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# Development of Risk Based Treatability and Engineering Measures for Reducing Exposure to Lead Contaminated Media in the Miami Inner City, Florida

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FLORIDA INTERNATIONAL UNIVERSITY

Miami, Florida

# DEVELOPMENT OF RISK BASED TREATABILITY AND ENGINEERING MEASURES FOR REDUCING EXPOSURE TO LEAD CONTAMINATED MEDIA IN THE MIAMI INNER CITY, FLORIDA

A dissertation submitted in partial fulfillment of the

requirements for the degree of

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in

CIVIL ENGINEERING

by

Tarla TaMia Toomer

2008

To: Interim Dean Amir Mirmiran
College of Engineering and Computing

This dissertation, written by Tarla TaMia Toomer, and entitled Development of Risk Based Treatability and Engineering Measures for Reducing Exposure to Lead Contaminated Media in the Miami Inner City, Florida, having been approved in respect to style and intellectual content, is referred to you for judgment.

We have read this dissertation and recommend that it be approved.

_____
Shonali Laha

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Walter Tang

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Janvier Gasana, Co–Major Professor

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Berrin Tansel, Co–Major Professor

Date of Defense: October 6, 2008

The dissertation of Tarla TaMia Toomer is approved.

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Interim Dean Amir Mirmiran
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Dean George Walker
University Graduate School

Florida International University, 2008

ACKNOWLEDGMENTS

Trust in the Lord with all thy heart, and lean not upon thine own understanding:

In all thy ways acknowledge him, and he will direct thy paths.

Proverbs 3:5-6

And to whomsoever much is given, of him shall much be required: and to whom they commit much, of him will they ask the more

Luke 12:48

Mr. Edward Alexander Bouchet, the first African American to obtain his Ph.D., in Physics from Yale University

and

Ms. Lilia Abron, the first African American woman in the nation to receive a Ph.D., in Chemical Engineering from The University of Iowa

Thank You

I am an individual who believes in divine order and predestination and a belief that God's hand is on everything act and deed that I touch. I thank God for allowing me to be in a position to bring what was is in my heart out onto paper in the area that I love. I thank my family who always believed in me not matter the subject, cost, or time commitment required to complete the task. I thank my friends who also are a constant group of support, insight, and words of encouragement. This journey began a while ago and I thank everyone involved on the ride to this exact moment in time. My prayer is that this is just the first in a long line of studies committed to this type of work.

I thank my professors who believed in my work and believed in me as an individual to accomplish this goal. First, I would like to thank Dr. Berrin Tansel, P.E., who took a calculated risk on me and my unique ideas and beliefs of incorporating issues relating to African Americans into my work from an engineering perspective. Dr. Tansel believed in me from day 1 and never wavered in her commitment to me and my life's work and purpose. I would like to especially like to thank Dr. Janvier Gasana who in the spring of 2000, would listen to me ramble along as I explained how childhood lead poisoning is linked to engineering and related more than the experts would admit to. I would also like to notably thank Dr. Shonali Laha, P.E., and Dr. Walter Tang, P.E. for also believing and allowing me to work and providing their industry and educational expertise of over 50 years of combined engineering work in an area where there isn't much work being studied from an engineering perspective.

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We did it

Thank You

ABSTRACT OF THE DISSERTATION

DEVELOPMENT OF RISK BASED TREATABILITY AND ENGINEERING MEASURES FOR REDUCING EXPOSURE TO LEAD CONTAMINATED MEDIA IN THE MIAMI INNER CITY, FLORIDA

by

Tarla TaMia Toomer

Florida International University, 2008

Miami, Florida

Professor Berrin Tansel, Co–Major Professor

Professor Janvier Gasana, Co–Major Professor

A major consequence of contamination at the local level's population as it relates to environmental health and environmental engineering is childhood lead poisoning. Environmental contamination is one of the pressing environmental concerns facing the world today. Current approaches often focus on large contaminated industrial size sites that are designated by regulatory agencies for site remediation. Prior to this study, there were no known published studies conducted at the local and smaller scale, such as neighborhoods, where often much of the contamination is present to remediate.

An environmental health study of local lead-poisoning data in Liberty City, Little Haiti and eastern Little Havana in Miami-Dade County, Florida accounted for a disproportionately high number of the county's reported childhood lead poisoning cases.

An engineering system was developed and designed for a comprehensive risk management methodology that is distinctively applicable to the geographical and environmental conditions of Miami-Dade County, Florida. Furthermore, a scientific

approach for interpreting environmental health concerns, while involving detailed environmental engineering control measures and methods for site remediation in contained media was developed for implementation. Test samples were obtained from residents and sites in those specific communities in Miami-Dade County, Florida (Gasana and Chamorro 2002).

Currently lead does not have an Oral Assessment, Inhalation Assessment, and Oral Slope Factor; variables that are required to run a quantitative risk assessment. However, various institutional controls from federal agencies' standards and regulation for contaminated lead in media yield adequate maximum concentration limits (MCLs). For this study an MCL of .0015 (mg/L) was used.

A risk management approach concerning contaminated media involving lead demonstrates that the linkage of environmental health and environmental engineering can yield a feasible solution.

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## 1. EXECUTIVE SUMMARY

Environmental contamination is one of the pressing environmental concerns facing the world today. Current approaches often focus on large contaminated industrial size sites that are designated by regulatory agencies for site remediation. However, there are no known published studies conducted at the local and smaller scale, such as neighborhoods, where often much of the contamination is present. In addition, within this large sites approach, studies have been conducted as it pertains to environmental assessment, health assessment, and risk assessment, however, to date there are not any known approaches with respect to risk management as the primary methodology, with the focus of engineering design and lead as the primary contaminant.

Lead is a multimedia toxicant that exposes its toxicity even when the specific exposures appear relatively modest. Lead is found in various conterminous media where the focus should be reduction, eradication and or elimination. At major industrial sites that contain this media, the general pathway includes soils: and dusts at superfund sites, paint in deteriorating housing, or other sites where public agencies have regulatory or intervention oversight. Characteristically, the remediation method employed by these agencies as it pertains to humans and lead exposure when long existing environmental lead concentrations in and around waste sites and adjacent communities are disturbed with lead remediation activities (Mushak 2003).

The assessment of changes in lead exposure takes place in the larger framework of human health assessment with the concentration on the measurement of biological markers at either exposure or early effect. Of all the biomarkers present to date, lead is the most widely and commonly used (Mushak 2003). Dust lead has been found to draw a

relationship strongly with soil lead concentration since soil renders considerably to support interior dust loading (Ren et al 2006).

Development of Risk based treatability and engineering measures for reducing exposure to lead contaminated media in the Miami inner city, Florida will be conducted using a risk management approach. Within the already defined risk management method, environmental assessment, risk assessment, and health assessment will be produced while a systematically methodology based on engineering design to contribute to the reduction and eradication of lead contamination at the local level.

A risk management approach concerning contaminated media involving lead demonstrates that the linkage of environmental health and environmental engineering yielded a feasible solution resulted in a hazard index resulting in values of 0.0313 (Child 2-6), 0.026 (Child 6-12), and 0.0187 (Adult); excess lifetime cancer cases resulted in 2,640 (Child 2-6), 1,476.7 (Child 6-12), and 457.3 (Adult) of a population of 100,000. Additionally, the environmental engineering control cost of phytoextraction is $2,145.39 per property yard. This study when implemented, demonstrates a successful solution can be established in bridging the environmental health and environmental engineering.

## 2. INTRODUCTION

### 2.1 ENVIRONMENTAL RISK ASSESSMENT

Environmental risk assessment is a useful method available to environmentalists for the management of hazardous waste and contaminated media. This risk based analyses provides methods to establish a relationship between levels of contaminants and public health (LaGrega et al 1994). It can also be utilized as guidance for developing

algorithms in relation to levels of exposure to those contaminants (Lorenzana et al 2003). Risk assessment has been used to describe the likelihood of different scenarios such as industrial explosions, workplace injuries, failure of machine parts, natural catastrophes, and injury or death due to an array of voluntary activities (Paustenbach 1989). The issues of risk being prevented or managed are best understood through the use of environmental controls. Environmental controls and engineering systems are laws regulating to and enforcing limits of access to areas that have a high potential for contamination (Lorenzana et al 2003).

A major problem of contamination at the local level as it relates to health is childhood lead poisoning. Current approaches of risk based assessments often focus on large contaminated industrial size sites that are designated by regulatory agencies for site remediation. However, there are no known current published studies conducted at the local and smaller scale, such as neighborhoods, where often much of the contamination is present. In addition, within this large sites, studies have been conducted as it pertains to environmental assessment, health assessment, and risk assessment, however, to date there are not any known approaches with respect to risk management as the primary methodology, with the focus of engineering design and lead as the primary contaminant.

### 2.1.1 LEAD AND ENVIRONMENTAL RISK ASSESSMENT

Lead is a multimedia toxicant that exposes its toxicity even when the specific exposures appear relatively modest. Lead is found in various contaminated media where the focus should be reduction, eradication and or elimination. At major industrial sites that contain this media, the general pathway includes soils and dusts at superfund sites, paint in deteriorating housing, or other sites where public agencies have regulatory or

intervention oversight. Characteristically, it is documented that remediation methods employed by agencies as it pertains to humans and lead exposure, when there exist, environmental lead concentrations are in and around waste sites the adjacent communities are disturbed with those lead remediation activities (Mushak 2003).

The assessment of changes in lead exposure takes place in the larger framework of human health assessment with the concentration on the measurement of biological markers at either exposure or early effect. Of all the biomarkers present to date, lead is the most widely and commonly used (Mushak 2003). Dust lead has been found to draw a relationship strongly with soil lead concentration since soil renders considerably to support interior dust loading (Ren et al 2006).

**2.2 RISK ASSESSMENT**

The purpose of the risk assessment is to aid policymakers, legislatures, and risk mangers with appropriate information so that the best management practices can be developed for risks. If the risk assessment is properly conducted, the assessment will obtain extensive acceptance and conceptualize the separation of toxic, hazard, and risk. Toxicity is an intrinsic property of all substances. All chemical and physical agents can produce adverse health effects at some dose or under specific exposure conditions (Paustenbach 1989). Hazard refers to the intrinsic potential of a waste to cause harm (LeGrange et. al 1994). Risk is the probability or likelihood that an adverse outcome will occur in a person or group that is exposed to a precise concentration or dose of the hazardous agent. As a result, risk is a function of the exposure or dose (Paustenbach 1989). Because of the use, misuse, and interchangeability of the term risk assessment, the National Academy of Science (Paustenbach 1989) has defined it as such:

> Risk assessment is the mean characterization of the potential adverse health effects of human exposures to environmental hazards. Risk assessments include several element description of the potential adverse health effects based on an evaluation of results of epidemiological, clinical, toxicological, and environmental research, extrapolation from those results to predict the type and estimates the extent of health effects in humans under given conditions of exposure, judgments as to the number and characteristics of persons exposed at various intensities and durations, and summary judgments of the existence and overall magnitude of the public health problem. Risk assessment also includes characterizations of uncertainties inherent in the process of inferring risk.

Zolezzi et al *(2005),* best describes risk assessment as procedures that are considered the best available tools for supporting, under scientific basics, decision making processes on a wide range of areas, from economic to environmental development for both generic and site specific assessments.

**2.3 RISK MANAGEMENT**

Risk management has often been confused with that of risk assessment. As a result, the National Academy of Science has defined risk management in the following manner:

> Risk management is the process of evaluating alternative regulatory actions and selecting among them. Risk management with is carried out by regulatory agencies under various legislative mandates, is an agency decision making process that entails considerations of political, social, economic, engineering information with risk related information to develop, analyze, and compare regulatory options and to select the appropriate regulatory response to a potential chronic health hazard. The selection process necessarily requires the use of value judgments on such issues as the acceptability of risk and the reasonableness of the costs of control. (Paustenbach 1989)

The goal of the risk management is to balance out the benefits of an action against the real and or perceived risk. Additionally, cost, feasibility, and reasonableness into the scientific justification of acceptable levels or exposure are ideal (Paustenbach 1989).

## 2.4 HEALTH RISK ASSESSMENT

The health risk assessment is the process or procedure used to calculate approximately the probability that humans or ecological systems will be affected harmfully by a chemical or a physical agent under a specific set of conditions. Specifically, it is a written document in which all relevant scientific information regarding toxicity, human experience, environmental fate, and exposure are collected, analyzed, and interpreted (Paustenbach 1989).

## 3. OBJECTIVES

Development of Risk Based Treatability and Engineering Measures for Reducing Exposure to Lead Contaminated Media in the Miami Inner City, Florida will provide a systematic methodology based on engineering design to contribute to the reduction and eradication of lead contamination at the local level. In conducting this study, a link will also be established between environmental engineering and environmental health. Thus, this problem will not be treated only as an environmental engineering problem, but also as a public (environmental) health problem. The link between engineering and environmental health carries out its mission through organized, interdisciplinary efforts that address the physical, mental and environmental health concerns of communities and populations at risk for disease and injury. Public Health's mission is achieved through the application of health promotion and disease prevention technologies and interventions designed to improve and enhance quality of life. Engineering then subsequently applies its specific application of scientific and mathematical principles to practical ends by the design, manufacturing, and or operation of efficient and economical structures, machines,

processes, and systems. Additionally, environmental engineering controls, which are laws regulating to and enforcing limits of access, can be included the removal or burial of lead burdened soils and dusts, coverage of soils with vegetation, and stabilization of soils from erosion. Furthermore, institutional engineering controls can include barriers and enforcing limits of access to areas that have a high potential for contamination (Lorenzana et al 2003). The overall objective of this work is to design a systematic methodology based on engineering design to contribute to the reduction and eradication of lead contamination at the local level with the following specific objectives:

- **OBJECTIVE 1**: Design and develop a comprehensive risk management methodology, with focus on a quantitative risk assessment that is applicable to the geographical and environmental conditions of Miami Inner City Area.
- **OBJECTIVE 2:** Develop a scientific approach for interpreting public health concerns with environmental engineering methods for remediation in contained media involving detailed techniques, procedures, and recommendation for the treatability for local community being studied.

To achieve these objectives, this research was categorized into two sections: The first section will focus on the design and development of a comprehensive risk management methodology as it related to the Miami Inner City area. The second section will focus on development of a scientific approach for interpreting public health concerns in conjunction with engineering controls methods for remediation in contained media.

# 4. METHODOLOGY

## 4.1 METHODOLOGY

**OBJECTIVE 1:** *Design and develop a comprehensive risk management methodology, with a focus on a quantitative risk assessment that is applicable to the geographical and environmental conditions of Miami Inner City Area.*

For this study, various scenarios of data where available to design and develop the comprehensive risk management applicable to the geographical and environmental conditions to Miami, Florida and the development of a scientific approach for interpreting public health concerns with engineering methods (environmental engineering control measures) for remediation in contained media. Various literature searches indicated that carcinogeneous and non carcinogeneous human health risks by using both the average and maximal intakes which considers the best and worst case risks and that was the approach that was used. Additionally, a range of institutional controls from several federal agencies' standards and regulations for the varied contaminated lead in media are available as a threshold source. Other controls include proposed maximum concentration limits (MCLs), proposed maximum concentration limit guidelines (MCLGs), and Water Quality Criteria for Fish and Drinking Water are also available for use. In this study, the Maximum Concentration Limits (MCLs) of .0015 mg/L, is used to complete the risk assessment. The institutional controls portion will be discussed further in section 2 on the methodology. For a complete literature search of the entire study, the reader is referred to Appendix A. Figure 1, the Methodological Process of the Quantitative Risk Assessment gives progressive instructions of the process flow of objective 1.

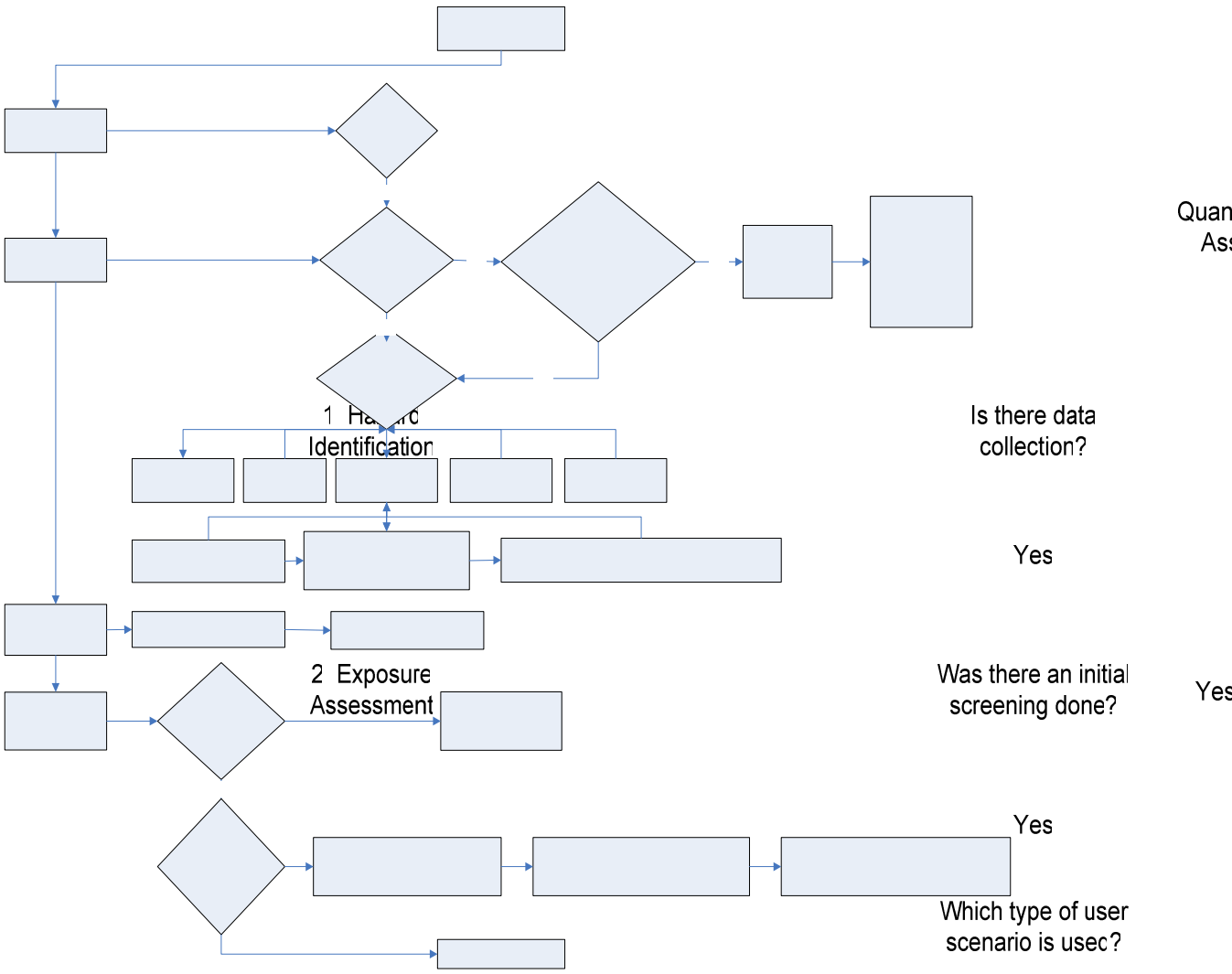



**Figure 1 Methodological Process of Quantitative Risk Assessment**

### 4.1.2 QUANTITATIVE RISK ASSESSMENT

The task of the Quantitative Risk Assessment has a four stage procedure that EPA has developed that includes the following:

1. Hazard Identification
2. Exposure Assessment
3. Toxicity Assessment
4. Risk Characteristics

### 4.1.3 HAZARD IDENTIFICATION

**DATA COLLECTION**

**FLORIDA CHILDREN'S ENVIRONMENTAL HEALTH ALLIANCE (FCEHA)**

The lead data was collected by Janvier Gasana MD, founder of the Florida Children's Environmental Health Alliance (FCEHA). Gasana established the Florida Alliance to Eradicate Childhood Lead Poisoning (FAECLP), an outgrowth of research he conducted in both Chicago and Miami. Later, the group changed its name to Florida Children's Environmental Health Alliance (FCEHA) to reflect its newly expanded area of environmental health (http://news.fiu.edu/releases/2003/04-23_janvier_gasana.htm). Indeed, 1995 Gasana moved to Miami to accept a faculty position at Florida International University where he studied local lead-poisoning data and found that Liberty City, Little Haiti and eastern Little Havana accounted for a disproportionately high amount of the county's reported lead poisoning cases. Gasana then assembled a team of 11 graduate students who obtained test samples from residents and sites in those specific communities. The data resulted in nearly two-thirds of the study sites returning one or more samples with lead levels greatly exceeding Environmental Protection Agency (EPA) guidance standards.

A study that was conducted as a result of FCEHA's work includes "Environmental Contamination in Miami Inner City Area" (Gasana and Chamorro 2002). In conducting this study power analysis indicated that a sample size of 137 was sufficient to test their specific hypothesis. The power analysis technique allowed the date to dictate and decide, while in the process of designing an experiment how large a sample is needed to enable statistical judgments that are accurate and reliable. In addition it illustrates how

likely the statistical test will be to detect effects of a given size in a particular situation (http://www.statsoft.com/textbook/stpowan.html). A random sample size of 137 children from the household in the areas was drawn to the researchers a sufficient amount. Lead inspections were performed at 121 homes. The inspections involved the collection of representative samples from the floors, windowsills, window wells, tap water, soil, and air. The environmental data that was collected is listed Table 1, Distribution of Lead Analysis Results in Different Media.

**Table 1 Distribution of Lead Analysis Results in Different Media (Gasana and Chamorro 2002)**

| Medium | HUD/EPA Standard | Sum | Mean | SD | Median | Mode | Maximum | Minimum | Range |
|---|---|---|---|---|---|---|---|---|---|
| Air (µg/$m^3$) (n=121) | 15 | 17 | 0.14 | 0 | 0.08 | 0.06 | 1.36 | 0 | 1.36 |
| Water Plug (ppb) (n=120) | 15 | 514 | 4.25 | 15 | 1 | 1 | 150 | 1 | 149 |
| Water Flow (ppb) (n=120) | 15 | 214 | 1.77 | 3 | 1 | 1 | 34 | 1 | 33 |
| Floor Dust (µg/$ft^2$) (n=121) | 40 | 1,667 | 13.77 | 20 | 8.3 | 13 | 150 | 0.8 | 149 |
| Window Sill (µg/$ft^2$) (n=121) | 250 | 11,709 | 96.77 | 417 | 11 | 17 | 3,500 | 0.69 | 3,499 |
| Window well (µg/$ft^2$)(n=118) | 400 | 127,583 | 1054.4 | 7,248 | 17 | 120 | 78,000 | 4 | 7,796 |
| Soil (ppm) (n=121) | 400 | 33,283 | 275 | 315 | 153 | 25 | 1,612 | 25 | 1,587 |

Because of privacy issues the Development of Risk Based Treatability and Engineering Measures for Reducing Exposure to Lead Contaminated Media in the Miami Inner City, Florida, the only data available for use is environmental data. There will not be any personal data (residential addresses, demographic information, health or medical information) used for this study.

**SOIL SAMPLE**

The focus of the study is on soil and every task and procedure throughout the study will have soil as the primary media unless otherwise noted. The soils were taken from a five part composite sample from bare unvegetated areas located near the dwelling of the children. Samples were collecting by coring or scooping the top half inch of soil

from five independent areas and combining them into a composite sample. These samples were then analyzed to the 18th edition of Standards Methods via atomic Absorption Spectrometer or Inductively Coupled Plasma (Gasana and Chamorro 2002).

The main goal of first the two techniques is to form a basis to decide, while in the process of designing an experiment, (a) how large a sample is needed to enable statistical judgments that are accurate and reliable and (b) how likely your statistical test will be to detect effects of a given size in a particular situation. The third technique is useful in implementing objectives *a* and *b* and in evaluating the size of experimental effects in practice (http://www.statsoft.com/textbook/stpowan.html).

### 4.1.4 EXPOSURE ASSESSMENT

#### INITIAL SCREENING

Industrial source pollution, leaded gasoline, the weathering of lead based paints are all variables that influences soil's contamination on lead and possible causes in this collection. The five part composite of soil was collected from 121 sites.

#### RESIDENTIAL SCENARIO

The residential population pathway has been determined to be scenario in regards to the characterization of which this population resides. The past, current, and future exposure scenario of this population has also been determined to include the residential setting.

#### ENVIRONMENTAL PATHWAYS AND EXPOSURE POINT CONCENTRATION

According to the Environmental Protection Agency (EPA), the major contributor from a hazardous waste standpoint and soil is the dust ingestion pathway. Where surface dust lead levels are elevated, the hand-to-mouth is the primary culprit. From this

perspective the focal point should be on environments impact by wastes from lead smelting and mining. Because there are not any known published studies, it is assumed that the primary environmental pathways to the lead contaminated soils are from the roads and highways that drift into the air and settle in the yards from observations.

**TOXICITY SCORE**

As mentioned previously, various institutional controls from different federal agency's standards and regulations for the various contaminated lead in media, other controls include proposed maximum concentration limits (MCLs), proposed maximum concentration limit guidelines (MCLGs), and Water Quality Criteria for Fish and Drinking Water are also available for use.

**NONCARCINOGENS**

$$TS = C_{MAX}/MCL \qquad (1)$$

**Equation 1 Toxicity Score**

where: TS = Toxicity Score

$C_{MAX}$ = Maximum Concentration Level

MCL = Maximum Contaminant Level ($\frac{mg}{L}$)

**Table 2 Lead Concentrations**

| Concentration (mg/L) Minimum | Concentration (mg/L) Mean | Concentration (mg/L) Maximum |
|---|---|---|
| 25 | 275 | 1612 |

Chronic Daily Inhalation Intake

$$I = \frac{C * CR * EF * ED}{BW * AT} \qquad (2)$$

**Equation 2 Chronic Daily Inhalation Intake**

where:

I = Intake (mg/kg of body weight* day)

C = Concentration at exposure point (mg/L in water or mg/$m^3$ in air)

CR = Contact rate (L/day or $m^3$/day)

EF = Exposed frequency (days/year)

ED = Exposed Duration (Years); For residential exposures, a default value of ED = 30 years is typically used.

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

Average Daily Intake from Dermal Contact with Soil

$$I_N = \frac{C * A * DA * Abs * SM * ED}{BW * AT} \qquad (3)$$

**Equation 3 Average Daily Intake From Dermal Contact With Soil**

where:

$I_N$ = Intake

A = Skin Exposed = 20 % ($cm^2$)

DA = Dust Adherence = $0.51 \frac{mg}{cm2}$

Abs = Skin Absorption Rate 6%

SM = Effect of Soil Matrix = 15% (because of the soil matrix, only 15% of contamination is actually available for contact)

EF = Two Exposure events per day; 156 exposure days per year

ED = 1 Year

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

### 4.1.5 TOXICITY ASSESSMENT

EPA's Integrated Risk Information System (IRIS)

According to the Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS), the Oral Rfd Assessment, Inhalation RfC Assessment, and Oral Slope Factor were not available for lead. Specifically, EPA published the following statement:

> "EPA considered providing an RfD for inorganic lead in 1985, and concluded that it was inappropriate to develop an RfD, as documented online in the following statement in 1988:
> A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead."

(http://www.epa.gov/iris/subst/0277.htm).

In addition to the above statement, the EPA's Integrated Risk Information System (IRIS) http://www.epa.gov/iris/, system provided confirmation through email and conversation that confirmed that there are not any known available data for there reference cases (Email, 4/10/07).

As a result, to run a quantitative risk assessment a suitable value was needed to complete the task. The substitute value determined to best guide the quantitative risk assessment was the maximum concentration limits (MCLs).

**Table 3 Drinking Water Contaminants (http://www.epa.gov/safewater/contaminants/index.html)**

| Contaminant (Inorganic) | Maximum Contaminant Level Goal (MCLG) mg/L | Maximum Contaminant Level (MCLs) or Treatment Technique (TT) mg/L | Potential Health Effect from Ingestion of Water | Sources if Contaminant in Drinking Water |
|---|---|---|---|---|
| Antimony | 0.006 | 0.006 | Increase in blood Cholesterol; decrease in blood sugar | Discharge from petroleum refineries; fire retardants; ceramics; electronics; solder |
| Arsenic | 0 | .010 (as of 01/23/06) | Skin damage or problems with circulatory systems, and may have increased risk of getting cancer | Erosion of natural deposits; runoff from orchards, runoff from class & electronic production waste |
| Asbestos (fiber > 10 micrometers) | 7 million fibers per liter | 7 MFL | Increased risk of developing benign intestinal polyps | Decay of asbestos cement in water mains; erosion of natural deposits |
| Barium | 2 | 2 | Increase in blood pressure | Discharge of drilling wastes; discharge from metal refineries; erosion of natural deposits |
| Beryllium | 0.004 | 0.004 | Intestinal lesions | Discharge from metal refineries and coal burning factories; discharge from electrical, aerospace, and defense industries |
| Cadmium | 0.005 | 0.005 | Kidney damage | Corrosion of galvanized pipes, erosion of natural deposits; discharge from metal refiners; runoff from waste batteries and paints |
| Chromium (total) | 0.1 | 0.1 | Allergic dermatitis | Discharge from Steel and pulp mills, erosion of natural deposits |
| Copper | 1.3 | TT Action Level 1.3 | **Short Term Exposure:** Gastrointestinal distress<br>**Long Term Exposure:** Liver of Kidney Damage | Corrosion of household plumbing systems; erosion of natural deposits |

| Contaminant (Inorganic) | Maximum Contaminant Level Goal (MCLG) mg/L | Maximum Contaminant Level (MCLs) or Treatment Technique (TT) mg/L | Potential Health Effect from Ingestion of Water | Sources if Contaminant in Drinking Water |
|---|---|---|---|---|
| Cyanide (as free cyanide) | 0.2 | 0.2 | Nerve damage of thyroid problems | Discharge from steel or metal factories; discharge from plastics and fertilizer factories |
| Fluoride | 4 | 4 | Bone Disease (pain and tenderness of the bones); Children may get mottle teeth | Water Additive which promotes strong teeth. Erosion of natural deposits; discharge from fertilizer and aluminum factories |
| Lead | 0 | TT Action Level .0015 | **Infants and Children:** Delays in physical or mental development; children could show slight deficits in attention span and learning disabilities<br>**Adults:** Kidney problems; high blood pressure | Corrosion of household plumbing system; erosion of natural deposits |
| Mercury (inorganic) | 0.002 | 0.002 | Kidney damage | Erosion of natural deposits; discharge from refineries and factories runoff from landfills and crop lands |
| Nitrate (measured as Nitrogen) | 10 | 10 | Infants below the age six months who drink water contaminant nitrates could become seriously ill and if untreated may die. Symptoms include shortness of breath and blue baby syndrome | Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits |
| Nitrate (measured as Nitrogen) | 1 | 1 | Infants below the age six months who drink water contaminant nitrates could become seriously ill and if untreated may die. Symptoms include shortness of breath and blue baby syndrome | Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits |
| Selenium | 0.05 | 0.05 | Hair or fingernails loss; numbness in fingers or toes; circulatory problems | Discharge from refineries; erosion of natural deposits; discharge from mines |
| Thallium | 0.0005 | 0.002 | Hair loss; changes in blood; kidney; intestine; or liver problems | Leaching from ore processing sites; discharge from electronics glass, and drug factories |

### 4.1.6 RISK CHARACTERIZATION

### HAZARD INDEX

$$HI = \frac{In}{RfC} \qquad (4)$$

**Equation 4 Hazard Index**

where:

HI = Hazard Index (dimensionless)

$I_N$ = Chronic daily intake of NonCarcinogen $\frac{mg}{kg * day}$

RfC = Reference Concentration $\frac{mg}{kg * day}$

MCL = Maximum Contaminant Level $\frac{mg}{L}$ or $\frac{mg}{m3}$

## 4.2 METHODOLOGY

**OBJECTIVE 2:** *Develop a scientific approach for interpreting public health concerns with environmental engineering methods for remediation in contained media involving detailed techniques, procedures, and recommendation for treatability for the local community being studied.*

### DATA COLLECTION

### FLORIDA CHILDREN'S ENVIRONMENTAL HEALTH ALLIANCE (FCEHA)

A study that was conducted as a result of FCEHA's work includes "Environmental Contamination in Miami Inner City Area" (Gasana and Chamorro 2002). Lead inspections were performed at 121 homes. The inspections involved the collection

of representative samples from the floors, windowsills, window wells, tap water, soil, and air. The environmental data that was collected is listed in Table 1, Distribution of Lead Analysis Results in Different Media.

