data utilized in this risk evaluation include all of the relevant surface soil data collected during the 2001, 2003, 2004, and 2005 sampling events. More detailed information on these data is provided in reports prepared by MEC (2002a, 2003, 2005) and Premiere Environmental Services (2005).

3.0 RISK ASSESSMENT METHODOLOGY

3.1 Risk Assessment Framework

The EPA has developed and refined a framework for evaluation of potential human health risk from exposure to chemicals in the environment (EPA, 1989, 1991a, 1997, 2001, 2002a). Risk assessment is a regulatory process that uses information about the toxicity of chemical substances to estimate a theoretical level of risk for people who might be exposed to those substances. It is extremely important to understand the context of the risk assessment process before drawing conclusions from this or any other risk assessment report.

The risk assessment process is used to determine if levels of environmental constituents pose an unacceptable risk as defined by regulatory standards and requirements. Risk estimates are calculations based on models – they are useful for ranking purposes, but are not necessarily predictive of any actual individual's risk of developing cancer or other adverse health effects. It should be recognized that no substance or activity is without some level of risk. Many of the activities we engage in on a daily basis would appear extremely "risky" or dangerous if viewed through the lens of the regulatory risk assessment process.

The central tenet of the risk assessment process is summarized by the expression shown below:

$$\textbf{Risk} = \textbf{Toxicity} \times \textbf{Exposure}$$

Very simply, risk is a function of the hazard (the inherent toxicity) of a chemical substance and one's exposure to that substance. If either of these components is absent, there is no risk. Accordingly, risk assessment requires information about both of these components in order to derive estimates of potential risk that can be used by decision makers to establish site-specific remediation goals. Since it is part of a regulatory decision-making process,

both the toxicity and exposure components of the risk equation incorporate highly conservative (i.e., health protective) factors. For this reason, RALs developed using the risk assessment process DO NOT represent levels above which adverse health effects are expected. Rather, the RALs developed using this process represent levels that will be safe for all individuals, including sensitive subgroups within the population. The following sections provide additional discussion of some of the health protective factors that make up the toxicity and exposure components of the risk equation.

3.1.1 Toxicity Component

The toxicity component provides a description of the relationship between a dose of a chemical and the potential likelihood of an adverse health effect. The purpose of the toxicity assessment is to provide a quantitative estimate of the toxicity of chemicals for use in risk characterization. Often, extrapolation of actual toxicity information from high doses to low doses is necessary since environmentally relevant exposure concentrations for humans are typically much lower than experimental exposure concentrations in animals where adverse effects were observed. Extrapolation of results from laboratory animals to humans is usually required.

In the context of the regulatory risk assessment process, potential effects of chemicals are separated into two categories: carcinogenic (cancer) and non-carcinogenic (non-cancer) effects. This division relates to current EPA policy that the mechanisms of action for these endpoints are different. Chemicals that are believed to be carcinogenic may also be capable of producing non-cancer health effects. Potential health risks for these constituents are evaluated for both cancer and other types of effects as described below.

For regulatory purposes, the EPA generally makes the conservative assumption that carcinogenic chemicals do not exhibit a response threshold[1] (EPA, 1986a), while non-carcinogenic effects are universally recognized as threshold phenomena. Recent scientific evidence clearly indicates that this assumption is an oversimplification of carcinogenic responses. A growing number of chemicals have been shown to elicit carcinogenic effects in experimental animals via mechanisms that are: (a) not relevant to human biological processes; or (b) are not expected to occur in humans at significantly lower, environmentally relevant doses (James and Saranko, 2000). The EPA has recently revised the *Guidelines for Carcinogen Risk Assessment* (2005a), in which they recognize these issues and provide alternative approaches for addressing them within the regulatory framework for cancer risk assessment.

[1] A threshold indicates that a minimum amount of drug or chemical agent is required to elicit an effect. For example, certain metals such as iron and selenium are toxic above a threshold dose but safe and, in fact, required dietary components at lower doses. For carcinogens, EPA assumes that no threshold exists and that there is some increased risk at every dose level.

It is well accepted that non-carcinogenic biological effects of chemicals occur only after a threshold dose is exceeded (Klaassen, 2001). This concept implies that a range of exposures up to some defined threshold can be tolerated without appreciable risk of harm. Potential effects may be minimized at concentrations below the threshold by pharmacokinetic processes such as decreased absorption, distribution to non-target organs, metabolism to less toxic chemical forms, and excretion. Once the threshold dose is reached, one or more of these processes may become compromised, potentially resulting in toxic responses.

Carcinogenic Chemicals

The EPA uses a two-step process for evaluating potential carcinogenic effects of chemicals. First, the available scientific data are reviewed to determine if there is an association between the chemical and cancer in humans or experimental animals. Based on this review, the substance is assigned a weight-of-evidence classification reflecting the likelihood that the chemical is a human carcinogen. Second, a cancer slope factor (CSF) is calculated for chemicals considered to be known or probable human carcinogens.

Potential carcinogenic effects resulting from human exposure to chemicals are estimated quantitatively using CSFs, which represent the theoretical increased risk per milligram of constituent intake per kilogram body weight per day $(mg/kg\text{-}day)^{-1}$. CSFs are used to estimate a theoretical upper-bound lifetime probability of an individual developing cancer as a result of exposure to a potential carcinogen.

CSFs are derived by the EPA from the results of chronic animal bioassays, human epidemiological studies, or both. Animal bioassays are usually conducted at dose levels much higher than those likely to be achieved by human exposure to environmental media. Such high levels are used in order to detect possible adverse effects in the relatively small test populations used in the studies. Therefore, a large degree of conservatism exists in the form of high-dose to low-dose extrapolation. Human epidemiological studies often are based on historical occupational exposures at levels much higher than those currently experienced in environmental settings, requiring quantitative extrapolation to account for the dose differences. As described in greater detail below, the cyclodiene chemicals that are the subject of this risk evaluation have never been demonstrated to cause cancer in humans, even those exposed to relatively high levels over long periods of time in occupational settings.

In the case of the cyclodiene chemicals that have been identified as carcinogens by the EPA, high-dose data from animal carcinogenicity studies were extrapolated to low-doses using mathematical models. Most commonly, the linearized multi-stage model is used to estimate the 95% upper confidence limit (UCL) linear slope at low extrapolated doses that are consistent with the data. This model assumes that the dose-response effect of the

carcinogenic agent on tumor formation seen at high doses in animal studies is essentially linear at low doses (i.e., the slope of the dose-response curve can be extrapolated downward to zero in a linear manner). The EPA's *Guidelines for Carcinogen Risk Assessment* (1986a) recommended that the linearized multistage model be employed in the absence of adequate information to the contrary, and that, in general, models that incorporate low-dose linearity are preferred. The 95% UCL slope of the dose-response curve is subjected to various adjustments and an inter-species scaling factor is usually applied to derive a CSF for humans.

In summary, CSFs are derived using extremely conservative (i.e., health protective) assumptions, and the models are believed to provide rough estimates of the upper limits on carcinogenic potency. The actual risks associated with exposure to a potential carcinogen are not likely to exceed the risks estimated, and may be much lower or even zero (EPA, 1986).

This is particularly relevant to the assessment of carcinogenic potential from cyclodiene chemicals. Not only have they never been demonstrated to cause cancer in humans, but the only positive responses in animals have been liver cancers in mice. Because of the extreme susceptibility of certain strains of mice to liver cancer, the relevance of this specific response to the assessment of potential carcinogenicity in humans is considered highly suspect (James and Saranko, 2000). Nonetheless, using the methodology described above, the EPA has concluded that certain cyclodiene pesticides, including dieldrin are "probable human carcinogens" (EPA, 2005b). As discussed below, epidemiologic studies of populations of workers exposed to these chemicals during the manufacturing process do not support this conclusion.

The majority of aldrin and dieldrin was produced at two facilities; one in Denver, Colorado and one in Pernis, Netherlands. Virtually all the workers at these two facilities have been followed in several epidemiological studies of the health effects of cyclodiene exposure (Amoateng-Adjepong et al., 1995; de Jong, 1991; Sielken et al., 1998). None of these studies showed a positive association between dieldrin exposure and cancer. In fact, the most comprehensive of these studies (Sielkin et al., 1998), not only showed no increase in cancer risk, but also a decrease in the probability of death from all causes. Clearly there is a discrepancy between the animal studies used to develop the CSF and the human studies. Possible explanations for this discrepancy are discussed below.

EPA summaries of the scientific literature related to dieldrin carcinogenicity reveal that the positive carcinogenicity studies are restricted to mice (EPA, 2003, 2005b). While mice seem to develop liver tumors, rats and humans do not, suggesting that there might be significant physiologically differences that alter a species susceptibility to cyclodiene induced tumors. Research has shown that dieldrin induces a species-specific P_{450} mediated increase in superoxide radicals in mice. This leads to an increase in transcription factors and

DNA synthesis in mouse liver. Similar events are not seen in liver cells from rats or humans (Klaunig et al., 1995). A second possibility is that cancer risk estimates based on the mouse studies falsely assume that the background carcinogenic transition rate in mice and humans is the same. This means that the baseline conversion rate of a liver cell from a normal to a cancerous state is similar between species. However, the incidence of spontaneous liver tumors in mice strains varies from 2% to nearly 50% while liver tumors in humans are three orders of magnitude less frequent (James and Saranko, 2000). Ignoring the background incidence of liver tumors in mice could introduce considerable error in the estimation of cancer risk. Both the mechanistic and epidemiological findings suggest that the cyclodiene CSFs developed by the EPA are likely overestimates.

Non-Carcinogenic Chemicals

In contrast to carcinogens, regulatory agencies acknowledge that non-carcinogenic effects occur through threshold mechanisms. Adverse effects are not expected at a range of exposures and resulting doses below the threshold dose. The threshold dose for a chemical is usually estimated from the no observed adverse effect level (NOAEL) or the lowest observed adverse effect level (LOAEL), as determined from animal studies or human data. The NOAEL is defined as the highest dose at which no adverse effects are identified, while the LOAEL is defined as the lowest dose at which adverse effects are detectable.

Safety factors are applied to either the NOAEL or the LOAEL to develop a safe dose called the reference dose (RfD). The RfD is expressed in units of daily dose (mg/kg-day) and includes standard safety factors to account for uncertainties such as: the extrapolation from animals to humans, the time period of exposure, and the potential for sensitive individuals within the human population. The RfDs for the cyclodiene chemicals considered in this risk assessment incorporate safety factors ranging from 100 to 1000.

The RfD is defined as an estimate of the daily maximum level of exposure to human populations (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 1989). The RfD provides a benchmark against which human intakes of chemicals in environmental media are compared. When environmental exposure results in a dose lower than the RfD, there is no appreciable risk for non-cancer health effects.

3.1.2 Exposure Component

Potential exposure to chemicals in the environment is dependent on their presence in environmental media (e.g., soil) and characteristics of exposure (e.g., frequency and duration of contact). The RALs developed in this report use exposure factors consistent with an individual's reasonable maximum exposure (RME). The RME is a term defined by the EPA to represent the "maximum exposure that is reasonably expected to occur at a site." The

EPA has indicated that individual factors included in the RME should result in a final exposure estimate that approximates an upper percentile from a range of possible exposures (EPA, 1991b). It is important to point out that RME exposure does not require that every exposure factor represent an upper percentile estimate. If upper percentile values are chosen for each exposure factor, the resulting exposure estimate is no longer "reasonable" and in fact, may exceed the realm of possibility altogether. Therefore, some of the estimates used in the RAL calculations are based on measures of central tendency (e.g., average, median, etc.).

3.2 Overview of the Risk Assessment for Cypress Creek

The investigation of Sub-Area III, including the development of RALs presented in this document has been an ongoing process incorporating the following risk assessment components:

1) Identification of Chemicals of Potential Concern (COPCs) – concentrations of detected chemicals are compared to conservative default screening values. Chemicals with concentrations above these screening values are retained as COPCs for further evaluation in the risk assessment. COPC identification for Sub-Area III occurred during the 2001 investigation (MEC, 2002a).
2) Exposure Assessment – exposure is defined for risk assessment purposes as contact with chemicals in environmental media through the gastrointestinal tract (for ingestion route), skin (for the dermal route), and lungs (for inhalation route). Exposure assessment is the process of measuring or estimating the intensity, frequency, and duration of exposure. It should be noted that some of the exposure parameters used to calculate default screening values are conservatively biased such that potential risks will be overestimated to avoid mistakenly excluding COPCs. It is often appropriate to refine these biased parameters during the risk assessment process.
3) Toxicity Assessment – a toxicity assessment provides a description of the relationship between a dose of a chemical and the potential for an adverse health effect. Its purpose is to provide a quantitative estimate of the toxicity of COPCs for use in risk characterization.
4) Risk Characterization – because risk is a function of both chemical toxicity and receptor exposure, the risk characterization integrates the results of the exposure and toxicity assessments into quantitative and qualitative expressions of risk. The risk characterization also includes an evaluation of the uncertainties associated with the risk assessment.

Risk Management comprises a separate step in which the magnitude of potential risks and the necessity of corrective actions to mitigate them are evaluated. In the context of Cypress Creek, risk management measures will be addressed as part of the Corrective Measures Study.

The Sub-Area III investigation has been conducted in a series of investigative phases which have each included risk evaluations based on some or all elements described above (MEC, 2002a, 2002b, 2003). Based on these previous assessments, Velsicol, TDEC, and the EPA are very familiar with the primary COPCs, their general distribution along the channel, and the screening levels that have been used to determine the need for more detailed evaluation. These factors suggest that a streamlined, pragmatic risk assessment approach in which all of the data are used to develop technically sound, risk-based RALs can be utilized.