Figure 2, the Methodological Process of the Environmental Engineering Controls and Institutional Control Measures gives progressive instructions of the process flow of objective 2.

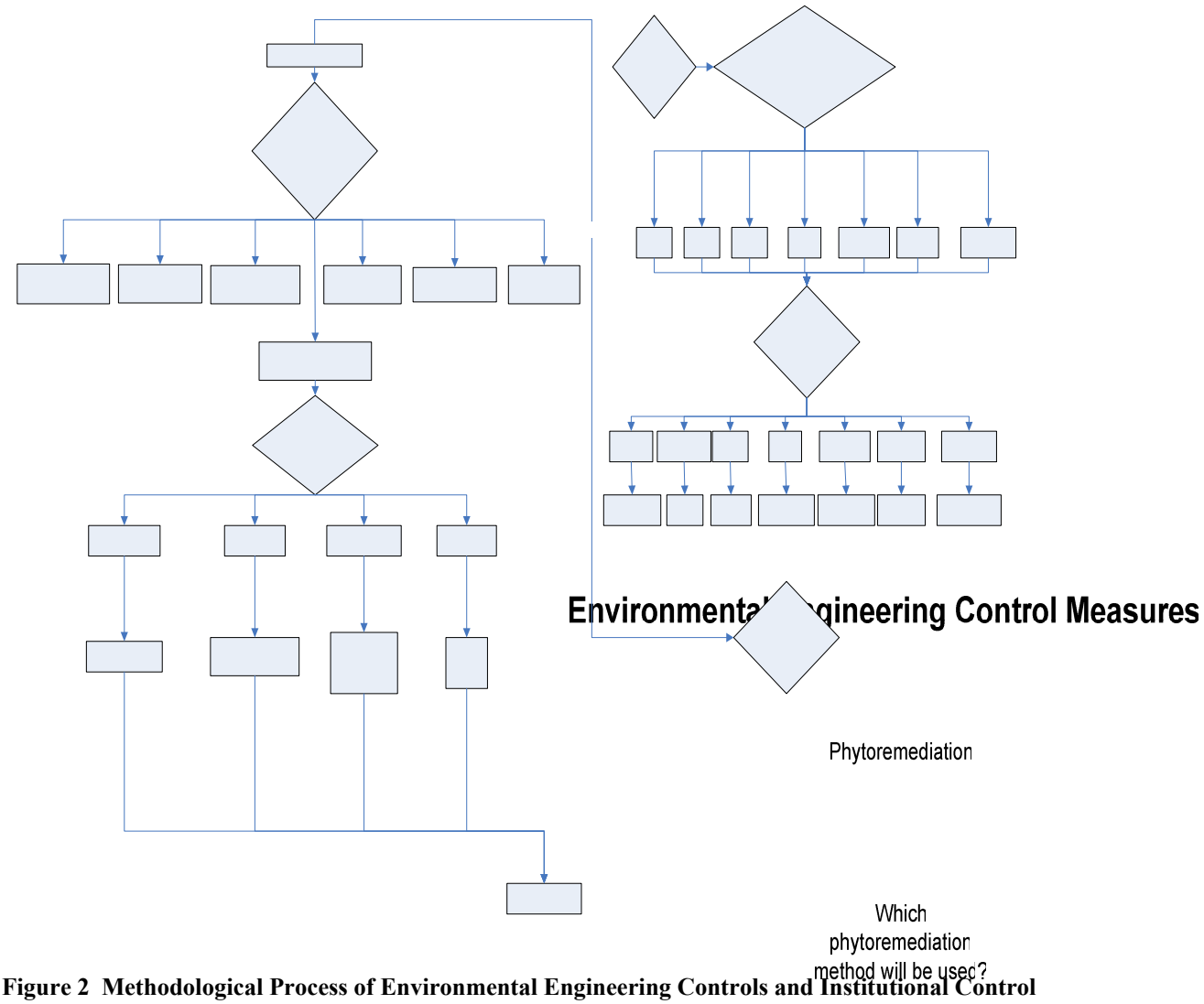



**Figure 2 Methodological Process of Environmental Engineering Controls and Institutional Control Measure**

### 4.2.1 INSTITUTIONAL CONTROLS MEASURES

### STANDARDS

Various federal agencies have provided advisory standards and or enforceable regulation that set the lead levels in different media. Table 4, Summary of Standard and Regulations provides various institutional control measures from those agencies.

**Table 4 Summary of Standards and Regulations for Lead (Moeller, D 1992)**

**Summary of Standards and Regulations for Lead**

| Agency | Media | Level | Comments | Source |
|---|---|---|---|---|
| Centers For Disease Control and Prevention | Blood | 10 µg/dL | Advisory: level pf concern for children | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Blood | 40 µg/dL | Regulation: cause for written notification and medical exam | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Blood | 50 µg/dL | Regulation: cause for medical removal from exposure | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Air (workplace) | 50µg/$m^3$ | Regulation: permissible exposure limit (8-hour average) (general industry) | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Air (workplace) | 30 µg/$m^3$ | Regulation: Action Level | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Air (ambient) | 1.5 µg/$m^3$ | Regulation: National Ambient Air Quality Standard; 3 month Average | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Soil (Residential) | 400 mg/kg | Soil Screening guidance | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Water Drinking | 15 µg/L | Action level for public supplies | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Water Drinking | 0 µg/L | Non enforceable goal; maximum contaminant level goal | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Consumer Product Safety Commission (CPSC) | Paint | 600 ppm or 0.06% | Regulation; by dry weight | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |

### 4.2.2 INSTITUTIONAL CONTROLS MEASURES EQUIPMENT

Within the area of Institutional Controls, measures are in place to assess the current use against the prescribed measure of that specified data.

**AIR**

Composite air samples were collected in 121 homes. The collection process took place in the bedroom, living room, and dinning area. The sampling media was placed at a height consistent with the children's primary breathing area. Once the air was collected, it was analyzed in the laboratories flowing EPA methods 7420 and 7421(Gasana and Chamorro 2002). Figure 3, Collected Air Samples presents the data.

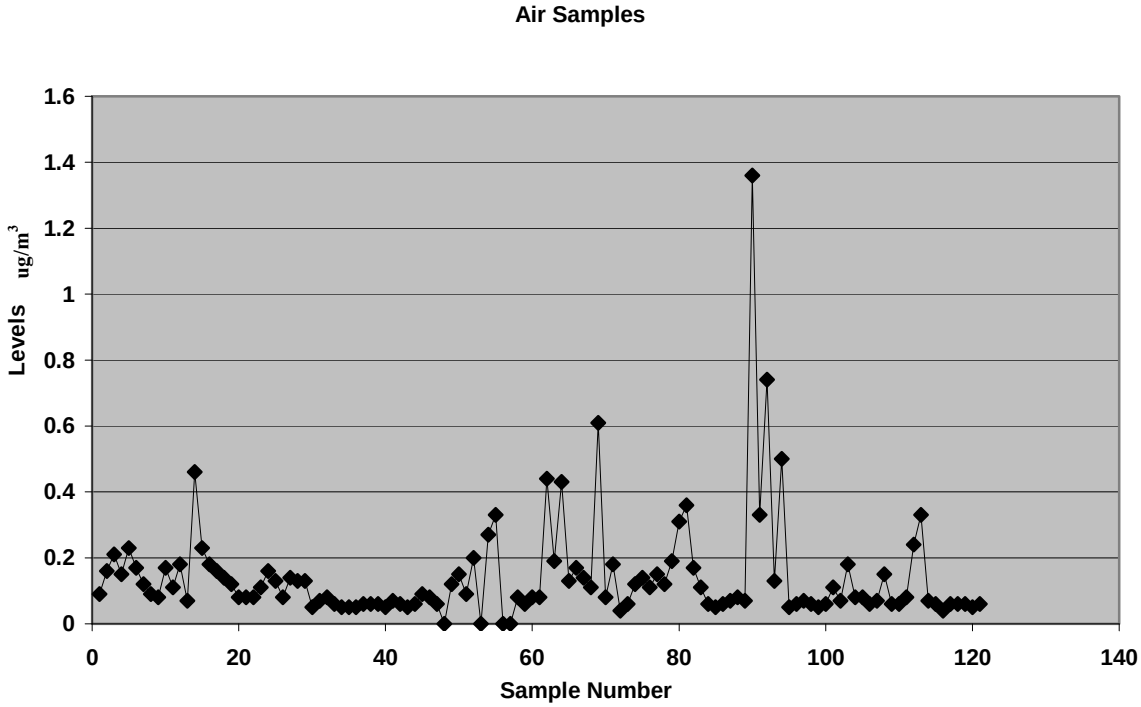



**Figure 3 Collected Air Samples (Gasana and Chamorro 2002)**

## FLOOR DUST

Surface dust testing for lead was utilized using the wipe sampling techniques. Three samples were taken from each dwelling using commercially available wipes moistened with a non alcoholic wetting agent. Once the samples were collected, it was analyzed in the laboratories following EPA methods 3050 and 7082(Gasana and Chamorro 2002). Figure 4, Collected Floor Dust Samples presents the data.

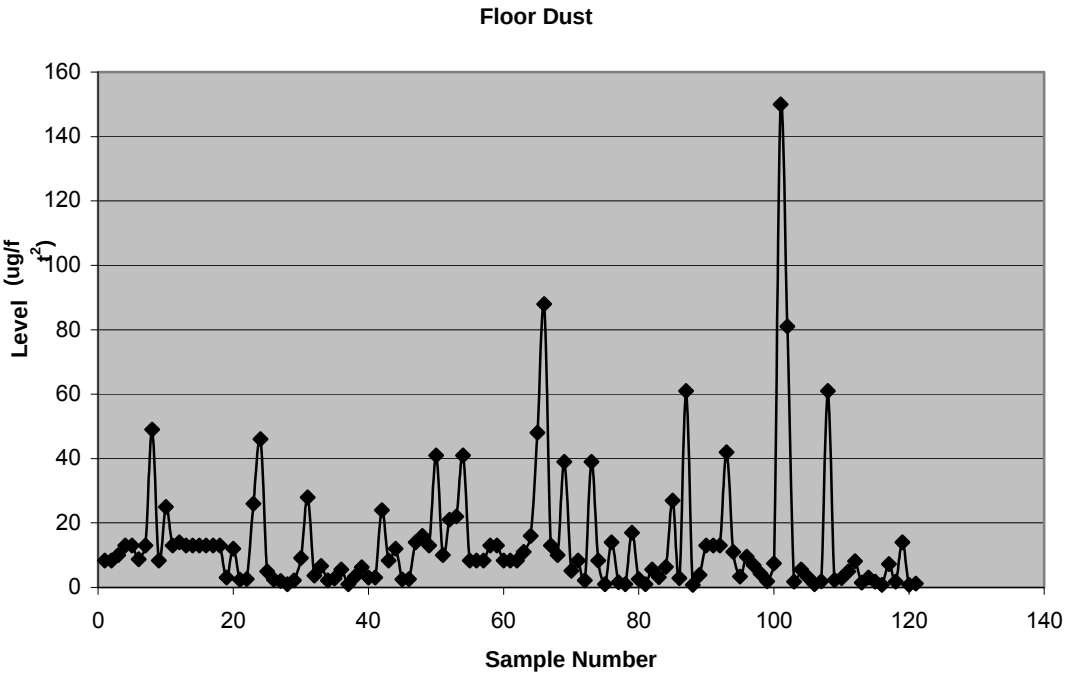



**Figure 4 Collect Floor Dust Samples (Gasana and Chamorro 2002)**

## LEAD BASED PAINT DETERMINATION BY XRF

The onsite inspection of lead based paint consisted of testing a maximum of three rooms. These rooms were selected based on motion and time studies based on the parents' affirmation. The lead based analysis was conducted by direct reading XRF using

a Niton XL spectrum analyzer (Gasana and Chamorro 2002). Figure 5, Collected Lead Based Paint Determination by XRF presents the data.

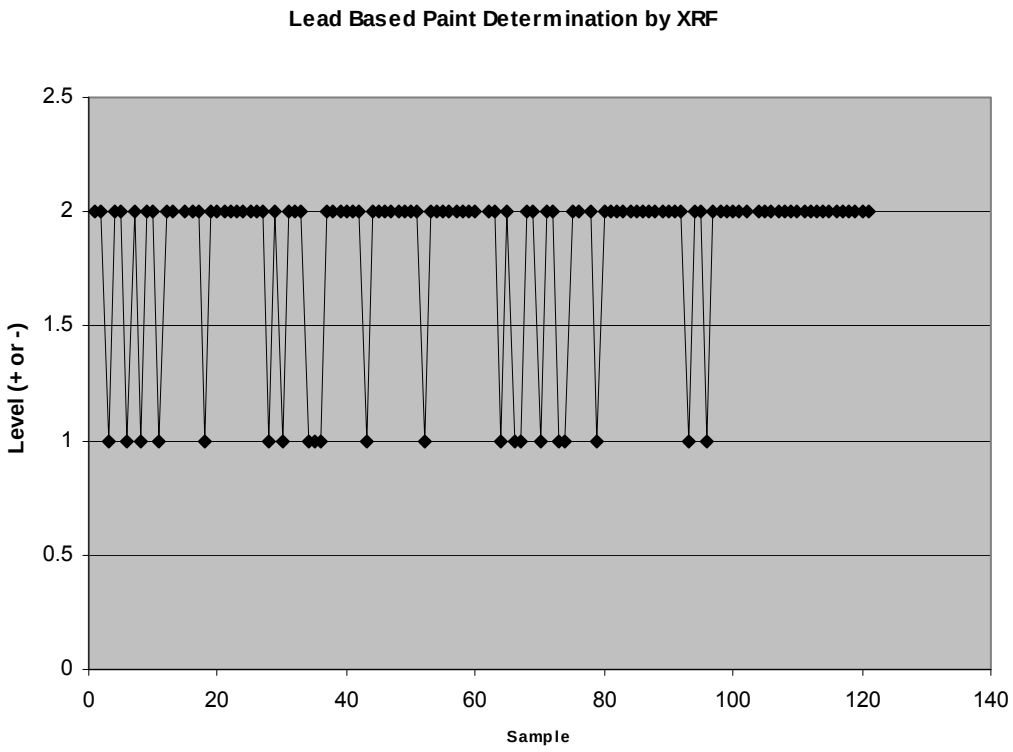



**Figure 5 Collected Lead Based Paint Determination by XRF Samples (Gasana and Chamorro 2002)**

## POTABLE WATER

There were two water samples collected from the faucet used to supply the child with potable water. The plug flow, which was the first draw, was followed by a water flow sample, which took place after 30 seconds of continuous water flow. It was ideal to take the water sample first thing in the morning. However, sample participants preferred the evening. The samples were prepared and analyzed according to the 18$^{th}$ edition of Standards Method via Atomic Adsorption Spectrophotometer or Inductively Couple

Plasma (Gasana and Chamorro 2002). Figure 6, Collected Potable Water presents the data.

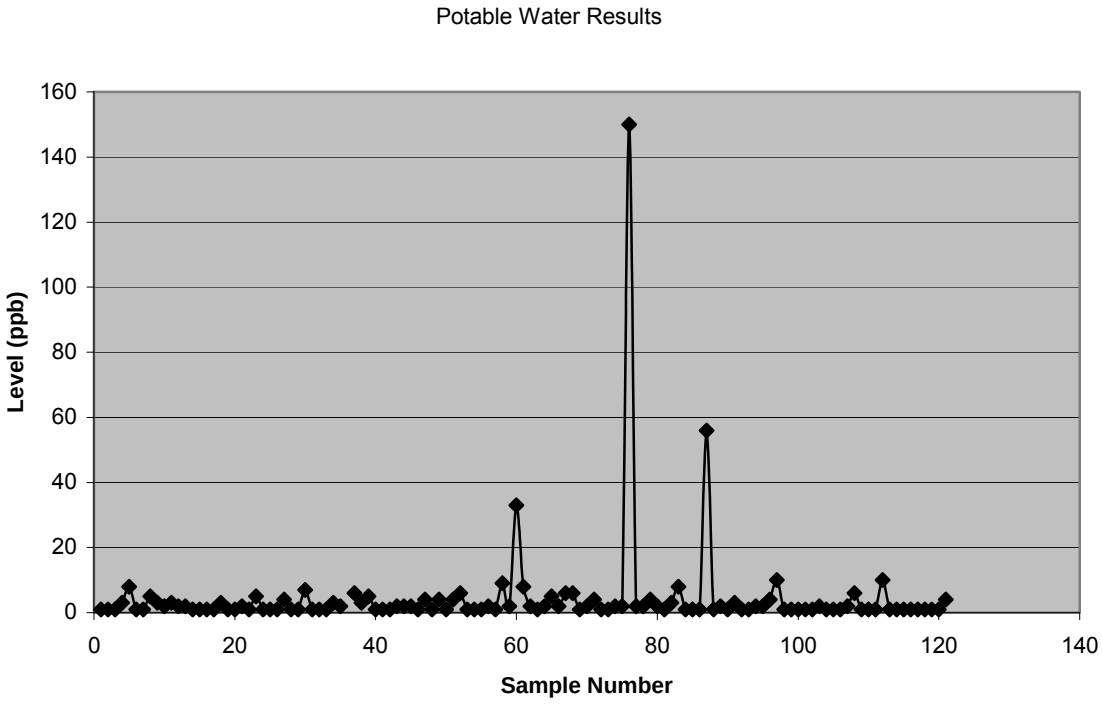



**Figure 6 Collected Potable Water Samples (Gasana and Chamorro 2002)**

## SOIL

The soils were collected from a five part composite sample from bare unvegetated areas located near the dwelling of the children. Samples were collecting by coring or scooping the top half inch of soil from five independent areas and combining them into a composite sample. These samples were then analyzed to the 18$^{th}$ edition of Standards Methods via atomic Absorption Spectrometer or Inductively Coupled Plasma (Gasana and Chamorro 2002). Figure 7, Collected Soil presents the data.

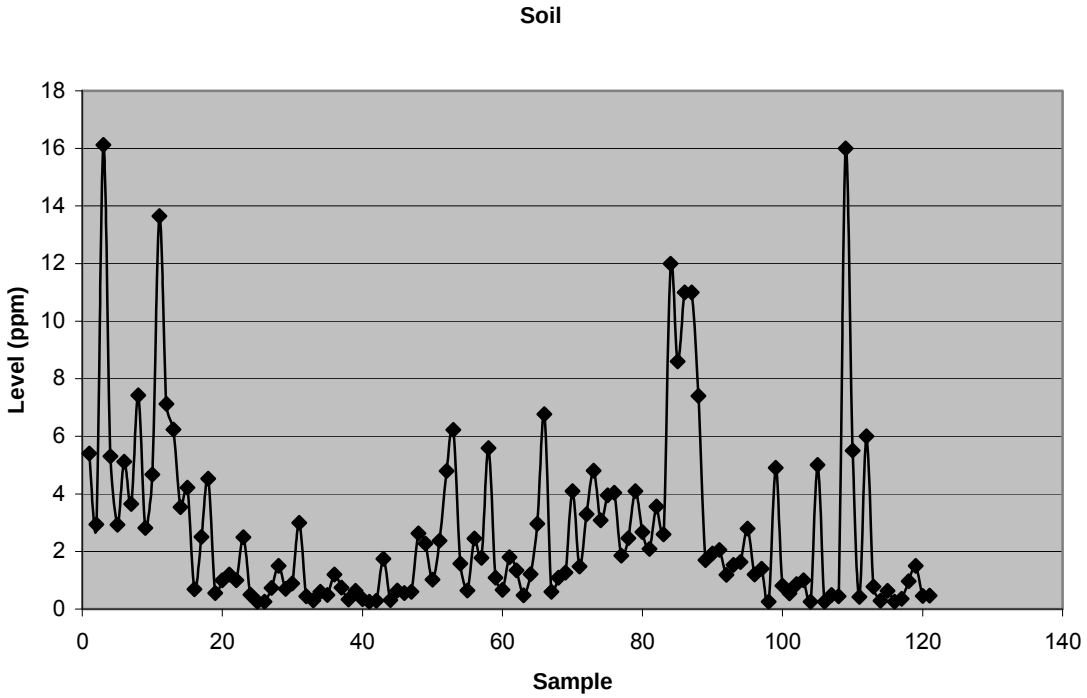



**Figure 7 Collected Soil Samples (Gasana and Chamorro 2002)**

## WINDOW SILL DUST

Surface dust testing for lead was utilized using the wipe sampling techniques. Three samples were taken from each dwelling using commercially available wipes moistened with a non alcoholic wetting agent. Once the samples were collected, it was analyzed in the laboratories flowing EPA methods 3050 and 7082(Gasana and Chamorro 2002). Figure 8, Collected Window Sill Dust presents the data.

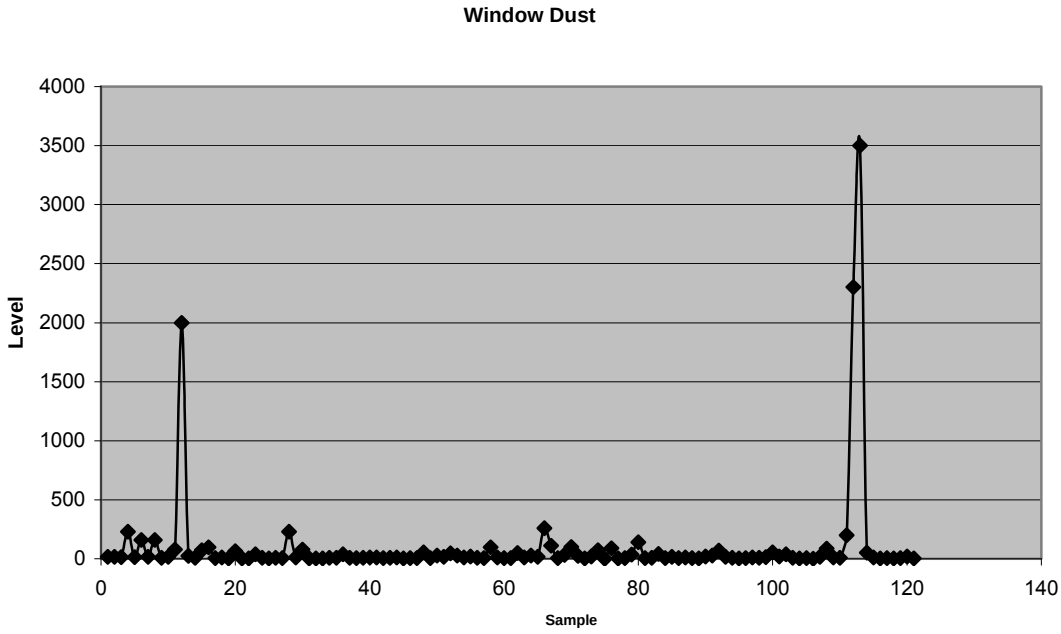



**Figure 8 Collected Window Dust Samples (Gasana and Chamorro 2002)**

## 4.3 ENVIRONMENTAL ENGINEERING CONTROL MEASURES

### PHYTOREMEDIATION

### PHYTOACCUMULATION

Based on the review of the study, the most suitable environmental engineering control is the use of phytoremediation also known as phytoaccumulation. This method is the uptake and translocation of metal contaminates from the soil into the plant matter via plant roots into the above ground portions of the plants (LaGrega et al 2001). Certain phytoextration methods of uptake have considerable amounts of metals in contrast to other plants and the ambient concentration. The uptake should be metal specific which allows the risk of impoverishing the surrounding soil. In addition to the metal specificity, a high transport of metals from the roots to the shoots should take place for this method

to be effective during remediation treatment (Suthersan 2002). The plants act as a filter or traps for the metals and remove them from contaminated soils. Once these plants have performed their function, they are harvested and incinerated with waste ash at hazardous waste landfills (LaGrega et al 2001).

**GENERAL DESIGN FACTOR PHYTOREMDIATION**

Since the design of the phytoremediation depends on the specific site and its characteristics more environmental information should be collected. In spite of this being an environmental health study and missing information, there are design considerations that could be used for all methods including the following:

1. Contaminant Levels
2. Treatability
3. Plant Selections
4. Cost

## 4.4 MONTE CARLO SIMULATION

Mathematical techniques and simulations can be utilized to assess both uncertainty and the sensitivity of final answers to individual input parameters when estimating exposures. The purpose of the Monte Carlo simulation is to run the simulations numerous times and as each run is determined, the output produces new vales of the random variables thus, producing a new risk.

### 4.4.1 MONTE CARLO METHOD

In running the Monte Carlo method, there are parameters that are fixed with one parameter varying. This analysis can then display the effect of a range of set values on that varying parameter, producing different outcomes of the simulated model. In

addition, the simulation can illustrate the need to collect any additional information that may be omitted (Loyd 2006).

This simulation procedure can facilitate the replacing of point estimates with random variables drawn from probability distribution functions when there is pertinent information missing or not defined. The model is ran repeatedly with the outputs of each run saved. The output of each run can then be used to determine expected values in addition to low and high end risks presenting the probability of incidences (Loyd 2006). This allows a set of sample results that can be displayed in a frequency output as opposed to one single risk estimate drawn from a defined sample size.

In addition, the Monte Carlo method characterizes uncertainties within the quantitative risk assessments. In conducting these quantitative risk assessments, there are innate and deficient uncertainties present in each of the four procedures. Hazard Identification is based on data in which the detection, identification, and quantification limits could introduce errors. As a result, the interpretation of the final results will already include their built in uncertainties, thus generating a more practical output. Please note that the reader must be aware that this is not a study of the Monte Carlo method. Monte Carlo method is only used to assist in one aspect of this study. Please refer to Herman (1957) or Hammersley (1964) for further study in this area.

### 4.4.2 MONTE CARLO COST

The economic data was instituted through a study from Chappell (1997). The study determined that Option 1 consisted of 10 acres of contaminated land opposed to Option 2 consisting of 1 hectare of various contaminates. The resulting costs have been

calculated based on the average lot size of 3600 $ft^2$ in Miami-Dade County Florida and is listed in Table 5, the estimated costs.

**Table 5 Estimated Cost (Chappell, J 1997)**

| Option | Contaminants | Phytoremediation Costs | Estimated Cost Using Other Technologies |
|---|---|---|---|
| 1 (Lead) | 10 acres lead contaminated land | $500,000 | $12 million |
| 2 (Various Contaminants) | 1 hectare to a 15 cm depth (various contaminants) | $2,500 to $15,000 | none listed |

### 4.4.3 SCENARIO ANALYSIS TOOL

Within the Monte Carlo tools, the Scenario Analysis tool was used to determine the economic cost and efficient and cost for remediation. The Scenario Analysis tool runs a simulation and then sorts and matches all the resulting values of a target forecast with their corresponding assumption values. This method allows for further investigation into combinations of assumptions values given for a particular result (Decisionengineering Inc 2007).

Chappell (1997), indicates that it is complicated to predict the cost of the phytoremediation because of it's innovativeness to the remediation arena and the lack of establishment through years of use as other technologies have been. Lab, pilot, and field study test have included monitoring procedures far above those expected at a site with a remediation goal. As a result, it is complicated to secure down precise costs. Nevertheless, the fundamental aspect of phytoremediation is the use of trees and or grasses, which then renders it by nature a much inexpensive and cheaper option when

compared to technologies that involve the use of large scale, energy consuming equipment.

Table 5 Estimated Cost, represents some estimates of phytoremediation's cost in relation to conventional technologies. This table characterizes ambiguous and variable estimates due to the current scarcity of cost information data (Chappell 1997). The bulk of this work is derived from poplar tree systems that are site specific. Table 6 list several fixed cost developed for this specialized area.

**Table 6 Fixed (Specific) Costs of Phytoremediation**

| **Ecolotree** | |
|---|---|
| Installation of trees at $1,450 tress per acre | $12,000 to $15,000 |
| Predesign | $15,000 |
| Design | $25,000 |
| Site Visit | $5,000 |
| Soil Cover and Amendments | $5,000 |
| Transportation to Site | $2.14 per mile |
| Operations and Maintenance | $1,500 per acre with irrigation<br>$1,000 per acre without irrigation |
| Pruning (not every year) | $500 |
| Harvest (during harvest year) | $2,500 |
| **Applied Natural Science** | |
| Treemediation program design and implementation | $50,000 |
| Monitoring Equipment | Hardware $10,000 |
| | Installation $10,000 |
| | Replacement $5,000 |
| Five Year Monitoring | Travel and Meetings $50,000 |
| | Data Collection $50,000 |
| | Annual Reports $25, 000 |
| | Sample Collection and Analysis $50,000 |

From Table 5 and 6, I derived variable costs from the fixed costs to run the simulation. The variable costs were calculated utilizing an upper and lower bound (+ or – 5% rate) on the fixed cost (Decisionengineering Inc 2007). The results of the variable costs are listed in Table 7, Variable Costs.

**Table 7 Variable Costs**

| | Variable Cost | | |
|---|---|---|---|
| **Option Various Contaminants** | | **Option Lead** | **Cost** |
| Option Various Contaminants (Fixed) | $516.25 | Option Lead (Fixed) | $4,130.00 |
| Option Various Contaminants ((Variable Cost includes labor and material at (+ and - 5%)) | $1,548.75 | Option Lead ((Variable Cost includes labor and material at (+ and - 5%)) | $12,390.00 |
| **Total Remediation Cost Various Contaminants (Fixed Cost + Variable Cost)** | $2,065.00 | Total Remediation Cost Lead (Fixed Cost + Variable Cost) | $16,520.00 |

Before running the Monte Carlo Scenario Analysis, the assumptions used are listed below in Table 8.

**Table 8 Assumptions**

| Assumptions | | | | |
|---|---|---|---|---|
| **Fixed Costs** | **Cost** | **Variables Costs** | **Cost** | **Total Estimated Remediation Cost** |
| 1 Option Lead (Fixed) | $4,130.00 | 1 Option Lead (Variable) | $12,390.00 | $16,520.00 |
| 2 Option Various Contaminants (Fixed) | $516.25 | 2 Option Various Contaminants (Variable) | $1,548.75 | $2,065.00 |

The Monte Carlo Scenario Analysis system, including the inputs and outputs, is displayed in the schematic below in Figure 9, the Initial Simulation Data.