3.3 Quantitative Evaluation of Dieldrin as a Indicator Compound

The risk evaluations conducted during the previous phases of the Cypress Creek investigation (MEC, 2002a, 2002b, 2003) consistently indicate that dieldrin is the major contributor to potential toxicity and risk. Representatives of TDEC and EPA have acknowledged this observation in oral and written communications over the course of these investigative phases. For example, a dieldrin concentration of 0.7 mg/kg was established to identify areas that required additional investigations as described in the *Interim Measures Work Plan* (MEC, 2004). For this reason, GeoSyntec focused on evaluating an action level for dieldrin as the basis for corrective actions. The use of dieldrin in this manner will streamline and expedite the remedy selection and remediation process. It will also foster a consistent approach to corrective action throughout Sub-Area III. The following criteria must be met in order to demonstrate that dieldrin is suited for this purpose:

1) dieldrin must be a significant contributor to the total toxic potency or cumulative risk within each exposure unit; and
2) concentrations of dieldrin must be significantly correlated (co-located) with other significant contributors to toxic potency, such that corrective actions to address dieldrin will also mitigate potential risks associated with other COPCs.

3.3.1 Data Selection for Dieldrin Analysis

The dataset used in the analysis presented in this document included the 263 surface soil samples collected from 141 properties located adjacent to the Cypress Creek channel in Sub-Area III. As described previously, 10 of these samples were collected in 2005, 80 samples were collected during 2004, 164 samples were collected in 2003, and nine samples were collected in 2001. The majority of these samples were collected by MEC and analyzed at GTW Analytical Services. Forty of the 263 samples were collected by the EPA and analyzed by EPA's Science and Ecosystem Support Division laboratory.

The EPA and MEC samples had a slightly different list of target analytes. Table 1 provides a comparison of the target analytes between the MEC and EPA samples. Despite this difference, all cyclodiene chemicals common to both the EPA and MEC datasets were included in this evaluation. With minor exceptions, the chemicals not common to both

analyte lists were detected less frequently and at lower concentrations. Therefore, their overall contribution to cumulative risk estimates is considered to be minor. Although the non-cyclodiene pesticides listed on Table 1 were detected infrequently and only at concentrations below conservative default screening levels, most were included in this evaluation so as to be as comprehensive as possible with respect to the evaluation potential risks associated with exposure to pesticides and related chemicals at Sub-Area III properties. Exceptions were toxaphene, which was never detected in any sample; and diethyl-p-nitro phenyl phosphate, which was only detected in one sample and is an organophosphate compound with an entirely different mechanism of toxicity. These two compounds were excluded from the evaluation.

Table 1. Target Analytes

MEC Parameters	EPA Parameters
Common Cyclodiene Chemicals	
Aldrin	Aldrin
Alpha-Chlordane	Alpha-Chlordane
Chlordene	Chlordene
Dieldrin	Dieldrin
Endosulfan I	Endosulfan I
Endosulfan II	Endosulfan II
Endosulfan Sulfate	Endosulfan Sulfate
Endrin	Endrin
Endrin Ketone	Endrin Ketone
Gamma-Chlordane	gamma-Chlordane
Heptachlor	Heptachlor
Heptachlor Epoxide	Heptachlor Epoxide
Hexachlorocyclopentadiene	Hexachlorocyclopentadiene
Isodrin	Isodrin
Methoxychlor	Methoxychlor
Non-Common Cyclodiene Chemicals	
Heptachloronorbornene (Hex VCL)	alpha-Chlordene
Hexachloronorbornadiene (Hex BHC)	beta-Chlordene
Octachlorocyclopentene	cis-Nonachlor
	Oxychlordane
	trans-Nonachlor
Non-Cyclodiene Chemicals	
4,4'-DDD	4,4'-DDD
4,4'-DDE	4,4'-DDE
4,4'-DDT	4,4'-DDT
Alpha-BHC	Alpha-BHC
Beta-BHC	Beta-BHC
Delta-BHC	Delta-BHC
Diethyl-p-nitro phenyl phosphate	Gamma-BHC (Lindane)
Gamma-BHC (Lindane)	Hexachlorobenzene
Toxaphene	Toxaphene

The concentrations of each chemical in samples collected from the same property (A and B samples) were combined to provide an average concentration for each property[2]. When an analyte was not detected in one of the two samples, a proxy value corresponding to ½ of the reported detection limit was used in the calculation. It should be noted that each of these samples represents a composite from five discrete locations. The five discrete samples comprising the "A" composite sample were collected in a narrow strip roughly corresponding to the construction easement where there is reason to suspect the presence of higher concentration of COPCs. The discrete samples comprising the "B" composite sample were collected from a generally larger area of the property outside of the construction easement. This sample bias in favor of areas associated with higher COPC concentrations and the general lack of sampling in areas at greater distances from the channel (e.g., front yards) where COPC concentrations are likely to be much lower, provides a conservative estimate (i.e., biased high) of the average COPC concentrations at individual properties. In teleconferences on November 8, 2004 and December 17, 2004 involving representatives of TDEC, the EPA, and the Memphis-Shelby County Health Department, it was agreed that although biased high, the average provides a conservative representation of the property-wide concentration (i.e., representative concentration), and should be used as the basis for developing RALs and implementing remedial decisions.

3.3.2 Criterion #1 – Contribution of Dieldrin to Total Toxic Potency

As described previously, potential carcinogens and non-carcinogens are treated separately in the EPA's risk assessment framework. This division relates to current EPA policy that the mechanisms of action for these endpoints differ in most cases. However, chemicals that are believed to be carcinogenic may also be capable of producing non-cancer health effects at some dose. Therefore many potential carcinogens for which EPA has developed CSFs, also have RfDs based on their non-carcinogenic effects.

Many of the chemicals detected in the Sub-Area III samples are by-products of pesticide manufacturing or products of the molecular transformation of the pesticides in the environment. Because toxicity values have not been developed by the EPA for most of these chemicals, surrogate toxicity values were selected in order to evaluate them in this analysis. When faced with assessing the potential toxicity of a chemical for which little or no experimental data exist, toxicologists commonly rely on toxicological information from chemicals with similar structures. This is because a chemical's structure can provide important information about its mechanism of action and potential for toxicity.

[2] There were 34 properties where, due to size constraints or other site conditions, only one sample was collected per property. For these properties, the COPC concentrations in the single sample were used as the representative concentrations for that property. Similarly, there were several properties where more than two samples were collected. At these properties, the representative concentration is the average of all samples.

For several chemicals where structural similarity was readily apparent, surrogates were selected on that basis. For example, the RfD for endosulfan (a mixture) was used to represent endosulfan I, endosulfan II and endosulfan sulfate. Likewise, the RfD for endrin was used to represent endrin aldehyde and endrin ketone. However, the structures of the cyclodiene chemicals are relatively closely related in general, so for any given chemical it is difficult to identify the "most" structurally similar compound to use as a surrogate. Therefore, for the other chemicals considered in this analysis without EPA toxicity values, endrin was used as a surrogate because it has one of the more conservative and therefore health-protective RfDs. It should be noted that no CSFs were used as surrogates because: i) the specific cancer responses in mice on which these CSFs are based, have questionable applicability to humans (EPA, 2003); and ii) the cancer responses in long-term rodent bioassays are highly variable even among this group of structurally similar chemicals. The use of toxicity surrogates in this evaluation is another protective measure intended to ensure that cumulative risk is not underestimated. The compounds for which surrogate toxicity values were used in this evaluation are shown in Table 2.

Table 2. Toxicity Surrogates

Chemical	Surrogate
4,4'-DDD	4,4'-DDT
4,4'-DDE	4,4'-DDT
Alpha-Chlordane	Chlordane
Alpha-Chlordene	Endrin
Beta-Chlordene	Endrin
Chlordene	Endrin
cis-Nonachlor	Endrin
Endosulfan I	Endosulfan
Endosulfan II	Endosulfan
Endosulfan sulfate	Endosulfan
Endrin aldehyde	Endrin
Endrin ketone	Endrin
Gamma-Chlordane	Chlordane
Hex VCL	Endrin
Hexachloronorbornadiene	Endrin
Isodrin	Endrin
Octachlorocyclopentene	Endrin
Oxychlordane	Chlordane
Trans-Nonachlor	Endrin

The CSFs and RfDs established by the EPA can be thought of simply as estimates of relative toxic potency of one chemical compared to another. For example, the CSF for dieldrin is 16 $(mg/kg\text{-}day)^{-1}$, whereas the CSF for heptachlor is 4.6 $(mg/kg\text{-}day)^{-1}$. Although there is substantial protectiveness in the manner in which EPA derives all of these theoretical potency estimates, the ratio of these two values can be taken to indicate that dieldrin is approximately 3.5 times (16/4.6 = 3.5) more potent than heptachlor. Even though the units of the EPA's RfDs are inverse to those of the CSFs, they can be used in a similar manner to derive relative potency estimates for non-cancer health effects.

EPA toxicity values were used in this manner to estimate the fraction of the total cancer and non-cancer toxic potency attributable to each COPC at each property. For every sampled property, the A-B average concentration of each COPC was multiplied (cancer slope factors) or divided (reference doses) by the EPA toxicity values for that COPC. The results were summed to provide an estimate of "total toxic potency" for each property. The percent of the total toxic potency contributed by individual COPCs at each property was then calculated. Finally, the summary statistics on the percent contribution of each COPC to the total toxic potency of properties within Sub-Area III were calculated. These summary statistics are provided for carcinogens and non-carcinogens in Tables 3 and 4, respectively. It is important to note that when the same conservative default exposure assumptions are applied at each property, as discussed in Section 5 , the contribution of each chemical to the estimate of "total toxic potency" at a property is equivalent to its contribution to cumulative risk estimates (both cancer and non-cancer) at that property.

Table 3. Contribution to Toxic Potency (Cancer) by Chemical

COPC	Cancer		
	Mean % Contribution to Potency	Median % Contribution to Potency	Range (%) of Contributions
Dieldrin	82.8	89.4	23.6 - 98.2
Aldrin	5.78	3.02	0.24 - 28.38
Heptachlor epoxide	5.27	3.70	0.16 - 51.5
Heptachlor	2.10	0.89	0.08 - 13.6
Alpha-BHC	1.72	0.57	0.03 - 11.92
Beta-BHC	0.48	0.18	0.01 - 3.17
Gamma-Chlordane	0.42	0.39	0.098 - 1.62
Gamma-BHC	0.36	0.12	0.01 - 2.51
Hexachlorobenzene	0.33	0.11	0.03 - 26.1
4,4'-DDT	0.25	0.09	0.0025 - 4.69
Alpha-Chlordane	0.23	0.13	0.0065 - 5.33
4,4'-DDE	0.14	0.05	0.0014 - 4.26
4,4'-DDD	0.08	0.02	0.0018 - 0.71

Note: Only chemicals with EPA cancer slope factors shown. The cancer slope factor for technical chlordane applied to both alpha and gamma chlordane.

Table 4. Contribution to Toxic Potency (Non-Cancer) by Chemical

COPC	Non-Cancer		
	Mean % Contribution to Potency	Median % Contribution to Potency	Range (%) of Contributions
Endrin ketone	26.6	25.9	1.79 - 62.1
Dieldrin	24.5	21.8	4.72 - 72.3
Endrin	14.2	14.7	0.15 - 34.7
Heptachlor epoxide	10.6	7.45	0.16 - 64.0
Hex VCL	9.19	9.05	0.05 - 48.4
Isodrin	4.12	2.78	0.20 - 27.4
Aldrin	3.12	1.57	0.10 - 19.2
Hex BCH	2.08	0.81	0.03 - 21.0
Chlordene	1.32	0.85	0.08 - 8.64
Trans-Nonachlor	1.01	0.25	0.08 - 11.5
cis-Nonachlor	0.75	0.63	0.08 - 2.96
Hexachlorocyclopentadiene	0.65	0.17	0.004 - 5.14
Gamma-Chlordane	0.54	0.51	0.12 - 2.21
4,4'-DDT	0.40	0.12	0.002 - 7.71
Hexachlorobenzene	0.36	0.03	0.004 - 4.41
Alpha-Chlordane	0.32	0.16	0.008 - 7.24
Octachlorocyclopentene	0.27	0.06	0.002 - 4.10
Beta-BHC	0.26	0.08	0.003 - 1.92
Delta-BHC	0.26	0.08	0.002 - 1.92
Gamma-BHC	0.24	0.07	0.002 - 1.92
Alpha-BHC	0.24	0.07	0.002 - 1.92
4,4'-DDE	0.20	0.07	0.001 - 4.32
Heptachlor	0.19	0.10	0.01 - 0.96
4,4'-DDD	0.17	0.05	0.0021 - 1.41
Alpha-Chlordene	0.16	0.10	0.01 - 1.14
Oxychlordane	0.15	0.10	0.01 - 0.68
Beta-Chlordene	0.14	0.06	0.01 - 1.14
Methoxychlor	0.12	0.03	0.0008 - 0.96
Endosulfan II	0.02	0.01	0.0001 - 0.14
Endosulfan sulfate	0.02	0.005	0.0002 - 0.14
Endosulfan I	0.02	0.004	0.0001 - 0.85

The results of this analysis support the conclusion that dieldrin is by far the most significant contributor to cancer risk throughout Sub-Area III. Dieldrin is also a very significant contributor to non-cancer toxic risk at Sub-Area III properties, although there is

more variability in the non-cancer risk drivers at individual properties. Specifically, dieldrin on average represents approximately 83 of the total theoretical cancer potency at Sub-Area III properties sampled. In contrast, the next highest contributors are aldrin and heptachlor epoxide with contributions of only 5.8 and 5.3, respectively. Clearly, dieldrin is the predominant contributor to theoretical cancer risk at these Sub-Area III properties. Several residential properties had mixtures of pesticides where dieldrin contributed less than 65% of the total cancer risks. However, each of these properties had very low levels of all the COPCs.