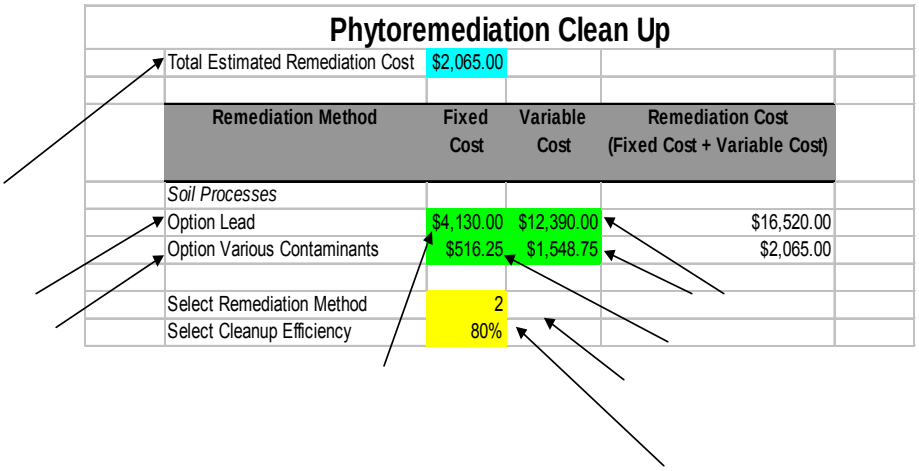



**Figure 9 Initial Simulation Data**

## 5. RESULTS AND DISCUSSION

### TOXICITY SCORE

Table 9, Lead Concentrations, is used in the calculation to determine the various Toxicity Score as depicted in Table 10.

**Table 9 Lead Concentrations**

| Concentration (mg/L) $_{Minimum}$ | Concentration (mg/L) $_{Mean}$ | Concentration (mg/L) $_{Maximum}$ |
|---|---|---|
| 25 | 275 | 1612 |

Figure 10 Toxicity Score, depicts the various concentration levels (minimum, mean, and maximum) when utilizing the lead MCL of .0015 mg/L as a score. Figure 10 further depicts that between the minimum and mean concentration levels there is only a difference of 166,666.6. However, the most significant jump is from the mean score to the maximum score of 891,333.4. This clarifies that as the concentration level increase so does the specific Toxicity Score.

**Table 10 Toxicity Score**

| Toxicity Score | |
|---|---|
| Concentration mg/L | Result |
| 25.0 | 16,666.7 |
| 275.0 | 183,333.3 |
| 1,612.0 | 1,074,666.7 |

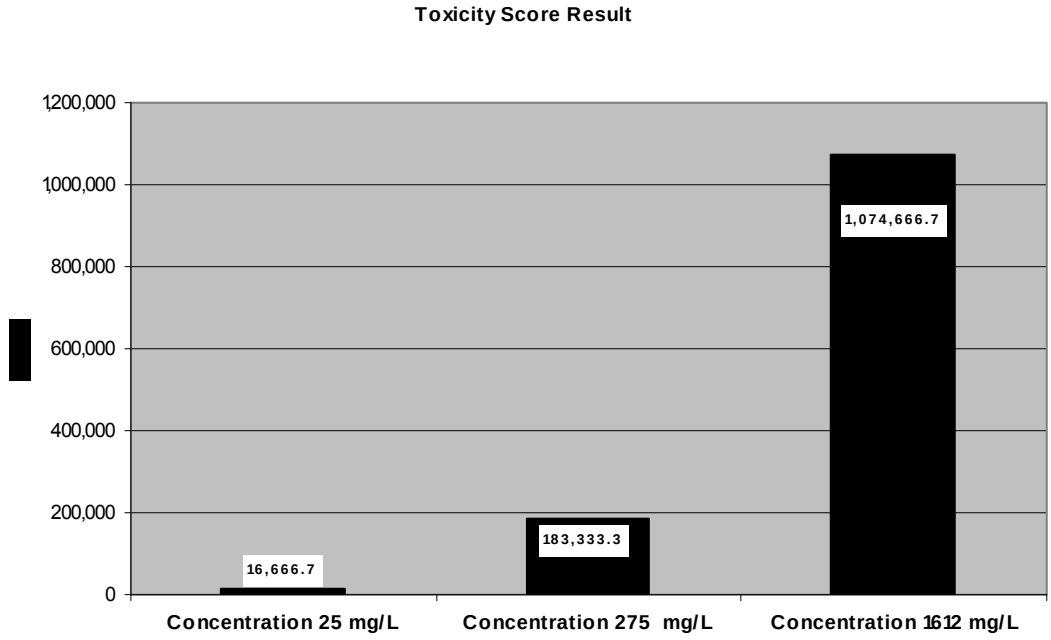



**Figure 10 Toxicity Score**

## CHRONIC DAILY INHALATION INTAKE

Table 11 and Figure 11 both present The Chronic Daily Inhalation Intake that fall below the Administered Dose of the Risk Assessment. The table sorts the values of the results as it relates to the separate age categories (Child 2-6, Child 6-12, and Adult). Various parameters such as body weight, absorption rate, soil ingested and other parameters were used to determine the results of the different intake levels. It is observed that at the different concentration levels, the data indicates that the younger the study groups, the higher the concentration levels. There are variations, but as mentioned previously, the most susceptible groups are children within the age group of 2-6, followed by child 6-12. At the Minimum Level of 25 mg/L, there is only a difference of 4.2 $\frac{mg}{kg * day}$ between the children's groups. At the Mean level of 275 mg/L there is only

a difference of 46.9 $\frac{mg}{kg * day}$ children's groups. At the Maximum Level of 1612 mg/L there is only a difference of 332.3 $\frac{mg}{kg * day}$ children's groups.

$$\mathbf{C_{MIN}} = 25\frac{mg}{L}, \mathbf{C_{MEAN}} = 275\frac{mg}{L}, \mathbf{C_{MAX}} = 1612\frac{mg}{L}$$

An example involving the children's group, age 2-6 at the minimum concentration is listed below.

**Child 2-6**

$$I = \frac{(25\frac{mg}{m3}) * (6\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(16kg) * (365 days)}$$

$$I = 281.25\frac{mg}{kg * days}$$

**Table 11 Chronic Daily Inhalation Intake**

| Inhalation Intake (mg/kg*days) | | | |
|---|---|---|---|
| **Age** | **$C_{MIN}$ 25 (mg/L)** | **$C_{MEAN}$ 275 (mg/L)** | **$C_{MAX}$ 1612 (mg/L)** |
| **Child (2-6)** | 281.3 | 3,093.8 | 18,135.0 |
| **Child (6-12)** | 285.5 | 3,140.7 | 18,467.3 |
| **Adult** | 213.4 | 2,347.7 | 13,804.6 |

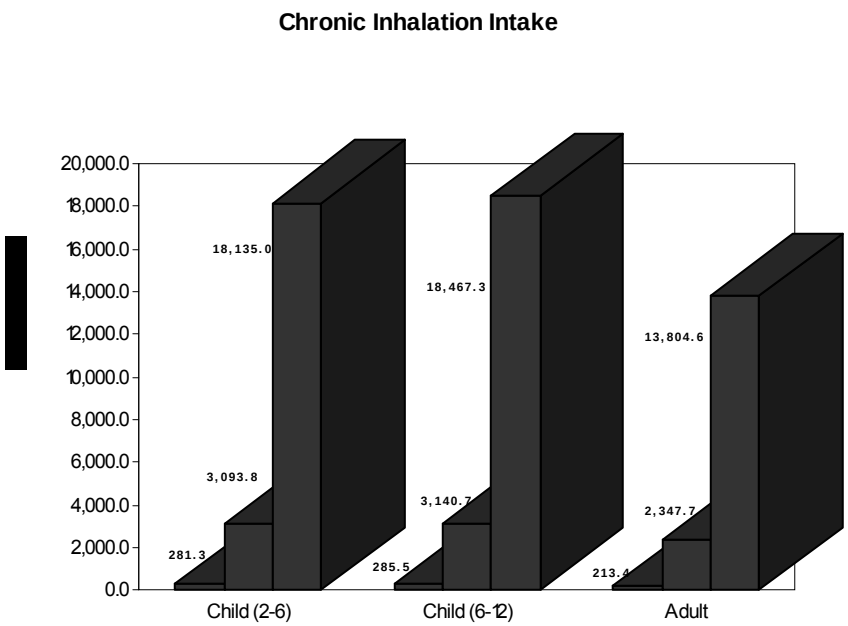



**Figure 11 Chronic Inhalation Intake**

## AVERAGE DAILY INTAKE FROM DERMAL CONTACT WITH SOIL

Table 12 and Figure 12 present The Daily Intake (Average) from Dermal Contact. Table 11, sorts out the values of the results as it related to the separate age categories (Child 2-6, Child 6-12, and Adult) and the minimum concentration of 275 mg/L. Various parameters such as skin exposed, dust adherence, skin absorption rate, and the effect of soil matrix are some parameters that were used to determine the results of the various daily dermal intake levels. It is observed that at the different concentration levels, the data varies from the previous observations and indicates that all of the category groups' levels are different by only a few data points. As in the previous observations, the most susceptible group are children in the age category of 2-6 at .000047 ($mg/m^3/kg*days$), followed by child 6-12 .000039 ($mg/m^3/kg*days$), and Adult .000028 ($mg/m^3/kg*days$).

These amounts differ of only .000008 (mg/$m^3$/kg*days), and .000011 (mg/$m^3$/kg*days) respectively.

$$\mathbf{C_{MEAN}} = 275\frac{mg}{L}$$

An example involving the children's group, age 2-6 at the prescribed concentration is listed below.

**Child (Age 2-6)**

A= (.20) * (6980 $cm^2$) = 1396 $cm^2$

$$I_N = \frac{275(\frac{mg}{m3}) * (1396cm2) * (.51\frac{mg}{cm2}) * (.06) * (.15\frac{2\,\mathrm{exp}\,osure}{day}\frac{events}{}) * (\frac{156days}{year}) *}{(16kg) * (365days)} 10^{-6}$$

$$\frac{kgsoil}{mgsoil}$$

$$I_N = 4.707 * 10^{-5}\ \frac{mg}{kg * day}$$

$$I_N = 4.7 * 10^{-5}\ \frac{mg}{kg * day}$$

**Table 12 Daily Intake from Dermal Contact**

| Daily Intake (Average) from Dermal Contact with Soil (mg)/(kg*days) | |
|---|---|
| **Child (2-6)** | $4.7*10^{-5}$ |
| **Child (6-12)** | $3.9*10^{*-5}$ |
| **Adult** | $2.8*10^{-5}$ |

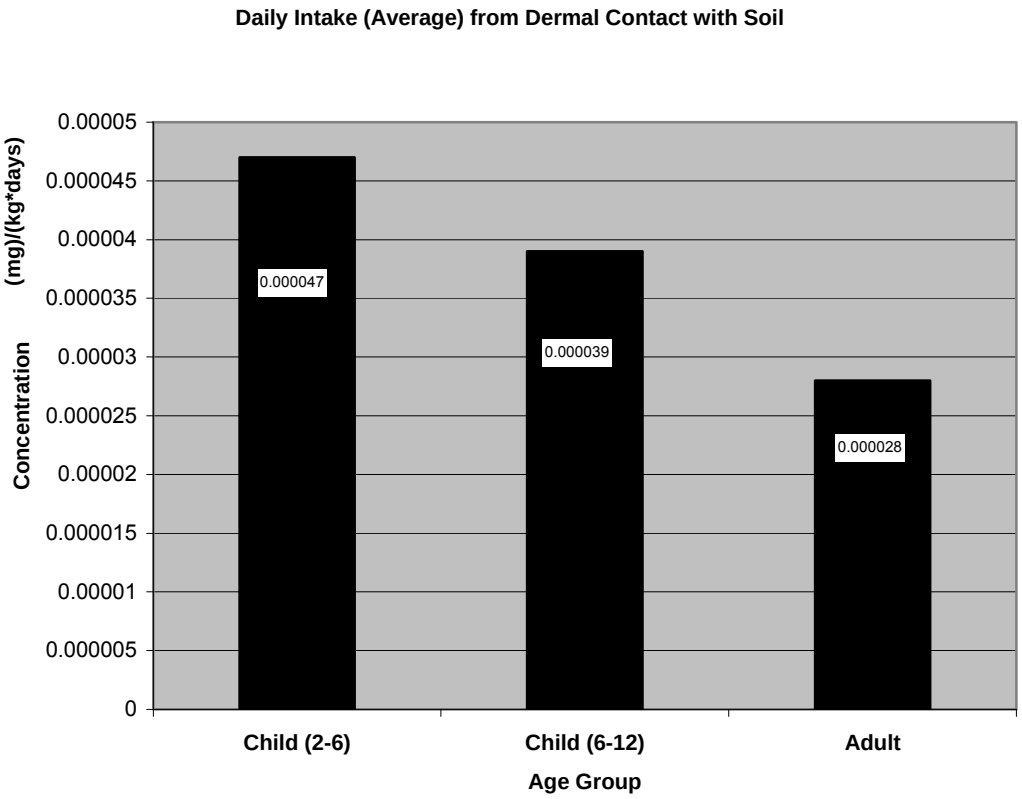



**Figure 12 Daily Intake from (Dermal) Contact with Soil**

## TOXICITY ASSESSMENT

EPA's Integrated Risk Information System (IRIS)

According to the Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS), the Oral Rfd Assessment, Inhalation RfC Assessment, and Oral Slope Factor were not available for lead. Specifically, EPA published the following statement:

> "EPA considered providing an RfD for inorganic lead in 1985, and concluded that it was inappropriate to develop an RfD, as documented online in the following statement in 1988:
> A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment

(OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead."
(http://www.epa.gov/iris/subst/0277.htm).

In addition to the above statement, the EPA's Integrated Risk Information System (IRIS) http://www.epa.gov/iris/, system provided confirmation through email and conversation that confirmed that there is know known available data for there reference data (Email, 4/10/07).

As a result, to run a quantitative risk assessment a suitable value was needed to complete the task. The substitute value determined to best guide the quantitative risk assessment was the maximum concentration limits (MCLs). Example for lead data is available in Table 13, for the Drinking Water Contaminants.

**Table 13 Lead Drinking Water Contaminants**

**(http://www.epa.gov/safewater/contaminants/index.html)**

| Contaminant (Inorganic) | Maximum Contaminant Level Goal (MCLG) mg/L | Maximum Contaminant Level (MCLs) or Treatment Technique (TT) mg/L | Potential Health Effect from Ingestion of Water | Sources if Contaminant in Drinking Water |
|---|---|---|---|---|
| Lead | 0 | TT Action Level .0015 | **Infants and Children:** Delays in physical or mental development; children could show slight deficits in attention span and learning disabilities<br>**Adults:** Kidney problems; high blood pressure | Corrosion of household plumbing system; erosion of natural deposits |

## RISK CHARACTERIZATION

## HAZARD INDEX

Table 14 and Figure 13 present The Hazard Index for each study group. The table sorts out the values of the results as it relates to the separate age categories (Child 2-6, Child 6-12, and Adult) with the maximum contaminant level .0015 mg/L. The hazard index in the ratio of the dermal intake to the maximum contaminant level (MCL) of .0015 mg/L. The pattern continues with the most susceptible group is the child 2-6 age at .03130 (L/kg*days), child age 6-12 .0260 (L/kg*days), and Adult .01870 (L/kg*days).

**Table 14 Hazard Index**

| Hazard Index | |
|---|---|
| **Child (2-6)** | 0.0313 |
| **Child (6-12)** | 0.026 |
| **Adult** | 0.0187 |

Based on the criteria of the hazard index being less than the prescribed value of 1, then this hazard is acceptable. However, when conducting risk assessments there are usually multiple chemicals and index are calculated for each chemical of concern and for each pathway and computed to either be greater than or less than 1. In this case, the values were less than the prescribed amount for each study group, but the results are not conclusive. In this case, the index will be reported with the value that was calculated.

An example involving the children's group, age 2-6 for the hazard index, which is dimensionless, is listed below.

**Child (Age 2-6)**

$$HI = \frac{.000047}{.0015} \text{ Unit less}$$

HI = .03133

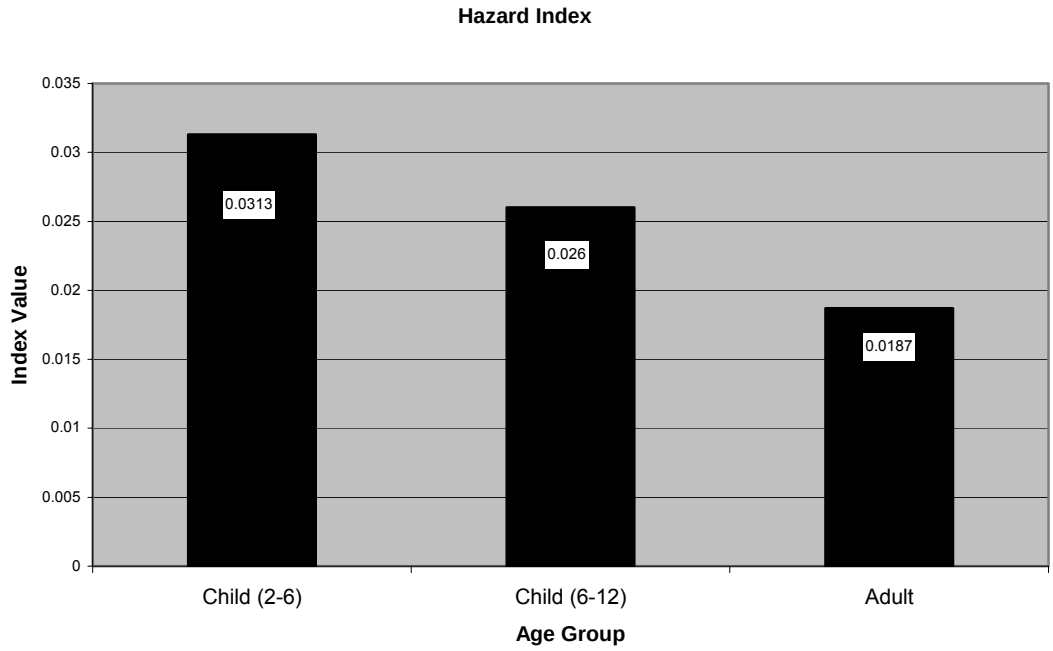



**Figure 13 Hazard Index**

## CANCER RISKS ON A POPULATION

## POPULATIONS ON 100, 000

**Table 15 Individual Cancer Risk**

| | Individual Cancer Risk | | |
|---|---|---|---|
| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
| **Child (2-6)** | 0.0264 | 0.2900 | 1.7002 |
| **Child (6-12)** | 0.0148 | 0.1625 | 0.9552 |
| **Adult** | 0.0046 | 0.0503 | 0.2958 |

**Table 16 Individual Cancer Risk Percentage**

| | Individual Cancer Risk Percentage | | |
|---|---|---|---|
| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
| **Child (2-6)** | 2.64% | 29.00% | 170.02% |
| **Child (6-12)** | 1.48% | 16.25% | 95.52% |
| **Adult** | 0.46% | 5.03% | 29.58% |

Tables 15, 16, and 17 presents the tabulated results of the different study groups based on the Individual Cancer Risk (Tables 15), Individual Cancer Risk Percentages (Tables 16), and Excess Lifetime Cancer Cases (Tables 17) all based on a population of 100,000.

**Table 17 Excess Lifetime Cancer Cases**

**Excess Lifetime Cancer Cases**

| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
|---|---|---|---|
| **Child (2-6)** | 2,640.0 | 29,004.4 | 170,015.6 |
| **Child (6-12)** | 1,476.7 | 16,245.0 | 95,520.5 |
| **Adult** | 457.3 | 5,030.8 | 29,581.3 |

Table 15 and Table 16 respectively, represents The Individual Cancer Risk with the minimum 25 mg/L, mean 275 mg/L, and maximum 1612 mg/L concentration levels reveals the various cancer risks in addition to the risk as a percentage. An example involving the children's group age 2-6 for the cancer risk, maximal cases, and the percentage per 100,000 populations at the minimum concentration.

**Child (Age 2-6)**

Population 100,000 Children

Children Weight = 16 kg

$$\text{Intake}_{\text{Child (2-6) MIN}} = 281.3 \frac{mg}{kg * day}$$

$$\text{Individual Cancer Risk}_{\text{MIN}} = (.0015 \frac{mg}{L}) \ (\frac{281.3 \frac{mg}{kg * day}}{16kg}) = .0264 \frac{mg}{L * day}$$

$$\text{Individual Cancer Risk}_{\text{MIN}} = (.0264) * 100\%$$

$$\text{Individual Cancer Risk}_{\text{MIN}} = 2.63\%$$

**Maximal Cases** $_{MIN}$

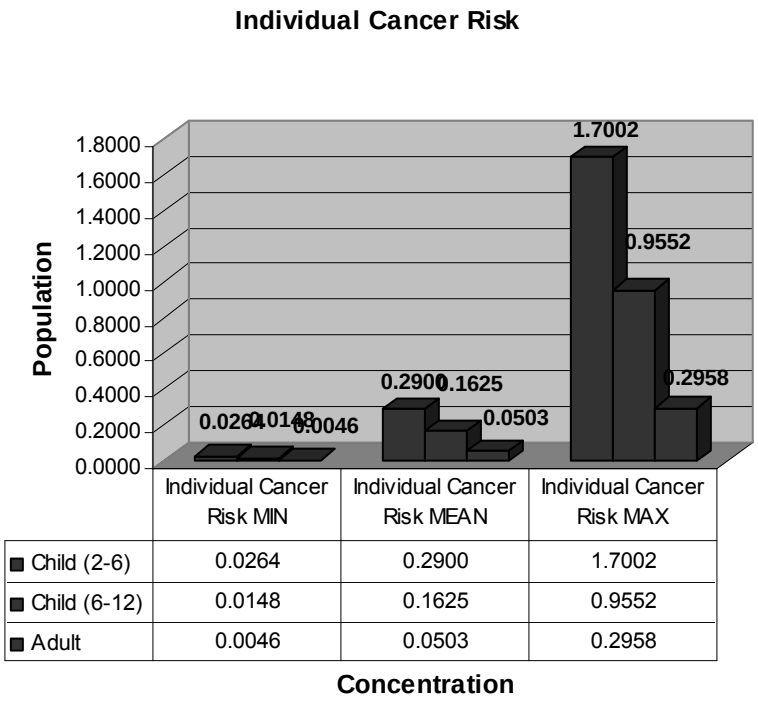
Maximum Cases $_{MIN}$ = (Risk) * (Exposed Population)

= (.0264)* (100,000)

= 2,640

**Excess Lifetime Cancer Cases** $_{MIN}$ = 2,640

Figure 14, represents the Individual Cancer Risk with the tabulated data. The table sorts out the values of the results as it related to the separate age categories (Child 2-6, Child 6-12, and Adult) with the various concentration levels.




| | Individual Cancer Risk MIN | Individual Cancer Risk MEAN | Individual Cancer Risk MAX |
|---|---|---|---|
| Child (2-6) | 0.0264 | 0.2900 | 1.7002 |
| Child (6-12) | 0.0148 | 0.1625 | 0.9552 |
| Adult | 0.0046 | 0.0503 | 0.2958 |

**Figure 14 Individual Cancer Risk**

Figure 15 presents the Individual Cancer Risk (Minimum Concentration), Figure 16 presents the Individual Cancer Risk (Mean Concentration), and Figure 17, Individual

Cancer Risk (Maximum Concentration) graphically displays the individual concentration levels in a pie chart.

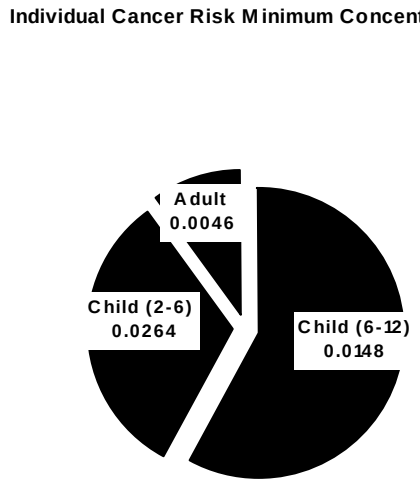



**Figure 15 Cancer Risk for Minimum Concentration**

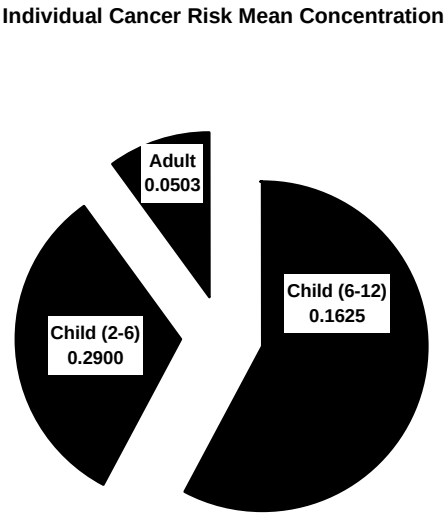



**Figure 16 Cancer Risk for Mean Concentration**

Figure 14, The Individual Cancer Risk further displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the mean concentration with a difference of .1275 between the two youngest groups. It is also observed that the youngest group (Child 2-6) is the most susceptible to cancer at the maximum concentration with a difference of .745 between the two youngest groups.

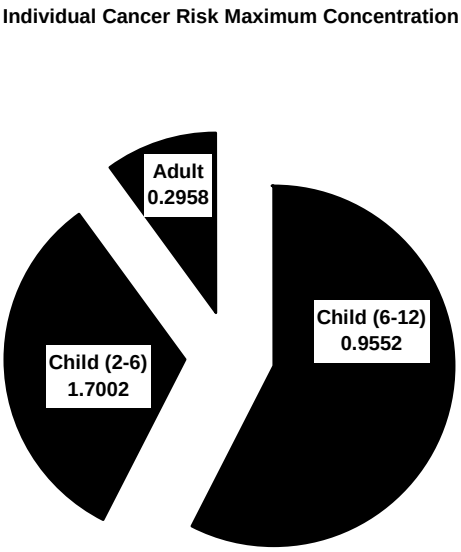



**Figure 17 Cancer Risk for Maximum Concentration**

Figure 18 presents the Individual Cancer Risk Percentages with the tabulated data. The table sorts out the values of the results as it related to the separate age categories (Child 2-6, Child 6-12, and Adult) with the various concentration levels. The noticeable differences are in the mean and maximum concentrations. However, the maximum concentrations for the two youngest study groups have a percentage of 170% and 96%

which indicates that at the maximum case, the percentage risk of cancer is almost 200 and 100 more likely than not.

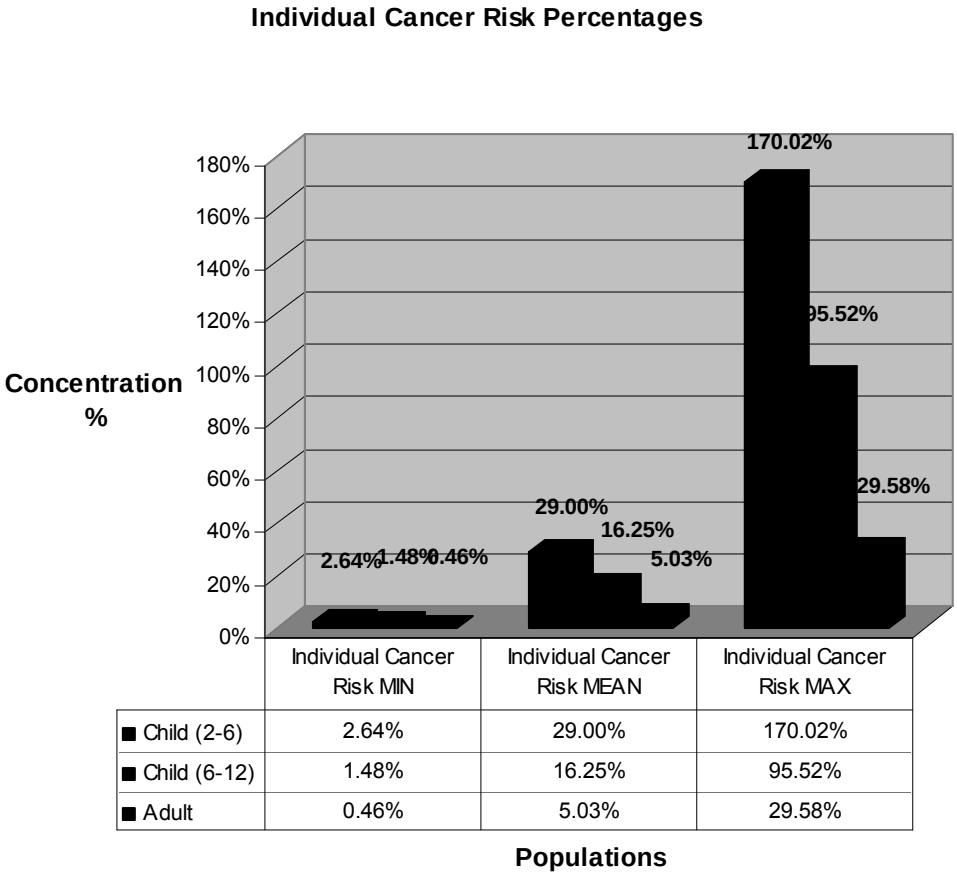



**Figure 18 Individual Caner Risk**

Figure 19 presents the Individual Cancer Risk Percentage (Minimum Concentration), Figure 20 Individual Cancer Risk Percentages (Mean Concentration), and Figure 21 Individual Cancer Risk Percentages (Maximum Concentration) graphically displays the individual concentration levels in a pie chart. Figure 19, further displays that in the observations that in this case the youngest group (Child 2-6) is the most

susceptible to cancer at the minimum concentration as a percentage with a difference of 1.16% between the two youngest groups.

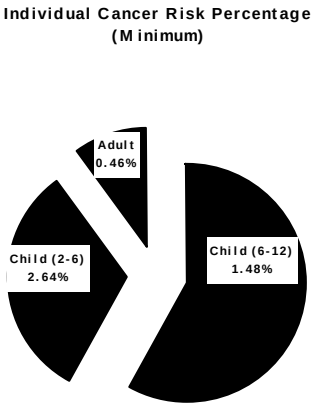



**Figure 19 Individual Cancer Risk (Minimum Concentrations)**

Figure 20, further displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the mean concentration of almost 30%.

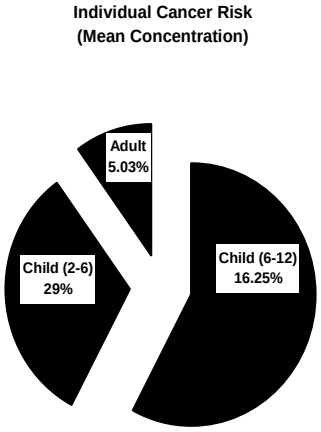



**Figure 20 Individual Cancer Risk (Mean Concentrations)**

Figure 21, further displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the maximum concentration as a percentage of 170% that indicates that at the maximum case, the percentage risk of cancer is almost 200 times more likely than not.

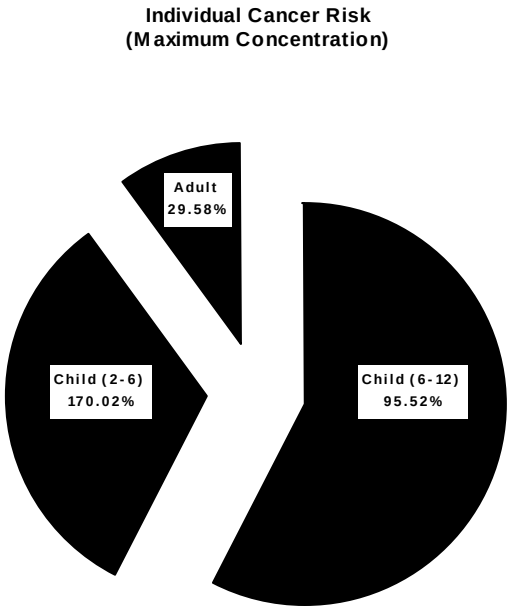



**Figure 21 Individual Cancer Risk (Maximum Concentrations)**

Figure 22 presents the Maximum Cases on a Population (Minimum Concentrations). The table sorts out the values of the results as it related to the separate age categories (Child 2-6, Child 6-12, and Adult) with the various concentration levels.

Figure 23 presents Maximum Cases on Populations (Mean Concentrations) graphically displays the individual concentration levels in a pie chart displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the mean concentration.

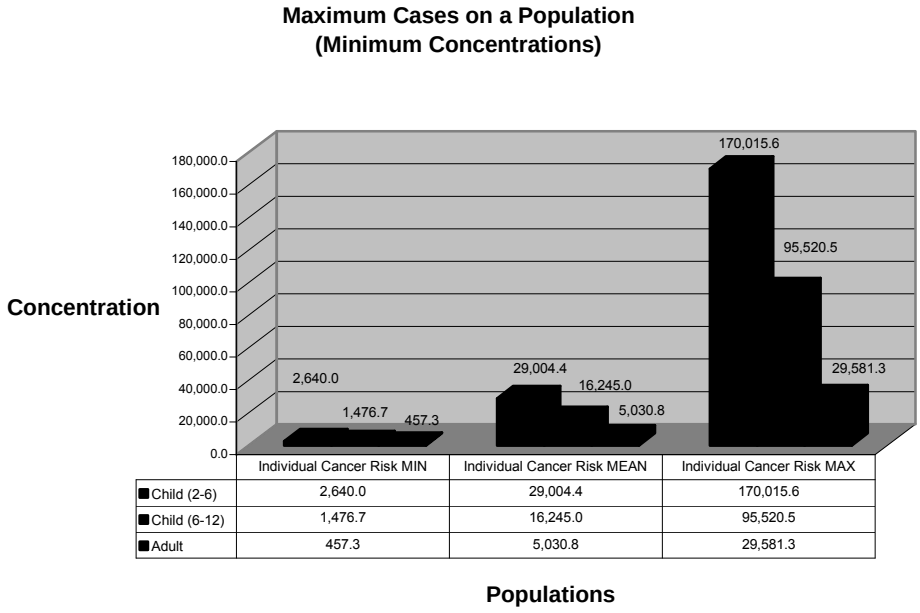



**Figure 22 Maximum Cases on a Population (Minimum Concentrations)**

Figure 24 presents Maximum Cases on a Populations (Mean Concentrations) further displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the mean concentration and that children in this age group are 5 times more likely to the risk of cancer than an adult.

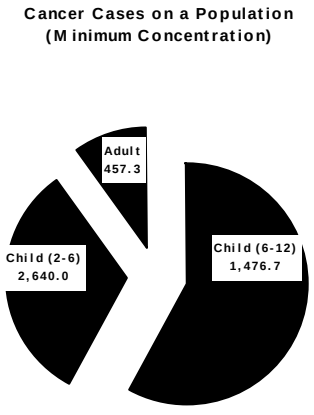



**Figure 23 Maximum Cases on a Population (Minimum Concentrations)**

Figure 25 presents Maximum Cases on a Populations (Maximum Concentrations) further displays that in the observations that in this case the youngest group (Child 2-6) is the most susceptible to cancer at the maximum concentration and that children in this age group are 5 times more likely to the risk of cancer than an adult.

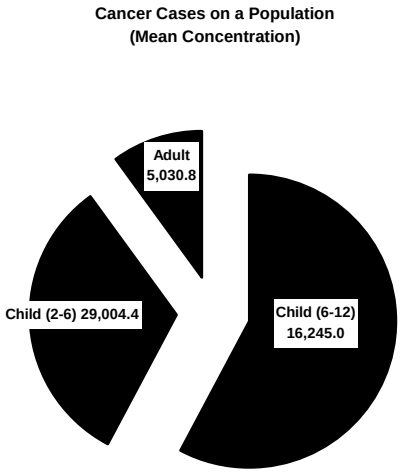



**Figure 24 Maximum Cases on a Population (Mean Concentrations)**

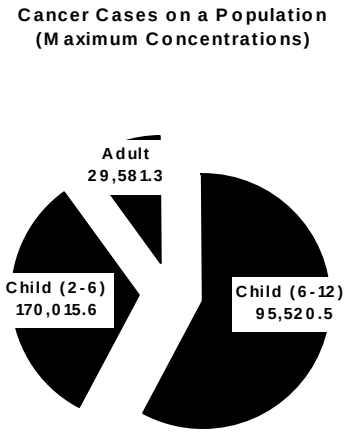



**Figure 25 Maximum Cases on a Population (Maximum Concentrations)**

## INSTITUTIONAL CONTROLS MEASURES

### AIR

The collected data samples ranged from 540 to 1277 l (Gasana and Chamorro 2002). Based on Figure 26, Air EPA Institutional Control Limit, and the standard of 1.5 $\mu g/m^3$, all of the samples were below the Air EPA Institutional Control Limit.

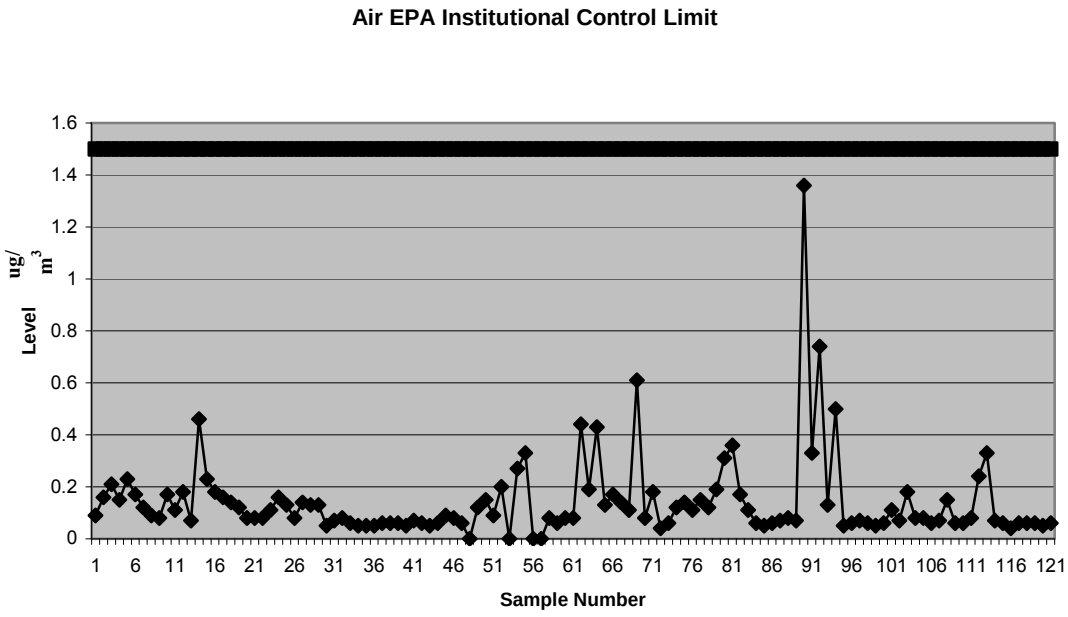



**Figure 26 Air Institutional Control EPA Control Limit**

### FLOOR DUST

The collected data samples ranged from 11 $\mu g/ft^2$ to 40 $\mu g/ft^2$ (Gasana and Chamorro 2002). Based on Figure 27, Floor Dust EPA Institutional Control Limit, and the standard of 40 $\mu g/ft^2$, thirteen samples where found to be above the Floor Dust EPA Institutional Control Limit.

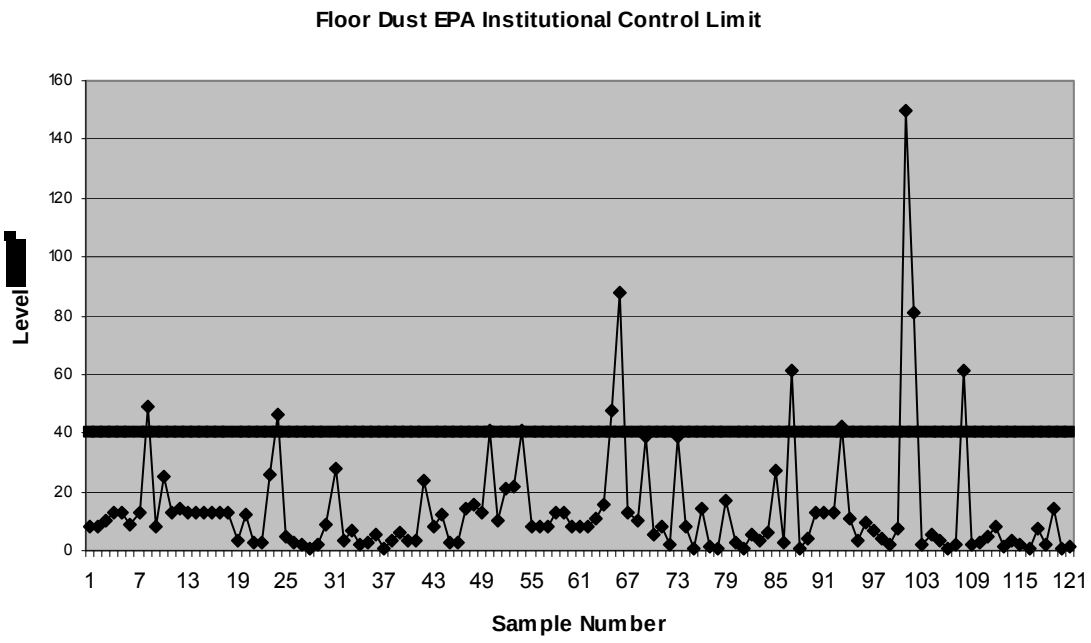



**Figure 27 Floor Dust Wipes Sample Results**

## LEAD BASED PAINT DETERMINATION BY XRF

The collected data samples were either negative or positive (Gasana and Chamorro 2002). To make the graph quantifiable, I assigned the value of positive (1) and negative (2). Based on Figure 28, Lead Based Paint Determination by XRF EPA Institutional Control Limit, and the standard of either positive or negative, twenty one sites were positive.

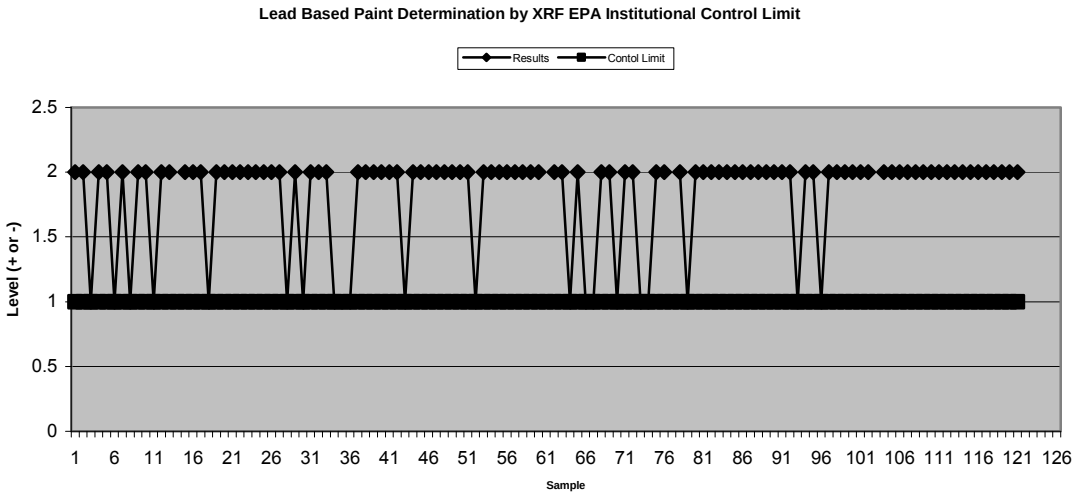



**Figure 28 Lead Based Paint Determination by XRF Institutional Control Limit**

## POTABLE WATER

The collected data samples ranged from 1 to 150 ppb (Gasana and Chamorro 2002). Based on Figure 29, Potable Water EPA Institutional Control Limit, and the standard of 15 ppb, three samples where found to be above the Potable Water EPA Institutional Control Limit.

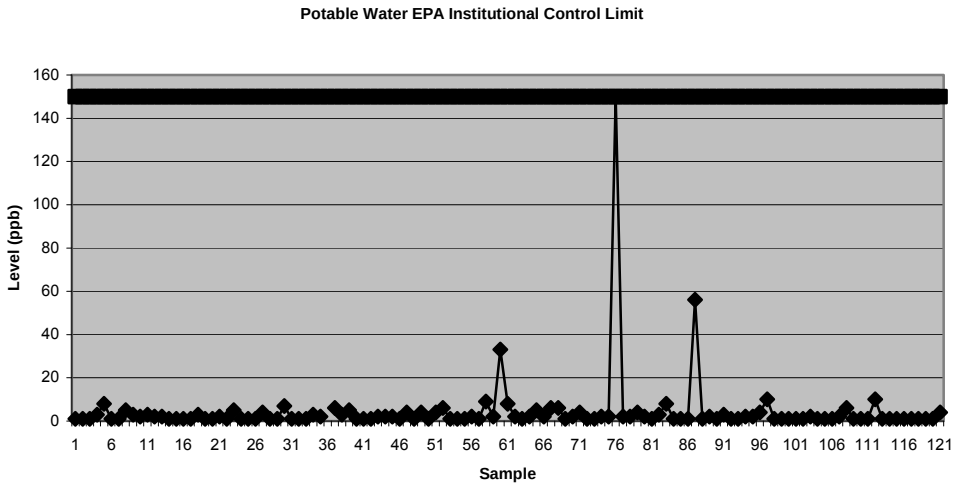



**Figure 29 Potable Water EPA Institutional Control Limits**

## SOIL

The collected data samples ranged from 25 to 1612 ppm (Gasana and Chamorro 2002). Based on Figure 30, Soil EPA Institutional Control Limit, and the standard of 400 ppm, thirty-three samples where found to be above the Soil EPA Institutional Control Limit.

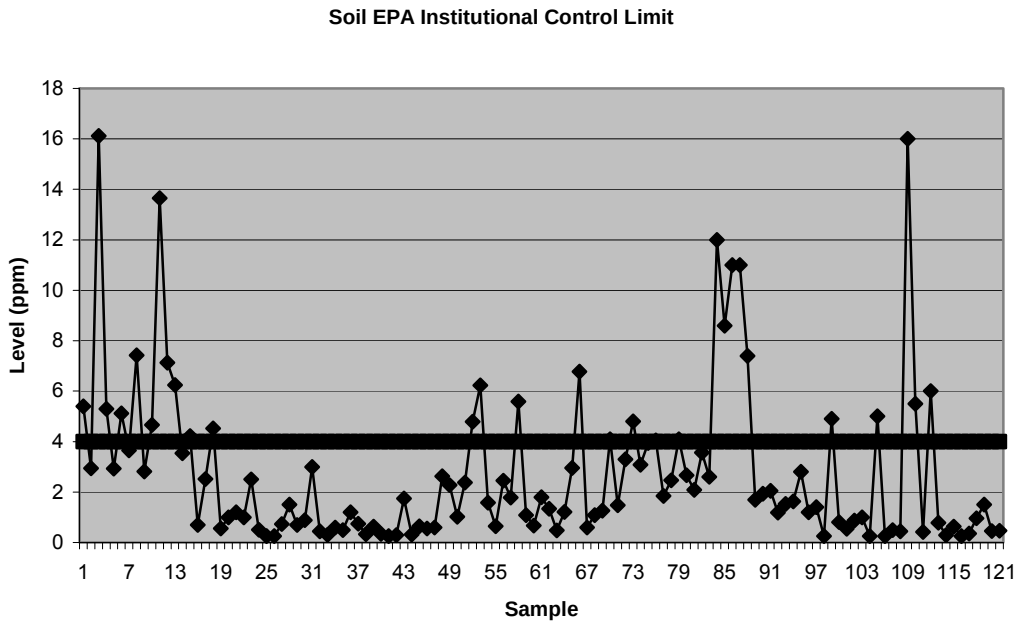



**Figure 30 Soil EPA Institutional Control Limits**

## WINDOW SILL DUST WIPES

The collected data samples ranged from 4 μg/ft$^2$ to 78,000 μg/ft$^2$ (Gasana and Chamorro 2002). Based on Figure 31, Window Sill EPA Institutional Control Limit, and the standard of 400 μg/ft$^2$, twenty-eight samples where found to be above the Window Sill EPA Institutional Control Limit.

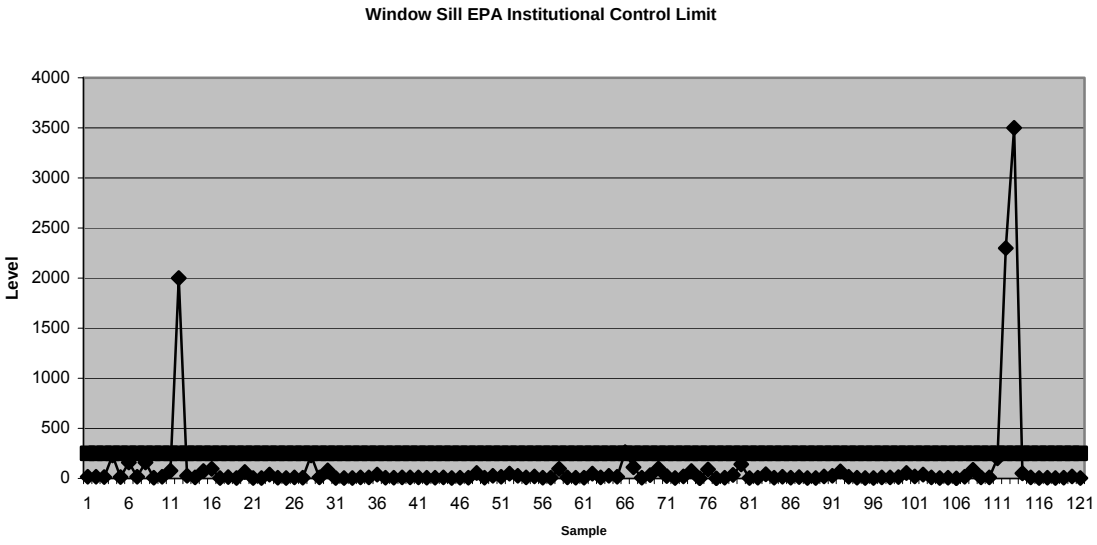



**Figure 31 Window Sill Dust EPA Institutional Control Limit**

## ENVIRONMENTAL ENGINEERING CONTROL MEASURES

## PHYTOREMEDIATION

## PHYTOACCUMULATION

Based on the review of the study, the most suitable environmental engineering control is the use of phytoremediation also known as phytoaccumulation. This method is the uptake and translocation of metal contaminates from the soil into the plant matter via plant roots into the above ground portions of the plants (LaGrega et al 2001). Certain phytoextration methods of uptake have considerable amounts of metals in contrast to other plants and the ambient concentration. The uptake should be metal specific which allows the risk of impoverishing the surrounding soil. In addition to the metal specificity, a high transport of metals from the roots to the shoots should take place for this method to be effective during remediation treatment (Suthersan 2002). The plants act as a filter

or traps for the metals and remove them from contaminated soils. Once these plants have performed their function, they are harvested and incinerated with waste ash at hazardous waste landfills (LaGrega et al 2001).

**GENERAL DESIGN FACTOR PHYTOREMDIATION**

Since the design of the phytoremdiation depends on the specific site and its characteristics more environmental information should be colleted. In spite of this being an environmental health study and missing information, there are design considerations that could be used for all methods including the following:

1. Contaminant Levels
2. Treatability
3. Plant Selections
4. Cost

**PLANT SELECTION**

When considering specific plants for the use of the design, plants with high amounts of biomass, produce exudates, grows quickly, have long growing seasons, have roots that extend to the depth of the contaminants, and have a high tolerance for concentrated contaminants (LaGrega et al 2001).

Useful biomass consists of parts of the plant that are available for evapotranspiration, which includes the leaf surface area and the plant root system. Trees are also utilized in this process because of their large biomass and their depth of root penetration. These roots are more beneficial because they may be able to reach a shallow saturated zone (LaGrega et al 2001). Plants that were selected and are currently available in the market include following in Table 16, Plant Selections.

**Table 18 Plant Selection**

Indian Mustard (Brassica Juncea)

Barely (Hordeum Vulgare)

Oats (Avena Sativa)

Alfalfa (*Medicago Sativa*)

## COST

## SCENARIO ANALYSIS TOOL

Within the Monte Carlo tools, the Scenario Analysis tool I used to determine the most economic option for remediation. The Scenario Analysis ran, based on the assumptions in Table 8, Assumptions. Figure 32, Simulated Cost is a schematic of the outputs of the simulation.

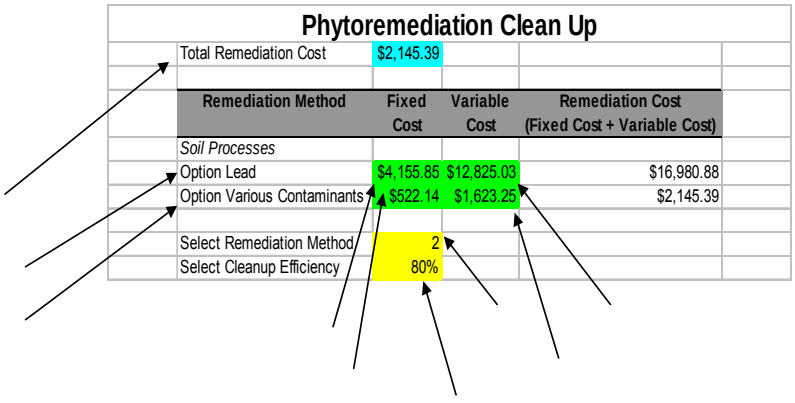



**Figure 32 Simulated Cost**

Based on the analysis, in Figure 32, the following Results of Simulation is listed in Table 19.

**Table 19 Results of Simulation**

| Itemized List Before Simulation Run | | | |
|---|---|---|---|
| **Option Various Contaminants** | | **Option Lead** | **Cost** |
| Option Various Contaminants (Fixed) | $516.25 | Option Lead (Fixed) | $4,130.00 |
| Option Various Contaminants ((Variable Cost includes labor and material at (+ and - 5%)) | $1,548.75 | Option Lead ((Variable Cost includes labor and material at (+ and - 5%)) | $12,390.00 |
| **Total Remediation Cost Various Contaminants (Fixed Cost + Variable Cost)** | $2,065.00 | Total Remediation Cost Lead (Fixed Cost + Variable Cost) | $16,520.00 |
| **After Simulation Run** | | | |
| **Option Various Contaminants** | | **Option Lead** | |
| Option Various Contaminants (Fixed) | $4,155.85 | Option Various Contaminants (Fixed) | $522.14 |
| Option Various Contaminants ((Variable Cost includes labor and material at (+ and - 5%)) | $12,825.03 | Option Various Contaminants ((Variable Cost includes labor and material at (+ and - 5%)) | $1,623.25 |
| **Total Remediation Cost Various Contaminants (Fixed Cost + Variable Cost)** | $16,980.88 | Total Remediation Cost Lead (Fixed Cost + Variable Cost) | $2,145.39 |

Table 19, presents the Results of the Simulation of both the Option Lead and Option Various Contaminants, simulated costs of the two phytoremediation options.

The simulated cost ranged from $$16,980 and $2,145. The simulation resulted in the selection of Option Lead as the Phytoremediation Option at a cost of $2,145.39. As a result of the simulation, the most economical cost is$2,145.39 as depicted in Costs of Options in Figure 33.

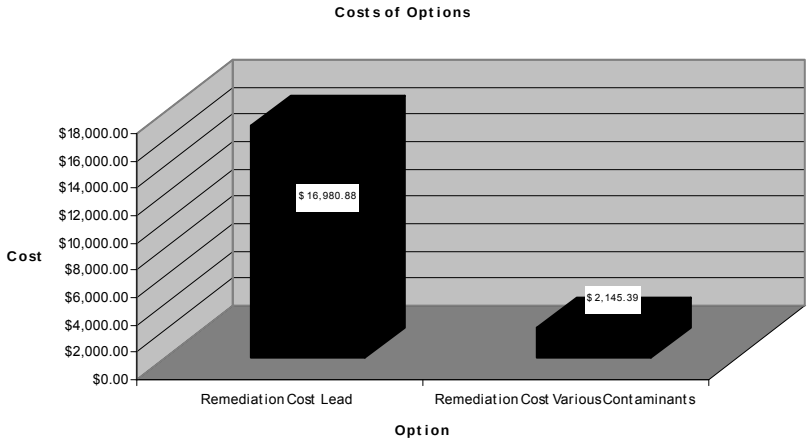



**Figure 33 Cost of Options**

## 6. CONCLUSION

**OBJECTIVE 1**: Design and develop a comprehensive risk management methodology, with focus on a quantitative risk assessment that is applicable to the geographical and environmental conditions of Miami Inner City Area.

This study provides a comprehensive risk management methodology with the focus that contributes to the reduction and eradication of lead contamination at the local level. By breaking the study down into two distinct parts a solution for both the public health (environmental health) aspect as well as the engineering aspect were achieved and provided proof that environmental health studies can be ling to environmental engineering study to provide substantial results. Specifically, the Quantitative Risk Assessment showed that there is a significant risk to the youngest study group (Child 2-6) and the second youngest group (Child 6-12). It is of importance to mention that adults did not escape this risk. From the study and utilizing the lead MCL of .0015 mg/L, the specific was detailed below:

- *The Toxicity Score* levels between the minimum and mean concentration levels there is only a difference of 166,666.6 as a score. However, the most significant jump is from the mean concentration to the maximum concentration of 891,333.4. This clarifies that as the concentration level increase so does the various Toxicity Scores.
- *The Chronic Daily Inhalation Intake* includes takes into account various parameters such as body weight, absorption rate, and soil ingested to determine the results of the different intake levels. It is observed that at the different concentration levels, the data indicates that the younger the study groups, the

higher the concentration levels. There are variations, but as mentioned previously, the most susceptible groups are the child 2-6 and child 6-12. At the Minimum Level of 25 mg/L, there is only a difference of 4.2 $\frac{mg}{kg * day}$ between the children's groups. At the Mean level of 275 mg/L there is only a difference of 46.9 $\frac{mg}{kg * day}$ children's groups. At the Maximum Level of 1612 mg/L there is only a difference of 332.3 $\frac{mg}{kg * day}$ children's groups.

- *The Daily Intake (Average) from Dermal Contact* with the minimum concentration of 275 mg/L as the variable in addition to various parameters such as skin exposed, dust adherence, skin absorption rate, effect of soil matrix were used to determine the results of the various daily dermal intake levels. It is observed that at the different concentration levels, the data varies from and indicates that all of the category groups' levels are differentiated by only a few data points. The most susceptible group is the child within the age group of 2-6 at .000047 ($mg/m^3/kg*days$), child 6-12 .000039 ($mg/m^3/kg*days$), and Adult .000028 ($mg/m^3/kg*days$). This amounts to a difference of only .000008 ($mg/m^3/kg*days$) and .000011 ($mg/m^3/kg*days$) respectively.
- *The Hazard Index* pattern continues with the most susceptible group is the child 2-6 at .03130, child 6-12 .0260, and Adult .01870. Based on the criteria of the hazard index being less than the prescribed value of 1, then this hazard is acceptable.

- *The Individual Cancer Risk (Minimum Concentration)* shows that the youngest group (Child 2-6) is the most susceptible to cancer at the minimum concentration with a difference of .0116 between the two younger.
- *The Individual Cancer Risk (Mean Concentration)* shows that the youngest group (Child 2-6) is the most susceptible to cancer at the mean concentration with a difference of .1275 between the two younger groups.
- *The Individual Cancer Risk Percentages* show a notably difference in that the maximum concentrations for the two youngest study groups have a percentage of 170% and 96% which indicates that at the maximum case, the percentage risk of cancer is almost 200 and 100 more likely than not based on a population of 100,000. This fact is the most startling in all of the observations.

The environmental engineering methods for remediation in contained media involving detailed techniques, procedures, and recommendation for the treatability for the local community being studied was also determined successful. As indicated in the study, the primary contaminated media was soil. As a result, objective 2 was explored based on that consideration. The detailed results are below:

**OBJECTIVE 2:** Develop a scientific approach for interpreting public health concerns with environmental engineering methods for remediation in contained media involving detailed techniques, procedures, and recommendation for the treatability for the local community being studied.

**INSTITUTIONAL CONTROLS MEASURES**

- *Air*

Based on the control limit, all of the samples were below the Air EPA Institutional Control.

- *Floor Dust*

Based on the control limit, 13 samples where found to be above the Floor Dust EPA Institutional Control Limit.

- *Lead Based Paint Determination by XRF*

Based on the control limit, 21 samples where found to be positive and above the Lead Based Paint Determination by XRF EPA Institutional Control Limit

- *Potable Water*

Based on the control limit, 3 samples where found to be above the Potable Water EPA Institutional Control Limit.

- *Soil*

Based on the control limit, 33 samples where found to be above the Soil EPA Institutional Control Limit.

- *Window Sill Dust Wipes*

Based on the control limit, 28 samples where found to be above the Window Sill EPA Institutional Control Limit

**ENVIRONMENTAL ENGINEERING CONTROL MEASURES**

It was determined that best environmental engineering control and use of phytoremediation measure is phytoextration/phytoaccumulation. This method is the uptake and translocation of metal contaminates from the soil into the plant matter via

plant roots into the above ground portions of the plants. These plants will act as a filter or traps for the metals and remove them from contaminated soils. Once these plants have performed their function, they are harvested and incinerated with waste ash at hazardous waste landfills (LaGrega et al 2001).

## GENERAL DESIGN FACTOR PHYTOREMDIATION

In this case, the design will focus on:

1. Contaminant Levels
2. Treatability
3. Plant Selections
4. Cost

## PLANT SELECTION

The specific plants for the use of the design, plants with high amounts of biomass, produce exudates, grows quickly, have long growing seasons, have roots that extend to the depth of the contaminants, and have a high tolerance for concentrated contaminants (LaGrega et al 2001). These plants include the following:

**Table 20 Plant Selection**

Indian Mustard (Brassica Juncea)

Barely (Hordeum Vulgare)

Oats (Avena Sativa)

Alfalfa (*Medicago Sativa*)

## COST

Table 19, presents the Results of the Simulation of both the Option Lead and Option Various Contaminants, simulated costs of the two phytoremediation options. The simulated cost ranged from $$16,980 and $2,145. The simulation resulted in the selection of Option Lead as the Phytoremediation Option at a cost of $2,145.39.