The contribution of dieldrin to total non-cancer risk in the Sub-Area III samples, while significant, is less than the contribution of dieldrin to theoretical cancer risk. Dieldrin represents approximately 24% of the total non-cancer toxic potency across Sub-Area III and is similar to the estimated contribution of endrin ketone at 27%. The next highest contributors are endrin and heptachlor epoxide with contributions of 14% and 10%, respectively. The fact that these other chemicals significantly contribute to the total non-cancer toxic potency in this dataset, indicate that the development of secondary criteria is warranted to ensure that the final dieldrin-based RAL also provides protection from other COPCs. The development and application of secondary criteria based on non-cancer effects is discussed in Section 5.5.

3.3.3 Criterion #2 – Spatial Correlation of Dieldrin to Other Risk Drivers

As described previously, cyclodiene chemicals were present in the sediments of the Cypress Creek drainage channel. Some of these sediments may have initially been carried onto adjacent properties during high rainfall periods or flood events. Some sediments are believed to have been excavated during the concrete lining and straightening of the channel during the 1960's and placed as backfill along the channel's retention wall. Finally, some sediments may have been used to fill low-lying areas of properties adjacent to the channel. Because these chemicals and/or their degradation products were initially co-located within the sediment, it is anticipated that they will display a similar degree of co-location in samples collected in areas of fill placement even though many years have passed. Many of the sample locations with a high concentration of one chemical also have high concentrations of other chemicals. These observations were statistically verified.

A statistical analysis was conducted to evaluate the spatial correlation between dieldrin and some of the other COPCs. Included in this analysis were chemicals that contribute significantly to the toxic potency at the site (>10% of the total hazard or risk potency in any one yard). As identified in Tables 3 and 4, these chemicals include aldrin, alpha-BCH, endrin, endrin ketone, heptachlor, heptachlor epoxide, Hex VCL, Hex BCH, and isodrin.

Correlation is the standard measure of association between two variables. The Spearman's Rank Correlation technique is the recommended method for calculating a

correlation coefficient for non-normal data, such as contaminant concentration data (Rosner and Belmont, 1990). The value of a correlation coefficient can vary from -1 to +1. A -1 indicates a perfect negative correlation, while a +1 indicates a perfect positive correlation. A correlation of zero means there is no relationship between the two variables. A positive correlation means that as the concentration of dieldrin increases, the concentration of the other compound increases as well. The standard error of a correlation coefficient is used to determine the confidence intervals around a true correlation of zero. The significance (probability) of the correlation coefficient is determined from the t-statistic where the probability of the t-statistic indicates if the correlation is significantly different than zero. The results of the correlation analysis conducted for dieldrin and other chemicals that contribute significantly to total toxic potency are provided in Table 5 below.

Table 5. Chemical Concentration Correlation with Dieldrin

Chemical	Correlation with Dieldrin[1]
Endrin Ketone	0.95
Endrin	0.95
Isodrin	0.89
Hex VCL	0.87
Heptachlor Epoxide	0.84
Heptachlor	0.80
Hex BCH	0.78
Aldrin	0.75
Alpha BHC	0.56

[1]**Spearman's Rank Correlation coefficient**

3.3.4 Conclusions for Dieldrin as an Indicator Compound

The results of this analysis demonstrate that concentrations of dieldrin are highly correlated with other chemicals previously shown to be significant contributors to total toxic potency in the samples collected from Sub-Area III properties. When these results are combined with those from the analysis addressing Criterion #1, they indicate that use of dieldrin as an indicator for corrective action decision making is unlikely to result in an underestimation of the risk posed by other COPCs present. Further, this analysis suggests that corrective actions to address dieldrin as an indicator of potential risk should also mitigate the potential risk from other COPCs. As described in Section 5, cumulative risk estimates for each property have been calculated to further evaluate the use of dieldrin concentrations at Sub-Area III properties as the primary basis for corrective action decisions.

3.4 Spatial Analysis of Cypress Creek Sub Area III Analytical Results

An analysis was conducted to determine the relationship between sample locations relative to the reported construction easement (within or outside) and the concentrations of pesticides. The results of this analysis are used to evaluate the protectiveness in using the average of multiple samples collected from each property as a representative concentration for that property.

Collocated A and B sample data were available from 100 individual properties. Dry weight soil concentration data was used for all analyses. For the purposes of this analysis, when chemicals were not detected in a particular sample, the full detection limit was used as a proxy concentration. Samples were designated as originating from the area corresponding to the reported construction easement (A) or from an adjacent area outside the construction easement boundary (B). All samples were collected as five-point composites. The total pesticide concentration in each sample was calculated by summing the dry weight analytical results for DDD, DDE, DDT, aldrin, alpha-BHC, alpha-chlordane, alpha-chlordene, beta-BHC, beta-chlordene, chlordene, cis-nonachlor, delta-BHC, dieldrin, endosulfan I, endosulfan II, endosulfan sulfate, endrin, endrin ketone, gamma BHC, gamma-chlordane, heptachlor, heptachlor epoxide, Hex VCL, hexachlorobenzene, hexachlorocyclopentadiene, hex BCH, isodrin, oxychlorocyclopentene, oxychlordane, and trans-nonachlor.

3.4.1 Comparison of Central Tendencies

A useful measure to compare the A vs. B samples is to evaluate central tendency estimates (e.g., mean, median, etc.). The A and B designations were based on historical records of the construction easement associated with the concrete lining of Cypress Creek. In every instance, the A sample location was located closer to the creek bank than the corresponding B sample within the same property. Soils removed from the creek may have been used as backfill material directly along the creek banks resulting in higher levels of pesticides in these locations. The data collected to date support this conceptual model.

Table 6 shows the mean and median dieldrin concentrations (mg/kg) based on all 2003 and 2004 sampling data from either the A or B samples. Both the mean (average) and median (50^{th} percentile) concentrations are higher in the A samples than compared to the B samples. Similar results were obtained when the total of the selected cyclodiene pesticides were compared from the A or B sample locations.

Table 6. Comparison of Chemical Concentration by Location

	A (within easement)	B (outside easement)
Dieldrin		
mean	4.8	0.89
median	0.62	0.26
Total Pesticides		
mean	210	23.4
median	13.7	4.0

Note: All concentrations are expressed in units of mg/kg.

3.4.2 Comparison of the Magnitude of the Differences

In the majority of the cases, samples collected from within the construction easement (A samples) were higher than those outside the easement (B samples). However, there are some exceptions. To further evaluate the difference in the A vs. B samples it was necessary to review the data from those properties where both A and B samples were taken. In 100 cases, data were available to directly compare the concentration of the A sample to the B sample on an individual property. The magnitude of the difference between the A and B samples was evaluated by dividing the concentration of one sample by the concentration of the other. For ease of discussion, this ratio is always discussed as the magnitude or fold increase of one sample to the other. In cases where sample A is larger than sample B, the ratio is computed as A/B and the result is discussed how many times A is larger than B. When sample B is larger than sample A, the ratio is calculated as B/A and the results are discussed as the fold increase of B over A.

Results of the magnitude analysis for dieldrin are presented in Table 7. Dieldrin concentrations in the construction easement (A) were higher than those in the adjacent area (B) at 69 property locations. The magnitude of the higher dieldrin concentration at A was, on average, 104-fold higher than those at location B and ranged from equal to 1180-fold higher. Location B has higher levels of dieldrin than location A at 31 properties. For those samples where the B location was higher than the A location, the magnitude of the difference was on average, only 2-fold higher and driven mostly by a few properties where the concentration of dieldrin was less than the 0.7 mg/kg screening level previously approved by TDEC in both the A and B samples.

Table 7. Comparison of Concentrations by Location within a Single Property

	Cases where A>B	Cases where B>A
Occurrences	69	31
Mean	104 fold	2 fold
Median	10 fold	2 fold
Range	1-1180 fold	1-120 fold

* There were only five properties where the dieldrin concentration in sample B exceeded sample A by more than 5-fold. In two of those cases, the higher B samples were 9.6 and 6.7 mg/kg. In the other three cases, the higher B samples were 0.18, 0.11, and 0.33 mg/kg.

This analysis demonstrates that in cases where B samples are greater than A samples the magnitude of the difference is much smaller than the magnitude of the difference when the A samples are larger than the B samples.

3.4.3 Use of Sample Averages as Representative Property Concentrations

Velsicol has developed a RAL based on dieldrin as an indicator compound for the evaluation of the contamination of properties along Cypress Creek. Estimating risks and determining how to implement RALs can be accomplished by selecting a single numerical concentration of dieldrin to represent each property. Most of the samples from properties along Cypress Creek were taken from residential backyards that border the creek. These properties typically consist of an area near the creek that approximates the original construction easement (A samples) and a generally larger area within the property but outside the construction easement (B samples). A few properties were sampled in the front or side yards further away from Cypress Creek and the construction easement. It is important to note that all of the samples collected from Sub-Area III properties in 2003, 2004, and 2005 are five-point composites and that the areas represented by the B samples are generally larger than the areas represented by the A samples. This significantly decreases the likelihood that areas of significantly higher concentration have been missed in this evaluation.

Risk assessment methodology considers the entire residential property as the exposure unit for calculation of risks from exposure to soil contaminants. EPA guidance specifies that "an individual receptor is assumed to be equally exposed to media within all portions of the exposure unit over the time frame of the risk assessment" (EPA, 2002b). For this reason the arithmetic average soil concentration is the best input for the calculation of risks associated with exposure to soil contamination within an exposure unit. Because the areas where the samples were collected vary in size for each property, it may be possible to develop area weighted averages for each property. However the distribution of COPCs across the entire property and receptor exposure patterns are not well defined. In the face of this uncertainty,

the additional refinement in exposure concentrations through weighted averaging is not warranted[3]. As an alternative, the simple average of the A and B samples could be used as the property concentration. Because the areas represented by the A and B samples vary in size; the simple average will tend to over-represent the smaller portion of the yard in the calculation of the average.

The area of the yard that represents the construction easement is typically a small portion of the entire yard. Results of this analysis suggest that samples from within the construction easement are more contaminated than those outside the easement. A representative property concentration using a simple A and B average for those properties will tend to be biased high (i.e., conservative) as compared to the true average property concentration.

For properties where the COPC concentrations in the B samples were higher, COPC concentrations were either generally low, or the B samples were only marginally greater than the A samples. In addition, use of the average concentrations in this manner makes the conservative assumption that the remainder of the property (i.e., not represented by the A or B samples) contains levels at least as high as those in the B samples. Because concentrations decreased with distance away from the creek, the concentrations of COPCs in the remainder of the property are most likely lower than those found in the B sample. Again, this supports the use of the simple average of the A and B composite samples as a conservative estimate of the COPC concentrations at each property.

4.0 CONCEPTUAL EXPOSURE MODEL FOR HUMAN HEALTH RISK

The Conceptual Exposure Model (CEM) is a tool to describe potential exposures to environmental media. As described previously, Sub-Area III is approximately 2.4 miles long and encompasses the stretch of the concrete-lined Cypress Creek that passes through mixed residential and commercial areas between Jackson Avenue and Evergreen Street. The creek is contained within a straight-banked concrete channel approximately 8 to 15 feet high with a fence on top throughout Sub-Area III. The land use of the approximately 170 properties in the Study Area is predominantly residential. An approximate breakdown of the specific land uses follows:

- 130 single family residences;
- 22 undeveloped open space parcels;
- 13 commercial/industrial properties; and
- 5 apartment complexes.

[3] Per teleconferences between representatives of TDEC, EPA, the Memphis-Shelby County Health Department, and Velsicol on November 8, 2004 and December 17, 2004.

It should be noted that the Cypress Middle School/University Park area are not included in the current Sub-Area III evaluation as that area was the subject of a previous investigation and risk assessment (MEC, 2002b), and corrective action completed by TDEC in 2003. The CEM for Sub-Area III is presented in Figure 1. Elements of this CEM are discussed in more detail in the following sections.