As a result of the simulation, the most economical cost is$2,145.39 as depicted in Costs of Options in Figure 33.

## 7. RECOMMENDATIONS FOR FUTURE WORK

Feasibility Study (FS)

Since this was not a designed risk assessment, certain functions and procedures were not applied. In order to conduct a successful risk assessment a Remedial Investigation (RI) and or Feasibility Study (RI) should be conducted. Within a Remedial Investigation (RI), a Feasibility Study (FS) should be conducted.

The major plans of an investigation and sampling plan should include:

1. Summary of site background information
2. Summary of assessment of existing data
3. Contaminants of interest (specifically more trace metals and carcinogens)
4. Sampling locations and frequency
5. Sample and testing procedures
6. Operation plan and schedule
7. Cost estimate
8. Other supporting document

a) Quality assurance and quality control plans

b) Health and safety plans

c) Data management plans

The benefit to this study is that most of this has been collected.

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# APPENDICES

## APPENDIX A
## LITERATURE REVIEW

10. Lead

10.1 Toxicity

Lead Pb, (L. plumbum) atomic number 82, is a multimedia toxicant that exposes its toxicity even when the specific exposures appear relatively modest. A soft, heavy, toxic and malleable poor metal, lead is bluish white when freshly cut but tarnishes to dull gray when exposed to air. Lead is used in building construction, lead-acid batteries, bullets and is part of solder, pewter, and fusible alloys. Lead has the highest atomic number of all stable elements.

**Table 21 General Characteristics of Lead (LeGrange et. al 2001)**

| General Characteristics | |
|---|---|
| **Name** | Lead |
| **Symbol** | Pb |
| **Number** | 82 |
| **Group** | 14 |
| **Period** | 6 |
| **Block** | p |
| **Standard Atomic Weight** | 207.2(1) $g*mol^{-1}$ |
| **Electron Configuration** | [Xe] $4f^{14}$ $5d^{10}$ $6s^2$ $6p^2$ |
| **Electrons per shell** | 2,8,18,32,18,4 |

10.2 Sources

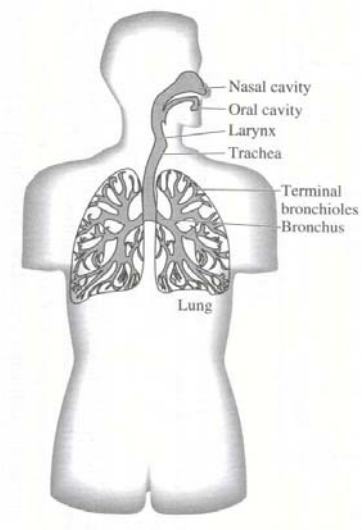
Sources of lead from the household environments include lead paint, automotive and industrial lead emissions, and lead present in food and water. Lead from paint and airborne emissions are deposited in house dust as a result of being tracked in on shoes brought in from the work place is highly documented (Sutton et. al., 1995). A further investigation done in Trinidad and Tobogo was conducted on the general population and

its association between occupational health and contaminated communities with very little information on Blood Lead Level (BLL) (Rajkumar et. al., 2005).

**Table 22 Physical Properties (LeGrange et. al 2001)**

| Physical Properties | |
|---|---|
| **Phase** | Solid |
| **Density (near r.t.)** | 11.34 g*cm-3 |
| **Liquid Density at mp** | 10.66 g*cm-3 |
| **Melting Point** | 600.61 K, 327.46°C, 621.43°F |
| **Boiling Point** | 2022 K, 1749°C, 3180°F |
| **Heat of Fusion** | 4.77 $kJ*mol^{-1}$ |
| **Heat of Vaporation** | 179.5 $kJ*mol^{-1}$ |
| **Heat Capacity** | (25°C) 26.650 $J*mol-1*K^{-1}$ |

10.3 Toxicology Effects of Lead




Regardless of the specific sources of lead integrates into critical organs systemically (Rosen 1995). Lead is exposed through the lungs and gastrointestinal tract (Glorennec 2005). Because children have a great affinity of hand-to-mouth contact, their risk is greater to higher lead levels than adults. As a result of this greater affinity, children have a higher efficiency for absorption (Rajkumar et. al., 2005).

**Figure 34 Gastrointestinal Tract (LeGrange et. al 2001)**

Adults absorb 35-50% of lead that they ingest as opposed to 50% by children. Ninety percent of the body that is burdened with lead is deposited in the bone. This then can complicate the long-term devolvement and growth of children because on the extent

of blood always being present. In fact, the movement of maternal lead from bone during pregnancy and lactation together with other environmental exposures increases the body's burden of lead in children (Ahamed 2005).

**Table 23 Toxic Effects of Lead (LeGrange et. al 2001)**

**Toxic Effects of Lead**

| Toxic Substance | Carcinogenetic Effects | NonCarcinogenetic Effects |
|---|---|---|
| Lead | Kidney Tumor (in test animals) | Reduced birth weight, anemia, increase blood pressure, brain and kidney damage, IQ impaired, decreased learning |

10.4 Environmental Health

Defining environmental health in terms of the type of problems solved as opposed to its systematic methodology should be the rational to the study of environmental health. These problems range from the treatment and disposal of liquid and airborne waste, the elimination or reduction of stress in the workplace, purification of drinking water supplies, and the impact of over population. From a professional standpoint, long range problems that are currently being solved include the effects of toxic chemicals and radioactive waste, acidic deposition, depletion of the ozone layer, and global warming. Because environmental health is a very broad area and encompasses many sub areas and when solving problems multidisciplinary approach should be utilized. Professions in this area include engineers, scientist, lawyers, mathematics, epidemiologist, scientist, and physicians (Moeller 1992).

10.4.1 Childhood Lead Poisoning

When studying environmental health as is applies to the environment, the most widely affected group and area are children and childhood lead poisoning. Although lead

exposure has been reduced in the United States, it still remains a public health threat, especially among children. The hand to mouth touching, absorption through the skin and the considerable amount of lead in soils are examples as to how small children are a primary target for lead poisoning. The discontinuance of lead in most of our everyday uses sources is a significant contributor to the reduction of lead exposure; however, the problem still exists.

Over the last twenty years, there has been a phase out process of lead from gasoline, food beverage cans, house paints, as well as the limitations on industrial emissions, drinking water, and other consumer goods and at hazardous waste sites. In the United States, the sources are magnified even more in older major metropolitan areas (Gasana and Chamorro 2002).

**Table 24 Categories of Estimation Methods for Children Exposed to Lead By Sources (Committee on Advances in Assessing Human Exposure to Airborne Pollutants 1991)**

| Categories of Estimation Methods for Children Exposed to Lead by Sources | | |
|---|---|---|
| **Source Category** | **Level of Precision** | **Basis of Exposure Measurements** |
| Lead in Paint | Potential exposure | Determination of numbers of children in housing with highest likely lead paint burdens |
| | Potential exposure with a better indication of actual exposure risk | Number of children estimated to be in lead paint housing with deterioration: peeling paint, broken plaster, damage |
| | Likely Actual Exposure | Use of specifically determined prevalence for an NHANES II stratum matching such children; other, regional survey data |
| Lead in Gasoline | Potential exposure (blood lead change) in a subset of US urban child population | Total number of young children in 100largets cities |
| | Actual exposure based on leaded gasoline combustion | Logistic regression analysis to estimate numbers of children falling below selected blood lead criterion values |
| Lead from Stationary Sources | Potential exposure | Total of young children in common cities within certain proximity of led operations |
| Lead from Stationary Sources | Actual exposure | Prevalence of indicated of lead at or above some criterion level in actual field studies of stationary sources |
| Lead in Dust and Soils | Potential exposure | Summing of potential exposure numbers from the above three categories |
| | Actual exposure | Summing of corresponding actual exposure numbers from first three actual exposure categories or use of multimedia regression equation (not possible with present data) |
| Lead in Drinking Water | Potential exposure | Number of young children either in homes with old lead plumbing or in law homes with old solder |
| | Actual exposure measurable, but not the highest risk of society | Number of young children in homes with lead in drinking water >20 µg/L |
| Lead in Food | Potential exposure at or near toxic magnitude | Number of children in age group |
| | Acute exposure | Fraction of potentially exposed children whose food lead intake might raise blood lead high enough to cause concern |

## 10.5 Blood Lead Levels (BLL)

### 10.5.1 Biomarkers

Blood Lead Levels (BLL) has extensively been used as a biomarker of lead exposure. *Schulte* (1995) defines a biological marker as a biological indicator that is used to represent an exogenous exposure, effects on exposure, early or frank disease, or susceptibility to any of these. The usefulness and the advantage of utilizing biomarkers is that it is primarily a resultant from its impendence to provide information with regard to the hazard or risk and ultimately to the prevention of the disease. This is critical and essential in those areas where there are breaches in the scientific knowledge foundation concerning exposure-disease relationships or characteristics. This case is a perfect example of a breach in the scientific knowledge. Human studies mainly use biomarkers to provide useful information to scientist and decision makers (Troast et. al, 2003).

Advantages to biomarkers use in humans, is those of recent exposure to lead, the biomarkers can be detected within the preceding 4 months (Committee on Advances in Assessing Human Exposure to Airborne Pollutants 1991). Another advantage to biomarkers is that it can be applicable to more than one event. The BLL of an individual is greatly influenced by the lead exposure intensity during the recent few weeks or few months prior to the measurement (Troast et. al, 2003).

Blood Lead Levels (BLL) as low as 10 μg/dL are associated with decreased intelligence and impaired neurobehavioral development. In fact, *Glorennec* (2005) generates it a step further and maintains that lead induces neurobehavioral and cognitive effects in children. Other effects of BLL ≥10 μg/dL have been associated with aggressive behavior, developmental affect, hyperactivity, weight loss, renal effects, anemia, and

effects on vitamin D metabolism in children (Sanchez-Nazario 2003). There is a misconception that with this low level BLL and with no distinctive signs there are not any negative health effects (Rajkumar et. al., 2005). Furthermore there are not any known published studies to this present date that show that at any threshold blood lead levels there exist no health effect ((Glorennec 2005). Death, comas, and seizures can be brought about with childhood lead levels at 70 μg/dL to learning and behavioral problems with lower levels (Meyer et. al. 2005).

10.6 Childhood Lead Poisoning and Hazardous Waste

According to the Environmental Protection Agency (EPA), the major contributor of childhood lead poisoning from a hazardous waste standpoint and soil is the dust ingestion pathway. Where surface dust lead levels are elevated, the hand-to-mouth is the primary culprit. From this perspective the focal point should be on environments impact by wastes from lead smelting and mining. As a result, the *Lorenzana* study specifies the approach used at these Superfund sites included a combination of engineering and institutional controls and public outreach. The engineering controls included the removal or burial of lead laden soils and dusts, coverage of soils with vegetation, and stabilization of soils from erosion. The institutional controls included barriers and enforcing limits of access to areas that have a high potential for contamination. The outreach aspect included programs that can include public education about lead exposure pathways and hazards and monitoring of blood lead concentrations in the community (Lorenzana et. al. 2003).

11. Institutional Controls Measures

11.1 Laws

11.1.2 Resource Conservation and Recovery Act (RCRA)

In 1976, the Resource Conservation and Recovery Act (RCRA) for the first time placed a significant role on the federal government for the management of hazardous waste. Within this office, a series of amendments were added to the Solid Waste Act of 1965, creating a separate Office of Solid Waste within Environmental Protection Agency. This office had the responsibility of establishing a comprehensive regulatory program that includes identifying which wastes are hazardous and to establishing a manifest system for tracking waste. The cradle-to-the-grave process is a product of this process (LeGrange et. al 2001).

The intent of Resource Conservation and Recovery Act (RCRA) is that future management of hazardous waste now stressed conservation and recovery of reusable sources, such as recycling, as opposed to disposal. The Resource Conservation and Recovery Act (RCRA) established an extensive regulatory process for newly created and generated waste, but nothing was done to help correct the results of poor disposal practice and inadequate technology that was done in the past. It was common practice for owners to abandon their plants and to leave tanks and other hazardous waste for others to deal with (LeGrange et. al 2001).

As a result of this issue and a major environmental episode (Love Canal), congress passed the Comprehensive Environmental Response Compensation and Liability Act (CERCLA) of 1980 was created. The Comprehensive Environmental Response Compensation and Liability Act (CERCLA) of 1980 was later amended in

1996 by the Superfund Amendments and Reauthorization Act (SARA). Superfund can be used for both of these laws (LeGrange et. al 2001).

11.1.3 Federal Hazardous Waste Regulations under Resource Conservation and Recovery Act

Within the Resource Conservation and Recovery Act (RCRA) congress directed the Environmental Protection Agency (EPA) to institute a major regulatory program for hazardous waste. This regulation define which waste were established, established an Environmental Protection Agency (EPA) notification process for organizations producing waste, and set up detailed regulations covering the generation, transportation, treatment, storage, and disposal of hazardous waste (LeGrange et. al 1994).

Under Resource Conservation and Recovery Act (RCRA) hazardous waste can be defined as a solid waste or combination of solid wastes, which because of it quantity concentration, physical, chemical, or infectious characteristics may

- Cause or significantly contribute to and increase mortality or an increase in serious irreversible, or incapacitating reversible, illness, or
- Pose a substantial present or potential hazard to human health or the environmental when improperly treated, stored, transported, or disposed of, or otherwise managed (LeGrange et. al 1994).

To further simplify this area as it pertains to waste, solid waste is defined as:

> any garbage, refuse, sludge from a waste treatment plant, water treatment plant, or air pollution control facility and other discarded material including solid, liquid, semi-solid, or contained gaseous material resulting from industrial, commercial mining, and agriculture operations, and from community activities, but does not include solid or dissolved materials in irrigation return flows or industrial discharges which are point sources subject to permits under Section 402 of the Federal Water Pollution

> Control Act, as amended (86 Stat. 880), or source, special nuclear, or byproduct materials defined by the Atomic Energy Act of 1954 as amended (68 Stat. 923) (LeGrange et. al 2001).

There are three ways in which a solid waste can be considered a hazard under Resource Conservation and Recovery Act (RCRA).

1. The waste is specifically listed in any of the four list provided in the Environmental Protection Agency (EPA) regulations.
2. The waste is tested and meets one of the four characteristics established by the Environmental Protection Agency (EPA). These four characteristics are ignitability, corrosive, reactive or toxic.

   **Ignitable Waste**. Ignitable Wastes are liquids with a flashpoint below 60 °C or solids capable of causing fire under standard temperature and pressure. Ignitable Wastes were assigned Environmental Protection Agency (EPA) Waste No. D001.

   **Corrosive Waste**. Corrosive Waste are aqueous waste with a pH below 2 or above 12.5, or which corrode steel at a rate to exceed 0.25 inch per year. Corrosive wastes are classifieds D0002.

   **Reactive Waste**. Reactive Wastes are normally unstable, react violently with air or water, or form potentially explosiveness mixtures with water. This category also includes waste threats that emit toxic fumes when mixed with water and materials capable of denotation. Reactive Wastes are classified as D003.

   **Toxicity**. The objective of this area is to determine whether its parameters constitute of toxic constituents in solid wastes leached into the groundwater if the waste is placed in a municipal solid waste landfill.

3. The waste is declared hazardous by the generator on the basic knowledge of the waste (LeGrange et. al 2001).

11.1.4 Lead Contamination Control Act (LCCA)

The Lead Contamination Control Act (LCCA) of 1988 authorized a Centers Disease Control (CDC) grant program to be established in childhood lead poisoning prevention. The program had three primary efforts. First, the CDC program increased emphasis on data collection and analysis by childhood lead poisoning prevention programs. A special software program, System for Tracking Elevated Lead Levels and Remediation (STELLAR), has been developed to assist childhood lead poisoning prevention programs in both case and data management. Secondly, increased emphasis has been placed on evaluating the impact of interventions.

The Lead Contamination Control Act (LCCA) of 1988 authorized the CDCP to make grants to state and local agencies for comprehensive programs designed to

- Screen infants and children for elevated BLL's,
- Ensure referral for medical and environmental intervention for lead-poisoned infants and children, and
- Provide education about childhood lead poisoning.

The LCCA of 1988 also has the responsibility to

- Develop programs and policies to prevent childhood lead poisoning,
- Educate the public and health-care providers about childhood lead poisoning,
- Provide funding to state and local health departments to determine the extent of childhood lead poisoning by screening children for elevated blood lead levels, helping to ensure that lead-poisoned infants and children receive medical and

environmental follow-up, and developing neighborhood-based efforts to prevent childhood lead poisoning, and

- Support research to determine the effectiveness of prevention efforts at federal, state, and local levels.

12. Standards

Various federal agencies have provided advisory standards and or enforceable regulation that set the lead levels in different media.

**Table 25 Summary of Standards and Regulations for Lead (Moeller, D 1992)**

**Summary of Standards and Regulations for Lead**

| Agency | Media | Level | Comments | Source |
|---|---|---|---|---|
| Centers For Disease Control and Prevention | Blood | 10 µg/dL | Advisory: level pf concern for children | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Blood | 40 µg/dL | Regulation: cause for written notification and medical exam | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Blood | 50 µg/dL | Regulation: cause for medical removal from exposure | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Air (workplace) | 50µg/$m^3$ | Regulation: permissible exposure limit (8-hour average) (general industry) | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Occupational Safety and Health Administration (OSHA) | Air (workplace) | 30 µg/$m^3$ | Regulation: Action Level | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Air (ambient) | 1.5 µg/$m^3$ | Regulation: National Ambient Air Quality Standard; 3 month Average | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Soil (Residential) | 400 mg/kg | Soil Screening guidance | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Water Drinking | 15 µg/L | Action level for public supplies | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| US Environmental Protection Agency (EPA) | Water Drinking | 0 µg/L | Non enforceable goal; maximum contaminant level goal | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |
| Consumer Product Safety Commission (CPSC) | Paint | 600 ppm or 0.06% | Regulation; by dry weight | http://www.atsdr.cdc.gov/HEC/CSEM/lead/standards_regulations.html |

In addition to the various institutional controls from the different federal agencies' standards and regulation for the various contaminated lead in media, other controls

include proposed maximum concentration limits (MCLs), proposed maximum concentration limit guidelines (MCLGs), and Water Quality Criteria for Fish and Drinking Water are also available for use.

**Table 26 Drinking Water Contaminants (http://www.epa.gov/safewater/contaminants/index.html)**

| Contaminant (Inorganic) | Maximum Contaminant Level Goal (MCLG) mg/L | Maximum Contaminant Level (MCLs) or Treatment Technique (TT) mg/L | Potential Health Effect from Ingestion of Water | Sources if Contaminant in Drinking Water |
|---|---|---|---|---|
| Antimony | 0.006 | 0.006 | Increase in blood Cholesterol; decrease in blood sugar | Discharge from petroleum refineries; fire retardants; ceramics; electronics; solder |
| Arsenic | 0 | .010 (as of 01/23/06) | Skin damage or problems with circulatory systems, and may have increased risk of getting cancer | Erosion of natural deposits; runoff from orchards, runoff from class & electronic production waste |
| Asbestos (fiber > 10 micrometers) | 7 million fibers per liter | 7 MFL | Increased risk of developing benign intestinal polyps | Decay of asbestos cement in water mains; erosion of natural deposits |
| Barium | 2 | 2 | Increase in blood pressure | Discharge of drilling wastes; discharge from metal refineries; erosion of natural deposits |
| Beryllium | 0.004 | 0.004 | Intestinal lesions | Discharge from metal refineries and coal burning factories; discharge from electrical, aerospace, and defense industries |
| Cadmium | 0.005 | 0.005 | Kidney damage | Corrosion of galvanized pipes, erosion of natural deposits; discharge from metal refiners; runoff from waste batteries and paints |
| Chromium (total) | 0.1 | 0.1 | Allergic dermatitis | Discharge from Steel and pulp mills, erosion of natural deposits |

| Contaminant (Inorganic) | Maximum Contaminant Level Goal (MCLG) mg/L | Maximum Contaminant Level (MCLs) or Treatment Technique (TT) mg/L | Potential Health Effect from Ingestion of Water | Sources if Contaminant in Drinking Water |
|---|---|---|---|---|
| Copper | 1.3 | TT Action Level 1.3 | **Short Term Exposure:** Gastrointestinal distress<br>**Long Term Exposure:** Liver of Kidney Damage | Corrosion of household plumbing systems; erosion of natural deposits |
| Cyanide (as free cyanide) | 0.2 | 0.2 | Nerve damage of thyroid problems | Discharge from steel or metal factories; discharge from plastics and fertilizer factories |
| Fluoride | 4 | 4 | Bone Disease (pain and tenderness of the bones); Children may get mottle teeth | Water Additive which promotes strong teeth. Erosion of natural deposits; discharge from fertilizer and aluminum factories |
| Lead | 0 | TT Action Level .0015 | **Infants and Children:** Delays in physical or mental development; children could show slight deficits in attention span and learning disabilities<br>**Adults:** Kidney problems; high blood pressure | Corrosion of household plumbing system; erosion of natural deposits |
| Mercury (inorganic) | 0.002 | 0.002 | Kidney damage | Erosion of natural deposits; discharge from refineries and factories runoff from landfills and crop lands |
| Nitrate (measured as Nitrogen) | 10 | 10 | Infants below the age six months who drink water contaminant nitrates could become seriously ill and if untreated may die. Symptoms include shortness of breath and blue baby syndrome | Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits |
| Nitrate (measured as Nitrogen) | 1 | 1 | Infants below the age six months who drink water contaminant nitrates could become seriously ill and if untreated may die. Symptoms include shortness of breath and blue baby syndrome | Runoff from fertilizer use; leaching from septic tanks, sewage; erosion of natural deposits |
| Selenium | 0.05 | 0.05 | Hair or fingernails loss; numbness in fingers or toes; circulatory problems | Discharge from refineries; erosion of natural deposits; discharge from mines |
| Thallium | 0.0005 | 0.002 | Hair loss; changes in blood; kidney; intestine; or liver problems | Leaching from ore processing sites; discharge from electronics glass, and drug factories |

12.1 EPA's Integrated Risk Information System (IRIS)

According to the Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS), the Oral Rfd Assessment, Inhalation RfC Assessment, and Oral Slope Factor were not available. Specifically, EPA published the following statement:

> "EPA considered providing an RfD for inorganic lead in 1985, and concluded that it was inappropriate to develop an RfD, as documented online in the following statement in 1988:
> A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead."

(http://www.epa.gov/iris/subst/0277.htm).

In addition to the above statement, the EPA's Integrated Risk Information System (IRIS) http://www.epa.gov/iris/, system provided confirmation through email and conversation that confirmed that there is know known available data for there reference data (Email, 4/10/07). Table 27 provides examples of other non carcinogen from EPA's Integrated Risk Information System.

**Table 27 EPA Compounds (http://www.epa.gov/iris/)**

| Compound | Case (CASRN) # | Oral Exposure (RfD) | Inhalation (RfC) | Oral Slope Factor |
|---|---|---|---|---|
| **Acrylamide** | 79-06-1 | $2 \times 10^{-4}$ mg/kg-day | | 4.5 mg/kg/day |
| **Benz[a]anthracene** | 56-55-3 | | | |
| **Benzidine** | 92-87-5 | $3 \times 10^{-3}$ mg/kg-day | | $2.3 \times 10^{2}$ mg/kg-day |
| **Benzo[a]pyrene (BaP)** | 50-32-8 | | | 7.3 mg/kg-day |
| **1,3-Butadiene** | 106-99-0 | | $2 \times 10^{-3}$ mg/m$^3$ | |
| **Captafol** | 2425-O6-01 | $2 \times 10^{-3}$ mg/kg-day | | |
| **Dibenz[a,h]anthracene** | 53-70-3 | | | |
| **Diethyl sulfate** | 64-67-5 | | | |
| **1,2-Diphenylhydrazine** | 122-66-7 | | | $8.0 \times 10^{-1}$ mg/kg-day |
| **Dimethyl sulfate** | 77-78-1 | | | |
| **Epichlorohydrin** | 106-89-8 | | $1 \times 10^{-3}$ mg/m$^3$ | $9.9 \times 10^{-3}$ mg/kg-day |
| **Lead and compounds (inorganic)** | 7439-92-1 | | | |
| **N-Nitroso-N-methylethylamine** | 10595-95-6 | | | $2.2 \times 10^{1}$ mg/kg-day |
| **Styrene** | 100-42-5 | $2 \times 10^{-1}$ mg/kg-day | 1 mg/m$^3$ | |
| **Tetrachloroethylene** | 127-18-4 | $1 \times 10^{-2}$ mg/kg-day | | |
| **Trichloroethylene** | 79-01-6 | | | |
| **1,2,3-Trichloropropane** | 96-18-4 | $6 \times 10^{-3}$ mg/kg-day | | |
| **Vinyl bromide** | 593-60-2 | | $3 \times 10^{-3}$ mg/m$^3$ | |

13. Environmental Engineering

13.1 Environmental Media

13.1.2 Contaminants Release, Transport, Transfer and Transformation

The discharge of chemicals into the environment can be introduced by various methods. These examples include natural processes, human activity, and accidents. Specific examples include leaching of soluble chemicals to the groundwater, designing the construction of site drainage channels, and as chemical spills (LaGrega et al 2001).

**Table 28 Contaminant Release Mechanisms (LaGrega et al 2001)**

| Media | Mechanism | Time Frame |
|---|---|---|
| Air | Volatilization<br>Fugitive Dust Generation<br>Combustion | Chronic<br>Chronic Episodic<br>Episodic |
| Soil | Erosion<br>Leachate Generation<br>Spills | Chronic Episodic<br>Chronic<br>Episodic |
| Surface and Groundwater | Leachate Generation<br>Spills | Chronic<br>Episodic |

Once the chemicals are known from their source, the next step is to determine how the chemical is transported, transferred, and or transformed. This transport involves the movement by advection and diffusion (LaGrega et al 2001).

**Table 29 Mechanisms of Environmental Transfer and Transformation of Chemicals (LaGrega et al 2001)**

| | Fate Mechanism | |
|---|---|---|
| **Media** | **Transfer** | **Transformation** |
| Water | Volatilization<br>Adsorption | Biodegradation<br>Photochemical degradation |
| Soil | Uptake by plants<br>Dissolution in rainwater | Biodegradation |
| Atmosphere | Washout by rain<br>Gravitational deposition | Oxidation by ozone |

13.1.3 Subsurface Environment

Civil engineers define soil as unconsolidated sediment. Others define it as the upper most covering mantel of material. Soil is formed with the weathering of parent rock or unconsolidated sediments as a result of the transport, deposition, and accumulation of this material. With the addition of geochemical processes, water movement, biological activity, and frost action this could cause further changes in soil.

Physical and chemical characteristics of soil varies with the location, depth, and time, depends primarily on the parent material, climate, and topography (LaGrega et al 2001).

Soil is a mixture of different inorganic and organic material. The inorganic portions consist mainly of fine grains subdivided into different sizes. The texture of the soil is classified by its percentages and based by its weight in gravel, sand, silt, and clay (LaGrega et al 2001).

Soil typically includes a considerable amount of organic material composing of mostly decomposing plant matter or humus. This organic matter acts as a stabilizer that binds the inorganic particles as aggregates (LaGrega et al 2001).

**Table 30 Average Concentration in Soil (LaGrega et al 2001)**

| Element | Average Concentration µg/kg |
|---|---|
| Arsenic | 6 |
| Cadmium | 10 |
| Nickel | 40 |
| Lead | 10 |
| Selenium | 0.2 |

Prevalent inorganic elements that are found in soil are soil silicon, aluminum, and iron with major contributions from both trace and micro elements. Many of the naturally occurring trace elements found in soils are hazardous (LaGrega et al 2001).

13.1.4 Potentially Exposed Populations

Exposure assessments determines those potentially populations that are exposed. Characteristics of the potentially expose populations include the following:

1. Present population in vicinity of the site
2. Future population in the vicinity of the site

3. Subpopulations of special concern (an example could be children exposed to lead poisonings)
4. Potential on-site workers during any remediation

(LaGrega et al 2001).

When determining the transport analysis of the chemicals, institutional controls such as, specific distances, can be identified to assess potentially exposed populations. For example, specific subpopulations may require special attention because of their higher toxicity level (LaGrega et al 2001).

13.1.4.1Exposure Use Scenarios

Once the potential population pathways have been determined, the characterization of the conditions under which the populations will be exposed is done. The exposure scenario involves both the evaluation of the current and future use of the site to establish a reliable set of conditions under which the exposure could occur (LaGrega et al 2001).

13.1.4.2 Worker Use Scenario

Is the site currently used for industrial activities? Are workers exposed to site related constituents under normal conditions? Could worked be exposed in the future, either because of a change in use of the site or because the workers would be involved in remedial activities (LaGrega et al 2001).

13.1.4.3 Trespasser Use Scenario

Is there evidence that trespassing may routinely occur at the site? Is there a fence that would limit access to the site? If so, is the fence in good condition? Have other measures been taken to limit access to the site (LaGrega et al 2001).

13.1.4.4 Residential Use Scenario

Is the site currently used for residential purposes? Will it or could it be used for residential use in the future? Are there any zoning or deed restrictions that would limit use for residential purpose? Are the residences single family dwellings? Is there the potential for residential use of groundwater (LaGrega et al 2001)?

13.1.4.5 Recreational Use Scenario

This is used to evaluate the potential risks associated with surface water bodies where people may swim, fish, and canoe for example (LaGrega et al 2001).

13.1.4.6 Construction Scenario

Are construction activities planned or likely at this site? Will the construction result in a potentially exposure for both on site receptors and off site populations (LaGrega et al 2001).

13.1.4.7 Soil Ingested By Adults

The daily uptake of soil of intentional ingestion for most people beyond the age of 6 is relatively low. However, the ingestion is through the use of fruits and vegetables. It is documented that most of these vegetables that contains most of the dirt are of the leafy variety. There have been investigations conducted at nuclear weapon sites that have revealed that particles that exceed 45μm are seldom retained on leaves. Moreover, the surface contamination on these vegetables and fruits of smaller particles are loss from the leaves through the rain or washing and the surface contamination exhibits little risk (Paustenbach 1989).

A study conducted by the EPA on growth of lettuce estimated that at high air concentrations (0.45 $mg/m^3$), the contribution of total dust contributes, and it is unlikely

that surface deposition alone can account for more than 0.6-1.5μg lead/g (2-5μg/g lead) on the surfaces of lettuce during a 21 day growing period. This data suggest that the daily ingestion of dust and dirt by adults is unlikely to exceed 0-5 mg/day. There is further evidence from the EPA with respect to lead that the worst case assumption of uptake of lead from vegetables is 100 μg/day (Paustenbach 1989).

13.1.4.8 Exposure From Dermal Contact

Quantitative estimates of dermal uptake of chemicals within dusts or soils contain more uncertainty than estimates from other entry routes of entry. The Centers for Disease Control's (CDC) estimate from a TCDD contaminated study assumed that the dermal exposure would follow "an age dependent pattern of deposition similar to soil ingestion". This investigation further provides the assumption that dirt would remain on the hand for a time period enough to bring about 1% absorption. This absorption percent determined in a study of rats exposed for 24 hours (Paustenbach 1989).