4.1 Human Receptors

For the purposes of risk assessment, the term "receptor" is used to describe a human that may be exposed to chemical hazards through specific types of actions or exposure scenarios. Because of the mixed land use in Sub-Area III, several general categories of receptors are currently present. These include:

- child and adult residents;
- commercial workers;
- recreators; and
- construction workers;

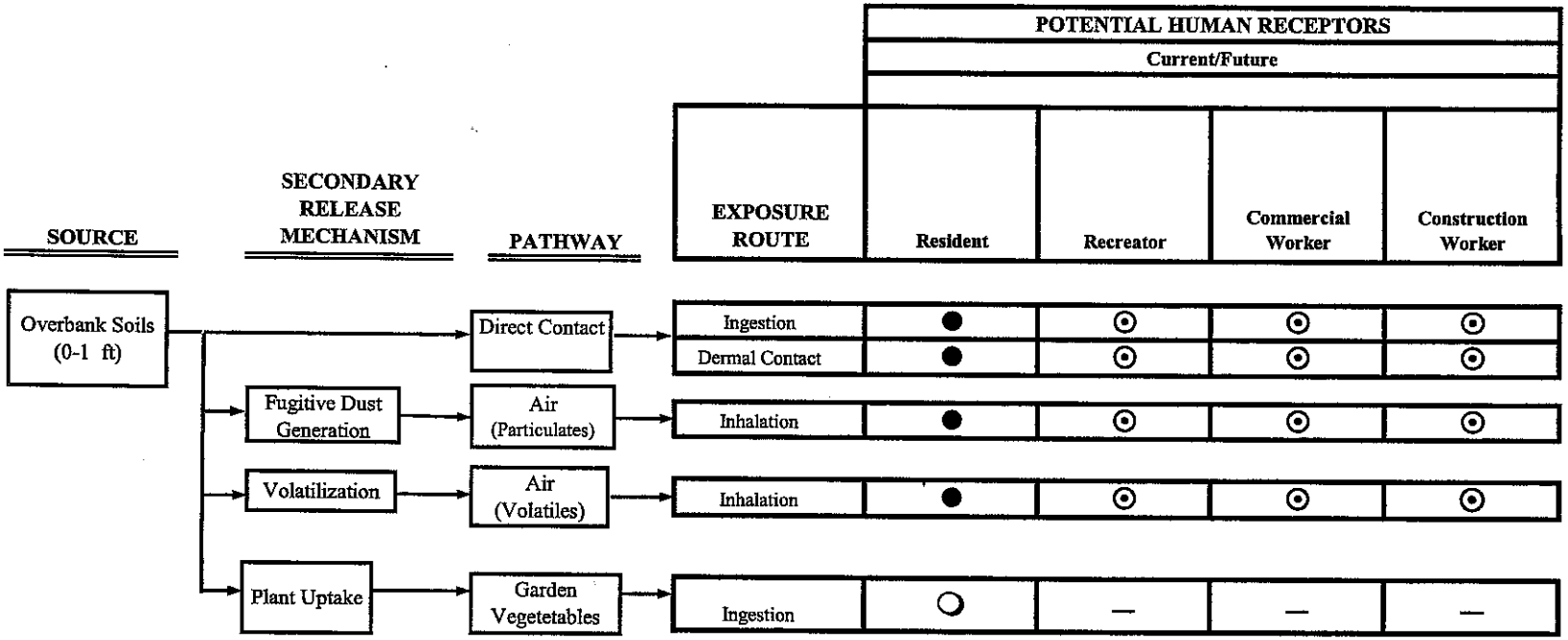
This report focuses on the development of risk-based RALs for adults and children at residential properties within Cypress Creek Sub Area III. It should be noted that this scenario uses a more conservative set of exposure assumptions than typically used for receptors based on non-residential land use. Therefore, remedial action levels based on residential use would be expected to provide a conservative representation of risk to other receptors potentially exposed at these properties. For comparison purposes, this report also presents risk estimates based on residential exposure assumptions for a number of non-residential properties. These risk estimates would only be valid if land use at these properties were to change to residential in the future.

4.2 Exposure Pathways

This section describes the exposure pathways by which receptors could come in contact with COPCs. These pathways are considered to be complete when links exist between impacted site media and a potential receptor. Because of the current and foreseeable future mixed land use in Sub-Area III, the receptors identified above may be exposed to a greater or lesser degree to COPCs in the following media while pursuing their daily activities:

- Surface Soil – child and adult residents, recreators, commercial workers, and grounds maintenance workers are potentially exposed to COPCs present in the surface soil via incidental soil ingestion, inhalation of volatiles and fugitive dusts in ambient air, and dermal contact.
- Subsurface Soil – there is a potential that future construction workers could be exposed to COPCs present in the subsurface soil via incidental soil ingestion,

Figure 1
Conceptual Exposure Model for Human Health Risk
Cypress Creek Sub-Area III



Legend

- → = Pathways -- past, current, and hypothetical future
- ● = Pathways evaluated quantitatively
- ⊙ = Receptor/pathway not quantitatively addressed
- ○ = Pathways evaluated qualitatively
- — = Incomplete pathways

dermal contact, and inhalation of volatiles and fugitive dusts in ambient air. Current and future residents may also be exposed periodically to subsurface soil during landscaping or gardening activities.

- Household Dust – It is reasonable to assume that a significant proportion of daily incidental soil ingestion occurs through ingestion of household dusts. Although exposure to indoor household dust is not explicitly evaluated in the RAL equations provided in Section 5, because soil ingestion rates used in this risk assessment reflect a total daily soil ingestion rate regardless of source (e.g., house dust, soil from the yard, soil from the school or jobsite), the use of soil sampling data from the individual properties (including samples immediately adjacent to the drainage channel) provides a worst case scenario for the evaluation of potential health risks to Sub-Area III residents. Further because soil-derived house dust consists of primarily near surface soil, it is likely that concentrations of COPCs in house dust are lower than the concentrations in samples collected from the property on which that house is located due to natural degradation processes that occur much more rapidly in near surface soil exposed to oxygen and sunlight.
- Homegrown Produce – based on survey information gathered during the 2003 sampling of residential properties in Sub-Area III, which coincided with the growing season, approximately 14 of 107 (13%) of the single family residences sampled had active flower or vegetable gardens. Although the survey did not differentiate between these two types of gardens, observations by MEC personnel indicate that vegetable gardens comprise a smaller fraction. This percentage is significantly lower than the 29% reported for urban residences in the EPA's *Exposure Factors Handbook* (EPA, 1997). Only a couple of these gardens were larger than 100-200 square feet in area. It is also reasonable to assume that some proportion of future residents will also garden and consume homegrown produce. There are reports in the scientific literature that indicate that chemicals in the cyclodiene family may be taken up from the soil into plant tissue.

Exposure to soil via incidental ingestion, dermal, and inhalation pathways are explicitly considered in the RAL model described in Section 5. Exposure to COPCs via ingestion of homegrown produce, however, is not considered in that model. The following sections present a qualitative evaluation of the plant uptake and exposure pathway.

4.3 Uptake of Organic Chemicals by Plants

A potential pathway of exposure for Cypress Creek residents is from the consumption of vegetables grown in soils containing COPCs. The quantitative evaluation of this pathway is complicated by the considerable uncertainty introduced by attempting to model contaminants from soil to develop a reliable exposure estimate. Both theoretical and empirical evidence is presented in the sections below that explore the basis for developing exposure estimates for this pathway.

4.3.1 Background on Plant Uptake

Plant uptake of organic chemicals from soil can occur in different ways. Some uptake into the root system is common. Depending on the hydrophobicity of the chemical and the plant transpiration rate, this may be followed by transport in the transpiration stream (Ryan et al., 1988). The octanol-water partition coefficient (K_{ow}) is an indication of hydrophobicity, with increasing K_{ow} values indicating greater hydrophobicity. Generally, transpiration stream transport is an inefficient chemical transport process for hydrophobic chemicals in plants (i.e., log K_{ow} values >4.5). As a result hydrophobic chemicals will tend to partition into the root tissue, a process particularly significant for plants having relatively high lipid contents. The chemicals in the cyclodiene family all fall into the "hydrophobic" category (i.e., log K_{ow} values >4.5), with the exception of endosulfan and endosufan sulfate (log K_{ow} values of 3.50 and 3.64 respectively).

Plants can metabolize a variety of organic chemicals, resulting in reduction in concentration of the parent compound while also potentially generating metabolic intermediates. Wet and/or dry deposition on above-ground plant parts may lead to chemical absorption and transfer. However, this process is often not a significant pathway for subsurface chemical sources. A comprehensive study on the metabolism of cyclodiene chemicals by plants has not been reported in the primary literature.

Soil processes such as sorption and degradation are important factors affecting the rate at which plants uptake organic chemicals. For hydrophobic chemicals, the most important soil property is the soil organic carbon content. The tendency for hydrophobic chemical uptake decreases as soil organic carbon content increases, due to sorption of the organic chemical by the soil organic matter. This makes it less readily available for uptake by the plant from the soil pore-water. This is likely to be a significant factor for vegetable crops, since typical gardening practices include the addition of soil amendments high in organic carbon.

4.3.2 Plant Uptake Models for Organic Chemicals

Plant uptake models were investigated to evaluate their utility for accurately estimating uptake of cyclodiene chemicals by vegetable crops. Plant uptake models can be grouped into two general categories, *mechanistic* and *empirical* models. Several mechanistic evaluations have been proposed (Trapp et al., 1994; Paterson and Mackay, 1994; Boersma et al., 1988, 1991; Lindstrom et al., 1991; Hung and Mackay, 1997; Chiou et al., 2001). However, these mechanistic approaches have not been widely adopted for risk assessment purposes, and their utility for the estimation of the plant uptake of cyclodiene chemicals for the Sub-Area III investigation is limited. Models based on empirically derived chemical/physical parameters have also been proposed (Briggs et al., 1982, 1983; Topp et al., 1986; Travis and Arms, 1988; Ryan et al., 1988). However, the validity of the relationship for hydrophobic chemicals, such as the Sub-Area III COPCs, is uncertain and

requires extrapolation of the empirical relationship. For higher K_{ow} compounds, there appears to be no clear trend in the validity of the model - some researchers report over-estimation while others report under-estimation of plant concentrations. Given that the pesticides under consideration have log K_{ow} values greater than the 2 to 3 range, the model's applicability to plant uptake is uncertain.

4.3.3 Reports of Plant Uptake of Cyclodienes in the Scientific Literature

A limited amount of research has been conducted on the uptake of cyclodiene pesticides by plants. Most of this research dates back to the 1960's and 1970's when cyclodiene pesticides were still being used for insect control on food crops. Most of the reports focus on the uptake of fresh pesticide in a relatively short period following its application (Cole et al., 1976; Kloskowski et al., 1981; Beall and Nash, 1969, 1971; Singh et al., 1990, 1991; Gupta et al., 1979; Beall et al., 1972; Nash and Harris, 1973). While most of these studies suggest some level of uptake of freshly applied cyclodiene pesticides by crops, the COPCs present in the Sub-Area III soils have been in place for more than 30 years. These studies are considered to be of limited utility in evaluating the potential for uptake of such weathered pesticides in soil by garden produce.

A more recent study of chlordane uptake in plants by Mattina and co-workers (2000) arguably has the most relevance to the situation in Sub-Area III. While chlordane is not a major COPC in Sub-Area III, chlordane is chemically similar to other cyclodiene compounds suggesting that some insight can be drawn from these studies. The researchers measured chlordane uptake in 11 vegetable crops grown in soil that was treated with chlordane more than 30 years earlier. The results indicated that even weathered chlordane is translocated into plant tissues and the extent of uptake is highly variable with plant type. Moderate uptake of chlordane into the root systems was observed for many plant types. However, uptake into edible portions of plants was highly variable suggesting complex mechanisms for the translocation of chlordane into edible plant tissues. Based on these observations, these researchers classified the crops into the following groups: i) *uptakers* - beets, carrots, lettuce, potatoes, spinach, zucchini; ii) *non-uptakers* – corn, peppers, tomatoes; and iii) *intermediates* – beans, eggplant. This research report suggests that moderate levels of chlordane can be detected in edible plant tissue even after years of weathering. The extension of these finding to other cyclodiene chemicals is uncertain, however, it suggests that plant uptake may contribute to the exposure pathway.

4.3.4 Field Data for Plant Uptake of Cyclodienes from Sub-Area III Soils

Analytical data for six types of vegetables were obtained from a residential garden located at 1930 Edward Avenue. Memphis Environmental Center (MEC) collected and delivered vegetable samples to GTW Analytical Services in Memphis, Tennessee. Samples

were analyzed for a suite of chlorinated pesticides using FDA Method 303 E1+DG1. Table 8 provides a summary of the analytical data from the collected vegetables.

Table 8. Vegetable Analytical Results

Vegetable	Dieldrin (mg/kg)	Endrin (mg/kg)	Endrin Ketone (mg/kg)	Hex VCL (mg/kg)
Rape Greens	ND	ND	ND	ND
Kale Greens	0.0034	ND	ND	ND
Sweet Potato	0.0047	0.012	0.017	0.037
Mustard Greens	ND	ND	ND	ND
Turnip Greens	0.0085	0.0038	ND	ND
Tomatoes	ND	ND	ND	ND

Notes:
(1) Aldrin, DDD, DDE, DDT, Alpha-BHC, Beta-BHC, Delta-BHC, Gamma-BHC, Alpha-Chlordane, Gamma Chlordane, Chlordene, Diethyl-p-nitro-phenyl phosphate, Heptachlor, Heptachlor epoxide, Technical Chlordane, Methoxychlor, Endosulfan I, Endosulfan II, Endosulfan sulfate, Hexachlorocyclopentadiene, Hexachloronorbornadiene, Octachlorocyclopentene, Isodrin, and Toxaphene were analyzed for, but not detected in any vegetable samples.
(2) Detection limits for Dieldrin, Endrin, and Endrin ketone were 0.003 mg/kg.

Three of the vegetables sampled (rape greens, mustard greens, and tomatoes) had no detectable levels of any of the pesticides on the analyte list. The other three vegetables sampled (kale greens, sweet potatoes, and turnip greens) contained only trace amounts of one to three analytes at levels near the method reporting limits. Of these, sweet potatoes generally contained higher levels of analytes. As a tuber, sweet potatoes are in much closer contact with the soil than any of the other vegetables tested. It is possible that some of the pesticide residues might be attributable to contaminated soil that was not completely removed during sample preparation.

Two soil samples, one in 2003 and one in 2004, were collected from the residential property containing the garden plot (MEC, 2003, 2005). The 2003 sample was a five point composite collected from within the construction easement that included, but was not limited to, the garden plot. The 2004 sample consisted of a five point composite soil sample collected from completely within the garden plot in which these vegetables were grown. These soil samples were analyzed for the same suite of pesticides as the vegetable samples. The results of these samples indicate that all of the chemicals detected in the vegetables were also detected in the soil from the garden plot, but not all contaminants found in the soil were detected in the vegetables.

4.3.5 Recommendations for the Plant Uptake Pathway

For the Sub-Area III COPCs, the modeled plant uptake provides bioconcentration estimates that vary considerably. For example, for hydrophobic chemicals with high Log K_{ow} values (i.e., > 4.5), plant uptake estimates calculated using the empirical models can differ by orders of magnitude. This variability introduces a high degree of uncertainty to the assessment of potential exposures related to ingesting home grown produce grown in soils containing cyclodiene chemicals. This uncertainty is compounded by research indicating that translocation of at least some cyclodiene pesticides into edible plant tissues is also highly variable among different plant species (Mattina et al., 2000).

Field data obtained from the garden at 1930 Edward Avenue, suggests that, at least for this garden plot, plants can take up cyclodiene pesticides from soils. Trace levels of some of the cyclodiene pesticides found in the garden soils were also found in the vegetables. However, the levels of pesticides found in these vegetables were well below those that would cause concern, for human consumption. Extrapolating the results from this single garden plot to other properties is not possible because of the variability associated with many parameters contributing to vegetable uptake (e.g., soil organic carbon content, watering frequency, fertilizer application, tilling techniques, etc.). However, available evidence indicates that i) few residents currently maintain vegetable gardens; ii) levels of COPCs in tilled near-surface soil are likely to be much lower when compared to levels in undisturbed soils; and iii) empirical data from a Cypress Creek garden plot indicate that COPC levels in vegetables are below well below levels that could present a health concern. Based on these factors, home grown vegetables are considered unlikely to present a significant source of COPC exposure for Cypress Creek residents.

5.0 DEVELOPMENT OF REMEDIAL ACTION LEVELS

All of the RALs presented in this section are based on residential receptors. Residential exposure assumptions typically account for the most frequent and intense contact with impacted soil, such that a RAL that is protective of a residential receptor will also be protective of most other types of receptors who might also be exposed. It should be noted that there may be some properties where it is more appropriate to develop and implement a RAL based on non-residential (i.e., commercial/industrial, recreational) exposure conditions. The development of exposure parameters and RALs associated with non-residential land use is beyond the scope of this report. Therefore, the same residential exposure assumptions have been used to calculate baseline risks for all Sub-Area III properties sampled to date. However, the non-residential properties have been segregated from residential properties, and the risk estimates presented in separate tables. As discussed previously, these risk estimates would only be applicable to non-residential properties if they were converted to residential use sometime in the future.

5.1 Remedial Action Level Equations

The equations for calculating RALs for dieldrin based on direct contact with soil are shown below. Since the EPA has developed both cancer and non-cancer toxicity values for dieldrin, one equation calculates a RAL based on cancer risk and another calculates a RAL based on non-cancer health effects. When a chemical has both cancer and non-cancer health effects, the final RAL should consider both endpoints. Both equations estimate chemical intake from incidental ingestion of soil, dermal contact with the soil, and inhalation of chemicals present in soil that have volatilized or have adhered to soil-derived particulates (i.e., dust).

RAL for cancer effects:

$$RAL = \frac{TR \times BW \times AT}{EF \times ED \times \left[\left(CSF_o \times IR_o \times 10^{-6}\,kg/mg\right) + \left(CSF_d \times SA \times AF \times DA \times 10^{-6}\,kg/mg\right) + \left(CSF_i \times IR_i \times \left(\frac{1}{VF} + \frac{1}{PEF}\right)\right)\right]}$$

RAL for non-cancer effects:

$$RAL = \frac{THI \times BW \times AT}{EF \times ED \times \left[\left(\frac{1}{RfD_o} \times IR_o \times 10^{-6}\,kg/mg\right) + \left(\frac{1}{RfD_d} \times SA \times AF \times DA \times 10^{-6}\,kg/mg\right) + \left(\frac{1}{RfD_i} \times IR_i \times \left(\frac{1}{VF} + \frac{1}{PEF}\right)\right)\right]}$$

Where:

RAL = remedial action level
TR = target cancer risk (unitless)
THI = target hazard index (unitless)
BW = body weight (kg)
AT = averaging time (days)
EF = exposure frequency (days/yr)
ED = exposure duration (years)
IR_o = ingestion rate, oral (mg/day)
SA = surface area of skin exposed (cm^2/day)
AF = adherence factor (mg/cm^2)
DA = dermal absorption (unitless)
IR_i = inhalation rate (m^3/day)
VF = volatilization factor (m^3/kg)
PEF = particulate emission factor (m^3/kg)
CSF_o = oral cancer slope factor $(mg/kg\text{-}day)^{-1}$
CSF_d = dermal cancer slope factor $(mg/kg\text{-}day)^{-1}$
CSF_i = inhalation cancer slope factor $(mg/kg\text{-}day)^{-1}$
RfD_o = oral reference dose (mg/kg-day)
RfD_d = dermal reference dose (mg/kg-day)
RfD_i = inhalation reference dose (mg/kg-day)

The equations are functionally equivalent to those used by EPA Region 9 in developing their Preliminary Remediation Goals (PRGs – EPA, 2004). However, as described in more detail below, several of the inputs to these equations have been refined to incorporate more

site-relevant data as well as more recent data on human exposure parameters. The inhalation component of the equations also includes intake from airborne concentrations of chemicals resulting from volatilization and airborne dusts. The PRG calculation for dieldrin, on the other hand, only includes intake from dusts. Some of the newer exposure parameters were taken from regulatory guidance issued by the Florida Department of Environmental Protection (FDEP, 2005). In fact, the RAL equations are identical to those used by the FDEP to calculate risk-based target levels for their corrective action programs. The input parameters used to calculate the dieldrin RALs are discussed in more detail in the following sections.

5.2 Exposure Parameters

The RAL equations shown above require the selection of toxicity values, exposure parameters, and several physical/chemical parameters for each chemical. The following discussions present the approach used to select exposure parameters for residential receptors. In accordance with EPA guidance (1989), these exposure parameters estimate the reasonable maximum exposure (RME) for a residential receptor. RME is defined as "the maximum exposure that is reasonably expected to occur at a site" and the EPA has indicated that individual factors included in estimating exposure for an RME receptor should result in a final exposure estimate that approximates an upper percentile from a range of possible exposure estimates (EPA, 1991b). It is important to point out that this requirement does not suggest that every exposure factor represent an upper percentile estimate. If upper percentile values are chosen for every exposure factor, the resulting exposure estimate is no longer reasonable and in fact, may exceed the realm of possibility altogether. The exposure parameters for each receptor scenario are discussed in the following sections.

Risk assessments typically evaluate the potential chronic health effects. This requires the estimation of cumulative exposure over a period of years or even decades. Typically, children are assumed to experience the greatest daily exposure to soil under residential land use scenarios. For this reason, the exposure period for the residential scenario includes time spent at the site both as a child and as an adult. Most physiological parameters such as body weight, skin surface area, and inhalation rate change with age. Other exposure parameters such as soil ingestion rate are also age-dependent. To account for this, time-weighted average values reflecting both childhood, adolescent, and adult exposures were used to calculate RALs for dieldrin as well as other COPCs effects. The individual exposed from childhood on through adulthood is called the "long-term resident."

As described previously, the FDEP has completed a significant amount of study in recent years to refine certain exposure parameters used in developing risk-based target levels for soil (FDEP, 2005). This involved the analysis of data from the third *National Health and Nutrition Examination Survey* (NHANES III) to develop the most up-to-date estimates for body weight, skin surface area, and inhalation rates. FDEP undertook this analysis

because the more recent information indicates that body weights have changed nationally since the NHANES II survey in the mid-1980s. Increases in body weights mean that surface areas have changed as well. Use of the more recent data provides a more accurate and contemporary view of these body parameters that affect risk. Since these parameters all change dramatically with age, the FDEP also refined the manner in which data for these parameters are time-weighted for use in risk assessment. This approach, made possible by the more comprehensive dataset available directly from NHANES III, offers more precise estimates of these exposure parameters.

The exposure parameters for the long-term resident are explained below.

- An incidental soil ingestion rate of 200 mg/day was selected as an average daily soil ingestion rate (IR_o) for children under six years old, while 100 mg/day was selected as the IR_o for older residents. To derive an IR_o for the long-term resident, the IR_o for the child (200 mg/day) and the older residents (100 mg/day) were time-weighted to derive an average ([(6 years × 200)+(24 years × 100)]/30 years) of 120 mg/day. It should be noted the EPA's *Child Specific Exposure Factors Handbook* (2002c) actually states that "100 mg/day is the best estimate of mean (IR_o) for children under 6 years of age," but notes that "200 mg/day may be used as a conservative estimate of the mean." Accordingly, the use of 200 mg/day to estimate the exposure of residents to soil is another protective assumption utilized in this evaluation.

- The body weight (BW) values used to develop the dieldrin RALs were taken directly from FDEP (2005). These values are based on refined body weight averages derived using NHANES III data. The BW used for the long-term resident is 51.9 kg and represents the time-weighted average BW for males and females between the ages 1 and 31.

- The skin surface area (SA) values used to develop the RALs were taken directly from FDEP (2005). These values are based on refined body weight averages derived using NHANES III data and an allometric scaling model developed by Burmaster (1998). The SA used for the long-term resident is 4810 cm^2 and represents average SA for the head, forearms, hand, and lower legs for males and females between the ages of 1 and 31.

- The adherence factor (AF) values used to develop the RALs were taken directly from FDEP (2005). These values are based on refined body surface area averages derived using NHANES III data and recent dermal exposure assessment guidance from the EPA (2000a). The AF used for children under six years old is 0.2 mg/cm^2, and represents the 95th percentile of observations of children playing at a daycare center. The AF estimated for older residents is 0.07 mg/cm^2, and is based soil adherence data from the 50th percentile of a high contact activity (gardening). To derive an AF for the long-term resident, the AF for the child (0.2 mg/cm^2) and the adult (0.07 mg/cm^2) were time-weighted to derive an average ([(6 years × 0.2)+(24 years × 0.07)]/30 years) of 0.1 mg/cm^2.

- The inhalation rates (IR_i) used to develop the RALs were taken directly from FDEP (2005). FDEP derived receptor inhalation rates from the average for long-term residents based on the year-by-year average inhalation rates presented in Table 5-23 of the *Exposure Factors Handbook* (EPA, 1997). The IR_i used for the long-term resident is 12.2 m^3/day and represents the average IR_i for males and females between the ages 1 and 31.

- The exposure frequency (EF) for the long-term resident is 350 days/yr. This assumes that residents are only absent from the home for two weeks out of each year. An exposure duration (ED) of 30 years was used to calculate an RAL for carcinogenic and non-carcinogenic health effects based on the long-term resident receptor.

- Using the standard EPA approach, the doses for non-cancer health effects are averaged over the specific period of exposure for a given receptor. The non-carcinogenic averaging time (AT_{nc}) is therefore calculated by multiplying the ED (in years) for the receptor by 365 days/year. As such, the AT_{nc} for the long-term resident ages 1 to 31 years is 10,950 days. Cancer risk estimates are calculated over a lifetime exposure. The EPA standard value for the average lifespan is 70 years. Therefore, the averaging time for carcinogenic health effect (AT_c) was calculated as 25,550 days.

5.3 Toxicity Values

As discussed in Section 3.3.1, the regulatory risk assessment process separates potential adverse effects of chemicals into two categories: carcinogenic (cancer) and non-carcinogenic (non-cancer) effects. This division relates to current EPA policy that the mechanisms of action for these endpoints are different in most cases. Chemicals that are believed to be carcinogenic may also be capable of producing non-cancer health effects.

Potential carcinogenic effects resulting from exposure to chemicals are estimated quantitatively using cancer slope factors (CSFs), which represent the theoretical increased risk per milligram of chemical intake per kilogram body weight per day (mg/kg-day)-1 for the oral exposure route; or inhalation unit risk (IUR) factors, which are the theoretical increased risk at a defined exposure concentration for the inhalation route. In this risk assessment, IUR factors (mg/m3)-1 were converted to inhalation CSFs (mg/kg-day)-1 assuming an inhalation rate of 20 m3/day and an average adult body weight of 70 kg. The uncertainties associated with the application of these values to dieldrin and other cyclodiene chemicals are discussed in Section 3.3.1. For other chemicals, inhalation and dermal CSFs were calculated using route-to-route extrapolation from the applicable oral CSF exactly as described in FDEP guidance (2005).

Potential non-carcinogenic health effects resulting from exposure to chemicals are estimated quantitatively using reference doses (RfDs). The RfD is an estimate of the daily maximum level of exposure to human populations (including sensitive sub-populations) that

is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1989). For inhalation exposures, EPA has derived reference concentrations (RfCs) for some chemicals. In concept, an inhalation RfC is similar to a RfD. If the concentration of a chemical in air to which a human is exposed is lower than the RfC then there is no appreciable risk for non-cancer health effects from that exposure. In this risk assessment, RfCs (mg/m3) were converted to inhalation RfDs (mg/kg-day) using a daily inhalation rate of 20 m3/day and a body weight of 70 kg. Such a conversion allows a total dose from all routes of exposure to be calculated. For other chemicals, inhalation and dermal RfDs were calculated using route-to-route extrapolation from the applicable oral RfDs exactly as described in FDEP guidance (2005).