**Table 31 Soil Skin Concentrations (Paustenbach, D.1989)**

| Amount of Soil Deposited on Skin (CDC Assumption) | |
|---|---|
| **Age Group** | **Soil On Skin (g*day)** |
| 0-9 Months | 0 |
| 9-18 Months | 1 |
| 1.5-3.5 Years | 10 |
| 3.5-5 Years | 1 |
| 5-70 Years | 0.1 |

In an EPA risk assessment, an alternative assumption was utilized for dermal exposure. This new data was based on field investigations, which were more "realistic" than those proposed than the CDC's. It documents that about 0.5 mg of soils per $cm^2$ of skin adheres to a child's hand after playing in or around the home. Assuming values of 500 μg/kg and 2000 μg/kg for lead concentration in rural and urban house dust, this data

indicate dust uptake due to mouthing tendencies at about 100 mg/day if all the dust of both sides of the hands were ingested or absorbed through the skin (Paustenbach 1989).

13.1.4.9 Exposure From Inhalation

The EPA, CDC and other scientific organizations have concluded that the exposure through inhalation doesn't pose an adverse heath effect due to the inhalation of airborne chemicals. The degree of inhalation hazard is generally dictated by the volatility of the chemical, the distinct toxicity, the proximity of the population to the waste site, and the amount of dust generated at the site (Paustenbach 1989).

When actual field data is considered, inhalation will usually contribute slightly to total absorbed dose. This is in contrast with what has often been assumed in risk assessments. Site specific information should always be used whenever possible and most often is collected during the feasibility phase of the project (Paustenbach 1989).

14. Quantitative Risk Assessment

14.1 Risk

Risk, in the most general sense is defined as the probability of suffering harm or loss. When there is the convenience of risk to be measured, risk is calculated as the probability of an action occurring multiplied by the relentlessness of the harm if the action does occur (LaGrega et al 2001).

$$\text{Risk} = (\text{Probability}) * (\text{Severity of Consequences}) \quad (1)$$

**Equation 5 Risk**

In determining risk, there are three distinct types of risks that are defined: background risk, incremental risk, and total risk. Background risk is what people are exposed to in the lack of a particular source of risk being studied. Incremental risk is

what caused that source being studied and the total risk are both the background risk and incremental risk combined (LaGrega et al 2001)

In dealing with risk, the source of the specific risk must be determined. The source hazard is properly defined as the intrinsic capability of the waste to cause harm. This hazard contains various functions and variables such as mobility, toxicity, and the persistence of that source and how it is preserved. As a result, these variables and functions represent the release and or potential release that represent a hazard, but doesn't represent a risk unless exposure, such as childhood lead poisoning has occurred (LaGrega et al 2001).

The U.S. Academies of Sciences created a four stage procedure that EPA codified to employ a concept of a quantitative risk assessment. In dealing with this quantitative assessment, the uses of scientific principles are utilized to calculate the quantitative risk assessment. The most widely used method used by industry are:

1. Hazard Identification
2. Exposure Assessment
3. Toxicity Assessment
4. Risk Characteristics

(LaGrega et al 2001)

14.1.1 Hazard Identification

Risk assessments require a clear understanding of what chemicals are present at the site, their concentration and spatial distribution, and how they move in the environment from the site to the potential receptor point. This phase examines the data for all contaminants detected at a site and combines the data to stress the chemicals of concerns.

A site investigation can create a huge amount of data, and certain steps should be taken in the hazard identification stage to smooth the advancement of this process (LaGrega et al 2001).

**Table 32 Site Data Needs (LaGrega et al 2001)**

| Data Needs |
|---|
| Site History |
| Land Use |
| Contaminate Levels in Media: |
| Air, groundwater, surface water, soils, and sediments |
| Environmental characteristics affecting chemical fate and transport |
| Geologic |
| Hydrogeologic |
| Atmospheric |
| Topographic |
| Potentially affected population |
| Potential affected biota |

The surrogate chemicals are selected on the basis of which compounds best represents the risk posed by the site include:

- The most toxic, persistent, and mobile
- The most prevalent in terms of spatial distribution and concern
- Those involved in the more significant exposure

The list of surrogate chemicals should encompass those chemicals that are estimated to account for 99 percent of risk at the site. It should contain compounds that will support adequate evolution of both carcinogenetic and noncarcinogenetic risk (LaGrega et al 2001).

Initial Screening

1. Sort the contaminant data by media for both carcinogens and non carcinogens
2. Tabulate for each detected chemicals the mean and the range of concentration values observed at the site

3. Identify the reference concentrations for non carcinogens and slope factors for carcinogens for each potential exposure route
4. Determine the toxicity score for each chemical in each medium
5. For each exposure rout, rank the compounds by toxicity scores
6. For each exposure route, select those chemicals comprising 99 percents of the total score.

NONCARCINOGENS

$$TS = C_{MAX}/RfC \qquad (1)$$

**Equation 6 Toxicity Score NonCarcinogens**

where: TS = Toxicity Score

$C_{MAX}$ = Maximum Concentration

RfC = Chronic Reference Concentration

Carcinogens

$$TS = SF * C_{MAX} \qquad (2)$$

**Equation 7 Toxicity Score Carcinogens**

where: SF= Slope Factor

(LaGrega et al 2001)

14.1.2 Exposure Assessment

The second process of the risk assessment is the exposure assessment. The purpose of this step is to approximate the exposure to the chemicals by the populations potentially at risk. To provide an all-inclusive view of this process, a proper understanding of the causes of contamination and the spatial distribution of contaminants at the site is needed. Once the step is identified as to how the contaminants were releases, it is then necessary

to estimate how the contaminants migrate. Once the current and potential receptor points are identified, the attention the turns to:

1. Identification of general and sensitive populations of current and potential receptors
2. Estimation of both short and long term exposure in terms of doses by exposure route

(LaGrega et al 2001)

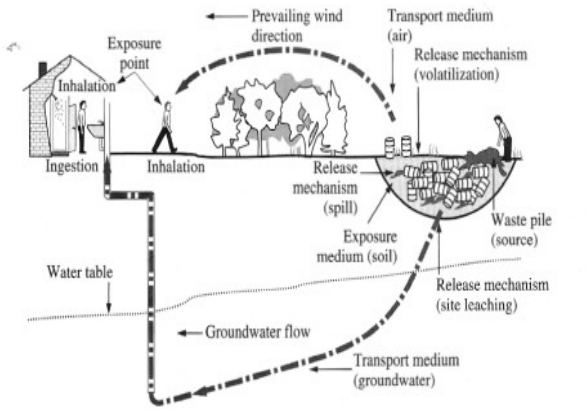



**Figure 35 Contaminant Release (LaGrega et al 2001)**

Environmental Pathways

In order for exposure to take place, an environmental pathway must be identified. That pathway determines the fate and transport analysis which includes:

- Source
- Chemical release mechanism
- Transport mechanism
- Transfer mechanism
- Transformation mechanism
- Exposure point
- Receptors
- Exposure routes

(LaGrega et al 2001)

14.1.3 Exposure Point Concentration

The Exposure Point Concentration is used to estimate the concentration of contaminants at the exposure points, pathways, and with respect to food. For exposures that are present, current monitoring data should be used when available. Site exposure point concentrations in soil and groundwater may be calculated as the arithmetic or geometric mean (depending on the statistical distributions of the site analytical concentration data) (LaGrega et al 2001).

Exposure Point

Exposure Points defines the location of the receptor for various scenarios. This information is identified for each exposure scenarios by applying the demographic information with the exposure pathways (LaGrega et al 2001).

Receptor Dose

The receptor dose is the final step in the exposure assessment phase. This dose is used to estimate the dose of the unique chemicals of concerns to which receptors are potentially exposed at the exposure points. With the receptor dose, the ingestion, inhalation, and dermal routes are determined with the administered dose, intake dose, and target dose. (LaGrega et al 2001).

14.1.4 Administered Dose

Chronic Daily Inhalation Intake

$$I = \frac{C * CR * EF * ED}{BW * AT} \qquad (3)$$

**Equation 8 Chronic Daily Inhalation Intake**

where:

I = Intake (mg/kg of body weight* day)

C = Concentration at exposure point (mg/L in water or mg/$m^3$ in air)

CR = Contact rate (L/day or $m^3$/day)

EF = Exposed frequency (days/year)

ED = Exposed Duration (Years); For residential exposures, a default value of ED = 30 years is typically used.

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

Average Daily Intake from Dermal Contact with Soil

$$I_N = \frac{C * A * DA * Abs * SM * ED}{BW * AT} \qquad (4)$$

**Equation 9 Average Daily Intake From Dermal Contact With Soil**

where:

$I_N$ = Intake

A = Skin Exposed = 20 % ($cm^2$)

DA = Dust Adherence = $0.51 \frac{mg}{cm2}$

Abs = Skin Absorption Rate 6%

SM = Effect of Soil Matrix = 15% (because of the soil matrix, only 15% of contamination is actually available for contact)

EF = Two Exposure events per day; 156 exposure days per year

ED = 1 Year

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

Administered Dose (Dust)

$$I = \frac{C * CR * EF * ED * RR * Abs}{BW * AT} \qquad (5)$$

**Equation 10 Administered Dose (Dust)**

RR = Retention rate (decimal fraction); A conservative approach would be 100% or 1.

Abs = Absorption into bloodstream; A conservative approach would be 100% or 1.

(LaGrega et al 2001)

Administered Dose (Air)

$$C = C_S * P_C \qquad (6)$$

**Equation 11 Administered Dose (Air)**

where:

$C_s$ = Concentration of chemical in fugitive dust (mg/mg)

$P_c$ = Concentration of fugitive dust in air ($mg/m^3$)

(LaGrega et al 2001)

14.2 Toxicity Assessment

The toxicity assessment defines the toxicity for each chemical of concern. The ultimate objective is to identify those substances that might injure humans who may come into contact with chemicals and prevent injury. In the most fundamentalist sense,

the concept of toxicity is that it establishes a relationship between the dose of an agent and the response that is produced in a mammalian system. There are three primary components of the toxicity assessment. First, the magnitude of the biological response is a function of the concentration of the agent at the site of action. Secondly, the concentration of the site of action is related in some expected and describable manner with the administered dose. And finally, the dose and repose are casually related (Paustenbach 1989).

Once the toxicity assessment is completed the qualifying risk as it applies to humans and chemicals are classified as carcinogenic and non carcinogenic. In some cases, some fall into the category of both carcinogenic and non carcinogenic (LaGrega et al 2001).

14.2.1 Sources of Toxicity Data

In conducting risk assessment it is common to use existing data found in standard sources of toxicological data, in addition to and from select appropriate mathematical descriptors of toxicity. Sources of data include:

1. Integrated Risk Information System (IRIS) www.epa.gov/iris
2. The Agency for Toxic Substances and Disease Registry (ATSDR) http://www.atsdr.cdc.gov/index.html
3. Toxics Release Inventory (TRI) http://www.epa.gov/tri/

Furthermore, there are specific databases that include pertinent information such as:

1. Health Effects Assessment Summary Table (HEAST)
    - Includes interim RfD and CPF values

- Prepared by the EPA's Environmental Criterion and Assessment Office (ECAO)

2. Toxicological profile prepared by US Agency for Toxic Substances and Disease Registry (ATSDR)
3. The International Programme on Chemical Safety (IPCS) Environmental Health Criterion documents published by the World Health Organization (WHO), Geneva, Switzerland
4. The Joint FAO/WHO Meeting on Pesticide Residues (JMPR) published by Food and Agriculture Organization (FAO), Rome, Italy

(LaGrega et al 2001).

14.3 Risk Characterization

The final process of the risk assessment is to estimate the risk. This is done by calculating the estimates of the carcinogenic and non carcinogenic risks. These risks are receptors for all exposure routes and for the maximum exposed individual in addition to the most probable exposed population (LaGrega et al 2001).

. When performing risk calculations, it is important to determine the average and minimum calculation, but also the range of the potential risk which can then be used to provide useful information regarding the potential hazards associated with a particular set of exposure conditions (LaGrega et al 2001).

In general, calculations of potential risk by using an average concentration permits a better estimate of risk associated with chronic exposure. If using a maximum value, the best is the estimation of short-term, subs chronic risks (LaGrega et al 2001).

Carcinogenetic Risk

Carcinogenetic Risk is defined as the chronic daily intake dose multiplied by the carcinogenetic slope factor. This is the probability of excess lifetime cancer from exposure to this chemical

$$\text{Risk} = I_c * SF \qquad (7)$$

**Equation 12 Risk**

where:

$I_c$ = Chronic daily intake of carcinogen $\frac{mg}{kg * day}$

SF = Carcinogenetic slope factor $\frac{kg * day}{mg}$

(LaGrega et al 2001)

**NON CARCINOGENETIC RISK**

Non Carcinogenetic Risk is characterized in terms of a hazard index. This hazard index is the ratio of the estimated intake dose from exposure to the reference concentration.

$$HI = \frac{In}{RfC} \qquad (8)$$

**Equation 13 Hazard Index**

where:

HI = Hazard Index (dimensionless)

$I_N$ = Chronic daily intake of NonCarcinogen $\frac{mg}{kg * day}$

RfC = Reference Concentration $\frac{mg}{kg * day}$

If the acceptable level of intake is equal to the reference dose, then a hazard index less than 1 is acceptable.

(LaGrega et al 2001)

**Calculating Procedure for Assessment of NonCarcinogenetic Risk**

1. Identify discrete exposure conditions
    - Exposure route
    - Frequency
    - Duration
    - Administered Dose
2. Derive appropriate RfD for each discrete set of conditions
3. Evaluate hazard for effects as a ratio of exposure dose to the recommended RfD
4. Aggregate hazard for multiple chemical agents and exposure pathways as a hazard index, where appropriate

(LaGrega et al 2001)

14.3.1 Uncertainties

In conducting risk assessments, it is widely known that there are going to be inherent uncertainties in each of the four steps. Within this inherent uncertainty, the uncertainty should be discussed and explained thoroughly. The computation of these risks can best be described as applied probability of extremely rare events. It isn't possible to specify every conceivable outcome, and credible worst case scenario, which can produce an inherent conservatism that often results is assessing different scenarios that may never be produced (LaGrega et al 2001).

With this methodology, the goal is to protect public health by ensuring that the risks are not understated. Even with the most conservation methods employed, uncertainties still exists. An example of this can be to underestimate of the risk of exposures in dealing with complex mixtures of toxic substances. Another example could be the underestimating of actual risk that is the present of sensitive subpopulations (LaGrega et al 2001).

14.3.2 Monte Carlo Simulation

Mathematically techniques and simulations can be utilized to assess both uncertainty and the sensitivity of final answers to individual input parameters when estimating exposures. Monte Carlo's overall function is a simulation procedure that utilizes the capability of replacing point estimates with random variable drawn from probability distribution functions. The purpose of the Monte Carlo simulation is to run the simulations numerous times and as each run is determined, the output produces new vales of the random variables thus, producing a new risk. In conducting this quantitative method, the calculations can then be summarized into a histogram of the specific risk values to determine a specific value to use (LaGrega et al 2001).

14.3.3 Monte Carlo Method Application to Lead

Currently, Environmental Protection Agency (EPA) has not determined a threshold limit for lead (http://www.epa.gov/iris/subst/0277.htm). This threshold limit is vital to running any risk assessment. For this reason, this study will include the development of a systematic methodology using other threshold limits such as Proposed Maximum Concentration Limits (MCLs), Proposed Maximum Concentration Limit Guidelines (MCLGs), and Water Quality Criteria for Fish and Drinking Water in

determining the exposure and assessment of risk. With the absence of a defined threshold limit, a probabilistic approach such as the Monte Carlo Method can be used in determining estimates when a range of possible threshold values are to be used (Lorenzana et al 2003).

Monte Carlo method can be applied to scenario-specific data which results in different probability distributions being utilized with different variables such as the various migration paths of contaminants of air, dust, soil, and water (Lorenzana et al 2003). An example of running this method is parameters that are fixed with one parameter varying. This analysis can then display the effect of a range of set values on that varying parameter, producing different outcomes of the simulated model. In addition, the simulation can illustrate the need to collect any additional information that may be omitted (Loyd 2006).

14.3.4 Monte Carlo Simulation Process

This simulation procedure can facilitate the replacing of point estimates with random variables drawn from probability distribution functions when there is pertinent information missing or not defined. The model is ran repeatedly with the outputs of each run saved. The output of each run can then be used to determine expected values in addition to low and high end risks presenting the probability of incidences (Loyd 2006). This allows a set of sample results that can be displayed in a frequency output as opposed to one single risk estimate drawn from a defined sample size.

In addition, the Monte Carlo method characterizes uncertainties within the quantitative risk assessments. In conducting these quantitative risk assessments, there are innate and deficient uncertainties present in each of the four procedures. Hazard

Identification is based on data in which the detection, identification, and quantification limits could introduce errors. Exposure Assessment consists of fate and transport models that rely heavily on adjusted models to coincide with site specific situations which can be complicated in determining. Toxicity Assessment, especially in this investigation, has a high rate of uncertainty because of the missing threshold limits of the slope factors and reference doses. Finally, the Risk Characteristic procedure collects the previous three phases including collective individual uncertainties (LaGrega et al 1994). As a result, the interpretation of the final results will already include their built in uncertainties, thus generating a more practical output. Please note that the reader should be aware that this is not a study of the Monte Carlo method. Monte Carlo method is only used to assist in one aspect of this study. Please refer to Herman (1957) or Hammersley (1964) for further study in this area.

14.3.5 Probable Carcinogen To Humans

According to the National Cancer Society and the International Agency for Research on Cancer (IARC), Lead falls into Group 2A, Probable Carcinogens To Humans. This category is used for agents, mixtures, and exposure circumstances for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals.

14.3.6 Dose Response Relationship for Carcinogens

In determining the dose response for carcinogens, two characteristics must first be established:

1. Is the tested chemical a carcinogen?
2. How are data from experimental animals applied to humans?

When assessing a lifetime cancer risk to humans, the exposure to a single molecule of a genotoxic carcinogen could result in one of the two mutations to initiate cancer. As a result, the dose response is asymptotic to zero incidences. For the reason above, there is not an acceptable or safe level of cancer (LaGrega et al 2001).

**Table 33 Known and Probable Carcinogens**

**(http://www.cancer.org/docroot/PED/content/PED_1_3x_Known_and_Probable_Carcinogens.asp#known)**

| | |
|---|---|
| Acrylamide | Human papillomavirus type 33 |
| Adriamycin | Indium phosphide |
| Androgenic (anabolic) steroids | IQ (2-Amino-3-methylimidazo[4,5-f]quinoline) |
| Aristolochic acids (naturally occurring mixtures of) | Kaposi's sarcoma herpesvirus/human herpesvirus 8 (KSHV/HHV-8) |
| Azacitidine | Lead compounds, inorganic |
| Benz[a]anthracene | 5-Methoxypsoralen |
| Benzidine-based dyes | 4,4´-Methylene bis(2-chloroaniline) (MOCA) |
| Benzo[a]pyrene | Methyl methanesulfonate |
| Bischloroethyl nitrosourea (BCNU) | N-Methyl-N´-nitro-N-nitrosoguanidine (MNNG) |
| 1,3-Butadiene | N-Methyl-N-nitrosourea |
| Captafol | Nitrogen mustard |
| Chloramphenicol | N-Nitrosodiethylamine |
| a-Chlorinated toluenes (benzal chloride, benzotrichloride, benzyl chloride) and benzoyl chloride (combined exposures) | N-Nitrosodimethylamine |
| 1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU) | Phenacetin |
| 4-Chloro-ortho-toluidine | Procarbazine hydrochloride |
| Chlorozotocin | Styrene-7,8-oxide |
| Cisplatin | Teniposide |
| Clonorchis sinensis (infection with) | Tetrachloroethylene |
| Dibenz[a,h]anthracene | ortho-Toluidine |
| Diethyl sulfate | Trichloroethylene |
| Dimethylcarbamoyl chloride | 1,2,3-Trichloropropane |
| 1,2-Dimethylhydrazine | Tris(2,3-dibromopropyl) phosphate |
| Dimethyl sulfate | Ultraviolet radiation A |
| Epichlorohydrin | Ultraviolet radiation B |
| Ethylene dibromide | Ultraviolet radiation C |
| N-Ethyl-N-nitrosourea | Vinyl bromide |
| Etoposide | Vinyl fluoride |
| Glycidol | Vinyl Chloride |
| Human papillomavirus type 31 | |

**Table 34 Known and Probable Carcinogens**

**(http://www.cancer.org/docroot/PED/content/PED_1_3x_Known_and_Probable_Carcinogens.asp#known).**

| Exposure Circumstances | Mixtures |
|---|---|
| Aluminum production | Alcoholic beverages |
| Arsenic in drinking water | Analgesic mixtures containing phenacetin |
| Auramine, manufacture of | Areca nut |
| Boot and shoe manufacture and repair | Betel quid with tobacco |
| Coal gasification | Betel quid without tobacco |
| Coke production | Coal-tar pitches |
| Furniture and cabinet making | Coal-tars |
| Hematite mining (underground) with exposure to radon | Mineral oils, untreated and mildly treated |
| Involuntary smoking | Salted fish (Chinese-style) |
| Iron and steel founding | Shale-oils |
| Isopropanol manufacture (strong-acid process) | Soots |
| Magenta, manufacture of | Tobacco products, smokeless |
| Painter (occupational exposure as a) | Wood dust |
| Rubber industry | |
| Strong inorganic acid mists containing sulfuric acid (occupational exposure to) | |
| Tobacco smoking | |

14.3.6 Dose Response Relationship for Carcinogens

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14.3.7 Slope Factor and Carcinogen Potency Factor

The Slope Factor is the slope of the dose response curve at very low exposures was formerly termed Carcinogen Potency Factor (CPF). The SF is the 95 percent upper

confidence limit of the slope dose response curve at very low exposures. The SF is expressed as the inverse of the daily dose $\frac{mg}{kg * day}$ (LaGrega et al 2001).

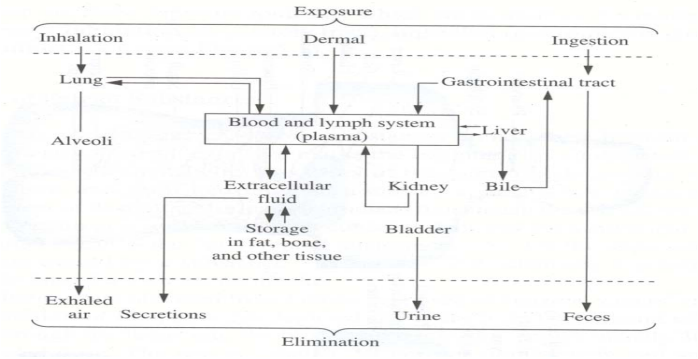


**Figure 36 Overview of Absorption, Distribution, Storage, Transformation, and Eliminations (LaGrega et al 2001)**

15. Toxicology

Most toxic agents do not cause harm at the entry points. Instead, this exposure marks the beginning of the metabolic process of the human body to interact, absorb, distribute, store, transform, and eliminate a substance. In order for the chemical agent to flourish in affecting a target organ, the chemical agent or its biotransformation product must reach that critical site at a suitable high concentration and for a sufficient length of time (LaGrega 2001).

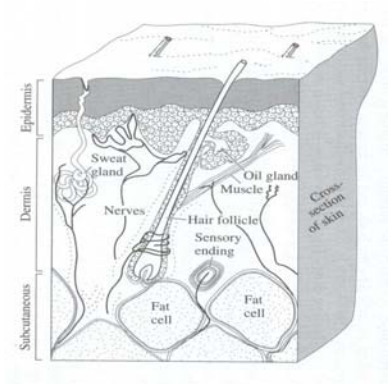


**Figure 37 Exposure Routes for Chemical Agents in Hazardous Waste (LaGrega et al 2001)**

15.1 Absorption

Absorption is the transport across any body barrier such as the skin, lungs, cells, and the lining if the gastrointestinal tract. A toxic chemical can enter the body by inhalation, ingestion, or dermal contact.

If the toxic agent is inhaled, the toxic agent must pass through the thin lining of cells covering the inside of the alveoli in the lungs. This connects the blood and air which may carry gaseous toxicants. If the toxic agent is passed through dermally, the agent must pass through the stratified layer of the skin. Although the administered dose is critical, the amount of toxic chemical absorbed through the body and the amount reaching the target organ is far more important (LaGrega 2001).

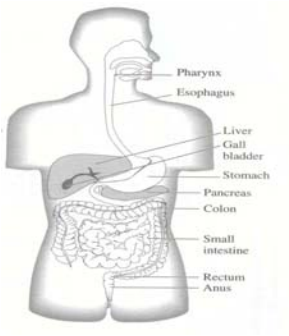


**Figure 38 Exposure Routes for Chemical Agents in Hazardous Waste (LaGrega et al 2001)**

15.1.1 Absorption via the ingestion route

Absorption of the toxic substance may occur along the entire length of the gastrointestinal tract, but will differ in the stomach compared to the intestine due to the lower pH in the stomach. There are an enormous amount of factors may alter gastrointestinal absorption (LaGrega 2001).

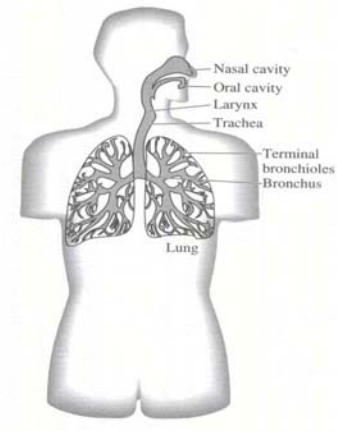


**Figure 39 Exposure Routes for Chemical Agents in Hazardous Waste (LaGrega et al 2001)**

15.1.2 Absorption via the inhalation route

In the gaseous state, the inhaled toxicant is transferred into the liquid layer lining the airway wall by diffusion. Capillary blood flow removes the dissolved gas on the other side of the mucous and separating tissue layers. For obtainable soluble gases,

uptake is linearly related to solubility. The tissue gas partitions coefficient linearly correlates with its fat gas and blood gas partition coefficients, providing an approach for estimating these parameters and blood gas partition coefficients, providing an approach for estimating these parameters. The relation between absorption in the lungs and air concentrations may be nonlinear as is the case for a poorly soluble gas (LaGrega 2001).

15.2 Distribution

Few toxic agents attack at the point of entry. They systematically rely on blood flow to reach other organs and tissue. Many factors influence the distribution of the agents. These factors include absorption, perfusion, exposure route, and tissue affinity. As a result, these toxic agents allocate partially and unequally to multiple compartments of the body as opposed to one or equally distributed to all. Perfusion is important in this stage because its use in the movement of blood through an organ tissue. Examples that are connected in this specific study are related to the liver and brain. The liver is well perfused which results in its total potential uptake as high. Equally, the brain, which is also well perfused, but its intake isn't high because it is protected by the blood-brain barrier (LaGrega 2001).

The point of absorption, which can be partly determined by the exposure route, is influenced by the distribution. One point of absorption may allow the toxicant to bypass the liver which is the body's primary detoxification site. An example given by *LaGrega*, indicates that toxicants absorbed through the lungs, skin, mouth, and esouphogus may temporarily bypass the liver, while those absorbed through the stomach and intestines will follow the bloods direct path to the liver (LaGrega 2001).

15.3 Storage

Many substances may amass at sites other than the target organs which can then allow the toxicant to be released over an extended period of time. The concentrations at these storage sites can be high. Depending on the chemical structure and division of the toxicant, determines into the amount of toxicant that is being stored. Some examples of storage sites include the following:

- Fat for nonpolar (lipophilic) compounds
- Blood for plasma for compounds bound by blood proteins
- Bone for lead, radium, and fluoride
- Kidneys for cadmium
- Thyroid glands for iodine

(LaGrega 2001)

16. EXPOSURE PERIOD

The period in which a dose is administered is important when determining the exposure. When an acute toxic dose is fractionalized into smaller portions and administered over a longer period of time, the toxic effect usually decreases (LaGrega 2001).

**Table 35 Exposure Period (LaGrega et al 2001)**

| Exposure Period | |
|---|---|
| Acute | One Day |
| Sub acute | Ten Days |
| Sub chronic | Two Weeks-Seven Years |
| Chronic | Seven Years-Lifetime |

16.1 Environmental Exposure Estimate

One of the most difficult issues in dealing with risk assessments is the degree of exposure to chemicals. This issue can be solved by modeling and or taking sampling from the source of concern. The model approach is more beneficial when determining possible future threats and concerns to exposure pathways. The distribution of a chemical into the environment varies and as a result, when coupled with its physical properties, can predict its behavior.

16.2 Human Exposure Estimation

The primary routes of exposure to chemicals in the environment are inhalation of dusts, vapors, dermal contact with contaminated soils of dusts, and ingestion of contaminated foods, water, or soil dust.

16.2.1 Uptake via Inhalation

To estimate the amount of a chemical absorbed by humans through inhalation, the following parameter must be measured or estimated:

1. Contaminant concentration in air (gas, vapors, or particulates)
2. Particle size distribution (for chemicals adsorbed onto particles)
3. Contaminant concentration in dust (may vary with particle size)
4. Respiration rate
5. Degree of pulmonary absorption (bioavailability)
6. Duration of exposure

(Paustenbach 1989)

16.2.2 Dermal Uptake

To estimate dermal exposure to contaminated soils, dusts, or liquids, and the subsequent absorption of the chemical contaminant, the following parameter need to be known or estimated:

1. Contaminant concentration in soil or dust
2. Soil dust deposition rate from air and from direct soil contact
3. Area of exposed skin
4. Dermal absorption coefficient (bioavailability)
5. Duration of exposure

16.2.3 Uptake Due to Ingestion

The risks associated with the ingestion of chemical contaminants are dictated by the following parameters:

1. Amount of contaminated medium ingested per day (soil, food, and liquids)
2. Contaminant concentration in each medium
3. Gastrointestinal absorption coefficient (bioavailability)

16.3 Classification of Toxins Actions and Effects

Toxic responses are manifested in behavioral and physiological terms and can range from headaches and nausea to convulsion and death (LaGrega 2001).

16.3.1 Classification by End Point

A common method to classify the toxic action of a chemical substance is by its end point. These endpoints are determined by their carcinogenic and non carcinogenic effects. In this manner, carcinogenic effects are defined with tumor induction as an end point and non carcinogenic effects comprising of all other effects (LaGrega 2001).

**Table 36 Toxic Effects of Lead (LaGrega et al 2001)**

| Toxic Effects of Lead | | |
|---|---|---|
| Toxic Substance | Carcinogenetic Effects | NonCarcinogenetic Effects |
| Lead | Kidney Tumor (in test animals) | Reduced birth weight, anemia, increase blood pressure, brain and kidney damage, IQ impaired, decreased learning |

16.3.2 Classification by Target Organ

In some instances, a specific target organ may be distinguishes and used as a basis for categorization purposes. For example, the bone marrow for benzene, the brain for methyl mercury, the liver for carbon tetrachloride, the lung for pesticide paraquat, the eye for the antimalarial drug chloroquine and kidney in case of cadmium(LaGrega 2001).

In some cases, it might not be easy to determine exactly what single toxins that target an organ. Several substances induce generalized symptoms of intoxication, where nausea with abdominal distress. Loss of appetite, headache, and drowsiness may be the only obvious symptom (LaGrega 2001).

16.4 Toxicological effect can be classifies as the following:

- Immediate versus delayed effects
- Irreversible versus reversible effects
- Local versus systematic effects

(LaGrega 2001)

16.4.1 Classification by Immediate Versus Delayed Effects

As discussed previously, toxicity action levels are characterized by either acute or chronic. However, acute or immediate toxicity as it relates to toxicity, results shortly

after an exposure, while a delayed toxic reaction have only latency periods (LaGrega 2001).

16.4.2 Classification by Irreversible Versus Reversible Effects

The process of involved between the exposure and reaching the target organ is usually irreversible. Depending on the organ and the toxin, depends on whether reversible and irreversible takes place. For example, skin and liver have a high repair capacity; therefore moderate damage induced at these sites is often irreversible. Injuries of the central nervous system caused by chemical are, on the other hand, mostly irreversible because of the slow or nonexistent regeneration power of these tissues (LaGrega 2001).

16.4.3 Classification by Local versus Systematic Effects

In the study of toxicology, there is a distinct difference between local toxins and systemic actions. Systemic poisons can exert their toxins at a point distant from the site of absorption where reactive chemicals cause toxic effects immediately when coming into contact with tissue (LaGrega 2001).

17. Dose Response Relationship

The dose of the toxins relies heavily on to the extent of damage the toxin will induce. As a result, a relationship has to develop as to the greater the dose the more severe the response. The susceptibility of an exposed individual relies on many variables such as age, sex, diet, genetics, health status prior exposure to agent, and exposure to other agents. Additionally, other variations in these variables can be utilized as well (LaGrega 2001).

17.1 Nature of Dose Response Relationship

A correlation exists between a dose and a specified frequency of a toxic end point. Specifically, the incidence of a specific toxic end point among individuals of a population is recorded as a function of dose. In any given population of living organisms that is exposed to an increasing amount of toxic compounds, the typical population will undergo various exposures of negative effects. With low doses, the population will experiences no deaths, a few deaths as the dose increases, and more deaths with higher doses until of the entire population is dead (LaGrega 2001).

Most plots of log dose versus cumulative mortality will display the nonlinear S curve which is referred to as median lethal dose ($LD_{50}$). This dose is at the 50 percent mark of the organisms that remain alive. If the dose is inhaled, it is referred to as the median lethal concentration ($LC_{50}$). The median lethal dose ($LD_{50}$) is expressed as milligrams per kilogram body weight while the median lethal concentration ($LC_{50}$) is expressed as the concentration of the substance present in a volume of inhaled air (LaGrega 2001).

17.2 Dose Effect Relationship

The Dose Effect Relationship is established in that a toxin is capable of causing adverse health effects at a dose significantly below the lethal level. This is demonstrated if the intensity level of the dose in an individual is plotted as a function of the effect of that dose (LaGrega 2001).

**NONCARCINOGENS**

The distinction between carcinogens and noncarcinogens is that noncarcinogens do not cause tumors. They include all of the toxicological responses. The most prevalent

and most toxic noncarcinogens response are those in which an agent effects the enzymes (LaGrega 2001).

**THRESHOLD**

In referring to a threshold of noncarcinogens, the toxicological end points, the dose effect, or dose response relationship is characterized by a threshold below which no effects can be observed on the cellular, subcellular, or molecular level. The damage of enzymes will have little effect on overall performance. It is only when a significant fraction of the targets have been eliminated by toxin actions above a certain threshold for the target dose, will a toxic affect occur (LaGrega 2001).

17.2.1 No Observed Adverse Effect Level (NOAEL)

The threshold value for a toxic substance can not be precisely identified. However, there are other methods such just of epidemiological data and animal tests that can be utilized. The concern is only in the toxicological significant effects, a variant of the NOAEL is utilized. The two other terms are:

- **Lowest Observed Effect Level (LOEL)** - The lowest dose tested for which effects were expressed; typically used when an effect is expressed at all dose
- **Lowest Observed Adverse Effect Level (LOAEL)** – A stricter version of LOEL to address only adverse effects

(LaGrega 2001)

17.2.2 Acceptable Daily Intake (ADI) and Reference Dose (RfD)

Acceptable Daily Intake (ADI) is used to represent the level of daily intake of a particular substance which should not produce and adverse health effect. The ADI are based on NOEL's and should not be construed as a strict physiological threshold that

when exceeded will result in adverse health effects. ADI's include safety factors to reflect the susceptibility in the human population and other uncertainties. An ADI is much small than the theoretical threshold and that is why toxicologist use the ADI instead of the threshold value (LaGrega 2001).

17.2.3 Reference Dose (RfD)

The Reference Dose (RfD) is the contemporary surrogate used by the EPA instead of the ADI. The development of an RfD follows a somewhat stricter procedure that used for an ADI, sometimes resulting in lower values for acceptable intake (LaGrega 2001).

**DERIVATION OF RFD**

It is common that chemical of concerns that fall in the realm of noncarcinogens frequently do not have a publishable or standard toxicological indices. In those instances, there are different methods to still perform the risk assessment. Those methods are listed below:

1. Use surrogate compounds with similar toxic activities and published indices
2. If no surrogate exists for noncarcinogenic chemicals, seek out other options of which are readily available. One method is employed this utilizes a rodent study using a No Observed Adverse Effect Level (NOAEL) has been determined and then dividing by a 100 fold safety factor to obtain an RfC

(LaGrega 2001)

**EPA SAFETY FACTORS**

The public health is the priority of the EPA when developing different standards and procedures. In establishing the reference dose and the carcinogen slope factors several safety factors are built in. The goal is to protect and ensure that risks are

overestimated rather than underestimated. Examples of the protective approach are as follows:

- For noncarcinogens, extrapolation of animal reference dose to humans utilizes at least two safety factors: one for animal-to-human extrapolation, and a second variation for toxic sensitiveness within the human population
- For carcinogens, the linearized multistage model assumes the upper bound 95 percent confidence level of extrapolated data
- For carcinogens, the linearized multistage model extrapolates data from the 10 to 90 percent carcinogens range observed in experimental animals to the regulatory target of 0.0001 percent carcinogenesis, a step that could overstate risk by several orders of magnitude
- Although evidence indicated that, like non carcinogens, nongenotoxic carcinogens have threshold below which they tail to influence cellular differentiation or division, they are treated mathematically like genotoxic carcinogens according to the linearized multistage dose response model

**DERIVATION OF RFD**

As stated previously, the RfD is a surrogate to the ADI. The development of the RfD is a stringent procedure than the ADI. To develop a RFD, certain standards must be followed:

1. Select the most sensitive species for which adequate studies are available.
2. Select the principle of critical studies using the appropriate route of exposure
3. Select supporting studies. Investigations from a wide variety of sources may prove additional aid in interpreting the results from the critical studies

4. Identify the NOEL, or if such data is not available, the LOAEL for the most sensitive end point
5. The NOAEL for the most sensitive point is adjusted downwards by order of magnitude to reflect uncertainty. These magnitudes are listed as such:
    - Reduce the NOAEL found in humans by an uncertainty factor of 10 to account for variations in the general population, thus protecting the most sensitive populations.
    - Reduce the NOAEL by an additional uncertainty factor of 10 when extrapolationing from animals to humans
    - Reduce the NOAEL by an additional uncertainty factory of 20 if the data are derived from a sub chronic instead of a chronic study
    - If the test data do not show a NOAEL, the LOAEL is selected and reduced by an additional factor of 10 to account for the uncertainty introduced by extrapolation

In conducting these derivations, the EPA supplies a "modifying factor" that ranges from 1 to 10, to reflect a qualitative professional judgment if uncertainties which are not accounted for in the other uncertainty factors.

18. Environmental Engineering Controls Measures

18.1 Biological Methods of Treatability applications

18.1.1 Slurry Phase Treatment – Ex Situ Systems

Slurry-phase systems involve the treatment of contaminated soils and or sludge's mixed with clean or contaminated liquids. This treatment technology is in its greatest use

as a potential use for biodegrading soils where difficult to treat soils such as heavy oils and PAH's.

18.1.2 Biological Treatment Systems – In Situ

Remediation can be accelerated by various in situ technologies for enhancing, stimulation and managing the actions of subsurface microbiological communities (LaGrega et al 2001). This major difference in remediation time extinguishes it from natural biodegradations which takes a longer amount of time

18.1.3 Xenobiotic Compounds Amenable to Biological Treatment

Some of the most important uses of microorganism to treat metal and inorganics include:

- Changing the valence state of metals, thus reducing the toxicity and or solubility
- Removing heavy metal and radionuclide from water adsorption
- Detoxifying cyanide
- Removing excess nitrogen compounds (ammonia and nitrate) from soils/groundwater through nitrification
- Changing the structure and properties of certain metals through metheylation (LaGrega et al 2001)

By utilizing indigenous microorganisms to modify the subsurface environmental for metals, treatment generates new dimensions to biological treatments process. Examples, give by *LaGrega* (et al 2001) present how microorganisms can be used to change the valence state of metals. Hexavalaent Chromium, which is a carcinogenetic, can be reduced to trivalent Chromium, which is a benign form of the metal. Uranium in batch

experiments has been reduced to highly insoluble uraninite following thee addition of ethanol as a carbon source and trimetaphosphate as an inorganic nutrient. Other examples include a sulfate-reducing bacteria that can be used to convert dissolved sulfate to sulfide and precipitating metals out as insoluble metals sulfides in the process. This treatment mechanics is potentially suitable for use with cobalt, cadmium, nickel, lead, and zinc (LaGrega et al 2001).

18.2 Phytoremediation

Phytoremediation is the use of plants to assist either direct or indirectly, in the attenuation of hazards contaminates present in soil. Depending on the topography, geographic location, site and contaminant suitability for phytoremediation, and type of contaminant the specific phytoremediation process is utilized. The basis of the process is that it takes on a multi-disciplinary approach involving ecotoxicity, soil microbiology, soil chemistry, and botany (LaGrega et al 2001).

These plants and their associated rhisosperic microorganisms remove, degrade, or contain chemical contaminants located in the soil, sediments, groundwater, surface water, and atmosphere (Chappell 1997). To date phytoremediation is partially effective in the cleanup of metals, pesticides, solvents, explosives, explosives, crude oils, polycyclic aromatic hydrocarbons, and landfills at certain sites (LaGrega et al 2001).

Phytoremediation is the most applicable for treatment of the vadose zone soil. It can however, be used for the treatment of shallow and saturated zone soil depending on the plant or tree root depth. Certain plants possess unique properties that can be used to reduce the toxicity or waste mobility waste constitutes. Plant selection is based on the capacity to draw constituents of concern up into plant stems and leaves, and ultimately

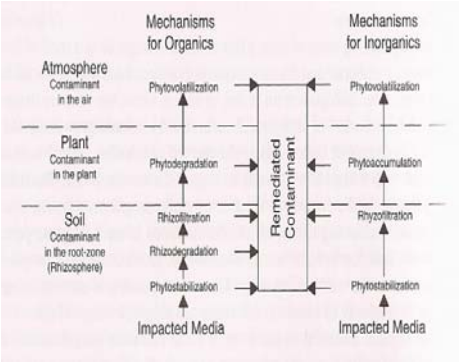


**Figure 40 Impacted Media (Suthersan 2002)**

harvested and removed them from the site. Phytoremediation is a cost effective way to remediate hazardous contaminants. (LaGrega et al 2001).

## ADVANTAGES

Advantages of phytoremediation included the fact that they can be applied to both in situ and ex situ methods. The ex situ method is used more because this method diminishes the surrounding environment and lessens the dispersion of contaminants throughout the environment. Another advantage is that both organic and inorganic compounds can be remediated through this process (Henry 2000).

**Table 37 Advantages and Disadvantages of the Phytoremediation Process (Henry, J. 2000)**

| Advantages | Disadvantages |
|---|---|
| Amendable to a variety of organic and inorganic compounds | Restricted to sites with shallow contaminants within rooting zone of remediated plants |
| In Situ / Ex Situ Applications | May take up to several years to remediate a contaminated site |
| In Situ applications decrease the amount of soil disturbance compared to conventional methods | Restricted to sites with low contaminant concentrations |
| Reduce the amount of waste to be landfilled (up to 95%) | Harvested plant biomass from phtoextraction may be classified as a RCRA hazardous waste |
| In Situ application decreases the spreading of contaminants via air and water | Climate Conditions |
| Does not require expensive equipment or highly specialized personnel | Introduction of non-native species may affect biodiversity |
| Easy to implement and maintain | Consumption of contaminated plant tissue is also of concern |
| Low cost compared to conventional treatment methods | |
| Environmentally friendly | |

**DISADVANTAGES**

A disadvantage of phytoremediation is the use of invasive or non-native species that can affect the biodiversity. The remediation process is restricted to the rooting depth of the plant. This concept must be keep in mind when dealing with this type of method since some remediation methods take several years to take effect and still may not be fully remediated. Additionally, once the remediation process has taken place and produced biomass, this newly produced waste can be classified as hazardous waste and may be subject to Resource Conservation and Recovery Act (RCRA) regulations of managing and removal (Henry 2000).

Within phytoremediation, there are sub-categories that are useful for the treatment and remediation. These include:

The primary remediation technologies currently being used are:

- Phytoaccumulation, Phytoaccumulation, Hyperaccumulators
- Phytostabilization
- Phytodegradation or Phytotransformation
- Phytovolatilization
- Rhizodegradation, phytostimulation or plant assisted bioremediation
- Rhizofiltration or contaminant uptake

18.2.1 Phytoaccumulation

Phytoaccumulation is the radiation of contaminated soils using non-food crops. This is also referred to as phytoextraction (Suthersan 2002). Phytoaccumulation is the uptake and translocation of metal contaminates from the soil into the plant matter via plant roots into the above ground portions of the plants (LaGrega et al 2001). Certain

hyperaccumulators methods of uptake have considerable amounts of metals in contrast to other plants and the ambient concentration. The uptake should be metal specific which allows the risk of impoverishing the surrounding soil. In addition to the metal specificity, a high transport of metals from the roots to the shoots should take place for this method to be effective during remediation treatment (Suthersan 2002). The plants act as a filter or traps for the metals and remove them from contaminated soils. Once these plants have performed their function, they are harvested and incinerated with waste ash at hazardous waste landfills (LaGrega et al 2001).

18.2.2 Phytostabilization

Phytostabilization requires the use of certain plant species to immobilize contaminants in the soil and groundwater through absorption and accumulation by roots, adsorption onto roots, or precipitation within the root zone and physical stabilization of soils (Suthersan 2002). This process allows the specific plant species to interact with the contaminants that retard the rate of contaminants leaching to the groundwater. Chemical processes, sorption, or the reduction of rainfall infiltration through increased evaporation assists in the phytostabilization to take full affect (LaGrega et al 2001). This method also stabilizes contaminated soil be decreasing wind and water erosion and decreases water infiltration and the subsequent leaching of contaminants. This method also reduces the mobility of the contaminants and prevents migration to the groundwater or air. In addition, it can be used to reestablish a vegetative cover at sites where natural vegetation is lacking due to high metal concentrations (Suthersan 2002). The only known disadvantage to phytostabilization, is that it requires the use of additional nutrients such as lime and or phosphate to the soil (LaGrega et al 2001).

18.2.3 Phytodegradation

Phytodegradation or phytotransformation as it is often referred to is the breakdown of contaminants taken up by plants through metabolic process within the plant or the breakdown of contaminants external to the plant through the effect of compounds, such as enzymes, are produced by the plants. The pollutants are degraded, used as nutrients and then incorporated into plant tissue. There are some cases when the metabolic intermediate or end products are released to the environment depending on the contaminant and plant species.

Phytodegradation and Phytotransformation can be summarized by the following:

- Plants form enzymes that degrade organic contaminants are isolated and metabolic pathways can be predicted
- Phytodegradation can be used for the treatment of soil, sediments, sludge, and groundwater depending on contaminant type and concentrations
- Differentiation between degradation by plant enzymes, rhizosphere microorganisms, and other breakdown process
- Development of engineered solution based on the use of monocultures versus multicultures found in wetlands and terrestrial communities is being further investigated
- Organic contaminants are the main category of contaminants with the highest potential of phytodegradation. Inorganic nutrients are also consumed through plant uptake and metabolism. Phytodegradation outside the plant does not depend on log $K_{ow}$ and plant uptake

(Suthersan 2002).

18.2.4 Phytovolatilization

Phytovolatilization is the uptake and transpiration of contaminants by a plant, with release of the contaminants or a modified form of the contaminants to the atmosphere from the plant. This method occurs as growing tress and other plants take up water, organic, and inorganic contaminants. Some of these contaminants can pass through the plants to the leaves and volatize into the atmosphere at comparatively low concentrations.

Phytovolatilization can be summarized by the following:

- Contaminants could be transformed to less toxic forms (elemental Hg and dimethyl selenite gas)
- The contaminants of hazardous metabolites might accumulate in the variation
- Significant reduction of TCE, TCA, and carbon tetrachloride have been achieved in experimental studies
- Groundwater must be within the influence of the plant; usually tree roots and soil must be able to transmit sufficient water to the plant
- Climatic factors such as temperature, precipitation, humidity, solar radiation, and wind velocity can affect transpiration rates and thus the rate of phytovolatilization
- Improved methods for measuring phytovolatilization, diurnal, and seasonal variations, and precipitation versus groundwater use need to be developed

(Suthersan 2002).

18.2.5 Rhizodegradation, phytostimulation or plant assisted Bioremediation

Rhizodegradation is the breakdown of contaminants in the soil through microbial activity enhanced by the presence of rhizosphere. This is also referred to as phytostimulation, rhizosphere, biodegradation, or plant assisted bioremediation.

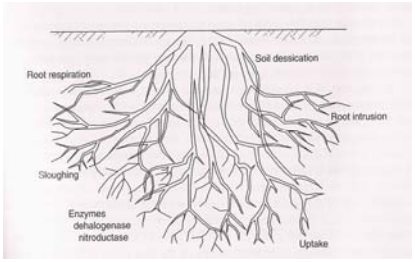


**Figure 41 Rhizodegradation Process (Suthersan, S. 2002)**

Microorganism such as yeast, fungi or bacteria consumes, degrade, or transform organic substances for the use as nutrient substances. Rhizodegradation is aided by the way plants loosen the soil and transport oxygen and water to that area. Plants also enhance biodegradation by other mechanism such as breaking apart clods and transporting atmospheric oxygen to the root zone.

Different plant species establish different subterranean floras. The differences are attributed to variation in rooting habitats, tissue composition, and excretion products of the plants. The primary root population is determined by the habitat created by the plant.

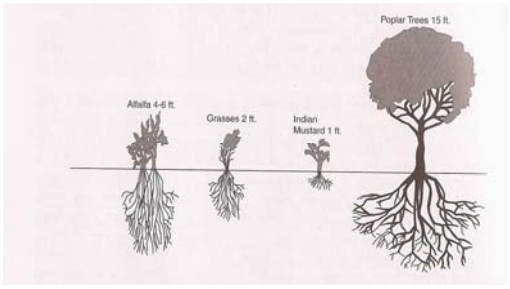


**Figure 42 Root Depths (Suthersan, S. 2002)**

The secondary flora depends on the activities of the initial populations. The age of the plant also factors in to the alterzation in the microbial population in the rhizosphere. These different fungi grow in association with the plant have unique enzymatic pathways, similar to white root fungus enzymes that help to degrade the organics that could be transformed solely bacteria. Typical microbial population in rhizosphere comprise: $5*10^6$ bacteria, $9*10^5$ actinomycetes, and $2*10^3$ fungi per gram of air dried soil.

Rhizodegradation can be summarized by the following:

- Contaminant degradation can be achieved in situ
- Various microorganisms' species and enzymes have been isolated which degrade different contaminants
- Translocation of the contaminant to the plant or atmosphere is less likely than other phytoremediation techniques since degradation takes place at the source of the contamination
- There are low installations and maintenance costs since there is no harvesting and disposal
- Analytic methods to better quantify treatment efficient and success are improving field management techniques for nutrients, water, and plant selection are advancing

(Suthersan 2002)

18.2.6 Rhizofiltration

Rhizofiltration is the adsorption or the precipitation of contaminants into plants roots or the adsorption of contaminants into the roots when contaminants are in solution

surrounding the root zone. In some uses, the plants are raised in greenhouses with their roots in water rather than in soil. Once a large enough root system is developed, the contaminated water is diverted and brought in contact with the plants and are moved and floated into the contaminated water. The goal is that either plant uptake, concentration, and or translocation might occur depending on the specific contaminants. This process is two-fold. First, the contaminant is contained either by immobilization, accumulation, or within a plant. And the final step is that the contaminants are then removed by removing the plant (Suthersan 2002).

**Table 38 Types of Phytoremediation Systems (Chappell, J. 1997)**

| Treatment Method | Mechanism | Media |
|---|---|---|
| Rhizofiltration | Uptake of metal in plant roots | Surface water and water pumped through troughs |
| Phytotransformation | Plant uptake and degradation of organics | Surface water and groundwater |
| Plant Assisted Bioremediation | Enhanced microbial degradation in the rhizosphere | Soils, groundwater within the rhizosphere |
| Phytoextraction | Uptake and concentration of metals via direst uptake into plant tissue with subsequent removal of the plants | Soils |
| Phytostabilization | Root exudates causes metal to precipitate and become less bioavailable | Soils, groundwater, mine tailing |
| Phytovolatilization | Plant evapotranspirates selenium, mercury, and volatile organics | Soil and groundwater |
| Removal of organics from the air | Leaves take up volatile organics | Air |
| Vegetative Caps | Rainwater is evaprtranspirated by plants to prevent leaching contaminants from disposal sites | Soils |

There are an abundance amount of treatability methods that phytoremdiation can be applied to. The most common in all of these cases is the use of plants to treat the contamination problem (Chappell 1997). It is essential to note that the optimal implementation of this newer technology of phytoremediation is crucial for this technique to gain a broader audience. Most of the phytoremediation elements are widely

recognized, but for those segment that aren't, additional research and development should be carried out to:

1. Obtain a better understanding of mechanism of uptake, transport, and accumulation of contaminants
2. Improve collection and genetic evaluation of hyperaccumulating plants
3. Obtain better understanding of interaction in the rhizosphere interactions among plant roots

(Suthersan 2002)

18.3 General Design Factor Phytoremdiation

The design of the phytoremdiation blueprint greatly depends on the specific site and its characteristics. However, there are design considerations that could be used for all methods including the following:

1. Contaminant Levels
2. Treatability
3. Irrigations
4. Agronomic Inputs (P, N, K, salinity, zinc)
5. Maintenance
6. Groundwater capture zone and transpiration rate
7. Contaminant uptake rate
8. Clean up time required
9. Plant Selections

When considering specific plants for the use of the design, plants with high amounts of biomass, produce exudates, grows quickly, have long growing seasons, have

roots that extend to the depth of the contaminants, and have a high tolerance for concentrated contaminants (LaGrega et al 2001).

Useful biomass consists of parts of the plant that are available for evapotranspiration, which includes the leaf surface area and the plant root system. Trees are also utilized in this process because of their large biomass and their depth of root penetration. These roots are more beneficial because they may be able to reach a shallow saturated zone (LaGrega et al 2001). Plants that are currently being researched for use in heavy metal treatment are:

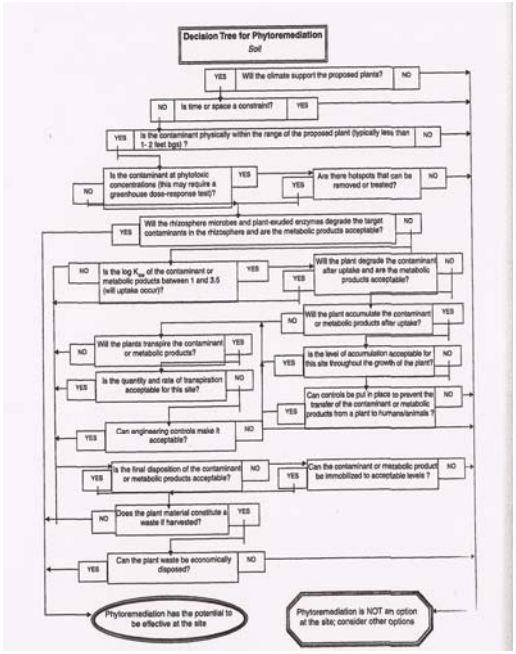


**Figure 43 Decision Tree for Phytoremediation of Soils (Suthersan, S. 2002)**

**Table 39 Types of Remediation Plants**

**Indian Mustard (Brassica Juncea)**

**Barely (Hordeum Vulgare)**

**Oats (Avena Sativa)**

**Alfalfa (*Medicago Sativa*)**

Other studies yielded the following:

**Table 40 Summary of Recent Field Applications Involving Lead (Chappell, J. 1997)**

| Site | Contractor / Vendor | Type of Application | Initial Contaminant Concentrations | Performance / Contaminant Removal | Site Conditions | Plant Species/Number of Crops |
|---|---|---|---|---|---|---|
| Open Burn / Open Detonation Area at the Ensign-Bickford | Edenspace Systems Corporation | Phytoextraction / Phytostabilization | **Area 1:** 500 to 5000 mg/kg<br>**Area 2:** 125 to 1250 mg/kg<br>**Area 3:** 500 to 2000 mg/kg<br>**Area 4:** 750 to 1000 mg/kg<br>**Area 5:** 6.5 to 7.5mg/kg | Total soil lead level concentrations were reduced from 635 mg/kg to 478 mg/kg<br>Average plant uptake was 1000 mg/kg | Soil consisted of a silt loam<br>Soil pH ranged from 6.5 to 7.5 | Brassica Juncea (Indian Mustard) first crop<br>Helianthus Annus (Sunflower) second crop |
| Confidential Superfund Site | Not Reported | Phytoextraction | Total soil lead contamination average 55480 mg/kg, with a maximum value of 140, 500 mg/kg | Growth chambers were used to assess some of the plant species abilities to uptake lead<br>Taraxacum Officinale extracted 1059 mg/kg of lead for the first crop and 921 mg/kg for the second crop<br>Ambrosia Artemisiifola (rag | Soil was alkaline (pH ranged from 7.5 to 8.1) | Agrostemma gilthago<br>Planago rugelii<br>Alliaria officinalis<br>Taraxacum officinale<br>Ambrosia artemisiifola (ragweed)<br>Acer rubrum (red maple) |
| Confidential Dump Site for Lead Acid Batteries | Not Reported | Phytoextraction | Total soil lead contamination average 29400 mg/kg, with a maximum value of 112, 500 mg/kg | Lead concentration of 1695 mg/kg were found in Ambbrosia artemisiifola (ragweed) | Ground cover of more than 85% | Secondary Growth:<br>Acer rubrum (red maple)<br>Rosa multiflora (multiflora rose)<br>Ambrosia artemisiifola (garweed)<br>T. officinale (dandelion)<br>Alliaria officinalis (garlic mustard) Plant |
| Open Burn / Open Detonation Area at the Ensign-Bickford | Edenspace Systems Corporation | Phytoextraction / Phytostabilization | **Area 1:** 500 to 5000 mg/kg<br>**Area 2:** 125 to 1250 mg/kg<br>**Area 3:** 500 to 2000 mg/kg<br>**Area 4:** 750 to 1000 mg/kg<br>**Area 5:** 6.5 to 7.5mg/kg | Total soil lead level concentrations were reduced from 635 mg/kg to 478 mg/kg<br>Average plant uptake was 1000 mg/kg | Soil consisted of a silt loam<br>Soil pH ranged from 6.5 to 7.5 | Brassica Juncea (Indian Mustard) first crop<br>Helianthus Annus (Sunflower) second crop |
| Confidential Superfund Site | Not Reported | Phytoextraction | Total soil lead contamination average 55480 mg/kg, with a maximum value of 140, 500 mg/kg | Growth chambers were used to assess some of the plant species abilities to uptake lead<br>Taraxacum Officinale extracted 1059 mg/kg of lead for the first crop and 921 mg/kg for the second crop<br>Ambrosia Artemisiifola (rag | Soil was alkaline (pH ranged from 7.5 to 8.1) | Agrostemma gilthago<br>Planago rugelii<br>Alliaria officinalis<br>Taraxacum officinale<br>Ambrosia artemisiifola (ragweed)<br>Acer rubrum (red maple) |
| Confidential Dump Site for Lead Acid Batteries | Not Reported | Phytoextraction | Total soil lead contamination average 29400 mg/kg, with a maximum value of 112, 500 mg/kg | Lead concentration of 1695 mg/kg were found in Ambbrosia artemisiifola (ragweed) | Ground cover of more than 85% | Secondary Growth:<br>Acer rubrum (red maple)<br>Rosa multiflora (multiflora rose)<br>Ambrosia artemisiifola (garweed)<br>T. officinale (dandelion)<br>Alliaria officinalis (garlic mustard) Plant |

| Site | Contractor / Vendor | Type of Application | Initial Contaminant Concentrations | Performance / Contaminant Removal | Site Conditions | Plant Species/Number of Crops |
|---|---|---|---|---|---|---|
| Bayonne, New Jersey | Edenspace Systems Corporation | Phytoextraction with EDTA | **Surface Soil:** (0-15 cm):1000 to 6500 mg/kg<br>**Average:** 2055 mg/kg<br>**Subsurface Soil:** (15-30 cm): 780-2100 mg/kg<br>**Average:** 1280 mg/kg | Soil lead levels were reduced on surface soils from 2300 to 420 mg/kg<br>Soil lead levels were reduced on subsurface soils from 1280 to 992 mg/kg | Soil was alkaline (pH=7.9)<br>Soil consisted of sandy loam | Brassica Juncea (Indian Mustard)<br>Three (3) crops were grown and harvested |
| Dorchester, Maine | Edenspace Systems Corporation | Phytoextraction with EDTA | **Surface Soil:** (0-15 cm):640 to 1900 mg/kg<br>**Average:** 984 mg/kg<br>**Subsurface Soil:** (15-30 cm):<br>**Average:** 538 mg/kg | Total soil lead level concentrations were reduced from, ad average of 984 mg/kg to 644 mg/kg in the surface soil<br>Lead levels increased slightly from 538 mg/kg to 671 mg/kg in the subsurface soils | Soil was acidic (pH ranged from 5.1 to 5.9)<br>Soil consisted of sandy loam | Brassica Juncea (Indian Mustard)<br>Three (3) crops were grown and harvested |
| Trenton, New Jersey | Edenspace Systems Corporation | Phytoextraction with EDTA | Lead contamination ranged from 200 to 1800 mg/kg | Total soil lead levels were reduced 13% on surface from 429 mg/kg to 373 mg/kg<br>Soils that exceeded 600 mg/kg of lead were reduced to 539 mg/kg. A difference of 21% | Soil pH ranged from 5.1 to 7.1 | Brassica Juncea (Indian Mustard)<br>Three (3) crops were grown and harvested |
| Twin Cities Army Ammunition Plant (TCAAP); Site C and Site 129-3 | US Army Environmental Center | Phytoextraction with EDTA and acetic acid | **Site C:** Averaged 2610 ppm in the subsurface soil<br>**Site 129-3: Averaged 358 ppm in the subsurface soil** | Results were not s good as expected.<br>Corn only averaged lead concentrations of .65% and .13% (dry weight)<br>White mustard was very low, averaging .083% and .034 (dry weight) of lead | Soil had a high sand content<br>Average annual temperature was 49.6°F | Zea Mayes (corn) first crop<br>Brassica (White Mustard) second crop |

18.4 Specific Phytoremediation Design Factors

- Topography
- Appropriateness of planting mixed stands or single species (monoculture) stands
- Determine the synergetic or adversarial effects in mixed stands
- Appropriate spacing for planting (allowing for plant growth)
- Planting depth
- Degree of plant root penetration
- Growth rates of plant in various levels of contaminates
- Ability of plants to control water infiltration
- Potential impact of natural plant success (the evolution through pioneer species from grasses to scrubs to tress)

(LaGrega et al 2001)

18.4.1 Monitoring Plan

The monitoring plan for this method should include erosion control evapotranspiration, the effectiveness of degradation (contaminant reduction), and the process of succession. The erosion control can be measured by the presence or absence or airborne particulate emissions and by the quality of water runoff (LaGrega et al 2001).