Where available, CSFs and RfDs for the COPCs were obtained first from the EPA Integrated Risk Information System (IRIS), followed by the EPA Health Effects Assessment Summary Tables (HEAST). Table 9 presents the cancer and non-cancer toxicity values used to calculate the RALs for dieldrin and other COPCs. If no toxicity data were available from any of these sources, a surrogate chemical was used as described in Section 3.3.2 and shown in Table 2.

5.4 Other Model Input Parameters

One of the RAL equation parameters, the particulate emission factor (PEF), is used to address intake from inhalation of chemicals on soil-derived particulates. This value is a function both of site and local climatic conditions. The formula for calculating a PEF value is taken from the EPA's Soil Screening Guidance (SSG – EPA, 1996). In calculating a PEF for Sub-Area III, default parameters from the SSG were used except for the soil particulate dispersion coefficient (Q/C) term. The default Q/C value reported in the SSG was replaced with a Q/C value for a 0.5 acre site in Little Rock, Arkansas, geographically the closest city with data available in the SSG, and therefore more representative of regional conditions.

Another of the RAL equation parameters used to assess the soil-to-air pathway of exposure is the volatilization factor (VF). This term is used to define the relationship between the concentration of the chemical in soil and the flux of the volatilized chemical to air. The VF is also calculated using an equation from the SSG. Parameters related to characteristics of both the chemical and the soil are used in the calculation of a VF. With the exception of the Q/C term, the default soil characteristics specified in the SSG have been used to calculate the dieldrin RALs. As discussed above, a Q/C for Little Rock, Arkansas is used rather than default Q/C from the SSG. Finally, chemical-specific information for most COPCs was obtained from the Superfund Chemical Data Matrix.

Table 9. Toxicity Values for Carcinogens and Non-Carcinogens

Chemical	RfDo (mg/kg-day)	RfDo Source	RfC (mg/m^3)	RfC Source	CSFo $(mg/kg\text{-}day)^{-1}$	CSFo Source	IUR $(ug/m^3)^{-1}$	IUR Source
Cyclodienes								
Aldrin	3E-05	IRIS	--		1.7E+01	IRIS	4.9E-03	IRIS
Chlordane	5E-04	IRIS	7E-04	IRIS	3.5E-01	IRIS	1.0E-04	IRIS
Dieldrin	5E-05	IRIS	--		1.6E+01	IRIS	4.6E-03	IRIS
Endosulfan	6E-03	IRIS	--		--		--	
Endrin	3E-04	IRIS	--		--		--	
Heptachlor	5E-04	IRIS	--		4.5E+00	IRIS	1.3E-03	IRIS
Heptachlor Epoxide	1.3E-05	IRIS	--		9.1E+00	IRIS	2.6E-03	IRIS
Hexachlorocyclopentadiene	6E-03	IRIS	2E-04	IRIS	--		--	
Methoxychlor	5E-03	IRIS			--		--	
Non-Cyclodienes								
4,4'-DDD	--		--		2.4E-01	IRIS	--	
4,4'-DDE	--		--		3.4E-01	IRIS	--	
4,4'-DDT	5E-04	IRIS	--		3.4E-01	IRIS	9.7E-05	IRIS
Alpha-BHC	--		--		6.3E+00	IRIS	1.8E-03	IRIS
Beta-BHC	--		--		1.8E+00	IRIS	5.3E-04	IRIS
Gamma-BHC (Lindane)	3E-04	IRIS	--		1.3E+00	HEAST	--	

5.5 RALs Based on Dieldrin

The equations and exposure parameters discussed in the previous sections were used to calculate potential RALs for dieldrin. These RALs represent chemical concentrations in soil that are considered protective of humans (including sensitive individuals) over a lifetime. They are calculated incorporating conservative estimates of residential exposure (i.e., 30 years of daily exposure to outdoor soil at one residence) and current EPA toxicity values. In accordance with EPA Region 4 guidance, three of these RALs are based on theoretical carcinogenic effects, correlating to different target risk levels of 10^{-6}, 10^{-5}, and 10^{-4}, spanning EPA's designated target risk range. The remaining RALs are based on EPA target hazard quotient (HQ) of 0.1, 1, and 3 for non-cancer health effects. This presentation of a range of potential RALs is consistent with current EPA Region 4 guidance (EPA, 2000b). The calculated RALs for dieldrin in soil are presented in Table 10. Figures 2 and 3 provide example calculations for the cancer and non-cancer based RALs for dieldrin, respectively.

Table 10. Dieldrin Remedial Goal Options

Risk Target	Dieldrin RAL (mg/kg)
1 in 1,000,000 (10^{-6})[a]	0.06
1 in 100,000 (10^{-5})[a]	0.6
1 in 10,000 (10^{-4})[a]	6
HQ = 0.1[b]	2
HQ = 1[b]	20
HQ = 3[b]	60

[a] based on theoretical excess lifetime cancer risk

[b] based on non-cancer health effects

EPA manages non-carcinogens and potential carcinogens differently in that risk from carcinogens may fall within a 10^{-6} to 10^{-4} range, whereas risk from non-carcinogens must generally fall below a threshold HQ of 1. Guidance from EPA Region 4 indicates that a 10^{-4} risk level and an HI of 1 are typically used as remediation "triggers" (EPA, 2000b).

Figure 2

Cancer RAL calculation for dieldrin based on a target cancer risk of 1×10^{-5}:

$$RAL = \frac{0.00001 \times 51.9\text{kg} \times 25550\text{days}}{350\text{d/yr} \times 30\text{yr} \times 1 \times \left[\left(16(\text{mg/kg/d})^{-1} \times 120\text{mg/d} \times 1\text{x}10^{-6}\ \text{kg/mg}\right) + \left(16(\text{mg/kg/d})^{-1} \times 4810\text{cm}^2\text{/d} \times 0.1\text{mg/cm}^2 \times 0.01 \times 1\text{x}10^{-6}\ \text{kg/mg}\right) + \left(16(\text{mg/kg/d})^{-1} \times 12.2\text{m}^3\text{/d} \times \left(\frac{1}{2.069\text{x}10^{6}} + \frac{1}{1.07\text{x}10^{9}}\right)\right)\right]}$$

$$\text{RAL} = \frac{13.26}{10500 \times \left[(1.92\times10^{-3}) + (7.70\times10^{-5}) + (9.45\times10^{-5})\right]}$$

$$RAL = \frac{13.26}{10500 \times (2.09\times10^{-3})} = \frac{13.26}{21.95} = 0.6\ \text{mg/kg}$$

**** Final RAL value rounded to 1 significant figure**

Given: CSFo = 16 (mg/kg/day)-1
CSFd = 16 (mg/kg/day)-1
CSFi = 16 (mg/kg/day)-1
VF = 2.069×106 m3/kg
PEF = 1.07×109 m3/kg

Figure 3

Non-cancer RAL calculation for dieldrin based on a target hazard quotient of 1.0:

$$RAL = \frac{1.0 \times 51.9\text{kg} \times 10950\text{days}}{350\text{d/yr} \times 30\text{yr} \times 1 \times \left[\left(\frac{1}{5\text{x}10^{-5}\text{mg/kg/d}} \times 120\text{mg/d} \times 1\text{x}10^{-6}\text{kg/mg}\right) + \left(\frac{1}{5\text{x}10^{-5}\text{mg/kg/d}} \times 4810\text{cm}^2\text{/d} \times 0.1\text{mg/cm}^2 \times 0.01 \times 1\text{x}10^{-6}\text{kg/mg}\right) + \left(\frac{1}{5\text{x}10^{-5}\text{mg/kg/d}} \times 12.2\text{m}^3\text{/d} \times \left(\frac{1}{2.069\text{x}10^{6}} + \frac{1}{1.07\text{x}10^{9}}\right)\right)\right]}$$

$$\text{RAL} = \frac{5.68 \times 10^5}{10500 \times \left[(2.4) + (9.62 \times 10^{-2}) + (1.18 \times 10^{-1})\right]}$$

$$RAL = \frac{5.68 \times 10^5}{10500 \times 2.61} = \frac{5.68 \times 10^5}{2.74 \times 10^4} = 20\ \text{mg/kg}^{**}$$

**** Final RAL value rounded to 1 significant figure**

Given: $RfD_o = 5.0 \times 10^{-5}$ mg/kg/day
$RfD_d = 5.0 \times 10^{-5}$ mg/kg/day
$RfD_i = 5.0 \times 10^{-5}$ mg/kg/day
$VF = 2.069 \times 10^6$ m^3/kg
$PEF = 1.07 \times 10^9$ m^3/kg

5.6 Development of a Secondary Criterion to Supplement Dieldrin RALs

The range of potential RAL values presented above is based exclusively on concentrations of dieldrin. As described in the preceding sections, selecting a final RAL on the basis of dieldrin concentrations is generally a conservative indicator compound to identify areas requiring corrective action. It has been demonstrated that dieldrin is the most significant contributor to cancer risk at the overwhelming majority of Sub-Area III properties included in this evaluation. As discussed in Section 3.3, however, there are a limited number of properties where COPCs besides dieldrin may contribute more than 10% of the total cancer or non-cancer risk. Therefore, a more detailed evaluation of cumulative risk at all properties was conducted to ensure that a final RAL based on dieldrin would also provide protection from other COPCs.

This is a much more significant issue for non-cancer risk, as there are several other chemicals that significantly contribute to non-cancer toxic potency (Table 4). As discussed previously, dieldrin concentrations are highly correlated with the concentrations of other significant contributors to toxic potency including aldrin, endrin, endrin ketone, heptachlor, heptachlor epoxide, and isodrin. To evaluate the potential significance of the other COPCs, non-cancer RALs for these COPCs were calculated according to the methodology and sources of input parameters identified in Section 5. These RALs are presented in Table 11.

Table 11. RALs for All Pesticides

Chemical	RAL at a Hazard Quotient = 1.0 (mg/kg)	RAL at a Cancer Risk = 10^{-5} (mg/kg)
Aldrin	10	0.6
Chlordane (alpha+gamma)	200	28
4,4'-DDD	--	42
4,4'-DDE	--	29
4,4'-DDT	200	29
Dieldrin	20	0.6
Endosulfan (alpha+beta+sulfate)	2000	--
Endrin	100	--
Heptachlor	200	2
Heptachlor epoxide	5	1
Hexachlorobenzene	200	4
Alpha-BHC	100	1
Beta-BHC	100	5
Delta-BHC	100	--
Gamma-BHC	100	7
Hexachlorocyclopentadiene	40	--
Methoxychlor	2000	--

5.6.1 Calculation of Cumulative Risk Estimates

The RALs provided in Table 10 were used in a ratio-based method to calculate cancer and non-cancer risk estimates for residential receptors at individual Sub-Area III properties. Using this method, the representative concentration (i.e., the property wide average) of each COPC is divided by the applicable RAL from Table 10. For carcinogens, these quotients are then multiplied by 1×10^{-5} to determine relative risk for that COPC. For chemicals without RALs in Table 10, surrogate values were applied based on the information presented in Table 2. Finally, the cancer and non-cancer estimates are summed to derive an aggregate risk and hazard for each medium.

The following equation was used to calculate cumulative cancer risk for carcinogenic COPCs:

$$CR = \sum_i \left(EPC/RAL\right)_i \left(10^{-5}\right)$$

Where:
CR = cumulative cancer risk
i = carcinogenic COPC i
EPC_i = property-wide average concentration of COPC i
RAL_i = RAL for COPC i (based on carcinogenic endpoints and target risk = 1×10^{-5}).

The following equation was used to calculate total non-cancer Hazard Index (HI) for COPCs. Note that the HI is a summation of the individual hazard quotients (HQs) for all COPCs in a given medium:

$$HI = \sum_j \left(EPC/RAL\right)_j$$

Where:
HI = hazard index for non-cancer effects
j = non-cancer COPC j
$EPCj$ = property-wide average concentration of COPCj
$RALj$ = RAL for COPC j (based on non-cancer endpoints and a target HQ=1).

An example calculation of cumulative cancer and non-cancer risk for a hypothetical property using this ratio-based method is provided in Table 12. This table is intended only to provide a basic example of the risk calculation methodology and is therefore limited to an abbreviated list of target analytes.

Table 12. Example Calculation of Cumulative Cancer and Non-Cancer Risk

Chemical	Representative Concentration (mg/kg)	Cancer RAL (mg/kg)	Non-Cancer RAL (mg/kg)	Cancer Risk	Non-Cancer HI
Aldrin	0.016	0.6	10	2.7×10^{-7}	0.002
α-Chlordane	0.003	28	200	1.1×10^{-9}	0.00002
Dieldrin	0.46	0.6	20	7.7×10^{-6}	0.02
Endrin	0.90	--	100	--	0.009
Heptachlor	0.011	2	200	5.5×10^{-8}	0.00006
Hex VCL	0.003	--	100	--	0.00003
			Cumulative:	**8.0×10^{-6}**	**0.03**

5.6.2 Risk Calculation Results

Tables 13 and 14 present the cumulative cancer and non-cancer risk estimates by property. Table 13 presents results for properties which have been identified as residential either by the presence of a home or apartments on the property, or the property's proximity to other residential parcels. Table 14 presents results for properties which do not currently support residential land use. As discussed previously, these risk estimates would only be applicable to non-residential properties if they were converted to residential use sometime in the future. Velsicol understands that TDEC may, in the future, call for risk evaluations based on actual site conditions at the non-residential properties, after the higher priority residential areas have been addressed.