18.4.2 Cost

Phytoremediation is growing to be a cost effective alternative to high energy and high cost methods. A study conducted involving one acre of sandy loam soil with a contaminated depth of 50 cm with plants was estimated at $60,000 - $100,000 compared to $400,000 for traditional excavation and disposal procedures (Chappell, J 1997).

There were few if any studies in this magnitude in Miami-Dade County as it pertaining to this type of technologies with respect to cost. However, in conducting various literature searches, the following was obtained:

**Table 41 Estimates of Phytoremediation versus Established Technologies Cost (Chappell, J 1997)**

| Contaminants | Phytoremediation Costs | Estimated Cost Using Other Technologies |
|---|---|---|
| Metals | $80 per cubic yard | $250 per cubic yard |
| Site contaminated with petroleum hydrocarbons (site size not disclosed) | $70, 000 | $850, 000 |
| 10 acres lead contaminated land | $500,000 | $12 million |
| Radionuclide in surface water | $2 to $ 6 per thousand gallons treated | none listed |
| 1 hectare to a 15 cm depth (various contaminants) | $2,500 to $15,000 | none listed |

18.5 Physiochemical Treatability applications

18.5.1 Stabilization and Solidification

Stabilization and solidification has been widely used in the management of hazardous waste. These technologies are widely used in the treatment of industrial wastes, the treatment of waste prior to secure landfill disposal, and the treatment of contaminated land where large quantities of soil containing contaminants are encountered (LaGrega et al 2001).

Stabilization is the process where additive are mixed with waste to minimize the rate of contaminant migration from the waste and to reduce the toxicity of waste and its hazardous constituents into a form that minimizes the rate of contaminate migration into the environment. It can also reduce the level of toxicity. Stabilization is accomplished through the addition of regents that:

- Improve the handling of the physical characteristics of waste
- Decrease the surface area across where transfer is loss or contamination can occur
- Limit the solubility of any pollutants contained in the waste
- Reduce the toxicity of the contaminants

(LaGrega et al 2001).

Solidification is a process where solidifying material, including solids, are added to the waste to result in a solidified mass. Solidifying the mass is accomplished through the addition of regents that increase the strength, but decreases the permeability, and compressibility of the waste (LaGrega et al 2001).

As a result of this physiochemical method the waste would be both reduced in its toxicity and mobility as well as to improve the engineering properties of the stabilized property. Stabilization and solidification is used interchangeably (LaGrega et al 2001).

18.5.1.1 Stabilization and Solidification Application

The three primary areas of the application for stabilization and solidification technologies are:

- Land Disposal
- Site Remediation
- Solidification of Industrial Waste

**LAND DISPOSAL**

Currently, US regulation bans the land disposal of liquid waste which increases the migration of contaminants. Wet sludge and liquid waste must be stabilized before being added to a landfill (LaGrega et al 2001).

**SITE REMEDIATION**

The remediation of contaminated sites having organic wastes, inorganic, wastes, and or contaminated soils may be accomplished by employing differing stabilization techniques.

The remediation of contaminated sites having organic wastes, inorganic, wastes, and or contaminated soils may be accomplished by employing differing stabilization techniques.

For sit remediation, stabilization is used to:

1. Improve the handling and physical characteristics of the wastes
2. Decrease the rate of the contaminants to migration by decreasing the surface area across which the transfer of pollution can occur

3. Limiting the solubility of pollutants to reduce the toxicity of certain contaminants

Stabilization is often termed a permanent remedial solution. This is often best utilized for sites where the hazards involve large quantities of soils contaminated at low levels. In many instances it may not be economically feasible or environmental sound to excavate, transport, and landfill soils contaminated with low level of pollutants (LaGrega et al 2001).

**SOLIDIFICATION OF INDUSTRIAL WASTE**

A wide variety of organic and inorganic industrial waste can be found in pits, pound, and lagoons because of bad past waste management practices. Solidification improves the engineering properties and may include the rate at which contaminants migrate into the environment. Many of these materials are frequently structurally unstable, aesthetically unsuitable, and their condition precludes other uses of the site area. (LaGrega et al 2001).

18.6 Remediation

18.6.1 Soil Washing

Contaminant sediments have been identified as one of the largest potential risks to water quality and the aquatic environment. Because of this non-point source of pollution, soil washing techniques has been applied (LaGrega et al 2001).

Soil Washing's objective is to separate contaminated solvents into two output stream. One stream is contaminated and the other stream is clean. As a result of this process, the concentration is reduced, producing a reduction of volume of the contaminated material. This soil washing may be done with water, aqueous extractive agents, solvents, or even air. The washing may take place on the entire soil matrix or on

selected portions that contains the contaminants that are separated from the clean portion by fractionation (LaGrega et al 2001).

As a result of surface changes associated with clay particles, inorganic contaminants are associated with the finer fraction of the soil matrix as opposed to the organics falling under humic matrixes. This separation leaves the remaining soil clean (LaGrega et al 2001).

18.6.2 Design Process Factor

**BERGMAN PROCESS**

The contaminated soils should be at least 60 percent course and the organic content should be no more than 20 percent. This is important in keeping with the separation of the soil into fractions of density and grain size differences within the soil matrix. Water is the added to the soil and then directs the slurry through a series of separating devices. Trimmer units are used to separate material coarsest than 6 mm, cyclone separators are for the removal of particles smaller than 45μm, and a dense media separator is used to remove surficial contaminants separated from the coarse fraction. A partition dewatering screen is used to recover the washed coarse materials and humic substances from their slurries, and a flocculation clarifier is used to separated the contaminated fines from the fines slurry stream (LaGrega et al 2001).

# APPENDIX B
# RISK ASSESSMENT CALCULATIONS

Risk Assessment Calculations

**Table 42 Distribution of Lead Analysis Results in Different Media (Gasana and Chamorro 2002)**

| Medium | HUD/EPA Standard | Sum | Mean | SD | Median | Mode | Maximum | Minimum | Range |
|---|---|---|---|---|---|---|---|---|---|
| Air (µg/$m^3$) (n=121) | 15 | 17 | 0.14 | 0 | 0.08 | 0.06 | 1.36 | 0 | 1.36 |
| Water Plug (ppb) (n=120) | 15 | 514 | 4.25 | 15 | 1 | 1 | 150 | 1 | 149 |
| Water Flow (ppb) (n=120) | 15 | 214 | 1.77 | 3 | 1 | 1 | 34 | 1 | 33 |
| Floor Dust (µg/$ft^2$) (n=121) | 40 | 1,667 | 13.77 | 20 | 8.3 | 13 | 150 | 0.8 | 149 |
| Window Sill (µg/$ft^2$) (n=121) | 250 | 11,709 | 96.77 | 417 | 11 | 17 | 3,500 | 0.69 | 3,499 |
| Window well (µg/ft2)(n=118) | 400 | 127,583 | 1054.4 | 7,248 | 17 | 120 | 78,000 | 4 | 7,796 |
| Soil (ppm) (n=121) | 400 | 33,283 | 275 | 315 | 153 | 25 | 1,612 | 25 | 1,587 |

## CONVERSIONS

**Air** $\frac{\mu g}{m3} = pptr$ **Water Plug** = ppb **Soil** = $ppm = \frac{mg}{L}$

**Table 43 Soil Lead Concentrations (Gasana and Chamorro 2002)**

| Concentration (ppm) Minimum | Concentration (ppm) Mean | Concentration (ppm) Maximum |
|---|---|---|
| 25 | 275 | 1612 |

## TOXICITY SCORES

Noncarcinogens

$$TS = C_{MAX} / MCL$$

*where:* TS = Toxicity Score

$C_{MAX}$ = Maximum Concentration $\frac{mg}{L}$ and MCL = Maximum Contaminant Level $\frac{mg}{L}$

$$\mathbf{C_{MIN}} = 25\frac{mg}{L}, \mathbf{C_{MEAN}} = 275\frac{mg}{L}, \mathbf{C_{MAX}} = 1612\frac{mg}{L}$$

$$\mathbf{MCL} = \text{Maximum Contaminant Level } .0015\frac{mg}{L}$$

$$\mathbf{TS_{MIN}} = \frac{25\,\frac{mg}{L}}{.0015\,\frac{mg}{L}} = 16{,}666.7$$

$$\mathbf{TS_{MEAN}} = \frac{275\,\frac{mg}{L}}{.0015\,\frac{mg}{L}} = 183{,}333.3$$

$$\mathbf{TS_{MAX}} = \frac{1612\,\frac{mg}{L}}{.0015\,\frac{mg}{L}} = 1{,}074{,}666.7$$

**Table 44 Toxicity Score**

| Toxicity Score | |
|---|---|
| Concentration mg/L | Result |
| 25.0 | 16,666.7 |
| 275.0 | 183,333.3 |
| 1,612.0 | 1,074,666.7 |

**ADMINISTERED DOSE**

**Chronic Daily Inhalation**

Intake $_{\text{Inhalation}}$

$$I = \frac{C * CR * EF * ED * RR * Abs}{BW * AT}$$

where:

I = Intake (mg/kg of body weight*day)

C = Concentration at exposure point (mg/L in water or mg/m$^3$ in air)

CR = Contact rate (L/day or m$^3$/day)

EF = Exposed frequency (days/year)

ED = Exposed Duration (Years); For residential exposures, a default value of ED = 30 years is typically used.

RR = Retention rate (decimal fraction); A conservative approach would assume the RR and Abs into the bloodstream would be equal to 100% or 1.0.

Abs = Absorption into bloodstream; A conservative approach would assume the RR and Abs into the bloodstream would be equal to 100% or 1.0.

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

**Table 45 Parameters (LeGrange et. al 2001)**

| Parameters | Adults | Child Age (2-6) | Child Age (6-12) |
|---|---|---|---|
| Average Body Weight (kg) | 70 | 16 | 29 |
| Skin Surface Area ($cm^2$) | 18150 | 6980 | 10470 |
| Water Ingested (L/day) | 2 | 1 | 2 |
| Air breathed ($m^3$/h) | 0.83 | 0.25 | 0.46 |
| Retention Rate inhaled air) | 100% | 100% | 100% |
| Absorption Rate (inhaled air) | 100% | 100% | 100% |
| Soil Ingested (mg/day) | 100 | 200 | 100 |
| Bathing Duration (minutes) | 30 | 30 | 30 |
| Exposure Frequency (days/year) | 365 | 365 | 365 |
| Exposure Duration (year) | 30 | 4 | 6 |

**Air Breathed Calculations**

**Child Age (2-6)**

$$CR= (0.25\frac{m3}{h})* (\frac{24h}{day}) = 6\frac{m3}{day}$$

**Child Age (6-12)**

$$CR= (0.46\frac{m3}{h})* (\frac{24h}{day}) = 11.04\frac{m3}{day}$$

**Adult**

$$\text{CR} = (0.83\frac{m3}{h}) * (\frac{24h}{day}) = 19.92\frac{m3}{day}$$

**Table 46 Lead Concentration Levels**

| Concentration (ppm) Minimum | Concentration (ppm) Mean | Concentration (ppm) Maximum |
|---|---|---|
| 25 | 275 | 1612 |

$$\text{I} = \frac{C * CR * EF * ED * RR * Abs}{BW * AT}$$

$$\mathbf{C_{MIN}} = 25\frac{mg}{L}, \mathbf{C_{MEAN}} = 275\frac{mg}{L}, \mathbf{C_{MAX}} = 1612\frac{mg}{L}$$

**Child Age (2-6)**

$$\text{I} = \frac{(25\frac{mg}{m3}) * (6\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(16kg) * (365days)}$$

$$\text{I} = 281.25\frac{mg}{kg * days}$$

$$\text{I} = \frac{(275\frac{mg}{m3}) * (6\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(16kg) * (365days)}$$

$$\text{I} = 3{,}093.75\frac{mg}{kg * days}$$

$$\text{I} = \frac{(1612\frac{mg}{m3}) * (6\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(16kg) * (365days)}$$

$$\text{I} = 18{,}135\frac{mg}{kg * days}$$

**Child Age (6-12)**

$$I = \frac{(25\frac{mg}{m3}) * (11.04\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(29kg) * (365days)}$$

$$I = 285.52\frac{mg}{kg * days}$$

$$I = \frac{(275\frac{mg}{m3}) * (11.04\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(29kg) * (365days)}$$

$$I = 3{,}140.69\frac{mg}{kg * days}$$

$$I = \frac{(1612\frac{mg}{m3}) * (11.04\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(29kg) * (365days)}$$

$$I = 18{,}467.25\frac{mg}{kg * days}$$

**Adult**

$$I = \frac{(25\frac{mg}{L}) * (19.92\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(70kg) * (365days)}$$

$$I = 213.43\frac{mg}{kg * days}$$

$$I = \frac{(275\frac{mg}{L}) * (19.92\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(70kg) * (365days)}$$

$$I = 2{,}347.71\frac{mg}{kg * days}$$

$$I = \frac{(1612\frac{mg}{L}) * (19.92\frac{m3}{day}) * (365\frac{days}{year}) * (30 years) * (1.0) * (1.0)}{(70kg) * (365days)}$$

$$I = 13{,}804.56\frac{mg}{kg * days}$$

**Table 47 Chronic Daily Inhalation Intake**

| | Inhalation Intake (mg/kg*days) | | |
|---|---|---|---|
| | **$C_{MIN}$ 25 (mg/L)** | **$C_{MEAN}$ 275 (mg/L)** | **$C_{MAX}$ 1612 (mg/L)** |
| **Child (2-6)** | 281.3 | 3,093.8 | 18,135.0 |
| **Child (6-12)** | 285.5 | 3,140.7 | 18,467.3 |
| **Adult** | 213.4 | 2,347.7 | 13,804.6 |

**DAILY INTAKE (AVERAGE) FROM DERMAL CONTACT WITH SOIL**

$$I_N = \frac{C * A * DA * Abs * SM * ED}{BW * AT}$$

where:

$I_N$ = Intake

A = Skin Exposed = 20 % ($cm^2$)

DA = Dust Adherence = $0.51\frac{mg}{cm2}$

Abs = Skin Absorption Rate 6%

SM = Effect of Soil Matrix = 15% (because of the soil matrix, only 15% of contamination is actually available for contact)

EF = Two Exposure events per day; 156 exposure days per year

ED = 1 Year

BW = Body Weight (kg)

AT = Averaging Time (days)

(LaGrega et al 2001)

$$I_N = \frac{C * A * DA * Abs * SM * ED}{BW * AT}$$

$$\mathbf{C_{MEAN\ (SOIL)}} = 275 \frac{mg}{L}$$

**Child (Age 2-6)**

A= (.20) * (6980 $cm^2$) = 1396 $cm^2$

$$I_N = \frac{275(\frac{mg}{m3}) * (1396cm2) * (.51\frac{mg}{cm2}) * (.06) * (.15\frac{2\,\text{exposure}}{day}\frac{events}{}) * (\frac{156days}{year}) *}{(16kg) * (365days)} 10^{-6}$$

$$\frac{kgsoil}{mgsoil}$$

$$I_N = 4.707 * 10^{-5}\ \frac{mg}{kg * day}$$

$$I_N = 4.7 * 10^{-5}\ \frac{mg}{kg * day}$$

**Child (Age 6-12)**

A= (.20) * (10470 $cm^2$) = 2094 $cm^2$

$$I_N = \frac{275(\frac{mg}{m3}) * (2094cm2) * (.51\frac{mg}{cm2}) * (.06) * (.15\frac{2\,\text{exposure}}{day}\frac{events}{}) * (\frac{156days}{year}) *}{(29kg) * (365days)} 10^{-6}$$

$$\frac{kgsoil}{mgsoil}$$

$$I_N = 3.8954 * 10^{-5}\ \frac{mg}{kg * day}$$

$$I_N = 3.9 * *10^{-5} \ \frac{mg}{kg * day}$$

A= (.20) * (18150 $cm^2$) = 3630 $cm^2$

**Adult**

$$I_N = \frac{275(\frac{mg}{m3}) * (3630cm2) * (.51\frac{mg}{cm2}) * (.06) * (.15\frac{2\exp osure}{day}\frac{events}{}) * (\frac{156days}{year}) *}{(70kg) * (365days)} 10^{-6}$$

$$\frac{kgsoil}{mgsoil}$$

$$I_N = 2.7976*10^{-5} \ \frac{mg}{kg * day}$$

$$I_N = 2.8*10^{-5} \ \frac{mg}{kg * day}$$

**Table 48 Daily Dermal Intake**

| Daily Intake (Average) from Dermal Contact with Soil (mg)/(kg*days) | |
|---|---|
| **Child (2-6)** | $4.7*10^{-5}$ |
| **Child (6-12)** | $3.9*10^{*-5}$ |
| **Adult** | $2.8*10^{-5}$ |

**RISK CHARACTERIZATION**

The Oral RfD is listed below:

Hazard Index

$$HI = \frac{In}{RfC}$$

*where:*

HI = Hazard Index (dimensionless)

$I_N$ = Chronic daily intake of NonCarcinogen $\frac{mg}{kg * day}$

MCL = Maximum Contaminant Level $\frac{mg}{L}$

If the acceptable level of intake is equal to the reference dose, then a hazard index less than 1 is acceptable.

(LaGrega et al 2001)

$$HI = \frac{In}{RfC}$$

**CHILD (AGE 2-6)**

$HI = \frac{.000047}{.0015}$ Unit less

HI = .03133

**CHILD (AGE 6-12)**

$HI = \frac{.000039}{.0015}$

HI = .0260

**Adult**

$HI = \frac{.000028}{.0015}$

HI = .0187

**Table 49 Hazard Index**

| Hazard Index | |
|---|---|
| **Child (2-6)** | 0.0313 |
| **Child (6-12)** | 0.026 |
| **Adult** | 0.0187 |

## CANCER RISKS ON A POPULATION

**Table 50 Chronic Daily Inhalation Intake**

| | Inhalation Intake (mg/kg*days) | | |
|---|---|---|---|
| | **$C_{MIN}$ 25 (mg/L)** | **$C_{MEAN}$ 275 (mg/L)** | **$C_{MAX}$ 1612 (mg/L)** |
| **Child (2-6)** | 281.3 | 285.5 | 213.4 |
| **Child (6-12)** | 3,093.8 | 3,140.7 | 2,347.7 |
| **Adult** | 18,135.0 | 16,467.3 | 13,804.6 |

## POPULATIONS

### Children (Age 2-6)

Population 100,000 Children

Children Weight = 16 kg

$\text{Intake}_{\text{Child (2-6) MIN}} = 281.3\,\frac{mg}{kg * day}$, $\text{Intake}_{\text{Child (2-6) MEAN}} = 3{,}093.8\,\frac{mg}{kg * day}$, $\text{Intake}_{\text{Child (2-6) MAX}} = 18{,}135\,\frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\,\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (2-6) MIN}} = 281.3 \frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MIN}} = (.0015 \frac{mg}{L}) \ (\frac{281.3 \frac{mg}{kg * day}}{16kg}) = .0264 \frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MIN}}$ = (.0264) * 100%

Individual Cancer Risk $_{\text{MIN}}$ = 2.63%

**Maximal Cases** $_{\text{MIN}}$

Maximum Cases $_{\text{MIN}}$ = (Risk) * (Exposed Population)

= (.0264)* (100,000)

= 2,640

**Excess Lifetime Cancer Cases** $_{\text{MIN}}$ = 2,640

Population 100,000 Children

Children Weight = 16 kg

Intake $_{\text{Child (2-6) MIN}} = 281.3 \frac{mg}{kg * day}$, Intake $_{\text{Child (2-6) MEAN}} = 3{,}093.8 \frac{mg}{kg * day}$, Intake $_{\text{Child (2-6) MAX}} = 18{,}135 \frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015 \frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (2-6) MEAN}} = 3{,}093.8\frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MEAN}} = (.0015\frac{mg}{L})\ (\frac{3{,}093.8\frac{mg}{kg * day}}{16kg}) = .2900\frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MEAN}}$ = (.2900) * 100%

Individual Cancer Risk $_{\text{MEAN}}$ = 29%

**Maximal Cases** $_{\text{MEAN}}$

Maximum Cases $_{\text{MEAN}}$ = (Risk) * (Exposed Population)

= (.2900) * (100,000)

= 2,900

**Excess Lifetime Cancer Cases** $_{\text{MEAN}}$ = 2,900

Population 100,000 Children

Children Weight = 16 kg

Intake $_{\text{Child (2-6) MIN}} = 281.3\frac{mg}{kg * day}$, Intake $_{\text{Child (2-6) MEAN}} = 3{,}093.8\frac{mg}{kg * day}$, Intake $_{\text{Child (2-6) MAX}} = 18{,}135\frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (2-6) MAX}}$ = $18{,}135\frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MAX}}$ = $(.0015\frac{mg}{L})\ (\frac{18{,}135\frac{mg}{kg * day}}{16kg}) = 1.7002\frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MAX}}$ = (1.7002) * 100%

Individual Cancer Risk $_{\text{MAX}}$ = 170%

**Maximal Cases** $_{\text{MAX}}$

Maximum Cases $_{\text{MAX}}$ = (Risk) * (Exposed Population)

= (1.7002) * (100,000)

= 170,015.6

**Excess Lifetime Cancer Cases** $_{\text{MAX}}$ = 170,015.6

**<u>Children (Age 6-12)</u>**

Population 100,000 Children

Children (Age 6-12) 100,000

Children Weight 29 kg

Intake $_{\text{Child (6-12) MIN}}$ = $285.5\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MEAN}}$= $3{,}140.7\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MAX}}$ = $18{,}467.3\frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (6-12) MIN}} = 285.5\frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MIN}} = (.0015\frac{mg}{L})\ (\frac{285.5\frac{mg}{kg * day}}{29kg}) = .0148\frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MIN}}$ = (.0148) * 100%

Individual Cancer Risk $_{\text{MIN}}$ = 1.48 %

**Maximal Cases** $_{\text{MIN}}$

Maximum Cases $_{\text{MIN}}$ = (Risk) * (Exposed Population)

= (.0148) * (100,000)

= 1,476.72

Population 100,000 Children

Children (Age 6-12) 100,000

Children Weight 29 kg

Intake $_{\text{Child (6-12) MIN}} = 285.5\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MEAN}} = 3{,}140.7\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MAX}} = 18{,}467.3\frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (6-12) MIN}}$ = $3{,}140.7\frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MEAN}}$ = $(.0015\frac{mg}{L})\ (\frac{3{,}140.7\frac{mg}{kg * day}}{29kg}) = .1625\frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MEAN}}$ = (.1625) * 100%

Individual Cancer Risk $_{\text{MEAN}}$ = 16.25 %

**Maximal Cases** $_{\text{MEAN}}$

Maximum Cases $_{\text{MEAN}}$ = (Risk) * (Exposed Population)

= (.1625) * (100,000)

= 16,245

Population 100,000 Children

Children (Age 6-12) 100,000

Children Weight 29 kg

Intake $_{\text{Child (6-12) MIN}}$ = $285.5\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MEAN}}$= $3{,}140.7\frac{mg}{kg * day}$, Intake $_{\text{Child (6-12) MAX}}$ = $18{,}467.3\frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{16kg})(\frac{mg}{L})$

Intake $_{\text{Child (6-12) MAX}}$ = $18,467.3\frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MAX}}$ = $(.0015\frac{mg}{L})\ (\frac{18{,}467.3\frac{mg}{kg * day}}{29kg}) = .9552\frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MAX}}$ = (.9552) * 100%

Individual Cancer Risk $_{\text{MAX}}$ = 95.5 %

**Maximal Cases** $_{\text{MAX}}$

Maximum Cases $_{\text{MAX}}$ = (Risk) * (Exposed Population)

= (.9552) * (100,000)

= 95,520.5

**ADULT**

Population 100,000 Adults

Adult Weight 70 kg

Intake $_{\text{ADULT MIN}}$ = $213.4\frac{mg}{kg * day}$, Intake $_{\text{ADULT MEAN}}$ = $2{,}347.7\frac{mg}{kg * day}$, Intake $_{\text{ADULT MAX}}$ =

$$13,804.6\frac{mg}{kg * day}$$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015\frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{79kg})(\frac{mg}{L})$

Intake $_{\text{ADULT MIN}} = 213.4 \frac{mg}{kg * day}$

Individual Cancer Risk $_{\text{MIN}} = (.0015 \frac{mg}{L}) \ (\frac{213.4 \frac{mg}{kg * day}}{70kg}) = .0046 \frac{mg}{L * day}$

Individual Cancer Risk $_{\text{MIN}}$ = (.0046) * 100%

Individual Cancer Risk $_{\text{MIN}}$ = .4573 %

**Maximal Cases** $_{\text{MIN}}$

Maximum Cases $_{\text{MIN}}$ = (Risk) * (Exposed Population)

= (.0046) * (100,000)

= 457.3

Intake $_{\text{ADULT MEAN}} = 2{,}347.7 \frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015 \frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{70kg})(\frac{mg}{kg * day})$

Individual Cancer Risk $_{MEAN} = (.0015 \frac{mg}{L}) \ (\frac{2{,}347.7 \frac{mg}{kg * day}}{70kg}) = .0503$

Individual Cancer Risk $_{MEAN}$ = 5.03.5%

**Maximal Cases**

Maximum Cases $_{MEAN}$ = (Risk) * (Exposed Population)

= (.0503)* (100,000)

= 5,030.8

**Excess Lifetime Cancer Cases** = 5,030.8

Intake $_{ADULT\ MEAN}$ = $13{,}804.6 \frac{mg}{kg * day}$

Slope Factor = Maximum Contaminant Level (MCL) = $.0015 \frac{mg}{L}$

Cancer Risk = $(SlopeFactor)(\frac{Intake}{Weght})$

Individual Cancer Risk = $(\frac{mg}{kg * day})(\frac{1}{70kg})(\frac{mg}{kg * day})$

Individual Cancer Risk $_{MAX} = (.0015 \frac{mg}{L}) \ (\frac{13{,}804.6 \frac{mg}{kg * day}}{70kg}) = .2958$

Individual Cancer Risk $_{MAX}$ = 29.6 5%

**Maximal Cases**

Maximum Cases $_{MAX}$ = (Risk) * (Exposed Population)

= (.2958)* (100,000)

= 29,581.3

**Excess Lifetime Cancer Cases** = 29,581.3

## CANCER RISKS ON A POPULATION

**Table 51 Individual Cancer Risk**

**Individual Cancer Risk**

| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
|---|---|---|---|
| **Child (2-6)** | 0.0264 | 0.2900 | 1.7002 |
| **Child (6-12)** | 0.0148 | 0.1625 | 0.9552 |
| **Adult** | 0.0046 | 0.0503 | 0.2958 |

**Table 52 Individual Cancer Risk Percentage**

**Individual Cancer Risk Percentage**

| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
|---|---|---|---|
| **Child (2-6)** | 2.64% | 29.00% | 170.02% |
| **Child (6-12)** | 1.48% | 16.25% | 95.52% |
| **Adult** | 0.46% | 5.03% | 29.58% |

**Table 53 Excess Lifetime Cancer Cases**

**Excess Lifetime Cancer Cases**

| | Individual Cancer Risk $_{MIN}$ | Individual Cancer Risk $_{MEAN}$ | Individual Cancer Risk $_{MAX}$ |
|---|---|---|---|
| **Child (2-6)** | 2,640.0 | 29,004.4 | 170,015.6 |
| **Child (6-12)** | 1,476.7 | 16,245.0 | 95,520.5 |
| **Adult** | 457.3 | 5,030.8 | 29,581.3 |

# APPENDIX C
# ENVIRONMENTAL ENGINEERING CONTROL MEASURES

**CONVERSIONS**

1 hectare = 2.5 acre

1 acre = 2.5 hectare

1 acre = 43,560 $ft^2$

Average lot size in Miami-Dade County = 3600 $ft^2$

Average lot size in Miami-Dade County = .0826 acre

**Table 54 Estimates Cost (Chappell, J 1997)**

| Option | Contaminants | Phytoremediation Costs | Estimated Cost Using Other Technologies |
|---|---|---|---|
| 1 (Lead) | 10 acres lead contaminated land | $500,000 | $12 million |
| 2 (Various Contaminants) | 1 hectare to a 15 cm depth (various contaminants) | $2,500 to $15,000 | none listed |

(Chappell, J 1997)

**Option 1**

10 acres of lead contaminated land = $500,000

1 acres of lead contaminated land = $50,000

**Option 2**

1 hectare = $2,500

2.5 acre = $6,250

**Option 1**

(.0826 acre)*($50,000) = $4,130

**Option 2**

(.0826 acre)*($6,250) = $516.25

# VITA

## TARLA TAMIA TOOMER

### Education

| | |
|---|---|
| Doctoral Candidate 2008 | Florida International University, Miami, Fl., Civil and Environmental Engineering |
| M.S. 2003 | Florida International University, Miami, Fl., Telecommunications and Networking |
| M.S. 2001 | Florida International University, Miami, Fl., Engineering Management |
| B.S. 2000 | University of Miami, Miami, Fl., Industrial Engineering |

### Honors

Tau Beta Pi Engineering Honor Society-Florida Theta Chapter (ΤΒΠ), Chi Epsilon National Honor Society (XE), Upsilon Pi Epsilon International Honor Society (ΤΠΕ), Order of the Engineer, Delores Auzenne Fellow, National Science Foundation-Florida Georgia Louise Stokes Alliance Scholar

### Professional Experience

| | |
|---|---|
| 2000-2001 Florida Department of Transportation (Contracted (FIU)) | Miami, Fl |
| 2000-2001UM/Jackson Memorial Hospital | Miami, Fl |
| 2002-2003 Miami-Dade Transit (MDT) | Miami, Fl |
| 2003-2007 United States Department of the Interior | Fort Lauderdale, Fl |
| 2007-Present United States Department of Energy | Washington, DC |

### Professional Societies

Tau Beta Pi Engineering Honor Society-Florida Theta Chapter (ΤΒΠ), Chi Epsilon National Honor Society (XE), Upsilon Pi Epsilon International Honor Society (ΤΠΕ), National Society of Black Engineers (NSBE), Florida Engineering Society (FES), American Society of Civil Engineers (ASCE), National Society of Processional Engineers (NSPE)