These tables also present the representative concentration of dieldrin at each property as well as the summed concentration of all pesticides and related COPCs considered in this evaluation. In each table, the properties are ranked in order from highest to lowest representative dieldrin concentration.

Table 13
Cumulative Risk at Sub-Area III Residential Properties
Properties Ranked by Representative Dieldrin Concentration

Location	Dieldrin [1] (mg/kg)	Total Pesticides [2] (mg/kg)	Cumulative [3] HI	Risk
V1	47.9	2525	28	1.0E-03
V2	15.1	620	7.0	3.4E-04
V3	12.3	349	4.0	2.3E-04
V4	11.5	583	6.6	2.7E-04
V5	11.2	300	3.6	2.1E-04
V6	9.5	276	3.2	1.8E-04
V7	9.3	174	2.2	1.7E-04
V8	6.3	214	2.4	1.3E-04
V9	4.9	158	1.9	9.6E-05
V10	3.8	60.2	0.8	6.5E-05
V11	3.5	94.6	1.1	6.5E-05
V12	3.5	161	1.8	7.7E-05
V13	3.0	81.7	1.0	5.6E-05
V14	2.7	24.0	0.4	4.8E-05
V15	2.7	88.2	1.0	4.7E-05
V16	2.6	38.9	0.5	4.7E-05
V17	2.6	57.8	0.7	4.9E-05
V18	2.0	42.3	0.5	3.5E-05
V19	2.0	45.3	0.5	3.5E-05
V20	2.0	40.5	0.5	3.4E-05
V21	1.8	41.8	0.5	3.3E-05
V22	1.7	55.6	0.7	3.7E-05
V23	1.6	38.1	0.4	2.8E-05
V24	1.6	26.8	0.3	2.7E-05
V25	1.5	26.5	0.3	2.7E-05
V26	1.3	38.8	0.5	2.5E-05
V27	1.3	29.2	0.4	2.3E-05
V28	1.3	23.3	0.3	2.2E-05
V29	1.3	17.3	0.2	2.1E-05
V30	1.2	17.0	0.2	2.0E-05
V31	1.1	20.7	0.3	2.2E-05
V32	1.0	16.2	0.2	1.9E-05
V33	1.0	27.0	0.3	1.9E-05
V34	1.0	20.2	0.2	1.7E-05
V35	0.93	18.4	0.2	1.7E-05
V36	0.91	6.1	0.1	1.6E-05
V37	0.89	20.0	0.2	1.6E-05
V38	0.89	19.7	0.2	1.7E-05
V39	0.83	12.8	0.2	1.5E-05
V40	0.83	21.9	0.3	1.6E-05
V41	0.82	13.1	0.2	1.5E-05
V42	0.80	13.6	0.2	1.5E-05
V43	0.74	13.9	0.2	1.3E-05
V44	0.73	5.45	0.1	1.3E-05
V45	0.67	4.74	0.1	1.2E-05
V46	0.65	11.6	0.1	1.2E-05
V47	0.64	1.6	0.04	1.1E-05
V48	0.64	16.0	0.2	1.2E-05
V49	0.62	7.4	0.1	1.1E-05
V50	0.60	7.7	0.1	1.2E-05
V51	0.60	13.7	0.2	1.1E-05
V52	0.60	8.9	0.1	1.1E-05
V53	0.56	7.3	0.1	9.8E-06

Location	Dieldrin [1] (mg/kg)	Total Pesticides [2] (mg/kg)	Cumulative[3] HI	Risk
V54	0.54	2.1	0.05	9.6E-06
V55	0.53	5.4	0.1	9.5E-06
V56	0.53	7.3	0.1	1.0E-05
V57	0.53	2.5	0.1	9.3E-06
V58	0.52	5.6	0.1	9.1E-06
V59	0.51	11.3	0.1	9.9E-06
V60	0.50	10.5	0.1	9.0E-06
V61	0.50	11.9	0.1	9.6E-06
V62	0.47	10.8	0.1	9.3E-06
V63	0.47	13.4	0.2	8.3E-06
V64	0.46	3.6	0.06	8.1E-06
V65	0.41	6.0	0.08	7.6E-06
V66	0.39	2.5	0.05	7.1E-06
V67	0.39	3.1	0.05	6.7E-06
V68	0.37	4.2	0.06	6.4E-06
V69	0.33	6.3	0.08	7.1E-06
V70	0.33	5.8	0.08	7.0E-06
V71	0.33	3.4	0.05	5.9E-06
V72	0.32	4.5	0.06	5.7E-06
V73	0.31	6.8	0.08	5.6E-06
V74	0.30	3.7	0.05	5.3E-06
V75	0.30	5.9	0.08	5.3E-06
V76	0.27	4.8	0.06	4.8E-06
V77	0.26	2.3	0.04	4.7E-06
V78	0.26	2.7	0.04	4.7E-06
V79	0.25	2.2	0.03	4.4E-06
V80	0.25	2.6	0.04	4.4E-06
V81	0.23	2.3	0.03	4.2E-06
V82	0.22	3.0	0.04	5.1E-06
V83	0.21	1.1	0.02	3.6E-06
V84	0.20	1.6	0.03	3.5E-06
V85	0.18	1.3	0.02	3.2E-06
V86	0.18	1.6	0.02	3.2E-06
V87	0.17	3.0	0.04	3.6E-06
V88	0.17	1.9	0.03	4.4E-06
V89	0.17	0.76	0.02	3.1E-06
V90	0.16	1.5	0.02	2.9E-06
V91	0.16	3.3	0.04	3.6E-06
V92	0.16	4.4	0.06	3.2E-06
V93	0.16	1.1	0.02	2.9E-06
V94	0.15	5.1	0.06	2.9E-06
V95	0.14	1.5	0.02	2.5E-06
V96	0.14	0.8	0.01	2.4E-06
V97	0.13	1.0	0.02	2.3E-06
V98	0.13	4.6	0.05	2.4E-06
V99	0.13	0.72	0.01	2.2E-06
V100	0.12	1.4	0.02	2.4E-06
V101	0.11	0.63	0.01	2.0E-06
V102	0.097	1.3	0.02	1.9E-06
V103	0.093	0.88	0.01	1.7E-06
V104	0.091	0.64	0.01	1.7E-06
V105	0.085	1.1	0.02	1.8E-06
V106	0.084	2.0	0.03	2.0E-06
V107	0.084	0.62	0.01	1.5E-06
V108	0.060	0.58	0.01	1.1E-06
V109	0.058	0.18	0.00	1.1E-06
V110	0.055	0.46	0.01	1.1E-06
V111	0.054	1.6	0.02	1.2E-06

Location	Dieldrin [1] (mg/kg)	Total Pesticides [2] (mg/kg)	Cumulative[3] HI	Risk
V112	0.044	0.82	0.01	9.8E-07
V113	0.041	0.27	0.01	8.5E-07
V114	0.025	0.25	0.004	5.5E-07
V115	0.014	0.14	0.002	3.7E-07
V116	0.014	0.65	0.01	8.6E-07
V117	0.0094	0.28	0.004	2.9E-07
V118	0.0033	0.13	0.002	2.0E-07
V119	0.0031	0.22	0.003	2.1E-07
V120	0.0030	0.17	0.002	1.8E-07
V121	0.0029	0.11	0.002	1.7E-07
V122	0.0029	0.12	0.002	1.8E-07
V123	0.0028	0.12	0.002	1.8E-07
V124	0.0028	0.10	0.001	1.7E-07
V125	0.0016	0.050	0.001	7.1E-08

Notes:

1. "Dieldrin" represents the property-wide arithmetic average concentrations of dieldrin.

2. "Total Pesticides" represents the sum of the property-wide arithmetic average concentrations of DDD, DDE, DDT, aldrin, alpha-chlordane, alpha-chlordene, beta-BHC, chlordene, cis-nonachlor, delta-BHC, dieldrin, endosulfan I, endosulfan II, endosulfan sulfate, endrin, endrin ketone, gamma BHC, gamma-chlordane, heptachlor, heptachlor epoxide, heptachloronorborene (Hex VCL), hexachlorobenzene, hexachlorocyclopentadiene, hexachloronorbornadiene (hex BCH), isodrin, octachlorocyclopentene, oxychlordane, and trans-nonachlor.

3. Represents cumulative Hazard Index (HI) and theoretical excess lifetime cancer risk (Risk) estimates from exposure to "Total Pesticides".

Represents properties with risks above the upper end of EPA's risk range (1E-4) or a hazard Index of 1.0 based on exposure to "Total Pesticides".

Represents the dieldrin concentrations at properties exceeding EPA Risk Targets.

Table 14

Cumulative Risk at Sub-Area III Non-Residential Properties[1]
Properties Ranked by Representative Dieldrin Concentration

Location	Dieldrin[2] (mg/kg)	Total Pesticides[3] (mg/kg)	Cumulative[4] HI	Cumulative[4] Risk
V126	11.0	316	3.7	2.04E-04
V127	5.4	31.4	0.5	9.10E-05
V128	5.2	135	1.6	1.11E-04
V129	3.2	103	1.2	5.92E-05
V130	2.5	42.3	0.5	4.24E-05
V131	2.4	57.2	0.7	4.23E-05
V132	2.0	62.2	0.7	3.86E-05
V133	1.2	24.1	0.3	2.19E-05
V134	1.1	8.6	0.1	1.89E-05
V135	0.99	25.9	0.3	1.98E-05
V136	0.85	14.4	0.2	1.48E-05
V137	0.80	19.6	0.2	1.66E-05
V138	0.58	10.4	0.1	1.04E-05
V139	0.43	6.6	0.09	7.57E-06
V140	0.38	6.7	0.09	7.19E-06
V141	0.31	3.9	0.06	5.51E-06
V142	0.23	3.4	0.05	6.71E-06
V143	0.18	4.3	0.1	7.32E-06
V144	0.13	3.3	0.04	3.20E-06
V145	0.06	2.0	0.02	1.67E-06
V146	0.01	0.13	0.002	3.19E-07

Notes:

1. The properties identified in this table do not currently support residential land use. However, to provide an equivalent metric for comparison of all Sub-Area III properties, the risk estimates provided in this table are based on exposure assumptions for a residential receptor.
2. "Dieldrin" represents the property-wide arithmetic average concentrations of dieldrin.

3. "Total Pesticides" represents the sum of the property-wide arithmetic average concentrations of DDD, DDE, DDT, aldrin, alpha-chlordane, alpha-chlordene, beta-BHC, chlordene, cis-nonachlor, delta-BHC, dieldrin, endosulfan I, endosulfan II, endosulfan sulfate, endrin, endrin ketone, gamma BHC, gamma-chlordane, heptachlor, heptachlor epoxide, heptachloronorborene (Hex VCL), hexachlorobenzene, hexachlorocyclopentadiene, hexachloronorbornadiene (hex BCH), isodrin, octachlorocyclopentene, oxychlordane, and trans-nonachlor.
4. Represents cumulative Hazard Index (HI) and theoretical excess lifetime cancer risk (Risk) estimates from exposure to "Total Pesticides".

Represents properties with risks above the upper end of EPA's risk range (1E-4) or a hazard Index of 1.0 based on exposure to "Total Pesticides".

Represents the dieldrin concentrations at properties exceeding EPA Risk Targets.

6.0 REMEDIAL ACTION LEVEL SELECTION AND IMPLEMENTATION

6.1 Context of the EPA's Target Risks for Remedial Decision Making

The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (EPA, 1986b) originally established the EPA's risk range for making remedial decisions at contaminated sites. The EPA has consistently incorporated the NCP criteria in their guidance for conducting human health risk assessments.

In 1991, for example, the EPA's Office of Solid Waste and Emergency Response (OSWER) issued a Directive that states:

> *"where cumulative carcinogenic site risk to an individual based on reasonable maximum exposure for both current and future land use is less that 10^{-4} and the non-carcinogenic hazard quotient is less than 1, action is not generally warranted unless there are adverse environmental impacts" (EPA, 1991c).*

This OSWER Directive goes on to state that

> *"the upper boundary of the risk range is not a discrete line at 1×10^{-4}, although EPA generally uses 1×10^{-4} in making risk management decision. A specific risk estimate around 10^{-4} may be considered acceptable if justified based on site-specific conditions, including remaining uncertainties on the nature and extent of contamination and associated risks. Therefore, in certain cases EPA may consider risk estimates slightly greater than 1×10^{-4} to be protective" (EPA, 1991c).*

This policy has been affirmed in subsequent EPA risk assessment guidance documents including *Risk Assessment Guidance for Superfund (RAGS) Part B* (EPA, 1991a) and *RAGS Part D* (EPA, 2001). Not only does supplemental risk assessment guidance from EPA Region 4 endorses the use of a target cancer risk range between 10^{-6} and 10^{-4} for the development of remedial goal options (EPA, 2000b), this guidance also recommends that remedial goals for non-carcinogens be developed based on a range of hazard quotients corresponding 0.1, 1.0, and 3.0 to address the uncertainty incorporated in the derivation of RfDs.

From this discussion, it should be evident that risk-based screening-levels, such as the Region 9 PRGs, developed by the EPA and State environmental regulatory agencies using a target 1×10^{-6} excess lifetime cancer risk and a target hazard quotient of 1.0 should not be taken out of context. These risk-based values are intended only for screening purposes to determine where chemical concentrations are high enough to warrant further study, not as triggers to require remediation. A brief historical perspective on the origins of 10^{-6} as a screening criterion may be helpful in illustrating this point.

The 10^{-6} cancer risk criterion appears to have first been used as a matter of policy by the FDA in 1977 to establish a screening level of "essentially zero" or "de minimis risk[4]" in guidelines for assay methods for residues of potentially carcinogenic veterinary pharmaceuticals in meat and dairy products that could be consumed by humans (FDA, 1977). As the basis for this criterion, the FDA cited a statistical article by researchers at the National Cancer Institute studying the number of animals required to establish the safety of a chemical (Mantel and Bryan, 1961). It is ironic that this research was not intended to establish the definition of a "safe" level. However, 10^{-6} was thus established as the level below which no further regulatory action would be taken over the levels of potentially carcinogenic animal pharmaceutical residues in food products.

Current statistics indicate that one out of every two men and one out of every three women in the United States will develop cancer over the course their lifetime (James and Saranko, 2000). This corresponds to a risk greater than 10^{-1}. The overwhelming majority of exposures to carcinogens come from naturally occurring carcinogens in our foods (Ames et al., 1990, Ames and Gold, 1997). The probability of developing cancer associated with ambient levels of chemicals in the environment has been estimated at 10^{-3} to 10^{-2} (Travis and Hester, 1991), which corresponds to only 1-2% of the total cancer risk. These risks are driven by chemicals in the air we breathe and the water we drink. Thus, the EPA's risk range of 10^{-6} to 10^{-4} is orders of magnitude lower than our current risk from background exposure to environmental contaminants and even more miniscule when compared with our overall cancer risk. Further, since cancer risk estimates have been based on EPA's conservative risk assessment framework, the relative difference may actually be much greater.

6.2 Selection of Residential RALs

The previous section provides a brief discussion of the range of cancer and non-cancer risks that EPA has deemed appropriate for consideration when selecting a final RAL. Other sections of this report have presented multiple lines of evidence to support the conclusion that the EPA risk assessment paradigm used in this evaluation tends to be biased towards overestimating potential risks from chemicals in the environment.

The analysis presented in this report indicates that a single RAL based on the indicator compound dieldrin, with consideration of secondary contributors to non-cancer risk, can address potential risks from all cyclodiene chemicals in Sub-Area III. As shown on Tables 13 and 14, an RAL of 3 mg/kg dieldrin is supported by the present analysis as health protective of both cumulative cancer and non-cancer health effects from all COPC in Cypress Creek Sub-Area III. Below this RAL, all Sub-Area III properties sampled to date have cumulative cancer risk estimates below 5×10^{-5} and non-cancer HIs below 1.

[4] The term de minimis is an abbreviation of the legal term "de minmis con curat lex," which means "the law does not concern itself with trifles."

7.0 REFERENCES

Ames, BN; Profet, M; and Gold, LS. (1990). Dietary Pesticides (99% all natural). Proceeding of the National Academy of Science, 87: 7777-7781.

Ames, BN and Gold, LS. (1997). The causes and prevention of cancer: Gaining perspective. Environmental Health Perspectives, 105(Sup. 4): 865-873.

Amoateng-Adjepong, Y; Sathiakumar, N; Delzell, E; and Cole, P. (1995). Mortality among workers at a pesticide manufacturing plant. *Journal of Occupational and Environmental Medicine*, 37: 471-478.

Beall, ML Jr. and Nash, RG. (1969). Crop seedling uptake of DDT, dieldrin, endrin, and heptachlor from soils. *Agronomy Journal*, 61: 571-575.

Beall, ML Jr. and Nash, RG. (1971). Organochlorine insecticide residues in soybean plant tops: Root vs. vapor sorption. *Agronomy Journal*, 63: 460-464.

Beal, ML Jr.; Harris, WG; and Nash, RG. (1972). Endrin transformations in soil. *Journal of Environmental Quality*, 1: 391-394.

Boersma, L; Lindstrom, FT; McFarlane, C; and McCoy, EL. (1988). Uptake of organic chemicals by plants: A theoretical model. *Soil Science*, 1466: 403-417.

Boersma, L; McFarlane, C; Lindstrom FT. (1991). Mathematical model of plant uptake and translocation of organic chemicals: application to experiments. *Journal of Environmental Quality*, 20: 137-146.

Briggs, GG; Bromilow, RH; Evans, AA. (1982). Relationships between lipophilicity and root uptake and translocation of non-ionized chemicals by barley. *Pesticide Science*, 13: 495-504.

Briggs, GG; Bromilow RH; Evans AA; and Williams, M. (1983). Relationships between lipophilicity and the distribution of non-ionized chemicals in barley shoots following uptake by the roots. *Pesticide Science*, 14: 492-500.

Burmaster, DE. (1998). Lognormal distributions for skin area as a function of body weight. *Risk Analysis*, 18: 27-32.

Chiou, CT; Sheng, G; and Manes, M. (2001). A partition-limited model for the plant uptake of organic contaminants from soil and water. *Environmental Science and Technology*, 35: 1437-1444.

Cole, LK; Metcalf, RL; and Sanborn, JR. (1976). Environmental fate of insecticides in terrestrial model ecosystems. *International Journal of Environmental Studies*, 10: 7-14.

de Jong, G. (1991). Long-term health effects of aldrin and dieldrin. *Toxicology Letters*, Supplement 1991.

(EPA) United States Environmental Protection Agency. (1986a). Guidelines for Carcinogen Risk Assessment. EPA Risk Assessment Forum, Washington, DC. EPA/630/R-00/004.

EPA. (1986b). National Oil and Hazardous Substances Pollution Contingency Plan (NCP). 40 CFR 300.

EPA. (1989). Risk Assessment Guidance for Superfund. Volume 1, Human Health Evaluation Manual (Part A), Interim Final. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/002.

EPA. (1991a). Risk Assessment Guidance for Superfund. Volume 1, Human Health Evaluation Manual (Part B), Interim Final. Office of Emergency and Remedial Response, Washington, DC. EPA/540/R-92/01B.

EPA. (1991b). Risk Assessment Guidance for Superfund. Volume I, Human Health Evaluation Manual (Supplemental Guidance), Standard Default Exposure Factors – Interim Final, OSWER Directive 9285.6-03.

EPA. (1991c). Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Office of Solid Waste and Emergency Response. Washington, DC. OSWER Directive 9355.0-30.

EPA. (1996). Soil Screening Guidance: Technical Background Document. Office of Solid Waste and Emergency Response, Washington, DC. EPA/540/R-95/128.

EPA. (1997). Exposure Factors Handbook: Volumes 1-III. Office of Research and Development, National Center for Environmental Assessment, Washington, DC. EPA/600/P-95/002Fa.

EPA. (2000a). Risk Assessment Guidance for Superfund: Volume I: Human Health Evaluation Manual (Part E) Interim Guidance. Office of Emergency and Remedial Response, Washington, DC.

EPA. (2000b). Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment Bulletins. EPA Region 4, November 1995, Website version last updated May 2000: http://www.epa.gov/region4/waste/oftecser/healtbul.htm.

EPA. (2001) Risk Assessment Guidance for Superfund – Volume I, Human Health Evaluation Manual (Part D). Office of Emergency and Remedial Response, Washington, DC. Publication number 9285.7-47.

EPA. (2002a) Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Solid Waste and Emergency Response, Washington, DC. OSWER 9355.4-24.

EPA. (2002b) Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites. Office of Solid Waste and Emergency Response, Washington, DC. OSWER 9285.6-10.

EPA. (2002c). Child-Specific Exposure Factors Handbook. National Center for Environmental Assessment, Washington, DC. NCEA-W-0853.

EPA. (2003). Health Effects Support Document for Aldrin/Dieldrin. Office of Water, February 2003, EPA 822-R-03-001.

EPA. (2004). Region 9 Preliminary Remediation Goals (PRGs) 2004. USEPA Region 9, San Francisco, CA. http://www.epa.gov/region09/waste/sfund/prg/index.htm.

EPA. (2005a). Guidelines for Carcinogen Risk Assessment. EPA Risk Assessment Forum, Washington, DC. EPA/630/P-03/001F.

EPA. (2005b). Integrated Risk Information System (IRIS). http://www.epa.gov/iris.

(FDA) United States Food and Drug Administration (1977). Compounds used in food-processing animals: Procedures for determining acceptability of assay methods used for assuring the absence of residues in edible products of such animals. Final Rule. *Federal Register*, February 22: 10412.

(FDEP) Florida Department of Environmental Protection. (2004). Technical Report: Development of Cleanup Target Levels (CTLs) for Chapter 62-777, F.A.C., Prepared for the Division of Waste Management, Florida Department of Environmental Protection. February, 2005.

Gupta, HCL; Kushwaha, KS; and Kavdia VS. (1979). Aldrin residues in soils and its translocation in maize and pearl-millet. *Indian Journal of Entomology*, 41: 47-57.

Hung, H and Mackay, D. (1997). A novel and simple model of the uptake of organic chemicals by vegetation from air and soil. *Chemosphere*, 355: 959-977.

James, RC and Saranko, CJ. (2000). Carcinogenesis. In: *Principles of Toxicology: Environmental and Industrial Applications, Second Edition.* (Williams, P.L., James, R.C. and Roberts, S.M., eds.), New York: John Wiley & Sons.

Klassan, CD, ed. (2001). *Casarett and Doull's Toxicology – The Basic Science of Poisons, 6th Edition.* McGraw-Hill, New York.

Kloskowski, R; Scheunert, I; and Klein, W. (1981). Laboratory screening of distribution, conversion and mineralization of chemicals in the soil-plant-system and comparison to outdoor experimental data. *Chemosphere*, 10: 1089-1100.

Klaunig, JE; Xu, Y; Bachowski, S; Ketcham, CA; Isenberg, JS; Kolaja, KL; Baker, TK; Walborg, EF; and Stevenson, DE. (1995). Oxidative stress in non-geneotoxic carcinogenesis. *Toxicology Letters*, 82/83: 682-691.

Lindstrom, FT; Boersma, L; McFarlane, C. (1991). Mathematical model of plant uptake and translocation of organic chemicals: development of the model. *Journal of Environmental Quality*, 20: 129-136.

Mantel, N and Bryan, WR. (1961). Safety testing of carcinogenic agents. *Journal of the National Cancer Institute*, 27: 455-470.

Mattina, MJ; Iannucci-Berger, W; and Dykas, L. (2000). Chlordane uptake and its translocation in food crops. *Journal of Agricultural and Food Chemistry*, 48:1909-1915.

(MEC) Memphis Environmental Center. (2002a). *Cypress Creek Drainage Channel Investigation and Preliminary Human Health Risk Evaluation.* January 10, 2002.

MEC. (2002b). *Cypress Middle School - University Park Area, Cypress Creek Investigation and Human Health Risk Evaluation.* September 18, 2002.

MEC. (2003). *Cypress Creek Sub-Area III Investigation Report.* October, 2003.

MEC. (2004). *Revised Interim Measures Work Plan for Cypress Creek Sub-Area III.* April 8, 2004.

MEC. (2005). *Cypress Creek Sub-Area III 2004 Interim Measures Investigation Report.* January 2005.

Nash, RG and Harris, WG. (1973). Chlorinated hydrocarbon insecticide residues in crops and soil. *Journal of Environmental Quality*, 2: 267-273.

Paterson, S and Mackay, D. (1994). A model of organic chemical uptake by plants from soil and atmosphere. *Environmental Science and Technology*, 28: 2259-2266.

Premiere Environmental Services. (2005). *Cypress Creek Interim Measures Investigation. October 2005.*

Rosner, B and Belmont CA. (1990). *Fundamentals of Biostatistics-3rd Edition*. Duxbury Press.

Ryan, JA; Bell, RM; Davidson, JM; and O'Connor, GA. (1988). Plant uptake of non-ionic organic chemicals from soils. *Chemosphere*, 1712: 2299-2323.

Sielkin, RL, Bretzlaff, RS, Stevenson, DE, de Jong, G. (1998). Updated mortality analysis of Pernis epidemiological data on human exposures to aldrin and dieldrin. *Human and Ecological Risk Assessment*, 4: 201-225.

Singh, G; Higginson, FR; and Fenton, IG. (1990). Translocation of aged cyclodiene insecticide residues f;om soil into forage crops under optimal glasshouse conditions. *Journal of Environmental Science and Health [B]*, 25: 295-308.

Singh, G; Kathpal, TS; and Spencer, WF. (1991). Dissipation of some organochlorine insecticides in cropped and uncropped soil. *Environmental Pollution*, 70: 219-240.

(TDEC) Tennessee Department of Environment and Conservation. (2004). Risk Assessment Directive for Sub-Area III of Cypress Creek. Letter dated July 29, 2004.

Topp, E; Scheunert, I; Attar, A; and Korte, F. (1986). Factors Affecting the Uptake of ^{14}C-Labeled Organic Chemicals by Plants from Soil. *Ecotoxicology and Environmental Safety*, 11: 219-228.

Trapp, S; Matthies, M; Scheunert, I; and Topp, EM. (1994). Modeling the bioconcentration of organic chemicals in plants. *Environmental Science and Technology*, 248: 1246-1252.

Travis, CC and Arms, AD. (1988). Bioconcentration of organics in beef, milk and vegetation. *Environmental Science and Technology*, 22: 271-274.

Travis, CC and Hester, ST. (1991). Global Chemical Pollution. *Environmental Science and Technology*, 25:814-819.